

Plant Pathology at Washington State University, 1891-1989,  
and Cereal Research at Pullman

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## FOREWARD

Washington became a state, November 11, 1889, and Governor Elisha P. Ferry signed legislation 28 March 1890 establishing the Washington Agricultural College and School of Science, changed in 1891 to "Washington Agricultural College, Experiment Station, and School of Science." At this centennial time we reflect upon our roots.

Major sources of information were the early Annual Reports of the Washington Agricultural Experiment Station (on film in the Owen Science Library, call no. S-0496), and "Plant Pathology in the State of Washington, Past, Present, Future," by former Department Chairman, George W. Fischer (Plant Disease Reporter Supplement 200, pages 42-55, 1951). Information about the university as a whole was obtained from "Historical Sketch of the State College of Washington," by President Enoch Albert Bryan, 1928; and "E. O. Holland and the State College of Washington, 1916-1944," by historian William M. Landeen, 1958.

After the manuscript was complete I read the 1987 M.A. thesis in History, WSU, of Robert W. Hadlow, "A History of the Development of Scientific Research at Washington Agricultural Experiment Station, 1890-1940," and "Creating the People's University, 1890-1990," by historian George A. Frykman, 1990.

The illustrations are from entries to a T-shirt design contest within plant pathology, 1990. Figure 1 is by Cheryl Campbell, Figures 2-3 are by David Christian.

The history is more than a story of the Plant Pathology Department. It contains much about the University as a whole that directly or indirectly affected the fortunes of plant pathology. WSU includes all activities in Washington, not just at Pullman.

I want to thank Jack Rogers and Otis Maloy who read most of it, Gardner Shaw who read much of it and Rollie Line who read the cereal disease section. Jane Lawford typed the manuscript in its entirety, and she was patient during its revisions. I wanted to make it a history of plant pathology in Washington, including forestry at the University of Washington, activities of the Washington Department of Agriculture, Plant Quarantine and Private Consultants. I gave up on this goal and settled upon Washington State University alone.

Included is much about finances, essential as background by which to judge progress within the institution. The first part is a brief history of plant pathology at WSU in general. The second part is a history of cereal research at Pullman, emphasizing the smuts.

## INTRODUCTION

The first part of this history records the arrival and departure of state and federal faculty concerned with plant pathology, first within Botany, later within Plant Pathology. The department at Pullman, the Main Station of the Agricultural Research Center, was static in number of faculty from its inception until about 1945. Until 1945, Washington State University expanded into new activities rather than strengthening existing units. In the beginning (the C. V. Piper and R. K. Beattie periods), plant pathology received its share of research support, considering the broad responsibilities of its few workers. H. B. Humphrey separated pathology from Botany in the experiment station and grouped pathology courses together in the Botany curriculum. I. D. Cardiff returned plant pathology to Botany within the experiment station. F. D. Heald established the Department of Plant Pathology, both within the experiment station and academically, and it has remained an independent department ever since. The teaching load at Pullman was oppressive until about 1946, and during many of the early years it was inequitably distributed among faculty. During the Heald period graduate students often served as full-fledged teachers, increasing the turn-over in the teaching faculty.

If the state had provided three or four full-time career pathologists at Pullman to assist Heald, this department could have become the leading plant pathology department in the nation. Heald was the scholarly equal of any pathologist of his time. The only other long-time state pathologist at Pullman during the Heald era was Dr. Leon K. Jones, an able helper. I blame the expansionist policies of President Holland for the lost opportunity to become a truly great department. For example, Plant Pathology was better supported at times at Puyallup than at Pullman, probably because the budget for the Puyallup station was independent until 1946.

Plant Pathology began to thrive under President Compton. Shortly after he became President of Washington State University (1945) and George W. Fischer became Chairman of the Department of Plant Pathology (1946), the faculty at Pullman began to balance capabilities with expectations, and stability came to the teaching faculty.

The fruit industry developed early with irrigation projects in the Okanogan, Wenatchee, and Yakima Valleys. Completion of Grand Coulee Dam, with eventual delivery of water to the "Columbia Basin" by the Columbia River Project, made irrigated agriculture the most valuable agricultural sector in the state. Plant pathology at Prosser expanded greatly in the 1960's and has remained important ever since. The plant pathology faculty at Wenatchee, which concentrates on apples and pears, has always been small. One plant pathologist has long been housed at Mt. Vernon. Having a single plant pathologist at Mt. Vernon is a questionable practice. Advantages include proximity to local problems and political considerations. Disadvantages include isolation from other pathologists and limited contact with scientists in related disciplines. In any case, the branch station system is firmly established, rooted in the ecological diversity of the state and in politics. One can conjure an image of the facilities of Pullman, Prosser, Puyallup and Wenatchee combined at a single central location. It would make a formidable facility.

It is timely to consider Washington as an agricultural state. It ranks about 20th in farm receipts, animals plus crops, exclusive of government payments, about 12-13th in cash receipts from crops. If total farm produce were the criterion, Washington plant pathology should rank 19-21st; if cash receipts from crops were the criterion, it should rank 12-13th. Receipts from livestock are derived indirectly from the grain, hay, and forage the livestock consume, so I consider total farm income (exclusive of government subsidies) as the best way to judge agricultural strength.

Cash receipts in millions, from farming, 1976 and 1985, an estimate of agricultural strength, excluding government subsidies.

	State	Livestock		Crops		Total	
		1976	1985	1976	1985	1976	1985
1	California	2953	4165	6148	9804	9101	13970
2	Texas	3189	5440	3109	3857	6298	9297
3	Iowa	4041	4811	2967	4389	7009	9200
4	Illinois	1866	2063	4243	5704	6110	7667
5	Nebraska	2176	4112	1690	3093	3867	7205
6	Minnesota	2181	3370	1720	3101	3902	6471
7	Kansas	1917	3263	1614	2477	3531	5741
8	Wisconsin	2486	4099	541	1011	3028	5111
9	Florida	692	1015	1840	3725	2532	4740
10	Indiana	1334	1727	1995	2869	3330	4596
11	Ohio	1163	1510	1617	2429	2781	3940
12	North Carolina	1073	1934	1748	1979	2821	3914
13	Missouri	1577	1930	1053	1737	2630	3668
14	Georgia	1165	1727	1103	1599	2269	3327
15	Arkansas	1059	1825	1237	1454	2296	3280
16	Colorado	1446	2019	530	1144	1976	3163
17	Pennsylvania	1300	2183	500	966	1800	3149
18	South Dakota	1417	1903	372	1075	1789	2979
19	Kentucky	729	1351	896	1519	1626	2870
20	Michigan	815	1231	884	1618	1700	2850
21	Washington	523	932	1231	1865	1754	2797
Rank		21	21	13	12	19	21

How many states above Washington in this ranking are stronger in plant pathology? Make your own estimate. By my calculation Washington has every reason to be proud of its Department of Plant Pathology.

Pullman is in the Palouse Hills at the extreme eastern edge of Washington, 8 miles west of the Idaho border. The Palouse River runs through Pullman, but it is not a beautiful stream. The Moscow Mountains to the east remind us that Pullman is not only near the eastern border of Washington, but it is near the transition from natural grass land to forest. The hills were formed by soil and volcanic ash deposited by wind. The native vegetation was tall grass, but the hills are now occupied by wheat, barley, peas and lentils. The wind-blown soil is free of stones so the combine sickle bar can ride on the soil surface, facilitating harvest of pea and lentil vines. Rainfall is sufficient for the above crops so that irrigation is not commercially used in the vicinity of Pullman. Rotation is minimal and soil fertility is maintained with commercial fertilizers. Soil erosion is the most important long-time problem of the Palouse, and tillage is minimal, with some no-till or direct seeding of crops into stubble, made possible only when herbicides give adequate control of weeds.

Pathologists have access to experimental plots at the Spillman Farm south of town, at the Soil Conservation Farm northwest of town, and on WSU land at the edge of the campus. The department and cooperating USDA together possess farm implements essential for field work, and irrigation water is available at two local sites. Greenhouse and growth chambers are adequate but the growth of molecular biology has made our laboratory space limiting.

The branch stations are distinct experimental units, each with its own character. This feeling is lacking at Pullman. Buildings housing the agricultural departments on the eastern side of the campus blend with the remainder of the university. The most distinctive building on the ag campus is a modified dairy barn converted into the alumni center. The setting of plant pathology on the campus diminishes the influx of farmers seeking direct assistance. Parking is a deterrent.

Classroom facilities in Johnson Hall are excellent, except for the necessity of having to use the virology laboratory of Wyatt for teaching virology. As long as classes are small all can gather around a demonstration or use a particular instrument. All classes are taught at Pullman, except Nematology and Virology Methods which are taught at Prosser. Students and faculty at Pullman have access to the library facilities of the university, including an extensive collection of plant pathology reprints in the George W. Fischer Agriculture Library in Johnson Hall. Heald Hall houses Botany, Johnson Hall houses Plant Pathology and a few other departments. The agricultural branch library is named after Fischer, so three pathologists have been honored in this manner at Pullman.

Department chairs are responsible for tenure and promotion of all faculty at Pullman and they share that responsibility with superintendents of branch stations for faculty not at Pullman. Budgets (operation funds) at branch stations are controlled by station superintendents, so there is shared governance. The system works.

## CHRONOLOGY

### *The Start*

Washington Territory, in the far northwest corner of the United States, developed slowly. Many of the pioneers who ventured this far west settled in the Willamette Valley of Oregon, but population growth accelerated after 1883 when the first railroad reached Washington Territory. Before a territory could become a state, it had to have a state constitution approved by Congress and a sizeable population. The first state constitution, submitted to Congress in 1879, was rejected, possibly because it permitted women to vote. In February 22, 1889, Congress passed legislation enabling Washington to become a state. A new state constitution was approved by the people of Washington on October 1 and Congress accepted it November 11, 1889. President Benjamin Harrison signed it and Washington became the 42nd state.

The vision of early American politicians is remarkable. Most schools of higher education were schools of law, philosophy, medicine, and theology, attended primarily by the elite or wealthy. Congress saw a need for schools that taught skills required to promote agriculture, engineering and commerce, i.e. skills to develop the natural resources and the common people of the nation. In July, 1862, President Lincoln signed the Land-Grant Act. Even the problems of the Civil War did not stop constructive thought and actions by federal legislators.

The Legislature of Washington, in response to the Land-Grant Act, set aside 90,000 acres for agriculture and 100,000 acres for mechanical arts (engineering in a broad sense, including mining and metallurgy) and the sciences, including military science. The Land-Grant institution was established at Pullman. Many land-grant schools were called A. and M. (Oklahoma A and M, Texas A and M, etc.) schools for agriculture and mechanical arts, but Washington State University was never called "A and M". It went by various names, such as the State College of Washington, Washington State College, and finally becoming on September 1, 1959, Washington State University. It was a university long before it was officially designated as one. I will refer to the institution as Washington State University (WSU) throughout, regardless of its name at the time. The legislature finances WSU as it sees fit, largely independent of the land-grant endowment.

Federal vision included the need for agricultural research in order to learn proper ways to farm the soil of the nation, feed the livestock, etc., leading to passage of the Hatch Act, March 2, 1887. This act provided

\$15,000 per year to each state and territory for agricultural research. The Morrill Act of August 30, 1890, initially provided \$15,000, increasing \$1,000 per year, until it reached \$25,000 to support teaching at the land-grant schools. The Morrill Act broadened the A and M concept to include English, mathematics, physical and economic sciences to support agriculture and engineering. According to the 1905 WSU agricultural experiment station annual report, the Adams Act provided \$5,000, increasing gradually to \$15,000 per year, to further support agricultural research. The Adams Act was passed unanimously by Congress, evidence of broad support for the concept of government-supported agricultural research. The Smith-Lever Act of July 1, 1914, was enacted to support agricultural extension. These acts established the tradition of the land-grant institutions; research, teaching, and extension. By law, appropriated funds must be spent according to the intent of the individual acts. Administrators in agriculture must spend Hatch and Adams funds for research, Morrill funds for teaching, and Smith-Lever funds for extension, making accounting more complex within the College of Agriculture than within the rest of WSU.

The goals of the new Agricultural Experiment Station were announced in its first bulletin, published in December, 1891. The goals were unrealistic, far exceeding the capacity of the tiny faculty. They were a "wish list", a guide to the future. Nevertheless some of the early workers took them seriously and tried to accomplish them. Some of the goals included: test cereal varieties; seeding rates, depth, time of planting; heading dates, maturity, straw strength, yield of grain and straw; method of smut control in wheat and oats; fertilizers, manures; test varieties of potatoes, sweet corn, garden peas, lettuce, cabbage, radishes, etc., fruit trees, shade and forest trees, 12 varieties of sugar beets; test insecticides; test animal feeds; record the native flora and fauna, etc. This is only a partial listing, but it suffices to illustrate the desires of those supporting the new experiment station.

John O'B Scobey became Agriculturist and E. R. Lake became Horticulturist in 1891. The Agriculturist had most of the duties of the present departments of Agronomy and Animal Science. The Horticulturist was responsible for vegetables, fruits, forest and shade trees. In 1892, G. C. Hitchcock became Chemist. The Chemist's duties included analysis of fertilizers, manures, soils, foods, feeds, and insecticides.

The land at Pullman was virgin bunch grass sod. It had to be tilled for a year or two before meaningful experiments could be conducted. Horses, livestock, implements, sheds, seeds, personnel, etc., had to be acquired. An artesian well, a reservoir, and piping were constructed in the second year to facilitate caring for the trees, livestock, etc. Working under these conditions, especially with such high expectations for the station, was difficult. Yet, progress was made.

The new faculty had little knowledge of local conditions and no knowledge based on local experiments, yet they were expected to assist and lead local farmers. In some cases this was not difficult. The veterinarian could discuss a disease of horses based on prior knowledge with some confidence. But what about the adaptation of cultivars of apples, wheat, grasses, etc.? This situation was handled skillfully. Within a month of establishing the experiment station, President Lilley and the faculty participated in public meetings called "Institutes". The President and three or four faculty (about all of them) presented talks. After the talks, farmers, grain dealers, etc., related their personal experiences. The entire proceedings, including all contributions from the audience, were recorded and published. Many farmers had lived for years in the region and their experiences were of real value to the new faculty. Farmers, bankers, and agri-business people as well as college faculty were featured speakers.

The only diseases specifically mentioned in the 1891 annual report of the experiment station were oat, wheat, and corn smuts. John Scobey, the Agriculturist, reported that the number and vigor of wheat seedlings was reduced by copper sulfate and hot water. Even though hot water killed smut spores on the wheat seed, it was impractical as a treatment for smut control on the farm.

Washington Agricultural College and School of Science opened its doors to students January 13, 1892. Agriculture, Mechanical Arts, and Domestic Economy (= Home Economics) were 4-year programs. Pharmacy was a 2-year program. Young men were welcomed to Agriculture, Mechanical Arts, and

Pharmacy, young women to Domestic Economy and Pharmacy (Bulletin 1). At a meeting in Colfax, January 30, 1892, President Lilley stated that teaching was the most important function of the college and that a broad education was its goal (Bulletin 2). At a meeting in Pomeroy, May 15, 1892, President Lilley stated that there were no quotas for students by county for admission, and that education was free for all. There was no tuition (Bulletin 5). Dr. Lilley served only one year as president and his successor, Dr. John W. Heston served four months.

There were 21 freshmen, 3 pharmacy students, and 61 students in preparatory status in 1892.

Chaos in financial matters developed quickly. Dr. Lilley had the noble concept of practically free education. Rooms (heat, light, water) in the dormatory were free and food was served "at cost", and students were liberally hired to work at 12.5 cents/hour. President Bryan commented that all sorts of "students" came under these conditions, and that deficits grew rapidly. Bryan raised the entrance requirements, essentially eliminating the preparatory status, and separated the housing and food service from the college budget. (In 1988-1989 food service at WSU encumbered significant debt because it retained the same number of employees previously used for 5,000 students while the number eating in dining halls declined to 3,500.)

Joint appointments in teaching and research existed from the beginning. Four members of the faculty served as teachers in the School of Science and also as researchers in the Experiment Station (Second Ann. Rept., AES, 1893). In that year only \$1,666.66 of Hatch funds went to salaries, so teaching funds supported a large portion of the experiment station salaries. Hatch funds in the beginning went mostly for barns, tools, livestock, seeds, etc.

The college catalog for 1893 lists President Bryan, LL.D., as Professor of History and Political Science (he taught seven courses). Edward R. Lake, MS, was Professor of Agriculture (= Farm crops, Livestock, Horticulture and Forestry), Mrs. Nancy L. Van Doren, Professor of English Literature, was the Librarian and Preceptress (equivalent of a house mother in a present day sorority). Charles V. Piper, BS, was Professor of Botany and Zoology. James Ferguson was Secretary to the Faculty and Professor of Stenography and Typing. Anderson R. Saunders, ME, was Professor of Mechanical Engineering and Physics. Osmer L. Waller, BS, was Professor of Mathematics and Civil Engineering. Elton Fulmer, MA, was Professor of Chemistry. Lyman C. Reed was Assistant Professor of Horticulture. Persons whose names are underlined were also members of the Experiment Station staff. President Bryan served as Director of the Experiment Station.

Frequent personnel changes hampered progress. John O.'B. Scobey was Agriculturist 1891-1892 and in 1893 he was replaced as Agriculturist by William J. Spillman. E. R. Lake was Agriculturist and Horticulturist, 1892-1893, and he was replaced by John Balmer in 1894. Chemist Hitchcock was replaced by Fulmer in 1893. In 1903, President Bryan lamented the frequent changes in personnel. Within a few years he appointed three Horticulturists. In 1903 Bryan wrote, "And my experience goes to show that few men, if any, can take up a line of research already in progress and prosecute it successfully."

Early administration of the Experiment Station. Until 1908 Presidents Lilley and Bryan wrote the annual reports of the agricultural experiment station (AES), and the presidents were closely involved in its activities. High USDA officials objected to the president serving as experiment station director and President Bryan appointed R. W. Thatcher (Chemist) Director of the Experiment Station in 1908. I. D. Cardiff (Botanist) was director from 1914 until 1917. For several years after the College of Agriculture became a unit within the University, the dean of the college served as Director of the AES. Dean E. C. Johnson, 1919, served in this capacity. Dean Johnson was a distinguished plant pathologist. In 1909, he was coauthor with E. M. Freeman of a study of loose smut of barley and wheat; in 1910 he published methods of breeding for rust resistance in cereals; in 1911 timothy rust; 1912 a USDA Farmers' Bulletin on smuts of wheat, oats, barley and corn; and in 1914 another bulletin on imperfect fungi isolated from

cereals. I found no evidence that Dean Johnson favored plant pathology during his long tenure as dean. He did no research at WSU.

### ***The Times of Charles V. Piper, 1893-1903***

The first person officially responsible for plant pathology at WSU was Mr. C. V. Piper from the University of Washington, hired in November 1892 as Botanist and Entomologist for the Experiment Station, and Botanist and Zoologist of the teaching faculty (the College). In reading this chapter, keep in mind that plant pathology was the least of his activities, being overshadowed by his teaching, entomology, and by his involvement in recording the flora of the region. In 1893 he published bulletin 8 on important diseases in the state, including loose smut of oats, stinking smut (bunt) of wheat, late blight and scab of potatoes, fireblight and scabs of pear and apple, leaf curl and powdery mildew of peaches, black knot, leaf blight of strawberry, and dodder. Most of the early publications were based largely on the literature, as this one obviously was, but Piper did experiments to kill dodder by sprinkling it with an iron sulfate solution. In the annual report of 1893 he reported that common bunt was serious because of careless seed treatment. The department needed better microscopes, library facilities, and better equipment for spraying orchard trees. His knapsack sprayer was inadequate. He commented that lack of funds prevented him from doing any real research (how about time?).

The State College Bulletin for the 1893-1894 school year lists Charles V. Piper as the Professor of Botany and Zoology. He taught Elementary Botany, Systematic Botany, Plant Physiology, Cryptogamic Botany (including economic fungi, their morphology, life cycles and control), General Zoology, General Entomology, Special Entomology and special problems (= he guided individual studies). This was a teaching load of seven regular courses plus special problems! In the 1894-1895 Bulletin he revised his zoological offerings to include Invertebrate Zoology, Vertebrate Zoology, Normal Histology, Comparative Embryology, General Entomology, Special Entomology, plus the same Botany courses! Two of Piper's courses included some mycology with some emphasis on plant pathogens, but it is obvious that the college started with a minimum of plant pathology.

President Bryan in the annual report of 1894, responded to charges of incompetency and corruption in the teaching part of the college and stated that it was necessary to raise the standards for admission of students and that the preparatory (high school) function of the college should stop. Bryan also tried to rescue the institution from political manipulation. He commented that the Domestic Economy course for young ladies was well attended. The teaching faculty of WSU at that time consisted of 14 males and 2 females; there were 67 male and 43 female students.

Many companies and individuals donated spray equipment and supplies and in 1894 Piper sprayed fruit trees in cooperation with Mr. J. B. Holt, Manager of the Snake River Fruit Growers' Association. A blight of tomatoes, now known to be curly top, was severe and Piper sprayed tomatoes at the H. H. Spaulding place on the Snake River with Bordeaux mixture in an effort to control it. Modern pathologists recognize the futility of this effort, an example of the value of knowing the cause of a disease. Wheat rust (type not specified) was the most serious in memory, but rust was considered less serious than stinking smut because it was more erratic in occurrence. An average of two letters a day were answered, evidence of citizen interest in the activities of the department. Piper complained of cramped quarters and Chemist Fulmer lamented the lack of natural gas.

In 1895 crown gall attracted much attention. The galls on apple were small, those on grape large. Only those two hosts were brought to Piper's attention, but he stated the disease was known to affect more species of plants. He didn't like the name "crown gall" because galls also occurred on other parts of the host. He didn't know if crown gall on apple was caused by the same thing as crown gall on grapes [the cause of crown gall, the bacterium *Agrobacterium tumefaciens*, was unknown at this time]. Orange rust, *Caecoma nitens*, was found on raspberry and he promised to eradicate it. Black spot of apple (*Sphaeropsis malorum* var. *lignicola*) was serious in Western Washington. Piper believed Bordeaux



mixture would control it, but he was so busy he had no time for field work. This surely saddened those who donated the spray equipment.

Piper found a *Sporotrichum* sp. that attacked insects. The herbarium now contained 6,000 specimens of phanerogams, 400 pteridophytes, 700 bryophytes and a large collection of fungi, mostly plant parasites. Thus, both the botanical and mycological herbariums were well underway. In addition to identifying weeds, medicinal and poisonous plants, he corresponded heavily and wrote numerous newspaper articles. At this stage of institutional development extension type activities were significant.

In 1895 a three-week Winter School for Farmers was attended by 304 farmers from 12 counties. The farmers were the most diligent students Pres. Bryan had ever seen. He praised the value of combined investigation and instruction in the college. (Italics provided by Bryan (Annual Report, 1895).

Knowledge of farm experience and problems was obtained by widely distributed questionnaires, called circulars. W. J. Spillman, Agriculturist, sent a 'circular' to farmers in the 17-20-inch rainfall belt concerning wheat varieties before he attempted to improve wheats by breeding. Farmers told him Little Club was the best wheat in the wetter region. The farmers in the high rainfall area wanted a winter wheat like Little Club (a spring wheat with strong straw, shatter resistant). Spillman crossed Little Club with the best winter wheats available. From these crosses came Hybrid 128 (Little Club x Jones Winter Fife). It had larger club heads than Little Club, stiff straw, good quality, winter hardiness, and it yielded far better than its parents (see Bulletin 89, published in 1909, for details of this work). This advance by Spillman is locally comparable to that by Dr. O. A. Vogel with semi-dwarf wheats many years later. The questionnaire guided Spillman in his selection of parents and desirable qualities (good straw, resistance to shattering, winter hardiness). It illustrates, along with the "institutes", the use of farmers' experience by experiment station personnel.

State law required establishment of substations, one of which must be west of the Cascade Mountains. The Puyallup station had its start by 1895 when D. M. and Charles Ross and other family members donated 40 acres of bench land and offered reasonable terms for leasing 20 acres of bottom land (5th Annu. Rpt., 1895).

The intent of legislators was for agricultural work at Pullman to be mostly basic. [The Pullman faculty has emphasized "basic" research, if publication in refereed journals is used as the criterion. Funds for basic research have recently grown significantly. Branch station scientists will have to increase their emphasis upon basic science or suffer from relative decreasing support, at least temporarily. Hard money (= federal formula funds and state support), the mainstay of applied research, is not keeping up with inflation.]

Refereed articles per year, state-wide and at Pullman

Years	State Total	Pullman Total	Years	State Total	Pullman Total
1970-1971	44	24	1980-1981	71	43
1972-1973	39	25	1982-1983	57	40
1974-1975	46	25	1984-1985	64	39
1976-1977	31	23	1986-1987	77	50
1978-1979	47	32	1988-	31	30
			Total	507	323

Puyallup was to stress practical control of diseases prevalent under the more humid conditions of Western Washington. The Puyallup station for many years was supported only by state appropriations because of fear of misinterpreting the laws governing land-grant institutions, i.e. could those funds be legally spent at a "branch" station of a land-grant college?

R. W. Doane was appointed Assistant Entomologist to help Piper in 1896. The "department" now had two scientists. Doane assumed much of the teaching on zoological subjects, so Piper had more time for other activities. The herbarium had grown to 10,000 specimens of phanerogams and 2,000 of fungi. J. B. Flett contributed 400 plant specimens from near Mt. Rainier and Tacoma and Kirk Whited sent 600 from near Wenatchee. Progress on a technical bulletin on native flora of the region was slow, according to Piper. Piper cooperated with the veterinarian, S. B. Nelson, in trying to kill ground squirrels with poisons and with a natural bacterial pathogen of the rodents. This effort at biological control failed and was abandoned. Isolations from blighted tomatoes were inconsistent, so the etiology of tomato "blight" was still unknown. A USDA scientist, Dr. A. W. Thornton worked independently of WSU on fiber flax in western Washington. He recommended at least a 5-year rotation in the Puget Sound area. The cause of flax wilt was still unknown. At this time there was little or no coordination of investigations by federal or state scientists within Washington, and there was friction among them.

1897 was a bad year. No annual experiment station report was found for that year and the Puyallup station was closed in April. Note the drop in funds, 1897-1899 biennium.

Board of Regents report, WSU receipts

Biennium	State appropriation	Total income, all sources
1891-1893	60,000	132,500
1893-1895	97,000	167,333
1895-1897	78,474	156,167
1897-1899	29,000	114,549
1899-1901	144,250	250,139
1901-1903	127,800	236,393

The 1899-1901 appropriation included \$50,000 for Science Hall and \$5,000 to reopen the Puyallup station.

In 1898 Mr. David A. Brodie received the first B.S. degree in agriculture from WSU. He was a Botany major so much of his training was by Piper. Mr. Brodie was made superintendent and assistant agriculturist at Puyallup. His duties were to study the use of fertilizers, forage plants, fiber flax, and fungus diseases of plants, particularly black spot of apples.

The university catalog for 1898-1899 listed a total faculty of 25, 10 of whom had experiment station responsibilities. These 25 persons taught courses now distributed among six colleges.

Science Hall, begun in 1898, was complete in 1900. Piper spent much of that year at Harvard University, doing herbarium work. Mr. R. Kent Beattie, Instructor in Botany (1899), took charge during Piper's absence. Both Piper and Beattie had skills in plant pathology, as did Brodie at Puyallup. Mr. W. H. Lawrence, with training suitable for work in entomology and plant pathology, was working in Puyallup on aphids attacking hops. Agriculturist Spillman (Bull. 41) divided the state into several ecological areas as a guide for selecting different forage grasses. By this time the ecological diversity of the state was widely recognized. Peas for seed were already important in eastern Washington and Spillman projected a bright future for chick peas. Spillman resigned at the end of 1901. President Bryan requested a full-time person to run the institutes (= adult education or agricultural extension) because they took too much time. He complemented Brodie's efforts at Puyallup and recommended generous support for the Puyallup station. No money was provided for irrigation studies.

In 1901 Beattie was officially appointed Assistant Botanist in the experiment station, and O. L. Waller was appointed Irrigation Engineer, so irrigated agriculture was now receiving some emphasis. S. W. Fletcher,

Horticulturist, emphasized the need to duplicate many studies east and west of the Cascade Mountains. Bull. 46, "Potato Blight and its Treatment", was published in 1901 by Brodie. Brodie did real plant pathology, determining the best number and timing of sprays (Bordeaux mixture) to control late blight. In my opinion, this study qualifies Brodie as the first plant pathologist trained at WSU because this paper reported original, on site experiments of consequence.

The year 1902 was a very bad one for bunt (stinking smut). Over 50% of the grain was docked at sale because of smut. Wheat was selling at 50 cents per bushel and the dockage was 5% of the sale price. This, coupled with yield loss and danger of threshing machine fires, made the disease a special problem. Bull. 54 by Kent Beattie describes the use of formalin to destroy bunt spores on the seed. Mr. Piper was still convinced that careful seed treatment should control bunt, even in winter wheat. S. W. Fletcher, Horticulturist, experimented with sprays to control apple scab in western Washington. He attributed a severe loss of prune trees in Clark County to cold. In 1902 Mr. William H. Lawrence received a MS degree in Botany, the first advanced degree awarded by WSU.

Attempts by Beattie and Mr. Davis to control crown gall with fungicides and disinfectants failed (the bacterial nature of this diseases was still unknown). They concluded that Mr. Tourney was in error: crown gall was not caused by a slime mold. Piper and S. W. Fletcher published Bull. 59 (1903) on *Armillaria mellea* root rot of fruit and other trees, accounting for the loss of prune trees in Clark County. That report concluded Piper's contributions to plant pathology in Washington.

The 1903 bulletin on *Armillaria* root rot included excellent descriptions of the rhizomorphs, of the white velvety mycelium below the bark, especially at the collar of the tree, and of the honey-colored toadstools at the base of the tree. A prune grower near Vancouver recorded the fate of his trees, 503 planted in 1894 and 550 planted in 1895. Six died in 1895, 77 in 1896, 40 in 1897, 64 in 1898, 154 in 1899, 125 in 1900, and 117 in 1901, or 583 out of 1053. Piper and Fletcher couldn't find *Armillaria mellea* in the woods near Vancouver and Castle Rock, but the fungus was common on prune plantings in both areas. They thought the fungus may have been introduced. By 1910, western Washington Experiment Station Bulletin 3, W. H. Lawrence believed there were variants (= races?) of *Armillaria mellea*.

C. V. Piper's greatest contributions to WSU were in documenting the early flora of Washington and adjacent Idaho [Flora of the State of Washington, 1906 (QK 192 P66). Flora of Southeastern Washington and Adjacent Idaho, 1914 with R. Kent Beattie (QK 192 P64)]. He resigned from WSU in 1903, as did his former student D. A. Brodie. W. H. Lawrence, also a student of Piper, completed some of Brodie's studies at Puyallup, after which he was moved to Pullman.

This period produced little original research in plant pathology and no formal course on the subject, but obviously pathogenic fungi were studied. The recipients of the first BS degree in agriculture (Brodie) and of the first MS degree in the university (Lawrence) were skilled in Plant Pathology. Mr. Piper, though a fundamental botanist, must have taught in a way favorable to the study of plant diseases. There were no more than three persons active in plant pathology in Washington at any one time during the Piper period.

Obituary of Charles Vancouver Piper, who died in 1926, is in Science 63:248-249, 1926.

### ***The R. Kent Beattie Period, 1904-1909***

The departure of C. V. Piper was a real loss, but his replacement, R. Kent Beattie, devoted some time to plant pathology in spite of his heavy duties as Botanist. Beattie was most active as a teacher and in continuing studies on the flora of Washington. Wheat smut was so severe in 1903 that Beattie directed a campaign against it in 1904. Yield losses up to 40 percent occurred and fires and explosions in threshing machines were common. Oat smut was also serious. In 1904, Zoology was removed from Botany as an academic department, making two academic departments instead of one.

William Lawrence remained at Puyallup until 1904 when he was brought to Pullman to offset the loss of Piper. In 1904 he published Bulletin 64 on the control of apple scab in Western Washington. Apple scab was so severe it partially defoliated the trees, reduced the yield, and deformed the fruit. Lawrence also published Bulletin 66 on 'black spot canker' of apples in western Washington, a disease called apple anthracnose by A. B. Cordley in Oregon in 1900. Cordley named the pathogen *Gloeosporium malicorticis*. Lawrence studied the pathogen in culture and on the host, determined the incubation period on trees by inoculations, and control. The transfer of Lawrence to Pullman left Puyallup with no pathologist in 1905. Lawrence assisted Beattie in teaching and in 1905 he published Bulletin 70 on important powdery mildews in Washington.

In 1906 Lawrence published Bulletin 75 on apple scab in Eastern Washington. Chemist R. W. Thatcher developed a more economical lime-sulfur formulation. Beattie reported that even with very careful treatment of winter wheat seed with formalin, some bunt developed. Lawrence was granted leave for one year to study plant pathology at Cornell. This is the second leave (Piper's to Harvard was the first) that I found.

In 1907 W. H. Lawrence published Bull. 83 in which he discussed many important diseases with their symptoms and what was known about their control. Club root of cabbage, crown gall, fire blight of pear, late blight of potato, peach leaf curl, witches' broom of cherry, apple scab, pear scab, bean anthracnose, black spot canker of apple (which could be reduced by double-strength Bordeaux applied in November, a new finding), potato scab, canker of soft maple, brown rot of prunes, plums and cherry, dry rot of potatoes, bunt of wheat and the use of formalin, oat smut, Armillaria root rot of prune and powdery mildews were discussed. This was the second general publication on plant diseases of the state. Bordeaux mixture was now used to control apple scab, but it often caused severe damage to the tree. Wheats from the crosses of Spillman were now recognized as having great value to Washington. President Bryan, who wrote the 17th annual Experiment Station Report (1907), stated, "The most important investigational work of this department (Botany) has naturally been in connection with the study of plant diseases of economic importance".

The year 1908 was important. The Botany Department now had three full-time members with the employment of Dr. H. B. Humphrey (Stanford University), the first person with a Ph.D. within the unit. Humphrey was a botanist and a marine biologist at Stanford, but his B.S. from Minnesota included a background in plant pathology. In 1908 W. H. Lawrence was promoted from Assistant Botanist to Plant Pathologist, the first WSU employee with that title, and he was transferred back to Puyallup as Superintendent of the re-opened station. As Superintendent he had many duties, but he continued to study diseases important in Western Washington. Lime sulfur, being less phytotoxic, was studied as a substitute for Bordeaux mixture for control of apple scab.

In 1908 President Bryan ceased to act as Director of the Experiment Station. Dr. Thatcher, though retaining his duties as Chemist, assumed this responsibility. Dr. Thatcher officially stated that Puyallup should be the site of studies of diseases favored by high humidity, especially of raspberries and blackberries.

Extension activities were continuing on a significant level. In 1908 there were 71 'institutes' throughout the state with a total attendance of 14,306. A new development, farm demonstration trips, was initiated when the Oregon Railway and Navigation Company (part of the Union Pacific system), the Spokane and Inland Empire Electric Railway, and the Northern Pacific Railway took farmers on special trips to see farm demonstrations near the railroads. In 1911 attendance at institutes was 10,465 and 42,658 participated in the train demonstration trips. This massive extension activity in these early years is remarkable.

By 1909 lime-sulfur was established as an improvement over Bordeaux mixture for apple scab. Director Thatcher expressed the need for botanists with no teaching or other college duties so they can work full-time on plant diseases.

This period could have been called the Wm. Lawrence period because Mr. Lawrence was the person most dedicated to plant pathology up to this date, but Beattie was Head of Botany, and even with his many duties he contributed significantly to plant pathology. Bulletins 107 on a *Sclerotinia* and 108 on bluestem of red raspberry by W. H. Lawrence were published in 1912, after he had resigned.

The finances of Washington State University improved dramatically during the "Beattie" time. Kent Beattie left WSU in 1912 for a distinguished career in forest pathology with the USDA.

Biennium	State appropriation	Total income
1903-1905	\$163,000	\$278,648
1905-1907	\$165,000	\$296,666
1907-1909	\$575,754	\$717,847
1909-1911	\$534,536	\$784,058

Beattie resigned when Plant Pathology was made distinct from Botany and subsidiary to it, and Humphrey was to head Plant Pathology. Beattie was senior to Humphrey and he felt injured by this action (p. 47 in Robert Hadlow's MA thesis).

#### Obituaries

Harry Baker Humphrey, 1873-1955, by H. P. Barss. *Phytopathology* 47:247.

Born on a homestead in Minnesota, he moved to Oregon in 1877. After 5 years he returned to Minnesota. He received a BS at the Univ. of Minnesota in 1899 in Botany and Modern Languages (this may explain the linguistic talents of Woolman, at least in part). Humphrey received his Ph.D. from Stanford in 1907 and worked three summers at the Hopkins Marine Biological Lab, Pacific Grove, California. In 1908 he became Prof. of Botany and Plant Pathologist at WSU. In the summer of 1912 he taught at the Marine Biological Lab at Friday Harbor in the Puget Sound. He obviously had a sound background in both botany and zoology. He worked with the USDA from 1913-1943 when he retired at 70. He edited *Phytopathology*, 1929-1945, and Barss credits him with elevating the standards of this journal. He taught French from 1922-1948 at a USDA "graduate school" after working hours. No wonder he had no trouble translating the works of Tillet.

Rolla Kent Beattie, 1875-1960, by John A. Stevenson. *Phytopathology* 51:135.

Beattie received an AB degree at Cotner University (Lincoln, Nebraska) in 1895, a BS and MS from the Univ. of Nebraska in 1896 and 1898, respectively. He taught in high school for two years, and moved to Pullman in 1899 to collaborate with C. V. Piper, helping to produce *The Flora of the Palouse Region*, *Flora of Southwestern Washington and Adjacent Idaho*, and *Flora of the Northwest Coast*. In 1903 he was Head of the Botany Department. His most important pathological research was developing the use of lime-sulfur to control apple scab and other spray works in collaboration with entomologist A. L. Melander. He left WSU in 1912 to work in Washington, DC, on chestnut blight. He had a distinguished career in forest pathology with the USDA.

#### ***The Harry B. Humphrey Period, 1910-1913***

In 1910 H. B. Humphrey was promoted to Plant Pathologist, becoming the second person with that title within the Botany Department. Humphrey is listed in the 1909-1910 college catalog as sole teacher of two general courses on fungi, a course on economic (= pathogenic) fungi, general bacteriology, cytology and embryology, algae, and he shared a course on general botany with Professor Beattie. In the 1912-1913 catalog Humphrey taught Cytology, Embryology, and Algae in the Botany curriculum and General Plant Pathology, Morphology and Systematics of Pathogenic Fungi, Slime Molds and Phycomycetes, Pathogenic Ascomycetes, Basidiomycetes and Fungi Imperfecti, and Forest Pathology within the

Pathology curriculum. Humphrey, according to Fischer (1951), initiated the moves to establish a curriculum in Plant Pathology separate from Botany, and his division of subjects placed Mycology within Plant Pathology. Humphrey was Vice Director of the experiment station, 1910-1913. While Director, he placed plant pathology in the annual experiment station reports under Plant Pathology rather than under Botany. In 1911 he was designated Professor of Plant Pathology, the first academic use of this title in the University. Dr. Humphrey resigned in 1913 to head research on diseases of field crops for the USDA in Washington, D.C.

The discovery of the role of soilborne spores in the epidemiology of common bunt in the Pacific Northwest was made during this period. According to Woolman and Humphrey (1924, Studies in the physiology and control of bunt or stinking smut of wheat, USDA Bull. 1239), A. M. Richardson presented evidence that infection can in some cases come from wind-borne spores (unpublished thesis of WSU in 1911). Humphrey began experiments in 1911 and concluded (1912, Popular Bull. 48) that seed treatment with either copper sulfate or formaldehyde will not guarantee against bunt. By 1914 Woolman (Popular Bull. 73) stated that soil infestation is important, that the spores free of the bunt balls do not live in soil from one season to another, that the effect of spores on the soil surface can be reduced by moldboard-plowing summer fallow just before seeding, but that this is not practical. Woolman and Humphrey did the critical experiments on the role of soilborne spores, their longevity in soil and the influence of seeding date on smut (= soil temperature plus survival of spores in soil) that laid the foundation for the studies of Heald and George (1918) in which, beginning in 1915, they recorded the time and rate of spore showers. These studies were fundamental to understanding the failure of local-action seed treatments in controlling common bunt in the PNW, and they rank among the most important experiments ever conducted in Washington. All faculty and graduate students should read the 1918 bulletin of Heald and George and the 1924 bulletin of Woolman and Humphrey.

Woolman and Humphrey translated and published the literature of Europe on bunt (USDA Bull. 1210, 1924). In 1937 Harry Baker Humphrey published the translation of Mathieu du Tillet's 1755 publication, "Dissertation on the cause of the corruption and smutting of the kernels of wheat in the head and on the means of preventing these untoward circumstances", making Phytopathological Classic No. 5, APS Press.

Humphrey attributed the tomato blight in Washington (= mostly curly top) to *Fusarium oxysporum* (Bull. 115, published in 1914).

In 1910 W. H. Lawrence (at Puyallup) published Bull. 93 on ridding land of stumps with explosives, Bull. 95 on a new forage plant as a possible livestock feed, Bull. 97, "Anthracnose of Blackberry and Raspberry", and Western Washington Experiment Station Special Bull. 5 on club root in the Puget Sound region. In 1911 Lawrence resigned, terminating the most concentrated career of some duration in plant pathology in Washington to that date. Fischer credited Lawrence with teaching the first real course in plant pathology at WSU, Economic Fungi, first taught in 1905. I give much credit to Piper who taught both Brodie and Lawrence.

H. P. Barss was hired as plant pathologist at Puyallup in 1911, after Lawrence resigned. Barss left after one year to go to Oregon State University. Smut was raging. A smut conference was held at Pullman, October 11, 1911, for dissemination of general information.

In 1912, Mr. D. C. George, BS, the first plant pathology major to graduate from WSU, was added to the Pullman Plant Pathology faculty, as was John G. Hall. Humphrey reported that the Plant Pathology Department got some new equipment, including a freezing microtome.

There is no experiment station report on plant pathology in 1913, probably the result of Humphrey's departure and the late arrival of Ira D. Cardiff to Head Botany and Plant Pathology in 1913. Note the reunion of Botany and Plant Pathology by Cardiff as before the time of Humphrey.

Bulletin 106, by A. L. Melander, Entomologist, and R. Kent Beattie, Botanist, reported that coarse droplets under high pressure penetrated the canopy of fruit trees better than mists under low pressure, an important contribution. D. F. Fisher, a USDA pathologist, worked in fruit pathology at Wenatchee from 1913 - 1947. His work will be covered in other parts of the history.

It is difficult to follow the chronology of the faculty during this period, but Beattie, in Botany, still had some interest in plant pathology. Lawrence and Humphrey were faculty in 1911, Barss, Humphrey and George in 1912, John G. Hall (WSU, Pullman), George, D. F. Fisher (USDA, Wenatchee), Humphrey in part of 1913, and apparently Woolman late in 1913, were plant pathologists during the Humphrey period, at least for short times. As near as I can tell, for a few months there may have been four full-time plant pathologists at Pullman in 1913 (Humphrey, George, Hall, Woolman), a possible record not equalled for years to come, and, in addition, Fisher at Wenatchee.

It is worth noting that H. B. Humphrey (in 1945) and H. P. Barss (in 1928) became presidents of APS.

#### Obituary

Horace Mann Woolman, 1853-1932. By Harry B. Humphrey. *Phytopathology* 23:931.

Woolman was a self-taught civil engineer who surveyed the route of the Chicago, Milwaukee, and St. Paul Railroad through the Coeur d' Alene Mountains of northern Idaho. At the age of 56 he enrolled at WSU to study botany, chemistry, and bacteriology. At Pullman he taught himself to read German and French and he and H. B. Humphrey translated the European literature on the smuts (USDA Bull. 1210, 44 p. 1924). Mr. Woolman was employed by WSU as an Assistant in Plant Pathology in 1914. He never completed his bachelor's degree, quitting a few hours short of its completion. In 1917 he became Field Assistant in the Division of Cereal Crops and Diseases until 1924, when he retired at the age of 70. (Woolman continued the studies on the epidemiology of common bunt initiated by Humphrey, and together they published "Studies on the physiology and control of bunt, or stinking smut of wheat," USDA Bull. 1239). Woolman studied the host-pathogen interactions in "Infection phenomena and host reactions caused by *Tilletia tritici* in susceptible and non-susceptible varieties of wheat," *Phytopathology* 20:637-652. Woolman and Humphrey published a summary of early literature on bunt starting about 500 BC (already mentioned, USDA Bull. 1210, 44 pp. 1924). If you want inspiration, read these three publications by a man who never graduated from college. Real scholarship!

Humphrey credits Woolman with playing a major role in selecting Oro and Rio winter wheats, important sources of resistance to common bunt. E. F. Gaines gave Woolman segregating populations of Turkey x Florence wheats which he tested for resistance.

"True to the tradition of his Quaker lineage and background, Mr. Woolman was wholly and genuinely devoted to the philosophy of service, not for self but for the advancement and benefit of others."

The obituary did not mention a wife or family. I suspect he lived a spartan life on a meager salary.

#### ***The Ira D. Cardiff Period, 1913-1916***

The brief time during which Ira D. Cardiff served Washington State University is one of the most important periods, influencing development of the experiment stations and ultimately the plant pathology department.

Ira D. Cardiff received a B. S. degree at Knox College, was a Graduate Student and Fellow at the University of Chicago, received his Ph.D. at Columbia University, served as Assistant in Botany at Columbia 1904-1906, as Assistant and Professor of Botany at the University of Utah, 1906-1908, Professor at Washburn College 1908-1912, and worked in summer school at the University of Kansas in

1911-1912, after which he came to Pullman as a Plant Physiologist. With the departure of Humphrey, Cardiff became Botanist and Plant Physiologist, and Plant Pathology reverted to Botany in the experiment station. Cardiff was Head of Botany and Plant Pathology, and he was also Director of the Experiment Station, 1914-1916.

The departure of Humphrey and Lawrence required employment of a person with advanced training to maintain quality instruction in plant pathology. John Galantine Hall taught all the plant pathology courses from 1913 until 1915, when he resigned. He had a BA from Kansas State University and an MA from Harvard. Hall had served as Assistant in Plant Pathology at North Carolina State (1906-1910) and as Associate Professor of Botany, Forestry and Bacteriology at Clemson (1910-1913) before coming to Pullman. While here, in addition to teaching, he wrote Popular Bulletin 56 outlining the fire blight situation in Washington and its control. D. C. George was listed as an Instructor in Plant Pathology so he must have assisted Hall with teaching. George was also Assistant Plant Pathologist in the Experiment Station. In 1914 H. M. Woolman was made an Assistant Plant Pathologist, but Woolman had no degree and no status for teaching.

As Head of Botany in the Experiment Station, Cardiff wrote the annual reports. In 1914 he added alfalfa downy mildew, sclerotinia on alfalfa, California peach blight on apricot, barley smut, gummosis of cherry, currant blight, and bacterial rot of onions to the list of important diseases. He stated that smut was the subject of the oldest and most important project of the experiment station. Horticulture had a project on fire blight.

The study of tomato blight by H. B. Humphrey (Bull. 115) was published in 1914. Humphrey doubted that it was the same as 'summer' blight described by R. E. Smith in the California valleys in 1906 (= curly top). Humphrey attributed the blight of tomato to *Fusarium oxysporum* and *F. orthoceras*. N. Rex Hunt and D. C. George helped in the investigations.

In 1914 as many as 6-8 fires a day started in threshing machines in Whitman County. In 1913 close to 300 threshing machines were destroyed, at a loss of \$1200-\$1500 each. I. D. Cardiff, Director of the Experiment Station; O. L. Waller, Civil Engineering; H. V. Carpenter, Mechanical and Electrical Engineering; G. A. Olson, Chemist; E. G. Schafer, Farm Crops and A. L. Sherman, Assistant Chemist, studied the cause of the fires (Bull. 117). They found that smut balls contained more moisture than healthy wheat kernels and that smut dust was more flammable than other organic dusts tested. Smut spores contained 4-5% oil. A flame or spark is needed to ignite the dust. The smut-air mixture can be ignited by static electricity or any spark (the problem was not sabotage). The year 1914 was a serious smut year, and the harvest season was very dry, resulting in the epidemic of fires. This study was followed by Extension Bulletin 46 on prevention of, and protection from, smut dust explosions and fires in threshing machines by G. E. Thornton, Department of Mechanic Arts and Engineering in 1918. In 1925 it was reported that the static electricity could be safely discharged by grounding within the machine, preferable to grounding to the soil. Apple scab was so bad in Spokane County in 1914 only 10-15% of the apples graded fancy or extra fancy. Spokane County was the only county clamoring for a county agent with pathology training in 1914.

Cardiff's 1915 report makes it clear that answering disease inquiries was the number one activity in the Botany Department and that much diagnostic effort, or extension, was done at Pullman. In 1915 Frederick D. Heald was hired to replace Hall, who had resigned.

Frederick DeForest Heald received his BS and MS degrees in Botany at the University of Wisconsin and his PhD from the Univ. of Leipzig. He was Professor of Biology at Parson's College 1897-1903, Adjunct Professor Plant Physiology at the University of Nebraska 1903-1905, was Associate Professor of Plant Pathology at the University of Nebraska 1905-1906, Professor of Agricultural Botany and Botanist at Nebraska 1906-1908, served as Head of the School of Botany at the University of Texas 1908-1912, was



a Collaborator or Agent in the USDA 1905-1914, mainly investigating chestnut blight in Pennsylvania. The overlapping dates represent dual appointments. Heald was President of APS in 1932.

Research in progress included studies of brown rot of prunes in Clark County, and a study of powdery mildew in the A. M. Pearce Orchard at North Yakima, including spraying and pruning. Tomato blight was studied at Pullman, and doubt was expressed that *Fusarium* caused the disease. By now, it was known that available seed treatments did not control bunt in winter wheat in eastern Washington, that resistance was the only practical control available. Horticulture discontinued its study of fire blight and began to study a rosette of apple trees.

Heald (Bull. 125) proved that *Bacillus amylovorus* (= *Erwinia amylovora*) could enter stomata and hydathodes on apple and pear leaves, and from there the bacteria could enter the twigs. Blighted apple leaves turned brown, pear leaves black. Note the speed of publication, normal for station bulletins at this time. Within two or three months of gathering the last data the paper was published (Heald came in 1915 and he published this finding in 1915). Between 1889 and 1920 bulletins and circulars of various types were the major publication outlets. Between 1921-1950, numbers of these types of publication and scientific journal articles were about equal. From 1951 onward scientific journals were by far the major outlet for research results. Bulletins, circulars, extension publications are still used, mainly to communicate with clientele.

Heald and Woolman (1915) published the first comprehensive treatment of stinking smut (bunt) of wheat in the state. It discussed crop rotation, clean seed, seed treatments, cultural practices, and breeding and selection of resistant varieties. Discussed under cultural controls were soil temperature at seeding, soil moisture, and depth of seeding. The latter information was from experiments by Woolman and Humphrey because Heald had just arrived. The USDA now contributed \$300 a year to study smut.

The County Agriculturists and State Horticulture Inspectors played a real role in providing specimens, data, and field observations. The State Horticulturists aided in spray trials in orchards. Travel from Pullman at this time was relatively arduous, making cooperation even more valuable than today. In 1915 James G. Dickson graduated with a B.S. in Agriculture and Botany from WSU. He left with a strong interest in physiology. Apparently Cardiff was a good teacher. In any case, Dickson went on to a distinguished career at the University of Wisconsin. He was president of APS in 1953.

In 1916 President Holland replaced President Bryan. Cardiff was still Director of the Experiment Station and Head of Botany, and Heald, George, and Woolman were the pathologists.

Extensive crop rotation studies were started while Spillman was Agriculturist. In a 15-year study at Pullman, in 1915 the highest wheat yield (46 bu/acre) was following a clover and timothy, oats and pea rotation. The second highest yield was continuous wheat, but the experimental land received 10 tons of manure per acre per year, plowed under in the fall. [There is no evidence of increased disease loss in continuous wheat, but obviously the average seeding date was late, which would control strawbreaker foot rot and cephalosporium stripe, and possibly the heavy use of manure controlled or reduced take-all.] In general, rotations produced slightly higher total yields than monoculture of wheat and fallow.

Financial support for the University continued to climb. In the 1911-1913 biennium the total from all sources was \$824,390, in the 1913-1915 biennium, \$1,088,452.

#### *The Cardiff Episode, 1914-1916*

A conflict occurred between Cardiff and extension. By state law (1913) county agents were under the Director of the Experiment Station. By federal law they were under a Director of Extension. Even though the Smith-Lever Act (1914) provided federal support for county agents, most of the money came from

local sources. Cardiff tried to retain administration of county agents within the experiment station but lost the battle. County agents were placed under the Director of Extension, a new position.

From 1900-1915 bankers made loans to farmers in dryland Washington, and dryland farmers were doing poorly. The Spokane Chamber of Commerce and Director Cardiff favored establishment of a dryland station. In 1915 Adams County provided 320 acres a few miles north of Lind. Equipment was provided by gifts from a Spokane man and the Chicago, Milwaukee, and St. Paul Railway. The USDA contributed \$2500. The station was started (26th Annual Report, 1916). In 1915 a subunit was established in Douglas County near Waterville on land leased from the school board. Fencing was supplied by the American Steel and Wire Company.

Mr. H. E. Goldsworthy was appointed Vice-Director of the Dryland Unit, but resigned in three months. Mr. M. B. Boissevain served briefly as superintendent of both Lind and Waterville units, but he resigned to be replaced by M. A. McCall (27th Annu. Report, 1917). The Waterville unit was closed in 1920.

In 1916 Director Cardiff noted the existence of 250,000 acres of irrigated land in Central Washington and stated that it was impossible to adequately serve this industry from Pullman, Lind or Waterville. The Prosser station (Irrigated Agriculture Research and Extension Center) was authorized by the legislature in 1917 and in 1918, after Cardiff left, it received 205 acres of unimproved land (covered with sage brush). The Northern Pacific Railway donated land and offered to sell other land cheaply if \$2,000 would be spent on improvements in one year. The citizens of Prosser raised \$3,000 and donated labor to prepare the land for cultivation. Mr. F. M. Rothrock sold land to the state at a favorable price. The Prosser station received its first real boost when Gordon Moores, a local legislator, obtained a \$35,000 appropriation for the new station.

Director Cardiff compared the average annual state support for agricultural research, 1911-1915, in Montana, Utah, Idaho, Oregon, and Washington in the 26th Annual Report (1916). Montana and Oregon provided the most support, Idaho and Washington, the least.

State	Assessed Valuation	Population	Total State Appropriation for Research
Montana	\$ 347,000,000	446,000	\$ 63,333
Oregon	905,000,000	809,000	47,517
Utah	200,000,000	424,000	15,000
Idaho	168,000,000	446,000	10,766
Washington	1,005,000,000	1,471,000	9,933

By any measure, state support of agricultural research in Washington was appalling. Cardiff stated that Kansas appropriated \$86,585 per year. Kansas had fewer crops and a more uniform environment, making research on its problems simpler. Cardiff estimated needs: \$30,000 for irrigated agriculture, \$27,000 for soils, \$23,000 for dryland, \$13,300 for forages, \$14,000 for marketing, \$17,000 for crop pests and horticulture and \$11,000 for animal diseases, each of which exceeded the total average state appropriation at the time. The legislature appropriated \$33,964.70 for the 1916-1917 fiscal year, a marked increase. I like to think Cardiff's efforts contributed to this response. In any case, after Director Cardiff published the report which reflected unfavorably upon support by the state of Washington for agricultural research, President Holland put all printing under the control of the President. Cardiff stated that no other Director of Experiment Stations in the U.S. was subject to such restriction. The Regents requested Director Cardiff's removal and President Holland dismissed Dr. Cardiff April 30, 1917, with salary until September 15. Today administrators have tenure within academic departments, protecting them in case of removal as administrators.

The Ira Cardiff episode was discussed in detail by Hadlow. To me Cardiff was a hero. He fought vigorously for greater support for the experiment station and was fired for his efforts. But the details presented by Hadlow show he was imprudent.

Alfred Charles True (p. 7 of Hadlow's thesis) became Director of the Office of Experiment Stations of the USDA in the 1890s, and he used this position to emphasize basic research in agriculture. He interpreted the language of the Adams Act of 1906, which provided funds for "original research," to mean fundamental research (p. 7), and some projects submitted during Cardiff's time as Director of the Experiment Station were rejected (p. 49) as not being basic enough for funding, and funding was delayed while Cardiff and True contended (p. 51). Cardiff argued strongly, even going to the U.S. Secretary of Agriculture, to no avail.

Cardiff used \$1300 of Hatch funds to publish 10 Popular Bulletins (p. 52). True disallowed this expenditure, saying that these funds were for investigations and experiments, not for publication of information already in existence. They were not for extension bulletins. The Hatch Act was passed before there was an extension service and its actual wording, to "aid in acquiring and diffusing among the people of the United States useful and practical information on subjects connected with agriculture" (Hadlow, p. 6). Congress did not just want experiments, it wanted information diffused to farmers. Cardiff lost this argument.

The figures used by Cardiff to compare state support in Washington with state support in Montana, Idaho, Oregon and Utah were erroneous in that figures for the above four states included money spent at the main station plus branch stations: the figures for Washington were for only the main station. If the figures for Washington had included Puyallup, Washington would have ranked third (Hadlow, p. 55). President Bryan and the Regents did not appreciate Cardiff's error. I do not know the appropriations for Puyallup during that period, but if we assume them equal to the figure for Pullman (\$9,933), Washington would average \$19,866, a poor third, and still very poor compared to Montana and Oregon.

In 1917 Cardiff went to Olympia without permission from Pres. Holland to lobby in the state legislature for greater support for agricultural research, stating that administrators at WSU only wished to expand the "cultural side of education" at the expense of agriculture (Hadlow, p. 57). In my opinion Cardiff, so impetuous and forceful, was lucky to be fired in 1917. The record of support for agricultural research under Holland - until 1944 - as judged by the fortunes of the Plant Pathology Department, is so dismal this man would have suffered in vain.

The fate of other departments in the College of Agriculture is beyond the scope of this history, but I couldn't help noting that Plant Pathology did not suffer alone. When veterinarian J. W. Kalhus (Hadlow, p. 73) was transferred from Pullman to Puyallup in the 1920's, WSU was without a single veterinarian at Pullman until after 1935.

#### *Cardiff Fights City Hall Expansion into New Areas*

A few items of the early history of Washington State University from President Bryan's book (1928) illustrate the rapid assumption of activities at the fledgling college. The first appropriation (\$1,500) for a library and the first librarian in 1899; piano instruction, drawing and painting in 1900; a major gymnasium, water and heating systems, voice, theory and art of music, band conducting in 1901. Home Economy was started in 1903 with the first BS in home economics in 1906. In 1905 a School of Music was organized along with a program leading to the BS in Pharmacy. A curriculum for a BS in Forestry was approved in 1906. A Department of Education and initiation of architecture in engineering was begun in 1907. The Departments of English and Modern Foreign Languages were begun 1910.

Cardiff didn't agitate in a vacuum. In 1909 the state legislature complained that it didn't want two 'universities', and in 1915 this argument about duplication between the University of Washington in Seattle and Washington State University in Pullman was so heated Bryan stayed until April 1916 to help Holland fight this battle in the legislature.

These and other developments led to criticism that the state college was trying to become a university, usurping activities that were more properly in the domain of the University of Washington and that duplication was wasting money. President Suzzalo of the University of Washington, wrote a letter to President Holland, in 1916, who had just come to WSU, "But the truth is that in America the heads of agricultural colleges brag about their agricultural work to the farmers as though it was the most respectable work in the world, when in their hearts they give agricultural and vocational education a second rate place, and secretly aspire to become liberal college presidents" (p. 31, Landeen, 1958). President Suzzalo feared that Washington would have two mediocre universities rather than a first class university and a first class agriculture college (and engineering). President Holland responded, "The mere training for a vocation must always be supplemented and reinforced by sufficient acquaintanceship with the liberal arts as to insure an understanding and appreciation of the accomplishments and heritage of the human race in its struggles toward enlightenment and freedom," p. 32-33, Landeen. WSU was the first land grant institution in the US to have a chapter of Phi Beta Kappa (1929), a liberal arts honorary, evidence of the unusual nature of WSU at this time.

Many college presidents desire growth, a form of which is expansion into new subjects. To raise quality within an existing unit results in little visible "growth". Growth into new areas at WSU proceeded relentlessly, resulting in starvation of agricultural research. A balance between institutional "growth" and internal development is better.

The relative emphasis upon agricultural research is illustrated by figures gleaned from President Bryan's book. In 1907 the Experiment Station had 16 workers, four were full-time scientists and 12 were 2/3-1/2 teaching, together equalling roughly 10 full-time scientists. The experiment station was responsible for chemistry, soils and agronomy, horticulture, plant pathology, entomology, animal science, veterinary medicine, forestry, extension, agricultural economics, and bacteriology. The experiment station faculty was the equivalent of less than one person per modern department in 1907. In 1910, the Department of Modern Foreign Languages had seven faculty members, and a photograph of the Music and Fine Arts Faculty in 1913 portrays nine individuals.

The tendency of two strong presidents (Bryan, Holland) to over-reach their resources is evident, and it exacted a heavy price from most of the faculty. Under President Holland (1916-1945) 30 new degree programs were created in various areas of study (Landeen, 1958. p. 328). Presidents Bryan and Holland shared the vision of a broad institution, not one restricted to agriculture, engineering and science. Rapid implementation of this vision resulted in low salaries and heavy teaching loads for many years. Washington presently requires two universities and it has the resources to support them.

The policies of President Holland (1916-1945) will become evident in the Heald period (1917-1941). Even though the University as a whole continued to grow, at no time, 1917-1945, were there over three state plant pathologists at Pullman, and in some years only one. In retrospect, the department at Pullman was constant (less than one to no more than three) from 1889 until 1945. In my opinion, President Bryan maintained a better balance between adding programs and strengthening existing programs than did President Holland.

My reading of the books by Bryan, Landeen, and Frykman strengthen my belief that WSU, by advancing relentlessly toward becoming a comprehensive university alienated many groups within agriculture, weakening the support for WSU in the state legislature.

### ***The Frederick D. Heald Era, 1917-1941***

With the firing of Cardiff, Heald became Head of Botany. He re-established Plant Pathology as a section of the Experiment Station (1918) and established Plant Pathology for the first time as an academic department, separate from Botany. Under Heald's leadership the first extension specialist in plant pathology was hired. Three were hired in 1918, but the first two, A. M. Christensen and N. F. Thompson, quit because of World War I. George I. Zundel was the last hired and he served until 1926 when he went to Connecticut. In a later report by the Board of Regents (p. 79, call letters PNC 378.797 WBr, Holland Library) his work is referred to, as follows.

"Many years ago an Extension plant pathologist was employed. The work of this staff member is still evident; for he was largely responsible for the introduction of copper carbonate treatment of wheat smut, which is now the most popular treatment. The many diseases of orchards, berries, nuts, truck crops, field crops, and flowers make the employment of an extension plant pathologist very desirable to the farmers of the state in maintaining the high quality production needed for effective marketing." This statement is remarkable because Washington did not replace Zundel (1918-1926) until Dr. Marion Harris was hired in 1946.

Because of the length of the Heald period, I will concentrate on teaching and personnel, ignoring chronology to a certain extent. When the period started (1917) Heald, George and Woolman were the pathologists, with Zundel added in 1918. Woolman resigned in 1918 to enter the USDA. Bliss F. Dana was hired in 1918 to replace Woolman. Dana had been in the Bacteriology Department, and he started in Plant Pathology with a 50-50 split appointment in the two departments.

In the 1917-1918 college catalog Heald is listed as the teacher of all the courses: General Plant Pathology; Crop Diseases (selected diseases important in the Northwest); Fungi of Plant Disease; Methods; Principles and Practices of Disease Control; Forest Pathology; Diseases of Cereal and Forage Crops; Diseases of Fruits; plus problems and research. In the 1918-1919 catalog, Dana helped with teaching, and in the 1919-1920 academic year Dana added a course of his own, Diseases of Truck Crops. In 1924-1925 Dana was still an Instructor and he and Heald were still the only members of the academic faculty in plant pathology. As if this weren't enough teaching, correspondence courses were offered in 1918 or 1919 (Extension Service Bull. 48). Plant Pathology offered four courses, the most of any department.

1. Introduction to Plant Pathology. \$5.00. Taught by F. D. Heald and B. F. Dana. "Fungous Diseases of Plants" by B. M. Duggar can be used (not required) as a text. Available, along with other supplies, at the Students' Book Company, Pullman. All bulletins furnished must be returned unless marked for keeping.
2. Advanced Plant Pathology. \$5.00. F. D. Heald. No text required.
3. Fruit Diseases. \$4.00. F. D. Heald. Text, "Manual of Fruit Diseases" by Hessler and Whetzel.
4. Diseases of Truck Crops. \$5.00. B. F. Dana. Text, "Diseases of Truck Crops and Control" by J. J. Taubenhaus.

In retrospect, research was a minor activity, with one or two persons teaching entire programs in one or several general subjects.

Comparing growth in university income with teaching loads shows little relationship between the two figures. The total income of WSU during the 1911-1913 biennium was \$824,390. The teaching load of Professor Humphrey was heavy in 1913. The teaching load was heavy when Dr. Heald taught all of the Plant Pathology courses in 1917-1918 and total income of the college was \$1,521,701. In 1924-25 when Heald and Dana did all the teaching total income for the biennium was \$2,564,776. President Holland saw no problem with expanding the University into new academic areas with little strengthening of

existing units. Little effort was made to provide time for research. A comparison with the situation at the University of Wisconsin is instructive.

Research was important from the beginning within the College of Agriculture at Wisconsin, with the teaching load per faculty member kept at a minimum. Mycology was part of Botany. Fewer courses were offered at Wisconsin than at WSU, the faculty was larger, and the faculty was stable, as judged by persons present in 1919 (L. R. Jones 1910-1935, A. G. Johnson 1912-1919, G. W. Keitt 1914-1959, R. E. Vaughan 1914-1949, J. W. Brann 1916-1947, J. G. Dickson 1919-1961, and J. C. Walker 1919-1964). The only person who didn't serve until death or retirement was A. G. Johnson, who left to lead cereal disease research in the USDA at Washington, D.C., in 1919. Keitt taught diseases of fruit crops, Dickson diseases of field crops, and Walker diseases of vegetables all their professional lives, another form of stability, whether good or bad. Wisconsin attracted good workers and held them.

Plant Pathology courses at the University of Wisconsin, 1918, 1925, and 1948.

Pathology	Instructors		
	1918	1925	1948
1 Elementary	Vaughan	Vaughan	Arny
2 Diseases of Plants	Jones	Jones	Backus
3 Methods	Jones	Keitt	Riker
4 Diseases of Field Crops	Dickson	Dickson	Dickson
5 Diseases of Orchard Crops	Keitt	Keitt	Keitt
6 Diseases of Vegetable Crops	Jones	Walker	Walker
Seminar	Jones	Jones	Keitt
<u>Mycology (In Botany Department)</u>			
7 Mycology	--	Gilbert	Backus
8 Advanced Mycology	--	Gilbert	Backus

In 1919 Heald, Dana, and Extension Specialist Zundel served at Pullman, USDA pathologist Fisher at Wenatchee. D. J. Crowley was hired in 1923 as a cranberry production leader at Long Beach. Most of his research had to do with culture of cranberries but he experimented with fungicides, published some articles on cranberry diseases, and was at times classified as a pathologist. He worked for many years at Long Beach. Peter Bristow believes he was first to use water sprays on any crop to control frost damage to blossoms.

E. E. Honey was hired at Pullman in 1925, making Professor Heald, Assistant Professor Dana, and Instructor Honey the academic faculty. In the 1926 catalog, Introductory Plant Pathology was taught by Dana and Honey. Fungi of Plant Disease (two courses), Methods, and Diseases of Fruits were taught by Honey. Dana taught Principles and Practices of Disease Control, Diseases of Truck Crops, and Diseases of Cereal and Forage Crops. Heald taught Forest Pathology. Honey stayed only one year.

Glenn Huber, Teaching Fellow, who received his Ph.D. in 1931 at WSU, replaced Honey in 1926. Dana and Huber taught four courses together, Dana taught four courses alone, and Heald taught Forest Pathology. Dana, who started in Plant Pathology in 1918, resigned as Assistant Professor in 1927.

George D. Ruehle was appointed Assistant Plant Pathologist in 1926. He surely assisted in teaching.

Heald's "Manual of Plant Disease", one of the most scholarly books on plant pathology ever written, was published in 1926. It was revised in 1933. It contains complete treatments of major plant diseases as well as detailed early histories of each disease, much of it translated from early European studies, mainly German. This book led to a shorter "Introduction to Plant Pathology" that served for many years in many countries as the text for introductory plant pathology courses. I used Heald's text as an undergraduate

student at the University of Arkansas in 1940. Heald's books brought much prestige to Washington State University during the long period of their use.

In September, 1927, Lee E. Miles, a Ph.D. from the University of Illinois, replaced Dana. Miles and Huber taught three courses together, Miles taught five courses alone, Huber taught one course alone, and Heald did no teaching. Miles resigned after about one year. G. A. Newton (BS, 1925; MS in 1927, WSU) was sent as pathologist to Puyallup, the first there for some years. E. L. (Bud) Reeves was appointed Field Assistant by the USDA at Wenatchee.

In 1928 Leon K. Jones, a PhD from Wisconsin, replaced Miles. Jones and Huber taught three courses together, Jones taught two courses alone, Huber taught one course alone, and Heald taught Forest Pathology. Dr. H. H. Flor was sent by the USDA to Pullman in 1928 to work on smut.

George D. Ruehle received the Ph.D. in Plant Pathology in 1930, the first Ph.D. granted in any subject by Washington State University.

In 1931 only Heald and Jones were listed as teaching faculty. Jones is listed as instructor in seven courses, Heald one, and the instructor of one course was not given. Flor was transferred to North Dakota to work on flax rust and C. S. Holton, USDA, was his replacement.

In 1932 and 1933, teaching was mainly by Jones and G. A. Huber.

In 1934 Huber was appointed pathologist at Puyallup and George W. Fischer, University of Michigan, was hired half-time as Instructor. Fischer taught four courses, Jones five, and Jones and Fischer shared one. Fischer resigned in 1936 to work full-time for the USDA at Pullman as a grass pathologist, specializing in smuts. W. Harley English, who received his Ph.D. in Plant Pathology at WSU in 1940, was hired as Instructor to replace Fischer on the state faculty. In 1937, 1938, and 1939 Heald taught the control course and shared two with Harley English. Jones and English taught the rest of the courses. Jack Meiners wrote that he took beginning plant pathology from Heald in 1938. Harley English was the lab instructor. Heald suffered ill health and took a leave-of-absence July 1937-June 1938. S. M. Dietz, Sr., was hired from Iowa State University as Head during this period. In 1939 English resigned to work full-time for the USDA in fruit pathology at Wenatchee.

Leo Campbell, who received his Ph.D. at WSU in 1935, worked at Bellingham on vegetable diseases until 1937 when he became vegetable pathologist at Puyallup. He served there until retirement. Richard Wellman, who received his Ph.D. from WSU in 1939, served one year as the first pathologist at Prosser (1938). Wellman was replaced in 1939 by James D. Menzies, who received his Ph.D. at WSU in 1943. Menzies served as a state pathologist, 1938-1945, except for periods as a student at Pullman, and then as a USDA pathologist, 1945-1959, at Prosser, the first "long-time" pathologist at Prosser.

Don M. Coe, who received his Ph.D. in Plant Pathology at WSU in 1943, did much of the teaching in Plant Pathology during 1940 and 1941. Don Coe was an excellent teacher according to Jack Schafer. Coe served as a full-time teacher while a graduate student. Jack Schafer wrote that he took beginning plant pathology from Dr. Heald during this period. The course was well organized, intensive and difficult. "Dr. Heald gave the lecture and Jim Menzies, then a Ph.D. candidate, taught the laboratory. There was much substance in Dr. Heald's course, but he was a very dry lecturer." Schafer commented that students with no particular interest in the subject did very poorly, many getting D's. Jim Menzies was a stimulating, excellent teacher. He never held an academic appointment.

According to the 1941 college catalog, Heald taught two courses, Coe five, and Leon K. Jones one. Heald was forced to resign in 1941.

*Advanced Degrees in Plant Pathology, 1917-1941*

Heald (1915-1941) and Jones (1928-1944) both had PhDs (Heald from Leipzig; Jones from Wisconsin) and they directed all advanced students during the Heald Period. Heald taught few formal courses during most of this period, but he was heavily involved in directing graduate students. By today's standards it is difficult to understand how a department with two Doctors of Philosophy could offer the PhD, but Heald was an intellectual giant and Jones was good.

In 1934 the American Council on Education surveyed universities for departments able to give credible graduate degrees. At WSU only Plant Pathology qualified (Landeem, p. 338).

*F. D. Heald's Students*

Walter J. Bach, MS, 1922; Charles S. Parker, MS, 1923; L. W. Boyle, MS, 1924; Carl H. Spiegelberg, MS, 1925; Roderick Sprague, MS, 1925; George A. Newton, MS, 1927; Glenn A. Huber, MS, 1929; Jess R. Kienholz, MS, 1929; George D. Ruehle, PhD, 1930; Glenn A. Huber, PhD, 1931; Kenneth A. Baker, PhD, 1934; Leo Campbell, PhD, 1935; Otto F. Schnellhardt, MS, 1935; E. L. Reeves, MS, 1937; Richard H. Wellman, PhD, 1939; W. Harley English, PhD, 1940; Carl W. Boothroyd, MS, 1941; and Sylvan L. Cohen, PhD, 1942.

*L. K. Jones' Students*

Grover Burnett, MS, 1929, PhD, 1932; Kenneth J. Kadow, MS, 1933; Earl J. Anderson, MS, 1934; Folke Johnson, PhD, 1939.

*Budget Factors*

Budgets for Washington State University expanded gradually from 1917 to 1927, and then declined into the great depression.

Biennium	State appropriation	Total income from all sources
1915-1917	833,836	1,232,122
1917-1919	971,102	1,521,701
1919-1921	1,763,540	2,500,132
1921-1923	1,742,086	2,564,776
1923-1925	1,796,654	2,785,162
1925-1927	2,503,203	3,941,071
1927-1929	2,450,092	3,693,146
1929-1931	2,270,522	3,601,523
1931-1933	2,050,207	3,222,919
1933-1935	1,448,636	3,147,306

The high turn-over in faculty at Pullman was due both to low salaries and to heavy teaching loads. The retirement system at WSU started in 1941 (Landeem p. 215).

Much of the high turn-over of faculty at Pullman was the result of employing graduate students as teachers (D. C. George, Glenn A. Huber, Harley English, Don Coe). They worked for low salaries that substituted for present-day assistantships. The departures of Dana, Honey, and Miles resulted from other factors. George Fischer only had a half-time salary (1934-1936) so he would naturally leap at the chance for a full-time salary with the USDA. The only conclusion I can make is that the budget for the Plant Pathology Department at Pullman was so low it at no time could support three full-time professors during



the entire Heald period. Heald labored to maintain a good graduate program under oppressive conditions. His scholarly books, his collection of reprints and books, and his direction of graduate students were his greatest contributions to WSU and the world.

#### Identifiable Appropriations for the Agricultural Experiment Stations

The annual reports, 1892-1905, list only the \$15,000 annual Hatch funds. The 1906-1910 reports likewise account only for federal money, Hatch (\$15,000 per annum) now augmented by Adams funds which started at \$5,000 and increased to \$15,000 by 1911. In the 1911 report President Bryan (also Director Bryan) noted that the state supplied \$10,000 for printing bulletins, renting land, etc., and that it furnished janitorial services, heat, electricity, water, etc., for experiment station activities. In the Piper and Beattie periods the experiment station at Pullman was supported mainly by federal funds, if these reports are true. In 1915, \$15,000 Hatch, \$15,000 Adams, and \$10,000 state support are listed.

In 1916 Director Cardiff published his criticism of the low level of state funding. Starting in 1917 state appropriations for agricultural research surpassed the \$30,000 from the federal government for the first time (state support \$33,964, increasing to \$58,176 in 1918). Starting in 1919 and continuing until 1946, the Pullman budgets included all money appropriated for all stations (Pullman, Lind, Prosser, etc.) exclusive of Puyallup, which had its own state appropriation. From 1919-1929 state support for agricultural research exceeded federal support, and state support for Puyallup about equalled state support for the other stations. Puyallup was not favored as much as parity in state appropriations indicates. Federal funds were all spent at the land-grant institution (Pullman). In 1922 federal funds were \$30,000, state support for Pullman and satellites was \$55,385 and for Puyallup, \$49,289.

During the depression federal support for agricultural research either held steady or increased while state support declined to disaster levels in some years.

	Federal	State	
		Pullman <sup>a</sup>	Puyallup
1931	90,000	46,799	38,591
1932	90,000	34,439	57,820
1934	90,000	13,193	14,560
1935	90,000	39,549	39,506
1936	97,378	16,432	38,798
1937	104,757	25,789	22,970
1938	112,136	89,171	71,569
1939	115,826	103,664	75,350

<sup>a</sup> includes outlying stations, exclusive of Puyallup.

Federal funds saved the research staff during the 1931-1937 period. The federal government used deficit spending. The state by its constitution cannot spend in excess of receipts.

At the risk of beating a dead horse, I add the following. Arkansas went through the same depression, yet its five full-time professors of plant pathology (V. H. Young, E. M. Cralley, H. R. Rosen) and of botany (Delbert Schwartz and Dwight Moore) at the time I was a student (1937-1941) worked their entire careers at Arkansas. The University of Arkansas offered three courses in the Plant Pathology Department and mycology was taught by the Botany Department. They did not offer the Ph.D. degree until at least 15 years after I left the University of Arkansas. Why did WSU grant the PhD degree when it had so small a faculty? I must add, that the recipients of advanced degrees in Plant Pathology who studied under Heald and Jones had professional records equal to those of graduates of larger faculties and they were obviously very well trained.

## Obituaries

Frederick Deforest Heald, 1872-1954, excerpted from K. F. Baker and G. W. Fischer. *Annu. Rev. Phytopathology* 21:13-20.

F. D. Heald was born in Midland City, Michigan in 1872. He received the B.S. (1894) and M.S. (1896) in Botany at the University of Wisconsin. He received the Ph.D. in Plant Physiology from W. F. P. Pfeffer in 1897 (three years from the B.S. with the final degree in German!). His son, Henry T. Heald served as President of Armour Institute of Technology, Chancellor of New York University, and finally as President of the Ford Foundation.

Heald often used his own funds to purchase items for the department - he certainly subsidized the fine library. Heald was a fine illustrator, both in photography and sketching. He was formal by present standards, wearing a coat or lab coat at work and a suit coat at home. Heald took six months leave without salary to finish his *Manual of Plant Diseases*, and, according to Baker and Fischer, "it is unlikely that the royalties ever equaled this lost income." He did not like scientific meetings and seldom attended them. A motto on his desk, late in his career, "Work like Helen B. Happy." He died in Spokane Washington, in 1954 at the age of 82.

Edward Franklin Gaines, 1886-1944, by H. B. Humphrey and F. J. Stevenson. *Phytopathology* 36:87. Gaines, though agronomist and wheat breeder, is included because of his extensive efforts to develop wheats resistant to bunt. He was born in Missouri but moved near Chewelah in the Colville Valley at 14. He graduated from the State Normal School (= teachers' college) at Cheney in 1907. He received a BS in 1911 and a MS in 1913 at WSU. He was assistant cerealist while a graduate student and he spent one summer with E. M. East at Harvard in 1915. He returned to Harvard in 1920 and received a D. Sc. degree from Harvard in 1921. In 1930 he travelled 6 months in Europe (President Holland was so impressed with Gaines that he arranged additional financial assistance for this travel). He was Professor of Agronomy at WSU until his death. He published extensively with E. G. Schafer, Jack Schafer's father. O. A. Vogel, a student of Gaines, named his first semi-dwarf wheat Gaines, followed by Nugaines, in his honor. Dr. James G. Dickson, who received his B.S. from WSC and was my major professor at Wisconsin, spoke very highly of Dr. Gaines.

Lee Ellis Miles, 1890-1941, by David C. Neal, *Phytopathology* 32:267.

Miles graduated from Wabash College with an AB degree in 1914, and he became a machine-gunner in the Indiana National Guard during World War I. In 1920 he received his Ph.D. from the University of Illinois, with a thesis on leaf spots of the American Elm. He worked for the Mississippi State Plant Board, 1920-1922, trying to insure pathogen-free nursery stock. He then worked at Auburn, Alabama until 1927 when he came to Pullman. After one year he returned to the Mississippi Agricultural Experiment Station to work on southern crops for the rest of his rather short life. He became a Fellow of the American Association for the Advancement of Science.

Leon K. Jones, 1895-1966, by Folke Johnson. *Phytopathology* 57:331.

Leon K. Jones was born in Seattle. He graduated from Oregon State University with a BS in 1917 and served one year in the army. He received a MS in 1921 and the PhD from the University of Wisconsin in 1922. He worked on diseases of fruits in Wisconsin, 1922-1925, and on diseases of vegetables at Cornell, 1925-1928. He came to WSU in 1928 as assistant plant pathologist and assistant professor, and was promoted to associate rank in 1929. One of his biggest contributions in Washington was proving that raspberry mosaic could be controlled by roguing. He joined the Plant Quarantine Service, USDA, in 1944 and served in Seattle and Hawaii until retirement in 1962. His mother was the third white child born in Seattle.

### ***J. G. Harrar and Earl J. Anderson, 1941-1945***

Heald was replaced in 1941 as Head by J. G. "Dutch" Harrar (1942-1943), University of Minnesota, who in turn was replaced by Earl J. Anderson (1944-1945), University of Maryland. Harrar taught seven courses alone in 1942 and co-taught three with Coe. Coe left to start the virus-free fruit tree station at Moxee. Harrar left in 1943 to head agricultural research for the Rockefeller Foundation, contributing to world-famous work with wheat, potato, and corn in Mexico. Earl Anderson taught seven courses in 1943. Three others were listed as being taught by the "staff". I do not know whether Heald, who was listed Emeritus Professor, taught, but Leon Jones surely helped. Jones resigned in 1944. Leon Jones (1928-1944) was a long-time state employee at Pullman during the Heald period. He at times escaped a heavy teaching load and accomplished significant research. Fischer (USDA) and Holton (USDA) were long-time employees at Pullman. In 1945 Anderson was the only person listed on the teaching faculty, except for Heald, who was listed Emeritus. Seth Barton Locke was hired in 1945. Anderson resigned in 1945 to go to the Hawaiian Pineapple Growers' Inst.

Harold B. Busdicker, U. S. Bureau of Entomology and Plant Quarantine, came to Pullman in 1944 to head the barberry eradication program and other activities of this unit.

In contrast to Pullman, the faculty at Puyallup (W. B. Courtney, USDA nematologist, 1933-1963; Glenn Huber, 1934-1943, and Leo Campbell, vegetable pathologist, 1935-60), was stable. Karl Baur served both as Assistant Pathologist and Assistant Soil Scientist until 1944 when he was assigned full-time to soils. Charles J. Gould, specialist in diseases of ornamentals and turf grasses, was hired in 1941 and he served at Puyallup until retirement in 1977. In 1945 the faculty at Puyallup included Courtney (USDA), Campbell, Gould, and Folke Johnson. Folke specialized in diseases of small fruits. He served from 1943-1973. The Puyallup station was independent of the main station until 1946, with its own budget and director and the plant pathology faculty at Puyallup (Bill Courtney, Glenn Huber, Leo Campbell, Chuck Gould, Folke Johnson) was greater during most of this period than the faculty at Pullman (Stew Holton and George Fischer, USDA, Leon K. Jones until 1944, plus state personnel coming and going). There never was high turn-over of faculty at the branch stations or in the USDA. Misery was concentrated among state personnel at Pullman.

J. D. Menzies, who received his Ph.D. at WSU in 1942, was a mainstay at Prosser. In 1945 he switched to the USDA, but remained at Prosser. A severe problem in the cabbage seed industry caused the USDA to move Glenn Pound to Mt. Vernon, 1944-1946. Glenn Pound, who subsequently went to the University of Wisconsin became president of APS in 1959.

### SALARIES

Landeen (p. 219) concluded that low salaries at WSU (up to 1944) were a tradition. When Holland became president in 1916, salaries ranged from \$800 to \$6,000, with department heads receiving \$1,600-\$4,000 per year. In 1918 WSU offered new faculty \$1,600-\$2,000 per year, the lowest salaries found in a survey of land-grant institutions. In 1920 County Agents received \$600-\$1,620 per year, and over half of them quit to enter some other business. After the loss of Professor Melander of Entomology in 1925, the faculty commented that salaries should be high enough to keep at least one person of national reputation in each department in fairness to students (1926, p. 205), and a definite salary schedule should be established and it should be accessible to all faculty. In 1943 important agricultural groups in the state believed poor research was related to poor salaries and they offered their help in seeking added funds. President Holland did not welcome or encourage their help. It would disturb the "balance" within the University.

Salaries within ranks were lower than at the University of Washington on an annual basis, even though most WSU years were 11 months; UW, 9 months. To aggravate the discrepancy, in 1943 the University of Washington had twice as many full professors as instructors, WSU had almost twice as many

instructors as full professors. In other words, salaries were lower for 11 months at WSU than for 9 months at UW, and most of the faculty at WSU was in the junior ranks, whether due to slow promotion or to high turn-over. I suspect relatively few people stayed at WSU long enough to advance far in rank.

Landeem (p. 225) believed that expansion into new activities at no time took enough money to affect the salary structure. This is subject to question. Units are additive, cumulatively over time, and each uses money. The 1943 Salary Committee requested a halt in added activities until the salaries could be raised (Landeem, p. 217). Frequent departure of faculty reduced productivity, in research more than in the class room.

Salaries, advancement procedures, tenure, retirement, sick leave, health plans and vacation times are now good to excellent, and the faculty has been stable since 1946. In addition, the facilities, including those at the branch stations, are now of high quality, well equipped and well maintained.

### ***The George W. Fischer and J. Walter Hendrix Period, 1946-1960***

In 1946 the Plant Pathology Department began to grow. Until then there were never more than three state pathologists at Pullman, more often than not, two or less. It is illogical to attribute this to hard times. There had to have been some good times in 50 years.

Wilson M. Compton replaced Holland as President of WSU in 1945 and he served until 1951. President Compton came from a distinguished scientific family and he appreciated the need for time, facilities and money to do research. One of his brothers was President of Massachusetts Institute of Technology (a land-grant institution) and the other was president of Washington University in St. Louis. It is my belief that the stalemate in number of faculty within units was broken by the change of presidents. If my assumptions be true, do not look too harshly upon President Holland. According to the WSU Foundation Annual Report, Hill Topics 19 (6): p. 5, 1988, President Holland is listed among those who contributed over \$100,000 to WSU. These were much bigger dollars than those of today. He gave total devotion to this institution, both of his energy and of his resources.

C. Clement French (1952-1966) succeeded Compton as president, and progress within the department continued. President French, a physical chemist, supported balanced growth of colleges within the University and balanced growth of research, teaching, and extension within the College of Agriculture.

According to Jack Schafer, a native of Pullman who received his BS from WSU and whose father was chairman of the Agronomy Department, the chairmanship of Plant Pathology was offered to George W. Fischer in 1941 but Fischer declined. F. D. Heald wanted to remain as chairman. The retirement program at WSU was nil at that time, and Schafer suspects finances were a strong factor behind Heald's desire to remain, but he was removed from office. Sam Dietz, whose father was chairman of Botany and Plant Pathology at Oregon State University, told me conditions at OSU were similar. Sam's mother suffered from lack of funds in her later years.

In 1946, following the turmoil of 1941-1945 at Pullman, Fischer resigned from the USDA and became department chairman. In 1946 George Nyland, who received his Ph.D. in Plant Pathology at WSU in 1948, joined the teaching faculty. Schafer took Advanced Mycology from Fischer, Mycology, General Plant Pathology and Forest Pathology from Fischer and George Nyland (1946-1948), Diseases of Horticultural Crops from Seth Locke, and Diseases of Ornamentals, Farm Crops and Advanced Plant Pathology from the staff (Fischer, Locke, Nyland). Mycology courses as such (called Mycology) started with Fischer, and Mycology is taught within the University by the Plant Pathology Department.

In 1946 Marion R. Harris (University of Wisconsin) became the first Extension Specialist in plant pathology since George Zundel left in 1926. The extension specialist lightened the load of the Pullman staff. For some years extension specialists were housed together because of ease of coordinating travel.

This arrangement was terminated and the specialists are housed within the departments of their specialties, strengthening both the extension specialists and the subject matter departments. Department Chairmen play the leadership role in determining tenure, promotion and salary adjustments for extension specialists.

Dr. D. M. McLean (USDA) replaced Glenn Pound (USDA) at Mt. Vernon on vegetable seed pathology. Frank V. Stevenson, hired in 1946, served one year at Prosser. A leading tree-fruit pathologist, Earle C. Blodgett (University of Wisconsin) was hired jointly by the Washington State Department of Agriculture (WSDA) and WSU to work primarily on virus-free fruits at Prosser. He supervised the virus-free Plant Introduction and Quarantine Station at Moxee, several miles west of Prosser.

In 1947, a famous grass pathologist, Roderick Sprague (University of Cincinnati), C. Gardner Shaw (University of Wisconsin), and Avery E. Rich (1947-1952, Ph.D. Washington State University, 1950) were added to the Pullman faculty. R. C. Lindner, a biochemist, was hired at Wenatchee to study virus diseases of tree fruits. C. M. Wright was hired by the WSDA to work with Blodgett at Prosser. The state had a net gain of four pathologists in 1947.

In 1948 George Nyland left for California. C. M. Wright transferred from Prosser to replace Nyland on the Pullman faculty. Shirl O. Graham replaced C. M. Wright as assistant to Blodgett at Prosser. H. C. Kirkpatrick (USDA) joined Lindner at Wenatchee, strengthening tree fruit virus research there. Kirkpatrick made a net gain of one.

In 1950 Sprague transferred to Wenatchee to study fungal and bacterial diseases of apples and pears and to continue his life-long interest in fungal diseases of cereals and grasses, exclusive of smuts and rusts. Meiners replaced Sprague at Pullman. Jack P. Meiners, who received his Ph.D. at WSU in 1949, took Research and Seminar from Fischer and Advanced Mycology from Shaw, Diseases of Field Crops from Sprague, and Diseases of Horticultural Crops from Locke. Shirl Graham resigned his job with Blodgett at Prosser to become a graduate student at Pullman. J. G. Barrat replaced Graham at Prosser and T. O. Diener was added to the Prosser group, making a gain of two within the state. T. R. Wright is listed as Associate Pathologist, USDA, Wenatchee by Fischer, but I do not know when he came or what he did.

Maksis Eglitis, a refugee from Latvia with a Doctor of Science and a distinguished career in Europe, joined the staff at Puyallup as Research Associate (1951-1962). He was particularly interested in the use of high energy radiation to kill organisms, scarify hard seed coats, etc.

J. Walter Hendrix (Univ. of Minnesota) came to Pullman from Hawaii in 1952 (until 1982). He taught the beginning course in plant pathology for most of his career and for several years he taught an advanced course on disease control. Walt Hendrix worked first with stem and stripe rust, then extensively with students from Brazil, Argentina and Chile, stressing septoria leaf blights of wheat and seed-borne fungi. He served as Acting Chairman 1958-1960. In 1952 Charles F. Pierson (Univ. of Wisconsin) came to Wenatchee (USDA) to study storage diseases of fruits (until his retirement in 1982). A gain of two.

The year 1953 was a big one at Pullman. Common bunt was devastating. Jack Meiners switched from state to federal, Edgar L. Kendrick (U. of Wisconsin) and L. H. (Hank) Purdy (U. of California, Davis), all joined C. S. Holton to form the famous USDA Pacific Northwest Cereal Smut Research Laboratory. Shirl M. Dietz, Jr., joined this unit in 1954, resulting in Holton, Meiners, Purdy, Kendrick, Dietz, and Jackson T. Waldher all concentrating on smuts, mainly common bunt, dwarf bunt, and flag smut. Holton continued his life-long leadership of oat smut research in the U.S.

George W. (Bill) Bruehl (1954-1984), University of Wisconsin, replaced Jack Meiners on the Pullman faculty. Bruehl taught Diseases of Field Crops until a revision of courses eliminated crop-oriented courses. After that he taught a general plant pathology course. Bruehl was responsible in his early years

for cereal diseases exclusive of smuts and rusts (Fischer, Purdy, Hendrix, and finally Roland Line studied the rusts and Holton, Fischer, Kendrick, Purdy, Duran and Hoffmann studied the smuts at some time during Bruehl's tenure). Bruehl was president of APS in 1977.

When Bruehl came to WSU, his laboratory facilities in Wilson Hall were in a general area, used by several in addition to being the space for the coffee club. This played a role in his emphasis upon barley yellow dwarf and some other virus diseases, dependent primarily upon greenhouse and field experiments, before the move to new facilities in Johnson Hall.

The Columbia Basin Irrigation Project was advancing rapidly, making irrigated agriculture increasingly important, resulting in strong growth in plant pathology at Prosser. I am going to depart somewhat from chronology and present part of this growth. Murit D. Aichele, WSDA (Washington State Department of Agriculture), 1955-1971, joined E. C. Blodgett. Paul Fridlund (1955-1988) (University of Minnesota) took charge of the new IR-2 project at Prosser and Moxee, becoming responsible for obtaining, maintaining and providing virus-free stone fruit tree propagative materials for the U.S. Douglas W. Burke, USDA, a WSU graduate, became dry edible bean (*Phaseolus vulgaris*) pathologist and breeder (1956-1984). He was also administrator of the USDA scientists at Prosser (1972-1984). Calvin B. Skotland (1956-1988) became a general pathologist, with special interests in verticillium wilt of peppermint and in obtaining virus-free hops. Richard O. Hampton (Iowa State University) worked with the Washington State Department of Agriculture and WSU at Prosser, 1957-1961, mainly on virus diseases of fruits. William G. Hoyman, USDA, 1957-1972, served as a potato breeder-pathologist, and in 1959 Lindsey R. Faulkner (1959-1972) (1974-present), University of Wisconsin, served as nematologist, the first nematologist hired by the state. He first drove to Pullman to teach nematology during the school year, but this was too strenuous. He then started a summer course in nematology at Prosser, with students using sleeping and eating quarters on the Prosser station. The personnel at Prosser do everything possible to make the students comfortable. In a short period six pathologists were added at Prosser and from this time on the Irrigated Agriculture Research and Extension Center at Prosser has been strong in plant pathology.

In 1956 (-1962) Walter Apt, a WSU grad, went to Puyallup to serve as nematologist. He received special training by association with William D. Courtney, USDA, before the latter's retirement in 1963. Apt went to the Hawaiian Pineapple Research Institute when he left Puyallup.

In 1957 Shirl (Sam) M. Dietz, Jr., left the USDA smut unit to work in the Plant Introduction Unit of the USDA at Pullman, a unit he still leads.

Ruben Duran, a WSU graduate, was hired in 1958 as a mycology teacher and smut expert. He left after a year to work with the USDA on fruit diseases at Riverside, CA, but returned to WSU in 1961 and stayed until retirement (January, 1989). Duran carried a significant teaching load during his entire career at WSU, teaching General Mycology, Lower Fungi, and Basidiomycetes. His career peaked with publication of a wonderfully illustrated book on the smuts of Mexico (1988).

In 1958 James A. Hoffmann, a WSU grad, joined the USDA smut research unit. Hoffmann performed research of the highest quality on dwarf bunt, especially on races of *Tilletia controversa*. Hoffmann left Pullman in 1972 to continue his dwarf bunt studies at Logan, Utah, until his retirement in 1987.

Western Washington received a boost in 1960 when Richard (Dick) L. Gabrielson (University of California, Davis) joined the faculty at Puyallup and William (Bill) A. Haglund (University of Minnesota) went to Mt. Vernon. Gabrielson specialized in diseases of vegetables, especially in producing pathogen-free vegetable seeds. Haglund worked with green peas for processing, specializing in fusarium wilt of peas, nematodes on local crops, and pea root rots in humid conditions in general.

Fischer stressed social life within the department at Pullman. He and his wife hosted the entire group (faculty, staff, and students) at their country home regularly. The Locke's also strongly supported social

affairs. When Shaw became chairman he attempted to keep this tradition alive, but on a reduced level. It has gradually dropped to a low level.

Of the 17 faculty hired between 1893 and 1920 only C. V. Piper, D. F. Fischer (USDA, Wenatchee) and F. D. Heald stayed for 10 years or more. Of 16 hired between 1921 and 1940, seven stayed 10 years or longer. Of those, E. L. Reeves, W. D. Courtney, and C. Stewart Holton were federal, G. W. Fischer and James D. Menzies served part of the time in the USDA, part in WSU. Leon K. Jones (1928-1944) and Leo Campbell (Mt. Vernon and Puyallup), were employees of WSU.

Of 40 persons hired between 1941 and 1960, 29 stayed 10 or more years. Chuck Gould, Folke Johnson, Dick Gabrielson, WSU at Puyallup; Bill Haglund, WSU, and D. M. McLean, USDA, Mt. Vernon; Earle Blodgett, Ted Diener, Paul Fridlund, Cal Skotland, and Lindsey Faulkner, WSU, Murit Aichele, WSDA, Doug Burke, and Bill Hoyman, USDA, all at Prosser; Bob Lindner, Rod Sprague, WSU, Hugh Kirkpatrick and Charlie Pierson, USDA, Wenatchee; Seth Locke, Gardner Shaw, Shirl Graham, Walt Hendrix, Bill Bruehl and Ruben Duran, WSU, and Harold Busdicker (USDA), Marion Harris (Extension), Ed Kendrick, Hank Purdy, Jim Hoffmann, USDA, and Sam Dietz (Plant Introduction), Pullman, are all long-time employees, using a 10-year minimum as the criterion.

Since 1961 there have been essentially no short-term state faculty, with the exception of those with temporary assignments (Michael Pratt, Carolyn Roybal Allan, and Debra Inglis) who taught beginning plant pathology during the overseas leaves of Gardner Shaw.

Johnson Hall, our present quarters, was dedicated in 1959. Each of us planned our own laboratory space in Johnson Hall. We had excellent facilities for the first time and we were happy. We included the best teaching rooms in the building, space for the mycological herbarium, and a departmental library. Seth Locke devoted much effort to the department library to which Dr. Heald had donated his extensive personal library. It included rare books and reprints of early, classic studies. The library has been enlarged by personal materials from Roderick Sprague, C. S. Holton, George Fischer, Gardner Shaw, and Jack Schafer, as well as by constant solicitation. For a time the university supported the departmental library by providing a part-time librarian. The department library contained tables for study.

The entire Pullman contingent is housed in one half of the third floor of Johnson Hall. We made a serious error, however. The Horticulture Department planned grandly, obtaining far more space within the building than they needed. Forestry, Entomology and Plant Pathology planned conservatively. As our department grew our initially spacious facility became cramped. Our conference room became part of our bacteriology lab. When the library was combined within the George W. Fischer agricultural library in Johnson Hall the library space was used to house graduate students. When Lee Hadwiger joined the faculty (1965) our general preparation laboratory became the plant physiology laboratory of Dr. Hadwiger. Under the leadership of R. J. (Jim) Cook, the Root Disease and Biological Control Unit of the USDA has recently become crowded. (Dave Weller and Linda Thomashow have significant space requirements.) The space situation will not be solved until the Entomology Department moves to a new building, probably in 1992. When you have an opportunity to plan new facilities, plan for growth.

Since World War II teaching loads have permitted scientists with joint appointments (teaching and research) to pursue research. In the 1946-47 biennium General Plant Pathology, Forest Pathology, Mycology, Advanced Mycology, Diseases of Horticultural Crops, Diseases of Ornamental Crops, and Diseases of Field Crops were taught by George Fischer, Seth B. Locke, George Nyland and Roderick Sprague. Avery E. Rich and C. Gardner Shaw were added to the teaching faculty in 1947. Shaw taught Forest Pathology and Mycology. Nyland and Rich taught General Pathology, Sprague taught Diseases of Ornamental Plants and Diseases of Field Crops. Locke taught Diseases of Horticultural Crops. Sprague and staff together taught Methods. Some of these courses were offered only on alternate years, so the teaching load per faculty member justified significant research expectations. Fischer ceased to teach after 1948.

During the period 1948-1954, courses on History of Plant Pathology, Advanced Principles and Theory, and a summer course, Field Mycology, were added. By 1954 the teachers included Locke, J. Walter Hendrix, Shaw, Charles M. Wright and Jack P. Meiners. Fischer moved into higher administration in 1958 and Hendrix served until 1960 as acting chairman.

In 1947 and subsequently the Puyallup budget was no longer separate and state funding for agricultural research far exceeds that from federal "formula" funds.

Funds for agricultural research, not for the entire University.

	Federal	State
1947	\$ 126,025	\$ 509,672
1948	151,804	933,414
1949	201,930	1,128,882
1950	238,323	1,177,435
1957	616,475	1,855,103
1958	620,911	2,371,837
1959	607,342	2,524,432

If a financial collapse occurred now, federal funds would be inadequate to stabilize the system as it partially did in the 1930's.

The Fischer-Hendrix period, 1946-1960, saw us become a full-fledged department, capable of both teaching and research on a substantial scale. Extension support was low, with a single plant pathology specialist in the state.

My conclusion that Presidents Bryan and Holland, both with liberal arts backgrounds, favored the "university" as a whole rather than agriculture, engineering and science, was confirmed by Frykman's account. Frykman, p. 271, states, "Nevertheless, Enoch Bryan has been the great spokesman for the modern university when he fought to enlarge the college's horizons, curriculum, and student body. The vision of the people's university began with him and gradually unfolded in the course of the century."

In contrast, Frykman, p. 128, records that Wilson M. Compton planned to develop "a great institute of technology...adapted to the abundant natural resources of the Pacific Northwest..." and he challenged the regents, "With your help we can build a great institution for the people of the state and provide a great service to agriculture, industry and commerce in the Northwest." Compton tried to reduce teaching loads to provide time for study, thinking and research (p. 137). Compton engaged the faculty in governing the university (p. 130). Written procedures for faculty advancement, with annual review to reward merit were established (p. 143). Sick leave, as we know it today, originated during Compton's tenure (p. 145). In short, WSU entered the 20th century with Compton, and by coincidence (?) the Department of Plant Pathology began to flourish and faculty turnover stopped.

Frykman (p. 148-149) stated that Compton thought much of the research at WSU was not worth the time or money spent on it, "but in the end he supported it."

Compton was fired by the regents.



### ***The C. Gardner Shaw Period, 1960-1972***

The remarkable growth of the Plant Pathology faculty during the Fischer period continued, with a net gain of three permanent USDA and four state pathologists from 1960 through 1972. The general orientation of the department was not changed, except for greater activity in forest pathology and strengthening of the mycological herbarium. During this period the state personnel at Pullman obtained adequate greenhouse space for the first time, including 'snow mold chambers' in the basement of the greenhouse headhouse. Greenhouse facilities for all departments that moved into Johnson Hall were supported by state and matching National Science Foundation funds.

In 1961 Raymond L. Clark (USDA) and Mathias J. Silbernagel (USDA) went to Prosser. Both are WSU graduates. Clark studied virus diseases of vegetables, including the life cycle of the vector of the curly top virus, *Circulifer tenellus*, in the vicinity of Prosser. Silbernagel joined Burke to strengthen bean research. Clark left for Iowa State (USDA) in 1968, but Silbernagel is still at Prosser (in 1989). P. C. Cheo, a biochemist-virologist (1961-1963) joined Lindner at Wenatchee to work on tree fruit viruses. Roderick Sprague, the state general tree fruit pathologist at Wenatchee, died suddenly of a heart attack in the spring of 1962. Sprague worked in what was the attic of the US Weather Station on the Tree Fruit Experiment Station grounds. His facilities were poor. He subscribed to several journals, providing his own library. In 1950 he published his monumental work on "Diseases of Cereals and Grasses in North America (Fungi, Except Smuts and Rusts)". Sprague devoted much effort to snow molds of winter wheat in Douglas County. This project was taken over by Bruehl in 1962. Sprague's life long study of grass leaf spots died with him.

In 1962 Ron P. Covey, Jr., University of Minnesota, became the pear and apple tree pathologist at Wenatchee, replacing Sprague. Covey stressed fire blight, powdery mildew, scab of apple, and *Phytophthora cactorum* collar rot of apple. USDA workers at Wenatchee studied apple physiology and storage rots in the Wenatchee Post Office Building until fine facilities were built on the experiment station. Gaylord I. Mink, Purdue University, became the first true plant virologist hired by the state of Washington. Mink is stationed at Prosser where he specializes in viruses attacking tree fruits. Mink developed a summer course in "Methods of Plant Virology" at Prosser. In the beginning, Mink had better equipment (including an electron microscope) than was available within the department at Pullman. He developed an extensive ELISA service for detecting plant viruses in host tissues.

In 1963 Gene D. Easton, University of Wisconsin, became the potato pathologist at Prosser, serving the by-this-time important potato industry. Easton's position (salary) was supported by the Washington State Potato Commission for some years, a procedure used when an industry feels a strong need that cannot immediately be supported by regular state funds. Easton emphasizes verticillium wilt of potatoes. The rapid expansion of faculty at Prosser resulted in a severe laboratory and office space crisis that was solved by the completion of a spacious, beautiful building in 1963. I remember visiting Ted Diener in his laboratory in the second floor of an ancient wooden building adjacent to a cattle feeding lot and watching flies walk around on his chromatography papers while they hung to dry. Diener left in 1971 to join the USDA Pioneering Laboratory for Virus Research where he discovered viroids. Facilities at Prosser, as at Wenatchee and Puyallup, are truly fine now.

The year 1963 was also a significant one at Pullman. Otis C. Maloy (Cornell University) became Extension Specialist at Pullman, succeeding Marion Harris. Maloy at first devoted all his time to extension, but he gradually became active in research and eventually taught courses in control, advanced plant pathology, and diseases of Washington crops. Jack D. Rogers, University of Wisconsin, strengthened the Mycology curriculum, teaching a course on Ascomycetes, completing the series (General Mycology, Lower Fungi, Ascomycetes, and Basidiomycetes). Rogers teaches Forest Pathology to Forestry majors and he has a 20% appointment in that department. Rogers was President of the Mycological Society of America in 1978. Shaw offered a course in Field Mycology in which students collected and identified their specimens, but that course has been dropped.

A seed treatment, hexachlorobenzene, brought common bunt under control and the USDA transformed its smut research laboratory into the Pacific Northwest Regional Cereal Disease Laboratory. E. L. Kendrick (USDA) left to become a USDA administrator in 1965. R. James (Jim) Cook, Univ. of California, Berkeley, joined that group at Pullman as a cereal root disease pathologist. Cook rapidly developed a strong program, at first on *Fusarium culmorum* and water stress, later gravitating toward *Pythium* and biological control. He and Kenneth F. Baker wrote two books, "Biological Control of Plant Pathogens" in 1974, and "The Nature and Practice of Biological Control of Plant Pathogens" in 1983. Cook became President of APS in 1984 and of the International Society of Plant Pathologists in 1988. Lee Hadwiger, Kansas State University, joined the faculty at Pullman in 1965 as a physiologist to study the nature of disease resistance in plants. He teaches an advanced course in plant physiology and genetics. He developed the use of chitosan as a seed treatment.

In 1966 John M. Kraft, USDA, Univ. of California, Riverside and Peter E. Thomas, USDA, Univ. of Wisconsin, joined the faculty at Prosser. Kraft specializes in soilborne diseases of peas and beans. Thomas specializes in virus diseases of potatoes and tomatoes, especially curly top.

In 1967 L. H. Purdy, USDA, after making outstanding contributions in smut research, left the Pullman Regional Cereal Disease Laboratory to head the Plant Pathology Department at the University of Florida. Purdy was president of APS in 1980. Arlen D. Davison, Oregon State University, became Extension Specialist at Puyallup in 1967, giving us an extension specialist on the west side (Davison) and on the east side (Maloy). Davison served at Puyallup until 1974 when he joined extension administration at Pullman.

In 1968 Roland F. Line, USDA, Univ. of Minnesota, joined the USDA Regional Cereal Disease Laboratory to specialize in the cereal rusts. He gradually replaced J. Walter Hendrix in this field. Line works to a lesser extent on flag smut and powdery mildew of wheat. He is an authority on the use of chemicals as seed treatments for rusts and smuts and as foliar sprays for rust control (1989). Rust research at Pullman went from Fischer to Hendrix to Purdy and R. E. Allan (USDA cereal geneticist) to Line (and Allan).

J. Lewis Allison, a forage plant pathologist, became superintendent of the Prosser station, 1969-1975, succeeding Harold P. Singleton, long-time superintendent.

In 1971 Roy Davidson, WSU, was hired at Puyallup to assist Davison by running the newly established Plant Disease Clinic. The urban population of Western Washington, with its many gardeners, exceeded the ability of Davison to service the area. Davidson was the only extension aide of this type in the state. He resigned in 1988.

Gardner Shaw, while chairman, became interested in international projects. In late September-early October, 1969 he went to India with the Rockefeller Foundation as a consultant on downy mildews. He also studied chickpea diseases near Lyallpur. He returned to Pakistan January-March, 1970, to work on chickpeas, and again in 1972 for 2-2 1/2 months. In 1970 Shaw suffered a heart attack. S. O. Graham served as chairman during periods of Shaw's incapacity and during his foreign assignments.

In the 1962-1964 catalog, during early years of Shaw's chairmanship, course offerings increased. Introductory Plant Pathology and Principles and Practices of Plant Disease Control were taught by Hendrix; Forest Pathology and Ascomycetes and Fungi Imperfecti were taught by Rogers. Diseases of Plants and Diseases of Field Crops by Bruehl. Lower Fungi, Basidiomycetes and General Mycology were taught by Duran. Field Plant Pathology was taught by Duran with the help of branch station pathologists. Field Mycology was taught by Shaw. Viruses and Virus Diseases, Physiology and Variation of Plant Pathogens, Diseases of Ornamentals and Advanced Methods and Techniques were taught by Graham. Noninfectious and Bacterial Diseases, Diseases of Horticultural Crops, Advanced Principles and Theory, and History of Plant Pathology were taught by Locke. Nematodes and Nematode Diseases of Plants was

taught by Faulkner at Prosser. The nematology course is very intensive (all day and into the night), making it possible to acquire significant knowledge and skill in a short time (two weeks during the summer). Gerry Santo, nematologist from U. of Calif., Davis, hired in 1974, continued the nematology course at Prosser. Methods in Virology, taught by Mink at Prosser, is handled in the same manner as nematology. Seminar was taught by the staff. This, in retrospect, appears to be more courses than needed. If all these courses were actually taught, Graham and Locke were burdened.

The expansion of the Mycology program within the department occurred while Shaw served as chairman. The General Mycology course (Plant Pathology 421) or its equivalent satisfied the core requirements for the Ph.D. degree in Plant Pathology. The more specialized courses were taken as electives. At one time a Ph.D. in Mycology was contemplated, but it did not go beyond contemplation.

The proximity of the University of Idaho and Washington State University led to joint offerings and co-listings of courses in Bulletins of both Universities. For years, even though such offerings would increase the choice of courses, few students drove between the schools (8 miles). Recently more use has been made of courses in both schools. In the 1986-1987 biennium, Economic Nematology, Biology of Fungi, Post-Harvest Pathology, and Seed Pathology were available to our students at the University of Idaho.

Because the degrees offered by this department are in Plant Pathology, the faculty devised a minimum core of Plant Pathology courses required for the Ph.D. degree, plus a thesis, foreign language, and supporting courses. In 1988 the core curriculum for the Ph.D. degree consisted of General Plant Pathology (Murray), General Mycology (Duran), or its equivalent, Diseases of Plants (Maloy), Viruses and Virus Diseases of Plants (Wyatt), Nematodes and Nematode Diseases of Plants (Santo), Phytobacteriology (Gross), and Physiology and Genetics of Parasitism (Hadwiger), plus Seminar. There was no formal core for the MS degree, nor a language requirement, but a thesis is required. Graduate students could take Principles and Practices of Disease Control (PI.P. 503, Maloy), Methods of Plant Virus Research (PI.P. 512, Mink), Basidiomycetes (PI.P. 522, Duran), Ascomycetes and Fungi Imperfecti (PI.P. 523, Rogers), and Lower Fungi (PI.P. 524, Duran) within this department as well as those offered in Idaho as electives in the major subject if they wish. In 1989 a minimum total hours of Plant Pathology was substituted for specific courses, and the foreign language requirement was dropped.

Undergraduate horticulture and agronomy students are required to take General Plant Pathology (PI.P. 429, taught by Murray) and all forest management majors must take Forest Pathology (PI.P. 331, taught by Rogers). Diseases of Washington Crops (PI.P. 405, Maloy) is designed primarily for students in Integrated Pest Management.

No graduate course remains within the department that is based on a particular group of plants on a crop basis (vegetables, fruits, ornamentals, field crops, forages, forest, etc.).

Following the concept that a university must add to knowledge as well as teach, WSU was a college as far as plant pathology was concerned until 1947 after which research became equal to or greater than teaching as an activity within the department. Research faculty at the branch experiment stations and USDA scientists were always primarily researchers, but as a group they have contributed significantly to teaching, especially in directing research of advanced students.

During the Shaw period grant funds began to become truly significant. The NSF grant for greenhouse facilities has been mentioned (\$80,000). Pabst Laboratories contributed \$23,000 for phytoactin research. The US Forest Service contributed \$292,000 for information retrieval (White Pine Blister Rust, Wood Degradation). The NSF sponsored a summer conference on teaching plant pathology (\$15,000). The Plant Pathology Department received the first National Defense Education Act fellowships (four) in the university, each supporting a graduate student and family for three years.

## Obituaries

Roderick Sprague, 1901-1962, by C. Gardner Shaw. *Mycologia* 54:587 (amended by Bruehl). Roderick Sprague was born in Skagway, Alaska. He spent his boyhood near White Bluffs where his father grew fruit. Sprague received his BS in 1924 and his MS in plant pathology at WSU. His PhD in Botany was received from the Univ. of Cincinnati in 1929. Roderick Sprague enrolled in Plant Pathology at the University of Wisconsin in 1925 to work for his Ph.D. The policy at Wisconsin was to have all PhD students take all the advanced courses, regardless of their previous background. Sprague was so disappointed with the fruit pathology course at Wisconsin that he quit within a few weeks and transferred to the University of Cincinnati where he obtained his doctorate in Botany. Sprague told me that he knew more fruit pathology than the teacher at Wisconsin. There is no need or way to judge the validity of Sprague's feelings, but it attests to the quality of training he received at WSU. He did his MS thesis on a fruit tree disease under Heald. Sprague was a nonconformist, independent soul and this trait kept him from receiving the recognition within the profession that he deserved. He worked on grass diseases in the USDA from 1929-1947, the first 11 years at Corvallis, Oregon (OSU). He made classic studies on the epidemiology of strawbreaker footrot. He was the first to obtain spores of *Pseudocercospora herpotrichoides* in culture. Most of his epidemiological studies of foot rot were in Klickitat, Co., Washington. This fungus sporulates in the cool, wet part of the year and Sprague drove a Model-T Ford to his plots (without heaters in those days). He contracted pneumonia during these efforts and was quite sick. In Oregon he worked on foot and root rots of wheat, *Septoria* spp., *Selenophoma* spp., common bunt, grass diseases in general. He transferred to the Mandan, ND, Northern Great Plains Field Station and there he further established himself as the world's leading grass pathologist. He moved to Washington State University in 1947 where he taught Diseases of Field Crops. In 1950 he was stationed at the Tree Fruit Research Station, Wenatchee, where he worked until his death, March 17, 1962. Sprague concentrated on applied research at Wenatchee, studying orchard sprays. Snow mold of wheat was acute in Douglas County, and he did extensive research on *Typhula*, *Microdochium* (*Fusarium*) *nivale*, and he reported the presence of *Sclerotinia borealis* at about 4,000 feet elevation in the Molson-Chesaw area of Okanogan County. His home was in East Wenatchee, which is in Douglas County, about 20-30 miles from the snow mold problem. To my knowledge he never had a grant from a "scientific" granting agency, even though much of his mycology was fundamental science. His greatest single contribution was his book on diseases of cereals and grasses, other than smuts and rusts (Ronald Press, 1950).

Wilbur Doane Courtney, 1896-1965, by Gerald Thorne. *Phytopathology* 55:603. Wilbur Courtney was born in Pomeroy, Washington and he died near Sumner, WA. He served two years in World War I. Courtney received a BS from WSU in 1922 and a MS in zoology from the University of Washington in 1929. He taught zoology at Oregon State University much of the time between 1922 and 1930. He was interested in nematodes and joined the USDA as nematologist at Salt Lake City, 1930-1933, after which he came to the USDA Ornamentals Crops Station at Puyallup (this unit is now at Corvallis, Oregon). Courtney transferred to the Western Washington Experiment Station at Puyallup in 1944, and served in that institution in the USDA the remainder of his career.

Charles Stewart Holton, 1904-1980, by George W. Fischer. *Phytopathology* 71:663. Stew Holton, born in Louisiana, received his BS from Louisiana State University in 1927 and his MS and PhD degrees from the University of Minnesota in 1929 and 1932, respectively. He replaced H. H. Flor of the USDA at Pullman in 1932, and he worked on smuts here for essentially the rest of his career, except for periods of important foreign assignments. He headed the Pacific Northwest Regional Smut Research Laboratory, established in 1953, and then administered its transition to the PNW Regional Cereal Disease Research Laboratory after common bunt was controlled. Holton collaborated with George Fischer in writing the book, "Biology and Control of the Smut Fungi," 1957, Ronald Press, probably the most complete single book on this subject ever published. Holton was editor of *Phytopathology* and of the Golden Jubilee Volume, 1958. He gave an invited address at Washington State University, 1968, on "Man's Unworthy Competitors for the World Food Resources." He received the E. C. Stakman Award

from the University of Minnesota in 1971. Holton was President of the Pacific Division of APS (1958) and President of APS in 1962. He was made Fellow in 1965. He was widely respected by the wheat industry.

### ***The John (Jack) F. Schafer Period, 1972-1981***

Jack Schafer (University of Wisconsin) served as a member of the famous Purdue University small grain improvement group and as Chairman of Plant Pathology at Kansas State University prior to returning to Pullman, his original home town. Schafer was President of APS in 1979.

Schafer brought a moderating, diplomatic leadership to the department at a critical time. Graduate training was a major activity at Pullman and the Pullman faculty regretted its weaknesses (virology, nematology, bacteriology). Some of us resented the presence of a nematologist (Faulkner) and of a virologist (Mink) at Prosser while no individual so trained was on the faculty at Pullman. For several years substantial increases in faculty occurred at the outlying stations with little expansion at Pullman. During Schafer's tenure as chairman these frictions were eliminated.

Several steps were taken to lessen rancor. Faculty off-campus were urged to join the Graduate Faculty and to serve as chairmen of theses committees, becoming major professors of graduate students. Faculty who did not teach formal courses, especially those at the branch stations, were invited to participate in a course on disease control initiated by Bruehl. Response was great, even though it meant much travel and personal sacrifice by the visiting professors. In advance of Bruehl's retirement (1984), Otis Maloy taught this course in 1983. In the early years this course served its purpose in presenting off-campus pathologists to our students and making off-campus professors more an integral part of the faculty during a period of intra-departmental friction.

The College of Agriculture had long recognized the need to foster unity and coordination within departments, especially after the branch stations became strong. For many years biennial conferences were held in which state-wide faculty, both state and federal, met to discuss research, teaching, extension, departmental matters, and to get better acquainted. Bruehl was embarrassed to meet Gerry Santo for the first time at an APS meeting. Santo was a member of this department and Bruehl didn't even know him. Biennial conferences at Pullman, Prosser, Wenatchee or Puyallup enable faculty to observe facilities state-wide. The department has not yet met at Mt. Vernon. The university expects participation in these conferences but it does not provide special travel funds. Individuals take their travel expenses from their research funds.

When graduate students do their research at the branch stations, it is difficult for them to take all the required courses at Pullman within two years. To relieve this problem, several courses that had been taught once every two years were taught every year. Even though class sizes were smaller, administration supported this accommodation.

Before getting into the chronology of the Schafer period, some other general subjects will be discussed.

Attendance at regional and national scientific meetings was more difficult in 1954 than it is now. In 1954 the department attempted to provide funds for each faculty member to attend one meeting every two years. Research grants were few, so ARC and teaching funds were used. About 1961 I was invited to talk in Tokyo, Japan, on barley yellow dwarf. The state would pay my way only to and from Seattle and I refused to go. Faculty can now attend any meeting that contributes significantly to professional growth without the use of personal funds.

A full professor is expected to represent the university with distinction, both in the scientific world and on the farm. Off-campus faculty are evaluated for tenure and promotion on essentially similar standards as those on campus at Pullman. Research personnel must stress contributions to 'scholarly journals', and not become too involved with 'direct' service to agricultural clientele, but leave much of the latter to

extension specialists. Those who do not teach broad formal classes should make special effort to maintain general competence, to avoid becoming too specialized or technically obsolete. Faculty should be encouraged, or required, to take advantage of the professional leave (sabbatic) program, a privilege within the university. Our department is weak in 'enforcing' participation.

In the 1973-74 biennium, Bruehl taught Diseases of Plants and Plant Disease Diagnosis; Hendrix taught General Plant Pathology, and Principles and Practices of Disease Control; Rogers taught Forest Pathology, and Ascomycetes and Fungi Imperfecti; Graham taught Viruses and Virus Diseases of Plants, and Methods; Shaw taught some sections of General Plant Pathology, Plant Disease in the Environment, and General Mycology; Duran taught Lower Fungi, and Basidiomycetes; and Hadwiger taught Physiology and Genetics of Parasitism. Note the relatively equitable distribution of teaching loads and the degree of course stability.

When George Fischer became Dean of the College of Agriculture he initiated mandatory evaluation of teaching, using a standard form. The manner in which evaluation was done caused widespread resentment within the faculty. Teacher evaluation is now less formal, less rigid. These evaluations influence tenure, promotion and salary. To my knowledge, only those who teach key undergraduate courses are considered for teaching awards within the College of Agriculture and Home Economics. This eliminates teachers who teach only advanced courses (most of the Plant Pathology faculty).

Department Chairmen use discretion in the use of teacher evaluations. D. A. Roberts of the University of Florida (Plant Disease 72:372, 1988) taught the same course 16 times in 8 years, keeping detailed records of his evaluations. He believed his teaching did not fluctuate much from class-to-class, but his evaluations did. He ranked each class from good to poor on the basis of student performance. When the class was poor, his evaluations were poor. When the class performance was good, his evaluations were good. Roberts emphasized the dangers this could present an untenured person, especially one who teaches once every year or two. Two poor classes in a row would be serious. According to my experience, small classes vary considerably in talent. (Gardner Shaw comments - "Actually at WSU faculty in Agriculture and Home Economics fail to get tenure, not because of poor teaching, but because of failure to do research, or failure to publish). My own feeling is that teaching receives fair emphasis in tenure and advancement.

Back to chronology. Arlen D. Davison (Oregon State University) was hired 1967 as extension specialist at Puyallup. When Lin Faulkner became superintendent of the Prosser station, after serving one year as Chairman of Plant Pathology at Kansas State University, Gerald (Gerry) S. Santo (1974-present) was hired as nematologist. Santo works on many crops, especially potatoes, and teaches nematology as a summer course. He is one of our best teachers. When Arlen Davison moved into central administration in extension at Pullman, Ralph S. Byther (Oregon State University) replaced Davison in 1975 as extension specialist at Puyallup. Ralph is particularly active in ornamentals and in berries. Peter R. Bristow (Michigan State University) in 1976 replaced Folke Johnson as small fruit pathologist at Puyallup. Gary A. Chastagner (Univ. of Calif. Davis) replaced Chuck Gould in 1978 as ornamental and turf pathologist at Puyallup. Gary has added Christmas trees to his list of duties.

In 1978 the USDA added two pathologists, Walter J. Kaiser (1978-present) and John O'Bannon (1978-retirement in 19??). Kaiser (U. of Calif., Berkeley) specializes on seed-borne pathogens in the Plant Introduction Unit here at Pullman, with special emphasis on edible peas and beans. He cooperates heavily with Stephen Wyatt, virologist. Kaiser is also involved in international plant pathology, having spent several years in Latin America and Iran. John O'Bannon (Univ. of Arizona) came to Prosser after a distinguished career in nematology. He worked primarily on nematodes of alfalfa and potatoes at Prosser. Alice Christen (1978-1982), WSU, worked on verticillium wilt of alfalfa at Prosser.

At Pullman, Michael J. Pratt (Oregon State University) in 1978-1979 taught Introductory Plant Pathology in the absence of Gardner Shaw, as did Carolyn Roybal Allan (WSU) in 1979-80. Both these pathologists

did an excellent job and served the department in an essential role. Stephen D. Wyatt (Univ. of Kentucky) was hired in 1978 as virologist at Pullman to replace the deceased S. O. Graham. Steve is committed to molecular biology. Shaw was Chief of Party in the U.S. Aid Project in Jordan during this period.

Debras Inglis, a graduate of WSU, also taught introductory plant pathology on a temporary appointment. She was so popular the students had a "gorilla" attend the final lecture as a token of appreciation.

In 1979 Dennis C. Gross (Univ. of Calif. Davis) became our first *bona-fide* bacteriologist-plant pathologist in residence at Pullman since Bliss. Dennis stresses tree-fruit and potato problems. Hiring Wyatt (virologist) and Gross (bacteriologist) added much balance to our Pullman teaching faculty.

Harold J. Larson (Oregon State Univ.) in 1979-1981 studied fungi that attacked the wood of fruit trees at Wenatchee. David A. Weller, USDA (Michigan State Univ.) in 1979 joined R. James Cook in the Biological Control and Root Disease Unit at Pullman. Weller specializes in seed bacterization.

The last real advance under Schafer was the appointment of Dennis A. Johnson (University of Minnesota) in 1980, as extension specialist at Prosser. We now have three extension specialists (Maloy at Pullman, Byther at Puyallup, and Johnson in irrigated agriculture at Prosser).

Bruehl served as acting chairman slightly over one year between Schafer and Davison, but no changes in policy or personnel occurred during this brief time.

#### ***The Arlen D. Davison Period, 1981-1986***

Arlen Davison, after serving as Extension Specialist at Puyallup and in extension administration at Pullman, was selected to lead the department in 1981. He returned to Puyallup in 1986 as Superintendent of that station and as Assistant Dean of the College of Agriculture and Home Economics, Western Washington. He administers the Long Beach cranberry unit, the Southwestern Washington Unit near Vancouver, and the Northwest Washington unit at Mt. Vernon. A large extension staff, serving urban and rural western Washington, is housed at Puyallup.

Shaw was in Jordan as Chief of Party for a second WSU-AID project and Hendrix had retired, so Debra Inglis (WSU) taught introductory plant pathology (1982-1983). Richard G. Gilbert (1982-1988, USDA) replaced Alice Christen (WSU) as pathologist with the alfalfa improvement group at Prosser.

Timothy (Tim) D. Murray (WSU) was hired in 1983 to continue the research of Bruehl on cephalosporium stripe and strawbreaker footrot of wheat and to teach introductory plant pathology. He made this course more rigorous. Hendrix retired in 1982, Shaw in 1983, and Bruehl in 1984, resulting in a net decrease in state faculty at Pullman.

Samuel H. Smith (Univ. of California, Berkeley), a contemporary of Walter Kaiser and James Cook at Berkeley, became President of WSU in 1985. President Smith, a virologist, has tenure within this department. This is the first plant pathologist, and the first agriculturist, to head this University. It is interesting to me that WSU had no president from agriculture until 1985, unusual for a land-grant institution. Henry Schmitz, a forest pathologist and life member of APS, is listed as President Emeritus of the University of Washington in the 1963 APS directory of members. James H. Jensen, a tobacco mosaic virus expert, is listed as President of Oregon State University in the 1963 APS directory. John W. Oswald, originally a California pathologist, served as president of both the University of Kentucky and of Pennsylvania State University. These are examples of pathologists who became presidents of major institutions.

Linda S. Thomashow (Univ. of California, Los Angeles) (USDA) joined the Root Disease and Biological Control Unit at Pullman in 1985. She genetically engineers rhizosphere bacteria in ways to make it possible to estimate the individual and combined effects of possession of substrate, siderophores, and antibiotics in disease control by rhizobacteria.

In 1986 two pathologists were hired at Wenatchee. Gary G. Grove (Ohio State University) was hired to augment Ron Covey in studying diseases in the orchard, and Rodney G. Roberts (University of Georgia), USDA, was hired to replace Charles F. Pierson (USDA) as a post-harvest pathologist, mainly on apples and pears. Pierson retired in 1982, so the post-harvest pathology position had been vacant for four years. WSU concentrates on diseases in the orchard, and the USDA concentrates on post-harvest pathology at Wenatchee.

The plant pathology faculty, state wide, has essentially 'matured'; growth in numbers has stopped.

Seminar is taken seriously at WSU, and students really work on their seminars. Leadership of seminars is rotated among the teaching faculty and some USDA workers, particularly Roland Line and David Weller. When I attended seminar at the University of Wisconsin in the 1940s it was organized like a course, with a central theme for an entire semester. History of Plant Pathology was the subject at regular intervals, such as once every two years. The suggestion was made in the past to use our seminar to fill gaps in formal training, but this idea was rejected by the faculty (I tried it once - on pathological histology, and the students didn't like it). Each advanced student presents his or her research in seminar, acquainting the faculty and fellow students with research activities of the department as a whole.

Washington ranks 25th among all states in state support for agricultural research, 10th in obtaining grant support, and 3rd in support from commodity groups. The latter ranking reflects the strong commitment of agricultural leaders within the state to agricultural research. Growers assess themselves at sale of products and contribute over \$1,000,000 annually for research on plants, much of which is for plant disease research. (In some years the Tree Fruit Commission alone contributed \$1,000,000 for research.)

Success in obtaining grants, for the entire Agricultural Research Center, not plant pathology alone, the 1983-1984 federal fiscal year.

	State	Federal formula funds	Grants	Total
Cereals and field crops	47%	16%	38%	\$3,836,848
Fruits, berries, nuts	56	16	28	4,275,185
Vegetables	53	19	28	2,827,339
Pasture, forages	49	30	21	333,889
Forestry	49	32	19	736,093
Ornamentals and turf	73	10	17	410,635
Livestock and poultry*	68	19	12	3,204,980

\* Added for comparison only.

Gross groupings conceal discrepancies. In tree fruits the Washington Tree Fruit Commission gives individual grants that exceed the entire research support provided by the small fruit commissions. It is no harder to study a leaf spot of apple than of cranberries or blueberries, but the research dollars provided by the small commodity commissions cannot approach those of large commodities, like apples, wheat, or potatoes.



### ***The Jack D. Rogers Period, 1986-***

Jack Rogers (University of Wisconsin), teacher of Forest Pathology and Ascomycetes, curator of the Mycology Herbarium, and student of the ascomycetes, became chairman in 1986. Rogers and Shaw hold the record for quick advancement to full professorship within this department. Rogers has been honored by the College of Agriculture for teaching and by the University for research. Rogers is continuing his research and teaching while serving as chairman.

Lori Carris (University of Illinois) became a fungal taxonomist within the department in April, 1989, replacing Duran. She will teach the mycology courses formerly taught by Duran, or the mycology offerings will be rearranged.

The position vacated by Hendrix was filled by Hei Leung, Univ. of Wisconsin, November 1, 1989. He will serve as a fungal geneticist and molecular biologist. Leung will perform fundamental research on subjects selected by him and he will teach an advanced course in fungal genetics.

Gross, Hadwiger, Mink, and Wyatt are classified as molecular biologists by the University, along with Linda Thomashow (USDA). The addition of Leung makes the faculty at Pullman strong in molecular biology and genetic engineering, capable of meeting the 'brave new world'. Balance in applied pathology will be provided mainly by branch station faculty.

Mink replaced Paul Fridlund as leader of the IR-2 project at Prosser in 1988.

The teaching faculty for 1990 is projected as follows, with the year in which the Ph.D. was earned following the name. Maloy, 1958; Mink, 1962; Hadwiger, 1962; Rogers, 1963; Wyatt, 1973; Santo, 1974; Gross, 1976; Murray, 1983; Leung, 1984; and Carris, 1986. Maloy may retire in 1991. The teaching faculty will remain relatively young.

Nonteaching faculty at Pullman (1990) includes R. James Cook, Walter Kaiser, Rollie Line, Linda Thomashow, and David Weller, all of the USDA. They belong to the Graduate Faculty and direct students toward advanced degrees. Line and Weller regularly take turns leading seminars.

Drs. Maloy and Debra Inglis have completed a book, "Diseases of Washington Crops," to be published by the extension service. It covers symptoms, control, disease cycles, and other pertinent factors and will be generously illustrated in color.

For several years we had a summer course in Field Plant Pathology, taught by Fischer, Shaw, and then by Duran, in which students toured the branch stations where they saw diseases of various plants in the field and heard discussions of these diseases by the scientists. This course died for lack of enrollment. It has been re-initiated by Maloy.

To minimize the time required for students to be in Pullman, core courses were offered every year. In 1988, due to decreased enrollment, consideration is being given to again offering them on alternate years.

Faculty listed in the University Bulletin, 1987-1989

at Prosser

G. I. Mink, G. S. Santo, G. D. Easton, L. R. Faulkner (Station Superintendent), P. R. Fridlund, C. B. Skotland, D. A. Johnson (75% Extension, 25% research), R. G. Gilbert, J. M. Kraft, M. J. Silbernagel, P. E. Thomas (= 10 active plant pathologists, six state, four federal).

at Puyallup

R. S. Byther (100% Extension), A. D. Davison (Station Superintendent and Associate Dean), R. L. Gabrielson, P. R. Bristow, G. A. Chastagner (= four active plant pathologists, all state)

at Mt. Vernon

W. A. Haglund (= one active plant pathologist, state)

at Wenatchee

R. P. Covey, G. Grove (75% research, 25% extension), R. Roberts, L. Parish (two state, two federal)

at Pullman

J. D. Rogers, R. Duran, L. A. Hadwiger, O. C. Maloy (75% extension, 25% research), S. H. Smith (President of the University), D. C. Gross, S. D. Wyatt, T. D. Murray (= seven active state). G. W. Bruehl, J. W. Hendrix, and C. G. Shaw, state professors emeritus. R. J. Cook, S. M. Dietz (Administration, Plant Introduction Unit), W. J. Kaiser, R. F. Line, L. Thomashow, D. M. Weller, USDA (= five active federal).

Total active, 20 state, 11 federal.

Leaders of strong agricultural commodity groups (wheat especially) have long looked to the federal government for solution of major problems in Washington. They consider federal pathologists superior to state pathologists, and they prefer to extract support from the federal rather than from the state budget. In recent years federal pathologists have done most of the research on wheat, peas and lentils, important cultivated crops near Pullman.

The Plant Pathology Department is proud of its ability to attract grants to support research. According to the 1986-1987 annual report of the graduate school and Vice-Provost for Research, the department ranks second among research units of the College of Agriculture and Home Economics.

Department	Outside support as a % of total support
Institute of Biological Chemistry	63.4
Plant Pathology	57.0
Horticulture	50.2
Agronomy and Soils	46.8
Food Science and Human Nutrition	35.9
Agricultural Engineering	25.8
Animal Science	23.5
Entomology	18.9
Agricultural Economics	16.1
Forestry and Range Management	12.3

The number of graduate students enrolled in Plant Pathology has declined from the 1977-1978 school year (38 students) to 1986-1987 year (28 students). When numbers decline below 20, classes are small and instructional costs per student become high. If half the students are foreign nationals, the cost per domestic student becomes truly great. Universities do not concern themselves much over these relationships, but government officials with responsibility to the taxpayers of the state and nation do.

I strongly urge the department to prepare an annual summary, like the "Aurora Sporealis" of the University of Minnesota or "The Pathogen" of Wisconsin. The newsletter, or whatever it is called, should include all persons - faculty, staff, and graduate students, statewide, noting especially new personnel, retirements or deaths, and major events. These, filed away, would make the task of future historians easier and more accurate.

Evidence presented earlier convinced me that the personal philosophies of university presidents affect what happens at the department level, but consider what happened in the Agricultural Research Center (ARC), 1970-1986, under the tenure of President Glenn Terrell, a social scientist. The number of scientific full-time-equivalents (FTE) in the ARC, statewide, declined from 138 in 1970 to 117 in 1986. The logical conclusion is that agricultural research suffered because of President Terrell's leadership. But is this true? The total cost per research FTE increased from \$57,065 in 1970 to \$234,689 per research FTE per year in 1986. With costs per FTE per year rising, maintaining numbers is difficult.

Cost of Research in the Agricultural Research Center, 1970-1986, per year, not per biennium.

Year	Faculty no.	Source of Funds, %			Total, \$	Total cost per faculty person*
		State	Federal formula funds	Grants		
1970	138	71	13	16	7,875,093	57,065
1972	128	65	16	18	7,792,714	60,880
1974	126	66	13	19	9,198,262	73,002
1976	131	65	13	22	12,533,509	95,675
1978	124	61	14	24	14,493,122	116,880
1980	138	55	13	32	19,366,459	140,336
1982	131	51	13	36	21,967,974	167,694
1984	122	46	13	41	26,136,095	214,230
1986	117	46	12	42	27,458,634	234,689

\* Includes all support people (administration, office personnel, graduate assistants, aides, and post-doctorates).

In terms of state appropriations alone, cost per faculty in 1970 was \$40,427; in 1986, \$107,554. In terms of inflation, state support in 1986 does not equal state support in 1970. Total support from all sources exceeds inflation.

These figures include the branch stations as well as the main station. The visible cost per research faculty FTE at the branch stations far exceeds that at the main station (Pullman). At the main station many large costs are borne by building and grounds and central administration budgets. Any way you look at the costs, however, they are staggering. Research is not cheap. Hiring a scientist is serious business. If the cost of a tenured faculty is figured for a life time, the individual obviously must do significant work to justify the cost.

#### Cost of Plant Pathology to the State

The simplest assumption is that state support relative to crop value was greatest in Pennsylvania (\$24,000,000 of crop per state pathologist) and least in Iowa (\$399,000,000 of crop per state plant pathologist), but there is nothing simple about these relationships. In Iowa a preponderance of crop is soybeans and corn. Some states have many minor crops, some specialize in perishable fruits and vegetables, and some have great ecological variations within their borders, all of which require greater effort in pathology per unit of crop. In 1985 California produced 5,203,000 tons of grapes, Washington 116,100 tons. This does not mean that the pathology effort on grapes in California should be 45 times greater than in Washington. Washington produced 62,370,000 cwt of fall potatoes, Pennsylvania,

5,720,000, but diseases are a greater threat in Pennsylvania than in Washington. Likewise, Washington produced 2,050,000,000 pounds of apples, Virginia 395,000,000, or about 1/5th as much, but Virginia apples have many problems that are not serious in Washington.

Table 4. Crop value (1985) per plant pathologist (1984-1985)

State	Crop value, millions	Pathologists*			Crop value in millions per pathologist	
		State	Federal	Total	State	State and Federal
Iowa	4389	11	4	15	399	293
Nebraska	3093	12	5	17	258	182
Indiana	2869	15	3	18	191	159
South Dakota	1075	7	0	7	154	154
Illinois	5704	40	6	46	143	124
Kansas	2477	17	3	20	146	124
California	9804	82	7	89	119	110
Minnesota	3101	25	6	31	124	100
Missouri	1737	16	2	18	109	97
Texas	3857	34	7	41	113	94
Ohio	2429	22	5	27	110	90
Colorado	1144	11	5	16	104	71
Michigan	1618	21	2	23	77	70
Kentucky	1519	22	1	23	69	66
Florida	3725	56	4	60	67	62
<u>Washington</u>	<u>1865</u>	<u>24</u>	<u>14</u>	<u>38</u>	<u>78</u>	<u>49</u>
Arkansas	1454	36	0	36	40	40
Georgia	1599	34	9	43	47	37
North Carolina	1979	54	10	64	37	31
Wisconsin	1011	27	7	34	37	30
Pennsylvania	966	40	3	43	24	22

\*Agricultural Handbook Number 305, 1984-1985 Directory of Professional Workers in State Agricultural Experiment Stations and Other Cooperating State Institutions.

It is notable that states heavily involved with cereal grains have the smallest pathology input per unit of crop, but even here real differences exist. Washington produces about 1/3 as much wheat as Kansas, but winter wheat in Washington is subject to more diseases of consequence than wheat in Kansas.

Washington rates fairly well in state support for plant pathology. Most pathologists in Pennsylvania, Wisconsin, and North Carolina are housed in one place, but this did not reduce cost per unit of crop value, a surprise to me. I supposed that the extensive branch experiment station system of Washington would inflate costs.

Total cost per research FTE in the Agricultural Research Center, 1986, was \$234,689. To allow for teaching and extension, I estimate \$300,000 per state pathologist as an average figure. Each state pathologist is responsible for an average of \$78,000,000 of commodity. The insurance cost for pathology is about 0.4% of crop value per year, a bargain ( $\$300,000 \div \$78,000,000$ ).

A factor in complexity of research is the presence of crop plants in several plant families. Wheat, barley, corn, and oats have some diseases in common, apples and pears share some important pathogens, etc., so the botanical diversity of important crops affects the cost of research.

Table 6. Value of major cultivated crops in Washington, 1984

Wheat	\$545,190,000	Onions	\$20,392,000
Apples	331,915,000	Lentils	12,933,000
Potatoes	264,701,000	Beans (dry edible)	12,443,000
Hay (mostly alfalfa)	224,917,000	Carrots	10,941,000
Barley	146,510,000	Peaches	9,239,000
Corn (grain, silage, sweet)	122,090,000	Red raspberries	7,675,000
Nurseries & greenhouses	100,000,000	Strawberries	5,424,000
Hops	87,168,000	Cranberries	5,408,000
Pears	56,450,000	Mushrooms	5,186,000
Peas (dry & processing)	41,856,000	Oats	3,672,000
Sweet cherries	40,654,000	Lettuce	3,373,000
Asparagus	37,454,000	Prunes	2,716,000
Mint oils	25,948,000	Blueberries	2,465,000
Grapes	20,853,000		

Forestry is almost eliminated from the Washington data because crop data are from cultivated land. Forestry (paper, lumber, plywood, logs) is a major Washington industry, but C. H. Driver, R. L. Edmonds, R. L. Rauch and K. W. Russell, forest pathologists of the University of Washington or the Washington Department of Natural Resources are not included in the calculations.

#### Summary

The philosophies of university presidents are major factors in determining the future of institutions, particularly if in office for many years, as were Presidents Bryan and Holland. The record makes me conclude that regents are important in selecting presidents, and in firing them, but at WSU, once in office, Presidents Bryan and Holland prevailed over several boards of regents.

The department of plant pathology at WSU did not begin to approach its potential until about 1947. Its early years were blighted by lack of support. Heavy emphasis upon teaching, with an unusually large number of classes per faculty member under Presidents Bryan and Holland essentially destroyed the opportunity for research by its most talented people. Heavy teaching loads upon a small faculty, along with low salary, led to high turn-over with little accumulation of experienced professionals. Misery was concentrated in the teaching faculty, 1917-1945. My conclusion is that the troops in the trenches pay the price of administrators who expand into new areas without strengthening existing units.

The wonder of it is how so small a faculty up to 1946 did so well.

#### Extra

I suspect that WSU is the only land grant institution in the nation that did not have a president from agriculture or engineering until 1985 (Pres. S. H. Smith, Agriculture). How long will the engineers wait at WSU?

## PERSONNEL

### *Department Chairmen*

Department chairmen serve a vital function within the university, described in this section by Jack Schafer. Selection of chairmen is democratic, with all tenured faculty voting on the candidates. Higher administration, to my knowledge, generally accepts the judgement of the faculty. The faculty votes every four or five years on retention of the person serving in that capacity. All chairmen have tenure within the department. Chairmen receive 10% extra pay for administrative duties, and their salary comes from research, teaching and extension budgets, commensurate with their responsibilities in each activity. Some chairmen devote full-time to administration while some retain some research and/or teaching while in the job.

#### Botany, Zoology, Entomology

1893-1903	Charles V. Piper
1904-1907	R. Kent Beattie

#### Botany and Bacteriology

1907-1910	R. Kent Beattie
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#### Plant Pathology

1910-1913	Harry B. Humphrey
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#### Botany

1913-1916	Ira D. Cardiff
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#### Plant Pathology

1917-1941	Frederick D. Heald
1938	S. M. Dietz, Sr., one year during Heald's period.
1941-1943	J. G. Harrar.
1943-1945	Earl J. Anderson.
1945-1958	George W. Fischer.
1958-1960	J. Walter Hendrix, Acting.
1960-1972	C. Gardner Shaw. Shirl O. Graham acted as chairman during the several protracted absences of Shaw.
1972-1980	Jack F. Schafer.
1980-1981	George W. Bruehl, Acting.
1981-1986	Arlen D. Davison.
1986-	Jack D. Rogers.

Until the mid-1940's, when Puyallup had an independent budget, the lead pathologist at Puyallup was Head of the Section at that station. After unification of the budgets, there has been only one Head, all of whom served at Pullman. A quote from Chuck Gould (1941): "I was hired by Dr. J. W. Kalkus (Supt. of the WWES) and Dr. Glenn A. Huber (Head of the Department of Plant Pathology at Puyallup." This does not happen today.

*Jack Schafer, reflections as a department chairman*

Except for periodic visits over the years, my next participation in WSU Plant Pathology was in 1972, just 30 years after leaving Pullman for war-time service. At the time I was the Plant Pathology Department Head at Kansas State University where I had gone from Purdue 4 ½ years previously. Walt Hendrix, the Search Committee Chair, had called me about a candidate to succeed Gardner Shaw, long-time department chair at WSU. Incidentally, he asked if I might be interested and I agreed that I could be, and subsequently interviewed for the job. I arrive in Pullman on September 1, 1972, to enter into the position previously held by Heald, Harrar, Anderson, Fischer, and Shaw and several others on an acting basis.

I believe that in 1972, WSU Plant Pathology was a distinguished department, with a highly recognized history and several outstanding faculty. Washington agriculture had expanded greatly in the previous 30 years, particularly with the development of irrigation in the Columbia Basin with water from the Grand Coulee Dam.

Although the WSU campus is in the midst of the important Palouse agricultural region, it is in a far corner of the state, away from population centers and other important agricultural areas. Over the years, several both large and small outlying Research and Extension Centers and Units were developed with professional staff stationed on site with local professional-level superintendents. Center staff were also members of agricultural academic departments. Plant Pathology faculty were located at four outlying locations, and these people comprised about three-fifths of the total departmental faculty.

My 8-year tenure as department chair was not one of great expansion or major retrenchment. I believe that we essentially held our own in magnitude of effort. The faculty as a whole was distinguished and generally on the older side. The Pullman group was equivalent in size to the Kansas State Department from which I had come, but the Department as a whole over twice as large. The whole department was of the same magnitude as that at Minnesota but much more fragmented geographically.

There are obvious agricultural merits to having professional faculty on the site of their agricultural areas. These include the close, continuing relation to the important problems, the development of appropriate scientific teams, and the maintenance of political support from the affected clientele. Disadvantages include the difficulty of maintaining departmental cohesion, fielding a broadly based teaching program, and bringing all scientific expertise to bear on a graduate study program without difficult logistical problems.

In retrospect, I believe that my most important contribution as Department Chair was in bringing the geographically separate faculty into a more integrated departmental whole. We worked particularly hard at broadening the graduate student program (that is, the major professor base) out to the outlying centers. Several of the individual faculty at outlying centers were especially cooperative and participatory in this development. This effort included generation of stipend support, having appropriate subject matter classes suitably available, and organizing schedules so that students could attend classes at Pullman, yet conduct research with continuity at the outlying research centers without excessive logistical problems. We evolved into a program in which about one-quarter of the graduate students had their research based with faculty at outlying stations.

A Department Chair, as such, has no real influence on the branch station system. His effect is in adapting program and encouraging people so as to achieve the most both for the various facets of the state's agriculture and for the development of a cohesive department while not detracting from the needed cohesiveness of the research centers and their team efforts.

I had previous experience in Kansas where the branch research stations were totally independent from departments, functioned as parallel units to departments, and competed with those departments having

the same subject goals. Plant Pathology, a small, growing department there, was not then represented at the branch stations, so I was able to observe branch station and department relations externally, and become involved only in a small way in some coordinated activity precisely because I was external to the activities of the units involved. This background influenced my efforts in attempting to make all of the Plant Pathology Department members at WSU a cohesive unit.

Going back much further, I was well aware of the outlying stations, having joined my father a few times on short visits with agronomic personnel at the stations. (An enjoyable later reverse follow-up was the day my father, retired Agronomy Head, at about age 90, joined me on a visit to Prosser where we "toured" the facility with Harold Singleton, the retired Superintendent, then past 80, he and my dad being close associates and good friends over the years.)

Washington has an outstanding commodity commission program. This provides an important base of financial support for research. Each participating researcher reports, at an annual session and maintains ongoing contact throughout the year. The relevant department chairs participate integrally in these reviews and their planning, a major investment of time and effort. Plant Pathology is involved in almost all of the programs that either Agronomy and Horticulture is in. Although the Plant Pathology relation may be a little less intense than theirs, the total magnitude is about the sum of the other two. This activity was one of the more enjoyable outreach functions, albeit a rather time consuming one, of the agriculture department chairs. We had close relations with commissions on wheat, potatoes, tree fruit, and peas and lentils.

Commission activity provided a forum for interaction with the other department chairs. As in my previous experience at Kansas State, the interaction with the other Ag department chairs was one of the important and rewarding aspects of my tenure as Department Chair. Possibly even more important was the interaction with Center Superintendents, who jointly supervised research programs. In my opinion this very complicated administrative system of interaction of Chairs and Superintendents, when properly and congenially handled, provided a more coordinated program and a better final product, considering the needs of the agricultural clients, the University, and the professional status of faculty, than would a simpler unilateral system.

A Department Chair has many chores. Personnel relations and financial management are among the more important and take much time. Among other chores are such things as college greenhouse coordinator and building chairman. Although the latter was a relatively small activity, it took considerable time from time to time over a 5-year period, with such items as scheduling classes in the classrooms of a 5-department building, and arbitration of small controversies. Even the manipulations of secretarial hiring within a very rigid Civil Service System are important. The System, in fact, is rather good and very supportive to the employees, at least if they are willing to move between positions. A Department Chair can expect to keep a good Secretary I for about six months and a Secretary II for about a year. Thus, one must always expect to be in the recruiting business. When filing a Secretary IV, one must know who all are on various "bubbles" around the campus and fully understand the priorities and absolutes of the system.

Probably the single most important thing a department chair does is to recruit. In a modern department this is not accomplished alone, of course, but the chair or an assignee must put together specifications, meet equal opportunity standards, and conduct extensive interviews. Much of my time at APS annual meetings during my Pullman years was spent interviewing. In this process, I met many splendid young scientists. Many continue as good friends even though not hired by us. During my eight years at WSU, we employed Gerald Santo, Peter Bristow, Ralph Byther, Steve Wyatt, Gary Chastagner, Dennis Gross, and Dennis Johnson. They are all still in their WSU positions, and I believe doing well. Recruiting is the significant legacy of any department chair.



The WSU Department of Plant Pathology has had much more than its share of leadership of the American Phytopathological Society. In the 80 years of the Society, six of its presidents have been from the WSU faculty. Dr. Heald held this office in the 30s, Dr. Fischer in the 50s, and Dr. Holton in the 60s. Early in my tenure at WSU, Dr. Bill Bruehl was elected into this office sequence, becoming President in 1976. Only two years later I followed, and five years after that Jim Cook took office. Like Stu Holton before him Dr. Cook was a USDA/ARS employee in the WSU department. Dr. Hank Purdy of the University of Florida, a long-time USDA/ARS employee at Pullman immediately followed me. Both Dr. Purdy and Dr. Ed Kendrick, another Pullman USDA/ARS employee, were long-time APS Treasurers.

There may be sad times involved with one's professional life; 1976 was particularly a year of these. In September of that year we lost Dr. Shirl Graham, a long-time distinguished faculty member, who had served as Acting Chair several times. For me, 1976 was a particularly heavy year. My Dad died in July at age 92; Shirl in September; and Ralph Caldwell, my initial employer and long-term colleague at Purdue, and my overall closest lifetime professional associate, in November.

Being a department chair is very rewarding. However, it can eventually be wearing. In contrast to the era of Dr. Heald and my Dad, I don't think that it should be a major lifetime occupation. After four years as Head at Kansas State and eight years as Chair at Washington State, I thought that I should not spend the rest of my professional career in this job. One makes his contribution and then the return is marginal, and it is someone else's turn. Opportunity for me to change arose with the chance to go to the USDA Science and Education Administration Staff as the national Integrated Pest Management Coordinator for that agency, on their Intergovernmental Personal (IPA) exchange program in which I could work for them without at that time leaving the WSU faculty, and WSU would be monetarily reimbursed for my services. I had just been reappointed for a third 4-year term as Chair, but with eight years of service, I thought that this was a reasonably respectable time to leave. Although not one of the longer-term faculty, I trust that I have an integral place in the history of the WSU Department of Plant Pathology.

The best life of F. D. Heald that I have seen, written by Kenneth F. Baker and George W. Fischer, is in Annual Review of Phytopathology 21:13-20, 1983.

*Personal Attributes of Administrators and Scientists*  
*Richard Wellman*

Notes taken by Bruehl during a lunch with Dr. Richard Wellman, date unknown - about 10? years ago?

Dr. Richard Wellman obtained his B.S. degree at WSU in 1936 and his Ph.D. in 1939. He spent one year at Prosser after which he joined the Union Carbide Corporation. He rose to Vice-President, Manager of Agricultural Chemicals. He left Union Carbide in 1974 to head the Boyce Thompson Institute for Plant Research, first at Yonkers and then at Cornell.

Scientists and Scientific Administrators both require high intelligence and intuition, both should make decisions promptly, both should have an independent spirit and self-reliance. Both need to be self-motivated and neither should glamorize the work. Differences between scientists and administrators are several.

Scientists are theoretically removed from daily concerns. Scientists should be "things" oriented. Scientists must be on the cutting edge of scientific disciplines and they must know what has been done and what others are doing. Scientists must prove their assumptions (or theories). Scientists shouldn't be too patient; impatience is frequently desirable.

Administrators should be practical, pragmatic, and they should get money for research. Administrators should be people oriented. Administrators must get cooperation between workers. Administrators must

be salesmen and they must convince people to support research. Administrators must sell their assertions and be able to make decisions on the basis of limited knowledge. Patience is frequently desirable in administrators. Administrators must encourage and develop subordinates, they must delegate authority, they must let people fail. People grow with failure. Administrators are facilitators rather than actual doers.

Speaking for industry, Dr. Wellman stressed that second-best products are losers. The clientele finds out when a product is second class. Second-best products get only a fraction of the market and usually lose out. A lot of time is spent selling and advertising, so only good products should be marketed.

Don't let a person (scientist? administrator?) become attached to a product.

Bruehl adds that administrators at all levels of WSU give scientists essentially complete freedom in choosing subjects to study.

### ***Faculty***

This department is primarily a research and graduate teaching department, with lesser emphasis on undergraduate training. The so-called "service" courses offered are of high quality and they are taught by excellent teachers, Murray, Maloy, and Rogers, but the future of the department, its national and international reputation, will be in direct proportion with the excellence of our faculty, state-wide. This department enjoys respect within the university, the state, and the nation. There is no doubt that selecting faculty with the ability to sustain high interest and energy, to grow in capability, and to pursue worthwhile objectives through a life-time career is the most important function of administration. This treatment of faculty deals with some of the changes that have taken place and are still in progress within the profession.

I made no special effort to state the origin of faculty, but the universities from which professors were trained has varied through the years. The University of Wisconsin supplied the most in the past, and more recently the University of California at Davis supplied several, but our faculty is diverse in origin, a source and of varied skills. Historically few from WSU are selected for teaching positions, an effort to avoid inbreeding and to maintain versatility.

### *Getting a Job*

In the early years, when few students attended universities, college degrees were special, and I believe, during the period of opening new experiment stations, jobs were easy to get. During the depression of the 1930s jobs were few and competition was keen. During World War II graduate training for many was interrupted, and in the post-war years jobs were plentiful. When I entered the job market, while still a student (1947) a USDA administrator, Dr. Cecil Salmon, spent two hours with me in the laboratory where I worked, visiting and looking at my histology slides on the campus at the University of Wisconsin. I was also invited to Purdue University, escorted around the department and the field plots by Dr. Ralph Caldwell. These constituted both "interview" processes. I had a choice of both jobs and took the USDA job at Brookings, South Dakota, because of romantic ideas about the prairies and Great Plains. Both interviews were pleasant experiences. When hired by WSU, no interview or visit occurred. It was sight-unseen for both parties.

It is with sympathy that I watch candidates arrive to undergo the present rigorous, structured interview process, taking considerable time and including a performance in a seminar. Without good slides and an excellent presentation you don't have a chance. In the 1940-1960s period the entire process was simple. From the 1970's onward a committee screens the written records of usually many candidates, selecting the most qualified individuals as the initial step. In the 1940s-1960s the letters of recommendation, your

thesis, and your transcript were about all that were considered. There has been a change from a sellers' to a buyers' market.

### *Post-Doctoral Training*

At some point, probably during the late 1930's to early 1940's, the Ph.D. degree became essential for faculty positions. During the 1940s only a few departments (Wisconsin, Minnesota, Cornell, U.C. Berkeley) produced many of the Ph.D. degrees in plant pathology, with some other departments making lesser contributions. In the 1940s and the 1950s the job market absorbed all new pathologists. Some time after World War II, particularly after increased financing for research became available, more departments began to produce Ph.D.s and the job market eventually became saturated. With this development, some young scientists accepted temporary employment as post-doctoral research associates. At the present time (1989) there are seven at Pullman (Brenda Callan, Catherine Daniels, David Heron, Bonnie Ownley, Sandy Pierson, Neil Quigley, and Larry Specht), the greatest number ever here. When I graduated (February, 1948) new Ph.D.s often had a choice of good, permanent positions. The increase in post-doctorates is sad to me.

Present post doctoral associates view the situation differently. The Ph.D. degree does not impart sufficient depth in specialized areas of research to enable successful competition in the grant markets or in the skills of complex molecular biology. Post doctoral associates select the expert with whom they choose to work. They concentrate full-time on research, unencumbered by teaching or worrying about grant proposals. They can increase their skills in research and writing full-time, for maybe the only such period in their life. Some enjoy this period and would choose it voluntarily.

In human medicine internships are required. The MD degree alone does not qualify one to practice. Plant pathology has now reached the stage where post-graduate training (post-Ph.D.) is almost essential. If plant pathology requires the Ph.D. plus "internship," salaries should be commensurate to compensate for so many years of preparation. Learning on the job is still possible, however, especially if sabbatic leaves are properly used.

### *Retirement*

On my first job (USDA, stationed at South Dakota State University, 1948-1952) I observed two elderly gentlemen make their way onto the campus almost every day. One occupied an office three doors from mine. He usually napped in his chair, visited a bit, and went home. Both retired before SDSU had a retirement system. Both had been leaders in their fields. By some legal (?) subterfuge they remained on the payroll, the only way SDSU could protect them in their old age. Their salaries were never high enough for adequate savings. I admire the administration for bending the rules. There was no objection from the legislature.

Retirement at WSU today is based upon the salary of the last two years and the years of service to a maximum of 25 years. An individual retiring with a salary base of \$60,000 after 35 years could receive  $2\% \times 25 \text{ years} = 50\% \times \$60,000 = \$30,000$  per year, not adjusted for inflation. WSU belongs to the Social Security system, which does adjust for inflation. This retired individual would be in the maximum social security bracket, so social security would add at least \$10,000 per year. If retirement was at age 65, in good health, after 18 years at an average of 4% inflation, social security would double to \$20,000. This hypothetical retirement would increase gradually from about \$40,000 to \$50,000 at death, for an average of about \$45,000 per year. The good old days were not so good. Retirement is no longer feared.

Retirement under the former USDA system was more generous, in that it was not limited to 25 years and that it was adjusted for inflation. An individual retiring after 35 years at \$60,000 per year would start at  $66.25 \times \$60,000$ , or \$39,750 per year, and theoretically, at 4% inflation, it would double to \$79,500 per

year at death, for an average of about \$59,625 per year. The medical provisions in the state system are apparently more generous than those in the federal system.

Retirement at age 65 was mandatory in the state system until about 1980, when retirement because of age was made illegal. Some individuals remain productive until age 70, some become unproductive well before age 65. The change in mandatory retirement age will average a loss to the university in my opinion.

### *Grants*

Government scientists in the past were supported by the employing agencies, state or federal, with little money from other sources. The funds were small by present standards, but little or no time was spent writing grant proposals.

The greatest outside support of research in plant pathology at WSU at first came from the agricultural industry itself. Washington laws enable producers of commodities (apples, wheat, cherries, etc.) to assess themselves at sale of product, amassing funds to advertise their products and to support research. Grant proposals for commodity commissions require little time to prepare; reporting results is likewise simple. The university collects no overhead from these grants. The apple growers, etc., are tax payers of Washington and the rationale is that they already support WSU. They support research to solve specific problems, the solution of which directly improves the economy of Washington, and indirectly of WSU. Scientists with grants of this type have a real responsibility. They represent WSU to the agricultural leaders of the state. If they are incompetent or misleading they will eventually be detected as such. These leaders of agriculture are our strongest supporters in the state and federal legislatures, and we must nurture their confidence.

Chemical companies make many small grants to individual scientists to help defray the cost of testing their materials. Some scientists (Line, Covey, Santo) receive enough of these grants that, when combined, are substantial. In the past some grantors of small grants requested detailed reports. Most of these grants are so small they in no way cover the cost of the work, leading to the criticism that WSU subsidizes chemical companies. The Agricultural Research Center (ARC) now requires small grants to be gifts, with no special requirements for detailed reports of results. In addition, the testing must be part of the scientist's regular activities, not an added activity. All grantors of substantial funds, other than Washington State commodity commissions, pay overhead charges to the university to help defray indirect costs of the research.

Federal granting agencies are now the greatest source of substantial funds, and these agencies favor technical, sophisticated, "cutting-edge" science. The highest paid scientists in the university are those most successful in obtaining federal grants. Young pathologists quickly learn that their future is influenced greatly by their ability to obtain substantial support. This has led to alterations in curriculum, with emphasis on advanced technology. In earlier days, most federal funds were Hatch, Adams, McIntyre-Stennis ("formula" funds), funds directed by experiment station directors with few restraints. The competitive grant system has reduced their autonomy.

University scientists today, for their personal advancement, can ill-afford to spend much time testing seed treatments, sprays, cultivars, etc., yet these are vital activities that must be done, especially by a competent, independent testing agency. It is time, in my opinion, for the state of Washington to assign these tasks to special extension personnel. These activities require training in the design of experiments, statistics, in epidemiology and in evaluating the results. Producers, the public, and industry deserve honest, adequate evaluations. Some companies are highly ethical and some are not. True evaluations protect everyone, including the ethical companies.

### *Teaching and Research Costs.*

In recent years the highest paid professors have been those who bring in the most grant money, particularly from granting agencies with high overhead payments. David E. Honig, formerly of the University of Washington (Newsweek, June 6, 1988, p. 8) stated that he spent 150 hours in class in four years; the rest of the time theoretically was devoted to research. He blames emphasis upon research as the reason why teaching costs at research institutions have increased faster than inflation. Faculty spend fewer hours in contact with students than in the past, greatly increasing teaching costs. Honig's statements are only partially true. One hour in class requires several hours in preparation, plus grading examinations, general reading and thinking. The effort in teaching should not be judged solely by hours spent in class.

Honig goes so far as to say that for faculty it is no longer publish or perish, but get money from the National Science Foundation or National Institutes of Health, or perish. Honig obtained \$750,000 in four years for research (some get much more), wrote papers, attended symposia, traveled widely, etc., worked 60-hour weeks, but spent 150 hours in class in four years. He documents the effects of extreme emphasis upon fund-getting upon teaching, especially at the undergraduate level.

The emphasis on winning federal grants in establishing salaries is not as strong at WSU as at the University of Washington, but it exists. Some scientists are so successful at obtaining grants that the overhead payments equal the scientist's salary.

The only college in the University of Washington or at Washington State University that attempts to document costs of teaching and research separately is the College of Agriculture and Home Economics.

### *Federal-State Relationships*

The distribution of USDA pathologists within Departments of Plant Pathology varies greatly. One wonders why. According to the Directory of Professional Workers in State Agricultural Experiment Stations and Other Cooperating State Institutions, 1984-1985 (Table 1, p. 94), there were no federal pathologists in the departments of plant pathology at the Universities of Arizona, Arkansas, California (Berkeley and Riverside campuses), Hawaii, or Massachusetts. The plant pathology faculty of the University of Kentucky had 4% federal workers, Montana State University 5%; in New York there were 7% at Cornell and 0% at Geneva, and the University of California, Davis campus, 21%. At WSU, 37%.

Federal pathologists reduce state expenditures for research, a bargain in the research budget. There is another aspect to federal-state relations that should be considered, however. The quality and prestige of graduate programs depends primarily upon the prominence of the classroom teaching faculty. Teaching professors should lead important research projects to insure their greatest possible growth. There are only so many prime research 'territories' within a state, and territorial rights should be considered if a department is to attain the greatest possible stature in its graduate training program. It is easier to attain prominence standing on a hill than in a valley.

### *Recognition by APS*

Of the faculty with a significant part of their careers in Washington, eight became presidents of the American Phytopathological Society (Heald, 1932; Humphrey, 1945; Fischer, 1957; Holton (USDA), 1963; Bruehl, 1977; Schafer, 1979; Purdy (USDA), 1980; and Cook (USDA), 1984). J. D. Rogers was president of the Mycological Society of America in 1978. H. P. Barss, who served one year at Puyallup, was president in 1928. J. G. Dickson, who earned his BS while Cardiff was Botany chairman, was president in 1953 and Glenn Pound (USDA) who served at Mt. Vernon two years was president in 1959. Al Dimond, who took his first plant pathology from Dr. Heald, was president in 1964. Harold H. Flor (USDA),

who worked three years on common bunt at Pullman, was president in 1968 and he received the Award of Distinction in 1980.

My access to records of presidents of the Pacific Division of the American Phytopathological Society are sketchy, but pathologists with experience in Washington are prominent among them (H. P. Barss, 1916; F. D. Heald, 1918; 1919; Bliss Dana, 1939; G. W. Fischer, 1948; Earle Blodgett, 1950; C. S. Holton (USDA), 1957; K. F. Baker (a graduate of this department), 1959; J. D. Menzies, 1960; John Milbrath (a BS major here), 1962; C. J. Gould, 1964; C. Gardner Shaw, 1966; G. W. Nyland (a graduate of this dept.), 1968; G. W. Bruehl, 1972; W. H. English (a student here), 1973; J. A. Hoffmann, 1977; T. Kosuge (a student here), 1978; R. J. Cook (USDA), 1979; M. F. Stoner (a WSU student), 1980. G. I. Mink, 1985.

APS Fellows include H. P. Barss, George W. Fischer, Harold H. Flor, J. George Harrar, C. S. Holton, Glenn S. Pound, K. F. Baker, T. O. Diener, G. W. Bruehl, Tsune Kosuge, C. J. Gould, R. J. Cook, George Nyland, L. H. Purdy, W. Harley English, Douglas W. Burke, and Jack F. Schafer (to 1988).

The Ruth Allan Award for advances in technology has been won by H. H. Flor, Austin G. Goheen and T. O. Diener. The Lee M. Hutchins Award for advances in fruit tree virology was won by Gaylord Mink.

This listing of honors is far from complete, but it suffices to establish significant leadership from this department.

#### *Visiting Professors and Sponsors*

Often individuals with special skills and interests join the faculty on a temporary basis. This exchange results in solving specific problems at little cost to the state, and many visitors teach new skills to their sponsors. Many come with salaries from their home institution, others are paid by WSU. They are usually given full faculty privileges and status while here.

My listing is inadequate, but it suffices to show the magnitude of the flow of talent through the department.

1967	Roy L. Millar. Bruehl.
1967-1968	William Q. Loegering. Hendrix?
1969	David Griffin. Cook.
	N. C. Schenck. Graham. (Date unknown)
1977	William G. Willis. Cook.
1977-1978	Yoshihisa Homma. Cook.
1981-1983	Bing-Xing Zhang. Cook.
1983	E. A. Stowell. Rogers.
1984-1986	Wen-hua Tang. Cook.
1985	Thomas Herring. Cook.
1985	Claude Vallavieille-Pope. Cook.
1985-1986	Liliane Petrini. Rogers.
1986	Guoxuan Li. Wyatt.
1986	Akira Ogoshi. Cook.
1986-1987	Antonio Traperero-Casas. Kaiser.
1987	Albert Rovira. Cook.
1987-1989	Ta-Li Kuan. Haglund (Mt. Vernon).

Richard C. Maxwell, State Extension Specialist in Pesticides. Assists in registration of fungicides for minor purposes and legal questions as EPA and Pure Food and Drug laws.

## Research Associates

1962-1965	K. M. Safeeula. Shaw.
1965-1966	Eva H. D. Fuchs. Hendrix.
1967-1971	Martin Schwochau. Hadwiger.
1969-1970	Robert Nyvall. Mt. Vernon.
1977-1981	Alice A. Christen. Prosser.
1978-1981	Ellis Darley. Wenatchee (Courtesy appointment).
1978-1981	John Gardner. Duran.
1979-1981	Rosy Chako. Shaw. Worked as an Ag. Res. Tech. II for a time and as an Instructor for a time.
1979-1980	Joanna Macinkowska. Prosser. Kraft.
1981-1982	Andrew P. Nyczepir.
1982-1983	Felix Mauch. Prosser.
1982-1983	Francis J. Grant. Hadwiger.
1982-1984	Ole J. Becker. Cook.
1983-1985	Wendy J. Wagoner. Hadwiger.
1983-1985	John N. Pinkerton. Prosser.
1983-1987	Edward N. Bassett. Cook.
1984-1985	Hassan Mojtahedi. Prosser.
1986-1987	Ann-Scott Ettinger. Hadwiger.
1986-1989	Kurt Regner. Gross.

Research Associate is a mixed category, but mostly it contains full-time employees of WSU, or in some cases employees paid by another institution.

## Post Doctoral Research Associates, State System

1969-1970	Haasan Melouk. Skotland, Prosser.
1971-	John A. Christenson. Hadwiger.
1974-1975	William E. Hunter. Bruehl.
1975-1977	John A. Fernandez. Duran.
1975-1977	Christian Sander. Hadwiger.
1978-1980	Robert A. Blanchette. Shaw.
1979-1982	Henry T. Wilkinson. Cook.
1980	Wojciech K. Kaniewski. Skotland, Prosser.
1980-1981	Takashi Naki. Cook.
1980-1981	David C. Loschke. Hadwiger.
1981	Tsungmin Kuo.
1981-1983	Edward B. Adams. Line.
1982-1988	Abdul Qayoum. Line.
1982	Josef Robak. Prosser?
1984-1985	Jon Jensen. Duran.
1985-1988	Robert Klein. Kaiser & Wyatt.
1985-1987	Jeanne C. Anderegg. Murray.
1985-1987	David J. Rhodes. Gross.
1985-1986	Yvonne S. Cody. Hadwiger.
1986-1987	Remi Rakotndrdona. Line.
1987-	Neil B. Quigley. Gross.
1987-1989	Lawrence P. Specht. Murray.
1987-	Catherine H. Daniels. Hadwiger.
1988-1989	Brenda Callan. Rogers.
1989-	Ming-Mei Chang. Hadwiger.

WSU faculty, USDA, WSDA scientist, and extension specialists in Plant Pathology, with the year of first employment. Persons 10 or more years in Washington are underlined.

### **1893-1920**

Charles V. Piper, WSU, 1893 Botany, Zoology, Entomology<sup>a</sup>  
R. Kent Beattie, WSU, 1902 Botany and Plant Pathology  
David A. Brodie, WSU, 1902 Botany and Plant Pathology  
Samuel W. Collett, WSU, 1907 Botany and Plant Pathology  
Harry B. Humphrey, WSU, 1909 Botany and Plant Pathology  
Rex N. Hunt, WSU, 1909 Plant Pathology (?)  
H. P. Barss, WSU, 1911 Plant Pathology Puyallup  
Ira D. Cardiff, WSU, 1913 Botany and Plant Pathology  
D. F. Fisher, USDA, 1913 Wenatchee  
D. C. George, WSU, 1914 Plant Pathology  
John G. Hall, WSU, 1914 Plant Pathology  
H. M. Woolman, WSU, 1914 Plant Pathology  
F. D. Heald, WSU, 1915 Plant Pathology  
A. M. Christensen, Extension, 1918 Plant Pathology  
B. F. Dana, WSU, 1918 Plant Pathology  
N. F. Thompson, Extension, 1918 Plant Pathology  
George L. Zundel, Extension, 1918 Plant Pathology

<sup>a/</sup> Only Piper, Fisher, and Heald stayed 10 years or longer. Score: USDA, 100%; WSU, 13% long-term faculty. Piper was actually hired in November, 1892.

### **1921-1940**

George D. Ruehle, WSU, 1926  
Lee E. Miles, WSU, 1927  
G. A. Newton, WSU, 1927  
E. L. (Bud) Reeves, USDA, 1927  
Harold H. Flor, USDA, 1928  
Leon K. Jones, WSU, 1928  
William D. Courtney, USDA, 1933 Puyallup  
C. Stewart Holton, USDA, 1931  
Glen A. Huber, WSU, 1932  
George W. Fischer, WSU, USDA, WSU, 1934  
Leo Campbell, WSU, 1935 Mt. Vernon, Puyallup  
W. Harley English, WSU, USDA, 1936 Pullman, Wenatchee  
S. M. Dietz, Sr., WSU, 1938  
Richard Wellman, WSU, 1938 Prosser  
D. M. Coe, WSU, 1939 Pullman, Moxie  
James D. Menzies, WSU, USDA, 1939 Prosser

Of those hired 1920-1940 who stayed 10 or more years, Jones and Campbell were with WSU the whole time, Fischer and Menzies worked for WSU and the USDA, and Reeves, Courtney and Holton were USDA employees.

### **Started 1941-1960**

Charles J. Gould, WSU, 1941 Puyallup



J. G. Harrar, WSU, 1941  
Earl J. Anderson, WSU, 1943  
Folke Johnson, WSU, 1943 Puyallup  
Harold B. Busdicker, USDA, 1944  
Glenn S. Pound, USDA, 1944 Mt. Vernon  
Earle C. Blodgett, WSDA, WSU, 1946 Prosser  
Marion R. Harris, Extension, 1946  
Seth B. Locke, WSU, 1946  
D. M. McLean, USDA, 1946 Mt. Vernon  
Jack P. Meiners, WSU, USDA, 1946  
George Nyland, WSU, 1946  
Frank J. Stevenson, WSU, 1946  
Robert C. Lindner, WSU, 1947 Wenatchee  
Avery E. Rich, WSU, 1947  
C. Gardner Shaw, WSU, 1947  
Roderick Sprague, WSU, 1947 Pullman, Wenatchee  
Charles M. Wright, USDA, WSU, 1947  
Shirl O. Graham, WSU, 1948  
Hugh C. Kirkpatrick, USDA, 1948 Wenatchee  
J. G. Barrat, WSU, 1950 Prosser  
T. O. Diener, WSU, 1950 Prosser  
J. Walter Hendrix, WSU, 1952  
Charles F. Pierson, USDA, 1952 Wenatchee  
Edgar L. Kendrick, USDA, 1953  
L. Henry Purdy, USDA, 1953  
G. William Bruehl, WSU, 1954  
Shirl (Sam) M. Dietz, USDA, 1954  
Murit D. Aichele, WSDA, 1955 Prosser  
Paul Fridlund, WSU, 1955 Prosser  
Walter Apt, USDA, 1956 Puyallup  
Douglas W. Burke, USDA, 1956 Prosser  
Calvin B. Skotland, WSU, 1956 Prosser  
Richard O. Hampton, WSDA, WSU, 1957 Prosser  
William G. Hoyman, USDA, 1957 Prosser  
Ruben Duran, WSU, 1958  
James A. Hoffmann, USDA, 1958  
Lindsey R. Faulkner, WSU, 1959 Prosser  
Richard L. Gabrielson, WSU, 1960 Puyallup  
William A. Haglund, WSU, 1960 Mt. Vernon

USDA workers Jack Meiners, Kendrick (1953), Purdy (1953), Dietz (1954) and Hoffman (1958) were part of the Regional Cereal Smut Laboratory at Pullman. Dietz transferred to the Plant Introduction Station at Pullman. Stability in state faculty came during this period and it has been permanent ever since.

### **1961 to 1988**

P. C. Cheo, WSU, 1961 Wenatchee  
Raymond L. Clark, USDA, 1961 Prosser  
Matthias J. Silbernagel, USDA, 1961 Prosser  
Ronald P. Covey, Jr., WSU, 1962 Wenatchee  
Gaylord I. Mink, WSU, 1962 Prosser  
Gene D. Easton, WSU, 1963 Prosser  
O. C. Maloy, Extension, 1963

Jack D. Rogers, WSU, 1963  
R. James Cook, USDA, 1965  
Lee A. Hadwiger, WSU, 1965  
C. Lee Parish, USDA, 1965 Wenatchee  
John M. Kraft, USDA, 1966 Prosser  
John McRitchie, USDA, 1966 Wenatchee  
P. E. Thomas, USDA, 1966 Prosser  
Arlen D. Davison, Extension, 1967  
Roland F. Line, USDA, 1968  
J. Lewis Allison, Superintendent, 1969 Prosser  
R. M. Davidson, Extension, 1972 Puyallup  
J. F. Schafer, WSU Chairman, 1972  
Gerald S. Santo, WSU, 1974 Prosser  
Ralph S. Byther, Extension, 1975 Puyallup  
Peter R. Bristow, WSU, 1976 Puyallup  
Gary A. Chastagner, WSU, 1978 Puyallup  
Walter J. Kaiser, USDA, 1978  
John O'Bannon, USDA, 1978 Prosser  
Michael J. Pratt\*, WSU, 1978  
Stephen D. Wyatt, WSU, 1978  
Carolyn R. Allan\*, WSU, 1979  
Dennis C. Gross, WSU, 1979  
Harold J. Larsen, WSU, 1979 Wenatchee  
David M. Weller, USDA, 1979  
Dennis A. Johnson, Extension, 1980 Prosser  
Debra Inglis\*, WSU, 1982  
Richard G. Gilbert, USDA, 1982  
Timothy D. Murray, WSU, 1983  
Samuel H. Smith, WSU President, 1985  
Linda S. Thomashow, USDA, 1985  
Gary G. Grove, WSU, 1986 Wenatchee  
Rodney G. Roberts, USDA, 1986 Wenatchee

\* These three pathologists on temporary assignments helped the department by serving as teachers of our introductory class during the absence of permanent faculty.

## **Students**

### *The BS Degree*

Forty-six individuals received the BS degree with majors in plant pathology, 1912-1950 (Fischer, 1951). The first 12 recipients, 1912-1925, all had careers in plant pathology, either directly or after obtaining advanced degrees in the subject. Between 1926 and 1940, 14 of 19 recipients had identifiable careers in plant pathology, and between 1941 and 1950, 4 of 11 became plant pathologists. These data make it obvious that those who chose to major in plant pathology as undergraduates were serious, and that the training was useful. Breaking the time periods 1912-1922, 1923-1932, 1933-1942, and 1943-1950, the percents who receive the BS degree in plant pathology who remained plant pathologists were 100, 87, 67, and 29, respectively.

Recipients of the Bachelor of Science degree in Plant Pathology through 1950. Those marked with an asterisk had identifiable careers in plant pathology. The history of the department by George Fischer (Plant Disease Reporter Supplement 200:42-55, 1951) was an invaluable source of information on the early years.

\*Earl J. Anderson, 1932  
 \*Kirk Athow, 1946  
 \*Walter J. Bach, 1921  
 \*Kenneth F. Baker, 1930  
 \*Karl E. Baur, 1936  
 Edwin A. Bornander, 1941  
 \*L. W. Boyle, 1923  
 \*Donald M. Coe, 1937  
 \*Silvan I. Cohen, 1939  
 \*D. J. Crowley, 1923  
 \*Bliss F. Dana (in Botany), 1916  
 E. G. Davenport, 1926  
 Harry Elcock, 1926  
 \*W. Harley English, 1935  
 Carl R. Frees, 1933  
 Elwood Fryer, 1940  
 \*D. C. George (in Botany), 1912  
 Ruth Halasey, 1941  
 \*John R. Hardison, 1939  
 Allan R. Hoagland, 1950  
 \*Glenn A. Huber, 1925  
 Paul A. Johnson, 1950

\*Jess Kienholz, 1928  
 \*Hugh C. Kirkpatrick, 1941  
 \*Charles F. Lackey, 1924  
 \*John Large, 1926  
 \*Lindsey Loring, 1931  
 \*Neil A. MacLean, 1943  
 \*John A. Milbrath, 1934  
 Henry C. Newbom, 1933  
 \*George A. Newton, 1925  
 \*George Nyland, 1940  
 \*Charles S. Parker, 1923  
 Clarence Piper, 1950  
 \*William E. Rader, 1940  
 Carl K. Riesenweber, 1938  
 \*George D. Ruehle, 1923  
 \*Jack F. Schafer, 1942  
 \*Otto F. Schnellhardt, 1933  
 Clarence H. Sonderman, Jr., 1948  
 \*C. H. Spiegelberg, 1924  
 \*Roderick Sprague, 1924  
 \*Richard H. Wellman, 1935  
 Heston O. Weyrich, 1936  
 \*John Y. Woo, 1950

For all I know, some of those not marked with an asterisk could have become county agents, farmers, etc., and used their plant pathology. Washington was a fertile source of plant pathologists.

The BS in plant pathology at WSU in the early years required about four courses in plant pathology, so depth of knowledge was obtained. The BS in agriculture at Arkansas (1937-1941) was an unspecialized degree. Specialization at the BS level came shortly after that. WSU may have been unusual in offering specialized BS degrees. The Heald family established a scholarship for the outstanding undergraduate major in Plant Pathology. The fellowship was changed so that it is awarded to an outstanding graduate student, where competition exists. For at least 30 years we have not encouraged BS students to major in plant pathology, but one or two usually do.

#### *Advanced Degrees*

In Europe in the 1850s to 1870s, when plant pathology was starting as a special science, most were trained as doctors, lawyers, biologists in general and even as clergy, with a "classic" education. H. B. Humphrey and F. D. Heald appear to be transitional figures, trained in biology yet with thorough skills in German, French, and Latin.

Heald's manual, 1933, p. 7, describes the training of a plant pathologist as follows. "The problems with which the professional plant doctor must deal call for the broadest kind of a training. He must be well grounded in the fundamentals of pure botany, including plant physiology, histology, and taxonomy of seed plants; on account of the importance of fungous diseases, a detailed knowledge of systematic mycology is an essential part of his equipment; the increasing importance of bacteria as producers of plant disease would make him something of a bacteriologist; the similarity of plant troubles to those caused by insects leads him into the field of economic entomology; without a thorough training in chemistry and physics, he could make but little real progress; since the final goal of the plant doctor is the prevention of crop losses, or the destruction of plants or plant products, he must be in sympathy with agriculture in general and should have a good working knowledge of agronomic, soil and horticultural science; and, finally, in order

that the researchers of the world may be an open book to him, the ability to read Latin, German, and French should be acquired."

The broad background stressed by Heald is no longer possible. Students today must take more chemistry and mathematics (and physics?) to become competent in the more advanced techniques and to properly use modern equipment.

The typical BS in Agriculture (1937-1941) included soils, genetics, horticulture, agronomy, entomology, botany, economics, agricultural engineering, animal science courses, and two chemistry courses (introductory and one semester of organic chemistry), but no physics or foreign languages. When I enrolled in graduate school at the University of Wisconsin I had to take Taxonomy, Plant Physiology, Plant Anatomy, and a Zoology course to correct deficiencies. The zoology requirement angered me because I had had two excellent entomology courses, courses including animal nutrition, enzymes and hormones in animals, their digestive systems, etc., but none of this satisfied the zoology requirement because they were not taught in a zoology department. I took Animal Parasites of Man, a course in the medical curriculum, to correct this deficiency. I still resent the denigration of zoology learned within the College of Agriculture, but such is bureaucracy. Plant Pathology was a biological science and biology included zoology and you couldn't get a Ph.D. at Wisconsin in a biological science without at least one zoology course. Statistics and Experimental Design were required by the College of Agriculture at Wisconsin. The Department of Plant Pathology required Mycology, Diseases of Plants (a general course), Diseases of Field Crops, Diseases of Fruit Crops, Diseases of Vegetable Crops, Methods, reading German and French in the 1940s. Note the University, College, and Departmental requirements. At WSU, other than a required number of graduate courses, the individual departments control requirements for advanced degrees.

At Wisconsin, at least from 1941 to 1948, the German exam consisted of a written translation from a German plant pathology book, with a dictionary, that required two hours of the student's time and that of the German professor who checked the translation. The French exam required 20 minutes, or even less. You took a French pathology book to the French professor, he selected a page, and you started to read. If you could read it, it was all over. And there was no charge for either exam. I still think the latter (French) the most efficient way to examine the student. The foreign language requirement at WSU for the Ph.D. degree was dropped in 1989.

Recipients of advanced degrees in plant pathology at Washington State University, date and major professor, 1922-1988.

Adams, E. B., Jr. 1982. Line.  
Allan, C. R. 1975. Duran.  
Al-Musa, A. M. F. 1979. Mink.  
Anderson, E. J. 1934. Jones.  
Anderson, J. F. 1967. Duran.  
Annone, J. G. 1984. Hendrix.  
Apt, W. J. 1958. Hendrix.  
Archuleta, J. G. 1979. Easton.

Babadoost, M. 1979. Gabrielson.  
Bach, W. J. 1922. Heald.  
Baker, K. F. 1934. Heald.  
Ballard, G. K. 1972. Mink.  
Bassey, E. O. 1982. Gabrielson.  
Becker, J. G. 1953. Shaw.  
Bjarko, M. E. 1986. Line.  
Blanchette, R. A. 1978. Shaw.

Bonman, J. M. 1980. Gabrielson.  
Boothroyd, C. W. 1941. Heald.  
Bourbals, D. 1958. Fischer.  
Boyle, L. W. 1924. Heald.  
Brandsberg, J. W. 1966. Rogers.  
Britt, J. L. 1979. Maloy.  
Brown, J. K. 1981. Wyatt.  
Buell, C. R. 1988. Hadwiger.  
Bull, C. 1987. Weller.  
Burke, D. W. 1955. Fischer.  
Burleigh, J. R. 1965. Hendrix.  
Burnett, G. 1929, 1932. Jones.  
Caetano, V. R. 1973. Hendrix.  
Callan, B. E. 1985, 1988. Rogers.  
Campbell, L. 1935. Heald.  
Carris, L. M. 1983. Bristow.

- Castello, J. D. 1976. Shaw.  
 Chamswarng, C. 1984. Cook.  
 Chang, K. F. 1983. Line.  
 Charchar, J. M. 1987. Santo.  
 Charchar, M. J. 1987. Kraft.  
 Chidambaram, P. 1976. Bruehl.  
 Christen, A. A. 1974. Rogers.  
 1978. Bruehl.  
 Christenson, J. A. 1971. Hadwiger.  
 Clark, R. L. 1961. Locke.  
 Cody, Y. S. 1985. Gross.  
 Coe, D. M. 1943. Heald.  
 Cohen, S. I. 1942. Heald.  
 Comai, L. 1978. Hendrix.  
 Cordas, D. I. 1971. Duran.  
 Cromarty, R. W. 1970. Easton.  
 Cummings, R. H. 1953. Wright.  
 Cunfer, B. M. 1972. Bruehl.
- Dade, C. A. 1957. Graham.  
 Damsteegt, V. D. 1962. Bruehl.  
 Daniels, B. A. 1975. Graham.  
 Daniels, C. H. 1987. Hadwiger.  
 Daniels, D. L. 1975. Hadwiger.  
 Davidson, R. M., Jr. 1971. Bruehl.  
 Davis, J. L. S. 1977. Maloy.  
 Dietz, S. M., Jr. 1963. Fischer.  
 Dilley, M. A. 1979. Covey.  
 Dobson, R. L. 1978. Hadwiger.  
 1982. Gabrielson.  
 Duran, R. 1958. Fischer.  
 Durrieu, G. 1966. Fischer.
- Echandi, R. J. 1962. ??  
 English, W. H. 1940. Heald.
- Fernandes, J. M. 1984, 1985.  
 Hendrix.  
 Fernandez, J. A. 1975. Duran.  
 Filer, T. H., Jr. 1964. Hendrix.  
 Frederick, L. 1952. Locke.  
 Fuentes-Davila, G. 1984. Duran.  
 1988. Gabrielson.  
 Fukui, R. 1988. Cook.
- Ghodbane, A. 1972. Hendrix.  
 Glawe, D. 1978. Skotland. 1982.  
 Rogers.  
 Goheen, A. C. 1953. Fischer.  
 Gordon, C. C. 1960. Shaw.  
 Goree, H. K. 1973. Shaw.  
 Govindu, H. C. 1958. Fischer.  
 Graham, S. O. 1954. Blodgett.
- Grand, L. F. 1968. Rogers.  
 Green, N. E. 1974. Graham.  
 Grove, M. D. 1976. Maloy.  
 Gurusiddaiah, S. 1970. Graham.
- Hajinikhassan, N. 1987. Covey.  
 Halisky, P. M. 1956. Holton.  
 Hamid, A. 1959. Locke.  
 Hammer, W. 1988. Chastagner.  
 Hannan, R. M. 1983. Kaiser.  
 Harder, D. E. 1968. Kirkpatrick.  
 Harrington, T. C. 1980. Shaw.  
 Harrison, W. C. 1966. Hendrix.  
 Harvey, A. E. 1968. Shaw.  
 Hassan, S. 1982, 1984. Thomas.  
 Hazelwood, D. 1983. Wyatt.  
 Hess, S. L. 1971. Hadwiger.  
 Hoes, J. A. 1963. Bruehl.  
 Hoffman, J. T. 1977. Rogers.  
 Hoffmann, J. A. 1961. Fischer.  
 Holtzman, O. V. 1955. Shaw.  
 Hopp, A. D. 1972. Bruehl.  
 Horvath, J. 1966. Locke.  
 Howell, C. R. 1967. Duran.  
 Howell, W. E. 1970. Mink.  
 Howie, W. J. 1985. Cook.  
 Huber, G. A. 1929. Heald.  
 Hudson, D. N. 1972. Maloy.  
 Hwang, S. F. 1983. Cook.
- Inglis, D. A. 1978. Maloy. 1982.  
 Cook.  
 Ingram, D. M. 1987. Cook.
- Jacobs, D. L. 1984. Bruehl.  
 Jafri, A. M. 1972. Mink.  
 Janisiewicz, W. J. 1980, 1983.  
 Skotland, Covey.  
 Jensen, J. 1980, 1984. Rogers.  
 Johnson, F. 1939. Jones.  
 Jones, B. L. 1967. Rogers.  
 Jong, S. C. 1970. Rogers.
- Kadow, K. J. 1933. Jones.  
 Kainz, M. S. 1980. Hendrix.  
 Kendra, D. F. 1984, 1986.  
 Hadwiger.  
 Kenerley, C. M. 1975. Rogers.  
 Kienholz, J. R. 1929. Heald.  
 Kiyomoto, R. 1975. Bruehl.  
 Knesek, J. E. 1973. Mink.  
 Koenigs, J. M. 1965. Graham.  
 1965. Shaw.

Kondo, W. T. 1967. Graham.  
Kosuge, T. 1955. Locke.

Lai, P. Y. 1967. Bruehl.  
Leben, S. D. 1985. Easton.  
Lin, Y. S. 1975, 1978. Cook.  
Lipps, P. E. 1977, 1979. Bruehl.  
Lloyd, E. H. 1968. Hendrix.  
Loman, A. A. 1968. Rogers.  
Long, L. E. 1977. Hendrix.  
Loschke, D. C. 1980. Hadwiger.  
Love, C. S. 1986. Bruehl.  
Lowther, C. V. 1948. Holton.

MacKenzie, M. 1981. Shaw.  
MacLean, N. A. 1949. Sprague.  
Magnuson, J. E. 1982. Easton.  
Malmgren, M. M. 1977. Rogers.  
Manandhar, J. B. 1972. Bruehl.  
Mankin, C. J. 1952, 1953. Fischer.  
Martin, N. E. 1972. Hendrix.  
McCoy, R. J. 1982. Kraft.  
McCracken, F. I. 1972. Hendrix.  
McDonald, G. I. 1969. Graham.  
McMillan, R. T., Jr. 1968. Locke.  
Meiners, J. P. 1949. Fischer.  
Menzies, J. D. 1942. Harrar.  
Michaels, E. M. 1983. Chastagner.  
Milne, D. R., Jr. 1967. Haglund.  
Milus, E. A. 1978, 1984. Line.  
Moore, K. J. 1978. Cook.  
Moulton, G. A. 1977. Schafer.  
Murray, T. D. 1980, 1983. Bruehl.

Nasser, L. C. B. 1983. Hendrix.  
Nelson, B. D. 1980. Duran.  
Neto, N. 1977. Hendrix.  
Newton, G. A. 1927. Heald.  
Nuss, T. S. 1980. Hendrix.  
Nyland, G. D. 1948. Fischer.

Ong, C.-A. 1987. Mink.  
Oster, J. J. 1981. Shaw.

Pachinburavan, A. 1981. Hendrix.  
Park, J. Y. 1951. Sprague.  
Parker, C. S. 1923. Heald.  
Pettey, T. M. 1982. Shaw.  
Pierobom, C. R. 1983. Hendrix.  
Prestes, A. M. 1974, 1976.  
Hendrix.  
Probasco, E. G. 1971. Skotland.

Qayoum, A. 1976. Line.

Rakotondradona, R. 1979, 1983.  
Line.  
Reeves, E. L. 1937. Heald.  
Regner, K. M. 1987. Johnson.  
Reis, E. M. 1980. Cook.  
Reyes, A. A. 1959. Hendrix.  
Rich, A. E. 1950. Locke.  
Rivera, J. E. 1958. Bruehl.  
Roberts, D. D. 1971. Kraft.  
Ruehle, G. D. 1930. Heald.

Saksena, K. N. 1967. Mink.  
Schnellhardt, O. F. 1935. Heald.  
Schultz, T. R. 1984. Gabrielson.  
1988. Line.  
Schuster, M. L. 1946. Fischer.  
Scott, R. S. 1985. Line.  
Seybert, L. J. 1980. Wyatt.  
Siang, W. N. 1953. Holton.  
Silbernagel, M. J. 1961. Holton.  
Sikorowski, P. P. 1964. Shaw.  
Sitton, J. W. 1975. Cook. 1984.  
Pierson.  
Smiley, R. W. 1972. Cook.  
Smith, T. J. 1975. Hendrix.  
Snow, M. D. 1974. Hendrix.  
Spalding, D. H. 1960. Bruehl.  
Spiegelberg, C. H. 1925. Heald.  
Spinner, G. A. 1977. Rogers.  
Sprague, R. 1925. Heald.  
Stahl, S. A. 1985. Rogers.  
Stiers, D. L. 1974. Rogers.  
Stoner, M. F. 1967. Graham.  
Strausbaugh, C. A. 1985. Maloy.  
1988. Murray.  
Strobel, J. W. 1959. Graham.

Sung, J. M. 1979. Cook.  
Sutherland, J. B. 1978. Cook.

Tachibana, H. 1963. Holton.  
Tarry, J. C. 1968. Rogers.  
Teasdale, J. R. 1975. Hadwiger.  
Thomas, A. G. 1950. Shaw.  
Thomas, J. M. 1971. Graham.  
Thyr, B. C. 1964. Shaw.  
Toko, H. V. 1957. Bruehl.  
Tu, J. C. 1967. Hendrix.

Uyeda, I. 1975, 1978. Mink.

Wadi, W. A. 1980, 1982. Easton.  
Waldron, H. M. 1969. Shaw.  
Wang, W. Y. 1983, 1985. Mink.  
Watts, D. K. 1972. Rogers.  
Weimer, R. J. 1971. Skotland.  
Wellman, R. H. 1939. Heald.  
White, J. C. 1970. Hendrix.  
Wicker, E. F. 1965. Shaw.  
Williams, R. E. 1972. Rogers.  
Wilson, A. D. 1988. Rogers.  
Woloshuk, C. P. 1986. Hadwiger.

Xu, G.-W. 1984, 1987. Gross.

Yerkes, W. D., Jr. 1952. Shaw.

Production of M.S. and Ph.D. degrees by year, 1922-1988.

<u>Year</u>	<u>No.</u>	<u>Year</u>	<u>No.</u>	<u>Year</u>	<u>No.</u>	<u>Year</u>	<u>No.</u>
1922	1	1942	2	1960	2	1976	5
1923	1	1943	1			1977	8
1924	1	1944	0			1978	11
1925	2	1945	0	1961	3	1979	8
1926	0			1962	2	1980	12
1927	1			1963	3	1981	4
1928	0	1946	1	1964	3		
1929	3	1947	0	1965	4		
1930	1	1948	2	1966	4	1982	10
1931	0	1949	2	1967	9	1983	12
1932	1	1950	2	1968	7	1984	12
1933	1	1951	1	1969	2	1985	9
1934	2	1952	3	1970	5	1986	4
1935	2	1953	5	1971	8		
1936	0	1954	1	1972	12		
1937	1	1955	3			1987	7
1938	0	1956	1			1988	7
1939	2	1957	2	1973	3		
1940	1	1958	5	1974	4		
1941	1	1959	3	1975	11		

During the Heald period (1917-1941) an average of 1.05 advanced degrees per year were given; 0.75 during the turmoil of Harrar and Anderson; 2.20 per year under Fischer; 5.17 under Shaw; 7.22 under Schafer; and 9.00 under Davison. The output per year has declined recently, and numbers will remain relatively low for some time.

*Memories, Jack Schafer, 1988*

My first contact with WSU Plant Pathology was as a faculty child in Pullman in the 1920s and 30s. Common bunt or stinking smut was a well-known entity to anyone who was aware of wheat in the Palouse during those years. My memory is that Dr. Heald, with his German doctorate, was the only agricultural department head with a doctor's degree during that period. With his two text books, he was considered a major scholar. He had had a broad professional experience before coming to WSU in his early 40s about 1915 and was somewhat older than the other department heads of the 1920s and 30s, most all of whom had extended tenure as heads during that period. Although my dad was the Agronomy head during those same decades, I don't have any personal-type contact memories of Dr. Heald from my early childhood.

Both the Agronomy and Plant Pathology programs were considerably expanded with the addition from the USDA of a cereal pathologist and a geneticist and a forage pathologist and a geneticist between about 1929 and 1936. In Plant Pathology this must have nearly doubled the professional faculty-level personnel. C. S. Holton, a new doctorate from Minnesota arrived in Pullman about 1932 to fill the cereals pathology program and provide a more intensive attack on wheat bunt. H. H. Flor had been there previously, but I did not know him at that time.

WSU was the major summer employer of Pullman kids. Because of rather strict nepotism rules, my job opportunities with the college were limited, at least in agriculture. Although I am sure that I was still



somewhat of an insider, I was able to get a job on Stu Holton's USDA cereal pathology crew the summer that I was 15, along with Ronnie Barbee, a close friend and another Agronomy faculty kid. George Nyland, the undergraduate assistant, was our straw boss. We spent the summer on our knees, with hand tally counters making the counts in smut-inoculated nursery rows that provided data for smut percentage evaluations. We worked 10 hours a day, six days a week, for 30 cents an hour. The knees did get a bit sore and stiff at times, but they were young and durable. Ronnie and I carried our lunches, and ate under the maples along the adjacent golf course, a rather pleasant mid-day break.

After three more summers working primarily for Dr. Holton, Orville Vogel, and D. C. Smith, the USDA geneticists, I was asked by George Fischer, the forage pathologist, as a college sophomore, to be his undergraduate assistant. I succeeded John Hardison who had just graduated and gone on to the University of Michigan, Fischer's alma mater, for graduate study. I remained three years in this position with Dr. Fischer. He was a "hands off" boss and a very enjoyable and interesting man for whom to work. As successor to George Nyland, Jack Meiners had a similar job with Dr. Holton.

I took the beginning plant pathology course from Dr. Heald that spring. This was a very well organized, intensive, difficult course. Dr. Heald gave the lecture and Jim Menzies, then a Ph.D. candidate, taught the laboratory. There was much substance in Dr. Heald's course, but he was a very dry lecturer. Some of my less interested friends were not motivated and were lucky to get D's (then K's, I believe) in this class. About this time I decided, nevertheless, to major in Plant Pathology. I liked plants and the economic mission of agriculture. I was still somewhat concerned about being able to handle the new-to-me microscopic world, however. Jim Menzies was a particularly helpful, knowledgeable lab teacher. I especially remember the exercise on stem rust and how impressed I was with the intricacies of spore stages and host specialization.

A year's sequence in mycology followed. Don Coe was the teacher, as a full-time faculty instructor but also a graduate student. I thought that Don was a particularly good teacher. The world of fungi became personal and exciting in his hands. Our field trips to Moscow Mountain in a moist fall opened up a whole new vista of the fruiting structures of mycorrhizal fungi. In hindsight, my five-course program in plant pathology as an undergraduate was surely too specialized and intensive, and excluded some relevant basic courses, but nevertheless was interesting, enjoyable, and motivating.

My close friend, Eddy Bornander, from a florist family in Tacoma, was in the mycology class, as I believe were all of the other undergraduate majors: Kirk Athow, Neil MacLean, Joe Rock, and myself. Eddy graduated that year, 1941, went immediately to the Army from R.O.T.C., and was subsequently lost in the war in Europe. I believe that year in mycology was the last that I saw him. His death was one of the many tragedies of that era, and a great loss to Plant Pathology.

As commonly, Plant Pathology was largely a graduate department. In addition to Coe and Menzies, I remember Sy Cohen, Dick Wellman, and Carl Boothroyd. Harley English had just finished, and Hardison went elsewhere to graduate school after a bachelor's degree at WSU. Cohen was quite an athlete and even as a busy graduate student was some sort of internal college handball champion three years in a row, I believe.

As part of its graduate program, the department maintained a seminar. I was able to participate in this as an undergraduate. This was a joint program with the University of Idaho, which gave us a broader horizon and further contacts. I first met Bob Patton in seminar, an Idaho graduate student, with whom I was later a co-student at Wisconsin, and who just recently retired as a forest pathologist after a long career at Wisconsin. I don't believe that I gave an oral report, but did prepare written literature reviews in lieu thereof. The one I remember best was on the genus *Trichoderma* as an antagonistic and antibiotic fungus in the soil, a forerunner of subsequent efforts on biological control.

Dr. Heald was forced into retirement at the end of the 1940-41 college year, much against his desires, I believe. I suspect that there was not a standardized retirement schedule, nor an adequate retirement financial program. In spite of his unhappiness, Dr. Heald at that time left his extensive personal library to the department to become the core of the subsequent outstanding departmental library.

It is my understanding that George Fischer turned down the department head offer in consideration of his close relations with Dr. Heald. The recruiters then went outside and brought in J. George "Dutch" Harrar, of subsequent Rockefeller Foundation fame. Dr. Harrar was a personable, aggressive, youngish new leader. He even taught a class in forest pathology which I took. I remember it was a good but not extra special course which he probably didn't quite have time for, in the process of developing his leadership in his new department. My very pleasant memory of Dr. Harrar was his helpful assistance in making graduate school applications. His contact at Minnesota was especially useful (although I had a better offer from Wisconsin). With World War II on us at that time, I obviously didn't immediately get to graduate school.

I believe that Dutch Harrar could have been a very successful department head, as evidenced by his later successes. However, bigger things came on quickly, and he moved to head the new Rockefeller Foundation development program in Mexico early the next year. As head of that program he hired Norman Borlaug, John Niederhauser, and Ed Wellhausen, highly successful plant pathologist-breeders, and subsequently became President of the Rockefeller Foundation succeeding Dean Rusk when he became Secretary of State.

My only classmate as an undergraduate in plant pathology was Kirk Athow. Kirk was drafted before the 1942 school year was out, so had to come back after the war to graduate. He subsequently went to Purdue for graduate study and stayed on the faculty there until retirement. When I arrived on the Purdue faculty in 1949, Kirk was an advanced graduate student, and we were colleagues there during my 19-year tenure. When Kirk retired in the mid-1980s he had been a Purdue staff member for about 40 years. Like Jim Menzies he is now retired in western Washington. Like Sy Cohen, he was also one of the student-athletes, being the Northern Division Intercollegiate 139-pound wrestling champion two years.

I continued to work for George Fischer during the summer of 1942, following my graduation, until I entered the Army in August.

I left Pullman in August of 1942 to enter the Army for a nearly 4-year stint. I visited periodically in Pullman in successive years, as this was my parents' home. I believe that I actually worked a few days in the lab for Dr. Holton during a furlough or leave in 1943. I also met Dr. Anderson, the WSU alumnus who succeeded Dr. Harrar early in 1943. Dr. Harrar stayed 1 1/2 years, and Dr. Anderson 2 1/2 years before returning to Hawaii where he had a long-time career. In 1945, George Fischer was made department head, four years after first being offered the position.

*Avery Rich, 1988*

I'll put down a few of my memories of the department from 1947 to 1951. Probably they are covered in George Fischer's history. Gardner Shaw, Chuck Wright and I arrived on the scene July 1, 1947. Other faculty at that time were Geo. Fischer, Head; Roderick Sprague, Seth Locke, George Nyland, and C. S. Holton, USDA. Gardner was a new Ph.D. from Wisconsin. He taught mycology, advanced mycology, and probably forest pathology. Chuck Wright worked at Prosser on Little Cherry disease most of the time. He was a new Ph.D. from Cornell. I had my M.S. from U. of Maine, and had worked at Rhode Island State College for four years. I came to WSU in a dual role as a full-time faculty member and also as a Ph.D. candidate. I taught the labs for the beginning plant path course. Geo. Nyland gave the lectures. In 1950 I organized and taught a new course called Practical Plant Pathology. It was designed for students without much background in Botany and Plant Science who might become county agents or Agricultural teachers. My research dealt with potato diseases, more specifically with leafroll and phloem necrosis.

The title of my dissertation was Phloem Necrosis of Potatoes. Apparently the commercial potato growers were having serious problems with this disease, and no one could decide whether or not it was the same disease which we had in Maine or a new disease. I obtained tubers from Maine which were infected with leafroll and/or net necrosis and grew them in Washington along side of Washington potatoes showing similar symptoms. The disease from the two areas proved to be identical when grown under the same conditions. Potatoes grown in Washington exhibited much more severe symptoms of secondary infection than did potatoes grown in Maine, probably due to the warmer and longer growing season which favored a large build-up of aphids. The spread of leafroll and development of current-season leafroll symptoms in Washington were very similar to those in Rhode Island, but net necrosis was never a problem in Rhode Island. I received my Ph.D. in 1950, based on my course work and dissertation, and was promoted to Asst. Professor. (I had to be listed as Instructor while I was doing graduate work.) I resigned my position in Sept., 1951 to accept a position as Associate Professor of Plant Pathology at U. New Hampshire.

The Shaw, Wright, and Rich families lived in North Fairway in 1947. Then they moved into new houses on Fisk St. on Military Hill, houses which the college purchased from the builders.

Seth Locke and I built a concrete potato storage cellar out in the field near our research plots beyond the orchard East of South Fairway. We used the plans for a tornado shelter from Oklahoma. It was built into a hillside and covered with earth. Potatoes kept very well all winter without freezing. I purchased a new Ford tractor and attachments (plow, harrow, etc.) for our field research also.

Pullman had no field equipment when I arrived. The branch stations, especially Prosser and Mt. Vernon, were well equipped. I purchased a new pick-up truck for my field work, too. The Station had some money left over which had to be spent before a certain date, and I took full advantage of it. I had research plots at Pullman, Prosser, Vancouver, Mount Vernon, Lynden, Long Beach, and Colville. My travel budget was larger than my entire budget when I first came here.

George Fischer was a wonderful chairman. He was sympathetic, supportive, accessible, and a lot of fun. He and Geneva frequently invited the entire department, including wives, out to their ranch for supper and square dancing. They took care of our two daughters for about a week while we attended meetings in Victoria, B.C. We hated to leave them, but the pull to return to New England was too great.

Possibly my greatest contribution was Popular Bulletin 195, Potato Tuber Diseases, Defects, and Insect Injuries by E. C. Blodgett and Avery E. Rich. It was published jointly with Idaho. Every disorder was illustrated with a full-page picture. I demonstrated that net necrosis of Russet Burbank (Netted Gem) potato tubers could be controlled by controlling aphids with some of the new insecticides. I taught the potato inspectors and seed growers to recognize bacterial ring rot of potatoes, caused by *Corynebacterium sepedonicum*. Their fields were loaded with it when I arrived, but they got busy and cleaned up their seed lots by practicing strict sanitation.

Contributions which I have made at UNH which helped this state and the nation are pioneer research dealing with salt injury to roadside trees; demonstrating the fact that *Pratylenchus penetrans* nematodes can cause black root rot of strawberry in the absence of fungi; release of two tomato cultivars resistant to late blight; identification of two apple virus diseases -- stem pitting and dapple apple; release of a new pea cultivar resistant to root rot caused by *Fusarium solani* f. sp. *pisi*; author of text and reference book, Potato Diseases, Academic Press, 1983.

Hopefully I have made and will continue to make a contribution through my former students. There are about twenty M.S. and fifteen Ph.D.'s who are out there with their microscopes, test tubes, electron microscopes, computers, cell cultures, etc., manipulating genes and doing other weird things which I never heard of in graduate school. Dr. Tseh-An Chen, Rutgers (Spiroplasmas and nematodes), Dr. Charles Niblett earned his B.S. at U.N.H. and his Ph.D. at U.C. Riverside (Chmn. of Plant Path., U. of Florida) and Dr. James E. Hunter (Assoc. Dir., N.Y. Agr. Exp. Sta., Geneva) are among my former

students. Other B.S. students who went elsewhere for their Ph.D. include: Dr. Wayne Sinclair (Cornell) and Dr. Wm. Merrill (Minn), now at Penn State. I could not have done these things if the Plant Path. Dept. at Washington State had not given me the unusual opportunity of coming to Pullman and working for my Ph.D. degree holding a full-time faculty position.

I spent considerable time this past year writing the history of the New Hampshire Agr. Exp. Station for our centennial. I have been working on a history of the Dept. of Botany and Plant Path. also.

*Jack Meiners, 1988*

I was an undergraduate student at WSU from 1937-42 - part of that time as a major in the department - but all of the time working part time in the Department. I was a graduate student from 1946-1949, working on grass smut under George Fischer. I was a faculty member in the department from 1950-53 and a part of the USDA Smut Lab from 1953-1958. I took beginning Plant Pathology from Dr. Heald in 1938 or 39. Harley English was lab instructor. Folke Johnson was a grad student. L. K. Jones was the only other faculty member at that time, though Dan Coe came a couple of years later and taught most of the courses. Dick Wellman was also a graduate student. Leo Campbell would come each winter and prepare his report of the summer's research.

#### *Some Contributions of Graduate Students to Research*

Some subjects, such as common bunt (*Tilletia caries* and *T. foetida*), were studied primarily by permanent faculty, state or USDA, while others, such as head smut of corn, were studied primarily by students. In most cases students and faculty studied the same subjects, with student studies in some cases supplementing faculty studies; in other cases student studies were primary with the faculty being supportive.

Students contributed strongly to cereal rust research after 1965. Student studies have significantly advanced our knowledge of wheat foot and root rots, snow mold and snow rot, Cephalosporium stripe, and barley yellow dwarf, all important problems in Washington. An interesting contribution to international plant pathology was the series of studies of *Septoria* diseases of wheat, primarily by students from foreign countries, mainly Brazil and Argentina. In forest pathology, studies of white pine blister rust and of the failure of the antibiotic phytoactin to control it are prominent. Fluorine damage to pines, wood rots, mycorrhizal fungi, role of fungi and associated organisms in rotting forest debris and slash, permitting recycling of nutrients are of interest. Fundamental mycology has long been a major activity within the department. Fungal and host physiology, especially the synthesis and role of natural antifungal substances (phytoalexins) have been a major study.

Diseases of vegetables, particularly of onions, asparagus, potatoes, peas, beans and cabbage have been extensively studied. The cabbage seed industry is important in Washington and seed-borne pathogens of cabbage seed have been extensively studied.

The chronology of studies of rots of apple and pear fruits is significant. Before the Tree Fruit Research and Extension Center was established, much of this research was done at Pullman, with 10 theses from 1922 to 1940, and only one, in 1984, after that. Students have contributed significantly to *Phytophthora cactorum* in apple orchards and to virus diseases of the tree fruits.

In general, diseases caused by fungi have been studied most, with virus diseases second, bacterial diseases third, with only two theses involving nematodes. Historically, the department has been strongest in studying fungi and their activities.

Theses are recorded chronologically in the departmental records, but I organized them by subject to emphasize the problems studied.

### Stinking Smut

- 1929 Kienholz, Jess R.  
(Heald) Cultures and strains of the stinking smut of wheat
- 1948 Lowther, Conley V.  
(Holton) Studies on Chlamdospore germination and other developmental phases in physiologic races of Tilletia caries and T. foetida
- 1953 Siang, Wan Nien  
(Holton) Studies on the biology of certain Tilletia species on grasses and wheat
- 1958 Duran, Ruben  
(Fischer) Monograph of the genus Tilletia
- 1961 Silbernagel, Matt J.  
(Holton) Hybridization between Tilletia caries and T. controversa
- 1965 Mujica, Francisco Lucas  
(Purdy) Interaction of common bunt (Tilletia caries and T. foetida) and stripe ruse (Puccinia striiformis) in the same wheat plant

### Head Smut

- 1952 Mankin, Cleon J.  
(Fischer) Studies of the biology of Sphacelotheca reiliana causing head smut of corn
- 1953 Mankin, Cleon J.  
(Fischer) The biology of head smut corn caused by Sphacelotheca reiliana
- 1975 Smith, Timothy J.  
(Hendrix) Head smut of corn: some factors associated with infection and prediction
- 1977 Long, Lynn E.  
(Hendrix) Some factors related to the disease complex of head smut of maize: Sphacelotheca reiliana (Kuhn) Clint.
- 1979 Britt, Jeffrey L.  
(Maloy) Methods for rapid screening of sweet corn for resistance to Sphacelotheca reiliana (Kuhn) Clint., the head smut fungus

### Kernel Smut

- 1984 Fuentes-Davila, Guillermo  
(Duran) Biology of Tilletia indica Mitra

#### Flag Smut

- 1975 Allan, Carolyn R.  
(Duran) Urocystis agropyri: Chemical-physical factors affecting spore germination
- 1980 Nelson, Berlin D., Jr.  
(Duran) Studies on the biology of Urocystis agropyri the causal organism of flag smut of wheat

#### Grass Smuts

- 1949 Meiners, Jack P.  
(Fischer) Studies on the biology of Ustilago bullata causing head smut of grass
- 1958 Govindu, Heirehalli C.  
(Fischer) Physiological variation in the stripe smut of grasses, Ustilago striiformis
- 1961 Hoffmann, James A.  
(Fischer) Induced hybridization between certain Graminicolous species of Ustilago
- 1963 Dietz, Sherl M.  
(Fischer) Fundamental studies of the biology and parasitism of the stem smut of grasses, Ustilago hypodytes (Schlecht.) Fries.
- 1971 Cordas, David I.  
(Duran) Some biological aspects of the Nolina smut fungus, Clintamra nolinae comb. nov.
- 1975 Fernandez, John A.  
(Duran) Sorosporium consanguineum and Ustilago enneapogonis morphology, cytology and dissociation in culture

#### Oat Smut

- 1956 Halisky, Philip M.  
(Holton) Inheritance of pathogenicity in Ustilago avenae
- 1967 Kondo, William T.  
(Graham) The responses of Ustilago avenae (Pers.) Rostr. to preservation by lyophilization

#### Ergot

- 1953 Cummings, Robert H.  
(Wright) Biologic and pathogenic studies of Claviceps purpurea as a contribution toward the production of alkaloids on artificial media

### Stripe Rust

- |      |                                    |  |
|------|------------------------------------|--|
| 1965 | Burleigh, James R.<br>(Hendrix)    | The winter biology of <u>Puccinia striiformis</u> West, in the Pacific Northwest   |
| 1967 | Tu, Jui-Chang<br>(Hendrix)         | The summer biology of <u>Puccinia striiformis</u> West in Southeastern Washington  |
| 1968 | Lloyd, Edward H., Jr.<br>(Hendrix) | Influence of low temperature on the behavior of wheat leaves infected with <u>Puccinia striiformis</u> West                              |
| 1970 | White, Joseph C.<br>(Hendrix)      | Factors influencing the quantity and quality of urediospores produced by <u>Puccinia striiformis</u> West                                |
| 1972 | Martin, Neil E.<br>(Hendrix)       | Anatomical and physiological features of baart wheat roots affected by stripe rust   |
| 1974 | Snow, Michael Dennis<br>(Hendrix)  | Influence of fall infection by <u>Puccinia striiformis</u> West. on the growth and yield of wheat  |
| 1976 | Qayoum, Abdul<br>(Line)            | Resistance of wheat to <u>Puccinia striiformis</u> West  |
| 1980 | Nuss, Terrina Stokes<br>(Hendrix)  | The effect of temperature and light on stripe rust caused by <u>Puccinia striiformis</u> West  |
| 1984 | Milus, Eugene Albert<br>(Line)     | Inheritance of HTAP resistance to stripe rust in wheat   |
| 1985 | Scott, Randolph S.<br>(Line)       | Control of stripe rust and leaf rust of wheat in Washington with foliar applications and seed treatments of sterol-inhibiting fungicides |

### Leaf Rust

- |      |                                |  |
|------|--------------------------------|--|
| 1978 | Milus, Eugene A.<br>(Line)     | Resistance to leaf rust of wheat   |
| 1979 | Rakotondradona, Remi<br>(Line) | Seed treatment to control stripe rust and leaf rust of wheat   |
| 1983 | Chang, Kan Fa<br>(Line)        | Urediospore germination and growth and development of <u>Puccinia recondita</u> in leaves of wheat cultivars |
| 1983 | Rakotondradona, Remi<br>(Line) | Use of seed treatments to control stripe rust and leaf rust of wheat in Washington                           |

#### Stem Rust

- 1972 McCracken, Francis I.  
(Henrix) The winter behavior of the uredial stage of Puccinia graminis tritici

#### Biocontrol Rust

- 1982 Adams, Edward Blair Jr.  
(Line) Use of a plant pathogen (Puccinia chondrillina) to control rush skeletonweed (Chondrilla juncea)

#### Dryland Foot Rot of Wheat

- 1963 Hoes, Josephus A.  
(Bruehl) Dynamics of the mycoflora of subterranean parts of winter wheat in the dryland area of Washington
- 1975 Sitton, Jerry W.  
(Cook) Survival of Fusarium roseum f. sp. cerealis cvs. 'Culmorum' and 'Graminearum' as chlamydo spores in soil
- 1978 Lin, Yi-Sheng  
(Cook) Mechanisms involved in the control of Fusarium roseum 'Avenaceum' by suppressive soil
- 1978 Sutherland, John B. IV  
(Cook) Microbial production of ethylene in soil
- 1979 Sung, Jae-Mo  
(Cook) Effects of water potential on reproduction and spore germination by Fusarium roseum graminearum, culmorum, and avenaceum
- 1982 Inglis, Debra Ann  
(Cook) The Fusarium roseum sens. Synder & Hansen complex in wheat and wheat-fields in Eastern Washington

#### Take-All

- 1972 Smiley, Richard Wayne  
(Cook) Relationship between rhizosphere pH changes induced by root absorption of ammonium-versus nitrate-nitrogen and root diseases, with particular reference to take-all of wheat
- 1978 Comai, Luca  
(Hendrix) Pathogenicity of Microdochium bolleyi (Sprague) de Hoog and Hermenides, Fusarium equiseti (Corda) Sacc., Fusarium culmorum (Smith) Sacc. and Gaeumannomyces graminis (Sacc.) Arx and Oliver and responses of wheat to different soil treatments in mist culture



- 1978 Moore, Kevin J.  
(Cook) The influence of no-tillage on take-all of wheat
- 1980 Reis, Erlei M.  
(Cook) Effect of mineral nutrition on take-all of wheat
- 1985 Howie, William Johnston  
(Cook) Factors affecting colonization of wheat roots and suppression of take-all by Pseudomonads antagonistic to Gaeumannomyces graminis var. tritici

#### Strawbreaker

- 1976 Chidambaram, Palani  
(Bruehl) Aspects of the life cycle of Pseudocercospora herpotrichoides (Fron) Deighton
- 1980 Murray, Timothy D.  
(Bruehl) Some factors associated with resistance in winter wheat to Pseudocercospora herpotrichoides
- 1983 Murray, Timothy Daniel  
(Bruehl) Resistance in winter wheat to Pseudocercospora herpotrichoides

#### Pythium Root Rot

- 1983 Carris, Lori Marie  
(Bristow) Movement of the systemic fungicide metalaxyl in soils and its translocation in plants
- 1984 Chamswarng, Chiradej  
(Cook) Etiology and Epidemiology of Pythium root rot of wheat

#### Head Blight

- 1985 Strausbaugh, Carl A.  
(Maloy) Fusarium head blight of wheat in central Washington.

#### Stored Grain

- 1964 Sikorowski, Peter P.  
(Shaw) The interrelation of fungi and insects to deterioration of stored grains

#### Powdery Mildew

- 1971 Thomas, Janis M.  
(Graham) Studies on the physiology of Erysiphe graminis tritici and its host

### Cephalosporium Stripe

- |      |                                   |  |
|------|-----------------------------------|--|
| 1958 | Rivera, Jorge Enrique<br>(Bruehl) | A study of methods of infecting wheat with <u>Cephalosporium gramineum</u> and factors influencing the percentage of infection |
| 1960 | Spalding, Donald H.<br>(Bruehl)   | Mechanisms of the stripe pathogen, <u>Cephalosporium gramineum</u> , in killing cultivated wheat                               |
| 1967 | Lai, Ping-Yuen<br>(Bruehl)        | Aspects of the saprophytic life of <u>Cephalosporium gramineum</u>   |
| 1972 | Hopp, Allan D.<br>(Bruehl)        | The influence of antibiotic production and soil pH on survival of <u>Cephalosporium gramineum</u> in infested wheat straw      |
| 1986 | Love, Connie S.<br>(Bruehl)       | Effect of depth of burial of inoculum and soil pH on Cephalosporium stripe of wheat  |

### Yellow Dwarf

- |      |                                      |  |
|------|--------------------------------------|--|
| 1957 | Toko, Harvey V.<br>(Bruehl)          | Some relationships of insect vectors to isolates of the cereal yellow-dwarf virus  |
| 1962 | Damsteegt, Vernon D.<br>(Bruehl)     | The inheritance of tolerance in barley to barley yellow dwarf  |
| 1973 | Caetano, Veslei da Rosa<br>(Hendrix) | Studies on vectors of the barley yellow dwarf virus on wheat in Brazil, in particular, on <u>Acyrtosiphon dirhodum</u> --Summary in English of Portuguese thesis |
| 1980 | Seybert, Laura J.<br>(Wyatt)         | Identification of barley yellow dwarf virus strains present in Eastern Washington  |
| 1980 | Kainz, Mark S.<br>(Hendrix)          | Response of cereal roots grown in mist culture to barley yellow dwarf virus  |
| 1981 | Brown, Judith K.<br>(Wyatt)          | Irrigated corn as a reservoir for barley yellow dwarf virus and its aphid vectors in Eastern Washington  |
| 1983 | Hazelwood, Donna<br>(Wyatt)          | Barley yellow dwarf virus in eastern Washington: I. Host range of the predominant isolate II. Virulence of various isolates on corn                              |

### Snow Mold and Snow Rot

- |      |                                  |  |
|------|----------------------------------|--|
| 1971 | Davidson, Roy M. Jr.<br>(Bruehl) | Factors affecting the efficiency of sclerotia of <u>Typhula idahoensis</u> as inoculum |
|------|----------------------------------|--|

1972	Cunfer, Barry M. (Bruehl)	The role of the basial stage in the life cycle of <u>Typhula idahoensis</u>
1975	Kiyomoto, Richard (Bruehl)	Mechanism of resistance of winter wheat to the snow mold pathogen, <u>Typhula idahoensis</u>
1977	Lipps, Patrick E. (Bruehl)	Pythium snow rot disease of winter wheat in Washington
1978	Christen, Alice A. (Bruehl)	Interspecific mating relationships between <u>Typhula ishkariensis</u> and <u>T. idahoensis</u>
1979	Lipps, Patrick E. (Bruehl)	Etiology of pythium snow rot of winter wheat
1984	Jacobs, Darrel L. (Bruehl)	Identification, distribution and saprophytic ability of <u>Typhula</u> species in Washington and Idaho

#### SELENOPHOMA AND SEPTORIA

1951	Park, Jai Young (Sprague)	Comparative studies of selenophoma leaf spot diseases on certain grasses
1972	Ghodbane, Allala (Hendrix)	<u>Septoria tritici</u> , a pathogen on wheat: sporulation and inoculation
1974	Prestes, Ariano M. (Hendrix)	Septoria leaf blotch of wheat: Varietal response and influence on growth and yield
1976	Prestes, Ariano M. (Hendrix)	<u>Septoria tritici</u> Rob. Ex. Desm.: Host relationships, varietal response, and influence on the development of wheat roots
1977	Neto, Nelson (Hendrix)	Pathogenic variation in <u>Septoria tritici</u> Rob. ex. Desm.
1981	Pachinburavan, Assanee (Hendrix)	Pycnidiospore germination, penetration, and pycnidial formation of <u>Septoria tritici</u> Rob. Ex. Desm.
1983	Pierobom, Carlos Roberto (Hendrix)	Studies on pycnidial and ascocarpic fungi which cause leaf spots on wheat in Washington
1983	Nasser, Luiz Carlos Bhering (Hendrix)	Studies on microorganisms carried by cereal seeds produced in Brazil and Washington: their influence on seedling vigor, chemical and biological control, and histology of <u>Septoria nodorum</u> (Berk.) Berk. on wheat seed
1984	Fernandes, J. M. (Hendrix)	Studies on <u>Septoria nodorum</u> Berk., a pathogen of wheat in eastern Washington

- 1984 Annone, Juan Gerardo  
(Hendrix) Cultural types of Septoria tritici Rob. ex. Desm.: Occurrence in Washington, stability and virulence
- 1985 Fernandes, Jose Mauricio  
(Hendrix) Epidemiological and biological aspects of Septoria nodorum Berk., a pathogen of wheat in Eastern Washington

#### FOREST PATHOLOGY

- 1955 Holtzman, Oliver V.  
(Shaw) Organisms causing damping-off of coniferous seedlings and their control
- 1964 Thyr, Billy Dale  
(Shaw) Three fungi associated with foliage diseases of Western pines
- 1965 Koenigs, Jerome Wm.  
(Graham) The cytochemical localization of specific dehydrogenases in blister rust cankers of Western white pine
- 1965 Koenigs, Jerome W.  
(Shaw) Histochemical localization of dehydrogenase enzymes in Cronartium ribicola
- 1965 Wicker, Ed F.  
(Shaw) Biology and control of dwarf mistletoes on Douglas fir and Western Larch
- 1966 Brandsberg, John W.  
(Rogers) A study of fungi associated with the decomposition of coniferous litter
- 1966 Harrison, William Clark  
(Hendrix) The influence of some environmental factors on the response of Ponderosa pine to atmospheric fluorides
- 1967 Jones, Billy L.  
(Rogers) The interrelationships among Chermes cooleyi Gill., fungi, and the foliage of Pseudotsuga menziesii (Mirb.) Franco
- 1967 Stoner, Martin F.  
(Graham) Chemistry and physiologic action of the antibiotic phytoactin
- 1968 Grand, Larry F.  
(Rogers) Habitat correlations, mycorrhizal synthesis, and cultural characters of some Suillus species in the Pacific Northwest
- 1968 Harvey, Alan E.  
(Shaw) Isolation, identification, purification and biological properties of the antibiotic phytoactin
- 1968 Loman, August A.  
(Rogers) The effect of some common heartwood inhabiting fungi of Pinus contorta var. latifolia on Pinosylvin, Pinosylvinmonomethyl ether, Pinobanksin and Pinocembrin
- 1968 Tarry, Jerry C.  
(Rogers) The dieback of Ceanothus species with emphasis on some of the physiological aspects of Armillaria mellea

- 1969 McDonald, GERALD I.  
(Graham) Resistance to Cronartium ribicola J. C. Fisch. ex Rabenh, in Pinus monticola Dougl. seedlings
- 1969 Waldron, HARVEY M.  
(Shaw) Biology of Polyporus volvatus PK.
- 1970 Gurusiddaiah, Sarangamat  
(Graham) Studies on Phytoactin B: Its accumulation stimulation of growth, and reaction with cell constituents
- 1972 Hudson, DAVID N.  
(Maloy) Microorganisms associated with decay in grand fir
- 1972 Williams, RALPH E.  
(Rogers) Fungi associated with blister rust cankers on Western white pine
- 1975 Daniels, BARBARA A.  
(Graham) Effects of nutrition and soil extracts on germination of Glomus mosseae spores
- 1976 Castello, JOHN D.  
(Shaw) The role of Dendroctonus pseudotsugae in the dissemination of Polyporus volvatus and Fomes pinicola
- 1977 Hoffman, JAMES T.  
(Rogers) Aspects of Cytospora species causing brown stain of coniferous woods
- 1978 Blanchette, ROBERT A.  
(Shaw) Biological decay of forest residues
- 1980 Harrington, THOMAS C.  
(Shaw) Cultural characteristics, bark beetle relationships, and dissemination of Cryptoporus volvatus
- 1981 MacKenzie, MARTIN  
(Shaw) The role of nitrogen-fixing bacteria in wood decay
- 1981 Oster, JEFFREY J.  
(Shaw) Decay in Pseudotsuga menziesii (Mirb.) Franco subsequent to attack by Dendroctonus pseudotsugae Hopkins
- 1982 Pettey, THOMAS MICHAEL  
(Shaw) Physiological characteristics, biology and dissemination of Cryptoporus volvatus
- 1985 Stahl, SHERRILL A.  
(Rogers) Investigations of conifer foliage diseases caused by Hendersonia pinicola and Mycosphaerella pini (stat. anam. Dothistroma septospora)

#### MYCOLOGY

- 1943 Coe, DONALD M.  
(Heald) A comparative study of Sclerotinia sclerotiorum (Lib.) Masee and Sclerotinia trifoliorum, Erik.

1948	Nyland, George D. (Fischer)	Studies on some unusual heterobasidmycetes from Washington State
1950	Thomas, Antoinette Gage (Shaw)	Cytological studies of <u>Apiosporina collinsii</u> (Schw.) V. Hornel
1953	Becker, Joseph G. (Shaw)	A study of fungi in domestic sewage treatment plants
1959	Reyes, Andres Arenas (Hendrix)	Some studies on <u>Verticillium albo-atrum</u> Reinke and Berthold isolates of local origin
1966	Durrieu, Guy (Fischer)	Etude ecologique de Quelques groupes de champignons parasites de plantes spontanees dan les pyrenees (Peronosporales, Erysiphacees, Ustilaginales, Uredinales)
1970	Jong, Shung-Chang (Rogers)	Cultural and developmental studies of conidial stages of <u>Hypoxyylon</u> and allied genera
1972	Watts, Dorothy K. (Rogers)	Aspects of the biology of <u>Poronia</u>
1973	Goree, Harold K. (Shaw)	The hysteriaceae and lophiaceae of Western United States
1974	Christen, Alice A. (Rogers)	<u>Collembola</u> and their fungal associates
1974	Stiers, David L. (Rogers)	Ultrastructure of asci and ascospores in <u>Hypoxyylon</u> , <u>Neurospora</u> , <u>Poronia</u> , and <u>Rosellinia</u>
1975	Kenerley, Charles M. (Rogers)	Aspects of the biology of <u>Hypoxyylon serpens</u>
1977	Davis, Joyce Lynn S. (Maloy)	The ultrastructure and development of <u>Sclerotium cepivorum</u> Berk.
1977	Malmgren, Marlene M. (Rogers)	Studies of <u>Hypoxyylon serpens</u> in culture
1977	Spinner, George A. (Rogers)	Cultural study of <u>Podospora pleiospora</u>
1980	Jensen, Jon D. (Rogers)	The life history of <u>Hypoxyylon serpens</u> in culture
1982	Glawe, Dean (Rogers)	Studies on diatrypaceous fungi
1984	Jensen, Jon Duane (Rogers)	Morphologic studies in the Pyrenomycetes

- 1985 Callan, Brenda E.  
(Rogers) Studies of the anamorphs of selected species of Xylaria, Biscogniauxia, and Rosellinia

#### PHYSIOLOGY

- 1959 Strobel, James W.  
(Graham) Physiologically induced morphologic and pathogenic variation of some Glomerella cingulata isolates
- 1971 Hess, Samuel L.  
(Hadwiger) Biosynthesis of phaseollin and induction of its biosynthetic pathway in Phaseolus vulgaris
- 1971 Christenson, John A.  
(Hadwiger) Effect of Pisatin on clones of Fusarium solani pathogenic and non-pathogenic to peas
- 1972 Manandhar, Jaju Bhai  
(Bruehl) Water relations of Fusarium and Verticillium
- 1972 von Broembsen, Sharon  
(Hadwiger) Early protein synthesis in gene-for-gene interactions of the flax-Melampsora lini system
- 1974 Green, Norman E.  
(Graham) Erysiphe graminis f. sp. tritici: Studies on nutritional requirements for axenic culture and on the physiology of the host-parasite interaction
- 1975 Daniels, Donna Lucille  
(Hadwiger) Fusarium solani culture filtrates eliciting pisatin production in pea tissue
- 1975 Teasdale, John R. (†) The role of spermine and spermidine in the resistance of pea pod tissue to Fusarium solani
- 1978 Dobson, Robin L.  
(Hadwiger) Specific nuclear proteins from Fusarium solani inducing pisatin in Pisum sativum
- 1980 Loschke, David C.  
(Hadwiger) Studies on the synthesis of phenylalanine ammonia-lyase in peas
- 1982 Magnuson, J. A.  
(Easton) A selective medium for Corynebacterium michiganense pv. sepedonicum
- 1984 Kendra, David F.  
(Hadwiger) Characterization of the smallest chitosan oligomer that is maximally antifungal to Fusarium solani and elicits pisatin formation in Pisum sativum
- 1986 Kendra, David F.  
(Hadwiger) Biological effects of chitosan on Fusarium solani and Pisum sativum
- 1986 Woloshuck, Charles P.  
(Hadwiger) Cutinase of Fusarium solani f. sp. pisi: Mechanism of induction and relatedness to other Fusarium species

## VEGETABLES

- 1929 Burnett, Grover  
(Jones) A bibliographic review of non-parasitic diseases of truck and vegetable crops

## ONIONS

- 1924 Boyle, Lytton Wesley  
(Heald) A Fusarium rot in onions
- 1963 Tachibana, Hideo  
(Holton) Life cycle phenomena and host-parasite-interaction in Urocystis colchici (Schlect.) Rabenh. (U. cepalae Frost)
- 1967 Anderson, John Frederick  
(Duran) Sexuality of Urocystis colchici teliospores in naturally infected and artifically inoculated onions
- 1967 Howell, Charles R.  
(Duran) Biochemical changes in onion seedlings associated with resistance to the onion smut fungus Urocystis colchici

## TOMATO

- 1931 Burnett, Grover  
(Jones) The interrelation of certain virus diseases of tomato, potato and tobacco
- 1982 Hassan, Sher  
(Thomas) Host range, symptomatology and transmission characteristics of tomato yellow top virus
- 1984 Hassan, Sher  
(Thomas) Etiology of tomato yellow top disease and resistance to it in Lycopersicon peruvianum (L.) Mill.

## ASPARAGUS

- 1942 Cohen, Sylvan I.  
(Heald) A wilt and root rot of Asparagus officinales L. var. altilis L. caused by Fusarium oxysporum (Schl.) Snyd. and Han., Form asparagi
- 1976 Grove, Melvin D.  
(Maloy) Pathogenicity of Fusarium species associated with asparagus decline in Washington
- 1978 Inglis, Debra A.  
(Maloy) The association of two Fusarium species with asparagus seed
- 1978 Uyeda, Ichiro  
(Mink) Identification, characterization, and incidence of viruses isolated from asparagus



## POTATO

- 1950 Rich, Avery E.  
(Locke) Phloem necrosis of Irish potato tubers in Washington
- 1959 Hamid, Abdul  
(Locke) The effect of temperature on virus survival and multiplication in potato tissue
- 1960 Gordon, Clarence C.  
(Shaw) The life cycle and parasitism of an undescribed species of the Meliolaceae attacking Solanum spp.
- 1961 Clark, Raymond L.  
(Locke) A study of the host range of the potato leafroll virus in the tuberous Solanum species
- 1966 Horvath, J.  
(Locke) Methods for the differentiation of potato viruses and the properties of the strains of potato virus Y/Marmor upsilon Holmes
- 1968 Harder, Donald E.  
(Kirkpatrick) The effect of polyploidy on plant virus replication and control
- 1968 McMillan, Robert T. Jr.  
(Locke) A study of auxin and chlorogenic acid in leaf-roll resistant and susceptible potato varieties
- 1970 Cromarty, Robert W.  
(Easton) Bacterial soft rot of potatoes
- 1979 Archuleta, Jose G.  
(Easton) The causal nature of deep pitted scab of potatoes in Washington
- 1980 Wadi, Jawad A.  
(Easton) Cause and control of early dying of potato
- 1982 Wadi, Jawad A.  
(Easton) Biological control of Verticillium dahliae on potato abstract
- 1984 Xu, Guo-Wei  
(Gross) Selection of fluorescent Pseudomonas strains antagonistic to Erwinia carotovora and their effect on colonization and infection of solanum tuberosum L. by Erwinia carotovora
- 1985 Cody, Yvonne Susan  
(Gross) Pseudomonas syringae pv. syringae: Mechanisms of iron acquisition, the effect of iron on siderophore production, and biological control by fluorescent pseudomonads
- 1985 Leben, Shelley D.  
(Easton) Effects of Pseudomonas fluorescens on potato plant growth and control of Verticillium dahliae

## PEAS

- 1932 Kadow, Kenneth John  
(Jones) Studies on Fusarium wilt of peas with special reference to dissemination
- 1934 Anderson, Earl J.  
(Jones) Investigations of the powdery mildew of peas
- 1935 Campbell, Leo  
(Heald) The downy mildew of peas caused by Peronospora pisi
- 1939 Johnson, Folke  
(Jones) Viruses of the garden pea (Pisum sativum L.) in Washington
- 1946 Schuster, Max L.  
(Fischer) The etiology and control of damping-off and root stem rots of peas in Washington
- 1971 Roberts, Donald D.  
(Kraft) The effect of cultivar, inoculum density, soil temperature, and root rotting pathogens on the development of pea wilt caused by Fusarium oxysporum f. pisi race 5.
- 1973 Knesek, John E.  
(Mink) Seed transmission, host range, purification and properties of pea seed-borne mosaic virus
- 1982 McCoy, Randy Jay  
(Kraft) Comparison of screening techniques and sources of inoculum for use in evaluating peas (Pisum sativum L.) for resistance to Rhizoctonia solani Kuhn
- 1983 Hwang, Sheau-Fang  
(Cook) Mechanisms of suppression of chlamydospore germination of Fusarium oxysporum f. sp. pisi in soils

## LENTILS

- 1975 Lin, Yi-Sheng  
(Cook) Fusarium root rot of lentils in the Pacific Northwest

## CHICKPEA

- 1983 Hannan, Richard Martin  
(Kaiser) Factors affecting and sources of resistance in chickpea to pre-emergence damping off Pythium ultimum

## DRY BEANS

- 1955 Burke, Douglas W.  
(Fischer) Soil microflora relationships in the development of bean root rot in Columbia Basin soils

- 1962 Echandi, Ronald J.  
(Locke) A study of the biochemical nature of resistance of Phaseolus vulgaris and Phaseolus coccineus to Fusarium solani f. phaseoli
- 1983 Wang, Wei-Young  
(Mink) Serology of bean common mosaic virus strains
- 1985 Wang, Wei-Young (Simon)  
(Mink) Production and characterization of hybridoma cell lines and a broad spectrum monoclonal antibody against bean common mosaic virus

#### ALFALFA

- 1942 Menzies, James D.  
(Harrar) Studies on the Witches' Broom disease of alfalfa in Washington

#### CABBAGE

- 1979 Babadoost-Kondri, Mohammad  
(Gabrielson) Alternaria species pathogenic on Brassica seed crops and their control in Western Washington
- 1980 Bonman, John M.  
(Gabrielson) Biology of Phoma lingam on cabbage
- 1982 Bassey, Ekanem Odo  
(Gabrielson) Factors affecting seed assays and seedling disease of cabbage (Brassica oleracea L.) caused by Alternaria brassicola
- 1982 Dobson, Robin L.  
(Gabrielson) The biology and control of Plasmodiophora brassicae
- 1984 Schultz, Tom R.  
(Gabrielson) Biology and control of Xanthomonas campestris pathovar campestris in western Washington

#### GRAPES

- 1958 Bourbals, Denis  
(Fischer) Contribution a l'etude des causes de la resistance des Vitacees au mildiou de la vigne (Plasmopara viticola (B. et C.) Berl. et de T.) et de leur mode de transmission hereditaire
- 1975 Uyeda, Ichiro  
(Mink) Purification and properties of grapevine fanleaf virus
- 1978 Glawe, Dean A.  
(Skotland) The dying arm disease of grapevines

## SUGAR BEETS

- 1952 Yerkes, William D. Jr.  
(Shaw) Downy mildew of spinach and related plants
- 1970 Howell, William E.  
(Mink) Epidemiology of sugar beet viruses in Central Washington
- 1972 Jafri, Ali Majid  
(Mink) Interaction of sugar beet Western yellows virus, vector, and hosts

## MINT

- 1971 Weimer, Robert J.  
(Skotland) Life history and control of Puccinia menthae on native spearmint, Mentha spicata

## HOPS

- 1971 Probasco, Eugene G.  
(Skotland) Purification and characterization of a rod-shaped virus found in hops, Humulus lupulus L.

## BERRIES

- 1925 Spiegelberg, Carl Henry  
(Heald) Fungi associated with a canker disease of the Evergreen Blackberry (Rubus laciniatus Wild.)
- 1925 Sprague, Roderick  
(Heald) A witches' broom of the service berry
- 1953 Goheen, Austin Clement  
(Fischer) Fungus diseases of the cultivated blueberry

## APPLE AND PEAR FRUIT ROTS

- 1922 Bach, Walter J.  
(Heald) Rot of apples
- 1927 Newton, George Albert  
(Heald) Some fungi of the Stemphiliium type and their relation to apple rots
- 1929 Huber, Glenn Anthony  
(Heald) The fungus flora of the normal apple; the aspergilli and their relation to decay in apples
- 1930 Ruehle, George D. (†) Fungi which cause decay of apples in storage
- 1931 Huber, Glenn A.  
(Heald) The fungous flora of the normal apple and the sources of contamination

- |      |                                      |  |
|------|--------------------------------------|--|
| 1934 | Baker, Kenneth F.<br>(Heald)         | A review of the literature concerning <u>Penicillium Expansum</u> Link with special emphasis on its relation to fruit decays |
| 1934 | Baker, Kenneth F.<br>(Heald)         | Investigations of the etiology and control of the blue-mold decay of apples caused by <u>Pennicillium expansum</u> Link      |
| 1935 | Schnellhardt, Otto F.<br>(Heald)     | Investigations on the gray mold of apples caused by <u>Botrytis</u> species of the cinerea type                              |
| 1939 | Wellman, Richard Harrison<br>(Heald) | The control of blue-mold decay of apples   |
| 1940 | English, William H.<br>(Heald)       | The fungi which cause decay of pears in Washington   |
| 1984 | Sitton, Jerry Webb<br>(Pierson)      | Interaction and control of alternaria stem decay and blue mold in d'Anjou pears  |

#### FRUIT TREES

- |      |                                   |   |
|------|-----------------------------------|---|
| 1923 | Parker, Charles S.<br>(Heald)     | Coryneum blight of stone fruits   |
| 1937 | Reeves, Enoch Lloyd<br>(Heald)    | Mottle-leaf of cherries   |
| 1952 | Frederick, Lafayette<br>(Locke)   | Growth variations in bacteria-free crown-gall tissue  |
| 1954 | Graham, Shirl O.<br>(Blodgett)    | Some physiological and environmental factors necessary in vegetative propagation of presumably virus-free <u>Prunus</u> understocks |
| 1955 | Kosuge, Tsune<br>(Locke)          | Studies on factors influencing organogenesis in bacteria-free crown gall tissue   |
| 1967 | Saksena, Krishna N.<br>(Mink)     | Purification, properties and serology of an apple latent virus  |
| 1972 | Ballard, Gary K.<br>(Mink)        | A comparison of strains of apple chlorotic leaf-spot virus and transmission studies from herbaceous to woody plants                 |
| 1977 | Moulton, Gary A.<br>(Schafer)     | The response of apple seedlings with mycorrhizal fungi to arsenic contaminated soils  |
| 1979 | Al-Musa, Abdullah M. F.<br>(Mink) | Studies on sweet cherry stem pitting disease; identification and characterization of viruses isolated from under sweet cherry trees |

- 1979 Dilley, Marc A.  
(Covey) A preliminary study of wood decay in apple trees in Central Washington
- 1980 Janisiewicz, Wojciech J.  
(Covey) Changes in susceptibility of apple rootstocks to Phytophthora cactorum in relation to vegetative bud development and some associated differences in organic chemical constituents of bark tissue
- 1983 Janisiewicz, Wojciech  
(Covey) The relationship of vegetative bud development and some chemical constituents of apple rootstocks to Phytophthora cactorum susceptibility and a study on biological control of the fungus
- 1987 Regner, Kurt M.  
(Johnson) Etiology of canker and dieback diseases of sweet cherry (Prunus avium L.) in Washington

#### ORNAMENTALS

- 1941 Boothroyd, Carl Wm.  
(Heald) Fungi associated with silver twig of trees and shrubs
- 1949 MacLean, Neil A.  
(Sprague) Botrytis diseases of ornamental plants
- 1957 Dade, Caroline Esser  
(Graham) The effect of light on growth and pathogenicity of a botrytis cinerea isolate from African violet
- 1958 Apt, Walter J.  
(Hendrix) Studies of the Fusarium disease of the bulbous ornamental crops
- 1964 Filer, Theodore Henry Jr.  
(Hendrix) Parasitic and pathogenic aspects of Marasmius oreades, a fairy ring fungus
- 1983 Michaels, Ellen Marie  
(Chastagner) Swiss needle cast of Douglas-fir Christmas trees: Impact in the Pacific Northwest and factors influencing inoculum availability

#### NEMATODES

- 1967 Milne, Dwight Read Jr.  
(Haglund) Phoretic relationships of rhabditoid nematodes with dipterous insects
- 1987 Charchar, J. M.  
(Santo) Effect of temperature on the life cycle of Meloidogyne chitwoodi races 1 and 2 and M. hapla on Russet Burbank potato

## **Support Personnel**

### *Technical Aides*

Directors of the experiment station consider technical aides a good investment, increasing the productivity of scientists to whom they are assigned. Ideally each scientist, state-wide, should have a technical aide on regular funds. In practise, some had two and some had none, a cause of dissention. When unable to have an aide for each scientist, some were shared. Sharing was disastrous, especially when field work away from the home station was significant. The aide, while away, is unavailable to someone for days at a time, and this is serious at planting or harvest times. In addition, aides usually have difficulty balancing their efforts between two or more bosses.

Technical aide positions in the state system pay better than in the federal system, and most recently hired technical aides have MS degrees. This is desirable because the additional training equips them to appreciate the need for experimental design, replicates, statistics, standard procedures, and especially to record data and techniques used with adequate detail and clarity. In this department most work is in the laboratory, much with sophisticated and expensive equipment, another reason for added training and maturity.

In my opinion technical aides should be under the same work rules as faculty. When off-station experiments are an important part of a project, an 8-hour day becomes a real problem. If you drive 6 hours to the plot, work 3 hours, and then take an additional hour to get to a motel, that is a 10-hour day. You repeat this the next day, another 10-hour day, = 4 excess hours. The technical aide is entitled to 4 hours of compensatory time for the travel. State laws, under strong labor union influence, dictate such policies. The cost of off-station work is increased, an incentive to work only close to the home base.

Technical aides for the most part are career workers. Stagnation, loss of interest, failure to grow in the job is a real danger. They should, when work schedules permit, be encouraged to attend seminars and to learn new techniques. The aide needs stimulation to keep productive and happy over a full career.

### *Laboratory Staff*

#### *Classified Laboratory Staff, State System, Pullman*

1952-1967	John Y. Woo. Senior Exptl. Aide. Holton, Graham, Duran, Locke.
1962-1967	Theresa Ann Meinhart. Lab. Aide. Locke.
1963-1968	James E. Gray. Expt. Aide. Hendrix, Bruehl.
1964	Arthur C. Washington. Expt. Aide. Hendrix.
1964-1965	John Burnett. Expt. Aide. Bruehl.
1964-1967	Lloyd Slusser. Senior Expt. Aide. Graham, Bruehl.
1965-1966	Barbara J. Collins. Lab. Tech. Shaw.
1966-1967	Susan Dawson. Lab. Tech. Hadwiger.
1966-1982	Myra E. Smart. Lab Tech. II. Hendrix, Hadwiger, Shaw, Gross, Wyatt.
1966-1975	Robert Cromarty. Res. Tech. III. Easton at Prosser, Duran.
1967-1971	Bert A. Freeman. Expt. Aide. Locke, Duran, Hendrix, Bruehl.
1969-1976	Norman Green. Res. Tech. Graham.
1970-1971	Jane Meyers. Senior Lab. Tech. Cook.
1970-1971	Ke-Cheng Hsiao. Expt. Aide. Hendrix.
1971-1976	Bruce V. Hewitt. Expt. Aide. Line.
1971-1985	Rollin Machtmes. Ag. Res. Tech. III. Bruehl, Murray.
1972-1974	Marianne Morgan. Greenhouse Attendant. Hendrix.
1974-	Michael Adams. Res. Tech. III. Rogers.
1974-1977	Lewis E. Stansbury. Expt. Aide. Shaw.
1975-1976	Janice E. Eddyvean. Lab. Tech. II. Hadwiger.
1975-	Peter Gray. Res. Tech. III. Duran, Gross.
1977-1979	Howard K. Morgan. Ag. Res. Tech. II. Hendrix.

1977-1979	Everett J. Nichols. Microbiologist I. Hadwiger.
1978-1981	Hamidah B. Ghazali. Lab. Tech. I. Line.
1979-1980	Jean M. Beckman. Microbiologist I. Hadwiger.
1979-1981	Teresa Wilkinson. Res. Tech. II. Wyatt.
1979-1983	Gary Radamaker. Res. Tech. I. Hendrix, Gross.
1980-1981	Christy Roberts. Microbiologist I. Hadwiger.
1982-1983	Catherine Knoeber. Ag. Res. Tech. I. Wyatt.
1983-1984	Ann M. Schueren. Res. Tech. I. Duran.
1983-1984	Denise Shook. Ag. Res. Tech. I. Wyatt.
1984-1985	Myrna C. Graham. Ag. Res. Tech. I. Cook.
1984-1986	Linda Green. Ag. Res. Tech. I. Wyatt.
1984-1985	Michael Mills. Res. Tech. III. Gross.
1985-	Cheryl Campbell. Ag. Res. Tech. II. Murray.
1985-1988	Steven Victory. Ag. Res. Tech. I. Hadwiger.
1986-	Keri Druffel. Res. Tech. I. Wyatt.
1986-1987	Heather MacCrimble-Zimmerman. Res. Tech. II. Gross.

#### *Office Staff*

The office staff plays a critical role in the smooth operation of the department, keeping financial records, typing teaching materials, manuscripts, correspondence, and numerous daily housekeeping chores as well as filing and maintaining records. A stable, competent staff is critical to the chairman, allowing concentration on personnel, policy, and administrative matters with few worries about day-to-day operations.

The Staff Personnel Office of the University and its policies affect the quality and stability of the staff. Starting in 1965 and continuing until about 1979 turnover was excessive. The policy was to hire student wives when possible. Many office personnel served for no more than 12 months. It is hard to imagine the stress this added in trying to run an efficient office, and the time spent interviewing candidates. In addition, for some years the testing of typing skills was so inadequate that some secretaries could not type and had to be released. The personnel office was functioning more as a social action program than in trying to improve efficiency. The policy of favoring student wives to the virtual exclusion of other candidates has stopped.

Comments by C. G. Shaw. One of the worst aspects of personnel management during this period was that numerous department chairmen gave high recommendations to incompetent secretaries and even recommended them for promotion just to get rid of them. I complained of this to some department chairmen and the Office of Staff Personnel and made no headway. The university refused to give the Agriculture College Class IV or Administrative Assistant rankings and this led to the loss of the best secretaries. Jim Engibous, Chairman of Agronomy, finally broke the system and since then conditions have been good.

I remember Marge Hoisington (1955-1965) with gratitude. She was not an 8-5 worker. She spent a Sunday afternoon with me showing me how to handle the department budget during my first year as chairman. It went zip, zip, zip, and I understood her!

Bruehl from here on.

Some instability in personnel results when a person must move to a different unit within the university to advance in rank. Offices are limited in the number of upper level secretarial positions they can have by state regulations. This forces some movement of staff from unit to unit, but it results in a tolerable level of instability, unlike the period when student wives made up the bulk of the employees. It is amazing, in retrospect, that the faculty tolerated the inefficiency and chaos resulting from that practice.



For many years the department maintained a library, and from 1954 to 1974 the university libraries helped by providing part-time salaries for library help. This stopped when the departmental library was incorporated into the George Fischer Agricultural Library in Johnson Hall.

Some turmoil occurred during periods of staff reductions, about 1980-1981, because of financial stress throughout the University. When reductions occurred, those with the least seniority were discharged. Those with more seniority replaced them, a practice called "bumping", and chain reactions were common.

The office was a model of efficiency under Margery Hoisington. She hired students or student wives on time-slip to help with typing as needed. In her day there were fewer grants and bookkeeping was relatively simple. In recent years the office staff has been unusually skilled and efficient, and the office is well equipped with modern secretarial machines. We have no complaints.

#### *Office Staff, Pullman*

Head secretaries are underlined. Personnel for less than one year are indicated by an asterisk.

- 1950 Leona Wilson. 1950-52  
Ida Dietz. Librarian. 1950-53.
- 1952 Margaret Fluharty. 1952-53.
- 1953 Ida Dietz. 1953-55.
- 1954 Lavonne Sears Stern. Librarian. 1954-58.
- 1955 Margery Hoisington. Secretary. 2/55-4/65.
- 1961 Marion A. Costner. Library Attendant. 7/61-6/63.
- 1962 Jackie Sue (Owen) Weitman. Stenographer. 6/62-7/65.
- 1963 Donna Larson. Librarian, Stenographer. 6/63-1/68.
- 1965 Yvonne Beasley. Secretary IV. 6/65-12/71.  
Evelyn Bergman. Secretary. 4/65-6/66.  
\* Bernice Hopper. Library Attendant. 6/65-2/66.  
\* Carole Selter. Clerk Stenographer. 8/65-6/66.  
\* Margaret Varner. Clerk Stenographer. 9/65-9/66.
- 1966 \* Judith Dahlin. Library Attendant. 1/66-1/66.  
\* Margaret Grohs. Library Attendant. 1/66-6/66.  
Beverly Heebner. Library Attendant. 7/66-8/68.  
\* Solange Henderson. Clerk Stenographer. 1/66-1/67.
- 1967 Karla Hanks. Secretarial Stenographer. 2/67-5/70.  
\* Laurel Henning. Secretarial Stenographer. 12/67-7/68.
- 1968 \* Christin Hopkins. Library Assistant III. 8/68-8/69.  
\* Linda Scott. Secretarial Stenographer. 9/68-4/69.
- 1969 \* Janice Ellingson. Library Assistant III. 9/69-7/70.  
\* Susan Ponder. Clerk. 9/69-7/70.
- 1970 \* Diane Burke. Secretary. 4/70-8/70.  
\* Shirley Johnston. Clerk. 9/70-10/70.  
\* Judy Lane. Clerk. 9/70-3/71.  
\* Susan Mayer. Secretarial Stenographer. 10/70-1/71.  
\* Glauca Sousa. Library Assistant III. 7/70-5/71.
- 1971 June Benson. Secretary IV. 7/71-10/74.  
\* Geraldine Knowles. Secretarial Stenographer. 12/71-2/72.  
Judy Walters. Secretary IV. 12/71-4/73.  
Leslie A. Vosburgh. Secretary IV. 2/72-5/74.
- 1973 \* Patricia Scott. Library Assistant II. 5/73-5/74.
- 1974 Fredda Buxton. Office Assistant III, Typing. 11/74-2/77.  
\* Leonore Garnett. Library Assistant II. 6/74-6/75.  
Mildred Lytle. Secretary IV. 10/74-9/79.

1975 Virginia Murtuza. Secretary III. 5/74-6/75.  
 Margaret Faler. Secretary III. 6/75-9/76.  
 Jean Booras. Office Assistant III, Typing. 12/75-10/76.  
 1976 \* Susan Montague. Secretary II. 9/76-9/77.  
 1977 \* Lynne Dunagan. Secretary II. 9/77-1/78.  
 \* Heidi Taylor. Secretary II. 3/77-1/78.  
 Nancy Torbek. Office Assistant III, Typing. 1/77-9/78.  
 1978 Lois Bender. Secretary, Accounting Assistant II. 4/78-2/81.  
 \* Lee Meier. Secretary II. 8/78-5/79.  
 \* Lauren Stinson. Secretary II. 2/78-8/78.  
 \* Gail Waggoner. Office Assistant III, Typing. 10/78-5/79.  
 1979 Marta Arenaz. Secretary IV. 9/79-7/81.  
 Barbara LaBudde-Arausa. Typing III. 6/79-9/80.  
 Janet Hallauer. Secretary III. 6/79-11/80?  
 \* Marilee Wilder. Secretary II. 5/79-6/79.  
 1980 \* Karen Neller-moe. Secretary II. 12/80-12/80.  
 1981 Phyllis Cheevers. Administrative Assistant. 7/81-9/85.  
 Alice Coil. Accounting Assistant II. 6/81-8/84.  
 \* Margaret Johnson. Typing III. 4/81-3/82.  
 Joyce A. Mickelson. Office Assistant III, Typing. 3/81-5/84.  
 \* Carol Valentine. Accounting Assistant II. 4/81-6/81.  
 1982 Ruby Latham. Secretary III. 9/82-9/88  
 \* Sally Tompkins. Office Assistant III, Typing. 3/82-4/82.  
 1984 \* Linda Wilson. Office Assistant III, Typing. 8/84-8/85.  
 1985 Susan Irish. Administrative Assistant. 10/85-8/87.  
 Jane Lawford. Program Assistant I. 9/85-  
 1987 Pamela Lindquist. Administrative Assistant. 8/87-  
 1988 Jackie Mraz. Secretary III. 9/88-  
 1989 No changes.

## BRANCH STATIONS

### Western Washington Research and Extension Center, Puyallup

In some respects the Puyallup station is the most interesting of the branch stations. When the main station at Pullman was authorized, the state legislature required the establishment of at least two other stations, one of which must be west of the Cascade Mountains. The legislature expected the Pullman station to do most of the basic research, and the western Washington station was to stress applied research, with emphasis in plant pathology on diseases favored by high humidity. At the outset a problem arose. Could federal funds allotted for research at land-grant colleges be used at a branch experiment station removed from the teaching campus? To avoid possible legal problems, the budget for the WWREC at Puyallup was a special item in the state budget. This practise continued up to the mid 1940's, after which state funds for Puyallup were undesignated within the total state experiment station budget. When the Puyallup station was united officially with the state system, all research was under the director at Pullman.

Selection of the site near Puyallup was determined by a gift of 40 acres of bench land and the offer of lease at reasonable terms of 20 acres of Puyallup River Valley land by the D. M. and Charles Ross families (5th Ann. Rpt., 1895). The 20 acres had some hops and fruit trees on it. The bench land was mostly cut-over timber land that required removal of stumps. The state legislature provided \$2,500 and the Tacoma Board of Trade gave \$500 to purchase the bottom land and start the station, to be called

"The Ross Station." F. A. Huntley of Rock Ford, Colorado, was the first station superintendent. He was hired July 1, 1894. The station was closed twice in its infancy (1897-1899 and 1903-1907) for lack of funds, but after that it grew steadily in staff, buildings, equipment, land, and financing, until today it is a major facility. Most of the plot land at Puyallup is level alluvium. If adequately drained and uniform in texture and drainage it contributes to precision in field experiments. Urban sprawl increasingly restricts the tilled acreage in the valley, but even small patches of berries, bulbs, or vegetable seed crops produce significant income and local employment. The sociological benefit when young local people are employed to pick berries or other things during summer is great. Dairying is the biggest agricultural enterprise in Western Washington, but research on pasture and forage plants by pathologists has been minimal.

#### *Laboratory and Office Facilities*

Plant Pathology moved into Kalkus Hall in 1969. Floor space was only equal to that in the old vacated quarters but it was better organized and more useful than the old space. In May 1984 a fire damaged the laboratories and those of Entomology on the floor above. Fortunately, few records or data were lost. Books and documents damaged by water were frozen to prevent further deterioration and shipped to California where they were vacuum-dried. Many books had to be rebound. During the 8-plus months of reconstruction, department members operated out of the old facilities. Since the move to Kalkus Hall in 1969, the old laboratories and offices have been used to house graduate students, for storage, and to conduct work that involved either dusty or dirty materials.

Pride can lift morale and enrich an institution. The maintenance people of the branch stations do their work with pride. A visitor from Pullman to the Puyallup station, especially when the rhododendrons are in bloom, turns green with envy. The lawn, flowers and shrubs are kept near perfection by the groundskeepers. The floors of Kalkus Hall, the home of plant pathology and associated workers, shine, always. Visitors tracking mud will be noticed. The faculty and staff respond to these high standards by doing all they can to maintain the beauty of the place. Beauty does not make for better science, but it surely can't hurt. In addition, Mt. Rainier is visible on clear days.

#### *Library Facilities*

Because of the long history of this Center, the diversity of disciplines represented, and the tradition of discarding little over the years, it has rooms filled with old journals, bulletins, etc. Considerable organization and discarding were accomplished by the former librarian. Unfortunately, much of the older material is poorly housed and library facilities at WWREC have been deteriorating for the past 15 years. Inflation increased the price of periodicals and the library budget remained constant. Subscriptions to Current Contents and a few abstracting journals, such as Review of Plant Pathology, consume most of the budget. Most scientists maintain ongoing Current Awareness searches, but these are channeled via individuals with Extension appointments because this federal service was withdrawn from state scientists several years ago. The main stream journals, such as Phytopathology, Plant Disease, and Annual Review of Phytopathology, are purchased by individual plant pathologists and not by the library.

The biggest setback to the library was the loss of a fulltime librarian in 1987. The secretary assigned part time to the library cannot provide the level of services needed. Also, our former librarian did graphic work as time permitted and those talents have not been replaced (even though we are moving in the direction of computer generated graphics).

#### *Graduate Students*

Until the mid-1970's few, if any, graduate students conducted their research with the scientists at WWREC serving as Thesis Committee Chairmen. Financial support for students was scarce and the logistics of housing and moving back and forth between Pullman and Puyallup were severe problems.

Student housing became available at Puyallup when the Superintendent's residence was converted to a guest house in 1976. Also, a small house on the Center was upgraded for use by a married student. Since 1976, several M.S. and Ph.D. degree students have completed the research portion of their degree under the direction of WWREC plant pathologists. The presence of graduate students at WWREC has been beneficial.

#### *Notes*

Contributions to Puyallup history were submitted by Chuck Gould (retired), Folke Johnson (retired), Peter Bristow, Gary Chastagner, and Arlen Davison. I prepared some aspects of studies by Leo Campbell (deceased), Wilbur Courtney (deceased), Dick Gabrielson, and Ralph Byther.

Chuck Gould had a rare dynamic quality that made him particularly effective in dealing with clientele. He was honored by the American Florists (1950), the New England Gladiolus Society (1950), and he became the first Honorary Member of the British Columbia Bulb Growers' Association (1953). He served as Secretary-Treasurer (1960-1963), Vice President (1963-1964) and President (1964-1965) of the Pacific Division of the American Phytopathological Society (APS). He was elected Fellow of APS in 1978. His A.B. degree was from Marshall University (1934) and his alma mater named him a Distinguished Alumnus in 1979. Chuck Gould and Roy Goss, extension turf specialist, accomplished much together. His record proves that applied scientists can receive recognition in plant pathology. We expected a let-down when Gould retired, but Gary Chastagner quickly earned the respect of the industries involved.

Dick Gabrielson has been honored for his efforts to provide pathogen-free vegetable seeds. Arlen Davison served with distinction in Extension, and his successor, Ralph Byther, likewise has done very well. Folke Johnson, in berry research, was made Fellow of the American Association for the Advancement of Science. His successor, Peter Bristow, in collaboration with Ralph Byther, essentially saved the blueberries from mummyberry disease, and they both have already made real contributions in several areas.

#### *Plant Pathology at Puyallup*

A U.S.D.A. scientist, Dr. A. W. Thorton, worked independently of WSU on fiber flax somewhere in western Washington (WAES Ann. Rpt., 1896). He recommended no less than a 5-year rotation to control flax wilt in the Puget Sound region. There was little or no coordination of investigations by state and federal workers at this time and there was much friction among them.

#### *David A. Brodie*

David A. Brodie, the first person to earn a BS degree in agriculture at WSU, studied late blight control at Puyallup. Late blight of potatoes was severe west of the Cascades. Brodie determined times and rates of applying Bordeaux mixture and gave instructions for its preparation (Bull. 46, 1901). This may be the first significant research in plant pathology in Washington.

#### *William H. Lawrence*

William Lawrence, the recipient of the first advanced degree conferred by WSU, the MS in Botany, worked at Puyallup 1902-1904, and 1908-1911. Lawrence did some work on aphids in hops and a variety of things, but his greatest first effort was to control apple scab (Bull. 64, 1904). Apple scab was so severe it partially defoliated the trees, reduced yield, and deformed fruit. Lawrence also published Bulletin 66 on 'black spot canker' of apples in western Washington, a disease called apple anthracnose by A. B. Cordley in Oregon in 1900. Cordley named the pathogen *Gloeosporium malicorticis*. Lawrence studied the pathogen in culture and on the host, and determined the incubation period by tree inoculations and its control.

When the station closed, Lawrence was transferred to Pullman where he continued his research and taught. When the station reopened (1908), he returned to Puyallup as station Superintendent and Plant Pathologist, the first person to be called a plant pathologist in the state. Bordeaux mixture was phytotoxic to apples and he experimented with lime-sulfur as a replacement. While in Puyallup, Mr. Lawrence's research centered on diseases of caneberries, particularly black raspberries, where he worked on anthracnose, crown gall, and Verticillium wilt. In 1910 Lawrence published Bull. 93 on ridding land of stumps with explosives, Bull. 95 on forage plants as livestock feed, Bull. 97, "Anthracnose of Blackberry and Raspberry", and Western Washington Experiment Station Special Bull. 5 on club root in the Puget Sound region. In 1911 Lawrence resigned, terminating the most concentrated career of some duration in plant pathology in Washington to that date. Bulletin 107 on a *Sclerotina* was published in 1912.

Black raspberries were subject to an unknown disease reported from several areas near Puget Sound. In 1907 growers near Puyallup and Sumner harvested only 150 crates of berries. Lawrence isolated a fungus which he named *Acrostalagmus caulivorum* sp. n. He studied this fungus in detail during a leave of absence at Cornell. He concluded the pathogen was soilborne but not sensitive to soil type. The fungus formed chlamydospores in chains in culture and it invaded xylem vessels. He named the disease bluestem of raspberries (Bull. 108, October, 1912), the first record of *Verticillium* on *Rubus* spp. His resignation in 1911 cut short his study of this disease. His illustrations convince me that the pathogen was *V. dahliae*.

The only formal Puyallup station report of the early years, which I did not find, was written by Lawrence for the 1907-1911 period, Bulletin 7 (WWES series). Reference to this report was found in the WWES report of 1928 prepared by Superintendent J. W. Kalkus.

#### H. P. Barss, 1911-1912

H. P. Barss was hired as Plant Pathologist at Puyallup in 1911, after Lawrence resigned. Barss left after one year to go to Oregon State University. H. P. Barss became President of APS in 1928.

#### David James Crowley

When cranberry growers lost 40% of their crop, they requested help. The College sent Plant Pathology senior David James Crowley (1923-1955?) to Long Beach during the summer of 1922 to survey the cranberry situation and to report back to the College. Based on his report, the legislature in 1923 appropriated \$8,000 for cranberry and blueberry investigations. Mr. Crowley, who graduated from WSC with a B.S. degree in plant pathology in 1923, was hired to head the new Experiment Station at Long Beach. He served in that capacity until his retirement in 1954-5.

One of the first major problems requiring his attention was the development of a spray program to control the fire worm. Damage from killing frosts was a serious chronic problem. The solution was to sprinkle-irrigate during frosty periods and allow the latent heat of fusion (heat released from liquid water as it turns to ice) to keep the temperature of the vines from dropping below 32°F. Preventing frost injury during flowering proved that losses occurring during bloom were mostly caused by cold temperatures and not fungal blossom blights. Sprinkling for frost protection is now widely used in all cranberry producing regions and the principle has been extended to many other crops as well. Peter Bristow believes this was the first use of sprinklers for this purpose.

Mr. Crowley initiated the annual Cranberry Field Day and authored the first Cranberry Production Guide for Washington growers. Most of his research had to do with culture of cranberries but he experimented with fungicides, published some articles on cranberry diseases, and was at times classified as a plant pathologist.

The Long Beach cranberry unit is considered part of the Puyallup contingent.

### George A. Newton

George Newton went to Puyallup immediately upon receiving his M.S. degree in Plant Pathology at WSU in 1927. He worked at least 3 years there. Newton, in cooperation with H. D. Locklin, Horticulturist, studied the effect of the hot water treatment (for control of nematodes in narcissus) on the vigor of the bulbs after treatment (WWES Bull. 13, new series, 1929). The Western Washington Experiment Station had its own series of bulletins and circulars for many years. Personnel at Puyallup in 1929 included Superintendent J. W. Kalkus, Veterinarian, two other veterinarians, an agronomist, two horticulturists, a poultry scientist, an entomologist, and a plant pathologist (George A. Newton).

The 1929 and 1930 annual reports make it clear that Newton seriously studied diseases of head lettuce, including grey mold (*Botrytis cinerea*), downy mildew (*Bremia lactucae*), anthracnose (*Marsonina panattoniana*), bottom rot (*Corticium vagum*), damping-off, slime (bacterial soft rot?) and tip burn (bright sun) (Bull. 19, WWES series, 1931). *Sclerotinia sclerotiorum* could be reduced by sanitation and rotation (1930 annual report). The 1930 annual report records aster yellows (Fusarium wilt), pea root rot (*Ascochyta pinodella*), and down mildew on Early Cluster hops near Puyallup. Fuggles hops were more resistant. Downy mildew drove hops from western Washington to the drier Yakima Valley. Newton found that hot water plus mercuric chloride was more effective against basal bulb rot of narcissus than hot water alone, he reported a severe outbreak of bacterial blight on lilac, and bacterial canker on tomato.

S. M. Zeller, extension pathologist in Oregon, reported in 1925 that bluestem of black raspberries was devastating on land previously in potatoes but not on raspberries in adjacent soil. He isolated *Verticillium albo-atrum* (= *dahliae*) from 49 of 51 diseased plants. George Newton (1929 annual report) confirmed Zeller's observation and cautioned growers not to plant black raspberries on potato or tomato land. Newton observed that bluestem was really a minor problem in general, and that viruses, streak and red raspberry mosaic, were the main cause of failure in black raspberries, not bluestem (later called Verticillium wilt of raspberry). He began to cooperate with Leon Jones at Pullman on viruses of raspberries but I found no further record of Newton's activities.

Formal comprehensive experiment station reports as done in the early years are invaluable to historians. Many workers publish little, yet annual reports in an available form inform us as to the problems they addressed at that time. Much of what I learned of Newton came from annual reports. In my opinion all experiment stations should be required to publish annual reports listing briefly the activities of every scientist in every department. Present public records tend to emphasize the stars and ignore the peasants.

### Wilbur D. Courtney

Wilbur Courtney (USDA), 1933-1963, the first full-time nematologist in Washington, spent most of his efforts on the bulb and stem nematode, *Ditylenchus dipsaci*. In 1936 he reported that *D. dipsaci* destroyed strawberry plants following red clover in the Puget Sound area. In 1950, he and Chuck Gould improved the treatment of iris bulbs with hot water by including a small amount of formalin in the water. In 1952 Courtney studied the nematode on teasel. In 1962 Courtney published Bulletin 640 of the WAES, "Stem Nematode of Red Clover in the Pacific Northwest." Nematodes from red clover were pathogenic to alsike clover, phacelia, and strawberry. Italian rye grass and vetch 'carried' the nematode but were not damaged by it. After red clover was grown for seed, heavy infestation persisted in the field for six years because of volunteer clover plants arising from hard seed that germinated occasionally during the cultivation of nonhosts.

The nematode resists desiccation, and is widely disseminated by hay. It migrates up the growing clover plant, becoming concentrated in the stem tips and upper leaves and flowers. Courtney found 167 nematodes in the bottom 2 inches, 475 in the 2-6 inch portion, 634 between 6 and 10 inches, and 4,886 in the terminal parts of six diseased red clover plants. It is also disseminated on seed. The ability of *Ditylenchus dipsaci* to survive dried on seed and in hay contributed to its rapid spread in the virgin soils of the Columbia Basin project.

The bulb and stem nematode was reported in the Yakima Valley on alfalfa in 1923, and by 1954 Courtney reported it widespread on this crop (report to Alfalfa Seed Growers, Yakima, January 27-29, 1954). Courtney summarized knowledge of this nematode for the growers and stressed means of limiting damaged and spread.

The seed gall nematode of bent grass, *Anquina agrostis*, was studied extensively. The wheat gall nematode (*Anquina tritici*) is easily controlled in wheat (an annual) by crop rotation because the galls disintegrate readily in or on moist soil, freeing the second stage larvae, after which they die if a host is not present. *Agrostis* spp. are grown as perennials and nematodes freed from galls find a continuous host. All that would be required to control the nematode would be to deprive it of a flowering host for one year. This could be done by plowing the grass under, making certain no plants survived to flower. Even when heavily pastured by sheep, some heads develop and the infestation persists. Many seed fields are level. Flooding with fresh or brackish water for 4 weeks does not destroy the nematodes. More experiments were presented, but the problem of destroying the infestation without destroying the host was not solved.

At one of our statewide biennial departmental meetings (1955) George Fischer had Courtney present a short course on nematology. Courtney called it a short, short course. This was done in recognition of our general lack of knowledge of nematodes and diseases they cause.

#### Glenn A. Huber

Glenn Huber, 1934-1944, received his Ph.D. in Plant Pathology at WSU in 1931 and served as Instructor until 1934 when he went to Puyallup as Plant Pathologist. Huber worked extensively with fungicides on ornamentals. He collaborated with C. D. Schartz, Horticulturist, in efforts to improve red raspberries by breeding. He collaborated with Karl Baur, Plant Pathologist and Soil Scientist, on boron deficiency in alfalfa.

Brown rot was epidemic in Clark County in 1936, destroying 90% of the sweet cherries, 60% of the Italian prunes, and 40% of the sour cherries. Huber and Baur (1941) reported extensive efforts to destroy mummies on the ground to minimize primary inoculum. Plowing them under was unreliable because in most springs the soil was too wet to work at the critical time. They found that 220 pounds of pulverized cyanamid per acre broadcast in the orchard destroyed apothecia as they formed and calcium cyanamid is most toxic and toxic for the longest time under cool wet conditions. The effect lasted the entire 2-3 wks during which apothecia formed. They also clarified the roles of *Sclerotinia laxa* and *S. fructicola* in the attacks. He and Leo Campbell (1941) tried to reduce net necrosis and ring rot of potatoes. In 1942 Leon Jones and Huber revised the control of late blight of potato, and he collaborated with Chuck Gould even after he left WSU. In 1944 he became a private vegetable seed grower and breeder and served as a consultant to the vegetable seed industry. According to his obituary he grew berries and vegetable seeds from 1955-1984, so private industry was his main career.

#### Leo Campbell

by Folke Johnson

Leo Campbell (1935-1960) was a candidate for a Ph.D. degree when he began his vegetable pathology work in western Washington. His thesis study (1934-35) was on downy mildew of garden peas. There was considerable interest in producing fresh garden peas in Thurston and Grays Harbor Counties. The main cultivar, Tall Telephone, grew prolifically under the moist conditions and reached a height of six feet or more. The crop was trellised and the growth dense. There was much loss of foliage and infected pods caused by a fungus identified by Leo as *Peronospora pisi*. Much effort was spent in finding an effective fungicide, but the best results were obtained with Bordeaux mixture. The problem was to get the sprays to wet and stick to the waxy surfaces of the plants. Penetrol oil, an adjuvant, was incorporated into the spray. It wet the plant surfaces and held material on the plants. Good control was obtained, but this practice was not accepted by the growers for several reasons. Most local producers didn't have the necessary equipment and it added to the cost of producing peas. In addition, preparation of Bordeaux is

a messy and cumbersome procedure. At that time the number of fungicide spray products was limited and the more effective materials now used were not available. The industry eventually closed down, but Leo wrote his Ph.D. thesis based on this study.

Campbell was transferred to Whatcom County where there was a sugar beet industry and a local sugar refinery. Many farmers diversified their dairy operations with sugar beets. One problem was the young seedlings "damped-off". The problem became so severe in certain fields that it became unprofitable to grow beets. Dr. Campbell, working out of the refinery laboratory, found the cause to be *Phoma betae*. Though much effort was spent on control, the disease continued and the sugar factory closed down.

While working on beets, another concern became evident. A boron deficiency was a problem in certain soils in Whatcom County. Leo demonstrated good control with boron soil application, but the beet root problem eventually contributed to the demise of the sugar beet industry in Whatcom County. (Could it have been *Aphanomyces cochlioides*?)

After these years Dr. Campbell was transferred to WWREC where he began working on strawberry diseases in the Puyallup Valley. His main effort was on red stele caused by *Phytophthora fragariae*. He developed a method of ridging the soil in rows 42" apart with ridges 12 inches high. Plants set on the ridges were mostly free of red stele. This technique worked where the ground water did not reach the main root system, and most of the plants (Northwest Cultivar) escaped severe infection. In one experiment ridged plots yielded 11.5 tons of fruit and plants not on ridges yielded 3.2 tons.

About this time a machine called the "Ironator" became available for Campbell's experiments in red stele control. This was a large, heavy machine that passed soil through a heat chamber with live steam, a continuous process as the tractor-drawn vehicle moved down the row. Strawberry plants were planted in the treated band of soil. After about two years this practice was dropped as the disease control was wanting.

Another project Dr. Campbell worked on was mint rust, *Puccinia menthae*. The industry was centered in Cowlitz County on river bottom soil. Rust infections caused severe leaf drop and this reduced the yield of distillate. In addition, the oil quality was confounded by certain weeds which would denature the oil when distilled with the mint. The problem then was to find a control of rust without affecting the oil quality and a solution to the weed problem. Weed control in the field at that time was by hand, a laborious and costly procedure. The mint fields were cultivated in early spring by a tractor-drawn contrivance pulled over the mowed fields. This practice loosened the soil without injuring the mint roots significantly and gave better mint growth. One application of dinitro amine herbicides applied in the spring when the new growth of mint was about 1-2 inches tall generally resulted in about 90% reduction of the rust. This practice increased mint "stooling", reduced the shedding of the lower leaves, and gave good control of annual broad-leaved weeds, resulting in a 30-40% increase of mint oil at a cost of less than the value of the previous weed control program. This became a general practice in mint oil production. The results were published in "Down to Earth" 10:6-7, 1955.

In the final years of these investigations V. L. Miller of the Agricultural Chemistry Department, also at WWREC, was a co-worker on the mint project.

Other studies pursued by Dr. Campbell included storage rots of squash, downy mildew of broccoli (*Peronospora parasitica*), club root of crucifers, (*Plasmodiophora brassicae*), white rot of beans (*Sclerotinia*), and gray mold (*Botrytis cinerea*) of beans.

Good control of club root was obtained by mixing PCNB into a band of soil and setting the plants in this treated band. Dr. Campbell retired in 1960 and was replaced by R. L. Gabrielson.



Karl E. Baur

Karl Baur received a BS in Plant Pathology at WSU in 1936 followed by an MS in Soils. He served at first as plant pathologist and later half-time in Plant Pathology and half-time in Soils at Puyallup. He quit the experiment station to work in the fertilizer industry. The Plant Pathology Department at Puyallup (1940) consisted of Glenn Huber, Leo Campbell, and Karl Bauer (half-time). West of the Cascades 43% of the tillable land was in hay, some of which was alfalfa. The most significant paper by Baur (Bull. 396 with G. A. Huber and L. C. Wheeting, 1941) that I found was "Boron Deficiency of Alfalfa in Western Washington". Boron deficiency was severe on 15,000 acres. Manures would not ameliorate the deficiency and applications of sodium borate (borax) were recommended.

Charles J. Gould

*by Chuck Gould*

I was hired on August 15, 1941 (1941-1977) by Dr. Glenn A. Huber (Head of Plant Pathology at WWREC) to work on the cause and control of diseases of ornamentals, particularly those on bulb crops. However, during wartime I was assigned primarily to vegetable disease research (seed treatment, pea footrot, spinach leaf spot, and ladino clover crown rot). After the war we returned intensively to bulb research and this continued to retirement in 1977. In the late 40's we also began research on turfgrass diseases. In addition, we investigated diseases of nursery stock. Most of the research was done with the cooperation of many individuals who are named in the pertinent sections. The major part of bulb disease research was in cooperation with Vernon L. Miller, Chemist-WWREC; that of forcing bulb crops with Dr. Neil Stuart (USDA-Beltsville); and with Dr. Roy Goss (turfgrass specialist WWREC) on turfgrass diseases.

During the 1940's and early 1950's we handled many extension-type activities, including disease diagnosis and recommendations for both homeowners and farmers, writing extension-type articles, giving talks, etc. In the 1940's this also included a regular radio program about every three months. About 20% of our time was spent on the above until John Dodge, Extension Horticulturist, was transferred to WSU Puyallup in 1955. He took a heavy load off of all our shoulders and much of the remaining burden was lifted when the first Extension Plant Pathologist, Dr. Arlen Davison, was appointed in 1967. We still continued to handle some inquiries in our specialties, but the above additions enabled all of us to expand our research programs appreciably.

Bulbs. The first commercial plantings of bulbs in Washington was by Captain George Gibbs on Orcas Island in 1892. He moved to Bellingham in 1899 and stimulated other farmers into growing bulbs. Favorable reports and samples resulted in USDA establishing a Bulb Research Station at Bellingham in 1908 "in order to encourage the growing of Dutch bulbs in this country on a commercial scale, and to provide American-grown bulbs of superior quality for congressional distribution." Dr. David Griffiths took charge about 1917, expanded the research program, and wrote several excellent bulletins for grower use. The acreage had been growing slowly in Whatcom, King, Pierce, and Thurston counties.

The next major impetus occurred when the USDA adopted a quarantine against foreign-produced daffodil bulbs on January 1, 1926 in an attempt to prevent introduction of certain nematodes and insect pests. Before the deadline, many Dutch bulb companies sent representatives and bulb planting stock to the U.S. and many local dairy and berry farmers diversified into bulb growing. The acreage expanded and Washington now leads all other states in Bulbous Iris, Narcissus and Tulips, with a total of 1992 acres in 1988, plus smaller plantings of lilies and gladiolus. These figures may not seem large to a wheat farmer, but they represent over 90,000,000 bulbs. One company in Skagit County is the largest grower of Iris, Narcissus and Tulips in the U.S. with over 1,300 acres.

Washington's main advantage is a favorably climate which produces more and larger bulbs with earlier and better flowers than those from other countries, including Holland. It's disadvantages are shortage of suitable land and skilled labor, plus high transportation costs to major eastern markets.

The rapid expansion of acreage during the mid-1920's brought with it an increase in pests and diseases, including the bulb and stem nematode, basal rot on daffodils, and *Botrytis* 'fire' on tulips. H. D. Locklin and G. A. Newton started research on control of the nematode in 1927 at WSU Puyallup; the USDA established an Insect Laboratory at Sumner in 1929 to work on insect pests; and also stationed T. H. Scheffer at WSU Puyallup to work on rodent control in the late 1920's. W. D. Courtney (USDA) was assigned in 1933 to WWREC to expand the research on nematode problems. Huber began working on disease control in 1934 and continued until Gould arrived in 1941. In 1955 the USDA stationed Dr. W. J. Apt at WWREC to work on root lesion nematodes, and in 1976 Dr. Robert P. Doss for research on physiology and forcing of Iris bulbs. Doss was transferred to the USDA Lab at Corvallis, OR in 1983.

We assisted three graduate students with their Ph.D. thesis research on bulb problems: Neil A. MacLean on *Botrytis* blights; Walter J. Apt on *Fusarium* basal rots; and James Van Laan on the physiology of Iris bulbs.

Although the mild, moist climate favors bulb growth it also encourages many leaf spotting fungi. Huber found that their control on Iris and Narcissus was improved by adding Penetrol (a sticking agent) to Bordeaux mixture, but it sometimes caused injury, particularly on Tulips. Later, the dithiocarbamate fungicides became available and we found these to be very effective. These were partially replaced next by the benzimidazole fungicides, but alternating application were recommended to growers to reduce the likelihood of resistant strains developing.

Although fusaria thrive best on bulbs under warm, moist conditions, such as exists in the eastern U.S., they can cause serious losses in western Washington on susceptible varieties. Mercury compounds gave good control, but sometimes caused bulb or flower injury. Hundreds of possible fungicides were tested, but none proved to be a satisfactory substitute until 1966 when the benzimidazoles became available. In testing solutions in commercial tanks, Miller and associates had found the concentration of fungicides (formaldehyde, mercury and later the benzimidazoles) was often so low that growers were probably spreading pathogens instead of controlling them. Many factors, including soil, number of dips, etc., were responsible. Miller and associates therefore devised simple testing kits which growers could use to test and maintain the proper concentration of fungicide under their own conditions.

Two other bulb rotting fungi with the potential of rendering continued production of Iris and Tulips uneconomical became increasingly important. Fortunately, we found that both Crown Rot and Black Slime could be eliminated in Iris planting stock by treatment in hot water plus formaldehyde. We also found that soil-borne infection could be prevented by applying PCNB to bulbs before planting or dusting it over bulbs in the furrow. Rotation plus the repeated use of the hot water treatment and PCNB has reduced Black Slime to a minor problem.

The Iris nematode (*Ditylenchus destructor*) became troublesome during the 1940's so we assisted Courtney in determining methods of control with hot water. We also cooperated with Apt in controlling the root lesion nematode which had appeared on Narcissus on light sandy soils in the Puyallup area.

Our Iris bulbs usually performed well when forced in greenhouses, but sometimes, after cool and cloudy summer weather, the results were poor. Therefore, in 1948, we launched a cooperative experiment with Dr. Neil Stuart and associates in the USDA at Beltsville, MD. Out of this developed a series of treatments, including heat curing, stabilizing, precooling, etc., which enabled greenhouse forcers to force Washington bulbs easily. When an extremely severe freeze during the winter of 1953/54 damaged Holland's Iris crop, Dutch exporters with orders to fill purchased Northwest Iris bulbs as replacement. The Washington bulbs performed so well that forcers in Europe requested our bulbs in following years until eventually about 35 million Iris were being exported. This export continued until the early 1970's when a combination of factors occurred and sales gradually declined.

Other forcing tests with Dr. Stuart showed: the stimulating effect of ethylene gas on Iris bulbs, and the benefit of precooling Tulip bulbs at lower than usual temperatures. All the above treatments are now in use in major bulb growing countries.

Among other contributions are: research on control of certain lily and gladiolus diseases; and the initiation (1948) of annual Bulb Grower Short Courses and Field Days to keep growers up-to-date on the latest discoveries and recommendations.

Turfgrass Diseases. Lawns in western Washington are often infested with Fairy Ring, caused by mushroom type fungi (usually *Marasmius oreades*). During the 1940's and 1950's we received more inquiries on Fairy Ring than on all other diseases of ornamentals combined. Therefore, research was initiated with Miller and others in 1949 to find a control. The best were mercurials but they were only partially effective. Complete control was obtained with methyl bromide fumigation followed by reseeding, but this procedure did not become popular. The most practical approach was to "live with" Fairy Ring, but to reduce its effects by keeping the grass vigorous with ample fertilization and watering.

Golf courses had additional problems. Roderick Sprague reported in 1950 that Fusarium Patch and Corticium Red Thread occurred in western Washington. However, most golf course superintendents attributed their problems to Dollar Spot (*Sclerotinia homoeocarpa*), a common disease elsewhere in the U.S. on golf greens. The only problem was that control measures effective against Dollar Spot did not work so the Northwest Turfgrass Association requested assistance from WWREC in 1955.

Our surveys of golf course greens in 1955 and 1956 showed that Fusarium Patch (*F. nivale*) and, to a lesser degree, Corticium Red Thread (*C. fuciforme*) were the most common diseases. Dollar Spot was not found in this nor in subsequent surveys. Later, Ophiobolus Patch (*O. graminis* var. *avenae*) (= *Gaeumannomyces*) plus other less important pathogens, were added to the list. The turfgrass disease complex in western Washington closely resembled that in England, so we were able to use many of their research results as a guidelines. Surveys of eastern Washington golf courses showed that the above fungi were also present there, plus the important snow mold (caused mostly by *Typhula incarnata*) as had been reported by Sprague, Bruehl and others.

Our first approach to control was fungicidal. Certain mercury and cadmium compounds proved to be reasonably effective. Later an alternating program (as with Fore or Benlate, alternating with Rhodia 26019) was recommended for Fusarium Patch since it controlled the fungus, reduced the probability of development of resistant strains, and presumably reduced the build-up of toxic residues in the soil.

Nutritional studies were started next. Roy Goss and I found that Red Thread could usually be controlled with nitrogen fertilizers although, when grass was growing slowly, fungicides were often needed. Ophiobolus Patch responded more to the use of ammonium sulfate than to fungicides and ammonium sulfate partially controlled Fusarium Patch. Since the effective fungicide Fore contained sulfur, we next studied sulfur alone and found it beneficial in reducing attacks by both Fusarium and Ophiobolus, in addition to producing a greener grass. Sulfur reduced, but did not eliminate, the need for fungicides.

Our third method of attack was to seek varieties of bentgrasses, fescues, ryegrasses, and bluegrasses resistant in western Washington to the common pathogens and, in Eastern Washington and Idaho, varieties of bentgrasses resistant to Fusarium and Typhula (in cooperation with Al Law (WSU) and Dr. Ron Ensign (Idaho)). Hundreds of available varieties were tested, not only for disease resistance, but for agronomic features as well. None was immune, but several satisfactory varieties were found and recommended for replacement of the common types in general use.

Miscellaneous Diseases. Numerous other diseases of ornamental plants came to our attention and a few, for one reason or another, were investigated in some detail.

Rhododendron diseases were important because the host is one of the most popular shrubs in western Washington as well as being our state flower. At first, this was primarily a survey project with Dr. Maksis Eglitis, occasionally involving control studies. However, in 1954 we found, for the first time in the U.S., a new rust (*Chrysomyxa ledi* var. *rhododendri*) which was widespread on certain rhododendron varieties in a large coastal nursery. The Forest Service became alarmed, fearing that the rust might spread to native spruce (the alternate host) as had happened in northern Canada and Europe on other species of spruce. Subsequently, obtained a special grant to investigate the life cycle, host range, and possible control of the rust with the assistance of Dr. Eglitis, Bill Scheer and Worth Vassey. Meanwhile, the State Department of Agriculture quarantined the infected nursery for a time. The results supplemented by Gould in nurseries in Germany and native locations in Switzerland, indicated that the rust had probably been present in a coastal nursery in another coastal state several years before appearing in Washington, that it was present only to a limited degree in inland locations in Washington on highly susceptible varieties, that a great variation existed in resistance of rhododendron species and varieties to not only this rust but to other rhododendron rusts as well, that fungicide applications helped reduce incidence but were inadequate in a commercial nursery, even when accompanied by removal of all obviously infected leaves, and, most important, that because of the difference in climatic conditions between Washington State coastal area and its native habitat in Switzerland, it was not likely to produce the spore state (telial) capable of infecting spruce here. Therefore, we informed the Washington State Department of Agriculture and Forest Service that they should keep infested nurseries under observation, but that the rust was unlikely to pose a problem to spruce here. So far as I know, this rust has never developed on spruce in the U.S.

Large amounts of Salal, a popular native 'brush' crop used in flower arrangements, had to be discarded because of excessive leaf spotting. At the request of brush pickers and wholesalers, we (with Dr. Eglitis and Darrell Turner) initiated a study to determine the types of causal agents and their control. A large number of fungi were found, with the most common being a *Phyllosticta*. Fungicide applications in the forest were tried but they were not of much benefit, presumably because of the tangled mass of vegetation and repeated discharge of spores. The only approach that proved reasonably practical was the application of nitrogen fertilizers which stimulated earlier emergence and maturing of leaves. The leaves were presumably more resistant by the time spores were discharged. Nitrogen also improved the quality of the foliage.

Lilacs are popular throughout the U.S., but to a lesser extent in western Washington because of the very serious bacterial blight (*Pseudomonas syringae*). We tried various chemicals but only streptomycin sulfate gave reasonable control. However, 10 to 12 sprays were required, and this was too much effort for the average gardener. Therefore, having observed some variation in resistance, we collected available species and varieties in the hope that resistant types could be found. By this time in my career I had become convinced that disease resistant plants, if available, were the best solution to many home gardener's problems. By 1970 we had obtained over 200 species and varieties. Some, particularly Asiatic types, were highly resistant and could presumably be used as a source of breeding material with the more popular European types. However, there were some reasonably resistant varieties of the latter which we recommended for trial planting.

The last large project, other than bulb and turfgrass diseases, was the testing of *Pyraacantha* (firethorn) species, varieties and new hybrids, primarily for Scab (*Fusicladium pyraacanthae*) resistance, starting in 1973. This developed partially as a result of a request by Dr. Don Egolf (USDA Arboretum, Washington, D.C.) to test his new hybrids for disease resistance (both scab and fire blight), cultural characteristics and climatic compatibility. In cooperation with W. E. Vassey, Dr. G. F. Ryan and Dr. Don Egolf, we obtained as many varieties and species as possible. Fortunately, six good varieties and new hybrids showed enough resistance to scab, fire blight and winter injury to justify recommending them in 1978. The results are also being used by Dr. Egolf in his hybridizing program.

### Folke Johnson

by Folke Johnson

After the resignation of Glenn A. Huber, Folke Johnson (1943-1973) was hired July 1, 1943 at the rank of Assistant Plant Pathologist to work primarily on vegetable diseases. Shortly thereafter, Leo Campbell assumed vegetable pathology, with Folke taking over fruit crops. This was during World War II and, at that time, western Washington grew a large acreage of canning pears and Italian prunes, mostly in Clark and Skamania Counties. Prune orchards in the vicinity of Vancouver, Washington, were severely beset with a "leaf scorch" which caused premature defoliation with injury centered near alumina smelters. In cooperation with Dr. D. F. Allmendinger, Horticulturist and Superintendent of the Vancouver Experimental Unit, and V. L. Miller, Agricultural Chemist from the Puyallup Station, we found the injury was induced by fluorine effluents from the alumina smelters (at Troutdale, Oregon and Longview and Tacoma, Washington). Gladiolus and certain Pinus species were very susceptible. We found higher levels of fluorine in leaves of resistant gladiolas than in susceptible gladiolas. The former remained alive; the latter died, stopping fluorine absorption (1950). Later these crops became established as indicator plants to fluoride pollution. (Gould says Folke is too modest about this fluorine research).

Also at this time research was begun to determine the cause of the "Blossom blast" and "Twig dieback" in both the canning and winter pear orchards in Clark, Skamania, and Klickitat Counties, a serious problem for many years. Pathogenic organisms were ruled out after much testing and the final conclusion was boron deficiency. Allmendinger and Miller helped to sort out this problem.

Boron deficiency had been described on pear in Oregon and British Columbia, but the situation in southwest Washington was atypical. There were no symptoms on the fruit, and trees did not respond to boron added to the soil (1955). They found that symptoms were severe in the season following a dry summer. The pears could not obtain boron in adequate quantities from relatively dry soil, and these orchards were not irrigated. Success was obtained when boron was sprayed on the trees (1 lb actual boron per acre) after harvest in the fall.

Blueberries. Near the end of World War II attention was called to a dieback of young, current season's stems of blueberry bushes in the Long Beach area caused by *Botrytis cinerea*. Late summer fertilizer applications produced succulent shoots which were weakened by early fall frost, predisposing the young stems to infection and degeneration. The cooperation of D. J. Crowley, Superintendent of the Cranberry, Blueberry Laboratory at Long Beach, was helpful.

The dreaded "mummyberry" disease of blueberries, caused by *Sclerotinia urnula* (also *Monilinia vaccinii-corymbosi*), became serious in the Puget Sound region in the early 1960's. In a few years it was present up and down the coast wherever blueberries were grown. The effect on small, neglected acreage was devastating. The fungus causes a blossom blast in addition to blight of young twigs and leaves. The fruit becomes a worthless mummy that falls to the ground, inoculum for new infections the following spring. To make matters worse *Godronia* "Stem canker", (*G. cassandrae* and *G. menziesia*) was identified. *G. menziesia* was destructive and common. Infections originate through the leaf scars after leaf fall and they often girdle whole stems. Surveys revealed many other fungi which, at that time, were of little or no importance.

A problem arose in the early 1970's when some blueberry plants in Lewis County showed a general decline, resulting in severe stunt, leaf cast, and death of whole plants. Observations in the spring revealed scattered, dark-brown leaf spots of irregular size and shape on affected bushes. From these spots and leaf tissue, a "Ringspot" virus thought to belong to the tomato or tobacco ringspot group, was identified. It was transmitted from diseased to healthy plants by *Xiphenema* spp. or *Longidorus* spp., or both.

Cranberries. Serious problems in the cranberry bogs in the Long Beach area developed in the period of 1950 and later. A twig blight and leaf cast caused by *Lophodermium hypophyllum* and *L. oxycocci*

reduced fruit yield seriously. The stems blighted and, in severe cases entire fields were taken out of production. At this period, Dr. Charles C. Doughty, a horticulturist, replaced D. J. Crowley, who retired from the Coastal Washington Research and Extension Unit at Long Beach. After a few years, Dr. Doughty moved to Western Washington Research and Extension Center, Puyallup, and Azmi Y. Shawa, also a horticulturist, took over the Coastal Washington Research and Extension Unit. Both cooperated in the pathology studies of cranberries. The expertise of Maksis Eglitis in the blueberry-cranberry investigations cannot be over-estimated.

Shortly after the Lophodermium problem was resolved by timely application of fungicides, a condition developed around the Grayland-North Beach areas in which certain bogs were affected by a root rot which killed plants in somewhat round to irregular areas similar to "Fairy ring" of turfgrass. Mushrooms growing in the outer margins of the dead vines were identified by Dr. Stuntz and his group in the Botany Department of the University of Washington as *Psilocybe agrariella*. Its pathogenicity for cranberries was verified from pure cultures.

In addition to these two major problems, there were a number of fungi that caused leaf spots or blights and fruit rots, including "Rose bloom", *Exobasidium oxycocci*, and "Red leaf" *Exobasidium vaccini*. In all, 19 genera of fungi were identified from cranberry fruit and other plant parts.

Brambles. In the 1970's, Washington was the leading state in production of red raspberries. In heavy or poorly drained soils a root rot was destructive. The cause was found by Dr. Stace-Smith, University of British Columbia, to be *Phytophthora* spp. Other problems were "Crown gall" *Agrobacterium tumefaciens*, "Cane and Spur blights" *Didymella applanata*, *Elsinoe veneta*, *Leptosphaeria coniothyrium* and "Leaf rust" *Phragmidium rubi-idaei*. In addition, the red raspberry was subject to several virus diseases; "Ringspot", mentioned earlier in the blueberry section, was important as the cause of a condition known in the late 1960's as "Calico". There were also "mosaic" viruses, but the complex was not entirely understood at that time.

On the Evergreen cutleaf blackberry, grown commercially in western Washington, another "Leaf rust", *Kuehneola uredinis*, was periodically destructive. But more damaging to this crop was "cane blight", caused by *Rhabdospora rubi*. In some seasons it destroyed the entire crop of that year.

Strawberries. In the 1940's the leading commercial variety was Marshall. It degenerated because of red stele, caused by *Phytophthora fragariae*, but the cultivar was also subject to winter injury and susceptible to several viruses which caused "yellows". Within a year or two after infection the plants degenerated and had to be destroyed. The new 'Northwest' cultivar, developed by Dr. C. D. Schwartze, plant breeder at the Puyallup station, replaced Marshall. The Northwest variety was a heavy yielder, winter hardy, and resistant to "Leaf spot" *Mycosphaerella fragariae*. In subsequent years, Northwest was replaced by several newer cultivars developed by Dr. Schwartze and in some cases new varieties were introduced from the U.S.D.A., A.R.S., Plant Breeding Program at Oregon State University, Corvallis, Oregon and by hybridizers from the University of British Columbia, Vancouver, Canada.

Maksis Eglitis. Maksis Eglitis (1950-1960?), a senior mycologist with a Doctor of Science degree, came from the University of Riga, Latvia, to Puyallup. Shortly after arriving Dr. Eglitis became interested in the use of radio frequency energy, also commonly called "diathermy" or "radio waves". A 4 kw generator was adjusted to 33 megacycles (mc) that produced deep heating in most objects that intercepted the energy. By holding soil heavily contaminated with damping-off fungi between the electrodes for 10 minutes or less, seedling survival could be increased from 30-40 percent in the untreated control soil to over 90% in the treated samples. Germination of "Hard seed" of alfalfa was increased from 60-70 percent to over 90 percent after a 30-second treatment and certain viruses within plant tissues could be controlled by diathermy.

Eglitis was senior author on papers describing the European rhododendron rust in western Washington (with Gould and Doughty, 1955) and on a paper on rhododendron disease with Gould (1956). In 1966 Eglitis with Gould and Johnson published "Fungi found on Ericaceae in the Pacific Coastal Area". Folke Johnson stated that Eglitis developed an extensive pathological herbarium of Ericaceae which was added to the departmental herbarium at Pullman.

#### Walter J. Apt

Walter Apt (1955-1960) strengthened the nematology effort. The seed gall nematode of *Agrostis* spp., discussed under Bill Courtney, was not controlled by grazing, flooding, or any other practical means in seed fields. Walter Apt, Bill Courtney and Herman Austenson, Agronomist, attempted to break the life cycle of *Anguina agrostis* by spraying the grass with growth regulators to prevent flowering (1960). The best chemicals largely suppressed flowering, but a few flower stalks developed, so control was not complete.

Many bulb fields in the Puyallup Valley were infested with the root lesion nematode, *Pratylenchus penetrans*, and Apt and Chuck Gould found that fumigants increased the yield and quality of King Alfred daffodils. If the soil was treated too severely, especially with methyl bromide, losses from Fusarium basal rot increased.

#### Richard L. Gabrielson

Dick Gabrielson (1960-present), who succeeded Leo Campbell as vegetable pathologist, is best known for his efforts to provide pathogen-free vegetable seeds. Washington produces 80% of the cabbage seed of the U.S. and 30% of the cabbage seed of the world, much of it in the Skagit Valley, as well as several other vegetable seeds. The summer (1977) issue of "Search", published by the American Seed Research Foundation, was dedicated to the work of Gabrielson and of J. D. Maguire (Agronomy Department, WSU, Pullman) on controlling *Phoma lingam* on crucifer seed crops. Dick has contributed significantly to the control of *Sclerotinia sclerotiorum* and several other pathogens of vegetables in Western Washington. He is now the senior pathologist at Puyallup. Much of Gabrielson's research is presented under the seed industry in the section on Mt. Vernon, even though Gabrielson has always been stationed at Puyallup. He studied diseases of cucumbers. There was a pickle factory at Enumclaw, about 20 miles east of Puyallup. A grower of winter squash on Whidbey Island was suffering severe losses in storage, in spite of careful handling. Dick devised a fungicidal treatment of the stem end that greatly reduced the loss.

Club root of cabbage, caused by *Plasmodiophora brassicae*, is a world-wide problem of crucifers in wet, acid soils. It increases with repeated crops of crucifers, and the soil remains infested for years. The fungus produces millions of small spores in rotted host tissue. These spores become mixed with the soil during tillage. In addition, when the resting spores germinate, zoospores that can swim short distances in water within the soil are formed. These zoospores are attracted chemically to the host. They penetrate root hairs of cabbage. Evidence suggests that in alkaline soils resting spores do not germinate so disease does not develop. Liming to reduce soil acidity is a major means of control, but in many cases, considerable disease developed even after liming. R. L. Dobson, Ph.D., 1982, investigated this problem with R. Gabrielson at Puyallup.

Dobson and coworkers found that the size of lime particles was important when liming was done shortly before planting. In heavily infested soil, even though coarse limestone raised the bulk soil pH to 7.1, 80% infection occurred. With medium coarse limestone, 48% infection; fine limestone (particles less than 0.5 mm in diameter), 27% infection, even though the bulk soil pH was 7.3 and infection should not have occurred. When finely powdered limestone was used, no infection occurred. With coarse materials acidic microsites in which the spores germinated were present within the soil; with powdered limestone thoroughly mixed with the soil no acidic microsites existed and no disease developed. In order for liming to dependably control clubroot the soil must be treated in such a way that it is uniformly slightly alkaline.

The limestone should be as finely ground as possible, thoroughly mixed with the soil, and enough time for the limestone to react with the soil must elapse before the cabbage is planted.

Dobson, Gabrielson, and A. S. Baker (1982) made an additional important discovery. The life cycle of *Plasmodiophora brassicae* includes two sets of swimming spores. The first set (primary zoospores) is produced by resting spores that germinate in the soil. Primary zoospores infect root hairs, the fungus develops slightly within them, and then a second crop (secondary zoospores) of swimming spores is released into the water in the soil. If plants are infected only by primary zoospores disease does not develop. When secondary zoospores infect the root, disease develops. In other words, two crops of swimming spores are essential, and the two types of spores are qualitatively different, with only one (secondary zoospores) capable of producing disease. They found that primary zoospores can infect under slightly drier conditions than secondary zoospores, that primary zoospores are smaller than secondary zoospores, that primary zoospores are motile in thinner water films within the soil than secondary zoospores, providing circumstantial evidence that the secondary zoospores liberated from infected root hairs fuse in soil water to form larger zoospores. The zoospores released from root hairs acted as sexual units (gametes) that fused to produce larger, disease-causing swimming spores (zygotes). These fundamental discoveries proved that the life cycle of this primitive fungus was more complicated than thought in earlier years.

Clubroot of cabbage is not a major problem in Washington, but these studies, both practical and fundamental, contribute to plant pathology as a world-wide science.

#### *EXTENSION, THE EARLY YEARS*

*By Arlen D. Davison, 1967-1974*

Prior to mid 1967, extension plant pathology had been handled officially out of the main campus at Pullman. Otis C. Maloy became Extension Plant Pathologist at Pullman in 1963, replacing Marion Harris. C. Gardner Shaw, Chairman of the Department, convinced Director John Miller and State Program Leader Harry Cosgriffe of the need for an extension pathologist in western Washington, and in August, 1967 Arlen D. Davison became the first Extension Plant Pathologist stationed in western Washington.

Prior to my assignment to Western Washington Research and Extension Center (WWREC), Otis Maloy provided technical expertise and conducted educational programs in western Washington to the degree that time and budgets allowed. Research plant pathologists Gould, Johnson, and Gabrielson served unofficially as extension pathologists to the commodity groups with which they worked. John Dodge, Extension Horticulturist at WWREC also provided substantial help to home gardeners and commercial producers.

I spent much of the first year becoming acquainted with the agricultural industries and urban gardening problems of the "westside". A Plant Disease Clinic was established to provide diagnostic services to County Extension Agents and "walk-in" clientele at WWREC. During the spring of 1968, Roy Goss, Extension Turf Agronomist, John Dodge, Extension Horticulturist, James Pennel, Extension Entomologist, and I convinced Extension administrators of the desirability of establishing a Plant Clinic, staffed by a highly skilled classified staff employee. Establishment of the Plant Clinic brought about a closer coordination of diagnostic, plant, and insect identification services. Roy M. Davidson, a 1968 (-1988) Ph.D. graduate of the Department of Plant Pathology, WSU, was hired to staff the Plant Clinic. He worked with each of the above faculty but was administratively responsible to me. During the first several years the number of specimens increased rapidly. Most samples were "cultural" problems followed by samples for insect identification and plant diseases.

The increased demand for service placed a heavy load on County Extension Agents, many of whom lacked training and experience in entomology and plant pathology. Those lacking such knowledge were sending samples directly to the clinic without attempting to handle the matter at the county level, burying



the clinic in samples. County Extension Agent training was an obvious partial solution. After two or three agent-training sessions and individual training provided to agents on their own "turf", an agent training session was planned. When agents arrived at WWREC for the training they were given about 40 samples which they were asked to diagnose and/or identify and provide a recommendation to address the problem. They were also to defend their diagnosis or identification before their colleagues. Initially the specialist feared a revolt but as the agents got in the spirit of the session, feelings and skepticism began to change. Most became enthusiastic. One purposes of the exercise was to illustrate the necessity of obtaining essential information from clients before submitting samples to the clinic. The specimens had ranged from those with complete background data to essentially no background information. Through this exercise they also learned the importance of careful observation at the site and examination of the samples. From that time on the number of repeat samples declined and the quality of the information provided with the samples improved.

With the establishment of the Plant Clinic and training of County Extension Agents the home gardening public gradually learned of Cooperative Extension's commitment to serve their need, and demand for assistance and diagnosis increased to the point that some urban county agents were spending nearly all their time in these activities. Something had to be done.

During the winter or spring of 1971 a meeting to discuss possible solutions was attended by Davison, Wesenberg, Pennel, and Goss, by Dave Gibby, County Extension Agent, King County, by Dodge, Hopp, and Jones, District Extension Supervisors, along with William Ackley, Chairman of the Department of Horticulture and Harry Cosgriffe, State Program Leader in extension. I presented the concept of recruiting and training knowledgeable volunteers who could provide disease diagnostic and insect identification services and cultural recommendations to the public at "Master Gardener Clinics" in shopping centers and/or other public areas. The concept was simple - to place a group of trained volunteers between the agents and the general public. The agents could thereby utilize their training and expertise to manage an enlarged program which provided gardening information to a larger segment of the population than was currently possible.

To test the feasibility of such a new approach, a "test" clinic was tried at the Tacoma Mall. King and Pierce County horticulture agents and specialists from WWREC staffed the "test clinic". Announcements were placed in the local papers. The response of the public was immediate and positive. Sunset magazine learned of the test clinic and sent a reporter. Partially as a result of the Sunset story and newspaper advertisements, over 1,000 people applied to become Master Gardeners in the King-Pierce County program. Several hundred received 50-plus hours of training, passed the state pesticide licensing exam, and volunteered a minimum of 50 hours of service to the program during the year following their training. Shortly after the King/Pierce County experience, a smaller program was initiated in Spokane, Washington, an urban area of 200-300,000 people.

To assist the County Extension Agents in recruitment, furnishing materials, scheduling "clinics" and nurturing the Master Gardeners, a staff position of Extension Assistant was established in King, Pierce, and Spokane Counties. Funding was furnished by the State Cooperative Extension Service.

The concept of the Master Gardener Program spread to over a dozen counties in Washington and within five years to 40 states and Canadian provinces. In addition, the concept of using trained volunteers resulted in such additional "Masters" programs as the Master Food Preserver, Master Food Shopper, Master Livestock Program and probably others.

In 1974, Dr. Davison resigned as Extension Plant Pathologist to become State Program Leader of Agriculture and Natural Resources at the main campus in Pullman. He was Chairman of Department of Plant Pathology at Pullman, 1981-9186, and in 1986 he returned to Puyallup as Assistant Dean, in charge of the Puyallup, Mt. Vernon, Vancouver, and Long Beach units of WSU.

Ralph S. Byther

Ralph Byther (1975-present) replaced Arlen Davison as Extension Specialist in Plant Pathology at Puyallup. Ralph is a gifted photographer and his excellent photographs have enriched talks and publications. He has wide responsibilities (turf, ornamentals, vegetables, fruits) which makes wide knowledge and good management of time essential. He is dynamic, forceful, and effective. He also engages in research to the extent time permits, even though he is officially 100% extension. He was a coworker with Peter Bristow on control of mummy berry of blueberries (discussed under Bristow).

Peter R. Bristow, 1976-present

by Peter Bristow

Peter Bristow replaced Folke Johnson as small fruit pathologist.

In 1976 and 1977 mummy berry disease (*Monilinia vaccinii-corymbosi*) of high bush blueberry was severe in many areas of western Washington due to lack of registered chemicals effective against infection by ascospores, poor timing of sprays because soggy soil prevented ground machine access to many fields, increase of overwintering primary inoculum following several years of poor control, and weather in the spring for several successive years favorable for disease development. A new fungicide, triforine (Funginex), was found, in cooperative research with Dr. Ralph Byther, Extension Plant Pathologist, to be extremely effective against infection by ascospores and conidia. A Section 18 Emergency Exemption allowed triforine to be applied to Washington blueberries in the spring of 1977, the first commercial application of this fungicide in the United States. Helicopters permitted applications when soils were too wet for ground equipment. Triforine does not control Botrytis blossom blight, so recommendations were adjusted to include the use of materials during bloom that would keep both diseases in check. It is difficult to find mummy berry disease today and some growers are able to skip spraying for a year or two because inoculum levels are so low.

We tried since about 1983 to identify the cause of blueberry scorch, which results in severe blossom blighting. Working with Robert R. Martin, a Plant Virologist with Agriculture Canada, a carlavirus was found to be the cause. An ELISA-based test allows rapid detection of infected plants. Certain varieties test positive yet exhibit no symptoms. Present efforts center on identifying the vector, establishing the latent period, and documenting yield losses.

Cranberry. Epidemiological studies (spore trapping, environmental monitoring, etc.) of twig blight (*Lophodermium oxycocci*) of cranberry showed that although inoculum (ascospores) is produced from late spring through harvest, infection occurs primarily in July and early August. As a result, fungicide applications were modified, reducing the applications from 5 or 6 to 3. Fungicide timing was further improved using a system which predicted when the infection period begins. The predictive system, a part of the proposed cranberry integrated pest management program, is based on monitoring the development of ascocarps in the spring. Chlorothalonil (Bravo 500 and 720) was superior to registered products for control of twig blights (and fruit rots).

Losses from fruit rots of cranberry in the field are generally only about 5% but losses of fresh fruit held in refrigerated storage after harvest can be up to 50%. Fungicides applied to control twig blight also control field storage rots. Fruit used for processing is frozen immediately after harvest, consequently it suffers no post-harvest losses due to fungal fruit rots.

Rose bloom (*Exobasidium oxycocci*) of cranberry is conspicuous because of the fleshy, pink-colored abnormal branches that form on infected uprights in late-spring. Disease incidence can be very high (>80%) in areas within bogs during certain years but we did not know what if any damage this disease causes. Cranberry yield is affected by number of flowers per flowering upright, fruit set, berry size, etc. By measuring each component on both infected and healthy uprights, we found the yield on infected uprights is reduced by about one-third. Because cranberries are perennial, it is possible to measure the

effect the disease one year has on next year's crop. It appears that slightly fewer fruit buds are set on infected than healthy uprights, suggesting that there is a slight negative carryover effect.

Red Raspberries. A root rot of red raspberries caused by *Phytophthora erythroseptica* has been serious for years, especially on heavier soils. The new systemic fungicide, metalaxyl (Ridomil), applied to the surface of the soil in the fall and again in the early spring controlled it and in some cases, diseased plantings were restored to vigor and productivity. After several years under Section 18 Emergency Exemptions, the fungicide was fully registered in 1985. After a method for screening raspberry seedlings for resistance was developed, some resistance was found in native populations of both North American and European red raspberry. Sources of what may be immunity were found in two subtropical *Rubus* species but both species have several undesirable horticultural characteristics. It is also unknown if they will cross readily with red raspberry.

*Leptosphaeria coniothyrium* is a wound parasite of red raspberry which can girdle canes at the point where the fruit-catching plates on mechanical red raspberry harvesters wound canes. Cane blight is a major limiting factor in mechanically harvested red raspberries in the United Kingdom and Europe. A cooperative project with pathologists from Scotland and West Germany found no difference in the virulence of isolates from the respective countries nor in the susceptibility of test cultivars grown in each of the areas. Despite the presence of the pathogen and wounds, growers in the Pacific Northwest have successfully harvested red raspberries by machine for nearly 15 years. We concluded that the normally warm, dry harvest period in the Pacific Northwest is unfavorable for disease development by promoting healing of wounds made by the harvester and reducing the production and dispersal of inoculum.

Increasing quantities of fresh red raspberries have been shipped by air to distant markets since 1980 and we demonstrated that application of fungicides between early bloom and harvest increased the shelf-life of this extremely perishable commodity. Cool, wet conditions which favor pre-harvest fruit rot, especially by *Botrytis cinerea*, occur about one year in every five. Vinclozolin (Ronilan) and iprodione (Rovral), both dicarboximide fungicides, control *Botrytis* fruit rot and each has been used under Section 18 Emergency Exemptions but as of this time neither has received full registration on this crop. Irradiating fresh red raspberries with gamma radiation at doses as high as 200 kilorads did not improve the shelf-life of treated fruit in refrigerated storage.

Strawberries. Year in and year out gray mold (*Botrytis cinerea*) is the most serious fungal disease of strawberries. Control depends on timely applications of fungicides. Weather conditions during bloom and fruit ripening are usually favorable for disease development (cool and rainy). Benomyl and captan have been the standards for years, but their effectiveness has diminished, most likely due to tolerant strains of the pathogen. Vinclozolin (Ronilan) was tested in the late 1970's and registered a few years later. It is excellent even in years when it is rainy during harvest. The spectrum of activity for vinclozolin is narrower than the fungicides it replaced; hence, common leaf spot (*Mycosphaerella fragariae*) has become serious on the susceptible cultivars 'Olympus' and 'Shuksan'. Most strawberry cultivars bred in the Pacific Northwest, however, are highly resistant to common leaf spot. Irradiating fresh strawberries with gamma radiation at doses up to 200 kilorads did not extend the shelf-life when fruit was held in refrigerated storage.

The new systemic fungicide, metalaxyl (Ridomil), controls red stele root rot (*Phytophthora fragariae*) of strawberry. The chemical not only protects new plantings made into infested soil, but partially restores infected established plantings by allowing runner plants to root and develop into productive plants. Registration of metalaxyl for this use is expected in the very near future.

Damping off in plum and cherry seedlings. A local nursery expanded into cherry and plum seedlings for use as root stocks. Damping off, both pre- and post-emergence, caused significant stand and quality problems. Soil-borne *Rhizoctonia* and *Fusarium* species were the predominant fungi isolated from

diseased seedlings. Treating cold-stratified seed of both crops with a mixture of benomyl plus captan immediately prior to planting eliminated the problem and this is now the standard practice.

Sensitivity of pollen to pesticides. Current disease control recommendations for blueberry (mummy berry and Botrytis blossom blight) and cranberry (blossom blights and, at times, twig blight) call for fungicide applications to be made during bloom. While most chemicals were toxic to pollen *in vitro*, there were few adverse effects when these materials were used in the field. Formulation also influences toxicity. The emulsifiable concentrate formulation of triforine was markedly more toxic to blueberry pollen than either the wettable powder or flowable formulations. Nearly all the toxicity of the emulsifiable concentrate formulation was accounted for by one of the inert ingredients in that formulation and not by triforine.

#### Gary Chastagner

*by Gary Chastagner (1978-present)*

The primary objective of my research, as successor to Chuck Gould, on the diseases of ornamental bulb crops, turfgrass, and Christmas trees is the development of effective disease management based on understanding the etiology and epidemiology of the important diseases on these crops, abiotic, fungal and viral.

Daffodils. Daffodil fire, caused by *Botryotinia polyblastis*, is common throughout production areas in western Washington, reducing yields 10-60%. Studies on factors affecting the production of apothecia and identification of infection periods resulted in practical control with vinclozolin.

Tulips. Studies on the survival of sclerotia of *Botrytis tulipae*, the cause of fire on tulips, demonstrated that the rotation period between tulip crops could be reduced from the 3-5 years commonly recommended to 1-2 years because of the limited survival of sclerotia *in vivo*. Dicarboximide fungicides, iprodione and vinclozolin, provide superior control of fire on tulips, particularly on the flowers, compared to the standard fungicide used by bulb growers. In 1980, the increased value of the Washington tulip crop due to increased yields of bulbs and flowers resulting from iprodione compared to mancozeb was estimated to be 2 million dollars.

Fire on tulips has been used in a model to evaluate the effectiveness of management practices in delaying or preventing the development of benomyl and/or iprodione resistant strains of *B. tulipae* under field conditions. During this five-year test, applications of benomyl in combination or alternation with other unrelated fungicides prevented the development of resistant strains of *B. tulipae*. Although iprodione resistant strains of *B. tulipae* are easily selected *in vitro*, iprodione resistant strains of *B. tulipae* have not developed *in vivo* regardless of fungicide-use pattern.

Cooperative work with Dr. John Hammond, a USDA Virologist at Beltsville, on the transmission of tulip break virus (TBV) in western Washington has identified periods of aphid transmission of TBV and identified aphid species during periods of virus transmission. Application of monoclonal antibody techniques to large scale testing of tulips that may not express symptoms of TBV infection will allow growers to improve the quality of their propagating stocks and lower the incidence of new infections by limiting the reservoir of virus-infected material in their plantings.

Lilies. Lilies are increasingly important in the Pacific Northwest. Fire, caused by *Botrytis elliptica*, the most important disease, requires multiple applications of fungicides. Cooperative studies with Dr. R. P. Doss, a USDA Plant Physiologist at Corvallis, resulted in the development of techniques for inoculum production and inoculation of lily leaves. These techniques help identify ornamental lilies with resistance. Histological and scanning EM studies have shown that most infections of lily leaves by *B. elliptica* are associated with the stomatal apparatus on the undersides of the leaf, emphasizing the need to direct sprays to the lower leaf surface. Currently, there are major problems associated with the control of several Botrytis diseases on bulb crops in western Washington and Oregon due to the development of resistance to benzimidazole and dicarboximide fungicides.

Turf. The possibility of nematode damage to putting turf was a major concern of golf course superintendents in the late 1970's. A survey of golf courses throughout Washington revealed high populations and the presence of several species associated with poor areas in putting turf, particularly west of the Cascades. However, nematode control indicated that reducing the nematode populations did not significantly improve putting turf quality.

Fusarium patch is controlled through management of soil fertility coupled with the use of fungicides. My research resulted in the registration of new fungicides which provide control with fewer applications. The lack of control following applications of iprodione was due to resistant strains of the pathogen, the first report of resistance to iprodione under field conditions.

Take-all patch, caused by *Gaeumannomyces graminis* var. *avenae*, is serious on bentgrass turf and efforts to control it with fungicides have been unsuccessful. *In vitro* tests have identified several ergosterol biosynthesis-inhibiting fungicides with excellent activity against this pathogen. The key is to make applications during the winter, not during early spring just prior to symptom development.

My research on turf diseases has recently focused on necrotic ring spot, caused by *Leptosphaeria korrae*. Necrotic ring spot, first identified in North America in 1984, is now recognized as probably the most important patch disease of bluegrass turf, particularly turf established by sod. By producing pseudothecia *in vitro* we can compare Washington isolates to isolates from other states. The susceptibility of 75 cultivars of Kentucky bluegrass is currently being tested under field conditions in eastern and western Washington. Efforts by homeowners and lawn care professionals to control necrotic ring spot were largely unsuccessful because many of the fungicides which control Fusarium blight do not control necrotic ring spot. We identified several fungicides including fenarimol which, if applied in the spring, provide control.

Fir trees. In the early 1980's, Swiss needle cast was a major problem of Douglas-fir Christmas trees. 84% of the trees in western Washington and Oregon were infected and growers lost an estimated \$3.4 million at harvest due to the premature loss of needles. Control could be obtained on Douglas-fir Christmas trees and forest trees using aerial applications of chlorothalonil. In addition to losses at harvest, the disease also reduced the postharvest quality of trees by increasing the rates of needle loss and moisture loss after harvest.

In 1984 and 1985, Douglas-fir Christmas trees were severely damaged in several plantations by stem cankers. Our research proved that *Melampsora occidentalis* caused this disease, identified periods of primary inoculum production, determined that high inoculum levels were necessary for the development of stem cankers and identified several fungicides that provide control if applied when new shoot growth was 1-2 inches long.

Although usually sporadic in western Washington and Oregon, Rhabdochline needle cast can severely damage Douglas-fir Christmas trees. By identifying periods of inoculum production and infection, control is possible with two properly timed applications of chlorothalonil.

The demand for Noble fir Christmas trees resulted in its increased planting in the Pacific Northwest. Current season needle necrosis is an abiotic disease reduced with foliar applications of calcium chloride or eliminated by shading trees with shade cloth during shoot elongation. Grovesiella canker was found in 10% of the plantations and the most likely way this disease is introduced into new plantings is on infected seedlings. Apothecia are capable of producing inoculum twelve months of the year. Work is in progress to identify infection sites and infection periods.

Phytophthora root rot and subsequent stem cankers can result in death of Noble fir in newly-established plantations. In cooperation with Dr. Phil Hamm at Oregon State University, our research has determined

that several species of *Phytophthora* cause this disease. We are determining the susceptibility of conifer species to *Phytophthora* spp. and the factors which affect infection and symptom development under field conditions.

Research on the keepability of Douglas-fir, Noble fir and Grand fir Christmas trees has identified threshold moisture levels that cut trees can dry to before their quality is affected. Pacific Northwest trees arrive at major Southwest markets with adequate moisture, but their quality is severely reduced by the time a consumer purchases them because of improper care on the retail lot. We identified methods that retail lot operators use to reduce moisture loss from trees, showed that stem diameter is an excellent indicator of the volume of water a tree will consume indoors, and compared the keepability of the major conifer species used as Christmas trees. The Christmas tree industry considers this work on factors affecting the postharvest keepability a model of quality research that has provided information to improve Christmas trees available to consumers.

#### *Communication*

A strong advocate of getting WSU's story before the public, I have taken every opportunity to encourage and provide television, newspaper and magazine interviews. I took a leadership role in the organization of the first meeting of Pacific Northwest research and extension personnel with responsibilities for Christmas trees attended by 28 individuals from Washington, Oregon, Idaho, Montana, and British Columbia. It served to facilitate communication and provide opportunities for cooperative research and extension programs. As a result of efforts by Drs. R. P. Doss, R. S. Byther and myself, WSU will host the International Society of Horticultural Science's Fifth International Symposium on Flower Bulbs in 1989. End of Chastagner.

#### *Cooperation*

For many years, first with barley yellow dwarf, then with strawbreaker foot rot, I (Bruehl) had experiments at Puyallup and Vancouver (the Southwest Washington Experimental Unit). These stations provided land and its preparation at no cost to my projects. At Vancouver, under superintendent Perry Crandall, every worker on the station helped plant by hand the entire world collection of spring oats and many spring wheats and barleys. (This can safely be told because Dr. Crandall has retired as have the administrators of that time.) Assistance to this degree should not be encouraged, but it eventually led to the release of Cayuse oats and other important benefits. James Cook and Rollie Line of the USDA at Pullman have worked for years at Mt. Vernon, again with excellent relationships. Dr. Haglund, an expert on soil fumigation with field applying equipment, has assisted Cook for years with soil fumigation. Others on the Pullman staff, especially Dennis Gross, have had similar assistance at Prosser. We owe a debt of gratitude to persons involved. Cooperation permits use of the special environments at the branch stations.

The many environments of Washington with many local problems have led to extensive use of farmers' fields. In wheat, for instance, flag smut experiments were concentrated in Klickitat County, snow mold experiments in Douglas and Okanogan Counties, all on private land. Progressive farmers enjoy seeing well-conducted and well cared-for experiments on their land. Experimenters who use private land should control weeds and respect the owner's rights as a polite guest. It is poor policy to use inoculum of soilborne pathogens on private land, unless the inoculum originates from the same farm, or near it, so experiments involving their use are best done on the experiment stations.

#### *Conclusion*

I just read the entire Puyallup section and find that, from Brodie to the present, proper use of fungicides is a major activity at WWREC. In my student days Cornell was famous for fungicide work, Wisconsin for environmental studies, and Minnesota for pathogenic specialization. Puyallup is our Cornell.

## **Irrigated Agriculture Research and Extension Center, Prosser**

Some of the history of the Irrigated Agriculture Research and Extension Center at Prosser has been covered, but additions will be made. Prosser is a busy town in the lower Yakima Valley with a food processing industry typical of sizeable towns in irrigated Washington. To the south is a high grassy plateau called the Horse Heaven Hills. To the north are the Rattlesnake Hills. Both names portray the general environment, which contrasts greatly with that of Mt. Vernon or Puyallup. The rainfall, not much over six inches per year, places Prosser in the driest region of the state. The drive from Prosser to the experiment station takes one across the Yakima River north and east through an unpromising area of rocky land. Upon entering the station, the land is choice and the station excellent.

In 1918 the Northern Pacific Railway donated some land and offered to sell other land cheaply if \$2,000 would be spent on improvements in one year. The citizens of Prosser raised \$3,000 and donated labor to prepare the land for cultivation. Mr. F. M. Rothrock sold land to the state at a favorable price. The Prosser station received its first real boost when Gordon Moores, a local legislator, obtained a \$35,000 appropriation for the new station.

According to H. P. Singleton, long-time (1929-1965) superintendent, the Prosser station might have closed in 1933 had it not received \$8,000 from the governor's emergency fund, the same Governor Martin for whom the WSU stadium is named and for which he contributed over \$1,000,000. The station grew steadily after its tenuous early years. 347 acres of the Roza Irrigation Project was added to Prosser in 1942. By 1961 an irrigated tract near Othello and by 1963 a tract near Royal Slope were ready for use in experiments, both administered through Prosser.

The station was started when the Yakima River Valley contained the greatest irrigated acreage, before the Columbia Basin Project existed. The Columbia Basin Project followed completion of Grand Coulee Dam. Prosser personnel travel more than formerly because the Columbia Basin Project is north of Prosser, important near Othello, Moses Lake, and Quincy.

The narrative about Prosser lacks a treatment of grapes, important in the Yakima Valley for grape juice and jellies (Concord types), and recently for fine wines (European grapes).

Superintendent H. P. Singleton instigated the provision of technical aides for scientists on regular state funds (hard money). The goal of the ARC, to provide each scientist with a technical aide, has not yet been fully achieved. J. Lewis Allison, superintendent from 1969-1975, complained that the IAREC had 75 WSU employees, faculty and staff, and only \$1,303,675 of state support when he resigned. Hard money went primarily for salaries, with little left for operations. When Allison came the station received about \$85,000 in grants. When he left it received \$339,716 from commodity commissions, mainly potato, tree fruit, hops, mint and asparagus, and in 1975 there were 1,250,000 acres of irrigated land. Allison spent much effort trying to convince the commissions that Prosser had the people but they needed greater financial support. Lin Faulkner, who succeeded Allison, continued in this tradition.

### *Curly Top and Sugar Beets*

Even though curly top of sugar beets was studied primarily in other states, the disease was so significant to our sugar beet industry a brief history is presented.

One objective of the experiment station (1892 annual report) was to determine the adaptation of sugar beets in Washington. Chemist E. Fulmer (1894) reported that beets grown in Washington were high in sucrose. He pursued beet trials for years, with sustained enthusiasm and the Utah and Idaho Sugar Company became interested. K. Fujinuma produced 80 tons of beets on two acres at North Yakima and W. L. Chamberlain produced 111 tons on three acres near Sunnyside in 1917 (p. 104 in "Beet Sugar in

the West," by L. J. Arrington, 1966, Univ. of Washington Press, Seattle). Sugar factories were started at Toppenish and Sunnyside in 1918. The early growing season was dry and viruliferous leaf hoppers migrated into the beet fields from the surrounding desiccating vegetation. Curly top was so severe only 4000 of 9700 acres were harvested and the average yield of the harvested acres was 3.5 tons per acre. Work on the factories was stopped.

Hoping that the plague would not return, work on the Sunnyside and Toppenish factories resumed. They were completed in 1919 but curly top struck again. The Sunnyside factory was dismantled and shipped to Raymond, Alberta, in 1919. The Toppenish factory was moved to Bellingham in 1924 where it was never profitable. The acreage of sugar beets in Whatcom and Skagit Counties seldom exceeded 3000, too few to make a factory worthwhile even though curly top was no problem west of the Cascades. A factory completed in 1917 at North Yakima was moved to Chinook, Montana, in 1918. The losses in moving factories around, as well as to farmers, must have been staggering.

Running from curly top would not solve the problem and efforts to develop resistant varieties began. In 1923 J.W. Timpson of the U and I company selected 1200 beets in diseased fields in the Yakima Valley and seed from these beets was sent to Eubanks Carsner (USDA) at Riverside, California. In 1926, 1500 beets were selected in devastated fields in southern Idaho and Utah and seed from these beets joined the collection at Riverside where the resistance work was concentrated. In 1928 seed of the best beet was sent to Twin Falls, Idaho for field testing. Seed of 400 mother beets of the best type, sugar content, and curly top resistance was bulked to make US No. 1 in 1929. Seed increase was rapid and in 1934 950 acres of US No. 1 were grown in the Yakima Valley. The beets did well even though 1934 was a severe curly top year. The Bellingham plant was still running and the beets were processed in it. US No. 1 survived in 1935, one of the worst curly top years known, and in 1936 plans were made to build a factory at Toppenish.

Most farmers were unwilling to grow sugar beets because of their previous losses. To counteract this resistance, 12 farmers from the Yakima district were taken in 1936 at the expense of the Northern Pacific Railway to see the success of resistant beets in the Salt Lake Valley. In 1937 beets again became a commercial crop. In 1947 the Toppenish factory ran continuously for 182 days, a record for any intermountain sugar factory. The Toppenish district produced 650,000 tons of beets with an average yield of 23 tons per acre in the early 1960's. Beets more resistant to curly top, higher yield and richer in sugar replaced US No. 1.

With the completion of the Grand Coulee Dam irrigation water in the Columbia Basin project began to flow in 1952. Beet yields in this region (Moses Lake, Othello, Warden, Quincy) exceeded those of the Toppenish district. In 1963 beets in the Columbia Basin project average 27.2 tons per acre, the highest yield in the nation in a district this large. The factory at Moses Lake, built in 1953, had by 1963 been expanded to a daily capacity of 6,250 tons of beets per day. Several years of low sugar prices led to abandonment of sugar production in Washington. But the enthusiasm of Chemist Fulmer about the adaptation of sugar beets to Washington was well founded, provided curly top was brought under control by resistant beets.

Ray Clark studied the vector, *Circulifer tenellus*, in the Prosser region. It has 2-3 generations a year here in contrast to 5-6 generations in parts of the American southwest. W. E. Howell under the direction of G. Mink, surveyed viruses other than curly top in beets in Washington fields in 1967-1969. Beet western yellows and beet mosaic viruses, both transmitted by aphids, were common. Beet mosaic was selected as the subject of Howell's thesis (MS, 1970). Beet mosaic virus is not seedborne and it has few hosts other than beets. Control is achieved by destruction of volunteer beets. The virus does not persist in the aphids, so the only real source of virus is infected beets. If beets are not allowed to overwinter there is no source of virus, a form of control by sanitation. Among Howell's recommendation were harvest all beets in the field and destroy volunteer and cull beets by any means. The green peach aphid (*Myzus persicae*) is the main vector, but it is more difficult to control than eliminating the source of virus. In seed



production, overwintering beets are essential, but Washington does not produce sugar beet seed, so control of mosaic was simple and inexpensive.

### *Curly Top of Vegetables, Especially Tomatoes*

The vector (*Circulifer tenellus*) survives the winter as adults in semi-arid regions, such as south of Prosser near the Columbia River. During winter on warm days the hoppers feed on various 'desert' plants from which they disperse in the spring. B. F. Dana (1918-1927) and J. C. Dodge (Acting State Extension Agent) reported in 1927 that curly top occurred anywhere in Washington depending on the winds, appearing occasionally in the Palouse, near Spokane, in the Puget Sound area, near Vancouver, but leaf hoppers do not overwinter there, and curly top is most important in irrigated south central Washington.

Dana and Dodge stressed that curly top was severe on tomatoes, most varieties of snap and dry beans, and some varieties of spinach, garden beets, Swiss chard, squash and pumpkin. Cabbage, onions, peas, asparagus, and rhubarb were not affected. Major efforts were made to develop tomatoes resistant to curly top by the USDA, Idaho, Utah, and Oregon experiment stations, but with little success. Dana and Dodge recommended setting more than one tomato transplant in a hill. Close-setting of tomato plants results in a greater number of healthy escapes. Leaf hoppers do not like tomato plants, do not increase on them, and they leave as soon as possible, accounting for the escapes. Resistant field beans, snap beans, squash and pumpkins were available. J. D. Menzies and N. J. Giddings (USDA workers at Prosser, WA, and Riverside, CA, respectively) determined in 1953 that 'green dwarf' of potatoes was caused by strain 12 of the curly top virus. The disease was sometimes severe in small plots near diseased tomatoes or sugar beets, but potatoes are not good hosts of the vector, and curly top is not an economic disease of potatoes.

Tomatoes are a major crop in the Sacramento Valley of California, even though the valley is in a curly top region. California workers attribute disease control to insecticides applied to the leaf hopper overwintering areas, direct seeding of tomatoes in the field resulting in relatively high plant populations, development of dense foliage as early as possible, and tomatoes with ability to escape infection (M. W. Martin and P. E. Thomas, 1986).

Evaluating tests for resistance to curly top in tomatoes, R. L. Clark (1968) planted one row of infected sugar beets to two rows of tomatoes, 1 spreader row of beets to 3 rows of tomatoes, 2:4, and 4:8. Diseased tomatoes decreased from 48% in the 1:2 planting to 19% in the 4:8 planting. When multiple rows of tomatoes were grown between the spreader rows, infection was 22% in center rows and 38% in border rows. These experiments are evidence of restricted movement of leaf hoppers among tomatoes. Clark also found that starting tomatoes early was advantageous because vector activity increased with the temperature. Clark found a high correlation between winter rainfall in the over-wintering sagebrush-weed vegetation and vector populations. Winter rain to support weeds increased the number of vectors in the spring.

The USDA supported an intensive effort to breed tomatoes resistant to curly top, assigning a plant pathologist, P. E. Thomas, and a plant breeder, M. W. Martin, to the task at Prosser. This project resulted in publications from 1969-1986, and to the development of highly resistant tomatoes (Rowpac, Roza, Salad Master, and Columbia). These varieties were released to seed companies. In spite of these efforts, no canner has elected to initiate a commercial operation in Washington. Thomas and Martin (1971) found that cultivars differed in the length of time leaf hoppers would spend upon them, leading to differing numbers of escapes due to nonpreference as plants upon which to feed. Thomas (1972) found that hoppers 'fed' on nonpreferred tomatoes 25-50 minutes, after which they left. In contrast, the hoppers seldom left sugar beets. The hoppers could not distinguish tomatoes from sugar beets until they 'fed' upon them. When leaf hoppers are confined on tomatoes (Thomas and R. K. Boll, 1977), they begin to die within 12-16 hours, and by 72 hours few were alive. Leaf hoppers fed only water died at about the same rate. Those receiving a 3% sugar solution lived up to two weeks. Apparently tomatoes really aren't

hosts at all, as the evidence is that they received no food from tomatoes even though they probed them and transmitted curly top virus to them.

#### *Witches' Broom of Alfalfa*

Heald and R. H. Wellman reported witches' broom of alfalfa in Washington in 1938. Jim Menzies made witches' broom the subject of his thesis (1946). It was prevalent and serious in the Methow Valley, a small irrigated valley bordered by arid, sagebrush-covered hills. Infected plants at first produce many fine stems with many leaflets, and farmers thought it could result in superb hay. The trouble was that the plants declined in vigor and died in a few years. Menzies believed the production of so many stems exhausts the reserves in the root, leading to little resistance to cold during the winter. Seed production is essentially eliminated.

The 'virus' could not be transmitted mechanically so Menzies doubted that it was spread by mowing and it was difficult to transmit by grafting. It was transmitted by a leafhopper, *Platymoideus acutus*. Ed Klostermeyer (entomologist at Prosser) and Menzies studied vector-'virus' relationships (1951) and now referred to the vector as *Scaphytopius dubius*. Using the vector they found that four *Medicago* spp., three *Trifolium* spp., *Melilotus alba* and members of four other genera of legumes were hosts. The time from inoculation until symptom expression was so long they found no annuals with symptoms. The 'virus' persisted in the vector. It is probable that the causal agent is the mycoplasma found to infect alfalfa in Australia (1969) long after Menzies worked on this disease and before mycoplasmas were discovered in plants.

Menzies established a 1/2 acre of alfalfa seeded broadcast and 1/2 acre seeded in rows two feet apart at Prosser, side-by-side, at the same time. After five years 31% of the alfalfa seeded in rows was diseased, 0.25% of that in the solid planting. The disease spread in older alfalfa fields in the Methow Valley, particularly as the stands became thin. Menzies concluded that witches' broom could be controlled by destroying all old, unthrifty fields and reseeded them to establish thick, dense stands. The farmers followed this recommendation on a community basis after 1946. In 1951 Menzies could not find witches' broom in the Methow Valley, even where fields previously had 50-70% infection, even in plantings three and four years old. Destruction of diseased fields and replacing them with new solid plantings led to virtual disappearance of witches' broom. Menzies felt certain the vector did not like shady, dense stands, that there was no large natural reservoir of witches' broom 'virus' in the miscellaneous plants in nonagricultural land adjacent to the Methow Valley, and that there was no evidence that the vector dispersed as widely as the curly top vector. His observations are classic in my opinion.

#### *Hops*

The use of hops in the brewing industry is reviewed by D. C. Smith in the 1937 Department of Agriculture Yearbook, p. 1215-1241. Either the Russians or Germans first used hops to flavor beer, at least as early as 768 AD. Several monasteries in Europe became famous for the beer to which hops had been added. It only takes from a half to a pound of hops to make a 31-gallon barrel of beer, so the quantity of hops required is small compared to the quantity of barley. Hops, though of high dollar value, occupy relatively small acreages. Hops were grown in New Netherlands (= New York) in 1629 and in Virginia in 1648. In 1849 about 1,500,000 pounds of hops were produced in the U.S., 70% of which were grown in New York. Hops were important in the Puyallup Valley before the WWREC was started, but downy mildew drove them out. In 1883, E. Meeker of Puyallup published, "Hop Culture in the United States." Being a practical treatise on hop growing in Washington Territory from the cutting to the bale. The Meeker home is a historical landmark in Puyallup. By 1909 California and Oregon together grew more hops than New York. In 1935 Oregon was the leading state and hops were no longer important in eastern U.S. Blue mold and downy mildew were largely responsible for removing hops from humid to irrigated, arid land in the U.S. Before modern systemic fungicides, hop growers in Bavaria sprayed hops as many as 10 times a season to control downy mildew. Downy mildew became important in California, further accelerating the shift of

hops to the Yakima Valley of Washington. The Washington hop crop in recent years is worth about \$90,000,000, and Washington produces 77% of the U.S. total. In 1915 Washington had 2,000 acres. In 1981 hops were grown on 31,000 acres, and yields often exceed 2000 lbs per acre, about double those in most hop regions of the world. A good part of the harvest is exported.

*Pseudoperonospora humuli* is the most important foliage pathogen of hops (*Humulus lupulus*) and even though high humidity is important, downy mildew can be significant in the Yakima Valley (D. A. Johnson and C. B. Skotland, 1985). Hops are perennial plants, and Skotland (1961) found that the fungus overwinters in infected crowns and roots, to appear on greatly stunted, deformed and chlorotic shoots early in the spring. It does not form oospores in Washington. Sporangia form on the lower leaf surfaces. They mature in early morning hours in response to night-time cooling and increased humidity. In most seasons losses are slight. Even though favorable weather may prevail in May, it normally turns hot and dry soon thereafter, stopping the development and spread of the fungus. Control involves removal of infected plants and application of fungicide when necessary.

Johnson and Skotland found 65% to be the minimum relative humidity for sporangia to form. In Washington very cool nights restrict sporangium production, so both temperature and humidity are normally unfavorable. In addition, free water on the foliage for at least three hours is required for infection. With rill irrigation conditions seldom favor downy mildew. Close monitoring of inoculum production within the hop yard and weather records led to effective, economical control, stopping the use of fungicides when unnecessary. It has been unnecessary to breed hops resistant to downy mildew for the Yakima Valley, and most of the varieties are highly susceptible. The ability to economically protect susceptible vines from serious losses with systemic fungicide (metalaxyl, D. Johnson and W. L. Anliker, 1985) is a great advantage to local hop breeders. They can concentrate on yield and quality.

Hops are propagated vegetatively from vine (bine) cuttings. If vines containing viruses are used, the plant produced from them are infected and infected plants with no or mild symptoms can be propagated unknowingly. Even though no real symptoms are visible, yields from such plants are significantly reduced. Hop mosaic and hop latent viruses are symptomless in commercial hop varieties, but the American hop latent virus produces tiny yellow flecks in some cultivars. The absence of distinct symptoms makes roguing difficult. Cal Skotland suspected that many cultivars were infected with various of these systemic, insidious viruses, and he began to test them for virus infection by various techniques, including the ELISA test. He produced virus-free clones, primarily by heat treatment, tested them for virus infection, yield and quality, and released virus-free clones for commercial production. Much of the yield advantage of Washington hops is directly traceable to the work of Skotland at the Irrigation Research and Extension Center. It is possible that his work increased the dollar value of Washington crops more than that of any other pathologist in this state.

For virus-free materials to be of lasting value in a perennial crop, virus spread must be limited. The hop aphid is a suspected vector of hop mosaic, hop latent virus, and American hop latent viruses. Prunus ringspot virus is spread mechanically by plant contact (Maloy, Handbook of Washington Plant Diseases). Hop yards should be monitored for virus spread or the gains of the past will be lost.

Virus-free plants are obtained through tissue culture or heat treatment. Virus-free clones are grown in a propagation yard and from it they go directly to the grower. Washington has the only hop certification program in the U.S. Skotland says that Earle Blodgett, an expert in propagation and certification of virus-free propagating materials, helped. Some hops grew on land deficient in zinc, and zinc deficiency symptoms are similar to those of prunus necrotic ringspot, complicating the situation. Farmers spraying with Zineb, presumably controlling downy mildew, noted improved growth. Most hops in the Yakima Valley are now sprayed with zinc, so this complication no longer exists.

E. G. Probasco (M.S. thesis, 1971) is the only graduate student to have worked on hop diseases in Washington. Probasco purified a virus with long, slender particles that produced no symptoms in most

hops. The virus was very unstable, losing infectivity rapidly in juice from infected plants. Prunus ringspot virus had been identified in hops by Skotland (1969). Basic research on hop viruses has been limited, but applied research has been outstanding.

### *Asparagus*

In 1923 asparagus was grown on 450 acres, in 1984 asparagus was grown on 29,000 acres, and Washington produced 60% of the processed asparagus and 21% of the fresh market crop of the US, second in total population. Commercial asparagus is grown on light soils in the Columbia Basin under irrigation, with Yakima, Franklin and Walla Walla Counties producing the most. The crop in 1984 brought \$37,454,000 on the farm. Climate, soil and irrigation contribute to high yields and quality, making us highly competitive in the processing industry. Asparagus does best on land upon which asparagus has not been previously grown. If disease-free propagating material is used on new land, fields remain productive for several years, even for 20 years in some cases. Establishing a field with roots is expensive. The longer a field remains productive, the more profitable it will be. The most important contribution to asparagus growers by plant pathologists is to extend the productive life of fields.

In 1942 S. I. Cohen (Ph.D. thesis) and F. D. Heald reported that *Fusarium oxysporum* f. sp. *asparagi* was an important fungal pathogen on sandy soils of the lower Yakima Valley, with as many as 25-50% diseased or dead crowns in some fields. *F. oxysporum* produces long-lived chlamydospores that infest soil for many years. Cohen and Heald attempted to get pathogen-free planting material and failed.

Mel Grove (Ph.D. thesis, 1976) and Otis Maloy found that both *F. oxysporum* and *Fusarium moniliforme* were pathogenic to asparagus. *F. oxysporum* was isolated most frequently from diseased plants and most of the isolates were pathogenic. All isolates of *F. moniliforme* were pathogenic. Seedlings grown in naturally infested soils were yellowed, stunted, wilted and they had many rotted roots. In most of the problem soils 50% of the seedlings died within four months. It would be difficult to start asparagus from seed in such fields. *Fusarium* spp. were isolated from many seeds, and from some seeds even after surface disinfection, so some seeds had fungi within them. Pathogens present on seeds can spread in the nurseries and many of the roots used to start new commercial fields would introduce the pathogens into the 'clean' soil.

Debra Inglis (MS thesis, 1978) and Maloy found *F. oxysporum* only on a few seeds, but many carried *F. moniliforme*. The seed coats are rough with cracks and crevices in which spores are trapped. Asparagus seeds are extracted from the berries by pulping (mashing) the berries and soaking the pulp in water, after which the debris is screened from the liquid mass. Often the berries are stored for some time before extraction, and *F. moniliforme* and *Penicillium corymbiferum* sporulated on the surface of the berries. *Penicillium corymbiferum*, while not considered a major problem, was found pathogenic to asparagus by J. D. Menzies in 1948, causing a blue mold and seedling decline. Inglis concluded that during the pulping process fungal spores are spread within the fermentation liquid to what had been pathogen-free seed, so high levels of seed infestation could result. Seed of this type would give rise to infected plants, even if transplanted to clean soil. The obvious solution, since most of the fungus was external, was to kill fungi on the seed. Grove, Inglis and Maloy developed a hot water-benomyl treatment that destroyed both *F. moniliforme* and *F. oxysporum*, as well as any *Penicillium* that might be on the seed. Chlorox was later substituted as a more practical seed disinfectant. Clean seed and clean nursery soil will insure high quality planting roots, especially important with a high value, perennial plant like asparagus.

Recent pathology on asparagus is being done by Prosser personnel, why this account is presented under Prosser even though much work was done at Pullman.

### *Fruit Tree Propagation*

The production of vigorous, healthy nursery stock is critical in establishing and maintaining productive orchards. Pathologists normally concern themselves with producing pathogen-free trees, but in Washington they contributed to tree propagation. The need for virus-free nursery stock was recognized when James D. Menzies, still a graduate student, was sent to Prosser in 1939. Menzies worked mainly on alfalfa, however. In 1946 Dr. Earle C. Blodgett was hired jointly by the Washington State Department of Agriculture and WSU to work at Prosser on virus-free pome and stone fruit trees, especially those used as sources of bud wood or scions. It was soon found, however, that some of the best rootstock - scion combinations were difficult to propagate. In response to this need, Shirl O. Graham was sent to Prosser in 1948 to work with Blodgett.

Graham improved the rooting medium, the use of growth regulators to initiate rooting, and devised rooting chambers that maintained favorable humidity, temperature and light. In high humidity, favorable for rooting, cutin did not form properly on above ground parts. The above ground part of the cutting was dipped in a dilute emulsion of paraffin to restrict transpiration. Foliar applications of nutrients and adenine increased the number of roots per cutting. Graham (Ph.D., 1954) improved rooting in *Prunus mahaleb*, *P. avium*, *P. persica* and *P. cerasifera*, rootstocks normally difficult to root. Studies of rooting are not typical activities of a plant pathologist, but they aided the nursery and fruit industry. Graham joined the faculty at Pullman where he did physiological research and taught methods and physiology.

In 1955 Murit Aichele was employed by the Washington Department of Agriculture to work full-time with Blodgett on the virus-free nursery stock program. After several demonstration plantings of diseased and healthy trees in side-by-side comparisons, the nursery industry saw the real value of the efforts. A committee devised legislation to enforce recommendations, and in 1963 a state law established legal procedures. Trees from the program bear a special certification tag, not guaranteeing them free of virus, but guaranteeing that all known methods to prove them virus-free had been followed. The Washington nursery certification program is the best in the nation, and it formed the basis of Federal regulations. A 1% assessment on retail sales nursery stock supports the certification program.

### *IR-2 Program*

Virus diseases (or mycoplasmas or rickettsia-like bacteria) of stone fruits, particularly peach yellows were so serious a prize was offered for yellows prevention in 1796 at Philadelphia, Pennsylvania (Plant Dis. 64:826, Paul Fridlund), and more problems were recognized through the years. Finally, in the early 1940's, an approach to a systematic, sustained attack began to be formulated. The effort in Washington, particularly by Earle Blodgett, was outstanding, and it attracted attention. Also, it was concluded that the climate at Prosser was suitable for growth of many types of fruit trees. The IR-2 (InterRegional) project began July 1, 1955, funded by the USDA through CSRS (Cooperative State Research Service). Paul Fridlund was its first administrator and scientist. The facility included a greenhouse, five screenhouses to exclude aerial vectors, and 19 acres of irrigated land at Prosser. A twenty acre irrigated plot near Moxee was used to maintain known virus-free trees in isolation. The Moxee site became useless as irrigation spread and varied plants, including fruit trees, were grown closer and closer to the Moxee site.

In 1984 the IR-2 project received about \$240,000 from the USDA to cover all salaries and expenses, including the salary of Fridlund (1955-1987), and now Mink. This annual grant is solely for IR-2 work. IR-2 is guided and governed by an Interregional Technical Committee with members from all participating regions in the US, the USDA, and the Washington State Department of Agriculture. Consultants include a representative of Agriculture Canada. In spite of this complex guidance mechanism, Fridlund, and now Mink, are employees solely of Washington State University and advancement and salaries are governed by WSU.

WAES Circular 401 covers the early history and activities of the IR-2 project.

### *Bee Management in Sweet Cherry Orchards*

W. E. Howell, Scientific Assistant, and Gaylord Mink (1988) reported a fascinating story. Prunus necrotic ringspot virus (PNRSV) is transmitted rarely by infected pollen. One strain causes cherry rugose mosaic, a severe disease. After the certified virus-free nursery stock program was initiated (1961), along with removal of any tree with rugose mosaic and use of local bees for pollination, the disease was controlled. Sometime during the mid-1970's cherry rugose mosaic began to spread. About this time bee keepers trucked their bees to California, where cherries bloom earlier, then trucked them back to Washington, enabling them to do double duty. In some cases the bees were in California at 5 p.m. and in Washington orchards the next day, so they were moved very promptly from California to Washington. Mink detected pollen with infectious virus on bees emerging from hives after shipment from California. With this knowledge, bees used in California were held two weeks before release in Washington orchards. The virus loses infectivity in the pollen rather rapidly and this holding practice stopped the introduction of infectious virus from California into Washington.

While monitoring for PNRSV in cherry trees by the ELISA test, PNRSV was found in symptomless trees. Virulent and avirulent strains spread at the same rate, to about 10% of adjacent trees per year, so virulent and avirulent virus were equally infectious. Detailed studies over several years proved that the avirulent virus gave excellent cross-protection against the virulent strain, and studies are in progress to determine whether it will pay to infect all cherries with the avirulent strain to achieve biological control.

Gaylord Mink received the Lee M. Hutchins Award for contributions to the tree fruit industry, largely because of this research. For details see *Phytopathology* 79:57.

### *Seed Treatments for Peas*

Washington leads the nation in production of dry peas for soups, export, and seed, and is second in processing (green) peas. Pullman is in the center of the dry pea industry but it is surprising how little pathology on peas has been done here. A noteworthy exception is the PhD thesis of Max Schuster, initiated under J. G. Harrar, continued under E. J. Anderson (fall 1942, completed May 27, 1946), and published in 1948 as Bulletin 499, "The etiology and control of root and stem rots of peas in Washington." The Blue Mountain Canneries, Inc., of Dayton, WA (= Green Giant) supported the research and its technical staff gave considerable guidance.

Schuster started by isolating fungi from diseased seedlings and older peas and concluded that *Fusarium solani* f. sp. *pisi* was the most important pathogen, as it is today. I can't help but wonder what his isolation techniques were because he did not (obtain?) work with *Pythium* spp. at all.

Peas in commercial fields are drilled solid, requiring considerable quantities of seed, a major expense in pea production. Schuster concentrated on fungicides to reduce loss of seedlings and to increase yield. His field experiments were at Pullman and in the Blue Mountain pea region, from Dayton west to Walla Walla. The Blue Mountain section is favored for green peas because peas can be grown from different seeding dates in fields differing in elevation, producing green peas over a wide period of time resulting in a long processing season and efficient use of processing facilities.

The seed treatments included coppers, sulfurs, mercuries, and the then new Arasan and Spergon. Both Arasan and Spergon were superior to the other treatments, but Arasan was either toxic or irritating to some workers, so Spergon became the favored material. Captan was not yet available.

Schuster determined the best dosages, proved that Spergon did not reduce nodulation of peas treated with nitrogen-fixing bacteria, and that treated seed could be stored for a year or two without reduced

germinability. He treated a wrinkled pea (Green Giant), and a smooth pea (Alaska). The rough-seeded pea was more prone to stand problems than the smooth-seeded pea. Emergence of one-year old rough peas untreated was 44%, two-year old seed, 25%.

Injuries to the dry seed were observed visually, or after soaking in iodine solution or after soaking in water. Visual examination of dry peas did not disclose some injuries to the seed coat. Blue areas in the seed soaked in iodine revealed injuries, but water soaking was equal to the iodine treatment, so water soak was established as the method for detecting injuries. Schuster emphasized that injuries occurred during threshing and probably during drilling, but the latter was not studied. Very wrinkled seed, even hand-threshed, had some injured seed coats, the result of distortion pressures during shrinking during natural maturation and drying. Wrinkled seed gave a stronger fungicide response than smooth seeds. When the seed was threshed by hand there was little response to seed treatment, demonstrating the importance of wounds caused by machine.

Water did not remove much of the protectant from the seed in greenhouse pot trials, when pots were watered immediately after planting. In field tests with wrinkled seed results varied greatly as to location, season, and seeding depth. In general, adverse conditions during germination and emergence increased response to seed treatment. The treatments were equally effective, whether applied by machine or "by hand" (experimentally), so his results were applicable to commercial operations. Seed treatments increased yield, whether weeds were present or not. The seed treatments did not control subsequent damage to the epicotyl or to the roots, so the benefit from these fungicides was limited to protecting the seed and seedlings during emergence.

I found this study typical of many early publications. The bulletin had six figures and 30 tables. Today this work would have been presented in two or three journal articles, not in a single comprehensive publication.

#### *Resistance to Pea Root Rot*

John Kraft, USDA pathologist, has worked for years to increase resistance in *Pisum sativum* to *Pythium ultimum* and *Fusarium solani* f. sp. *pisi*, both of which are important. *F. solani* appears to be especially severe in the PNW. I remember being called to Walla Walla by the county agent to meet with pea processors and pea growers about pea root rot, mainly *F. solani*. I knew nothing about it but no one else was available, so I consulted Walker and found that rotations of five years between pea crops were recommended. I reported this to those present. It was obvious they didn't want to hear anything about long rotations. Peas are still grown, mainly in three-year rotations, leading to the conclusion that resistance is the main hope.

At first Kraft and Roberts (1969) determined how soil temperature and moisture, alone and in combination affected disease produced by both pathogens, alone and together. In cool wet soil *P. ultimum* was favored; in drier, warmer soils *F. solani* was favored. Under moderate temperature and moisture the worst disease occurred when both pathogens were present. Next (1973) they identified peas resistant to both pathogens. Some lines are so resistant, seed treatments with metalaxyl, *Trichoderma* spp., or Captan did not significantly increase yield in infested soil favorable to both pathogens (Kraft and George Papavizas, 1983). These results prove the great potential of resistance.

An interesting example of the complexity of developing resistant varieties is seen in a study by Randy McCoy and Kraft (1984). Epicotyl damage by *Rhizoctonia solani* was severe on many selections resistant to *F. solani* and *P. ultimum* and light on Dark Skin Perfection, the commercial cultivar used as a susceptible standard in the above root rot studies. Resistance to *R. solani* was greatest when the epicotyl was thick and least with thin epicotyls (very high negative correlation, -0.91, between thick hypocotyls and disease severity). The correlation was so reliable they predict that resistance to

Rhizoctonia disease can be determined by measuring epicotyl diameters, that the pathogen will be unnecessary.

#### *Pea Seed-borne Mosaic*

In 1969 Gaylord Mink, John Kraft, John Knesek and Ali Jafri reported a seed-borne mosaic virus in pea fields in Washington and Idaho. Infected plants were highly deformed and unproductive. The virus is transmitted by the green peach aphid (*Myzus persicae*) and by the pea aphid (*Acrythosiphum pisum*) as well as mechanically and through seeds of infected plants. All commercial pea cultivars were susceptible. The virus infected several legumes, including vetches, sweet peas and lentils. It also infected New Zealand spinach, periwinkle, globe amaranth, beets, swiss chard, and lambsquarters. This discovery caused alarm. J. E. Knesek, (PhD, 1973) under the direction of Mink, determined its host range, the number of seeds per plant that carried virus, and the physical properties of the virus itself. Mink subsequently developed means of assaying seed lots for the virus, leading to practical control by using only seed free of virus. Apparently there is no natural reservoir of virus in the vegetation of the region, in spite of its wide host range.

#### *Bean Root Rot*

When land in the Columbia Basin Project was first brought under irrigation, dry beans were a favored cash crop because of very high yields on virgin soil. In many cases farmers getting started were unduly pressed for money, leading them to grow beans repeatedly on the same land without rotation. Bean yields decreased rapidly in many fields, and J. D. Menzies (1952) documented this yield decline. In contrast, 'old' cultivated land in the Columbia Basin was not subject to this rapid decline in yield. The difference in response of beans to recropping on new and old cultivated land was so marked D. W. Burke (Ph.D., 1955), under the direction of G. W. Fischer, attempted to determine its cause. At the time Burke worked it was possible to collect similar soils that had been cropped for several years and adjacent uncropped soils. The difference between these 'old' and 'new' soils was caused by living organisms in the soil, not by differing structural, textural, or chemical soil properties. Cropping (alfalfa, asparagus, pasture, etc.) altered the soil microflora significantly from that of the sage-brush, sparse vegetation of virgin soils. The main pathogen of the beans was *Fusarium solani* f. sp. *phaseoli*. When equal numbers of the pathogen were introduced into old cultivated soil and virgin soil, bean root rot was more severe in virgin soil. Burke inoculated 'old' and 'new' soils and found that in old soils unfavorable to disease the fungus grew extensively in the soil but that it formed small, weak chlamydospores. In soils favoring disease the fungus grew little but formed large, vigorous chlamydospores. The strong chlamydospores survive longer and produce more vigorous germlings, indicating that in old soil the microflora permitted the fungus to extend itself considerably, but that this was actually weakening it, reducing its ability to survive and accumulate. Soils that had produced several crops of miscellaneous plants were relatively free of the disease. Burke was unable to determine the specific cause of the suppression of bean root rot, but his observations of chlamydospore (resting spore) biology are pioneering studies in soil microbiology. In retrospect, much loss could have been prevented if the farmers with new land could have been convinced of the necessity of rotating crops on this virgin soil. Once soil is heavily infested with *F. solani* it is almost impossible to clean the soil. Prevention is the best control.

Burke found that, on soils infested with *Fusarium solani* f. sp. *phaseoli*, early planting (1964) and heavy stands (1965) resulted in highest yields. He found that the pathogen did not spread easily from roots to the hypocotyl of the same plant, that it did not spread to any extent from one plant to another within the bean row, even when plants were as close as 1/2 inch, and that water did not move spores far within soil. He considered the fungus essentially immobile in soil. This means that severe disease depends upon a high population of thoroughly distributed spores within the soil to initiate many primary lesions.

By 1969 Burke, V. E. Wilson, and W. J. Zaumeyer released Big Bend, a Red Mexican bean particularly adapted to the Columbia Basin. It was early maturing, resistant to bean mosaic, and had a significant



level of resistance to bean root rot. On clean soil UI-36 outyielded Big Bend. On root rot soil Big Bend outyielded UI-36.

Burke (1968) found that in some soils tillage compaction at plow depth prevented bean roots from growing beneath the plow layer, restricting the roots to the tillage layer. A restricted root system made the plants more subject to water stress and to bean root rot. By 1972 Burke and D. E. Miller, a USDA soil scientist, recommended subsoil tillage directly beneath the bean row at seeding time to improve the root system and reduce root rot. Subsoiling infested soil increased the yield of Royal Red, a susceptible bean, from 927 kg/ha to 2475 kg/ha, illustrating the importance of subsoil tillage. Big bend yielded 2251 kg/ha without subsoil tillage, 3838 kg/ha with subsoil tillage. Note the large increase when resistance and subsoil tillage are combined: 927 kg/ha Royal Red not subsoiled, 3,838 kg/ha Bigbend subsoiled.

When wheat is grown under some conditions without rotation for several years, take-all, a root disease, becomes very serious in the first few years, then declines. This is called take-all decline. When beans were grown 15 years and peas 6 years on the same soil, root rot was severe. No decline occurred (Burke and J. M. Kraft, 1974). The pea pathogen (*Fusarium solani* f. sp. *pisii*) is very similar to the bean pathogen, but the bean pathogen (f. sp. *phaseoli*) did not damage peas, and the pea pathogen (f. sp. *pisii*) did not damage beans, suggesting that beans and peas, even though both legumes, could be grown in the same rotations.

Occasionally, especially in rill irrigation, parts of fields are temporarily flooded. D. E. Miller, Burke and J. M. Kraft (1980) found that even short periods of flooding produce an oxygen shortage in the soil and that oxygen stress for 72 hours predisposes plants to root rot. Plants flooded for short periods in pathogen-free soil recover quickly with no permanent damage.

Burke and coworkers developed integrated controls for bean root rot, including rotation, subsoiling beneath the row just before planting, proper fertilization, resistant varieties, seeding early and relatively heavily. Plant numbers are better increased with narrower rows than with more plants within the row.

#### *Bean Quality*

Much of the dry bean (*Phaseolus vulgaris*) produced in Washington are used as seed or canned. Both markets respond strongly to seed quality: for seed, high germinability and seedling vigor; for processing, clean beans that are not broken or cracked. Matt Silbernagel and Doug Burke (Bull. 777, 1973) described from cutting the vines to final sale, how to produce a maximum quality dried bean, emphasizing moisture content and reduction in physical injuries. Silbernagel cooperated with S. R. Drake and R. L. Dyck (1984) in a study that proved that optimum irrigation and subsoiling produced the highest quality green snap beans. High quality meant the beans had low shear values when cut during processing, and lost less dry matter during blanching and draining.

#### *Potato Scab Suppressive Soils*

Jim Menzies was at Prosser when the virgin lands of the Columbia Basin Project and some other irrigation projects were brought under cultivation. He observed that scab was more severe on potatoes grown in the new land than in land that had been cultivated for several years, even though the soils were similar chemically and physically, and even though the same cultivars were grown with the same management. He filled beds at Prosser with similar cultivated and virgin soils and grew potatoes from infected seed in them the first year. Subsequent potato plantings were from clean seed. Infected seed resulted in severe scab, regardless of the soil history, so temperature, moisture, oxygen, etc., and cultivar favored scab. In subsequent years, replanting with clean seed, scab was slight in the "old" soils, severe in the "new" soil. Menzies found that the old soils possessed a "disease suppressive" power that was destroyed by heat or sterilants; it was biologic in nature and it was transferrable from the old to the new

soil. To my knowledge the organisms responsible for this disease suppression are still unknown, but this pioneering research (1959) has stimulated many studies on "suppressive soil".

### *Shipping Potatoes*

Losses in shipment of fresh potatoes to distant markets can be severe. Shipping potatoes in refrigerated rail cars had been studied for many years, mostly by federal pathologists in the marketing service, and mostly long ago, but an effort to improve the system was made recently. Between 1965 and 1969 about 5% of Washington potatoes rotted in transit to the Eastern Seaboard. Five percent is a lot of potatoes to dispose of at the terminal. Most of the rot was bacterial soft rot and *Pythium ultimum* 'water rot'. Bob Cromarty (Ph.D., 1970), working with Gene Easton, studied the effects on rot of pre-chilling both the potatoes and the boxcars, reducing temperatures as rapidly as possible before shipment. If potatoes enter cars warm, even though the cars were iced after loading, temperature of potatoes within the car remained high for at least 24 hours. Potatoes in burlap sacks had no oxygen problems but prompt refrigeration was especially important when potatoes were shipped in cartons. High respiration by the potato and restricted ventilation within the carton predisposed the potato to rot because of incipient anoxia.

If potatoes were packed in cartons wet, just after washing, rot increased, primarily due to increased bacterial growth. Surface disinfecting tubers, however, increased rot, leading Cromarty to believe that many of the bacteria on the tuber surface restricted growth of pathogens. Cromarty concluded that rot started at the tuber surface, especially in wounds.

### *Phloem Necrosis in Russet Burbank Potato*

The potato leaf roll virus (PLRV) is spread by aphids. PLRV is systemic in the phloem, moving to all parts of the plant. When a healthy plant is infected, no external symptoms develop the first season (called the current season), but when tubers from such plants are used for seed, the crop from infected tubers develops stunted, thickened, curled leaves (= leaf roll) and severe yield and quality losses occur. In the current season crop, however, even though the foliage is normal, darkened phloem (vascular tissue) within the tuber (= phloem necrosis) may develop in storage. Discolored vascular strands ramify through the normal white tissue and they cannot be removed by peeling. Tubers with extensive phloem necrosis are unfit for sale as fresh market potatoes or for processing. Leaf roll can be controlled by use of virus-free seed and aphid control, but when studied by Avery Rich, Ph.D. 1950, it was so serious in Russet Burbank that many growers were forced to grow varieties of inferior cooking quality that did not develop phloem necrosis in current season infections.

Avery Rich found that the earlier the infection in Russet Burbank, the more severe the effects. The greatest incidence of phloem necrosis occurred in Grant, Yakima, and Kittitas Counties, and the percent of tubers with phloem necrosis increased during storage. In one sample from Prosser, 12% of the tubers had phloem necrosis in October, 27% in November, 40% in December and 60% in January. The temperature of storage affected the development of phloem necrosis, but for practical purposes the storage temperature could not be varied enough to significantly limit the progress of the net necrosis within the tuber. Treating the tubers with germination inhibitors did not stop development of phloem necrosis during storage. Phloem necrosis was less severe in early-planted potatoes than in late plantings, and early harvesting sometimes reduced the damage. Fertilizer treatments had no effect. Avery concluded his study by recommending virus-free seed, aphid control and resistant varieties. He firmly established the relationship of current season infection with phloem necrosis in Russet Burbank.

Avery reported 0 spread of leaf roll in 1949 at Long Beach, Lynden and Port Angeles, all in western Washington; 0 spread at Coupeville, Colville, Spokane, Port Angeles, and Lynden in 1948. Greatest spread occurred at Pullman and Prosser in 1948 and in Prosser in 1949. Trials of this kind are useful in that they suggest places for the production of virus-free seed potatoes.

In spite of much effort by Seth Locke and others, leaf roll is still a problem. A crop that started virus-free but became infected with leaf roll virus is symptomless at harvest. Peter Thomas developed the use of the ELISA test to sample healthy appearing potatoes. If infection is significant, the potatoes can be sold fresh or processed rapidly, before net necrosis develops in storage, eliminating the loss.

#### *Root-knot of Potatoes*

Second-stage juvenile of *Meloidogyne chitwoodi* (Columbia root-knot nematode) and *M. hapla* (northern root-knot nematode) penetrate potato tubers through lenticels and develop into females and deposit eggs in an egg mass within the vascular ring. During this process pimple-like bumps develop on the surface of the tuber infected with *M. chitwoodi* but not *M. hapla* and brown spots about the egg masses develop within the tuber. Such tubers may be rejected by processing plants because the blemishes are not removable by peeling. A condemned crop is a great loss to a grower. The potato processing industry, which utilizes about 80% of the Washington crop, uses a grading system for nematode injury developed at Prosser. Usually tubers with six or more nematodes (internal brown spots) are rejected as culls.

In the early years of the Columbia Basin Project *M. hapla*, found in Block 73 near Quincy in 1960, was the only root-knot nematode known on potato. The host range of *M. hapla* was such that it could be controlled by rotation with grasses (oats, wheat, barley, corn, sorghum, etc.), so practical control was possible (Faulkner and Fred McElroy, 1964). In addition to rotation, about \$9,000,000 annually is spent on soil fumigation to control root-knot. In 1968, 3000 acres were fumigated. In 1970, about 25,000 acres. In 1984, 60-70% of potato land was fumigated.

The situation seemed sustainable with known controls, but failures of both rotation and fumigation began to be reported. A new nematode, *M. chitwoodi*, was described in 1978. This nematode increases on wheat, corn, oats and barley, the main economic rotation crops, making control by rotation essentially impossible (Santo, et al, 1980). Alfalfa is a none to poor host of *M. chitwoodi* race 1, but it is a good host of *M. chitwoodi* race 2 and *M. hapla*, so alfalfa is not much help. Soil fumigation is less effective against *M. chitwoodi* than against *M. hapla* because the *M. chitwoodi* completes more generations on potato than *M. hapla*. Thus, a higher degree of control is required for *M. chitwoodi* than *M. hapla*. In addition, *M. chitwoodi* is able to migrate upwards faster and farther than *M. hapla* (John Pinkerton, et al, 1987). Thus, deep placed *M. chitwoodi* may be able to escape fumigation and later reinfect the fumigated zone. In 1984 (Santo and M. Qualls of Stauffer Chemical Company, Ephrata) reported that soil-infected with 1,3-dichloropropene was the most common control, but metham sodium was gaining in use because it not only controls nematodes but it also reduces *Verticillium dahliae*. About 90% of our potatoes are produced using over-head sprinkler irrigation and metham sodium can be distributed in irrigation water pre-plant (in the fall prior to the next potato crop). This application, properly done, penetrates the soil deeply (at least 91 cm) so it greatly reduces *M. chitwoodi*. Both *M. hapla* and *M. chitwoodi* attack peas and beans (1985). Race 1 does well on carrots, race 2 does not (1988). The combination of these two root-knot nematodes, with their distinct biologic characteristics, poses a grave threat to our potato industry and a challenge to our nematologist, Gerry Santo.

#### *Nematodes in Irrigation Water*

The virgin soils of the Columbia Basin quickly became infested with many important plant pathogens, in spite of the presence of competent pathologists. Lin Faulkner and W. J. Bolander (1966) published a classic study of the role of irrigation water in spreading nematodes. They blame infested water in accelerating the spread of important plant-parasitic nematodes.

In the Columbia Basin, water is introduced in ditches at the upper end of the field, and excess water that flows from the lower end of the field is gathered in ditches from which it enters lower fields. If a field at the top of the sequence becomes infested, water from it passes successively from field to field until

discharged back into a river system. The buoyancy of nematodes is such that they do not settle in the moving water and they occur essentially uniformly distributed within the water. Their oxygen requirements are so low that they remain vigorous even after extended periods in the ditch water. Nematodes varied from 20 to over 200 per gallon of water, and in main ditches from 2 to  $16 \times 10^9$  nematodes passed a given point in a day. Plant parasitic nematodes were 10-20% of the total. They concluded that each time an acre of land was irrigated in the Lower Yakima Valley, from 4 to over 10 million plant-parasitic nematodes were introduced. These studies illustrate an important advantage of irrigating from wells, but mainly, they show the difficulty of preventing the spread of plant pathogens once the latter become established in a region.

### *Verticillium Wilts*

Verticillium wilts are treated under Prosser because of prolonged study at that station, but the first study of Verticillium wilt in Washington was at Puyallup by W. H. Lawrence. Black raspberries suffered from an unknown malady in the Puget Sound area (1904-1907). Lawrence named it bluestem and described the pathogen as *Acrostalagmus caulophagus*.

L. K. Jones and Glenn Huber (1934) reported a severe wilt of greenhouse chrysanthemums caused by *V. dahliae*; the first use of that name in Washington. Greatest losses occurred when chrysanthemum cuttings came from infected mother plants, but infections also arose from virgin forest soils of western Washington. They recommended healthy mother plants and sterilized soil.

### *Stone Fruits*

Earle Blodgett apparently observed Verticillium wilt in stone fruits in 1946, the year he came to Prosser, but *V. dahliae* was officially recorded when identified by Austin Goheen in 1949. Blodgett and J. A. Twomey, Horticulturist, by 1955 had established that Verticillium wilt was a serious problem in sweet cherry, apricot, peach and prune in the Yakima Valley. It was severe in 1962 and 1963. Bing cherries on Mazzard rootstock were particularly susceptible. The disease was most serious in young trees. It was minimized by management practices (proper pruning, fertilizing, watering, disinfecting soil in replant situations, clean propagating materials). *V. dahliae* has not been serious on stone fruit west of the Cascades, probably because of generally acidic soils there.

At about the time *V. dahliae* was receiving emphasis in stone fruits, Cal Skotland studied cantaloupe wilt. Cantaloupe wilt was severe the year Cal arrived (1956) and in 1957, 1959, and 1960. The summer of 1958 was very hot, with an average daily high of 32°C between June 9 and August 26, and Skotland believed heat suppressed the disease in 1958. Experiments with several fumigants demonstrated how hard microsclerotia are to kill, with only chloropicrin, methyl bromide, and isothiocyanate effective. Economic control was not attained because cantaloupes are no longer commercially grown in the Yakima Valley.

### *Mint*

Jim Menzies found Verticillium wilt on mint near Mabton in the Yakima Valley in 1955. It apparently had been introduced on native spearmint from Indiana. Native spearmint (*Mentha spicata*) is most resistant, Scotch spearmint (*M. cardiaca*) has some resistance, and peppermint (*M. piperata*) is most susceptible. The *V. dahliae* that attacks mint is a special mint strain, unlike the wide host range strain commonly spread by potatoes (the one that attacks cantaloupes, stone fruit trees, and many other plants). From about 1910 when mint was first commercially grown in the Yakima Valley until the mint strain of *V. dahliae* was established, mint fields were productive for 10-20 years. Mint is grown from vegetative cuttings, so establishing a new field is not cheap. The first crop is small and the first two or three years provide little profit. When wilt was recognized as important in mint, it became Skotland's problem.

Washington peppermint is not of superb quality and it does not command a premium price. Sufficient chloropicrin to kill *V. dahliae* is costly and, even with cuttings certified *V. dahliae*-free, fumigation of

infested soil has not proven economical. With a long-lived perennial host like mint, inoculum that is not killed increases with time.

Water from Grand Coulee Dam reached some 'new' soil of the Columbia Basin Project in 1952, but several years elapsed before much new land was brought under irrigation. An effort was made to prevent introduction of the mint strain into the new lands by requiring use of cuttings certified wilt-free. The effort failed. Not only was *V. dahliae* introduced, but the northern root-knot nematode as well, mainly by corporate farmers, according to Skotland. It is relatively easy, however, to maintain clean land with water from wells, certified propagation materials, and sanitation.

Faulkner, W. J. Bolander, and Skotland, 1969 and 1970, found that the nematode *Pratylenchus minyus* increased the susceptibility of peppermint to *V. dahliae*, even with one part of the split root system exposed to the nematodes, the other part to the fungus, evidence of a more fundamental relationship than just physical wounds made by nematodes as portals of entry for the fungus.

Before retirement, Skotland and C. E. Horner, Oregon pathologist, worked cooperatively to produce Scotch Spearmint as resistant as native spearmint by mutation breeding, using radiation. Murray, a private breeder in the midwest, had produced resistant peppermint by radiation and this encouraged the local workers.

#### *Potatoes*

Potatoes played a major role in disseminating and increasing the wide host range general strain of *V. dahliae*, mainly by microsclerotia on tubers and to a limited extent by internal infections of seed tubers. George Newton, 1927-1930, cautioned raspberry growers in Western Washington not to establish plantings following potatoes. Blodgett cautioned against planting stone fruits following potato, tomato, eggplant, peppers or melons. Even in 1990 Otis Maloy and Debra Inglis were warning strawberry growers of the danger of potato land. *V. dahliae* on potatoes in Washington is most important in the neutral to alkaline soils of the Columbia Basin.

It was inevitable that the new lands of the Columbia Basin became infested, especially when potatoes were grown with little or no rotation. Yields at first were very high, but with repeated monoculture yields and quality declined, leading to recognition of the early dieing syndrome and continuous potato plots were established at Prosser in 1958 to study the problem close-hand.

Recognition that tubers were the main means of introducing *V. dahliae* to clean land, Bill Hoyman sampled certified seed potatoes from several states and Canada in 1962 for internal infection and found low percentages in most seed lots. Even killing microsclerotia on the surface of the tubers, possible with seed treatments, would not completely protect against spreading the fungus. Gene Easton tested certified seed potatoes in 1968 and 1969, again from several states and Canada, for both external and internal fungus. It was clear that seed potatoes produced conventionally could not be guaranteed free of *V. dahliae* and the Washington State Seed Certification Program makes no effort (1982) to guarantee seed free of this pathogen. Propagules of *V. dahliae* were numerous in irrigation water in ditches at the lower end of infested fields (Easton, 1969), so once introduced the pathogen spread rapidly in the Columbia Basin.

Cal Skotland stated in a lecture that rotation was the main control in potatoes. It is possible that if good rotations had been followed from the beginning (prevention), wilt of potatoes would not have become a serious problem, but farmers planted potatoes repeatedly on the best land. In 1964 Easton found that true nematicides did not control early dieing; fumigants with chloropicrin or methyl isothiocyanate (Vorlex) did, increasing the cost of fumigation. The cheapest and easiest way to apply the fumigant (1974) was to shank it in shallow (9 inches) and solid, not trying to fumigate only under the rows. Annual spring fumigation for five successive years reduced populations of *V. dahliae* in the soil, delayed symptoms, and increased yield. Burning the vines prior to harvest did not improve benefits from fumigation alone

(Easton, 1972). Here again, if potatoes were grown on clean land, vines burned prior to harvest, with a good rotation, fumigation may not have become essential. Prevention is cheaper than cure. Some fumigants, especially Vapam, are applied with irrigation water in circle systems. A 1982 extension circular estimated the loss in potato yields, quality, and fumigation costs at \$86,000,000 annually in Washington.

Annual fumigation may lead to soil or ground water problems, and chemicals may be condemned. A rotation of green peas followed immediately by Sudan grass controlled early dieing on heavily infested land with excellent yield and quality (Easton, 1977-1981 seasons). The Sudan grass grew 8 feet tall by November when it was rototilled into the soil. A thorough study of what happens to the microsclerotia and infection rates should be made. This rotation, or one similar to it, could have great value to the potato industry.

#### *Alfalfa*

Alfalfa is so resistant to *V. dahliae* that it is used in crop rotations, but in much of Europe an alfalfa strain of *Verticillium albo-atrum* causes the most serious disease of alfalfa. Alfalfa is a major hay crop in Washington (about \$200,000,000 annually) and Washington produces alfalfa seed. When specimens collected in 1976 by R. N. Peaden, Agronomist and alfalfa breeder at Prosser, were found to contain *V. albo-atrum*, alarm was great. *V. albo-atrum* was known to exist in infested debris among alfalfa seed, and a quarantine against untreated alfalfa seed from Europe had long been in place, but the fungus had somehow been introduced. A quick survey by J. H. Graham of Beltsville, Maryland, Peaden, and D. W. Evans, also of Prosser (1977), revealed the fungus in all parts of the Yakima Valley and the Columbia Basin, in Whatcom and Pierce Counties in western Washington, and near Umatillo, Oregon. A similar survey by J. W. Sheppard in British Columbia found it in the Okanogan and Kootenay Valleys (1977). Alice Christen and Peaden, 1981, estimated that *V. albo-atrum* has been introduced about 1972-1973. This is particularly interesting because *V. albo-atrum* had been reported in Quebec, Canada in 1962 and about the same time in British Columbia, but these early introductions died out. *V. albo-atrum* is now firmly established in North America.

Verticillium wilt is less prevalent in seed fields in Washington than in irrigated hay fields, probably because of fewer cuttings and fewer irrigations.

Europeans found no internal infection of alfalfa seeds, so seed treatments such as thiram should make the seed safe for importation, but Sheppard and Needham in Canada in 1980 found 0.03% internal infection in a lot of Washington seed, enough to put 750 infected seeds in a hectare. Alice Christen confirmed internal infection (1981) and stressed that removing light seeds with wind during threshing removed most internally infested seeds. Christen, 1982, found that as many as 25% of small seeds produced by plants inoculated within two weeks of pollination had internal infection. External seed contamination (Christen, 1983) was found in 5 of 20 seed lots in 1979 and in 2 of 20 in 1980. Systemic seed treatments are required for safety. Christen estimated under ordinary irrigation and cutting, 50% of the plants would be infected within 2-4 years of initial establishment, so spread is surprisingly rapid. The fungus was almost universal in alfalfa stands older than 1 year. Christen and Peaden, 1981, found dryland alfalfa in Stevens County healthy while irrigated alfalfa next to it was not.

*V. albo-atrum* on alfalfa is relatively insensitive to soil types, doing well in acid soils west of the Cascades and in neutral or alkaline soils in the Columbia Basin. Only one strain of the fungus has been found on alfalfa, and resistant individual plants occur in most cultivars. *V. albo-atrum* lacks microsclerotia and rotation with weed control would eliminate or greatly reduce it in infested soils, but control will be achieved by resistance. Alfalfa breeders at Prosser are developing alfalfas resistant to *V. albo-atrum* and to the stem and bulb nematode. Vernema, one of their alfalfas, is moderately resistant to Verticillium wilt and resistant to the stem nematode.

### *Farm Exports*

Washington farm exports were the highest in 1988 since 1984 when the state Department of Agriculture began keeping records. Washington accounted for 4% of the nation's farm exports. 1988 figures in relation to 1987.

Exports		
Wheat	\$652,000,000	up 84%
Apples	112,000,000	up 51%
Beef	98,000,000	up 33%
Potatoes	52,000,000	up 17%
Hops	52,000,000	up 9%
Hay	50,000,000	up 59%
Dairy	36,000,000	up 79%
Seeds	35,000,000	up 10%
Sweet corn	26,000,000	up 34%
Sweet cherries	24,000,000	down 25%

### ***Tree Fruit Research and Extension Center, Wenatchee***

Wenatchee, on the west side of the Columbia River in central Washington, stretches north-south in the narrow valley between the river and the foothills of the Cascade Mountains. The Wenatchee River joins the Columbia just north of the main part of town. The Burlington Northern (formerly the Great Northern) Railroad crosses the Columbia here and heads west up the Wenatchee River Valley to the crest of the Cascades and beyond to the Puget Sound at Seattle. U.S. Highway 2 followed the same path from Wenatchee westward. As Wenatchee grew it over-flowed along the east bank of the Columbia to form East Wenatchee. Apple orchards occupy the tillable and marginal soils in the valleys and lower slopes along the river systems of this region, and Wenatchee and adjacent towns are filled with fruit storage and packing concerns. Tree Top apple juice and apple sauce factory is located at Cashmere, the first town west of Wenatchee in the Wenatchee River Valley. It is not surprising that a tree fruit experiment station developed here.

The federal government established a tree fruit entomology laboratory near Yakima. In spite of this substantial laboratory, entomologists also work at Wenatchee. Ten federal entomologists (no plant pathologists) were located in the USDA Yakima lab in 1988; four WSU entomologists were present at Wenatchee. The policies leading to and sustaining these efforts in entomology in two laboratories would make an interesting study.

D. F. Fisher (1913-1947), USDA, was the first pathologist at Wenatchee, Chelan Co. He worked in private orchards and in a USDA "field laboratory," whatever that was. He was responsible for both orchard and storage diseases and he made important contributions in both. Advances in the control of two storage diseases of apples, scald and blue mold, and of powdery mildew in the orchard, were his greatest contributions.

The 1926 Annual Report of the WAES noted that Chelan County had supported research for seven years. The county requested WSU to station a horticulturist and an entomologist at Wenatchee to study fruit production, handling, and marketing. In 1927 WSU responded and Chelan County paid their expenses. In addition, \$46,000 was requested from the state legislature to establish a tree fruit experiment station, but the request, not part of the WSU budget, was vetoed by the governor. Not until 1937 was a request (for \$60,000) prepared by Dean E. C. Johnson included in the WSU budget. It was approved immediately. The station started with 60 acres of orchard, 45 purchased and 15 donated, and a few buildings. The difficult birth of the TFREC is further emphasized by the fact that a WSU plant pathologist, Roderick Sprague (1950-1962), was not sent to Wenatchee until 1950. Sprague was housed in the attic

of the US Weather Service building on the TFES grounds. His facilities were crude. Federal physiologists and pathologist Charlie Pierson, (1958-1981), who specialized in post-harvest diseases and apple storage, were housed in the US Post Office Building in downtown Wenatchee. The building had offices, laboratories, and cold storage rooms and it was within a few blocks of large commercial apple packing and storage units, also in downtown Wenatchee.

The Tree Fruit Research Center served special needs. In entomology and pathology precise timing of pesticide applications is essential, and scientist should be close to the orchards. At Wenatchee the laboratories are surrounded by fruit trees, as close to the subject as you can get. The US Weather station adjacent to the WSU buildings aids in epidemiological studies. In addition, the Environmental Protection Agency (EPA) had four chemists at Wenatchee who studied such things as when it is safe to enter an orchard after pesticide application, exposure of pesticide applicators and orchard workers to pesticides, disposal of protective clothing, development of more comfortable yet effective clothing, etc. This admirable union of pathologists, entomologists, climatologists and pesticide (EPA) scientists at one location was weakened by the recent withdrawal of EPA from the location.

Facilities at Wenatchee are excellent, well maintained, and attractive. The faculty (1988) contains horticulturists, entomologists, plant pathologists, and plant physiologists. "Scientific isolation" is not a problem and staffing is stable.

WSU had 100 acres at Wenatchee, 100 acres at Columbia View plots in Douglas County near Orondo, and the Columbia River Orchards southeast of Wenatchee. The latter was donated to WSU by the Alcoa aluminum company. For some years there were real and claimed damages from fluorine effluent from the Alcoa aluminum smelter. Columbia River Orchards (apples, pears, cherries, apricots, plums, grapes) was established by Alcoa down wind from the smelter. Emission control was excellent and the down wind fruits flourished. Profits from this orchard supported tree fruit research until WSU sold this property.

#### *Post-Harvest Pathology*

Pioneer orchardists discovered they had the highest yields and the best quality apples in the nation. They also discovered that their greatest problems came after harvest, in keeping the apples until consumed. In 1889, 7080 tons of apples were produced, mainly in the Yakima and Wenatchee areas. By 1910-1912 the orchards were expanding rapidly so that by 1925-1934 the crop averaged about 700,000 tons. It stabilized and remained at this level until 1965-1974 when the effects of controlled atmosphere storage began to be felt. In the decade 1975-1984 the crop averaged over 1,200,000 tons annually. In 1984, 1,092,500 tons were sold fresh, 194,500 tons went into juice, and 49,000 tons were canned. Controlled atmosphere storage makes it possible to market fresh apples all year long, greatly increasing the market. The Washington State Tree Fruit Research Commission receives money for each ton of fruit sold, so increases in sales benefit research funds.

This increase would not be possible if apple rots and storage disorders had not been controlled. Controlled atmosphere storage reduces the rate of development of storage diseases but it does not stop them. Post harvest pathology is a major aspect of apple production.

#### Scald

Scald rendered more stored apples unfit for the fresh market than any other disease. Scald develops after apples have been stored for several weeks. The surface cells die, resulting in a blotchy brown exterior. The damage is superficial but the beauty of the apple is destroyed. Brooks, Cooley, and Fisher (1919), all of the USDA, found that apples from Virginia developed scald quicker than those from Wenatchee, but the varieties were not the same. At Wenatchee heavy irrigation and picking Grimes apples before maturity, the worst combination, resulted in 95% scald. Constant air movement within the stored apples (1/8-1/4 mi/hr) prevented scald or reduced it to negligible quantities. The effect of good ventilation was not due to increased oxygen.



Brooks, et al cited a paper by Powel and Fulton (1903) in which wrapping apples in paper impregnated with paraffin reduced scald. Brooks, Cooley and Fisher found 70% scald in plain paper wraps, 35% with paraffin, 1% with either vaseline or cocoa butter wraps, and 0% when the paper was impregnated with beeswax. Scald is caused by a volatile substance released from apples during storage, and the volatile metabolite is trapped or inactivated in proper wraps. By 1923 they had studied seven different mineral oils. All were effective. The oil should not be placed directly on the apple because it impairs the flavor. The papers should contain about 15% oil by weight. In 1930 they found that shredded, oil-impregnated paper placed among the apples worked, that the apples did not have to be individually wrapped. Wrappers or paper impregnated with mineral oil was the major control until about 1962 when antioxidants replaced mineral oil. Brooks and Cooley, with whom Fisher collaborated, worked in the East.

In 1958-1969 Huelin et al in Australia found the real cause of scald, alphafarnesene. Treating apples with diphenylamine (DPA) controls scald by scavenging free radicals. The combination of controlled atmosphere storage and picking fruit at proper maturity enhances the effect of DPA. The concentration of DPA in the treatment solution, temperature of the water, and duration of the treatment must be proper for best results (Charlie Pierson, 1979 lecture notes). Apparently alphafarnesene is also a pheromone for codling moths and fire ants.

#### Blue mold

Brooks, Cooley, and Fisher (1920) reported that *Penicillium expansum* caused about 75% of rot in apples caused by fungi, so it was by far the most serious storage rot fungus. These workers considered it a wound parasite, following finger nail scratches, insect injuries, scabby spots, bruises and apples punctured by stems during handling. Baker and Heald (1932) found that *P. expansum* could also invade lenticels. *P. expansum* rarely attacks apples on the tree, but over ripe apples are susceptible. Control in the early days relied mainly on avoiding all possible wounds, cooling apples to 32°F within 1-2 days of picking, and wrapping the apples individually in paper (the fungus can spread from one apple to another in storage). Apple rots develop slowly at 32°F, but if the rot starts before cooling it develops more rapidly than at a constant 32°F.

Spoiled apples are a great loss, particularly after shipment across the nation. Records of rot were made at eastern shipping terminals, and Heald and Ruehle (1931) examined these records for the 1922-1925 period. At that time an average of 21.3% of the car loads rejected at the eastern terminals were rejected because of fruit decay, mostly blue mold (*Penicillium expansum*). In order to prevent serious losses apples must be in excellent condition when shipped. Dr. F.D. Heald came to Pullman in 1915. By 1921 he was publishing results of studies on rotting apples, so the problem must have been serious. At that time apples were grown commercially near Pullman. Heald and several of his students studied blue mold until 1934. Even though Heald contributed to many things, including stinking smut of wheat, these studies on blue mold (and other fruit rots) may have been his greatest direct contribution to Washington agriculture.

Control of the codling moth required several insecticide applications during the growing season, and lead arsenate was the chief insecticide. Little rain fell during the growing season and sprinkler irrigation was not used until recently. Lead arsenate accumulated on the fruit in our dry climate. Addition of mineral and fish oils to the spray aggravated the problem. Arsenous oxide tolerance established by the Federal Food and Drug Act of 1906 was less than 0.01 grain per pound of fruit. England began to embargo U.S. fruit in 1925 because of arsenic. Heald, E. L. Reeves, K. F. Baker, D. F. Fisher, and others studied the effects of arsenic on blue mold.

Jonathan and Delicious apples were susceptible to arsenical burning in the calyx basin of the fruit, predisposing it to fruit rot. In addition, removal of the toxic residues from the fruit required either brushing, rubbing, washing, or some means of getting the lead and arsenic to safe levels, and each operation involved wounds or actions that increased subsequent rot of fruit. During 1926 and 1927, Heald and

coworkers found that rubbing to remove chemical residues embedded fungal spores in the wax on the fruit, in lenticels and cracks, but that it did not materially increase blue mold. G. A. Huber (1931) found that brushing the fruit also spread spores.

When fruit was submerged in liquid baths spores were thoroughly disseminated, and a cubic centimeter of wash water at the end of the day contained from 200-12,300 spores. By 1927 it was known that rot was correlated with the number of wounded apples and the spore content of the wash water. The wash tanks tended to equalize batches of apples. A batch of apples with much inoculum would lose some spores in the wash, a clean batch would gain spores. Even if rotted apples were removed before dumping into the wash vats, sound apples near the rotted apples had heavy spore loads. Washing increased the uniformity of rot in storage. Hydrochloric acid solutions at the strength used to facilitate removal of the arsenicals did not kill the spores, even within 24 hours, but Fisher and Reeves (1928) found that alkaline washes reduced their viability somewhat. Water heated to 110°F facilitated cleaning, and if the fruit was moved promptly, it did not harm the apples. Baker and Heald (1932) found that the hot water, whether it contained acid or alkaline materials, killed most of the spores within 24 hours, making it unnecessary to empty the tanks and replace the wash water or to treat the tanks with formaldehyde.

In many packing sheds *Penicillium* grew on wood kept moist by vapor from the washing vats. Huber (1931) found great variation from packing shed to packing shed in the spore content of the air, emphasizing the need for packing shed sanitation. Huber also found 12,000 to 40,000 spores per square inch of gloves worn by fruit packers.

Should the apples be dried after washing before they are packed? It was feared that water on the fruit would favor germination of the spores. Fruit was at that time normally stored at 30-32°F and 83-85% RH. Heald and others (1928) packed Jonathan apples, wet or dried before packing, at 81-90%, 81-100%, and 68-87% RH ('normal', 'wet', 'dry' atmospheres). In no case did it matter whether the apples were dried or wet before packing. D. F. Fisher (1927) found that spores of *Penicillium expansum* can germinate at 32°F and that rot develops over long periods of time even at that temperature. Cold storage greatly retards rot but it doesn't stop it all together. This narrative, extracted largely from K. F. Baker (1934), could continue, but it suffices to illustrate some of the problems that faced the apple industry and how they were solved.

Removing apples from the large field boxes by submersion rather than dumping greatly reduces stem wounding and bruising, a major aid. Modern insecticides, making arsenic obsolete, have eliminated arsenic residues from the fruit. Extensive cleansing is no longer necessary. Harley English, Wright and Smith (1948) used sodium chlororthophenylphenate in wash water to kill spores. This material killed *Penicillium* and *Botrytis* spores but some packing house workers were allergic to it. Sodium-orthophenylphenate (SOPP) is relatively nonallergic and it replaced the former material in the wash water. Charles Pierson (1960,1966) found benzimidazoles superior to SOPP, and benomyl in the wash water and in the wax used to coat the fruit provides protection against blue mold and some other fruit rots. Benomyl is not used in the orchard because use in the orchard and also in the packing house would favor development of resistant strains of the fungi (Pierson, lecture notes, 1977). This is also noted in the PNW Disease Control Handbook.

In a 1979 report at the Wash. Hort. Association annual meeting, Charlie Pierson stressed that control of some storage rots started in the orchard. Bull's eye rot and side rot don't appear until 4-5 months after harvest, but two sprays in the orchard with zinc-containing fungicides had essentially controlled these storage rots. Management of the grass on the orchard floor to prevent excessive humidity reduces gray-mold rot (*Botrytis cinerea*). Picking pears at just the right stage of maturity reduces abrasion of the skin in the packing line and internal break down due to over-ripeness. He stressed proper training and supervision of pickers to avoid stem end punctures and bruises--and not to put apples from the ground among those from the tree. Apples from the ground usually rotted and rot spreads from apple to apple,

and over-ripe apples emit volatiles that affect the sound fruit. Also, in handling the large apple bins used in the orchards, be sure the handling area is well sodded or covered with sawdust to avoid getting soil among the apples.

Note my use of the Proceedings of the Washington State Horticulture Association (call letters SB1 W31). Scientists present papers at these meetings and these and all the activities of the association are printed and bound. They form an invaluable resource to historians. It is sad that other societies (wheat, potatoes, barley?), at least all who could afford it, don't do the same.

Rodney G. Roberts, USDA pathologist at Wenatchee, began working in Wenatchee, WA in July, 1986 for the USDA-ARS Tree Fruit Research Laboratory. His formal training was in mycology and plant pathology. His research is concerned with 1) determining whether *Erwinia amylovora* occurs on mature, symptomless apple fruit, and whether such fruit poses a quarantine risk to countries where fire blight does not occur, 2) investigating the possible uses of chlorine dioxide as an alternative to sodium hypochlorite used in dump and flume waters used to process cherries and apples, and 3) isolate, screen, identify, and develop delivery systems for biological control agents (primarily bacteria and yeasts) for use in controlling postharvest diseases of apples, pears and cherries caused by fungi.

#### *Diseases Mainly in the Orchard*

##### Powdery Mildew

W. H. Lawrence (1905), while at Pullman, published an extensive list of powdery mildew species in the state including *Sphaerotheca humuli*, which attacks several hosts (surely including hops, but he didn't mention hosts), *S. mors-uvae* (severe on gooseberry), *Erysiphe graminis*, which he surprisingly did not consider very abundant, *E. cichoracearum* and *E. polygoni* (both listed as common), *Uncinula necator* common on grapes, *U. parvula* on hackberry, *U. salicis* on cottonwood and willow, *Phyllactinia corylea* as common, *Podosphaera oxycanthae* on cherries, apples and snowberries, *Microsphaera alni* on alder and *M. diffusa* on snowberry. This paper (reprint 45977) is one of the first pure mycological papers published here. It included more species than I listed, but the mycological herbarium was well-started by this time.

In a companion paper in the same year (reprint 6089) Lawrence discussed the life cycle of the mildews, effects upon the host and symptoms, spread, etc., more a pathological treatise. His perspective is of interest. In general he did not consider the powdery mildews very important, with the possible exception of mildew on grape. But he then said powdery mildews were occasionally serious on apple, peach, the entire gooseberry crop may be destroyed, also peas. It seems obvious that, because there were only two or three active pathologists in the entire state, a problem had to be major to warrant more than passing interest.

D. F. Fisher (1918), USDA pathologist at Wenatchee, published a thorough treatment of powdery mildew on apples. Jonathan, Black Ben Davis, and some other varieties were often severely damaged, both in quantity and quality of fruit. Powdery mildew in mild form on fruit causes a russetting which makes them unfit for the fresh market. When severe the apples were so distorted they couldn't be peeled by machines. Fisher, after his review of the literature, concluded that powdery mildew was not serious east of the Rocky Mountains. Trees were sprayed frequently in humid climates to control apple scab and other fungal problems, and these sprays also controlled powdery mildew. He also thought hard showers typical of the East probably helped control powdery mildew (this is true). One of the big advantages of apples in Washington was the paucity of fungal diseases dependent upon rains and high humidity during the summer. All that is needed for powdery mildew on apple is dew, which frequently forms in orchards near Wenatchee.

C. W. Gilbreath, State Horticultural Inspector, reported losses of 25-50% near Walla Walla in 1915. The disease caused much russetting in the Yakima Valley. Near Wenatchee in 1914 and 1915 Jonathan and Black Ben Davis were near complete failure.

For the first time, in 1915, systematic spraying for a fungus disease was begun in the Wenatchee Valley. In that year about 49,000 pounds of dry sulfur and 11,600 gallons of lime-sulfur solution were sold at Wenatchee. Lime-sulfur causes burning of the fruit if very hot weather follows the spray. Three sprays were recommended by Fisher in his 1918 bulletin, the first when blossoms were in the "pink" stage, the second when the petals had fallen but before the calyx is closed. Lead arsenate for codling moth was included in the second and third sprays. He recommended ammoniacal copper carbonate instead of lime-sulfur in the third spray, applied 3-4 weeks after the second. Gray or silvery twigs (covered with mildew) should be removed during the winter prunings.

It is possible that apple production became more intense in the Wenatchee Valley between 1905, when Lawrence wrote his assessment of powdery mildews, and 1914-1915 when powdery mildew was recognized as a major problem in the Walla Walla, Yakima and Wenatchee districts. Heald (1933, p. 575) stated that powdery mildew is common on apples west of the Cascades, but it is seldom damaging there.

Roderick Sprague, the first Washington State University faculty pathologist assigned to the Tree Fruit Research Center (1950), was responsible for control of fungus and bacterial diseases in the orchards. His primary assignment was to improve the control of powdery mildew on apples. He did, however, continue working on leaf spots of grasses and snow mold of wheat in which he also made significant contributions. During his tenure the major advancement in apple mildew control was the substitution of Karathane for sulfur products. This led to mildew control later in the season than was safe with the sulfur sprays.

Roderick Sprague (Bull. 560, 1955) stated, "The classical study of apple powdery mildew is that of D. F. Fisher, USDA, who worked for many years on the problem at Wenatchee. His USDA Farmers' Bulletin 1120 issued in 1920 was for many years the standard source of information."

While apple powdery mildew (*Podosphaera leucotricha*) of apple was known to overwinter on infected twigs, it was not until recently that Gary Grove proved that cherry powdery mildew (*P. oxycanthae*) does not overwinter in this manner in Washington (WA Hort. Assoc. Proc. 1988). There is no evidence of survival on dormant cherry buds. Initial infections in the spring are correlated with maturation and release of ascospores. This advance in understanding will contribute to more efficient use of fungicides on cherry.

Ron Covey was assigned to the Tree Fruit Research Center in July of 1962 following Dr. Sprague's death the preceding March. The snow mold portion of Dr. Sprague's assignment was shifted to the Pullman faculty so that Dr. Covey's total responsibility involved tree fruit diseases. Apple powdery mildew studies continued, and early on the miticides Morocide and Morestan were found to control apple powdery mildew. This led to the recommendation that if either of these materials was applied for mite control no other mildewcide need be applied at that time. These findings about Morestan were particularly significant because the prebloom spray for control of apple mildew russet on pear fruit corresponds with the Morestan timing for control of pear psylla and pear rust mite. During this period it was found that winter temperatures below -10°F reduced the carry-over population of mildew. Since apple fruit is only susceptible early in its development, reduced initial inoculum reduced the amount of mildew russet. Covey found that rusty spot on peaches and perfection spot on apricots were the result of mildew infections. About 1970 the pesticide industry became interested in sterol inhibiting fungicides (SI's). Some of the earliest orchard testing of the SI's for the control of apple powdery mildew were performed at the Tree Fruit Research Center.

In October 1986 Gary Grove was added as a new plant pathologist at the Tree Fruit Research Center. Dr. Grove is partially supported by a grant from the Washington Tree Fruit Research Commission and has some extension responsibility as a specialist in plant pathology. His main research interests are in

ecology and epidemiology. In the short time Dr. Grove has been here he has found that cherry mildew overwinters as cleistothecia. He has defined the time, temperature, and relative humidity parameters for conidial germination of this fungus. He has determined the rate of disease increase and incubation period on one sour and three sweet cherry varieties. He has identified several fungicides that warrant further evaluation for the control of cherry powdery mildew.

Dr. Grove is currently (1988) studying *Cytospora* canker and has developed a semi-selective medium for its isolation. In the process of developing this medium he identified several sterol-inhibiting fungicides that warrant testing as control agents. Dr. Grove is also doing research on the bull's eye-perennial canker complex. Again, he has developed a semi-selective medium for isolation of *Neofabraea perennans*. Fruit that has overwintered on the orchard floor, as well as fruit thinnings, are potential sources of inoculum. His initial studies on fruit infection indicate that it can occur over a wide range of temperature with as little as one hour of wetting. Dr. Covey has prime responsibility for soil-borne diseases and fire blight while Dr. Grove is primarily responsible for all of the other above-ground diseases. Dr. Covey (1989) suffered a second attack by a brain tumor and he died in the spring of 1990.

#### Apple Scab

I suspect that apple scab (*Venturia inequalis*) was influential in shaping the apple industry of Washington. Apple scab was disastrous in western Washington (Wm. Lawrence, 1904, Bulletin 64). It was also a problem in parts of Spokane County (Wm. Lawrence, Bulletin 75, 1906). George L. Zundel, our first extension pathologist, described the situation in Extension Service Bulletin 99 (1923). Apple scab was severe west of the Cascades but it was present in only a few abandoned orchards in the Wenatchee district and absent in the Yakima Valley. Severity fluctuated in the extreme eastern part of the state. His control recommendations included planting varieties in solid blocks so that sprays could be applied to trees in the same stage of development. Prune well to facilitate air and sunlight penetration into the canopy. Do anything you can to destroy old dead leaves on the ground. In western Washington he recommended three spray applications, a fourth if damp weather persisted. A delayed dormant spray may also be necessary in the White Salmon area. In eastern Washington (Spokane County?), two sprays.

F.D. Heald, in his "Manual of Plant Diseases", second edition (1933) p. 613, stated that the most important apple region in the US in which control of apple scab was unnecessary was around Wenatchee and the Yakima Valley. Heald also stated that scab was devastating in western Washington and Oregon and variable in the Inland Empire (= near Spokane?). Where irrigation was required, scab was not serious. To support the probable role of scab in determining our apple region, I include data from an economics bulletin.

#### *"Growers' and Packers' Cooperatives and Associations"*

E.F. Dummeier, Bulletin 194 (1925) described the role of cooperatives in establishing quality standards. Rather than selling apples on an individual basis, growers founded the Yakima County Horticultural Union in 1903, our first cooperative. The produce was combined and sorted as to size, color, etc., and packed uniformly. The Blue Ribbon and Red Ribbon brands added greatly to the prestige of Washington apples, and essentially set the standards adopted for the entire US apple industry. Data on carloads of apples shipped in the 1923-1924 season follow.

#### Wenatchee and Okanogan

Skookum Packers' Association	3579
Wenatchee-Okanogan Cooperative Federation	2006
Wenatchee District Cooperative Assoc	1179
Omak Local Warehouse Co.	<u>150</u>
	6914

### Yakima Valley

Yakima County Horticultural Union	1774
Yakima Fruit Growers' Assoc.	<u>1550</u>
	3324

### Walla Walla

Inland Empire Cooperative Federation	900
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### Spokane Area

Spokane Valley Fruit Growers' Union	225
Arcadia Valley Fruit Growers' Assoc. (Deer Park)	150
Otis Orchards Apple Co.	<u>38</u>
	413

### White Salmon

Star Fruit Co	109
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Note the correlation between apple production and the ease of controlling apple scab prior to the use of overhead irrigation. The main production by 1923-1924 was in area in which scab is easiest to control and it still is today. According to Maloy and Inglis (1989) apple scab occurs regularly in the Green Bluff area near Spokane, the White Salmon area, and in all of western Washington.

Over-tree irrigation has made scab more common in the Wenatchee, Okanogan and Yakima regions. Ron Covey regrets its adoption on a wide scale, because of its effect in increasing scab on apples, phytophthora collar rot of apple trees and some other problems. It reduces one of the major advantages of fruit production in central Washington, mainly low humidity within orchards during the warm part of the growing season. A horticulturist hired by the Regal Fruit Cooperative of Tonasket (Proc., WHA, 1983), a Mr. Noble Law, states that scab can be controlled with few fungicidal sprays. Limit overhead irrigation to 12 hour sets. Turn irrigation off after a rain to let the orchard dry out at least two days. Irrigate during April and May only when soil moisture is deficient (test soil water content prior to irrigating). He said even sprinklers under the trees are not truly safe--significant scab can develop on lower parts of the tree. Manage sprinklers alike, whether over the tree canopy or under it. This report was based mainly on practical experience. He also discussed the use of fungicides, but regulation of irrigation was of greatest interest to me.

### *Fire Blight*

The severity of fire blight east of the Rocky Mountains is the main reason the dessert pear industry is located in the far west. Even though important in Washington, it is far more damaging in the East.

In a single typewritten page, probably for a press release, Kent Beattie in 1905 (?) stated that fire blight of pear and apple was common in eastern Washington and it was "beginning to be found" in western Washington. His control measures consisted mainly of careful pruning to remove infections with disinfection of pruning tools between cuts. He advised disinfecting the cut ends of branches with mercuric chloride before they were stacked and burned. He advised monitoring service berries and hawthorns, minimal irrigation and minimal cultivation to harden wood and removal of water sprouts. Much was included on one side of a single page of paper. John Hall (1913, Popular Bulletin 56) reported that fire blight was prevalent in eastern Washington but in 1913 only three doubtful cases were found west of the Cascades. This Bulletin contains needed detail for growers and adds sodding orchards to further harden the wood. He cautioned growers to purchase trees from nurseries free of fire blight.

F. D. Heald proved the bacteria could infect leaves through stomata, wounds, and hydathodes. He suspected leaf infection July 6, 1915 at North Yakima, isolated the pathogen, inoculated twigs on July 31, had symptoms August 8, and published Bull. 125, dated September, 1915. How is that for speed of publication?

Fire blight was epidemic in 1914-1915 in the Yakima Valley (Heald's book, p. 343). It is serious when late blooms are abundant and warm humid weather coincides with these blooms. Ron Covey recently improved fire blight recommendations based on this relationship. Hail wounds also favor fire blight.

Prior to 1970 the major effort on fire blight consisted of unsuccessful attempts to find more efficacious and safer spray materials. In 1972 Covey found resistance in Washington to the fire blight control bactericide, Streptomycin. Surveys in the next few years showed that resistance was present in all major pear growing districts except White Salmon. Later studies have involved the epidemiology and cultural control of fire blight. Because the fire blight bacterium does not infect blossoms until the mean temperature exceeds 60°F, spraying during the cooler primary bloom was generally futile. In detailed studies on the location of the infection, it was found that in excess of 80% of the infections originate in secondary bloom. When frequent cutting out of fire blight throughout the summer was compared with a single postharvest removal it was found that up to seven times the weight of wood was removed when cutting was postponed. Reading the Proceedings of the Washington Horticultural Association, 1977-1988, impresses one with how little progress has been made in fire blight control for several years. Fire blight recurs with frequency. Covey (1982) commented that it was on the program almost every year for the past 10 years. He also stated that the total effort by the USDA and state experiment stations of California, Oregon and Washington devoted to fireblight was about equal to one full-time scientist. Five or six in the region dabble with control, according to Covey! Warm weather during the main bloom, a little rain and light hail resulted in extensive losses in the Yakima Valley in 1988 according to Michael Willett, Yakima County Extension Agent. My suspicion is that knowledge exists to greatly reduce these losses, but slowness to act in response to weather conditions at critical times is crucial.

#### *Pear Decline*

A disease new to the Pacific Northwest was first observed in pear trees in British Columbia in 1948, and by the mid 1950's it was important in Washington and frightening pear growers. A survey of pear orchards in Yakima Co. in 1956 by Cyril Woodbridge (Horticulturist), Earle Blodgett and Ted Diener, revealed as high as 99% infected trees. By that time "quick" decline and a "slow" decline were recognized. In quick decline a tree wilts before or after harvest and dies in a single year. In slow decline the tree loses vigor gradually. Quick decline was relatively rare, with most trees undergoing slow decline. At this time the cause was unknown. Woodbridge, Blodgett, and Diener noted (1957) that pears with Japanese rootstocks, *Pyrus serotina*, (= *P. pyrifolia*) were susceptible. Pears in the early years were on imported French *Pyrus communis* rootstocks. About 1920-1935 *P. serotina*, rootstocks were widely used. After that Bartlett seedlings (*P. communis*) were used. Decline was prevalent in orchards 20-35 years old, coincident with the used of *P. serotina* as a rootstock. This observation that rootstocks might be important was verified by several studies, and it was the key. In 1960 *Pyrus ussuriensis*, also from the Orient, was proven highly susceptible. In susceptible rootstocks phloem necrosis below the graft union girdles the tree, leading to quick or slow decline, depending upon severity of the necrosis.

Pear decline illustrates how much can be gained from a thorough survey made by experienced workers. They did not just record disease; they inquired about many things and found rootstocks to be a real clue. In the early years Washington trees were resistant and the disease unknown. Then a period of susceptible trees. Then a period of resistant trees. The susceptible trees have since died and been replaced by trees on resistant rootstocks, making pear decline an historic disease in Washington.

In 1960 Bob Lindner, Plant Pathologist, Everett Burts (Entomologist) and Nels Benson (Soil Scientist) associated the pear psylla with pear decline. They believed (1962) the psylla injected toxin into the trees

causing the disease. By 1963 Earle Blodgett, Murit Aichele, and J. L. Parsons had evidence that the pear psylla acted as a vector, presumably of a virus, and that pear psylla toxin did not cause the disease. This was corroborated by Shalla, Chiarappa and Carroll in California the same year. It wasn't until 1970 that the true cause, a mycoplasma, was discovered by California scientists. In 1971 George Nyland reported its control in infected trees with annual tetracycline injections, a discovery which maintained the productivity of diseased pear trees in California.

Everett Burts reported that the last paper on pear decline in the proceedings of the Washington State Horticultural Assoc. was presented by L. P. Batjer in 1962. Burts concluded that research after that date has been academic as far as Washington is concerned. Pear decline is included in the Wenatchee story, even though Cyril Woodbridge was stationed in Pullman, Earle Blodgett and Murit Aichele at Prosser.

### *Brown Rot of Stone Fruits*

"Brown rot of stone fruits" appeared in *The Washington Horticulturist*, volume 9, issue 8 (in June, 1915). In it Heald described the blossom blight, leaf and twig blight, canker, rotting fruit and mummies formed from old, diseased fruit that remained hanging on the tree or fell to the ground. He attributed the disease to *Sclerotinia cinerea*. Heald stressed the importance of mummies as a source of asexual (conidia) or sexual spores (ascospores) after overwintering. Control included destruction of fallen fruit and removal of mummies from the tree (bury them several inches deep in soil). He recommended three applications of self-boiled lime sulfur and timed them roughly, obviously from recommendations developed in the east and middle west, as he admitted, because there were no local data. He stated that losses of 20-50% occurred in prunes and cherries in western Washington, but the disease was not a problem in the drier, irrigated regions. Brown rot in the orchard is favored by warm, humid conditions. This publication, useful to fruit growers, typifies the use of information not based upon local results. Heald came in 1915. He couldn't have based this upon his own experiments. Local data were obtained later by Charles Brooks and D. F. Fisher, collaborating.

Fisher sprayed cherries near Salem, Oregon, and Felida, Washington, to control brown rot. (Felida is west of the Cascades, a few miles northwest of Vancouver.) About 1% of the cherries rotted in the orchard, 24% in transit or after harvest when sprays were not applied in the orchard (Brooks and Fisher, 1921). Sprays in the orchard reduced after harvest rot to 6%. Unsprayed Italian prunes suffered 4.6% loss at harvest, 28% loss post-harvest. When sprayed in the orchard, 1.6% loss at harvest, 7% post harvest. The greatest benefit from the sprays was after harvest. The last spray, about 3-4 weeks before harvest, was the most important.

By 1924 Fisher and Brooks (USDA Farmers Bulletin 1410) had refined their recommendations, aimed mainly at cherries and prunes west of the Cascade Mountains. In humid weather blossom blight could lead to severe losses, and they recommended spraying just before the blossoms open, just after petal fall, when the husks are shed, and 3-5 weeks before harvest. The last spray was particularly important on prunes. They recommended fall and winter plowing to bury fallen fruit and thorough cultivation during the blossoming period to reduce formation of the sexual spores that are so important in blossom infections. They also recommended thorough pruning to improve aeration within the tree.

Heald (1933, p. 523) states that brown rot is rare or unknown in the dry sections of the Pacific Northwest east of the Cascades, but is serious on cherries, prunes and peaches west of the Cascade Mountains, another example of the climatic advantage of growing fruit in a relatively dry climate, using irrigation.

### *Peach Leaf Curl*

B.F. Dana (1922), Extension Service Bulletin 81, listed peach leaf curl as the most frequently reported disease of peach in Washington. It is favored by cool, wet springs. Dry weather when the buds are swelling and the leaves are unfolding results in little disease. It is most common in orchards along the



major rivers and in the Puget Sound area. According to Dana, a single thorough spray in fall or early spring will control the disease. The fungus, Exoascus (= Taphrina) deformans, overwinters as spores on the surface of the tree. If the spray penetrates the cracks and crevices, control will be excellent. If done in the spring, it must be done before the buds begin to swell. He suggested Bordeaux or lime-sulfur, the latter having the advantage of activity against San Jose scale. Fall applications are easier because the soil is firmer (not wet as in early spring) and there are fewer demands on the farmer's time in the fall.

It appears that, even though plant pathology started in Washington about 1892, it was several years before real, locally proven spray schedules for use in orchards were developed.

*Virus Program at Wenatchee  
by Lee Parish*

The USDA transferred E. L. (Bud) Reeves to Wenatchee in the 1940's to work on stone fruit viruses. (According to the history written by George Fischer, Bud Reeves went to Wenatchee in 1927 to work as a field assistant on tree fruit diseases in general.) At that time there were no field plots for virus work and transmission trials could not be done in grower plots, so much of the early work was done on the county poor farm. Phil Cheney soon joined him as an assistant. The Washington State Horticultural Association later bought an isolated tract of land for the virus work. As it was bare virgin land, Phil Cheney and Bud Reeves cleared the sage brush off and put in an irrigation system to establish what is now the Columbia View Plots. They made many of the early contributions in reporting stone and pome fruit viruses. Some of their notable contributions were their work with cherry mottle leaf, cherry twisted leaf, X-disease (Western X), little cherry (K & S little cherry), apple fruit marking disorders.

In the 1950's the virus program was at its highest effort. WSU had Robert Lindner, and the USDA had E. L. Reeves, Phil Cheney, and Hugh Kirkpatrick; in addition Homer Wolf and Ted Anthion had joint appointments between USDA and WSU to work on insect vectors. The effort started to dwindle. Homer Wolf took a job with what is now EPA working on pesticide residues and Ted Anthion became a WSU stone fruit entomologist. Bud Reeves retired in the early 1960's and Robert Lindner became station superintendent. In the mid 1960's C. L. Parish was hired by the USDA to work on stone and pome fruit viruses. In the late 1960's the USDA transferred Hugh Kirkpatrick to Byron, Georgia, to work on stone fruit viruses in the Southeast. Phil Cheney retired in the early 1970's. By the mid 1970's none of the original workers were left. The total effort of what had been a 6-man effort was left with a relative new comer, C. L. Parish.

Parish continued the work of his predecessors. Recent efforts demonstrated cherry rasp leaf and flat apple disease are incited by the same virus. Cherry rasp leaf is field spread (nematodes) and the recommendation was made to pull the cherry trees and plant apple trees. In light of the cherry rasp leaf - flat apple relationship, this recommendation was no longer valid. An ELISA test was developed and used for virus indexing on a large scale to detect tomato ring spot virus. This is important as at that time the Pacific Northwest produced over 50% of the apple trees and over 80% of the apple rootstock for the United States. Indexing the source nursery rootstock stooling beds for tomato ringspot virus became extremely important to insure tomato ringspot virus-free nursery stock. Many of the rootstock stooling beds were planted on old berry ground and tomato ringspot-infected nematodes were present in the soil. This meant indexing literally miles of rootstock and removing and fumigating infected areas. Five apple bark disorders were shown to be graft transmissible. These were previously thought to be genetic and/or nutritional. A suspected calcium disorder of pear was shown to be graft-transmissible; however, the symptoms are alleviated by calcium sprays. Dead spur of red delicious was shown to be graft-transmissible and related to long, leggy growth observed in other apple cultivars including some rootstocks. Apple Decline was demonstrated to be induced by a mycoplasma-like organism and the steady decline of the tree can be reversed by oxytetracycline injections.

Little cherry was detected in 1933 on the west side of Kootenay Lake near Nelson, B.C. Within 15 years it had ruined the cherry orchards of that region. In 1969 it was found near Penticton in the Okanogan Valley. By 1975 it was found in Washington just across the Canadian Border at Oroville alarming local growers. An extensive survey in 1976 found one tree near Oroville and one near Ellesford. In 1977 a few infected trees were found as far away as the Yakima Valley and even near Kennewick (Proc. Wash. State Hort. Assoc., 1977, Lee Parish). The disease has been controlled by eradication and sanitation.

Parish (Proc. Wash. Hort. Assoc., 1988) reported that all trees with apple decline tested positive for mycoplasma-like-organisms, that 82% of adjacent trees were positive, and 0% in trees two- or more trees from infected ones. Response to oxytetracycline injected into the tree after harvest supported the mycoplasma etiology. There was no response to Ridimil (for Pythium or Phytophthora control).

#### *Other Problems*

In the mid 1970's a survey was made by Harold Larson and Ron Covey to determine the causal agents of wood rots of apple in Washington. While as many as 16 species were involved, *Polyporus versicolor* was involved in more than 50% of the cases.

In the late 1960's Dr. Covey became more interested in soil-borne diseases. With the increased plantings of susceptible clonal dwarfing rootstocks, crown rot had become more of a problem. Studies on chemical control resulted in an improving series of recommendations from copper trunk paints (1970) to Ridomil 2E (1986). Early trapping studies demonstrated that as in British Columbia *Phytophthora cactorum*, the causal organism, is distributed by surface irrigation water. Later studies proved that *P. cactorum* was in canal water the entire irrigation season. It was further found that the fungus could be found throughout the orchard floor but was present in higher numbers close to the trunks of diseased trees. In studies on the susceptibility of rootstocks in relation to bud development it was found that all apple rootstocks are very resistant during the dormant period and that most reach their peak of susceptibility just past bud break. Apple rootstocks MM104 and MM106 are most susceptible; M7, M9, M26 most resistant. These modern rootstocks, many used for dwarfing, are vegetatively propagated, making them uniform in disease reaction. When rootstocks are from seedlings, they are variable in reaction. Golden Delicious scion wood is quite susceptible. Growers generally place the bud union below ground level when planting on seedling roots, making scion wood important. Cherry rootstocks are very resistant when dormant but reach the maximum susceptibility shortly after shoot elongation begins. The possibility of biological control of crown rot by a soil-borne bacterium has also been demonstrated in research conducted at the Tree Fruit Research Center.

Sometime in the 1930's it became evident that apple trees did not grow well in soils that had been previously planted to apples. The high levels of arsenic (sometimes in excess of 300 ppm), toxic to many other crops, was generally blamed. In studies in 1969 and 1970 Dr. Nels Benson, Soil Scientist, Tree Fruit Research Center, developed data that shed some doubt that the sole cause of the replant difficulty was due to arsenic. In the spring of 1971, Drs. Benson, Covey and Dr. William Haglund, Plant Pathologist of Northwest Washington Research and Extension Center, collaborated in fumigating high arsenic soil. The trees in soil fumigated with chloropicrin grew very well. Over the next three years this team developed practical means of fumigating replant sites in replant situations and changed the fumigant from chloropicrin to methyl bromide. Seven years after planting, many of the fumigated trees produce more than twice what the unfumigated checks produce. In recent studies with Dr. Frank Peryea the fumigant Vapam has given early growth increases comparable to those after use of methyl bromide. Only time will tell if it is a satisfactory substitute.

Endomycorrhizae can be implicated with both arsenic toxicity and soil fumigation. From 1977 to the present Dr. Covey, in cooperation with various soil scientists at the Tree Fruit Research Center, has studied the effects of these beneficial fungi on apple tree growth. In greenhouse studies, *Glomus* spp. overcome both zinc and phosphorous deficiency.

### ***The Northwest Washington Research and Extension Unit, Mount Vernon***

The first research facilities at Mt. Vernon, obtained in 1943, consisted of space in a building at the Skagit County Fairgrounds. The research unit was called the Northwest Seed and Truck Crop Laboratory. In 1947 the present station was started and by 1961 it had over 50 acres of Skagit River Valley land. By 1976 it had grown to 80 acres owned and 20 leased. The Northwest Agricultural Research Foundation augments appropriated funds with about \$40,000 a year. James Hulbert, one of the farm leaders who helped stimulate research in northwest Washington, served as a regent of WSU and is honored by Hulbert Hall on the WSU campus. Hulbert Hall houses the administrative offices of the College of Agriculture and Home Economics as well as some other units of the College.

The Mount Vernon station is within 10 miles of Puget Sound and about 40 miles south of the Canadian border. Like Puyallup, it is in a region of beauty, including the Puget Sound and Mt. Baker, a volcanic peak. The limited tillable land of northwest Washington is devoted to dairying, berries, bulbs, green peas, and vegetable seeds. Most of the cattle feed, other than corn silage, is purchased (high quality alfalfa hay from the Columbia Basin, plus grain). Washington dairy cows are the most productive in the nation, due to progressive dairymen, the high quality alfalfa hay, lush pastures in the usually mild, cool coastal climate that Holstein-Friesian cows appreciate.

#### *Cabbage*

A large population of German descendants led to a significant sauerkraut industry near Racine, Wisconsin. Cabbage was grown near the kraut processors repeatedly on the same soil for years. By 1910 cabbage failed because of cabbage yellows (*Fusarium oxysporum* f.sp. *conglutinans*). Resistant cultivars were developed and local seed growers increased the resistant cultivars. *Phoma lingam*, a pathogen dispersed by rain, infected the seed and black leg decimated the yellows-resistant cabbages. Hot water treatment would kill *Phoma* but it weakened the seed (p. 119, Williams and Marosoy). Professor Walker divided a seed lot in half, increasing half in Wisconsin and half in the Skagit Valley. The seed produced at Mt Vernon in 1921 was free of *Phoma lingam*. Seed of yellows-resistant cabbages was subsequently produced in Washington, reducing the seed industry of Wisconsin, but the cabbage seed industry of Skagit County was begun.

Control of black leg (*Phoma lingam*) in humid regions depends upon clean seed and crop rotation. Without clean seed, there is no practical control. The cabbage seed industry is concentrated in the Skagit Valley in Western Washington and in parts of Oregon because in dry summers the seed is free of the pathogen. In wet (rare) seasons even Washington seed can be infected.

Russia, England, Holland, and Belgium were important sources of cabbage seed (and that of many other vegetables) for the U.S., and these seed sources were lost during World War II, creating an emergency. Seed produced in the Skagit Valley, was not adequate. J. C. Walker, the leading vegetable pathologist in the U.S., recommended that Glenn Pound be sent to the Skagit Valley to facilitate increased seed production. Under an emergency agreement between WSU and the USDA, Pound arrived in Mt. Vernon in late May, 1943. He left in April, 1946, to become a professor at the University of Wisconsin (and subsequently Chairman of the Department of Plant Pathology, Dean of the College of Agriculture, and for a short time, President of that university). According to Glenn Pound (p. 36), the seed industry at Mt. Vernon had too little research and diseases had increased in the various seed crops because of intensive cultivation in a small area. His facilities were part of a county agent's office, a microscope, a car, and greenhouse space rented from a florist (p. 37).

Pound diagnosed the main problem in cabbage seed production as cabbage mosaic. Seedlings were grown near the over-wintered seed plants and aphids moved freely from old plants to young plants. Seedling beds were established 40 miles up the Skagit Valley, away from the seed production fields.

Both beets and cabbage were subsequently handled this way and seed yields doubled immediately. Pound arrived in late May, 1943 and left in April 1946. He came, he saw, he conquered. In retrospect, it causes concern that so much effort was expended trying to isolate young plants from sources of virus. L. K. Jones recommended this for beets years earlier.

In 1946 and in 1973 weather in the Skagit Valley favored *Phoma lingam*, and serious losses resulted in humid cabbage-growing regions (O. H. Calvert and Glenn Pound). Cabbage seedlings were grown in dense plantings (plant beds) prior to transplanting. The fungus sporulates quickly, and if it spreads within the plant beds many infected transplants result. It is important to maintain the dependability of our seed, and J. M. Bonman (Ph.D., 1980) began a detailed study of seed infection under the direction of R. L. Gabrielson. Bonman found only a few seeds infected, but only a few seeds infected with virulent fungus can cause severe losses. Bonman was unable to identify infected seed pods in the field before harvest, so the seed producer cannot tell in advance whether seed is infected or not. Only 1% of the seed pods were infected, with only 0.25% of seed from such pods infected. Only a weakly virulent strain of the fungus was found in the seed. Apparently infection by the local, nonvirulent strain occurs with some regularity, but virulent strains infect the seed only in unusually wet summers.

Gabrielson and M. W. Mulanax (1976) found the sexual state, *Leptosphaeria maculans*, of *Phoma lingam* in Washington and Oregon. It produces ascospores that are ejected into the air, and these spores can infect the seed pods, increasing the danger of having infected seed. Ascospore cultures (the sexual spores) in Washington produced only weakly virulent strains. Ascospores from Wisconsin produced virulent strains (1980). Bonman concluded that the ability of virulent strains to persist in the seed-producing regions of the West is not known.

The presence of avirulent strains in seeds complicates seed sales. Most countries require a phytosanitary certificate. If *Phoma* is found on the seed it should be tested for virulence. If avirulent, the seed is still safe to use. If virulent, the seed should be condemned. All seed labs are not equipped to perform the virulence test. G. S. Pound in 1947 described an avirulent strain of *Phoma lingam* which he called the "Puget Sound" strain, and by 1951 Pound and coworkers in Wisconsin concluded that the Puget Sound strain was so avirulent it was no threat in humid areas. The studies of Bonman confirmed the avirulence of strains that persist in the Pacific Northwest.

White blight (*Sclerotinia sclerotiorum*) is serious on seed cabbage and D. M. McLean devised an effective but expensive control using cyanamid, wide row spacing and hand-tying the plants in 1959. Gabrielson, W. C. Anderson, and Bob Nyvall (1973) devised a cheaper control using benomyl sprays and cyanamide. Gabrielson, R. K. Guilford, and D. R. Coahran (1971) also used benomyl to control white and gray molds of green table beans in Western Washington.

#### *Table Beets*

Beets are biennial, like cabbage, with seed produced by over-wintered beets. In seed production, seed is sown in beds in June. In late fall the roots (stecklings) are removed and stored during winter, usually in pits, to be transplanted in spring for seed production. By 1929 seed yields were half what they had been in 1924. Leon K. Jones attributed the decline to general infection of the mother beets by beet mosaic virus. The mosaic virus can be transmitted mechanically, facilitating assay for virus. Many fields of mother beets were 100% infected. Jones reported that symptoms were easy to see June 13, but as the season progressed symptoms became obscure. In 1929 Jones grew seedlings from 1000 seeds collected from diseased mother plants and not one seedling had mosaic, so the virus was not seed-borne, corroborating results of earlier workers in Europe. Spread by aphids was most rapid in the field between July 17 and September 3. By September 3 it was obvious that steckling (young) beets near mother beets were heavily infected. Little mosaic developed very near the Puget Sound. It increased as you moved inland. Aphids are transported by gentle wind, and winds are often from the water inland. Jones recommended growing the steckling beets near Puget Sound in isolated localities. Even though

Jones recommended isolation of the seedling beets from older beets infected with virus, control was not achieved.

D. M. McLean (1953), a USDA scientist who replaced Glenn Pound (1944-1946) at Mt. Vernon, reported that virus yellows was serious. The virus is efficiently transmitted by the green peach aphid (*Myzus persicae*) and to a lesser extent by other aphids. Although the virus has a wide host range, the beet itself is the most important source of virus. The aphids are difficult to control adequately with insecticides to prevent virus transmission. The most important control was to plant the seed beds in isolated spots away from other beets, plus aphid control in the steckling beds. Jones first, then Pound, and finally (?) McLean recommended isolation of young biennial plants from old ones.

### *Peas*

The pea seed industry of Washington has strong historical ties with Wisconsin, the most important producer of processing peas (green peas for canning and freezing). Washington is second. Pea production became a major industry in Wisconsin at an early date, but severe losses from foliage diseases (*Ascochyta* spp. caused failures before 1915). These fungi are seed-borne in humid climates. Richard E. Vaughan, the first extension pathologist in Wisconsin, nearly lost his job when he recommended purchasing pea seed from the West, where peas matured in a dry climate and seed-borne pathogenic fungi and bacteria were minimal or absent. Wisconsin seed producers did not want to lose the pea seed business, but necessity led to its moving west (as did the bean seed industry - most of the latter to Idaho).

*Fusarium oxysporum pisi* race 1 and race 2 are wide-spread, essentially present wherever *Pisum sativum* has been extensively grown. Race 1 causes common Fusarium wilt, Race 2 causes near wilt. Wilt resistant varieties were grown widely and the disease was controlled. Peas for processing (green peas) do very well in the cool, relatively humid climate of western Washington and peas were grown with short rotations. The processing pea industry began in Western Washington in 1924 and by 1930 race 1-resistant cultivars were required. Control was maintained until 1963 when race 5 was detected. Race 6 appeared in 1970. Bill Haglund and John Kraft described race 5 in 1970 and race 6 in 1979. Haglund and Wilbur Anderson developed varieties resistant to races 1, 2, 5, and 6, making economic pea production possible on infested land. Robert F. Nyvall and Haglund (1972) found that race 5 invaded susceptible peas more rapidly than other isolates of *F. oxysporum*.

Haglund, in cooperation with H. S. Pepin of Canada (Can. J. Plant Pathology 9:59-62), recently found another race in British Columbia. Washington State University selection 31 is resistant to the new race, but no commercial varieties are. For some reason new races of this fungus develop more rapidly near Puget Sound than any where else in the world. Pea research must be maintained if peas are to be grown in this region.

### *Soil Fumigation*

Bill Haglund maintains an active fumigation program against nematodes in bulb crops and berries. Chuck Gould stated that proper use of fumigants, perfected by Haglund, was a major benefit to narcissus and iris growers.

### *Present Faculty*

The faculty at Mt. Vernon (1989) consists of Wilbur Anderson, Horticulturist and station manager, Robert Norton, Horticulturist, Bill Haglund, plant pathologist, and Stott W. Howard, a weed specialist. The station is oriented toward horticultural crops. Bill Haglund works part-time as a consultant as well as station pathologist. Research is concentrated on vegetables, berries, ornamentals, Christmas trees, testing

cereals, with some work on tree fruits and grapes. Leon Jones and Leo Campbell worked in the region before there was a local experiment station.

## PART II

### CEREAL RESEARCH AT PULLMAN

#### *Smuts*

Before common bunt (*Tilletia caries* and *T. foetida*) was brought under control in the late 1950s by seed treatment with hexachlorobenzene, the Pacific Northwest was the "smut capitol of the world" and much effort was expended on common bunt from the inception of the experiment station until the 1960s, a big part of our total history. Wellman and Humphrey reviewed European common bunt literature (1924) and Holton and Fischer (1936) published a 211 page book on bunt. The first general treatises on local smuts were produced by George L. Zundel, "Some Ustilagineae of the State of Washington" and "Smuts and Rusts of Northern Utah and Southern Idaho". In 1926, just before he left Washington, he published a final note on *Tilletia*, *Ustilago*, *Sphacelotheca*, *Cintractia*, *Doassansia*, *Entyloma*, and *Urocystis* spp. in Washington.

Flag smut of wheat was first reported in the U.S. in Illinois in 1919, and it was believed to have been introduced from Australia. Zundel found *Urocystis agropyri* on grass leaves in the mycological herbarium that had been collected in 1899 by W. N. Suksdorf near Bingen, Klickitat County, and in 1919 Zundel found flag smut on grass leaves near Port Townsend, La Conner Flats, and Walla Walla, evidence that *Urocystis agropyri* had been within the state for a long time. It was not found on wheat in Washington until much later, however (Heald and Holton, 1940).

Fischer and E. Hirschorn, a visiting scientist from Argentina, published Bulletin 459 on smuts of Washington in 1945. "Biology and Control of Smut Fungi", the most comprehensive general treatise of the smuts, was published by Fischer and Holton in 1957. Durán and Fischer published a taxonomic monograph of *Tilletia* in 1961 and in 1987 Durán published a book on the smuts of Mexico. This partial listing of major works records the sustained significant interest in smut fungi at WSU.

It always puzzled me that some smuts go to grasses in many genera, yet single genes within a species can stop them. To the smut, sister wheats a and b are often more different than grasses in different genera, and even in different tribes. In these cases individual smut resistance genes have more effect than entire genomes exclusive of the resistance genes.

#### *Chapter I. Common Bunt of Wheat*

*Tilletia caries* (DC.) Tulasne, 1847

*T. tritici* (Bjerk.) Winter, 1881

*Tilletia foetida* (Wallr.) Liro, 1920

*T. levis* Kühn, 1873

Common bunt or stinking smut of wheat is so important in the history of plant pathology in Washington that it is given detailed coverage. Common bunt, hereafter referred to as bunt, is caused by *T. caries* and *T. foetida*, two species so closely related that they hybridize readily. The spore wall of *T. caries* has reticulate ornamentation, the wall of *T. foetida* is smooth. Their life and disease cycles are similar. Both invade wheat seedlings shortly after the wheat germinates, are systemic within the host, and they

sporulate within the ovary, completely replacing normal ovary contents with black, spherical spores. The ovary wall remains intact, firmly enclosing the spore mass to form bunt balls. The bunt balls are easily crushed, releasing the spores and the foul odor typical of 'stinking' smut, attributed primarily to trimethylamine. When wheat is threshed most of the bunt balls rupture and loose spores contaminate healthy kernels and some are blown from the rear of the machine. Some bunt balls escape rupture and remain within the threshed grain. Their size and shape make bunt balls difficult to remove with grain-cleaning equipment. Bunt balls are slightly lighter than kernels and can be removed from seed by flotation in salt brine. The best description of bunt that I have seen is that of Heald (1933, p. 719-721), and readers are referred to it. In Heald's time dwarf bunt (*T. controversa*) was not distinguished from common bunt. The spores of *T. controversa* are reticulate like those of *T. caries*, and Heald stated that *T. foetida* causes a 'tall' bunt, *T. caries* a 'short' bunt. This statement should be discounted as both species of common bunt cause some degree of stem shortening not comparable to shortening caused by dwarf bunt. Dwarf bunt has a different life cycle and will be treated separate from common bunt.

Geographic distribution. *Tilletia caries* and *T. foetida* occupy the same niche, doing the same thing in the same place at the same time, but, according to dogma, they should not coexist long in the same place. If one dominates in one area and the other dominates in another, there is a reason. They appear equal in their ability to overcome resistance genes in the host. Wheat has been moved repeatedly around the world, mostly before the use of seed treatments that killed spores on the seed, and before plant quarantines. Both pathogens were surely introduced several times into the major wheat regions of the world.

In northern and western Europe *T. caries* dominates (Feucht, 1932; Gassner, 1938). Hanna (1934) stated that only *T. caries* had been reported in Great Britain and that it was dominant in France and Germany. Jamalainen (1941) found that 97% of the collections in Finland were *T. caries*, 3% *T. foetida*. In southern Europe extending eastward to the base of the Caucasus Mountains, *T. foetida* is dominant (Feucht, 1932; Gassner, 1938). Gassner sampled Turkey extensively and reported 2003 heads of *T. foetida*, 274 *T. caries*, 5 infected with both. *T. foetida* is most common in Hungary, with *T. caries* being present mostly in wetter areas (Bohus and Podhradsky, 1952). In Australia both were common, but Churchward (1932) reported *T. foetida* more common. Hanna (1934) stated that *T. foetida* was more common in Western Australia, *T. caries* in New South Wales. Mitra (1935) reported *T. caries* in Kashmir near the Himalayan Range and *T. foetida* in the Simla Hills and in Baluchistan. Mitra believed *T. caries* was favored by slightly cooler soil than *T. foetida*. The common bunts were not found on the plains of India where the soil is above 20°C at sowing time and shortly thereafter.

In the central and southern Great Plains of North America *T. foetida* was almost exclusively present (Tisdale, et al, 1927). It was the only bunt detected in wheat at terminal elevators in Colorado, Kansas, Nebraska, Oklahoma and Texas. In a detailed survey of Kansas, 1928-1931, not a single specimen of *T. caries* was found, even though it had been reported earlier by Kellerman and Swingle (in Melchers, 1934). Melchers in 1950 again reported only *T. foetida* from Kansas. Use of seed treatments to control bunt declined, and in 1984 Williams and Gough (PD 68:537) reported *T. caries* in five southwestern Oklahoma counties. No details are given, but this may result from a recent introduction that increased without competition from *T. foetida*.

In the northern Great Plains a more complex situation existed. In an early period *T. foetida* dominated hard red spring wheat, *T. caries* on durum (Tisdale, et al, 1927). The dominance of *T. caries* on durum was later explained on the basis of races adapted to the particular durums grown at that time. In the Canadian prairies *T. foetida* dominated on hard red spring in the southern portions, *T. caries* became more important to the north (Hanna and Popp, 1930).

West of the Rocky Mountains the situation varied locally. *T. caries* dominated in California (W. W. Mackie, in Stephens and Woolman, 1922). West of the Cascade Mountains in Washington and Oregon both species were equal. East of the Cascades in Oregon, only *T. caries* was found. Dominance by *T.*

*caries* also existed in eastern Washington and northern Idaho. In 1919 Kienholz and Heald found *T. foetida* only twice among 631 collections. The absence of *T. foetida* in eastern Oregon so interested Stephens and Woolman (1922) that they inoculated wheat separately with both species and grew it in western Oregon and east of the Cascade Mountains. Both smuts did well in all three nurseries and Stephen and Woolman were unable to explain the difference in distribution of the two species. Why wasn't *T. foetida* established in eastern Oregon? They observed that *T. caries* might be favored in spore showers because the spore masses were more powdery, less sticky than those of *T. foetida*. This could be a factor in eastern Oregon, Washington and northern Idaho (the Pacific Northwest = PNW), in which soilborne spores from spore showers are important, but why is *T. caries* favored in many more humid areas such as northern Europe?

By the time Flor (1933) made his detailed survey, *T. foetida* had increased in eastern Oregon and Washington and in northern Idaho but *T. caries* was still dominant. Flor found *T. caries* in every smutted field, *T. foetida* in 60% of the fields. Between Heppner, Oregon and Dayton, Washington 100% of the smut was *T. caries*. Flor commented that many of the fields contained cultivars with no resistance to either species.

Hanna (1934) believed chance introductions responsible for the differences in distribution. Feucht (1932) and Gassner (1938) believed that continental climates favored *T. foetida*, maritime climates favored *T. caries*. Both smuts are of ancient origin (Woolman and Humphrey, 1924), and both pathogens are ideally adapted to seed dissemination. These two smuts differ in some subtle way not yet explained by their temperature, moisture, longevity, or virulence relationships as revealed in existing literature. Maybe Hanna is right, but it is hard to believe that prior establishment could be so powerful as to explain the dominance of *T. foetida* in Kansas or *T. caries* in Britain. Future scientists should explore the competitive relationships of *T. caries* and *T. foetida*. The most useful data on distribution are the early data obtained before these smuts were controlled by modern seed treatments.

Longevity of spores. Spores of both species remain viable for years when dry, either free as spore powder or within the bunt balls. They live as long as wheat seed, but after 5 years a decline in speed of germination occurs (Table 1, from Lowther, 1950). Spores stored for 11 years as dust in sealed vials germinated slightly more slowly than spores from bunt balls. Old wheat seed would likewise germinate more slowly.

Table 1. Time in days for fresh and 11-year old spores of *T. caries* to reach maximum germination (Lowther, 1950).

Race	8°C Old			20°C Old		
	Fresh	Balls	Powder	Fresh	Balls	Powder
T-3	8	14	14	4	7	7
T-4	9	14	22	5	7	7
T-1	9	22	22	5	9	9
T-2	10	22	22	5	9	9
Average	9.1	17.0	19.0	4.5	7.3	7.5

*T. caries* and *T. foetida* consist of races described on the basis of resistance genes in wheat that they attack. T-races are races of *T. caries* (synonym, *T. tritici*). L-races are races of *T. foetida* (synonym, *T. levis*). At the time Flor designated T and L races, *tritici* and *levis* were the recognized specific epithets, not *caries* and *foetida*. Smut workers retained the T and L designations.

Mechanisms of spore germination. Teliospores, hereafter referred to as spores, of *T. caries* and *T. foetida* do not require light to germinate; they usually require 5-7 days even under favorable conditions and they are not prevented from germinating in soil by fungistasis. They survive for years dry on stored



seed, and under favorable conditions they germinate more-or-less simultaneously, characteristics that adapt them to being seed-borne and to infect seedlings. Seed is contaminated during threshing and the spores germinate when the seed is sown in cool, moist soil.

Mature spores contain the only diploid nucleus in the life cycle (Dangeard, 1893). The spores are highly dehydrated (2.2% water, Trione, 1977) and they hydrate slowly, reaching 8.5% water in 24 hours. In 50 hours the spores contained 28.3% water and they had increased 117% in volume. During germination the diploid nucleus divides meiotically and mitotically within the spore according to Rawitscher (1922, in Kolk, 1930). The spore wall ruptures and a short, stout, aseptate germ tube called the promycelium by Tulasne, emerges. Haploid nuclei migrate into the promycelium at the tip of which four to 16, usually 8-16, slender hyaline spores called sporidia, also named by the Tulasne brothers, form. The haploid nuclei migrate from the promycelium into the sporidia, typically one nucleus per sporidium, and the promycelium is left enucleate.

The above sequence is essentially as I presented it to my classes, but Goates and Hoffmann (1986, Can. J. Bot. 65:512-517), studied nuclear phenomena during spore germination in *T. caries*, *T. foetida*, and *T. controversa*, and found them similar in the three species, and each species had four chromosomes. As presented in the preceding paragraph, no nuclear divisions occurred in the promycelium, but sometimes more primary sporidia formed than there were haploid nuclei at the tip of the promycelium. After migrating into primary sporidia, some nuclei divided and the extra nuclei migrated from nucleate sporidia to anucleate sporidia. Any supernumerary nuclei remaining in primary sporidia or in the promycelium lysed.

A hyphal bridge (plasmogamy) forms between some of the adjacent slender primary sporidia, forming what the Tulasnes called H-cells. DeBary postulated that the hyphal bridge made possible the movement of a nucleus from one sporidium into another, that the H-cells were evidence of a sexual process. Rawitscher illustrated this postulated nuclear passage in 1914 according to Kolk (1930). The migration of the nuclei is essential (Flor, 1931; Hanna, 1934) for pathogenicity. When primary sporidia are removed from the promycelium before H-cells form, and then grown in culture, they produce many secondary sporidia (or in some cases, mycelia). When secondary sporidia of a single primary sporidium are used as inoculum, no disease develops. When mixed in compatible combinations, disease development is possible. Monosporidial cultures are designated + or -, and + cultures alone or paired with other + lines are nonpathogenic, and - cultures likewise are nonpathogenic alone or paired together. Both *T. caries* and *T. foetida* are heterothallic, and single haploid lines are nonpathogenic. Flor's (1932) matings were 'imperfect' in that some + and - combinations did not result in disease, leading him to believe that more than two sexes (compatibility groups) existed. His conclusions were based upon ability to produce disease, not upon hyphal fusion. Flor found + and - in both *T. caries* and *T. foetida* and that each species recognized the + and - of the other, and that the species could hybridize and produce pathogenic offspring (Table 2).

Table 2. Production of disease (D) by paired monosporidial cultures of *Tilletia caries* and *T. foetida* (Flor, 1932).

	<i>T. caries</i> monokaryon				<i>T. foetida</i> monokaryon			
	209	263	303	306	223	285	286	308
<i>T. caries</i>	+	-	-	-	+	+	+	-
209+	0 <sup>a</sup>	D	D	D	0	0	0	D
263-	D	0	0	0	D	0 <sup>b</sup>	D	0
303-	D	0	0	0	D	D	D	0
306-	D	0	0	0	D	D	D	0
<i>T. foetida</i>								
223+	0	D	D	D	0	0	0	D
285+	0	0 <sup>b</sup>	D	D	0	0	0	D
286+	0	D	D	D	0	0	0	D
308-	D	0	0	0	D	D	D	0

<sup>a</sup> 0 = nonpathogenic

<sup>b</sup> 0 = expected to be pathogenic if only two mating types.

Hanna (1934) made similar crosses and he found only + and - monosporidial lines, so the fungus is heterothallic by the simple situation called bipolar with only two alleles (+ and -) at a single locus. Holton and others, especially Kendrick (1968), removed all doubt. Kendrick observed both fusions and pathogenicity between monosporidial pairings. Monokaryons of three races of *T. foetida* (= L-races) and 15 races of *T. caries* (= T-races) were inter- and intra-species compatible in a bipolar (+ and -) manner. Many compatible matings as determined by fusion were not pathogenic and compatibility and pathogenicity are inherited independently (Kendrick, 1968). The aberrant mating of Flor (1932, Table 2) was caused by a nonpathogenic offspring of monokaryons 285 x 263.

Once fusion between primary sporidia (= H-cells) has occurred, one of the pair gives rise to either a binucleate secondary sporidium or directly to an infection hypha. Penetration of the host is direct by the infection hypha, and the mycelium within the host is septate, normally with two nuclei (+ and -) within each cell (Dangeard, 1892), forming a dikaryon. The dikaryotic condition is maintained by simultaneous, conjugate division of the + and - nuclei. The compatible nuclei, even though within the same cell, do not fuse until within the teliospore, completing the life cycle.

Note that the term secondary sporidia is used for two unlike spores. Spores produced in culture (haploid, monokaryotic) by primary sporidia are called secondary sporidia. Spores produced on fused primary sporidia (dikaryotic, + plus -) are also called secondary sporidia. The reader must determine what is meant by the text. Primary and secondary sporidia are sexual spores, not conidia, and the term sporidia is justified.

A. H. R. Buller and T. C. Vanterpool made a special study of the production of primary and secondary sporidia of *Tilletia caries* and *T. foetida* in 1925-1926 (published by Buller in volume 5, p. 207-278, 1933). Any one interested in the formation of these structures and their role in infection should read this work. The smut spores were germinated on solid media in the laboratory, but their observations probably duplicate much of what happens in nature, especially when spores germinate on the soil surface. The paired primary sporidia, when borne on a vertical promycelium, may fall to the substrate. The pair can there produce a secondary sporidium that is forcibly ejected from a small sterigma into the air. Usually, secondary sporidia are ejected while the primary sporidia are still attached to the promycelium. According to Buller and Vanterpool secondary sporidia can be propelled upward 1 mm, and horizontally slightly further, far enough to propel them into air currents for greater dissemination. The total contents of the fused primary sporidia enter the secondary sporidia, so the cytoplasm is dense. Secondary sporidia showered upon germinating wheat and very young seedlings produced disease. During laboratory

experiments they observed that spores exposed to air currents germinated readily but germination in a closed vessel was greatly restricted. H. M. Woolman and H. B. Humphrey made a similar observation and they attributed reduced germination in a closed vessel to lack of oxygen. Buller and Vanterpool ruled that out. They believe that either the lack of air movement over the spores or a saturated atmosphere or both together reduce germination. We know the spores germinate well within soil where air currents are surely minimal and where the relative humidity of the soil atmosphere is high, so the observations of Buller, Vanterpool, Woolman and Humphrey--all excellent scientists--are hard to explain.

A modern treatment of the formation of secondary sporidia by *Tilletia foetida* with scanning electron micrographs is provided by Goates and Hoffmann (1986, Mycologia 78:371-379).

When Lowther (1950) retrieved immature, hyaline spores from immature bunt balls he was unable to germinate them. When the normal color began to form, germination increased as the spores became more mature within the spore ball. He found no after-ripening requirement for germination. A mature bunt ball contained 8,000,000 spores according to N. A. Cobb in Australia and Heald (1921) found 6-9,000,000 per bunt ball in Washington.

The promycelia are negatively geotropic, whether the smut spores germinate upon or within soil (Sartoris, 1924). The promycelia and sporidia from seed-borne inoculum grow appressed to the surface of the host. Sartoris observed the formation of infection hyphae within soil from fused primary sporidia. According to him the infection hypha grew 300-400  $\mu\text{m}$  before penetrating the host. This may be erroneous because Woolman (1930) and others have observed penetrations in a linear arrangement beneath an external hypha. Penetrations could have occurred at multiple sites beneath a long infection hypha, and Sartoris may have seen only the final penetration. He observed that after (the final?) penetration the external infection hypha collapsed rapidly.

Germination inhibitors. Spores born in masses, as in sori of rusts, often contain germination inhibitors that prevent germination until the spores are diluted by dissemination. Trione (1977) reported water soluble germination inhibitors in both *T. caries* and *T. controversa*. These spores do not germinate rapidly but it is unlikely that germination inhibitors are responsible. When a smutty crop is threshed, the seed may be so covered with spores the grain is visibly discolored. Heald (1921) calculated that such seed averaged 40,000 spores per kernel and produced a crop 70-73% smutted. Germination inhibitors from within the spores must not have been important. Many experimentors add smut spores to the carrying capacity of the seed and heavy smut results.

Some type of internal germination control operates. Slow germination is not due to trimethylamine (Trione, 1977). Some races of *T. caries* produce little trimethylamine (Hanna, 1932), and Hanna (1934) found that the production of the fishy odor was inherited in a dominant fashion in some crosses. An unusual type of nitrogen metabolism may partially regulate germination (Singh and Trione, 1969; Trione, 1973 and 1976). The spores are unusually high in amino acids, and synthesis of proteins may be required prior to germination. The slow rate of spore hydration is also a factor.

Nutrients. Nutrients in the germination medium restrict germination of teliospores. Brefeld (in McAlpine, 1910, p. 71) recommended germinating teliospores in water, then adding nutrients after germination or transferring the sporidia to nutrient media. Stakman in 1913 found that nutrients, other than soil extract, reduced germination (in Lowther, 1950). Glucose and sucrose reduced germination and 0.1% peptone inhibited it completely (Rabien, 1927). Kienholz and Heald (1930) recommended water agar or soil extract agar for both species. Lowther (1950) incubated spores of three races each of *T. caries* and *T. foetida* on 1.5% water agar and upon 2.0% Difco potato dextrose agar which contains 1.5% agar. Germination in 4 days at 16°C averaged 56% on water agar and 3% on the potato dextrose agar. The adverse effect of organic nutrients on spore germination is well established but unexplained.

These relationships establish *T. caries* and *T. foetida* as obligate parasites. In nature they obtain significant energy only from a host.

Inoculation. *Tilletia caries* is the first plant pathogen with which successful inoculations were made (Tillet, 1755). Common bunt was so serious that the king of France offered a reward for the best study of its cause and amelioration. In a time when superstition and speculation were the rule and experimentation rare, Mathieu du Tillet, born in Bordeaux, France in 1730, began to study bunt. Tillet had no training in biology, but his studies are classics of experimental design. He performed field work for 5 years during which he recorded the number of diseased plants in systematic experiments. He put the bunt dust on kernels, on the soil, and passed it through his horse. He concluded that bunt dust on the outside of the wheat kernel was the main means by which the disease developed. Tillet did not consider the bunt dust alive. To him it was a nonliving toxin somehow absorbed by the plant to reappear in the kernels. His study, published in 1755 when he was 25 years old, won the royal prize. We owe a debt of gratitude to the judges who honored this pioneer during his life time.

Tillet distinguished between common bunt (*caries*) and loose smut (charbon) of wheat. He considered them separate entities. When charbon dust (*Ustilago tritici*) was placed upon wheat kernels, charbon did not develop. He also placed corn smut spores (*Ustilago maydis*) on corn kernels, and common smut of corn did not ensue. Only common bunt was produced by inoculating the kernels. When bunt dust was sprinkled on the soil surface a day before wheat was seeded some bunt developed, so he also proved that bunt could develop from soilborne inoculum.

Well before establishment of the germ theory of disease, Prévost (1807) stated that the bunt dust consisted of living spores. He germinated the spores and postulated that the spores germinated on the kernels in the soil, the fungus entered the host, remained alive but unseen within it, until its appearance within bunt balls in the developed wheat plant. Prévost's conclusions were considered highly speculative by his contemporaries and they were not accepted. Prévost illustrated the promycelium, sporidia and secondary sporidia. The Tulasne brothers confirmed the work of Tillet and Prévost and named the genus *Tilletia* and the family Tilletiaceae in honor of Tillet.

Modern workers make a heavy suspension of spores in methylcellulose and apply the suspension to the seeds as a slurry, dry the seed, and plant it. Slurries are of value as a means of reducing the danger of accidental contamination in laboratories using different races of pathogens.

Number of spores on the seed. Early seed treatments that killed spores on the seed (copper sulfate, formaldehyde, mercurials) were obnoxious to work with, and they usually reduced seed germination and seedling vigor. If the relationship between the number of spores per seed and the amount of smut that could be expected in a crop from that seed were known, in cases of low infestation a farmer could be saved the expense and discomfort of treating the seed. In 1902 H. L. Bolley washed grain, centrifuged the wash water, and estimated spore load per seed (Heald, 1921). Aware that many factors affect the amount of smut, Heald (1921) never-the-less attempted to determine how many smut spores per kernel are required to produce a smutted plant. The most smut Heald found was 88% of heads smutted. Seed from such a crop was contaminated by 50,000 to 150,000 spores per kernel during threshing. Heald found that washing kernels in a standard volume of water for 30 minutes removed the spores and that counting them directly in the water suspension was easier than employing a centrifuge and then counting them as done by Bolley.

Table 3. Jones Winter Fife, results all from nature (Heald, 1921).

% of smutted heads in the crop furnishing the seed	No. of spores per kernel	% of smutted plants from the seed
0.49	937	10.40
2.20	833	12.95
5.16	937	17.01
12.35	3437	23.21
17.66	4583	31.61
82.05	40104	70.50

In general, the more smut in the crop, the more spores on the seed and the more smut in the succeeding crop if no seed treatment is used. Heavy straw was burned prior to seeding to eliminate spores on the soil surface so all infection was from seed-borne spores. In addition to correlating the amount of smut in the field with the amount of smut in a crop produced from seed in that field, Heald added known amounts of spores to clean seed and correlated spore load with disease incidence (Table 4).

Table 4. Amount of smut in Jenkins' Club inoculated with known quantities of spores (Heald, 1921).

Weight of smut per 100 grams of wheat	Number of spores per kernel	Per cent of smutted	
		Plants	Heads
0.0	104	0.49	1.89
0.005	458	3.63	2.45
0.01	533	9.52	5.99
0.1	5333	60.79	32.80
0.25	20687	66.23	43.77
0.5	36770	90.82	84.65
1.0	65229	94.43	92.92
2.0	166971	93.38	92.55
3.0	164208	98.88	94.58

From 36,000-150,000 spores per kernel were required to produce maximum smut. At 3.0 grams of smut per 100 g of seed the amount of spores the kernels can hold was exceeded. Most subsequent studies of common bunt followed these results: 100 g of seed was inoculated with 0.5-1 g of spores in routine studies. A 'resistant' spring wheat, Marquis, and a susceptible wheat, Jenkins, were inoculated with graded amounts of smut spores per 100 g of wheat. The amount of smut per unit of inoculum was influenced by the resistance of the host (Heald, 1921).

Leukel (1937) studied the combined effect of spore load, seeding date and wheat variety on the incidence of smut in winter wheat in Virginia. Smut was influenced less by the spore load on the kernels than by the seeding date. Small numbers of spores on the seed are of greatest importance when wheat is seeded in cool soil.

Table 5. Rate of seed inoculation and seeding date affect the per cent common bunt (Leukel, 1937, data of Purplestraw and Fulcaster wheats combined).

Spores: seed, g:g	Seeding date				Average
	9/20	10/2	10/16	11/2	
1:1000	T	3.9	7.0	22.6	8.37
1:500	0.35	6.4	12.3	38.4	14.36
1:250	0.45	7.7	16.3	47.5	17.99
1:100	1.25	10.6	16.7	57.1	21.41
1:50	2.40	10.5	22.6	65.0	25.13
Average	0.89	7.82	14.98	46.12	

Many have wondered why so many spores per kernel are required to produce a smutted plant. Abbé Tessier in France in 1783 found that spores on the embryo end of the kernel produced more smut than those on the brush end. It is probable that no 'synergism' between spores occurs, rather that position of the teliospore germlings relative to the embryonic tissues of the host is important. Inoculum on the seed is relatively fixed in position and the majority of the spores are situated in places from which they cannot incite bunt. Hyphal development from the spores is limited.

Spores in the soil. Common bunt spores have no endogenous means of preventing germination. If the soil is somewhat cool and moist, the spores germinate whether a host is present or not. There is no saprophytic survival in soil and germlings die in the absence of a host. Tillet (1755) produced smut experimentally by scattering spores over the soil surface a day before seeding in November in France. The spores were alive when the wheat was seeded, so infection from spores in soil was possible. H. L. Bolley, working in North Dakota, reported in 1897 that bunt spores did not overwinter in North Dakota. Bolley worked where only spring wheat was grown, and spores disseminated during threshing in the previous summer died before spring wheat was seeded. Appel and Riehm (1913) in Germany and Maddox in Tasmania observed an increase in smut when winter wheat was seeded near the threshing machine shortly after threshing, but by 1914 Appel and Riehm concluded, as had Bolley, that few of these spores survived the winter on or in soil. Only one race of common bunt (T-18) has some ability to overwinter loose in soil (Kendrick, et al, 1964) and this race has some characteristics of the dwarf bunt fungus, *T. controversa*.

Hungerford (1922) added spores to moist soil on 4 September and seeded wheat 0, 7, 20, and 28 days later. Infection declined with days between soil inoculation and seeding from 33% at zero days to 1% at 28 days after inoculation. He concluded that the spores died rapidly in moist soil. In water-saturated soil only a few spores germinated, and after 35 days in saturated soil, when moved to favorable conditions, spores did not germinate and were presumed dead (Woolman and Humphrey, 1924).

Some bunted heads escape harvest and are left in the stubbles. Some bunt balls escape rupture during threshing and are sown with the seed. McAlpine (1910, p. 75) stated that when bunt balls remain unbroken in the soil the spores remain viable until the following crop year and infection can then occur. Heald and Woolman (1915) stated that spores in unbroken bunt balls survive overwinter. They obtained few viable spores after two years when bunted heads were buried. Infection from spores from bunt balls left in the field is not sufficient for significant losses, however.

The one region in which soilborne spores were important was the Pacific Northwest (Oregon, Washington, northern Idaho), the reason the PNW became the bunt 'capital of the world'. When common bunt became important in Washington is not known, but it was already serious when the experiment station began. According to the early literature any disinfectant that kills spores on the surface of the

seed should control common bunt. The first agriculturists at Pullman used copper sulfate, formaldehyde, and hot water, but bunt was not controlled. C. V. Piper in 1893 blamed this failure upon careless seed treatment.

The first two bulletins (Sta. Bull. 54 and a Press Bulletin) published by Assistant Botanist R. Kent Beattie in 1902 explained in detail how to treat seed with formaldehyde and bluestone solutions. Beattie in 1905 published a press release, "Stop raising smut". He concluded his release with a note, "Write for information, or tell your troubles to the Washington Agricultural Experiment Station at Pullman, Washington". Between 1907 and 1917 at least 14 publications on bunt were published in the state, evidence of the extent of the problem (Heald and George, 1918). This steady effort is the result of failure of available seed treatments to control bunt, even though at least 98% of all seed was treated as carefully as possible.

The first clue for the failure of contact fungicides to control bunt in Washington appeared in an unpublished thesis of A. M. Richardson in 1911, according to Woolman and Humphrey (1924) and Heald and George (1918). A suspicion that spores distributed by wind during threshing contaminated the soil was expressed. Spore germination trials in 1913 determined that spores germinated promptly in cool, moist conditions (they had no endogenous dormancy) but in very warm, moist soil they did not germinate, even when the temperature was subsequently lowered (they presumably had died). When a thick layer of straw was burned on the soil surface just prior to seeding undisturbed fallow soil, smut from treated seed was greatly reduced (1915). No smut developed when treated seed was seeded in November on fallow soil, evidence that the soilborne spores had germinated and died by November. Seed treatment had always controlled smut in spring wheat. By 1914 Woolman, in Popular Bulletin 73, stressed the importance of contaminated soil, but the concept of soil infestation was still hazy. In 1915 Woolman and Humphrey (1924) seeded spore-free seed on land on which wheat had never been grown. Seeding began in July. No smut developed in seedlings on or before August 31. Smut in the September 2 seeding was 9%, reaching a maximum of 59% in the September 19th seeding, after which it declined to 0.5% in the October 25th seeding. The only source of inoculum had to be spores from the air. By October 25th in this year most spores of the spore shower had germinated and died. Heald and Woolman (1915), Bulletin 126, explained the failure of available seed treatments: they did not protect against new spores in the soil. The coleoptile, emerging through infested soil, became infected. Bulletin 126 stated, "The Office of Cereal Investigations of the Bureau of Plant Industry, USDA, contributed \$300 toward these investigations."

On September 9, 1915, D. C. George counted 145,272 *Tilletia caries* spores on a single apple leaf in the college orchard at Pullman. During the first week in September, 3,058 spores per square inch were caught on a spore trap. In the week of September 5-11, 1916, one spore trap caught 10,788 spores per square inch. The average total spore-fall per square inch at Pullman in 1916 was 36,113. These and other experiments established the importance of spore showers as sources of soilborne inoculum in the Pacific Northwest in winter wheat. Soilborne spores were of little significance in spring wheat because spores freed from the bunt balls during harvest germinated on or in cool, moist soil before winter.

In the summer fallow system a crop is grown every second year in a field, and the field without a crop is cultivated shallowly to conserve moisture. Harvested fields and bare lands are interspersed. Spores from the harvest accumulate on the dry surface of the fallow fields. The spores remain viable until the soil cools and rains begin in the fall. At seeding the spores are mixed with the surface layers of soil and seedlings emerged through the infested soil (Heald and George, 1918; Woolman and Humphrey, 1924). The 36,060 spores per square inch of surface soil provide 769 spores per cubic cm to a 7.5 cm depth, or about 592 spores per gram of soil.

In desperation, plowing the summer fallow with a moldboard plow to bury the contaminated surface soil prior to seeding was tried. Smut was greatly reduced, but the tillage resulted in lost moisture and added

expense. Burying the surface soil is impractical. Spores on the soil surface can be destroyed by burning large amounts of straw on the soil surface, but this is impractical.

By 1918, Heald and George (Bulletin 151) had put the clues together and explained why the Palouse became known as the bunt capital of the world. Wheat seedlings become infected during the early stages of seedling development, either from spores on the seed or in the soil near the base of the coleoptile. Copper sulfate, hot water, and formaldehyde are effective only against spores on the seed, not those in the soil. During harvest spores in the air contaminate the surface of the fallow soil. The winds are mostly from the west and southwest, so many spores arrive in eastern Washington. Rainfall at Pullman, 1915-1917, in July averaged 0.41 inches, in August 0.41 inches, and in September 0.72 inches. The dry surface of the fallow soil during harvest prevented germination of the spores. During shallow tillage and seeding, viable spores are mixed in the surface inches of soil so that they contact seedlings during germination and seedling emergence. In most parts of the world spores fall on moist soil and the spores germinate before winter wheat is planted; there is no soil infestation and surface disinfectants of seed control bunt. Although Heald and George put the story together, credit should be given to Humphrey and Woolman for experiments that established the role of spores in soil in the PNW, most of which was done before the Heald and George bulletin was prepared.

Copper carbonate. The next real advance was substitution of copper carbonate for copper sulfate or formaldehyde. Darnell-Smith in Australia found copper carbonate superior to the other chemicals and this was supported by experience in California. Copper carbonate is essentially insoluble in water and it is applied dry as a dust. Copper sulfate and formaldehyde are applied to seed in water. George Zundel (1921) proved that Washington wheat was unusually susceptible to injury because very dry, brittle kernels sustain microscopic cracks during threshing through which toxic chemicals in solution enter the kernels. Hand-threshed wheat treated with copper sulfate germinated as well as or better than untreated kernels (96-99%). Germination of machine-threshed kernels treated with copper sulfate was 43%, without treatment, 96%.

Heald and L. J. Smith (1922), an agricultural engineer, developed a seed treater for copper carbonate. Seed treated with copper carbonate had 97% germination; with copper sulfate, 66%; and with formaldehyde, 46%. Even though all these treatments were effective only against spores on the seed, not those in the soil, copper carbonate was a benefit to farmers because they could reduce their seeding rate and still obtain better, more vigorous stands of wheat. Zundel (1923) found that some farmers had troubles with copper carbonate because some merchants supplied a poor product. To be effective, at least 90% must pass through a 200 mesh screen and it must contain at least 50% metallic copper. It should be fine for better adherence to the seed and it should be applied in a closed machine. It quickly replaced copper sulfate and formaldehyde.

Hexachlorobenzine. The role of spores on the surface of the kernels made common bunt a prime candidate for control by seed treatment, more so because in Europe, where the early work was done, soilborne spores were of little significance. The early history of practices to remove or kill spores on the seed surface, starting with the use of salt water in 1650, is reviewed by Fischer and Holton (1957, p. 423-431). In more-or-less chronological sequence, salt, lime, urine, wood ashes, copper sulfate, mercuric chloride, formaldehyde, copper carbonate, organic mercuries and hexachlorobenzene (HCB) are discussed. Copper carbonate was the first material that killed spores on the seed without excessive phytotoxicity. Organic mercuries largely replaced copper carbonate even though they are hazardous to man and animals.

Mercury was expensive and hard to get in France during World War II and hexachlorobenzene (HCB) was found to inhibit germination of common bunt spores. HCB has a narrow spectrum, lacking adequate efficacy for essentially anything but bunt. Holton and Purdy (1954) and Purdy (1955) found that at high but practical dosages HCB was effective against both seed- and soilborne spores. HCB was the first treatment that controlled common bunt where soilborne inoculum played a major role.



In experiments HCB was effective but it failed to control bunt in some fields when first used commercially; some fields developed 50% smut. The early commercial failures were the result of nonuniform seed treatment by the commercial seed treaters of the time. The machines were modified to apply HCB in slurries and the problem was overcome. Soon after the adoption of HCB common bunt was controlled for the first time (about 1956) in the PNW. Research on common bunt in this region stopped within a few years. HCB (also PCNB) is no longer an approved fungicide because of its long residual 'life' in soil. Carboxin, a systemic fungicide, though less effective than HCB for common bunt of wheat, also controls loose smut and flag smut.

The role of soilborne inoculum in the epidemiology of the common bunts in the PNW was confirmed by success with hexachlorobenzene (and PCNB), the first seed treatment effective against soilborne inoculum (Purdy, 1958). Hexachlorobenzene vapors prevent teliospores in the vicinity of the coleoptile from germinating (Purdy and Holton, 1956).

Infection. In general usage, infection refers to establishment of the pathogen within the host. Disease does not necessarily follow. Smut pathologists, however, generally use infection to refer to disease development following inoculation. This is technically incorrect, but I follow smut pathologists in discussing bunt.

Host penetration. *T. caries* penetrates the host directly, mostly near the root node (= scutellar node) according to Kühn in 1859. Wolff in 1873 reported entrance through the coleoptile. No one has observed penetration of the host by *T. caries* or *T. foetida* by the promycelium itself. Formation of the infection hypha follows production of primary sporidia and their fusion. There is no evidence that penetrations of the pericarp or tissues below the coleoptile play any role in disease development (Swinburne, 1963), so the penetrations of the scutellar node observed by Kühn are of no consequence. Kühn in 1859 and Lang in 1917 commented upon the difficulty of following the fungus once it is within the host. In both inter- and intra-cellular hyphae the cytoplasm is concentrated behind the growing hyphal tips with thin-walled empty hyphae behind. The active, assimilative portion of the hyphae of smuts is sparse within the host during vegetative development of the plant, and this characteristic has made pathologic histology difficult to study (see Kolk, 1930 and Woolman, 1930, for reviews of early studies).

No host penetration had occurred 3 days after inoculated seed was incubated in moist soil at 15-18°C (Swinburne, 1963). The primary sporidia had fused (= H-cells) and the plumules were 2 mm long. In 4 days the coleoptile was 5 mm long and penetration of the pericarp, but not the coleoptile, was seen. In 6 days the coleoptiles were 25 mm long, they were beginning to emerge from the soil, and penetration near their bases was in progress. On day 7 halos were present in epidermal cells about the penetration sites in the lower 6 mm of the coleoptiles and the fungus was within the epidermal cells.

When the coleoptile emerges through 5.5 cm of moist, cool soil heavily infested with teliospores, 75% of the penetrations occur in the basal 2.5 cm of the coleoptile (Woolman, 1930), even though the entire coleoptile is exposed to germinated spores. Over 100 penetrations per plant, either resistant or susceptible, were the rule in heavily inoculated soil. Penetration (phase 1 of Woolman) was complete when either inter- or intra-cellular penetration of the coleoptile had occurred. The coleoptile was penetrated more quickly and over a wider area from soilborne inoculum (Woolman, 1930) than from seed-borne spores (Swinburne, 1963). Hansen (1958) found callosities, even in susceptible wheat, in the epidermal cells of the coleoptile at the penetration site.

Development within the host. After initial penetration, (phase 1 of Woolman) the fungus advances more quickly in susceptible than in resistant wheats to phase 2, which is development within the coleoptile and penetration of sheaths of true leaves within the embryo (Woolman, 1930). In Hybrid 143, very susceptible, 25% of the initial infections from soilborne spores reached phase 2 in 7 days. In Martin and Hussar, resistant wheats, about 5% of the infections reached phase 2 in 20 days. In phase 3 the fungus

developed intercellularly in very young leaf blades, nodes, internodes and the growing point. In susceptible wheats 1 to 4 infections reached phase 3 in 20 days, while in resistant wheats none did. Woolman concluded that in resistant wheats the fungus progressed so slowly it failed to establish within the growing points before they developed beyond reach. No more than four advanced infections of the 100+ initial penetrations were found in susceptible wheat, evidence of fungal retardation even within highly susceptible wheats. Most of the susceptible plants had one or two advanced invasions.

Progress within the host is so slow that when germinated wheat seeds with plumules 2-5 mm long were dipped in compatible sporidial suspensions of *T. caries*, a procedure which accelerates the time from inoculation to host penetration, *T. caries* reached the growing point in 30 days at both 3 and 15°C (Hansen, 1959). In 31 days the fungus from spores on the seed was in the terminal meristem of a spring wheat (Swinburne, 1963). It enters tiller buds from subtending, infected leaf bases. If internode elongation begins before infection of terminal meristems the latter escape infection. Once hyphae reach the primordium of the spike the fungus is carried upward passively by stem elongation. Both Hansen (1958) and Swinburne (1963) occasionally found living hyphae in nodes and leaves after stem elongation. Swinburne found no hyphae in the rachis or auxiliary floral structures at heading, but only in the pistils of the developing spike. Two weeks later live fungus was present in the young bunt balls, the last internode, and occasionally in the upper two nodes beneath the spike. At no time was the fungus able to penetrate differentiated vascular tissue. Trione (1972) isolated *T. caries* readily from young ovaries and from about 70% of the last stem internode within 2 cm of the spike. The fungus was not recovered below that level. Even though the leaves were flecked the pathogen was not isolated from leaves.

Depth of seeding and position of spores. Deep seeding increased common bunt of both spring and winter wheat, whether the inoculum was on the seed or in the soil (Woolman and Humphrey, 1924) (Table 6).

Table 6. Smut (%) in spring- and fall-seeded wheat, seeded at various depths from 1.25-7.5 cm deep, Pullman, Washington, 1914. (Woolman and Humphrey, 1924).

Seeding depth, cm	Spores on seed		Spores in soil	
	March 17 <sup>a</sup>	Oct 4 <sup>b</sup>	March 17	Oct 4
1.25	35	12	48	50
3.75	99	92	75	62
7.50	100	97	91	82

<sup>a</sup> Wilbur wheat

<sup>b</sup> Hybrid 143 wheat

The necessity of the fungus to maintain its position relative to host development is illustrated by the effect of seeding depth, even when the spores are on the seed (Swinburne, 1963). Deep seeding retards development of the coleoptile, increasing the period during which the terminal meristem is accessible. When seeded 4 cm deep, at 23 days the first leaf was 100 mm long and the second leaf was 46 mm long. When seeded 7 cm deep, the first leaf was 48 cm and the second leaf was not visible. Smut averaged 38% in the 4-cm seeding, 51% in the 7-cm seeding. Swinburne attributed the 13% increase in smut to slower host development from deeper seeding.

Table 7. Smut in Cappelle-Desprez at different seeding depths and dates from spores on the seed (Swinburne, 1963).

Depth, cm	Dates				Average
	Oct 7	Nov 7	Nov 29	Jan 16	
4	1.6	58.0	58.6	32.6	37.7
7	4.8	83.9	75.1	41.7	51.4

The effect of depth of seeding on the incidence of smut is not simple (Woolman and Humphrey, 1924), but as the depth of soil above the infested layer increased, smut increased.

Pre-germination of spores. Small differences in speed of spore germination among races of the pathogen are not important, yet rapidity of infection can be a factor under some conditions according to trials with pre-germinated spores. When smut-free seedlings of differing ages were inoculated with ungerminated teliospores or with sporidia, older seedlings were more susceptible to sporidial inoculum (Sartoris, 1924). When seedlings up to 4 days old were inoculated, no real differences were observed. In seedlings 7 days old or somewhat older, sporidial inoculum was more successful than ungerminated spores (Table 8). Using sporidia as inoculum lengthened the susceptible period about 3 days. The temperature of this trial was not given but it was obviously favorable to common bunt.

Table 8. % infection of seedlings of different ages by teliospore and sporidial inoculum (Sartoris, 1924).

Inoculum	Age of host when inoculated, days												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Teliospores	100	100	100	88	74	74	11	11	2	2	0	0	0
Sporidia	100	100	100	91	87	82	63	65	42	13	3	2	0

This experiment demonstrates the duration of susceptibility in the developing seedlings, and it may explain why small differences in rapidity of germination of teliospores among races are unimportant. In this trial wheat was fully susceptible to ungerminated spores applied to seedlings 3 days old, so the period of full susceptibility is long enough that under favorable conditions all viable spores should have germinated. Selection to synchronize host and pathogen doubtless began with the earliest threshing procedures, predating written records.

Temperature. Spore germination. The response of teliospores to temperature was first studied by Prévost (1807) who reported that spores of *T. caries* germinated in 2.5-3 days at 17-18°C, yet no worker has studied a complete, detailed range of temperatures. Most have used three or four temperatures between 5 and 25°C. Many list the minimum near 4 or 5°C, but this is erroneous. J. Hoffmann (personal communication) reports that teliospores can germinate down to -1 to -2 C, depending on the substrate. Woolman and Humphrey (1924) saw early stages of germination at 1.4°C in 25 days. Most workers did not expose spores to such low temperatures or wait so long to observe the results. Wheat seed germinated more rapidly than teliospores at 1.4°C, with the plumule reaching 1 mm in 10 days and 7 mm in 25 days. *T. caries* spores do not germinate at 28-34°C (Volkart, 1906). Woolman and Humphrey (1924) reported germination in 2 days at 19°C, the most rapid, and no germination at 29.1°C. When spores incubated 18 days at 29.1°C were moved to a favorable temperature they did not germinate and were presumed dead. The survival of these spores in warm, moist soil is limited.

The literature is replete with conflicts as to minimum, optimum and maximum temperatures, and Lowther (1950), who worked after races were known, explained many of the conflicts. He studied 16 races of *T. caries* and nine of *T. foetida* simultaneously under the same conditions. As a species, *T. foetida* germinated slightly more rapidly than *T. caries* on 4% water agar, but much variation existed among

racess within species. T-5, T-6, T-7, and T-8 of *T. caries* germinated relatively rapidly at all temperatures, T-10 and T-13, slowly. The optimum temperature, or the temperature at which 75% germination was first reached, varied as much as 5°C between races. Some races were fast at all temperatures, some slow. No one temperature was optimum for all races. It is no wonder that results of various workers, who worked with different spores of different collections and races on various media, differ. See Lowther (1950) for a review of temperature relations.

Variations among races within the species in response to temperature may be evidence that precise adjustment of the pathogens to temperature is not advantageous. Wheat is seeded under different conditions from year to year. Hybridization occurs within and between species and races, and if a particular temperature response was advantageous, natural selection theoretically should have accomplished more uniformity.

All Washington studies since Flor (1933) (Purdy, Kendrick, Holton, Lowther, Fischer, Meiners, etc.) used the same fungal accessions (races) as defined by virulence on differential varieties. These races are defined only by virulence. Would the background genome of L-2 from Kansas, Washington, or Europe, or from a different fungal cross, actually be the same? The same arrangements of virulence genes could arise from different sources with different background genomes, and accordingly, L-2 accessions from different sources may not have the same response to temperature. To my knowledge no one has compared races of different origins in this way.

Speed of spore germination and competition. Rapidity of germination may not affect competitive relationships. When T-5, T-6, T-7 and T-8, all pathogenic on Elmar and all rapid germinators (Lowther, 1950), were mixed in equal proportions, T-6 produced smut in 29/30 plants (Kendrick and Holton, 1961). T-6 did not dominate by speed. In the field in early days races L-1 and T-1 were prevalent in the absence of specific resistance genes in the host (Kendrick and Holton, 1961). Both races are in the second-fastest germination group (Lowther, 1950). Race T-5 of *T. caries* responded to temperature in moist natural soil in about the same way it did in culture (Purdy and Kendrick, 1957), evidence of agreement between germination in soil and on agar media.

Rodenhiser and Holton (1953) mixed T and L races to study their competitive abilities on a fully susceptible wheat. L-2 and L-8 quickly eliminated T-2 from the mixtures, and T-1 eliminated L-2 after several passages through the host (Table 9). What determines success in mixtures, obviously not speed of spore germination, is not known.

Table 9. Percent of the L-races after mixing equally with T-races in Ulka wheat at Aberdeen, Idaho (Rodenhiser and Holton, 1953).

Spore Mixture	Year							Germination Speed	
	1	2	3	4	5	6	7	L <sup>a</sup>	T <sup>a</sup>
L-2 + T-1	27	22	16	16	5	0.1	0	3 <sup>b</sup>	2
L-8 + T-1	39	34	32	29	16	6	4	2	2
L-2 + T-2	79	98	100					3	2
L-8 + T-2	79	85	100					2	2

<sup>a</sup> L = L-races, T = T-races

<sup>b</sup> Speed of germination classes, 1 = fastest, 5 = slowest, after Lowther, 1950.

Temperature. Disease development from seed-borne spores. After reviewing the voluminous literature, Holton and Heald (1941) concluded that soil temperatures of 5-15°C were most favorable to smut development, that either higher or lower temperatures retarded it. The importance of temperature was realized early in Europe, and in 1906 Volkart found that at 5-6°C, his lowest temperature, wheat seed germinated in 12 days, spores of *T. caries* in 15 days. At his highest temperature, 28-34°C, wheat seed

germinated but the spores did not. Hecke in 1909 expanded studies of the relative response of host and pathogen to temperature. Smut was favored by seeding in cool soil, whether in fall or spring.

Unexplained and contradictory results of seeding in very cool soil were reported. Feucht (1932) seeded winter wheat in Germany, October 26 to December 6, 1928. The soil temperature declined from 10°C in the earliest date to 2.2°C at the final seeding, and it declined to 0°C shortly after. No smut developed in the last seeding. Woolman and Humphrey (1924) seeded winter wheat from July to November 21 in Washington. No smut (*T. caries*) developed in the earliest dates when the soil was 21°C or above. When the soil was 10-4°C, smut incidence was above 80%. In the November 21 seeding the soil temperature averaged 2.7°C from seeding to 10 days after seeding. The wheat did not emerge until the following spring, but 21% of the plants were smutted. Melchers (1941) had similar experience in Kansas. A seeding date trial in 1926 in Kansas started with 0 smut (*T. foetida*) in Sept 4 and 11 seedings, high amounts of smut in seedings between October 9 and November 14, and 20% smut in the last seeding date (November 21) in which plants survived the winter. The soil temperature at the November 21 planting was slightly above freezing. In Feucht's trial, no smut developed in the coldest soil. In the other trials smut developed even when the wheat did not emerge until the following spring. Did the fungi develop slowly within the dormant host or were the spores and germlings preserved external to the host in Washington and Kansas by cold and invade during the following spring?

Both *T. caries* and *T. foetida* infected many seedlings at 5 and 10°C, somewhat fewer at 15°C, with little disease at 20°C or above from seed-borne inoculum (Faris, 1934, Table 10). The soil was pH 7 at 62% moisture holding capacity and the seedlings were incubated to the two-leaf stage at the indicated temperature after which they were all placed at 20°C or higher.

Table 10. Percent of smutted plants from inoculated seed (Faris, 1934).

Inoculum	Temperature, 0°C					
	5	10	15	20	25	30
<i>T. caries</i> from Washington	54	56	35	3	0	0
<i>T. foetida</i> from Washington	56	50	31	3	0	1
<i>T. foetida</i> from Missouri	<u>49</u>	<u>66</u>	<u>31</u>	<u>2</u>	<u>0</u>	<u>0</u>
Average	53	57	32	3	0	0

Faris marked the seedlings that emerged first. At 5°C smut averaged 82% in the fastest, most vigorous seedlings; 49% in the slower less vigorous seedlings. Results at 10 and 15°C were similar. The host did not outgrow the fungus at temperatures favoring smut. Faris concluded that rusts and smuts, unlike *Gibberella zeae*, were not dependent upon predisposition of the host. Seedlings were most vigorous in the 5-15° range, the temperatures favoring smut. Leukel (1937) also found that the most vigorous seedlings at 6-15°C had the most smut and did not grow away from the fungus, but at higher temperatures it is believed to do so.

In contrast to the results of Faris (Table 10) when Leukel (1937) compared *T. caries* and *T. foetida*, *T. foetida* differed from *T. caries* in being more effective at higher temperature (Table 11). Leukel speculated that this response to temperature might affect the geographic distribution of the species.

Table 11. Percent smut in four spring wheats (Leukel, 1937).

	Temperature, °C				
	6	10	15	20	24
<i>T. caries</i>	52	75	40	0	0
<i>T. foetida</i>	40	73	63	5	0

The time required for establishment within the host to produce systemic infection was determined by Kendrick and Purdy (1962). Heavily infested seed was incubated in moist sand at 5, 10, 15 and 20°C. Seedlings were removed daily and surface-sterilized with an organic mercurial fungicide after which the plants were grown in smut-free soil. The transplanted seedlings were incubated at 21°C for further development. Establishment within the host beyond the reach of the mercury began in 15 days and was complete in 24-25 days at 5°C. It began in 9 days and was complete in 13-16 days at 10°C. It began in 6 days and was complete in 9-11 days at 15°C. It began in 4 days and was complete in 7-8 days at 20°C: the seedling emerged in 5 days so the fungus was still establishing itself after the host had emerged. T-5 germinates rapidly, L-4 and L-9 intermediate, and T-13 relatively slowly at all temperatures. The final results do not clearly demonstrate that differences in rapidity of germination influenced the results (Table 12), except that T-5, the fastest germinator, was more effective at 15 and 20°C.

Table 12. Systemic infection of wheat by *Tilletia caries* (T-5, T-13) and *T. foetida* (L-4, L-9) at different temperatures (Kendrick and Purdy, 1962).

Race, germination	Temperatures, °C				
	5	10	15	20	Average
T-5 (fastest)	88	80	70	70	77
T-13 (slowest)	77	85	61	45	67
L-4 (medium)	70	80	39	47	59
L-9 (medium)	79	80	38	25	55
Average	79	81	52	47	

The effect of soil temperature on the percent of smut is usually determined prior to seedling emergence (Faris, 1924), but Leukel (1937) subjected this generality to test. Inoculated seed were incubated in soil at 6, 13 and 26°C until emergence after which the seedlings were subjected to differing post-emergence temperatures (Table 13). Pre-emergence temperatures were very important.

Table 13. Effect of pre- and post-emergence temperature upon systemic infection by *Tilletia foetida* (Leukel, 1937).

Pre	Temperature			Temperature			Temperature		
	Post	Smut		Pre	Post	Smut	Pre	Post	Smut
6	6	96		13	6	94	26	6	12
6	13	94		13	13	93	26	13	0
6	26	83		13	26	96	26	26	0

The studies of Faris (1924, 1934) and Leukel (1937) established the importance of pre-emergence soil temperatures to common bunt. They represent the normal, expected host-parasite interactions. Some atypical effects of post-emergence temperatures upon smut development are known, however. They usually result from interactions with resistance genes in the host. When Hope wheat was held at 9°C until maturity it was 100% smutted. When moved to 21°C after emergence it was 2% smutted (Smith, 1932).

Post-emergence resistance developed in Hope at the higher temperature. Baart is highly susceptible, Baart 38 contains Martin factors (*Bt1* & 7) for resistance (Zscheile, 1955). When Baart (susceptible) was inoculated with T-1, incubated at 10°C until emergence, and either held at 10°C or moved to higher temperatures after emergence, all plants were smutted. When Baart 38 (with Martin resistance) was moved to a warm greenhouse after emergence no smut developed. When Baart 38 was left at 10°C past the seedling stage smut developed. The resistance of Baart 38 could be partially overcome by continued cool conditions.

Temperature. Spores in the soil. Woolman and Humphrey (1924) observed that at 20°C wheat was practically immune to infection from spores on the seed, but not necessarily from spores in soil. Convinced that relative rapidity of host and pathogen development at 20°C could explain the low incidence of bunt from seed-borne inoculum, Purdy and Kendrick (1963) gave the fungus a head start. They inoculated soil 7 days before planting or at planting time with T-5, a race well adapted to a wide temperature range (Table 12). When the host and pathogen were introduced into moist soil simultaneously, disease incidence was high at 5 and 10°C. When they gave the fungus a 7-day head start in moist soil, disease incidence was high at 5-20°C, with some infection to 25°C (Table 14). The head start enabled the fungus to establish itself in rapidly developing seedlings.

Table 14. Systemic infection of wheat when the soil was infested 7 days prior to planting and at planting time with teliospores of T-5 (*T. caries*) (Purdy and Kendrick, 1963).

Soil infested	5	Temperatures				
		10	15	20	22	25
prior to planting	95 <sup>a</sup>	97	93	80	10	3
at planting	85	90	40	3	0	0

<sup>a</sup> Averages for Palouse silt loam at 13-24% water contents

Photoperiod. In a standard smut nursery grown over a wide geographic area, Rodenhiser and Holton (1942) noticed that smut in some wheats (Marquis, Canus) was highly variable and constant in others (Ulka). The variations were not attributable to races of the pathogen. To investigate this, Rodenhiser and Taylor (1943) grew Canus and Ulka under 11-hour and 24-hour (constant light) photoperiods. Both wheats headed in 53 days under constant light. Canus headed in 83 days in the 11-h photoperiod, Ulka in 87 days, so developmentally they both responded similarly, but not to smut (Table 15). They concluded that the smut reaction of Canus was strongly influenced by light. Accelerated host development under constant light did not reduce smut as would seem logical. Some wheats are more susceptible when seeded in the fall than in the spring. Whether this is due to longer exposure to low soil temperatures from fall seedings or to photoperiod is not known. Differences of this type are rare.

Table 15. Response of Canus and Ulka to *T. foetida* and *T. caries* under different light regimes (Rodenhiser and Taylor, 1943)

Inoculum	Canus		Ulka	
	11 h	24 h	11 h	24 h
<i>T. foetida</i>	3	49	75	87
<i>T. caries</i>	3	41	73	85

Water. Teliospores of *T. caries* were germinated by Prévost (1807) who observed promycelia, primary and secondary sporidia in water. Water quality was important. Teliospores germinated in rain water but not in well water that contained copper from a pipe. When spores were deep within a water droplet the

promycelia were long and septate and the primary sporidia formed above the surface of the droplet (McAlpine, 1910, p. 70-71). When not deep in the droplet the promycelia were aseptate, short and stout (normal). Trione (1977) observed that a thick water film on an agar surface tended to retard germination.

Spore survival after extreme dehydration is probably enhanced by slow hydration. Air-dried spores of *T. caries* with a water content of 2.2% required 24 hours to reach 8.5% water and by 50 h they contained 28.3% water and the volume increased 117% (Trione, 1977). In an effort to study the effect of hydration on spore germination, Lowther (1950) germinated spores on water agar varying in agar content from 1% (very weak) to 6% (very hard and strong). The strong agar delayed the germination of *T. caries* more than *T. foetida* (Table 16). Because spores of *T. foetida* are smooth and those of *T. caries* are ornamented with reticulations, it is possible that a larger portion of the smooth spore wall made contact with the hard agar surfaces, enabling those of *T. foetida* to absorb water more rapidly, or are spores of *T. caries* more slowly hydrated for some other reason?

Table 16. Germination of spores of *Tilletia caries* (3 races) and *T. foetida* (3 races) on the surface of water agars at 16°C within 4 days (Lowther, 1950).

	% Agar in medium							
	1.0	1.5	2.0	2.5	3.0	4.0	5.0	6.0
<i>T. caries</i>	51	37	42	33	22	11	10	6
<i>T. foetida</i>	75	75	75	75	75	65	61	54

<sup>a</sup> Lowther considered 75% germination as maximum because he could not count germinated spores among promycelia and sporidia beyond that condition.

Strong agar retarded germination but it did not prevent it. By 5 days spores of T-5, T-10, L-8 and L-9 reached maximum germination (75%) on 6% water agar and T-11, the slowest, reached 75% germination in 9 days. It is possible that hydration of wheat seed on the various agars would vary similarly. *T. foetida* is strongly dominant in drier parts of Hungary (Bohus and Podhradsky, 1952), and they reported that *T. foetida* spores germinated best in a brown clay at 59% moisture holding capacity (MHC), and *T. caries* at 71-84% MHC. Clay at 71-84% MHC is too wet to till properly.

In Palouse silt loam, germination of race T-5 spores of *T. caries* was greatest at the water holding capacity of the soil (24% by weight, about -0.3 bars) and least at 9% water by weight. The water content at permanent wilting point is 10.8% (approximately -15 bars) (Table 17). Ten days is a long time at 20 and 25°C. Data taken also at 5 days would have been useful.

Table 17. Percent germination of *Tilletia caries* spores, race T-5, in soil after 10 days (Purdy and Kendrick, 1957).

Soil water content, %	Temperature, °C					Average
	5	10	15	20	25	
9	0	2	2	0	0	1
11	60	60	26	2	2	30
13	82	70	63	10	10	47
18	90	85	80	42	39	67
24	<u>90</u>	<u>90</u>	<u>90</u>	<u>48</u>	<u>43</u>	72
Average	64	61	52	20	19	



Early experiments on the effect of soil water on disease development are difficult to interpret because water contents are generally not expressed in terms that we can visualize. Smut is decreased in saturated soil, soil wetter than its water holding capacity (Hungerford, 1922; Woolman and Humphrey, 1924). In normal field practice wheat is seeded in soils dry enough to permit tillage without harming soil structure, well below the moisture holding capacity (MHC). Hungerford (1922) obtained little smut from seed-borne inoculum in soil at 32% water (28-32% is near saturation in this soil), with 40-90% smut between 14 and 22% soil water content.

The effects of soil water content on spore germination and disease incidence may differ. Woolman and Humphrey (1924) inoculated soil with spores of *T. caries*, brought it to certain initial water contents by weight and planted it. No water was added until the seedlings were transplanted. They, like Purdy and Kendrick (1957), obtained their soil from near Pullman. If the two soils were fairly similar, the reference points given by Purdy and Kendrick are useful (24% = moisture holding capacity, 10.8% = permanent wilting point). Smut incidence was greatest in their drier soils (Table 18).

Table 18. Effect of soil moisture on smut incidence in inoculated soil (Woolman and Humphrey, 1924).

Water content in % at		
Seeding	Transplanting	Smut, %
15.8	11.1	27
22.6	17.6	31
26.2	20.5	13
30.0	25.0	2
32.6	27.5	0

In summarizing the literature Holton and Heald (1941) concluded that smut was favored by soils drier than 60% MHC. Soil moisture content, according to my interpretation of the results of Purdy and Kendrick (1957, 1963), is of minor consequence within practical soil water contents. In soil heavily infested with race T-5 and seeded the same day, soil water from just above the permanent wilting point (10.8% water) to the moisture holding capacity (24% water) had little effect on disease incidence between 5 and 15°C. Spore germination was retarded in the driest soil, but host development must have been equally retarded because disease development was not. These results from simultaneous soil inoculation and seeding are probably equivalent to what would happen from spores on the seeds. This speculation is supported by the conclusion of Fischer and Holton (1957 p. 200) that the optimum temperature for common bunt, based mostly on seed contamination, is 6-10°C.

When the soil was inoculated 7 days prior to seeding (Table 14), giving the fungus a head start, soil water content was not a major factor. In very dry soil [10% water (PWP=10.8%)] smut was 100% at 5°C, 96% at 10°C, 45% at 15, 30% at 20°C and 0 at warmer temperatures. Purdy and Kendrick (1963) demonstrated a remarkable adaptation of *T. caries* to soil water contents encountered by wheat seedlings during emergence. I conclude that when soil can be tilled and seeded and moisture is adequate to sustain germination and emergence of wheat, *T. caries* will infect the wheat.

The observations on the response of spores to water on agar surfaces and in soil are not in conflict. 'Thick' water films on an agar surface, which retard germination, are far thicker than a water film in normal soil at its water holding capacity. At the permanent wilting point water films are very thin. Statements that wet soil reduces smut (Hungerford, 1922) are based upon soil wetter than at moisture holding capacity, soil essentially saturated and low in oxygen (Leukel, 1937). (Rollie Line - this statement applies equally to flag smut.)

Assuming that differences in geographic distribution of *T. caries* and *T. foetida* are the result of adaptation and not of chance, comparisons of their distribution with total annual precipitation is dangerous. The only time that soil water affects the infection process from seed-borne inoculum is at and shortly after seeding.

Soil type and pH. When seed inoculated with spores is sown in light sandy soils less smut develops than when it is sown in clay loam or rich black loam (Leukel, 1937). When a sandy soil was adjusted to pH 5.6 and 7.9, smut increased from 6% in the acid soil to 42% in the alkaline soil (Leukel, 1937). Rabien (1927) gives pH 5.0 as the lower limit for germination of *T. caries* spores in soil (in Leukel, 1937). I found few studies on the effect of the soil itself or soil pH on common bunt.

#### *Breeding for resistance to common bunt in the Pacific Northwest*

William Farrer was breeding wheats resistant to common bunt in Australia in 1901 (Churchward, 1932), before such efforts in the Pacific Northwest (Oregon, Washington, Idaho), but breeding for resistance received more emphasis for a longer period of time in the PNW than anywhere else in the world because of the problem with soilborne smut spores in winter wheat. Seed treatment controlled bunt on spring wheat because spores in the soil did not survive the winter in substantial numbers so breeding for resistance was important only in winter wheats. From the time of realization that available seed treatments would not control bunt, breeding for resistance was the first line of attack.

Major genes are those which confer a high level of resistance to a given race or to several races of *T. caries* or *T. foetida*. Minor genes confer lower levels of resistance to one or several races. Minor genes are more difficult to study, and because of their lower level of effectiveness, they have been little studied. Because this is an historical account I refer to major resistance genes as done in early papers. Resistance genes were named after the wheat in which they were found. Farrer developed Florence, a wheat with a major recessive gene for resistance (Churchward, 1932). The resistance gene of Florence was transferred by E. F. Gaines to a hard red winter wheat called Ridit in 1915. Ridit was released in Washington in 1923. The gene from Florence became known as the Ridit gene, *rd*. My apologies to Australia.

The first successful hard red winter wheats grown in the US were 'Turkey' wheats. They were introduced mostly from Russia near the Black Sea and north of the Caucasus Mountains. Several of these wheats possessed what became known as the Turkey gene (*T*) but many did not. Stephens and Woolman (1922), after testing many wheats, recommended Turkey C.I. 1558A and B, Crimean C.I. 2903-5, Turkey 3055, Crimean C.I. 4430, and Turkey 1561C as sources of resistance among Turkey-type wheats. Rio (C.I. 10061) was selected from a Crimean wheat in 1920 at Moro, Oregon (Clark and Bayles, 1942). Rio possessed a gene for resistance designated as the R gene.

High resistance was found in Martin, a soft white wheat. Martin actually contained two major genes,  $M_1$  and  $M_2$ . In the early years the resistance of Martin was attributed to a single gene ( $M_1$ ), because discovery of the second gene was not possible until knowledge of races of the pathogens had advanced to a higher level. Martin genes were present in some other wheats, either alone or together, but Martin was the parent used most by breeders. E. F. Gaines released Albit in 1926. It had the  $M_1$  gene. Albit resulted from a cross between Hybrid 121 X White Odessa (C.I. 4655). The  $M_2$  gene is present in Federation, which was bred by Farrer in Australia.  $M_1$ ,  $M_2$ , *T*, *R*, and *rd* are the genes for resistance most widely used in the region and the resistance genes to which the pathogens were most exposed through time.

Sequence of cultivars in the PNW. Between 1900 and 1930 most of the wheats grown in Washington and Oregon, including Turkey types, Goldcoin, and Hybrid 128 were highly susceptible (Kendrick and Holton, 1961). Hybrid 128 could be seeded either in the spring or fall, and its high yield potential made it the leading wheat. It has no known genes for resistance. The introduction of Federation ( $M_2$  gene) from Australia, the presence of some Turkey types with the *T* gene, the release of Ridit (*rd*) in 1923 and Albit

( $M_1$ ) in 1926 exposed the smut population to resistance genes. The resistant cultivars were agronomically inferior to the highest yielding susceptible wheats, and susceptible wheats dominated.

Flor (1933) made the first significant survey of races of *T. caries* and *T. foetida* present in the region and found races T-1 and L-1 most common. T-races are *T. caries*, L-races are *T. foetida*. Dwarf bunt races (*T. controversa*), are called D-races. T-1 and L-1 are avirulent races. Virulent races capable of attacking resistance genes were present but in low numbers (Flor, 1933).

Between 1931-1947 susceptible Turkey types were still important. Rio, with the *R* gene, was released in 1931 but it was not widely grown. Rex, with  $M_1M_2$  genes, was released in Oregon in 1933. It had poor quality and most of the soft white and club acreage was still occupied by susceptible wheats. According to Kendrick and Holton (1961), in spite of the prevalence of susceptible wheats, bunt was not serious during this period.

Between 1948 and 1952 Golden and Elgin became important. They have no major genes for resistance. Rex ( $M_1M_2$ ) was joined by Hymar ( $M_1M_2$ ). The number of wheat varieties declined during this period, but T-1 and L-1 were still the prevalent races, so the  $M_1M_2$  genes in the unpopular cultivars had not yet significantly altered the general race population.

In 1949 Elmar (Elgin x Martin) was released and it quickly became the leading wheat. For the first time the  $M_1M_2$  genes were in a popular, widely grown genotype. Races attacking  $M_1M_2$ , called Martin races, multiplied quickly and a major epidemic followed. Brevor ( $M_1M_2rd$ ) was important in parts of the region in 1952-1962. Even though Brevor was resistant to the prevalent Martin races, Elmar dominated in much of the region because of its superior yields and plant type.

In 1955 a wheat resistant to all known races, Omar, with (*TM*) resistance genes, was released. In 1957, T-18, a race attacking Omar, was found in the field. By this time the new seed treatment, hexachlorobenzene (HCB) was being used and common bunt from this time to the present has been no problem. The soil infestation disappeared quickly.

Up to the time of the use of the HCB seed treatment every resistance gene or combination of them was overcome by the pathogens. The dismal prediction of Mourashkinsky in 1933 that breeding would prove essentially futile is true in a region with a soilborne infestation not controlled by seed treatment. Although the emphasis on resistance to common bunt has declined in the PNW, breeders still test their materials with selected races of common bunt. Testing for common bunt is useful in respect to dwarf bunt, and no truly effective seed treatment is available for dwarf bunt.

In the PNW bunt-resistant wheats were developed primarily by straight crosses, resistant x susceptible, until Elmar (= Elgin x Martin, backcrossed two times to Elgin). In California, an adapted genotype (susceptible) was made resistant using the back cross technique. Baart 38 resulted from Martin x Baart with six backcrosses to Baart. The early attempts to develop resistant wheats in the PNW (Ridit, Albit) were not wholly successful because the wheats were of low agronomic competitive ability. But in the long run the straight crossing method was more productive (Brevor, Burt, Gaines) of superior, resistant wheats. Agronomic characteristics are maintained by backcrosses, but agronomic advances, as well as losses, are made in straight crosses.

The severity of the smut problem in 1956 is illustrated by a survey of the percent of smutty grain delivered to elevators in north central Oregon, eastern Washington and northern Idaho. In 1956 42% of all the grain was Elmar and 54% of all Elmar graded smutty. Brevor made up 18% of the sample and 5.6% graded smutty. Turkey made up 7% of the total and 0.9% graded smutty. Turkey was grown in the drier areas in which wheat is typically seeded in warmer soil, unfavorable to smut, so the conclusion that the *T* gene was nearly immune from attack is not warranted. The  $M_1M_2$  genes of Elmar were essentially worthless, and the  $rdM_1M_2$  combination in Brevor had been seriously overcome. In Asotin County,

Washington, and the Camas Prairie region of Idaho, where Brevor was the leading wheat, smutty wheat in Brevor was 18%, in Elmar, 87%. Races attacking *rd* as well as  $M_1M_2$  were increasing. (Data taken from a survey led by C. S. Holton in cooperation with the Pacific Northwest Grain Dealers' Assoc.)

Table 19. Identification of genes for resistance to bunt, using the old and new designations.

New	Source	Old	
<i>Bt1</i>	Martin	$M_1$	Briggs, 1926
<i>Bt2</i>	Hussar	H	Briggs, 1926
<i>Bt3</i>	Ridit	<i>rd</i>	Churchward, 1932
<i>Bt4</i>	Turkey	T	Briggs, 1932
<i>Bt5</i>	Hohenheimer	Ho	Gaines and Smith, 1933
<i>Bt6</i>	Rio	R	Sanford & ?, 1941
<i>Bt7</i>	Martin	$M_2$	Briggs and Holton, 1950
<i>Bt8</i>	Yayla 305		Waud and Metzger, 1970
<i>Bt9</i>	Sel. M69-2073		From crosses with P.I. 178383 (see Hoffmann and Metzger, 1976)
<i>Bt10</i>	Sel. M69-2094		From crosses with P.I. 178383 (see Hoffmann and Metzger, 1976)
<i>Bt11</i>			Resistance genes <i>Bt8</i> occurs in P.I. 178210, <i>Bt9</i> in C.I. 7090, <i>Bt10</i> in P.I. 116301, and <i>Bt-11</i> in P.I. 166910, Metzger, 1980, personal communication

*Bt12* and *Bt13* known now according to J. Hoffman. Identified by Metzger.

*Bt4* and *Bt6* are closely linked, at least in some wheats (Sanford, 1941)

Genes do not belong to varieties (*rd* from Ridit from Florence from ?), and geneticists now call them bunt genes 1, 2, 3, etc. = *Bt1*, *Bt2*, *Bt3*, etc., numbered chronologically in accordance with their discovery, making  $M_1M_2$  into *Bt1* *Bt7*. More sources of resistance are known, but the identification of resistance genes ceased with the retirement of Metzger. R. Metzger (1980, personal communication) stated that a race of *T. controversa* that attacked all 10 described resistance genes was found by Hoffmann in northern Utah. No commercial wheat contains all these resistance genes, but Hoffmann states that the cultivars Cache and Wasatch with the Ridit (*Bt3*) gene resisted dwarf bunt for years, and no race consistently attacks *Bt5* or *Bt8*.

#### Identification of resistance genes

Identification of resistance genes depends upon how disease notes are taken. Gaines (1923) grouped plants into three categories; healthy, partially smutted and completely smutted. Gaines obtained complicated results indicating quantitative inheritance. Briggs (1926) used only two classes, healthy, and any degree of smutting. Briggs also used a single source of smut spores. He determined that resistance was inherited in a simple Mendelian fashion, with a single completely dominant gene for resistance in Martin ( $M_1$  or *Bt<sub>1</sub>*) and an almost completely dominant gene for resistance in Hussar (*H* or *Bt<sub>2</sub>*). Briggs developed the methods used by all subsequent breeders, namely, use pure (single race) inoculum and only diseased and healthy (two) classes of wheat.

The *H* gene is weak in that few wheats possessing this resistance were grown, yet races attacking *H* are common in both *T. caries* and *T. foetida*. The Martin genes were used early and extensively, so these resistances had to be overcome by the pathogens. The *T* and *R* genes of Turkey types and the *rd* gene were difficult to overcome, even though they were present in extensively grown cultivars for many years in the main dwarf bunt areas of Idaho and Utah. An effective chemical will be required if dwarf bunt is to be permanently controlled.

Hussar (*H*) and Hohenheimer (*Ho*) were little used because agronomically acceptable segregates were rare. Hussar actually contains the main Martin and the Hussar gene ( $M_1H$ ) (*Bt1*, *Bt2*), but they have been separated so that *Bt2* (*H*) can be studied independent of  $M_1$ . Likewise  $M_1$  and  $M_2$  of Martin were separated in crosses, as were *Bt9*, *Bt10* and a third resistance gene in PI 178383. Part of the work in studying resistance is separating the genes when they occur in combinations.

Breeding for resistance involved screening large numbers of wheats to discover sources of resistance and gaining or applying correct inoculation techniques and understanding environmental conditions favorable for bunt development. Identification and maintenance of pure races of the pathogens, and selections from wheat crosses that permit identification of resistance genes was likewise important. Much work was omitted from the discussion, but the thread of progress made, both by the breeder in developing resistant wheats and by the pathogen in combining virulence genes is apparent.

Only a fraction of the races of *T. caries* or *T. foetida* are required in experiments to inform the breeder of the major genes in a wheat. In 1961 there were 28 known races of *T. caries* and *T. foetida*, and Kendrick (1961) devised a scheme in which L-1, T-6, L-7, T-13, T-15, T-16 and T-18 could be used to identify the presence or absence of all of the known major resistance genes. In early stages of varietal development a selected composite of races can be used. A necessary precaution is not to include a race or races which attack all the possible resistance genes in the cross or all would be smutted and no selection within the cross would be possible. Holton and Heald (1936) cautioned that in complex composites (10 or more races) the inoculum of a particular race might be so dilute as to make the wheat appear resistant when it might not be so.

Blair J. Goates cautions me to state that some wheats resist all known collections of *T. controversa*, at least up to 1990, so all resistance genes have not been described.

Spectrum of major resistance genes. The number of races of a pathogen and the number of resistance genes that can be identified in a highly developed host-parasite relationship depends largely upon the nature and number of differential varieties used. The major bunt resistance genes confer resistance to several races, leading to the question: why or how were so many races described? In the beginning more differentials were used. With reduced numbers of differentials several races appear alike. When a full complement of differentials is used, however, the described races differ, and there is reason to believe that a gene-for-gene relationship exists (Holton, Hoffmann, Durán, 1968).

The spectrum of race coverage of the major genes is impressive, and it greatly simplified breeding for resistance. At the time when Omar was developed, 16 T-races and 10-L races were known. Omar resisted all 26 races. Omar contains at least the *T* and  $M_1$  gene (Table 19, 20). *T* confers resistance to all but T-16 and L-8, and  $M_1$  resists T-16 and L-8, so these two major genes conferred resistance to all known races (up to 1956).

Table 20. Major genes for resistance are effective against several races.

gene	T-races																L-races									
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	1	2	3	4	5	6	7	8	9	10
$M_1$	R	-	R	-	-	-	-	-	R	R	R	-	-	-	-	R	R	R	R	-	-	-	-	R	-	-
<i>T</i>	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	-	R	R	R	R	R	R	R	-	R	R
<i>H</i>	R	R	-	R	-	R	-	-	R	R	-	R	R	-	-	-	R	R	-	R	-	-	-	-	-	-
<i>Ho</i>	R	R	R	R	R	R	R	R	-	-	R	-	R	R	-	R	R	R	R	R	R	R	R	R	R	R
<i>rd</i>	R	R	R	R	R	R	R	R	R	-	R	-	R	R	R	R	R	R	R	R	R	R	R	R	-	I
$M_2$	-	-	-	-	-	-	-	-	-	R	R	-	R	R	-	-	-	-	-	-	-	-	-	-	-	R

Races T-1 and L-1 were predominant during the early periods. The only major gene they attack is  $M_2$ , which is present in Federation. The  $M_2$  gene was particularly valuable in combination with  $M_1$  and  $rd$ . The only races not covered by  $rd$  were T-11, T-13 and L-9.  $M_1$  resisted T-11, leaving T-13 and L-9, both of which were resisted by  $M_2$ . The combination of  $M_1M_2rd$  gave Brevor and Burt resistance to all but one known race at the time of their release. Thus by the end of the 'Golden Age' of breeding for bunt resistance in the PNW breeders knew what combinations of genes would give the greatest protection against known races. Burt, Brevor and Omar were developed by USDA breeder, Orville Vogel.

The use of T and L races as an aid in developing resistance to D-races is important because of the ease of inoculation with common bunt and because of the greater dependence of dwarf bunt upon environmental conditions. In a 16-year period, fewer than half of the dwarf bunt nurseries had sufficient smut to make them useful. Holton, Bamberg and Woodward (1949), summarized the results of field experiments for five years at Bozeman, Montana and at Logan, Utah, and for one year at Troy, Idaho (Table 21). This was the first real evidence that tests for resistance to common bunt, which are easy to make, could be useful in testing for resistance to dwarf bunt. Their 1949 results led them to refer to the dwarf bunt race.

Table 21. Correlation of resistance in wheats to race T-16 of *Tilletia caries* and to dwarf bunt in field nurseries, Holton et al 1949.

	Resistance	Smut, %		Reaction	
		Dwarf bunt	T-16	DB	T-16
Hybrid 128	O	67	63	S	S
Rio	R	50	57	S	S
Oro	T	37	30	S	S
Hohenheimer	Ho	8	19	S	S
Ridit	rd	13	6	S	S
Albit	M	4	1	R	R
Hussar	$HM_1$	3	1	R	R
Martin	$MM_2$	0	1	R	R

Anything with over 5% dwarf bunt was considered susceptible. Hard red winter wheats prevailed at Bozeman and Logan, Utah, and the  $T$  gene for resistance had been overcome. Note that the  $R$  gene is less effective than the  $T$  gene.  $T$  and  $R$  genes conferred little protection.

Minor genes for resistance. 'Minor' genes, are those which confer a low level of resistance to a given race or races. Holton (1959) illustrated the relative effectiveness of major and minor genes with the same spectrum of action (Table 22).

Table 22.

Wheat Gene	Groups of similar races													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Oro T	R	R	R	R	R	R	R	S	R	R	R	R	S	R
Rio R	R-	R-	R-	R-	R-	R-	R-	S	R-	R-	R-	R-	S	R-
Sel. 1001S(XY)	r	r	r	r	r	r	r	S	r	r	r	r	S	r

The *T* gene conferred the highest resistance, the *R* gene slightly less, and *X* and *Y* genes conferred only a low level of resistance (the capital and lower case letters do not denote dominance or recessive in this table, but degree of protection). *T* and *R* are major genes, *X* and *Y* are minor genes. Gaines detected what he called 'dilute' resistance in 1923 (in Holton, 1959) in crosses of two susceptible wheats.

Virulence genes in *T. caries*, *T. foetida*, and *T. controversa*. The virulence genes in the three bunts were summarized by Hoffmann and Metzger (1976). Their table included 28 T-races, 11 L-races and 17 D-races, or 56 races. The 56 races contain 39 different combinations of major virulence genes. Virulence against resistance gene *M*<sub>2</sub> occurred 48 times, *H*, 37 times, *M*<sub>1</sub> 34 times, *T* and *R* 22 times each, *Ho* 17 times, *rd* 15 times, *Bt-10* eight times, *Bt-9* five times, *Bt-8* no times. The *H* resistance gene is weak because it is essentially absent in commercial wheats, yet virulence against this gene occurred 37 times within the 56 races. The *Ho* resistance gene likewise had little exposure. The abundance of virulence against *M*<sub>2</sub>, *M*<sub>1</sub>, and *T* is expected because these resistance genes were widely used with a lesser degree of exposure to resistance gene *rd*. The compilation of Hoffmann and Metzger (1976) included seven T-races bred in the laboratory. Removing them leaves 49 races from nature. In them *M*<sub>2</sub> virulence occurred 42 times, *H* 31, *M*<sub>1</sub> 29, *T* and *R* 17 times each *rd* 14 times, *Ho* 17 times, *Bt10* five, *Bt9* four, and *Bt8* no times.

Holton, Bamberg and Woodward (1946) noted that the reaction of wheats to dwarf bunt in the field correlated with their reaction to races T-16 and L-8 of common bunt, evidence that at that time there may have been only one widespread dwarf bunt race in the PNW.

D-races in general attack more virulence genes than L- or T-races. Resistance-susceptible reactions are likewise less sharp in D than in L and T races. (Breeders say the resistance genes for dwarf smut 'leak'.)

Inheritance of virulence. In a cross of T-9 (*Ho*, *M*<sub>2</sub>) x L-8 (*H*, *T*, *R*, *M*<sub>2</sub>, *Bt9*) an F<sub>2</sub> line with reticulate spores was intermediate in virulence against both *T* and *Ho* resistance (Holton, 1938). Virulence to *Ho* came from T-9, to *T* from L-8. In a more extensive study (Holton, 1942), some hybrids were more, some less, and some similar to the parents in virulence. Spore wall markings and virulence were inherited independently. Some segregates were virulent on wheats resistant to all field races at that time. When the cross T-8 (*M*<sub>1</sub>, *M*<sub>2</sub>, *H*) x T-9 (*M*<sub>2</sub>*Ho*) was increased on Hybrid 128 (fully susceptible), and then F<sub>2</sub> segregates were propagated for three generations on Hybrid 128 or on wheats with *M*<sub>1</sub>, *Ho* and *HHo* resistance, the wheat upon which the lines were grown favored certain virulences (Table 23).

Table 23. Virulence of smut lines from the crosses T-8 x T-9 after three generations on a given host (Holton, 1942).

Line	Three generations on	Resistance genes in tester			
		O <sup>a</sup>	<i>M</i> <sub>1</sub>	<i>Ho</i>	<i>HHo</i>
50a <sup>b</sup>	Hybrid 128	83	46	3	1
53a	Hybrid 128	64	2	6	1
50b	Hohenheimer	84	10	57	2
55b	Hohenheimer	86	73	61	4
50c	Sel. 10068-1	78	21	55	14
55c	Sel. 10068-1	87	8417		51

<sup>a</sup> O = Hybrid 128, *M* = Albit, *Ho* = Hohenheimer, *HHo* = Sel. 10068-1, Hussar x Hohenheimer.

<sup>b</sup> 50 = the same line, 50 a, b, c = fractions taken from the F<sub>2</sub> 50 and subsequently increased 3 generations on a different wheat before testing on the differential varieties.

Line 55 grown on Hybrid 128 (= 55a) was avirulent on *H* and *Ho*, fraction b grown on Hohenheimer was virulent on *Ho*, and fraction c, grown on Sel. 10068-1 was virulent on *Ho* and *H*.

The absence of resistance genes in a host (Hybrid 128) tended to select races of low virulence. None of five lines after three generations on Hybrid 128 was virulent on *H* or *Ho*. Holton concluded that this experiment paralleled experience in the field, i.e. that when resistant wheats were grown, virulent races increased. He cited earlier studies in which workers inoculated a relatively resistant wheat with spores from that wheat and the percent smutted plants increased with repeated passages through the host. He emphasized the results of Mourashkinsky in 1933 in Siberia who was so impressed with the increased virulence after five passages through a formerly resistant host that he concluded that breeding for resistance could not succeed. Mourashkinsky did not know whether his resistant wheats selected virulent strains from a mixture or if they resulted from hybridization. Holton's experiment demonstrated the effect of hybridization in the smut and resistance in the host upon increased virulence.

Hoffmann cautioned that resistant cultivars do tend to increase races with the required virulence, but to remember that races existed that attacked *H*, *Ho*, *Bt9* and *Bt10* even though the smut had not been exposed to wheats containing these resistances.

Maintenance of genetic stability. Up to now we have considered only virulence genes, or genes effective against specific resistance genes, but the entire genotype is important. Many compatible combinations of primary sporidial cultures do not result in pathogenic dikaryons capable of perpetuation in the host. In interspecific crosses made by Holton (1942) 17% failed to sporulate in the host, and 41% of the compatible combinations of inter-race crosses failed to survive, even in a universal susceptible. Hoffmann and Metzger (1976) commented that aggressive hybrids that survive and sporulate in the host are not common, that possession of virulence genes is not the only requirement.

In the experiments cited above, new genotypes were without competition with fit, established genotypes. Competition can be severe, even between fit races (Table 9). When Elmar was the leading wheat, 90% of the total collections of the PNW were obtained from this one wheat, and 58% of the total collection was race T-6. When T-5, T-6, T-7 and T-8 were mixed equally, all of which are virulent on Elmar, after a single passage through Elmar, 29 of the 30 heads tested contained T-6 (Kendrick and Holton, 1961). These experiments illustrate competition among established genotypes on a susceptible wheat in which host resistance is not exerting known selection pressure.

Stabilizing selection. Stabilizing selection exists when the prevalent races of a specialized pathogen contain the minimum required virulence genes. Stabilizing selection tends to maintain the bulk population in as avirulent a condition as possible (Vanderplank). In the first systematic survey in the PNW, Flor (1933) found T-1 and L-1 prevalent. Hybrid 128, very susceptible, was the leading wheat. Virulent races were present but T-1 and L-1 made up 84% of the population. Flor noted that the least virulent races seemed favored. He next studied flax rust (*Melampsora lini*) in North Dakota. In a classic paper (Flor, 1953) he reported the number of virulence genes present in the flax rust population, 1931-1951. When most of the flax contained few resistance genes, most of the rust contained few virulence genes. In the initial period (1931-1940) 94% of the collections had one or two virulence genes. In the second period, 1942-1947, 86%. In the third period, 1948-1951, 39%. Flor observed that some selection mechanism favored races with the fewest virulence genes in flax rust. *Melampsora lini* is autoecious and it goes through the sexual stage every year in North Dakota. The least virulent races of *T. caries* and *T. foetida* were dominant in the U.S., Canada and Mexico on old wheats that lacked specific resistance genes (Rodenhiser and Holton, 1945).

The only evidence we have for stabilizing selection in dwarf bunt is the observation of Holton, Bamberg, and Woodward (1949) of the dwarf bunt race prevalent in the PNW. Methods of inoculating with individual collections were unknown prior to 1957-1959, so there could be no real knowledge of early



racess (Hoffmann). There are no early studies on the prevalence of D-races, but Hoffmann and Metzger (1976) found D-13 the most common. D-13 is highly virulent, attacking  $M_1$ ,  $M_2$ ,  $T$ ,  $R$ ,  $H$ , and  $Bt10$ . The race(s) observed by Holton et al 1949 was virulent on  $H$ ,  $T$ ,  $R$ ,  $Ho$ ,  $M_2$ . By the time any real knowledge of dwarf bunt was attained the pathogen had been exposed to  $M_1$ ,  $M_2$ ,  $rd$ ,  $T$ , and  $R$ . It is really not known whether stabilizing selection functions to any degree in this smut. Hoffmann commented that stabilizing selection seems to work against  $M_1$ ,  $T_1$ , and  $rd$ , but not the rest.

The different life cycles of common and dwarf bunts may affect host-race relationships. Sporidia of *T. controversa* are mostly produced at the soil surface and infection occurs in a relatively dormant host, mostly during winter. The relatively dormant host may reduce the sharpness of host-parasite interactions and reduce stabilizing selection.

Inheritance of pathogenicity (not virulence as used in the smuts). The first requirement for pathogenicity is the fusion of two compatible sporidia (+ and -). Kendrick (1968) mated compatible monokaryons of three L-races and 15 T-races in all possible combinations (544 matings). Compatibility was determined by fusions (plasmogamy). Only 37% of the compatible matings resulted in symptoms in Red Bobs, a fully susceptible wheat. Many compatible matings result in offspring unable to reproduce. Kendrick concluded that pathogenicity is inherited independent of compatibility factors and that pathogenicity is complex in its inheritance.

Highly virulent races have no more pathogenic offspring than weakly virulent races (Table 24). T-4 averaged 50% pathogenic offspring, T-6, 29%, yet they possess the same virulence genes. General "pathogenicity" is the first requirement; acquisition of virulence genes the second. The extensive matings by Kendrick (1968) firmly established the bipolar nature of both species and that + and - factors are the same in *T. caries* and *T. foetida*.

Table 24. Pathogenic matings (percent) resulting from pairing compatible monokaryons of different races of *Tilletia caries* (= T-races) and *T. foetida* (= L-races) (Kendrick, 1968).

Race	Virulence genes	Mating type		Pathogenic offspring, Average
		+	-	
L-1	7 <sup>a</sup>	42	59	50
T-4	1,7	47	53	50
T-8	1,2,7	41	47	44
T-15	1,2,5,7	26	59	42
L-8	2,4,6,7,9	44	53	48
	Average			37

<sup>a</sup> Virulence genes according to Hoffmann and Metzger, 1976. The average at bottom is the average of all data, not just those present in this table.

In a review of genetics of the smut fungi Holton (1954) stated that in general, culture type, spore morphology, symptomatology and virulence were inherited independently. In some crosses of *T. caries* x *T. foetida*, smooth spore is dominant, in some, reticulate spore is dominant, and in some dominance is partial.

Infection of grasses. Grasses have played little or no role in the epidemiology of common bunt in wheat in the PNW. The grasses could, however, protect a species from local extinction. Individual races of highly specialized pathogens, such as powdery mildew, the cereal rusts, and the bunts can be controlled by single genes in wheat and yet they can attack unrelated grasses in other genera. Specific resistance genes within wheat hinder a given race more than the entire genomes of several genera. Meiners (1956) inoculated many grasses with nine races of *T. caries* individually but he did not report the details of the study (which grasses succumbed to which races). He found hosts in three tribes, Aveneae, Festuceae, and Hordeae. Susceptible hosts were found in *Arrhenatherum*, *Koeleria*, *Bromus*, *Dactylis*, *Festuca*, *Agropyron*, *Elymus*, *Hordeum*, and *Lolium*, illustrating the ability of the bunts to tolerate a wide range of cytoplasm.

The background cytoplasm of *Agropyron cristatum* and *A. pauciflorum* is congenial to *T. caries* and *T. foetida*, but specific genes for resistance occur within these grasses just as in wheat. Fischer (1939) inoculated accessions of the above grasses with several races of *T. caries* and *T. foetida* and reported the results in detail (Table 25). I arranged the results in a way to reveal the relationship between known virulence genes (Hoffmann & Metzger, 1976) against resistance genes in wheat and the reaction of five accessions of each grass. The grasses reacted as specifically as wheat, yet there is no correlation between virulence genes expressed in the wheat system and in the *Agropyron* systems. If this be true, the bunts have a great reserve of potential virulences in that they can accommodate not only the resistance genes in wheat but in addition, the different resistance genes in different grasses.

Table 25. Smut (%) in five accessions each of *Agropyron cristatum* and *A. pauciflorum* and major virulence genes in each race of *Tilletia caries* (T) and *T. foetida* (L).

Race & virulence genes in the wheat system	<i>A. cristatum</i> accession						<i>A. pauciflorum</i> accession			
	28	29	32	36	37	62	68	69	75	288
T-1, $M_2$ (Bt-7)	0	0	50	40	0	13	17	0	13	0
L-1, $M_2$ (Bt-7)	0	50	0	0	25	13	16	0	13	0
L-2, $M_2$ (Bt-7)	50	0	0	0	33	28	30	0	28	40
T-2 $M_1M_2$ (Bt-1,7)	63	0	0	22	25	19	20	0	6	0
T-4 $M_1M_2$ (Bt-1,7)	38	25	25	33	40	29	25	0	5	14
T-6 $M_1M_2$ (Bt-1,7)	100	0	0	17	0	42	14	0	0	0
L-4 $M_1M_2$ (Bt-1,7)	33	0	0	14	57	23	20	6	33	0
T-5 $M_1M_2H$ (Bt-1,2,7)	28	0	0	0	0	32	33	6	6	29
T-7 $M_1M_2H$ (Bt-1,2,7)	0	25	0	0	0	27	17	0	14	0
T-8 $M_1M_2H$ (Bt-1,2,7)	33	0	100	28	33	0	17	0	0	0
L-5 $M_1M_2H$ (Bt-1,2,7)	50	0	33	10	0	16	25	20	0	0
L-6 $M_1M_2H$ (Bt-1,2,7)	14	0	0	33	0	24	12	0	0	0
L-7 $M_1M_2H$ (Bt-1,2,7)	50	0	0	0	33	19	13	17	0	0

Purity of races. Races of *Puccinia graminis* are maintained by asexual urediospores, and they are genetically stable. Races of the smuts are maintained as teliospores, and each generation involves karyogamy and meiosis, presenting a constant opportunity for recombination. Flor (1933) recognized this and considered it a probable problem in maintaining the stability of smut races for laboratory studies. Holton and Rodenhiser (1942) stated that of the 19 races of *T. caries* and *T. foetida* known at that time,

only T-11 seemed to have changed. As originally described, T-11 was virulent only on *H* and *rd*, but it probably gave rise to T-13, which is virulent on  $M_1$ , *H* and *rd*.

The purity, or relative homozygosity for virulence of some races, was determined by selfing several of them and testing the segregates (Holton, 1953). Upon selfing, T-10 and T-15 gave parental types; T-4, T-6, T-8, and T-12 gave parental types or lines of reduced virulence. He commented that T-4 was now less virulent on Albit (=  $M_1$ ) than originally. Race T-9 was heterozygous (Table 26).

Table 26. Response of race T-9 and of two of its inbred lines to resistance genes (Holton, 1953).

Fungus	Resistance genes			
	O	T	Ho	HHo
Race T-9	84	4	65	2
Inbred 5	65	2	62	3
Inbred 3	75	65	30	46

Inbred 5 was like the original T-9 and inbred 3 was like T-16. New races can originate by mating the sporidia from a single teliospore of a described, relatively stable race. Race T-16 from nature was inbred by Hoffmann and Metzger (1976) and a new race, T-24, was obtained. T-16 is virulent on *H*, *T*, *Ho*, *R*,  $M_2$ . T-24 lost its virulence on *Ho* and it gained virulence on *rd*, becoming the first race of either *T. caries* or *T. foetida* virulent on both *rd* and *T*. They produced T-25 and T-26 by selfing T-5. Race T-5 is an 'old' race. If it is possible to obtain new races by selfing, what maintains their stability in nature? Possibly most new genotypes cannot compete with "mother", i.e. the adapted parent genotype.

In smutty wheat thousands of spores coat the outside of the kernel in close proximity to each other, and hybridization by primary sporidia on different promycelia is possible. In nature threshing mixes smut and where soilborne inoculum is important, spores from many fields could be mixed in the spore showers. The opportunity for crossing exists, but plasmogamy normally occurs while primary sporidia are still attached to the promycelium, greatly limiting outcrossing. It seems obvious that some strong selectivity, including competition among genotypes, is functioning to provide some stability to the population.

#### Remarks

It is sad that modern fungicides have controlled *T. caries* and *T. foetida*. Pathologists and breeders no longer have the challenge of trying to keep up with these pathogens. It seems obvious, however, that the fungi would win without effective seed treatments.

Why do *T. caries* and *T. foetida* differ so greatly in prevalence in different geographic areas?

In chronological order, the following contributors to our knowledge of bunt who worked at Pullman became presidents of the American Phytopathological Society: Harry Humphrey, Frederick Heald, Harold Flor, Stewart Holton, George Fischer, and Hank Purdy. In addition, Ed Kendrick was treasurer of the national society and Jim Hoffmann was president of the Pacific Division. In varying degrees, their studies of the smuts enriched their careers and the reputation of pathology in Washington.

#### Dwarf Bunt of Wheat, Synopsis

Dwarf bunt (*Tilletia controversa*) presents a very different problem than common bunt, even though *T. controversa* is so closely related to *T. caries* that Holton and Matt Silbernagel, a student of Holton, crossed the two species and produced viable, pathogenic offspring. Dwarf bunt was never epidemic in

Washington because it requires rather specific environmental conditions for infection. Conley Lowther (PhD, 1948) worked when dwarf bunt was considered a peculiar 'race' of *T. caries*. He studied requirements of spore germination, using facilities provided by N. S. Golding and D. Miller of the Dairy Husbandry Department, suggesting to me that equipment for controlled temperature studies was not available in the Plant Pathology Department at that time. Lowther found that spores of *T. controversa* germinated best at 5°C, and that germination was slow, requiring 3-5 weeks, depending upon the collection.

The spores of *T. controversa*, unlike those of common bunt, possess endogenous dormancy and soil infestation persists for several years. High infection is associated with snow cover. Infection occurs mainly between December and February (Purdy, et al). Inoculum on seed is essentially ineffective, except to disseminate the pathogen. Spores are sprayed on the soil surface in inoculation trials rather than placing them on the seed. Light stimulates spore germination. This may be a major factor in preserving spores when soil is tilled because many spores would be within the soil in the dark. Dwarf bunt infection occurs when soil temperatures are low but not steadily below freezing, the reason why snow or straw mulch increases disease incidence. Very young tillers are invaded at the soil surface. Because of this, control by seed treatment is seldom satisfactory. The systemic fungicide thiabendazole (TBZ) is useful as a seed treatment in late seeding but not in early seedings.

Races of *T. controversa* have been studied extensively by James Hoffmann, a former student of this department. Some of his studies were done here at Pullman and some at Logan, Utah, often in cooperation with Robert Metzger, USDA geneticist at Corvallis, Oregon. Local breeders tested materials for resistance, mainly in cooperation with Hoffmann and Don Sunderman, USDA, Aberdeen, Idaho, both of whom have retired. Their combined efforts reduced losses in southern Idaho and northern Utah where dwarf bunt was a production problem.

In Washington dwarf bunt, called TCK (for *T. controversa* Kühn) by the press, is mainly a marketing problem. Some countries, particularly the Peoples' Republic of China, do not permit importation of wheat from regions in which dwarf bunt occurs. There is no hope of eradicating dwarf bunt so efforts have been made to kill spores in wheat shipments. This approach has little chance of success. Farmers of the region wish to sell their wheat to China, and political pressure has resulted in obtaining special TCK research funds, some of which went to Ruben Durán and some to Lee Hadwiger.

*Dwarf Bunt of Wheat*  
*Tilletia controversa* Kühn 1874  
*T. brevifaciens* Fischer 1952

Mr. Enos Holmes, a farmer in southwestern Michigan, noted 'high' and 'low' stinking smut in his wheat (Potter and Coons, 1918). *T. foetida* shortened the plants only 5-10 cm. 'Short' smut (believed then to be *T. caries*) shortened the stems a full 30 cms, so much that many heads escaped harvest and were left intact in the field in the stubble. Potter and Coons believed the difference in height was the simplest way to distinguish "*T. caries*" from *T. foetida*, the dominant bunt fungus in the region. In low smut the heads were enlarged because more of the florets develop into bunt balls than develop into normal kernels in healthy wheat. The short (dwarf) smut was affected by soil conditions; it was more spotty in the fields than the tall smut (*T. foetida*).

Two smut collections, labeled *T. caries*, were studied by Faris (1934). The collection from Washington (*T. caries*) applied to seed resulted in much bunt. The collection labelled *T. caries* from New York (*T. controversa*) was almost ineffective as a seed-borne pathogen. Some of the observations of Potter and Coons (1918) and Faris (1934) were based upon *T. controversa*, the dwarf bunt fungus. Spores of *T. caries* and *T. controversa* are both reticulate and their spore morphology overlaps to some extent; those of *T. foetida* are smooth.

Confusion ended with the publication of a remarkable abstract, never followed by a full paper, by P. A. Young (1935) in Montana. Young stated that the reticulations on the spores of *T. controversa* were higher and the areolae larger than in *T. caries* and that the dwarf bunt spores failed to germinate on water agar at 12°C or at 25°C within the time that he observed them. Dwarf bunt shortened the stems and increased the number of tillers. The awns of bearded wheats diverged at a wider angle than in common bunt, and the bunt balls were harder. Seed inoculation with *T. caries* was highly successful, but not with the dwarf bunt. Young considered the dwarf bunt fungus a distinct variety of *T. caries*. Following the appearance of this abstract, dwarf bunt of wheat was reported in Utah, Idaho, Washington, New York, British Columbia, Ontario, Germany, and Austria (see Connors and Skolko, 1953 and Purdy et al, 1963, for details). Dwarf bunt was hereafter not treated as a peculiar type of *T. caries*.

Dwarf bunt is common in eastern Turkey, Iran, and northern Pakistan (Trione, 1982). Hoffmann (1982) believes *T. controversa* originated in Asia in regions of much snow where the soil does not freeze deeply beneath the snow. Its host range among grasses is wide and similar to that of *T. caries* (Purdy et al, 1963). Even though it is rarely seed-borne, spores and bunt balls were spread with wheat seed, especially before seed treatments and quarantines. Upon introduction it would survive in regions favorable for its reproduction, mostly on fall-sown wheat in regions with much snow.

*T. controversa* was described from *Agropyron repens* near Halle, Saxony, by Julius Kühn in 1874 (Connors, 1954). It is common on *Agropyron* spp. in snowy areas of central Europe. It probably escaped detection as a pathogen of wheat because of the similarity of its spores, smell, and bunt balls to *T. caries*. Kühn distinguished it from *T. caries* on the basis of higher reticulations, larger areolae, and difficulty in germinating the spores under conditions favorable for *T. caries*. Fischer (1952), unaware of Kühn's work, described the dwarf smut fungus as *T. brevifaciens*. *T. brevifaciens* was placed in synonymy with *T. controversa* by Connors (1954), a decision with which Fischer agreed.

Kühn spelled the specific epithet *contraversa*. Durán and Fischer (1961) established *controversa*, replacing an a with an o, for reasons of grammar. D.B.O. Saville in 1962 agreed with the corrected spelling, so the name is *T. controversa* (Purdy et al, 1963). Fischer (1952) described the hyaline sheath which characteristically protrudes beyond the ridges of the reticulations. According to Hoffmann (1982) the sheath of *T. controversa* swells when hydrated to such an extent that, when dwarf bunt balls are placed in water the spore balls rupture, releasing spores into the water. Uninjured bunt balls of *T. caries* and *T. foetida* do not rupture in water, except after very long exposure.

*T. caries*, *T. foetida*, and *T. controversa* can interbreed (Purdy et al, 1963), they have similar virulence genes, and identification of races of all three is based upon a common set of differentials (Hoffmann and Metzger, 1976). Spores of race T-18 (*T. caries*) survive in soil 2-3 years, longer than any other race of *T. caries* or *T. foetida*, its spores require 14 days to reach maximum germination at 10°C, and Kendrick et al (1964) believe it a hybrid of *T. caries* x *T. controversa*, with characteristics of both species. When T-12 (*T. caries*) and L-9 (*T. foetida*) were crossed with *T. controversa* experimentally (Holton, 1941), the offspring varied in spore morphology and degree of dwarfing of the host. Holton studied offspring more like common bunt than dwarf bunt because he inoculated the seed and studied offspring that infected the host from that position.

*T. caries* and *T. foetida* are so close they faithfully recognize + and - mating types reciprocally. *T. controversa* is more distant in that in most interspecies crosses with *T. caries* and *T. foetida* it does not recognize + and - mating types of the common bunts. Silbernagel (1964) noted that in some matings a *T. controversa* monokaryon was compatible with either a + or a - monokaryon of *T. caries*. When monokaryons of *Typhula idahoensis* and *T. ishikariensis* were mated in interspecies crosses, the compatibility factors were inoperative because matings occurred between them promiscuously. This was interpreted as evidence of considerable genetic separation (Bruehl, et al, 1983). By the same logic, *T. caries* and *T. foetida* are very close, *T. controversa* more distant. Differences in the life cycle of dwarf bunt and common bunt warrant separate treatments of dwarf and common bunt.

Hoffman and Kendrick (1969) reported multiple alleles in *T. controversa*, which would increase outcrossing in this species.

The effects of dwarf bunt upon the host are evidence of strong hormonal differences between common and dwarf bunt. Dwarf bunt increases tillering by about 50% and shortens the straw by about 25 - 66 % (Purdy et al, 1963). The degree of dwarfing varies with the host genotype and the race of smut. In some combinations, common bunt shortens stems moderately, some dwarf bunt host combinations are relatively tall, but they seldom overlap. Dwarf-bunted heads are enlarged and squarose rather than cylindrical. In dwarf bunt the bunt balls are smaller and more spherical, less kernel-shaped than in common bunt. In dwarf bunt usually all florets of the spikelet are bunted (5-7), while in common bunt 3-5 are bunted. The spherical bunt balls of dwarf bunt force the lemma and palea out at large angles exposing the bunt balls. This, along with the greater number of smut balls per spikelet, results in heads wider than normal. Healthy wheat seldom forms kernels in all florets, so with dwarf bunt it exceeds its "normal" activities. A common morphogenetic host response to both bunts is that heads of club wheats in some race-cultivar combinations become elongated, like common wheat heads, an example of host retrogression to ancestral form.

The host range of *T. controversa*, briefly reviewed by Purdy et al, 1963, is extensive. Grasses in four tribes, 14 genera and 55 species were known hosts in 1963, with the largest numbers in *Aegilops*, *Agropyron*, *Bromus*, *Elymus*, *Festuca*, *Hordeum* and *Lolium*. Grasses probably function mainly to perpetuate the fungus but they are not important in epidemiology. Hardison, et al (1959) seeded many grasses in May, 1957 near Elgin, Oregon; Worley, Idaho; and Pullman, Washington. In October the plots were heavily inoculated with *T. controversa* spores from wheat. The fungus from wheat produced bunt in many species and genera, resulting in a rather similar host range to that of *T. caries*. As in common bunt, the fungus is a poor taxonomist, being able to produce disease in several very different grasses but little in a resistant wheat.

Race determination in *T. controversa* is much more difficult than in *T. caries* or *T. foetida*, primarily because of the special conditions required for disease development and because of different inoculation techniques. Another factor is that 'immunity' may not exist: even the most resistant wheat may develop a small amount of dwarf smut under severe conditions. Despite these difficulties, the first real survey (Hoffmann et al, 1962) revealed eight races in the Pacific Northwest (USA) on seven differential varieties.

The two bunts, though not identical, have enough in common that resistance tests for common bunt have value in at least initial screening for resistance to dwarf bunt. This relationship was emphasized by a test of several wheats in which wheats were tested against T-16, a dwarf bunt collection, and a hybrid of *T. caries* x *T. controversa*. In the combinations used, the results were essentially equal (Holton et al, 1949 see Table 21, common bunt).

Wheats with resistance to snow molds in Washington were tested for resistance to dwarf bunt in the field by J.A. Hoffmann in 1964. A surprising number were promising (C.I. 9342, P.I. 56238, P.I. 81793, P.I. 119342, P.I. 166262, P.I. 166315, P.I. 166797, 166820, 166843, 166886, 166944, 167457, 167822, 173437, 173438, 173440, 173451, 177958, 178030, 178201, 178383, 180591, 182909, 182910, 190970, 215626, 228295, 243767, and 251397. Most came from eastern Turkey, the region from which most of the wheats with resistance to snow mold came. There is no genetic association of snow mold resistance and dwarf bunt resistance, however. P.I. 172582 and 181268 are good sources of snow mold resistance but highly susceptible to dwarf bunt. Also, segregates from crosses often differ markedly in resistance to the two problems.

#### Compatibility relationships among *T. caries*, *T. foetida* and *T. controversa*.

Fusions between monosporidial lines of three races of *T. foetida* and 15 of *T. caries* revealed a simple bipolar compatibility within and between both species of common bunt (Kendrick, 1968). Incompatibility

within *T. controversa*, however, is not simple. Fusion (plasmogamy) tests between monokaryons from 13 teliospores from 10 races of *T. controversa* gave results not explainable on a simple + and - hypothesis (Hoffmann and Kendrick, 1969). They erected 5+ and 4- alleles in *T. controversa*. The + and - monokaryons of T-16 (*T. caries*) fused with *T. controversa* monosporidial lines in such a way as to correspond to A4(+) and A1(-) of *T. controversa*. They only tested one set of monokaryons from a common bunt with *T. controversa*, but the study of Kendrick (1968) established that all lines of *T. caries* and *T. foetida* had the same incompatibility alleles. This indicates that, in relation to *T. controversa*, all + lines of both common bunts correspond to +A4 and all minus lines correspond to -A1 of *T. controversa*.

The discovery of multiple alleles in *T. controversa* explained results of earlier studies in which + and - monokaryons of *T. caries* fused with the same monokaryon of *T. controversa*. When matings contain no common alleles, differing alleles overpower the + and- in these interspecies matings (Table 1).

Table 1. Effect of alleles in matings of monokaryons of *Tilletia caries* and *T. controversa* (Hoffmann and Kendrick, 1969).

T-16	<i>T. caries</i>		<i>T. controversa</i>							
	A4+	A1-	A3+	D-5	A4+	D-7	A1-	A5+	D-10	A4-
A4+	-	+	+	+	-	+	+	+	+	+
A1-	+	-	+	-	+	-	+	+	+	+
<u>D-5</u>										
A3+	+	+	-	+	-	+	-	-	+	+
A1-	+	-	+	-	+	-	+	+	-	-

Note that within species, + monokaryons do not fuse with + monokaryons even though they have different alleles, but that between *T. caries* and *T. controversa* monokaryons fuse if the alleles differ. Different alleles over-ride + and - in these inter-species crosses. This does not occur in crosses within a species.

The life cycle of *T. controversa* leads me to believe it adapted to survive before man threshed grain, that *T. caries* originated with wheat culture (threshing) when a dwarf bunt spore germinated promptly when contaminated seed was planted. The existence of multiple sex alleles in *T. controversa*, response of its spores to light, and their greater longevity in soil indicate an older biologic system, one that gave rise to the biologically more simple common bunt system.

Spore germination. Common bunt spores begin to germinate within 2-3 days at optimum conditions, enabling infection of very young seedlings, mostly prior to seedling emergence. Dwarf bunt spores germinate very slowly, with infection occurring almost exclusively after seedling emergence. The times required for spores of *T. controversa* to germinate, as given by various authorities, vary considerably. These variations are not errors. Spores of the races differ, and collections of the same race differ. Lowther (1948) reported maximum germination in 49-70 days at near 5°C. No spores germinated at 10°C. Meiners and Waldher (1959) reported germination in 26-60 days under ideal conditions. Hoffmann (1982) gives -2°C, 3-8°C and 15°C as minimum, optimum and maximum temperatures for germination.

Unlike common bunt, light greatly facilitates germination of *T. controversa* teliospores. Light is effective after the spores have been hydrated and continuous light of low intensity has the greatest effect (Baylis, 1958, gives early literature, as do Purdy et al, 1963). The optimum light intensity is about 2000 lux, but as little as 25 lux is helpful (Schauz, 1977). Ultraviolet (340nm) was most stimulatory with blue light (445nm) second. Schauz reported 74, 55, 15, 7, 5 and 4% germination respectively under ultraviolet, blue, green, yellow, red, far red and darkness. When light and dark were alternated, short days were most stimulatory. Baylis (1958) deduced from the response of spores to light that infection probably occurred

near the soil surface. In contrast, common bunt spores germinate well either on or in the soil. Dwarf bunt spores germinate mainly on the soil surface or very close to it.

The rate of spore germination is under strong internal control. As in common bunt, no one has been able to accelerate germination of dwarf bunt spores, other than normal responses to temperatures and light. Dewey and Tyler (1958), noting that no one had germinated *T. controversa* spores in less than 3-4 weeks, tried growth regulators, inorganic acids, bases, heat treatments and scarification without success. Teliospores of *T. controversa* contain lipid germination inhibitors soluble in absolute methanol that are almost insoluble in water (Trione, 1977). Possibly, they must be metabolized and this might function to delay germination in nature. Both common bunt and dwarf bunt spores contain water soluble inhibitors, but they probably don't function in *T. controversa* because the spores do not germinate for long periods even when hydrated on an agar surface. The water soluble inhibitors in *T. caries* function somewhat on a 3% agar surface. Spores at 3, 9, 30, 100, 302, and 1000 spores per mm<sup>2</sup> germinated 79, 77, 62, 34, 12 and 0% respectively (Trione, 1977). This is strange because common bunts infect wheat at high spore concentrations on the seed (as high as spores can be held by the seed).

Under favorable conditions, a short, stout promycelium with 14-30 terminal primary sporidia is produced (Hoffmann, 1982). H-cells form as in common bunt (Holton, 1943), and cytological events are similar except for more mitotic nuclear divisions. The promycelia and sporidia respond negatively to gravity, growing upward, making aerial spread by sporidia probable. If not desiccated, sporidia are viable 8-12 weeks between -5 and +5°C. The soil surface is typically humid under snow or straw during winter. Tyler and Jensen (1958) reported increased incidence of dwarf bunt in wheat in small depressions in the field. This may be evidence of movement of sporidia by water.

Unlike spores of *T. caries* and *T. foetida*, which tend to germinate simultaneously and completely under ideal conditions, germination of teliospores of *T. controversa* is protracted, beginning in 3 weeks, increasing during the 3-5 weeks period, so that maximum germination under ideal conditions requires 6-8 weeks in most collections (Hoffmann, 1982). Ideal conditions for laboratory germination are a soil extract agar medium, 3-5°C, and continuous low intensity light.

In nature teliospores on the soil surface germinate beginning in mid-October and continuing through January - February. Common bunt spores are adapted to germinate promptly on seed surfaces when the seed is planted in moist, cool soil, followed by invasion of the seedling through the coleoptile in the dark beneath the soil, typical of seedling-infecting smuts. Teliospores of *T. controversa* are adapted to germinating over a prolonged period on the soil surface, infecting very young shoots at the soil surface, typical of shoot-infecting smuts.

In retrospect it is easy to understand the difficulties early workers experienced working with *T. controversa* spores. Their knowledge of *T. caries* and *T. foetida*, whose spores germinate promptly (in 3-7 days) at moderate temperatures in light or darkness, couldn't have helped. Who saves dishes 40 - 100 days to check for germination? Who would go to special effort to provide 5°C or close to it, and who would concern themselves with light? The fungi differ in their basic biology, which is reflected in the disease cycles and epidemiology.

Spore longevity. Dwarf bunt is much more dependent upon the environment than common bunt, and epidemics do not result from seed-borne spores. The sporadic occurrence of the dwarf bunt increases the importance of longevity of spores in soil. Dwarf bunt may appear in significant quantities 4-7 years after the last severe outbreak, evidence of spore longevity (Holton et al, 1949). According to Baylis (1958) and Hoffmann (1982), spores free in the soil survive at least 3 years. Tyler and Jensen (1958) state that viability diminishes rapidly at first, but that some infectivity remained after 10 years (also true for flag smut according to Line). Holton et al (1949) concluded that rotation is impractical.



When submerged in anhydrous l-propanol, the majority of the spores of 25 races of *T. caries* and six species of grass smuts (*T. fusca*, *T. bromi-tectorum*, *T. guyotiana*, *T. holci*, *T. elymi* and *T. scrobiculata*) collapsed. Over 90% of *T. controversa* spores remained spherical (Trione and Krygier, 1977). Whether resistance to this alcohol treatment represents a mechanism contributing to spore longevity in soil is not known. This resistance may reflect some property of the sheath, spore wall or membranes that contributes to survival.

Common bunt is soilborne to a significant degree only where very dry conditions prevail between harvest and the seeding of winter wheats. Dwarf bunt is soilborne throughout its geographic range, including relatively humid countries like Germany and Switzerland (Purdy et al 1963).

Host invasion. In a parallel study of host invasion by *T. caries* and *T. controversa*, Hansen (1958) saw no differences in the details of pathologic histology. Host penetration and subsequent development of the fungus with respect to tissues were the same in common and dwarf bunt. Infection from spores on the seed is not normal in dwarf bunt, but Hansen developed an early seedling inoculation method. When the plumule of the seedling was 2-5 mm long, she cut off the tip of the coleoptile and submerged the seedling in a sporidial suspension to inoculate it. Hyphae grew in the spaces between the embryonic leaves, invaded them, and reached the growing point in 30 days at 3°C. At 15°C, the fungus made little progress. If the plants were first held at 3°C, then moved to 15°C, bunt developed. In common bunt, handled similarly, bunt developed with early incubation at either 3 or 15°C.

In a highly resistant wheat, P.I. 178383, the fungus penetrated the host directly, proceeded intercellularly through the coleoptile into the first and second leaves, and failed to develop further (Fernandez et al, 1978). In resistant wheats (Requa and Nugaines) the fungus advanced into primordial leaf and nodal tissues, but failed to establish within the terminal meristem. In a susceptible wheat (Red Bobs) it reached the terminal meristem, maintained this position through stem elongation, and, when ovaries were differentiated it filled the ovary with teliospores. Smuts of this type remain minimally active during the vegetative stages of host development and accelerate greatly nearing the end of host development. Sporulation normally does not occur in vegetative tissues. The intact ovary, as in common bunt, forms the outer covering of the bunt ball.

Time and place of infection. Unlike common bunt in which infection occurs shortly after germination of the seed infection by *T. controversa* occurs over a prolonged period of time after seedling emergence. Wheat was seeded 27 September 1954 and spores were applied to the soil surface October 5. By November 22, long after the wheat emerged, a gray growth of sporidia was visible on the soil surface (Meiners et al, 1956). This, along with the increase in smut from shallow seeding led Meiners et al to conclude that infection occurred at or close to the soil surface, often long after seedling emergence. Fungicides applied to the soil surface in Japan, Europe and Utah (in Purdy, 1957) controlled dwarf bunt. Dwarf bunt was controlled by HCB on the soil surface applied 4 weeks after emergence (Purdy, 1957), evidence that infection by dwarf bunt occurs much later than in common bunt.

Direct evidence of the time of infection was obtained by growing wheat plants from disinfected seed in sterile soil, then transplanting to infested soil (Tyler, 1958). Infection was heavy (86% or greater) in plants 20-45 days old at inoculation and 27% in the 52-day old plants. The oldest plants had 7 main tillers, 7 small tillers and 17 tiller buds per plant at the time of inoculation, yet some infection occurred. Tyler suspected that infection occurred during winter, because several workers reported that a straw covering of the soil increased dwarf bunt. Tyler and Jensen (1958) found that covering wheat with straw in December in New York increased dwarf bunt. They concluded that winter was a critical period for infection, and that the straw cover was even more effective in increasing dwarf bunt than snow.

When plants grown in infested soil were removed from the field in November or early December, very little or no smut developed (Purdy et al, 1963). Plants were removed from the field, washed, submerged in a

mercurial fungicide and transplanted to smut-free soil. Most of the plants became infected from late December through February, but systemic infection was possible to some degree until early April.

Factors favoring dwarf bunt. Dwarf bunt is common in areas in which the soil is covered with snow during December through February and the soil is not frozen solid all winter. *T. controversa* develops at low temperature but it is doubtful that it progresses much when temperatures are more than a degree or two below freezing. Snow moderates winter temperatures at the soil surface and protects against desiccation. Breeders, rather than relying upon capricious snow cover, often cover wheat with straw after the plants have become dormant, usually in late November or early December if snow has not yet covered the soil (Tyler and Jensen, 1953, 1958; Dewey, 1961). Some trials involved seeding wheat in rather deep trenches, then spreading vermiculite in the row about the wheat plants as a substitute for straw.

In contrast to common bunt, shallow seeding increases disease (Meiners et al 1956). Tyler and Jensen (1958) reported a small but consistent increase in dwarf bunt in a planting 2.5 cm compared to 5 cm depth. Deep seeding (6 cm) reduced dwarf smut in all but the earliest seeding date (August 19) compared to very shallow (1 cm) seeding (Hoffmann and Purdy, 1967). Most workers believe that shallow seeding concentrates the young tillers near the soil surface, leading to greater infection, but Hoffmann and Purdy found the crowns of both seeding depths were near the soil surface.

Early and very late seedings reduce dwarf bunt but drastic modifications of seeding date are impractical. Early seedings exposed wheat to Hessian fly in Ontario, and when farmers seeded after the Hessian fly free date, dwarf bunt increased (Connors and Skolko, 1953). Tyler (1958) concluded that the older tillers of early seeded wheat were not susceptible, that the tissues had matured to the extent that they had become resistant. Hoffmann and Purdy (1967) found the fungus capable of infecting plants in various seeding dates, but that plants with no or few tillers when winter began were most susceptible (Table 2).

Table 2. Effect of seeding date on dwarf bunt (Hoffmann and Purdy, 1967).

	Seeding Dates				
	Aug 9	Sept 2	Sept 15	Oct 1	Oct 14,
% infected heads	33	50	73	86	41
% of plants with all heads smutted	6	20	53	71	84
Tillers per plant, Nov. 4	24	8	3	1	0

The younger plants from later seedings were most frequently completely smutted, with only 6% of the oldest plants having all the heads smutted. Hoffmann and Purdy emphasized that the fungus must have considerable ability to penetrate host tissues. (Maybe some cultivars continuously produce young tillers before winter so that succulent shoots are present even in early seedings. Tyler reported 7 main tillers, 7 small tillers, and 17 tiller buds on early-seeded winter wheat in New York, 1958.) Seeding date is important in common bunt through its effect upon soil temperature at the time of seeding. Seeding date in dwarf bunt may influence the number of young, susceptible tillers present during winter when most infection occurs. At Logan, Utah, 1984-1985, with four seeding dates, the yield potential was greatest from the August 30 seeding, the least from the October 11 seeding (Hoffmann and Sisson, 1987), but early seeding had little effect in reducing smut in this trial.

Circumstantial evidence is strong in Washington that early seeding in summer fallowed soil reduces dwarf bunt. Seeding in the snow mold region is typically early and deep in warm, soil and dwarf bunt has not

been sufficient to depress yields for 20 years. Six wheats were seeded with farm drills on naturally infested soil August 29 and October 4 by R. J. Cook and J. Waldher (1979, unpublished) in Lincoln County, Washington under farm conditions. Smut beyond trace amounts developed only in the later seeding on Wanser and Nugaines (Table 3). Sprague has the same resistance genes as Nugaines (*Bt1*, *Bt4*) yet it escaped infection. Does it have more determinate tillering?

Table 3. Influence of seeding date on dwarf smut on two farms, Williams and Burgeron. R. J. Cook and J. Waldher, 1980.

Variety	Tillers With Dwarf Smut (%)			
	Early Seeding		Late Seeding	
	Williams	Burgeron	Williams	Burgeron
<u>Resistant to Smut</u>	%	%	%	%
Hansel	0	0	tr	0
Moro	0	0	tr	tr
Luke	0	0	0	0
<u>Susceptible to Smut</u>				
Wanser	tr	tr	22.5	28.3
Nugaines	tr	tr	27.5	26.2
Sprague	0	0	tr	tr

Each value is an average for four reps. Early seeding was Aug. 29, 1979; late seeding was October 4, 1979.

Factors such as compact soil (Holton, et al 1949) and snow drifts (references in Hoffmann, 1982) result in local variations in dwarf bunt incidence within fields. Earlier, when the moldboard plow was extensively used, dwarf bunt often developed in an alternate crop year sequence (Holton, et al 1956). Stubbles of a heavy smut year were plowed under. The next crop had light smut. The next plowing brought the inoculum near the surface, with a high smut incidence. Most land is now tilled with implements that do not invert surface soil, so a biennial incidence of dwarf smut is seldom observed today.

Chemical control. Thiabendazole (TBZ), a systemic fungicide, is registered for dwarf bunt control (Hoffmann and Sisson, 1987). It is effective only when wheat is seeded late. Applied to the seed TBZ is inadequate in early seeded wheat, either because it is metabolized or because it is diluted greatly within large plants that develop from early seeding. The main infection period, December through February, occurs long after application of the fungicide to the seed in early seedings. In late seedings, with no tillers at onset of winter, thiabendazole is effective (1% smut from October 11 seeding), ineffective from August 30 seeding (69% smut).

It is logical to assume that *T. controversa* could be eradicated by foliar sprays with systemic fungicides applied in the spring. No material applied to foliage at practical rates in the spring significantly reduced smut, however. For some reason *T. controversa*, when established within the host, is resistant to systemic fungicides (Dewey, et al 1983).

Competition between common and dwarf bunt. When common bunt was rampant dwarf bunt was unimportant and it was little studied. In the years after common bunt was controlled dwarf bunt became a major problem in some chronic snow areas, such as in northern Utah and southern Idaho. This increase in dwarf bunt may be due to reduction in common bunt. Bamberg et al (1947) found that common bunt suppresses dwarf bunt. Susceptible wheats were seeded in fields naturally infested with *T. controversa* in Utah and Montana. Half of the seed was inoculated with spores of various races of common bunts, half was free of common bunt spores (Table 4). A small number of plants were infected by both smuts,

but these are omitted from the table. *T. foetida* was established within the host before *T. controversa* spores had germinated and the growing points of many plants were occupied by *T. foetida* before *T. controversa* began to progress.

Table 4. Suppression of dwarf bunt (*T. controversa*) by common bunt (*T. foetida*) (Bamberg et al, 1947).

Inoculum on the seed	Infected with (%)	
	Dwarf bunt	L-2
L-2 <sup>a</sup>	12	68
none	56	0
		<u>L-7</u>
L-7	14	70
none	57	0
		<u>L-8</u>
L-8	9	68
none	59	0

<sup>a</sup> L-2, L-7 and L-8 are races of *T. foetida*. The dwarf bunt was whatever race(s) was (were) present in the field.

When common bunt was controlled by HCB, dwarf bunt either increased or was more noticeable, or both. Purdy et al (1963) found it necessary to state that treating the seeds with HCB did not directly increase dwarf bunt; it removed competition with common bunt.

Seed-borne inoculum. Spores of *T. controversa* on seed cause so little infection that dwarf bunt is not considered seed-borne (Holton et al, 1949). Spores or spore balls introduced with the seed are of greater danger to the next crop of wheat than to the current crop. If heavily infested seed is planted very shallowly (1.3 cm), however, some disease in the current crop can develop (Grey, et al, 1986). In common bunt, 0.5 to 1.0 g of spores per 100 kg of seed, is about all the spores the seeds will retain.

Table 5. Effect of seeding depth and inoculum level on dwarf bunt (Grey, et al 1986).

Seeding depth	Check	Inoculum, g spores/100 g seed			
		0.001	0.01	0.1	1.0
<u>Idaho</u>					
1.3cm	5	4	2	1	26
5.0cm	3	0	3	5	2
<u>Utah</u>					
1.3cm	2	9	5	46	65
5.0cm	0	5	0	0	17

Grey et al (1986) calculated that the 0.1 g of spores per 100g of seed represented 20,000 teliospores per seed. No grain lot from PNW ports was found during 1983 that was infested at the 0.1g/100g of seed level, so the conclusion was reached that, except under conditions very conducive to dwarf smut, the dwarf smut content of grain from commercial fields would be of slight importance other than to introduce the fungus into a new area. In most trials at several locations 0.1g of spores/100 grams of seed had no effect on the amount of dwarf bunt present. Little wheat is seeded at a depth of 1.3 cm in normal practice. Grey et al speculated that even though the spores were applied to the seed in a slurry of

methylcellulose and dried, some spores rubbed off during planting and what infection occurred may have come from spores in or on the soil rather than from those on the seed.

Pathogenic specialization. Until 1952 there was little evidence for more than one race of *T. controversa* in the PNW, probably because of the prevalence of common bunt and of little research on dwarf bunt. Races of dwarf bunt had been identified in Europe, usually no more than two in a country (see Hoffmann, et al 1962). In a study of 49 collections, Hoffmann et al (1962) identified eight races from Washington, Oregon, Idaho and Montana. They were identified using the differentials used to identify races of *T. caries* or *T. foetida*. Race D-1 was virulent only on the  $M_2$  gene, making it the equivalent of T-1, L-1 and L-2. In the 1976 study D-1 was still the least virulent dwarf bunt race, but by then they found it virulent against the *Ho* (*Bt5*),  $M_2$  (*Bt7*) and *Bt10* genes. Maybe the cut-off percent for calling a wheat susceptible (5% smutted) should be raised for dwarf bunt because of the 'leakiness' of resistance genes to dwarf bunt when very high inoculum levels, shallow seeding and favorable conditions exist. Under field conditions common bunt and dwarf bunt did not differ as greatly (Table 21 in common bunt section) as in a severe trial (Table 23, common bunt series).

By 1976 17 D-races (*T. controversa*) were described (Hoffmann and Metzger, 1976). The only D-races with equivalent common bunt races listed by Hoffmann and Metzger were D-8 and T-19, D-4 and T-15, D-7 and T-24. They listed no equivalent L-races (*T. foetida*).

I believe the capabilities of these fungi to overcome resistance genes in wheat exceeds the genes available in the host. When common bunt was important and inoculum in the soil was abundant, races capable of attacking new resistant wheats were either already present or were discovered, often before the new wheat reached the farm. These smuts appear to be genetically more competent than the rusts. Is this because each generation goes through sexual reproduction, or is it because of superior potential to adapt to the host?

### Taxonomy

I have discussed the bunts according to the taxonomy accepted at Pullman until the present. There was never any doubt that *T. caries* and *T. foetida* are very closely related, differing primarily in having ornamented (*T. caries*) or smooth spore walls (*T. foetida*). Flor (1932) found them completely interfertile in the first matings made of these species, as did Fischer and Holton (1957) when they studied inheritance of spore wall types in inter-species crosses. In 1969 Hoffmann and Kendrick (Table 1 under dwarf bunt) found that monokaryons of *T. caries* mated normally with A4+ and A1- of *T. controversa*, very strong evidence of a close relationship. L. M. Kawchuk, W. K. Kim, and J. Nielsen (1988) proposed the following nomenclature.

*Tilletia tritici*  
*T. tritici* var. *tritici* (*T. caries*, syn.)  
*T. tritici* var. *levis* (*T. foetida*, syn.)  
*T. tritici* var. *controversa* (*T. controversa*, syn.)

The proposed combination of *T. caries*, *T. foetida*, and *T. controversa* as varieties of *T. tritici* can do no harm, whether justified or not. But students of the smuts should remember the many differences between dwarf bunt and common bunt. (*T. controversa* - presence of a sheath and larger areolae than *T. caries*, greater number of primary sporidia per promycelium, difference in longevity of spores in cool moist soil, response of *T. controversa* spores to light, lower temperatures, slow germination, more complex incompatibility system, time of host infection and differences in effect upon tillering, stem length, spike morphology).

### Final Note

After preparing the above treatment, I learned that, due to changes in the Botanical Rules, *T. tritici* (= *T. caries*) and *T. levis* (= *T. foetida*) are the presently accepted names, bring us back to the time of T- and L- races of *T. tritici* and *T. levis*. Rather than change the text I leave it as it was, to illustrate what shifts in nomenclature do to the student.

*Partial Bunt of Wheat (Karnal Bunt)*  
*Tilletia indica* Mitra  
*Neovossia indica* (Mitra) Mundkur

Partial bunt of wheat was discovered near Karnal in the Punjab in May, 1930 (Mitra, 1935), and the disease became known as Karnal bunt. It was found in the Punjab and the Northwest Frontier (Mitra, 1935) at lower elevations on the plains. Mundkur (1943) found it also at Saharanpur in the United Provinces (Dupler et al, 1987) adjacent to the Punjab. It is now known in Uttar Pradesh, West Pakistan, Nepal, Iraq and Afghanistan. Durán (1972) found *T. indica* on wheat from Sonora, Mexico. It was probably introduced on seed from India.

The spores of *T. indica* are difficult to germinate and Mitra (1935) concluded that a rest period is required. Germination of spores increased after 4 months, and light stimulated germination (Smilanick et al, 1985). The spores are adapted to germinate during flowering of a new wheat crop, not during growth of the crop that produced them. Mitra (1935) obtained germination between 15 and 25°C, but not at 10 or 32°C. Spores germinate down to 5°C, and maybe lower, but germination is slow (Hoffman). The promycelium is indeterminate in length, ranging all the way from 10 to 1500 µm. The protoplasm migrates to the tip where Mitra counted 32-128 primary sporidia per whorl. Unlike sporidia of the common bunts, he observed no fusions (no H-cells). Mundkur (1943) believed the long promycelia are adapted to produce primary sporidia above the soil surface by teliospores slightly buried in soil. Holton (1949) confirmed the observations of Mitra (1935) on the great variation in length of the promycelia, and the number of primary sporidia ranged from 26-171 per promycelium with an average of 117. Neither Mitra nor Mundkur (1943) observed sporidial fusions, but Holton found a single H-cell among hundreds of primary sporidia, so fusion is very rare. Fusions do not occur in *Neovossia*, and this is one reason Holton favored this genus rather than *Tilletia* for the pathogen.

The spore-bearing hyphae give rise to lateral 'bud clusters' which develop into teliospores. The epispore is highly reticulate (more so than in *T. caries* and *T. controversa*) and it has a sheath (Mitra, 1935). The spores are larger than in common bunt, black, and a short attachment hypha (appendage) remains on some of the spores (Mitra, 1935). In *Neovossia* the appendage on the spore is hyaline and usually as long or longer than the diameter of the spore (Fischer and Holton, 1957, p. 27). In *T. indica* it is short, one reason Fischer favored *Tilletia* as the genus.

In partial bunt infection is local, not systemic, and not all spikes and not all kernels are bunted (Mitra, 1935). Usually there are only 1-5 spikelets per spike with bunted kernels. At first the diseased kernels are hidden behind the lemmas and paleas, but as the grain expands and ripens the kernels are exposed to view. In severe cases the hulls spread widely and may fall off. Kernels may also fall to the ground. The smut has a fishy smell like the other bunts of wheat. The embryo is intact and partially bunted kernels germinate more quickly than healthy kernels (they probably imbibe water more rapidly through the ruptured pericarp). There is little aerial spread of spores from diseased kernels at flowering time as is the case in loose smut (*Ustilago tritici*). This description of Mitra is given in some detail because I consider his study a classic.

The epidemiology of partial bunt was clarified by Mundkur (1943a). Mundkur (1943a) obtained some infection with teliospores soaked four days and then applied to the spikes by Moore's vacuum method of inoculating with loose smut. Mundkur speculated that probably few of the teliospores actually

germinated. Spraying spikes at anthesis with sporidial suspensions resulted in more smut. Mundkur saw no symptoms prior to flowering. The erratic and sparse distribution of infected kernels indicated local infection by air-borne spores. He believed the spores on the soil surface germinated in January and February in India when moisture was present and temperatures were moderate. Mundkur speculated that teliospores function primarily not by being air-borne themselves, but through the sporidia. In 1942 ample rain fell in the second half of January and in February, and partial bunt was epidemic at Karnal. In 1943 only traces of rain fell during heading and flowering and partial bunt was rare. The temperature was the same in both seasons, so he stated that moisture at and after flowering favored infection. At Gurdaspur in East Punjab sporidia functioned if it was rainy and not too hot at flowering or just afterwards.

Inoculating seed and soil failed to produce disease (Mundkur, 1943b). When the flowers were open and susceptible, sporidia from teliospores of the previous crop of spores produced partial bunt (Mundkur, 1943a). The role of airborne sporidia was conclusively proven by Bedi et al, 1949, when bagged heads remained free of partial bunt.

The supposition that primary sporidia contain haploid nuclei, and that some sort of fusion is necessary to establish a dikaryon was proven by Durán and Cromarty (1977). Single primary sporidial cultures were increased in shake cultures at 24°C. Secondary sporidia were abundant. Use of monosporidial cultures resulted in no disease. When paired, compatible cultures were identified by disease development. Four incompatibility alleles were identified. The fungus is heterothallic and bipolar with multiple incompatibility alleles at a single locus. Only two mating types were obtained from sporidia from a single promycelium. Three mating types,  $a_1$ ,  $a_2$ , and  $a_3$  were obtained from spores from a single sorus, evidence that the sorus represented infection by more than a single pair of compatible sporidia. Aerially disseminated primary sporidia that are individually unable to reproduce seems to be an inefficient system. Each ovary must be infected by at least two compatible primary sporidia.

Dhaliwal et al, 1983, studied the orientation of naturally infected kernels within individual spikes and spikelets at Gurdaspur. Previous workers believed each infected kernel was the result of an individual local lesion, but the fungus is capable of infecting more than one kernel within a spikelet, and in some cases, of progressing to an adjacent spikelet. A single infection can, therefore, result in a few or even several infected kernels. The kernel closest to the rachis in each spikelet was most damaged, indicating that it is the site of initial infection.

According to Dhaliwal et al (1983) the fungus can spread to new florets from primary infections as late as the dough stage. This means it can penetrate relatively mature tissues, that it is not restricted to meristematic or very young tissues during its vegetative activities. Durán and Cromarty (1977) obtained disease both by spraying spikes during anthesis and by injecting sporidial suspensions into spikes still within the boot. Fuentes-Davila and Durán (1986) inoculated individual kernels in the milk stage and smut developed. How long tissues are susceptible is still only vaguely understood.

Teliospores are initially binucleate, becoming uninucleate during maturation (Fuentes-Davila and Durán, 1986). Meiosis apparently occurred within the germinating teliospore, followed by several mitotic divisions, resulting in the many haploid nuclei that subsequently migrate into the primary sporidia. *T. indica* is typical in that the sporogenous hyphae are dikaryotic and single haploid lines are nonpathogenic.

Resting spores germinate equally on autoclaved or on natural soil, (Dupler et al, 1987) so fungistasis plays no role in the biology of partial bunt. Only spores on the soil surface or very near it can produce aerially disseminated sporidia. Teliospores do not germinate rapidly, even under optimum conditions, so moist soil for some time prior to and during heading is critical. Dupler et al (1987) suggest that under irrigation agriculture allowing the soil surface to remain dry during this period could be important in control.

Even though this smut is not known in the US it has been studied by Holton, Fischer, Durán, Cromarty, and Fuentes-Davila of WSU, and by Hoffmann at Logan, Utah. Its presence in the CIMMYT (Rockefeller, Ford Foundation) wheat breeding program in Mexico is a real handicap because quarantines to prevent the spread of *T. indica* into the US and other countries restrict distribution of seed of valuable crosses.

Holton and Fischer used to argue whether to call this pathogen *Neovossia* or *Tilletia*, Holton favoring *Neovossia*, Fischer favoring *Tilletia*.

### *Flag Smut of Wheat*

Flag smut of wheat, caused by *Urocystis agropyri*, was important in South Australia before 1868, with losses in occasional fields near 100% (see L. H. Purdy, 1965, for a thorough review). McAlpine (1910) reviewed flag smut, with emphasis on Australian observations. By the time McAlpine wrote his book it was known that the flag smut fungus of wheat did not infect rye, and that the flag smut fungus of rye (*U. occulta*) did not infect wheat; that early seeding of winter wheat (= warm soil) and relatively dry soil at seeding favored disease, that seeding in cool, wet soil controlled it; on virgin soil the disease increased with successive wheat crops; that infection resulted from both seed- and soilborne spores; that seedlings were susceptible before emergence; that seed treatments only prevented infection from spores on the seed. (The Australians learned about seed- and soilborne infestations of flag smut before the Americans understood this relationship in common bunt in the PNW.) Seed treatments of McAlpine's day, and until systemics became available, were ineffective against soilborne inoculum.

McAlpine could not germinate newly collected spores, but 40% of year-old spores germinated in tap water. He suspected that a resting period or form of transitory endogenous dormancy existed in the spores. After-ripening, or resting period, mentioned also in partial bunt (*T. indica*), refers to a period of aging required to make the spores germinable. True after-ripening cannot be overcome by short exposures to special temperatures, chemicals, etc. (Griffin, 1981 - p. 265-266).

Taxonomy of the pathogen. Spores from diseased wheat in Australia were identified by R. Wolff in 1873 as *Urocystis occulta*, the flag smut of rye pathogen. F. Koernicke in 1877 concluded that the Australian wheat flag smut fungus differed morphologically from that from rye, and he named it *U. tritici*. McAlpine (1910), finding that the two were pathogenically distinct, accepted Koernicke's *U. tritici* as the name of the wheat flag smut fungus. Inoculation trials by subsequent workers corroborate the distinct host preferences of these fungi. In 1943 G. W. Fischer concluded that *U. tritici* is a synonym of *U. agropyri* described from *Agropyron repens*. In cross-inoculation trials, spores from wheat resulted in smut on eight species of *Agropyron*, three of *Elymus* and one of *Hordeum* (Fischer and Holton, 1943). *U. agropyri* was found in nature on *Sitanion jubatum* in Klickitat County, Washington, before it was found locally on wheat. When wheat was inoculated with a composite of spores of *U. agropyri* collected from several grasses, 6% of the wheat plants became smutted. When spores from these wheat plants were used to inoculate wheat, 17% of the wheat plants were smutted. Fischer and Holton (1943) concluded that *U. tritici* arose from *U. agropyri*, and that *U. agropyri*, by priority, is the valid name of the wheat flag smut fungus. Grasses are not considered an important source or inoculum or a factor in epidemiology.

Pathogenic specialization. Pathogenic races of *U. agropyri* complicate the development and use of resistance, but to a small degree. The most extensive study of pathogenic specialization is that of Johnson (1959). US race 1 was somewhat similar to Chinese races 1 and 2. US race 2 was similar to race 2 of Chile and somewhat similar to Australia 2A and South Africa 2B. Race 3 (Japanese) and race 4 (from India) and all other races from China differed from US 1 and 2. Hafiz (1951) found US-1 only from the US, US-2 from Italy and Australia, and a group 3 from China and Cyprus. Smut from Pakistan had no counterpart among his collections from other countries. Races exist, but their number within a geographic region is small. Pathologists should be cautious because C. S. Holton found that the variety Rex was susceptible to US-2 in the greenhouse, but it was resistant in infested commercial fields.



Geographic distribution. Flag smut of wheat has been reported from all continents (Purdy, 1965), and, being seed-borne, it has probably been introduced frequently into many regions. *U. agropyri* was important in parts of Australia, China, South Africa, northern Egypt, the Punjab of India and Pakistan, and in local parts of the U.S. Local farming practices and differences in the inherent resistance of wheats in a region influence the prevalence of flag smut, but climate is the main factor.

In South Australia, Victoria, and New South Wales, where the disease has been important, the summers are dry with most of the precipitation in the cool months. Wheat is seeded in late summer or fall to be harvested the next summer, as in Washington, Oregon, and in the Punjab of India and Pakistan. Flag smut is primarily a disease of winter wheat. The general opinion is that spring wheat develops so rapidly the fungus does not maintain the necessary relationship with the meristem of the shoot. More important, probably, is that spring wheat is seeded relatively shallow in cool moist soil (Rollie Line agrees).

In the US flag smut in wheat was first recognized in 1919 in Madison County, Illinois, where it was identified by J. G. Dickson. It was found in 111 fields in 1920, and was soon found in Missouri and Kansas (Griffiths, 1924). Seed treatments, later seeding, rotation and resistant varieties eliminated flag smut in central US. Its elimination before the development of systemic seed treatments that protect against soilborne inoculum is evidence of lack of adaptation of the pathogen to this region.

Flag smut was found on wheat in Klickitat County, Washington in 1940 and soon thereafter just across the Columbia River in Wasco County, Oregon. It persisted in and was restricted to this area for 20 years, until very susceptible club wheats were widely grown in eastern Washington and Oregon. It then spread in winter wheat grown on summer fallow. Extreme host susceptibility (cv Omar, then Paha) increased its geographic range, but according to Rollie Line, losses outside Klickitat County have been insignificant. Hoffmann reports it appeared in the 1980's in Box Elder and Cache Counties, Utah, and that it apparently died out shortly after. It is present in Salt Lake County.

Soil temperature. In southern Australia, seeding winter wheat early when the soil is warm and relatively dry favors flag smut. Seedings in central US resulted in a range of 61% smut from an October 17 seeding to 0% after November 14. Seeding late enough to control the disease was considered agronomically unacceptable (Humphrey and Johnson, 1919). At Kuming, Yunnan Province, China, Yu et al (1945) stated that seeding about 15-20 October when soil temperatures were in the 13-25°C range, favors flag smut. This is near the ideal time to seed winter wheat in this region. Late seedings (late November) usually had little smut.

In the Punjab near Lyallpur summer temperatures are consistently above 27°C, and wheat seeded before 15 October escapes because of high soil temperature. Satter and Hafiz (1952) recommended seeding before October 15 at Gurdaspur and Karnal, before October 21 at Gurgaon, and before October 25 at Lyallpur, all in the Punjab but at different elevations above sea level. These dates were only about 1 week before customary seedings, so in the Punjab adjusting the seeding date was practical.

Experience in lower Egypt, near Kokki, was similar (El-Helaly, 1948). The mean air temperature in mid October was near 25°C. Smut was greatest in wheat seeded near December 1 when the mean air temperature was 20°C and falling. Where summer temperatures are very high (India, Egypt) early seeding controls flag smut. Where summer temperatures are moderate, as in Washington, early seeding favors flag smut. The soil temperature at seeding time and for a week after is very important.

Soil moisture. When inoculated seed was sown in soil at a temperature of 15.5-19.7°C, smut was 0 at 28% water content, 5 at 21.7%; 10 at 17%, 65 at 11.3%, 55 at 8%, near 40 at 5.5%, and 20 at 4.5% water (Sattar and Hafiz, 1952). These water contents are hard to interpret, but smut was prevented in the wettest soil and favored by moderately dry to dry soil (was this soil very sandy? How can seedlings emerge in a soil at 4.5% water content?). Line (unpublished) found that flag smut is favored by the same moisture levels that favor seed germination and seedling emergence.

Soil moisture x temperature. Faris (1933) reported that when soil is rather dry, high infection occurred from 10-20°C; when soil was moist high infection occurred from 10-15°C. When soil was very wet high infection occurred only at 10°C. As soil water increased soil temperature became more limiting.

The most precise data are those of Purdy (1966, Table 1). The soil he used was at permanent wilting point at 10.8% water content (-15 bars) and at water holding capacity at 24% water by weight (= -0.3 bars). Purdy inoculated the soil and used smut-free seed, so all measurements are from soil-borne spores. The plants were removed shortly after emergence, washed, disinfected, and transplanted to smut-free soil. All plants were watered the same and they were held at the same temperature after transplanting, so only conditions during germination and emergence varied. Disease was greatest at 11-13% moisture and 10-20°C.

Table 1. The effect of soil moisture and temperature upon flag smut of wheat from soilborne spores (Purdy, 1966).

Temperature	Moisture, %					Smut, average
	10	11	13	18	24	
5°	2	10	10	7	1	6
10°	30	75	80	60	30	55
15°	65	68	78	50	45	61
20°	67	75	77	55	35	62
25°	0	0	0	0	0	0
Smut, average	33	46	49	34	22	

Soil temperature is more critical than soil moisture, with low infection at 5°C and no infection at 25°C. Verwoerd (1929) reported a trace of infection at 28°C in South Africa. It is possible that spores of different origin differ, because several workers obtained some infection at what must have been 25°C or higher.

Seeding depth. McAlpine (1910) stated that dry soil and early seeding (warm soil) favored flag smut; that late seeding (cool or cold soil) in wet soil greatly reduced smut, but field observations are confounded with seeding depth. When the surface soils are relatively dry, farmers seed more deeply to reach moisture. When the soil is wet they seed relatively shallow. Soil moisture and seeding depth normally vary inversely. In summer fallow in Washington wheat is seeded beneath a dust mulch into relatively warm, moderately moist soil, ideal for flag smut infection.

When Miller and Millikan (1934) seeded winter wheat 5 cm deep in Australia, 51% smut resulted. When seeding was as shallow as possible, 22% were smutted. In Egypt, where soil infestation is important, Jones and El-Nasr (1940) seeded wheat at different depths in soil with different irrigation regimes. Very shallow seeding reduced smut. El-Helaly (1948) repeated these trials and verified that deep seeding increased smut (Table 2).

Table 2. Effect of seeding depth on flag smut (El-Helaly, 1948).

Smut, %	Planting depth, cm					
	0.5	5	7.5	10	15	20
	29	35	46	42	48	58

Jones and El-Nasr (1940) developed a concept, based on several seedling-infecting smuts, to explain the effect of depth of seeding. Seedlings are generally susceptible only until the coleoptile is ruptured by the emergence of the first true leaf at emergence of the seedling from the soil. Deep seeding prolongs this period and increases the time for the fungus to establish itself in the terminal meristem. They commented that soilborne inoculum was relatively unimportant in Australia because seed treatments, mainly coppers, were effective in Australia but they reduced smut only by about half in Egypt.

Mechanical operations. Jones and El Nasr attributed the relative importance of soil infestation in Egypt to the method of threshing. Flag smut sori are mainly in stems and leaves. In Egypt the wheat was cut, chopped up and beaten and winnowed by tossing the refuse and wheat into the wind. The heavy kernels were separated from straw and chaff, and the spores were disseminated by the wind. In central-eastern Washington and Oregon, where *U. agropyri* has persisted, the summers are dry and stubble mulching is common to preserve moisture. The straw and refuse are not plowed under as with a moldboard plow. With the passage of time and with each tillage operation the above-ground residues gradually disintegrate. After 1 year of fallow, the refuse is largely gone. Tillage spreads spores in the tillage layers as the enclosing host tissues deteriorate. Fewer spores are liberated into the air during threshing than in common bunt. In the latter all the spores are in the heads. If wheat with flag smut is lodged, however, and the sickle is lowered, much stem and leaf tissue is threshed, resulting in increased spore showers.

R. Line (personal communication) believes that flag smut was favored in Klickitat County, Washington, because cattle were an important part of the farming system. Line believes leaving the stubble untilled until the following spring, allowing the cattle to graze the refuse, retarded the break down of refuse left in the fallow field. A New South Wales leaflet for farmers by F. C. Butler (1949) recommended early plowing when wheat must follow wheat, with thorough cultivation before sowing in the belief that incorporating the spores into the soil as soon as possible favors early breaking of spore dormancy with some lethal germination in the absence of a host.

Summer rain. It is easy to assume that arid summers as in the south-western South Africa, most of southern Australia, and in Oregon and Washington favor survival of spores in the soil between crops of winter wheat. Sattar and Hafiz (1953) in the Punjab region of northern India and Pakistan, however, noted that flag smut was most severe where summer rain was substantial. In this area of Pakistan summer temperatures are consistently above 27°C. High soil temperatures probably prevent spore germination, even when the soil is moist. They made the following observations. Summer = June 1 to November.

<u>Location</u>	<u>Summer rain, inches</u>	<u>Flag smut</u>
Gurdaspur	32.3	high
Hoshiarpur	26.	"
Lyalpur	7.9	trace
Montgomery	6.0	0
Multan	5.2	0

When spores of Punjab origin were coated onto dry seed or introduced into soil shortly before planting little smut developed. When dry spores were mixed with dry soil and irrigated 12 days before seeding, then seeded with clean wheat seed on November 22, 5.4% smut developed. When inoculated seed was planted November 22 and irrigated immediately, 2.4% smut developed. They concluded that most of the spores remained dormant, even those wetted 12 days prior to seeding.

To investigate the possible effect of summer rain, they heavily inoculated the surface 5-10 cm of soil June 1 with fresh, new-crop spores at Lyallpur. Part of the soil was watered every 7 days. These plots received a total of 35-36.3 cm of water during the summer in addition to rain. The plots all received 7.5 cm of water in early November, and 12 days later they were planted with clean wheat. Soil containing the

spores wetted frequently produced 47 and 71% smutted plants in the first and second crop, respectively. Soil containing spores subjected to only the sparse summer rain (6.3-11.5 cm per summer) produced 4 and 16% smut in the first and second crops, respectively, even though 7.5 cm of water was added 12 days prior to planting. Summer wetting and drying at high temperatures did not destroy the spores; if anything their germinability was greatly increased. They did not study the effect of summer watering on longevity beyond the time between successive wheat crops.

Longevity of spores on seed and in leaf refuse. Spores on the outside of infested wheat kernels live so long that it is safe to assume they remain infective as long as wheat seed remains viable. Noble (1934) cut dried wheat leaves with mature sori into small pieces and exposed them to different relative humidities at room temperature. In dry air (0-33% RH) survival was excellent for 10 years, after which the trials were terminated. At 50% RH, survival was good for 3 years, with a trace germination for 3 more years. At 64% RH, survival was only 2 years. At 73 and 89% relative humidity *Penicillium* spp. and other xerophytes destroyed the leaves.

Longevity in soil. Wheat is the only economic host, so longevity in soil has significance in devising rotations. Verwoerd exposed pots containing heavily infested soil outdoors in South Africa. In each of four years some of the pots were planted to wheat. In the first year, 87% smut; year two, 54%; year three, 33%; year four, 10%. Significant numbers of spores were present three years after the initial infestation. Rotations of this duration are uneconomic in most dryland wheat regions because few cash crops other than wheat are available.

At Lyallpur in the Punjab some spores were alive after 3 1/2 years in soil under natural conditions (Sattir and Hafiz, 1952). An area averaging 27% smutted plants was divided with solid walls between plots to minimize contamination. All seed was smut-free. In treatment A wheat was grown each year for four years (41, 45, 57, 62% smut). In treatment B, the land was fallowed the first year (= -, 18, 29, 35% smut). Treatment C included two fallow years (-, -, 5, 10% smut). Treatment D, no wheat for three years (-, -, -, 0.2% smut). This experiment demonstrated the decline of soilborne spores with time and its increase with successive wheat crops. Some spores survive 4 years in soil but rotations are beneficial. Purdy (1965) gives examples of longer survival in soil, especially in Australia.

Viable spores in soil could be detected after six years using the very susceptible cultivar Paha (Line).

Practical inoculation methods. Miller and Millikan (1934) performed extensive field experiments on resistance and soil factors and concluded that inoculating the seed was the easiest and most practical method. Use of infested host material applied to soil was too laborious and results were less uniform.

In the Punjab, where new spores produce low amounts of smut when applied to seed, Sattar and Hafiz (1953) used soil containing spores conditioned by weekly wetting and drying as described earlier (under summer rain). Wheat was inoculated by coating the seeds with infested soil or by covering the seed lightly with infested soil. When the seed was coated, 18% smut resulted. When clean seed was dropped in a furrow in clean soil, covered lightly with infested soil, followed by clean soil to completely covering the seed, 47% smut resulted. Under their conditions covering seed with soil containing conditioned spores was most effective.

Rollie Line in Washington tests fungicides for efficacy and breeding lines of wheat for resistance. At first resistance trials were in Klickitat County in infested fields. Line obtains satisfactory results at Pullman by inoculating the seed and the soil in the seed furrow with dry spores and by seeding the wheat early and deep, making it unnecessary to travel to conduct flag smut trials.

Spore germination. The inability of early workers to germinate spores of *U. agropyri* consistently hindered many studies (McAlpine, 1910).

Noble (1923), working with new crop spores from Australia and the US, studied germination. He reported the optimum temperature for soaking (conditioning) was near 20°C.

Putting spores 6-14 months old into nutrient solutions of various types or pH levels did not stimulate germination (Noble, 1923). Soaking the spores 3 days in water, and then adding bits of live wheat seedlings to the water, induced 60-70% germination. These spores had already gone through after-ripening. Without addition of the bits of live wheat or bits of some other seedlings to the soak water, few spores germinated, even after 9 days in water. If conditioned spores respond to host exudates in soil in this manner, the spores respond to fungistasis. The gradual decline of spores in soil in the absence of a host, as in the survival experiments of Verwoerd and of Sattar and Hafiz indicated a relatively straight line loss of vigor, a lack of true adaptation to long-term survival in soil, and fungistasis, if present, is of little survival value to these spores.

Early workers, including McAlpine and Verwoerd, found that teliospores of *U. agropyri* that matured normally in wheat plants did not germinate until after an indeterminate rest or after-ripening period, varying from 28-82 days in different collections. Allan and Durán (1979) collected spores from mature sori in early August, 1972, at Pullman, Washington. After 2 months they extracted the spores and stored them in plastic vials at 18-20°C in darkness for 3 months, so they began their studies with spores 5 months old. They tested germination at monthly intervals after floating spores on the surface of distilled water for 72 hours at 19°C, ideal conditions for hydration. The floated teliospores were then streaked on 2% water agar. Dry teliospores were also streaked on the agar as controls. Germination of hydrated spores was low (about 10% for 5-23 months) but at 24 months germination abruptly increased to 34%, at 25 months, 40%; from 27-29 months, 47% germinated). Dry spores dusted on the agar did not exceed 3% germination regardless of age. The abrupt increase in germination at 24 months may be evidence of a second restricting mechanism that is released by age.

After conditioning at 20°C for 6 days, wheat seedling extract was added and the watch glasses were placed in incubators with different temperatures (Noble, 1923). In 3 days 77% germinated at 5°C, in 2 days 97% germinated at 13°C, and after 1 day germination was high at 18, 20, 24°C (91, 92, 88%, respectively), and 81% at 27°C. A few germinated at 32°C. Germ tubes were strongest at 24°C, and strong between 18 and 27°C. Conditioned spores, according to these in vitro results, should be able to infect wheat over a considerable temperature range, with optima between 18 and 24°C. 81% germination at 27°C is at odds with the low disease development in soil that warm.

Spores germinated at pH's between 3.6 and 7.1 in Noble's 1924 trials, with the optimum between pH 5.1 to 5.7. Sporidia were not produced at the pH extremes. Miller and Millikan (1934) state that in Victoria, Australia, flag smut occurs on a variety of soils, but alkaline soils favor flag smut. South of the Mallee Region, Foster and Vasey found most flag smut in plots receiving the most lime (near pH 8.3). In a variety of experiments flag smut was severe in soils ranging from 5.5 to 8.7 (the entire range of soils available?). The soils of the semi-arid regions of Washington in which flag smut is adapted were mostly neutral to slightly alkaline from 1940 to at least 1970. Field results could be biased because relatively dry, warm soil favors flag smut, and seeding winter wheat in warm, relatively dry soils is common in semi-arid regions, in which soils were naturally neutral or slightly alkaline.

Carolyn Roybal Allan (1975) studied the effect of pH on conditioning. She presoaked spores at 19°C for 3 days in solutions of differing pH and then streaked them on water agar. No germination occurred after soaking at pH 2 and 11. Germination within 24 h increased from about 3% at pH 3, 8% when soaked at pH 4, 40% at pH 5, peaking at 46% at pH 6, about 40% at pH 7, 30% at pH 8, 10% at pH 9, and 3% at pH 10. These experiments on the effects of differing presoaking conditions on spore germination are more compatible with field infections in alkaline soils in Victoria, Australia than the results of Noble. Allan used potassium diphthalate buffer solutions for the 3-day soak period but she spread the spores on regular water agar for actual germination so her methods did not truly duplicate those of Noble. It would be profitable to presoak and germinate the spores at the same pH values.

The mystery of spore germination continued. Sattar and Hafiz (1952) tried Noble's method of soaking mature spores in water for 3 days and then adding host material. Spores from Australia germinated well, those from the Punjab did not. They floated fragments of diseased leaves on the surface of water for 3 days. The spore material was then placed in a watch glass containing a 1:200 dilution of sap extracted from wheat seedlings (much stronger than in the Noble technique), and then they inverted a watch glass over it and sealed it with vaseline. Spores from the Punjab germinated 45-48% in 18-24 hours at 22-24°C. In another trial, 55% germinated between sealed watch glasses, 5% in open watch glasses. Using spores in sealed watch glasses they obtained 0 germination at 5-12°C, 12% at 15-17°C; 25% at 20-22°C, 56% at 22-24°C, 8% at 25-27°C, and 0 at 30-37°C.

Even after much work, the factors governing germination of *U. agropyri* spores are still mysterious. It is obvious that many things may influence their response - age, presoaking, temperature, plant exudates and volatiles present in soil, and differences between fungal strains.

Conclusion. Flag smut, like common bunt, is easily controlled by local action seed treatments except in regions where spores survive in soil. Common bunt is favored by seeding in cool, moist soil; flag smut by seeding in relatively warm, dry soil. Both are favored by deep seeding, in contrast to dwarf bunt which is favored by shallow seeding. If we ever lose the use of systemic seed treatments flag smut could be controlled in Washington by resistance.

#### *Other Smuts*

Loose Smuts of Wheat and Barley. The only times I have seen significant loose smut has been when seed of a new malting spring barley was brought to Washington from the mid-west, usually from North Dakota. Older, wiser pathologists (Marion Harris, Stew Holton) advised me that no control measure was necessary. Even when smut incidence is high in the first crop, it diminishes rapidly thereafter with no control measures. The spores of *U. nuda* are liberated when healthy barley is flowering. High humidity is essential at flowering time for spore germination and infection and this is rare in Washington. Maybe if all chemicals are lost, Washington can grow barley and wheat seed for other regions. The seed would also be free of many other seed-borne pathogens (if produced without sprinkler irrigation). No real research effort has been made on loose smuts here.

Oat Smuts. Horses and mules, prime consumers of oats, were the source of farm power when the experiment station began, and oats were second to wheat among the cereals for many years. Unlike common bunt, the only effective inoculum of oat smuts is externally seed-borne. Copper sulfate and formaldehyde were reasonably effective. George Zundel surveyed 167 fields in Whitman County in 1918 and all had covered smut (Heald, 1919). West of the Cascades covered and loose smuts were about equal. Loose smut was prevalent east of the Rocky Mountains. Heald suspected that differences in soil temperature at seeding time affected regional distribution of the seedling-infecting smuts of oats. Heald believed that if you had less than 5% smut it wouldn't pay to treat the seed! If we had 5% smut in our crops today farmers would take it very seriously.

C. S. Holton monitored races of the oat smuts for the entire US for years.

Head Smut of Corn. In most of the US common corn smut (*Ustilago maydis*) is the important smut, but in Washington head smut (*Sphacelotheca reiliana*) is more important. Dana and Zundel reported head smut at Pullman in 1919 and in 1921 C. S. Parker reported it in several areas, particularly in the Yakima Valley. About 1963 head smut became a problem in Golden Jubilee, a highly susceptible sweet corn, near the Yakima River. Walter Hendrix and several of his students tried unsuccessfully to develop control measures. Head smut infection results primarily from spores in the soil and available seed treatments were ineffective (Tim Smith, MS thesis, 1975). Tim found that infection occurred over a prolonged period of time. Corn germinated in clean soil and transplanted to infested soil after 1 day was 90% smutted;

transplanting after 3-9 days into infested soil = 71-81% smutted plants; 12-24 days, 47-64%. Smith developed a soil assay to determine the level of soil infestation.

Twin City, the main processor, enforced a five-year rotation by not purchasing corn grown with shorter rotations. When the problem first became serious they tried washing the ears, but according to Philip E. Bloom, retired county agent of Kittitas County, this only contaminated all the corn. They quit accepting corn with any smut. Farmers rogued the fields prior to mechanical harvest, removing all smut sori. The problem has since been controlled by growing a less susceptible sweet corn and using rotations. Field corn is more resistant and head smut has not been a production problem in it.

Smuts of grasses. George Fischer concentrated on grass smuts. Stew Holton on cereal smuts. Studies of grass smuts have great fundamental importance, enlarging our knowledge and filling gaps in understanding this important group of plant pathogens. Rubén Durán also devoted much of his effort to grass smuts. Several students did thesis research on grass smuts, as did extension pathologist George Zundel years ago. These studies contributed to establishing Pullman as the smut (study) capitol of the world.

Fischer and his students hybridized many smuts to study their true relationships and to grasp their evolution. He emphasized spore morphology and inheritance of spore morphology, and these studies led to some revisions in their taxonomy. In 1939 he found that *Ustilago hordei*, covered smut of barley, attacked several grasses (*Agropyron*, *Elymus*, *Hordeum nodosum*, and *Sitanion*). *Ustilago levis* (covered smut of oats) is indistinguishable morphologically from *U. hordei*, but it does not attack barley and has a distinct host range of its own.

Stem smut (*Ustilago spgazzini*) is very common on quack grass along mowed roadsides in Whitman County, and Fischer studied the taxonomy of stem smuts in detail.

By 1940 Fischer was convinced that *Ustilago bromivora* and *U. lorentziana* were synonyms of *U. bullata*. He identified eight races of *U. bullata* on the basis of their reaction on 14 differential species, and in 1953 Jack Meiners and Fischer identified 12 races. *U. bullata* was so common on cheat grass (*Bromus tectorum*) that it was thought of as a possible biological control but this did not work out. *U. bullata* is a real problem in seed production of several grasses. The Soil Conservation Service saw usefulness in Mountain Brome and marked differences, important in seed production, were found among accessions (1947). Fischer and Meiners (1952) tested seed treatments for the control of head smut (*U. bullata*) in several grasses.

This sample of research on grass smuts gives an idea of the nature of most of the studies.

## **Other Wheat Diseases**

### *Wheat Rusts*

Stripe Rust. *Puccinia striiformis*. Stripe rust is most serious in regions with moderate winters, cool springs and moderate summers. It is the most serious rust of wheat in Washington. According to H. B. Humphrey, C. W. Hungerford, and A. G. Johnson (1924), C. V. Piper collected stripe rust in western Washington in 1892. The early collections, mostly on grasses, were misidentified as leaf rust, but E. Bartholomew suspected a collection in Wyoming was *P. glumarum*, a synonym of *P. striiformis*. F. Kølpin Ravn of Denmark visited the far western states and positively identified stripe rust in 1915. Stripe rust was common in the San Juan Islands and Skagit Valley, and, on the basis of grasses attacked, Humphrey et al speculated that stripe rust came to America from Asia via grasses along the Pacific Coast from Alaska southward. Stripe rust is the accepted name in the US, as recommended by the above authors (Humphrey, Hungerford, Johnson), but it is called yellow rust in Great Britain and some other European countries.

Rollie Line stated that identification of stripe rust in the far West by Ravn in 1915 alarmed the USDA. Stripe rust might spread into the Great Plains and be truly serious. The USDA stationed C. W. Hungerford, followed by W. M. Bever (1928-1937), at Moscow, Idaho, to study stripe rust. When the USDA became convinced that stripe rust was no great threat to the wheat industry, the work was terminated and Wayne Bever left for the University of Wisconsin where he obtained his Ph.D. in 1940.

Hungerford (1923) found viable urediospores all winter long at Corvallis, Oregon 1917-1918, and he found that *P. striiformis* overwintered as mycelium in live, infected host leaves at Moscow, Idaho. He believed the urediospores of *P. striiformis* were shorter-lived than those of the other cereal rusts. Stripe rust, with the synonym *P. glumarum*, can develop on the pericarp of kernels. Hungerford determined that spores on or mycelium in wheat kernels were not a source of inoculum: *P. striiformis* is not seed-borne.

Hungerford, in cooperation with C. E. Owens of Oregon State University, began a search for resistance and a study of races of the pathogen (1923). Wayne Bever (1938) tested 317 American wheats, 1284 foreign wheats, 365 barleys, and 11 ryes for resistance. Stripe rust research in Idaho (and Washington) essentially died with the departure of Bever (1937) and it remained dead in Washington until about 1957.

Between 1937 and about 1957, no single wheat dominated central Washington, and several Turkey-type wheats were resistant. In retrospect, this mix of wheats and the nature of their resistance (high temperature, adult plant) did much to control stripe rust. I came to Washington in 1954, and for a short time thereafter I considered stripe rust more as a curiosity than as a real problem. The most stripe rust I saw would be isolated patches, maybe in a low corner of a field. The release of Omar, a winter club wheat of superb quality, smut resistance, and high yield changed everything. By 1960 Omar occupied 67.5% of the wheat acreage of the entire PNW (J. Walter Hendrix, WAES Circular 424, 1964). This club wheat was widely grown, even in the dryland, summerfallow regions west and south of the Palouse. It was highly susceptible to stripe rust. For the first time a highly susceptible wheat dominated the entire region. Rust spores blew from the earlier areas south and west of the Palouse, in a sense a repetition of the smut spore shower phenomenon described by Heald and George. In 1960 Hendrix estimated a 7,500,000 bushel loss, in 1961, a 15,000,000 bushel loss. Yields of susceptible wheats at Pullman were reduced 45% on average. Susceptible wheats were grown on 81%, intermediate wheats on 8%, and resistant wheats on 11% of the total acreage in the PNW. Brevor (resistant), the second most important cultivar, was grown on 6.9% of the land, illustrating the dominance of Omar. I witnessed the stripe rust epidemics of the early 1960s and believe Hendrix was conservative in his estimates of losses. Complacency vanished. All breeders in the region concentrated on replacing Omar with suitable resistant wheats, and these efforts continue to the present.

Hungerford stressed overwintering. Hendrix stressed oversummering. The urediospores of *P. striiformis* are not adapted as survival structures and living mycelium in the host is the main means of persisting during adversity. Summers of the PNW east of the Cascade Mountains are so dry the grasses either die or go dormant, and wheat and barley mature, so a period lacking green leaves is normal in July and August. Hendrix tried to stop the use of wheat as a summer cover crop under irrigation. Rollie Line found a wheat cover crop in an irrigated orchard in the Columbia River Gorge, a perfect location to be a source of urediospores blowing up the Gorge to the Horse Heaven Hills and northcentral Oregon and from there to the Palouse.

During the early 1960's, personnel at the Army Biological Laboratory at Ft. Detrick, Maryland became interested in stripe rust, especially in its epidemiology. The result of that interest was the allocation of research grants to scientists in California, Oregon, Montana, and Washington. Walt Hendrix was the recipient of this support for some years.

L. H. Purdy, USDA, started to work on stripe rust. Prior to this, Purdy had been primarily responsible for research on flag smut and chemical control of common bunt. There soon developed a territorial conflict between Purdy and Hendrix which required the intervention of state and federal administrators before it



was settled. The result limited Purdy to studying resistance and identification of races and Hendrix to the biology of the rust and chemical control. Purdy left the project in 1966 and Rollie Line replaced him in January 1968.

The number of races of *P. striiformis* increased rapidly as breeders released resistant cultivars, and the increase in races with efforts to breed for resistance continues. By 1980 Line had identified 21 races, called CDL races, after the now defunct Cereal Disease Laboratory. Line, Clarence Peterson and Robert Allan emphasize nonspecific resistance because of this trend in race proliferation. The nonspecific resistance of Nugaines, at first called mature plant resistance, is temperature sensitive, functioning best as the season progresses and temperatures rise. The resistance of Nugaines has been unaffected by new races. Some cultivars now combine specific and nonspecific resistance, the ideal combination. In addition, R. E. Allan developed the multiline Crew, consisting of a composite of individuals with different specific resistances.

Line developed precise recommendation for proper use of Bayleton (and other foliar fungicides) for stripe rust control when resistance fails or when rust develops on a susceptible wheat. In conjunction with these efforts, Stella Melugin Coakley used meteorological data and Line's rust data to develop predictions of the probability of losses. Winter temperatures are very critical, and these are known well in advance of the time wheat would be sprayed, so these studies are useful.

When stripe rust, which develops during cool weather early in the season, occupies the leaves leaf rust is "controlled" because the leaves are already parasitized, leaving no tissues upon which leaf rust can develop.

Stem Rust. Stem rust (*Puccinia graminis*) has historically received very little study in the PNW. E. C. Stakman surveyed stem rust west of the Rocky Mountains to the Pacific in 1918 from a railroad car. Whenever the train stopped near a field or grass he ran out and grabbed some material and got back on the train. He never missed the train. Races west of the Continental Divide differed markedly from those of the Great Plains (in "E. C. Stakman, Statesman of Science," by C. M. Christensen). The best review of the literature that I have seen is in the thesis (1972) of Francis McCracken, a graduate student with Walt Hendrix. He cites a paper (1935) in which the authors claim *P. graminis* can overwinter as uredia in the PNW. To my knowledge there is no evidence to prove this, at least not east of the Cascades, even though George Fischer implied in a seminar report (1944) that it might do so in very mild winters. Francis McCracken found no evidence of uredial overwintering in 1959, 1960, and 1961. Holton believed the rust depended upon the barberry. Line (1989) knows of no evidence for uredial overwintering east of the Cascades.

The studies of McCracken indicate a slow decline in vigor and eventual death of *P. graminis* hyphae and urediospores during the protracted low temperatures of late fall and winter. Bruehl (Ann. Rev. Phytopath. 14:254, 1976), in discussing the role of sexual states of fungi, made this statement. "Three rusts attack wheat in Washington. The perfect stage is important only in *Puccinia graminis*. Unlike *P. striiformis* and *P. recondita*, *P. graminis* is unable to overwinter here on living wheat leaves."

The "Inland Empire" is isolated in all directions, free of substantial spore showers originating in other areas, making it dependent upon local primary inoculum. Convinced of this, Washington and the USDA cooperated in a barberry eradication program, 1944 until some time in the 1970s when the program was abandoned. Convictions of local pathologists played the determining role. There was no official barberry eradication program in Idaho or Oregon, but I suspect progressive wheat growers eradicated many bushes themselves. Rollie Line has evidence that the barberries are essential. Recent local outbreaks were traced to surviving barberries, and collections of *P. graminis* from this area contain several races, many of which are relatively avirulent, evidence of a sexually produced population not yet exposed to significant selection (Line).

Fischer (seminar summary, 1914-1944) reported epidemics in 1916, 1937, 1938, 1941, 1942, and 1943 in the Palouse. No correlation with severe or mild winters was found, but rainy days averaged 11 in June and 3.5 in July in rust years; 5 and 1.7 respectively, in the other years, indicating moisture in June and July as a limiting factor. Between 1952 and 1972 McCracken reported only one stem rust year (1958). Fischer reported six in 30 years, McCracken 1 in 20 years, circumstantial evidence that barberry eradication paid? Or was spring wheat more important in the earlier years?

Stem rust is active on wheat at higher temperatures than leaf or stripe rust, and in Washington it is usually dangerous only to spring wheat. In the early periods spring wheat was widely grown. It matures later in warmer weather, making it subject to damage by stem rust. Spillman, in improving winter wheat for the area, provided the impetus for the dominance by winter wheat. In recent decades spring wheat occupies about 10% of the wheat acreage, except in years of severe winter kill when dead winter wheat is replaced by spring wheat.

Local breeders have not bothered to breed for stem rust resistance in winter wheat, but in recent years losses occurred on Luke and Lewjain, late-maturing winter wheats. Rollie Line states that stem rust was important on spring barley and wheat in 1980-1984. My own feeling is that winter wheat breeders can continue to concentrate on other problems if late-maturing wheats are avoided. Also, extension should periodically remind farmers in the wheat area of the danger from barberry and encourage them to eradicate the bushes when found.

Leaf rust. Cereal pathology in the PNW was dominated by smut research for so long, accurate early records of the importance of rusts are largely lacking. As expressed in one of the first annual reports, common bunt was important to some extent every year. Rust (whatever rust) was sporadic. I think it safe to say that, historically, leaf rust was least important.

Leaf rust became important after stripe rust-resistant wheats dominated the region. Stripe rust, because it develops earlier under cooler conditions, essentially excludes leaf rust from the leaves.

Breeding for leaf rust resistance in winter wheat is a relatively recent effort. Rollie Line stated that in 1980 about 99% of the wheat grown in the PNW was classed as susceptible, based on reaction type (pustule characteristics). This statement is misleading, however. Several of the spring wheats are "slow rusters", delaying pustule development and reducing losses. Line and Gene Milus studied slow rusting in detail. I cannot help but conclude, however, that historically leaf rust has been a minor problem. This does not mean that real losses do not occur. They do, and leaf rust resistance is a valuable trait.

The leaf rust (*Puccinia recondita*) that race situation is unclear to me. In a seminar in 1978 Gene Milus stated that WPR (Western *P. recondita*) race 1 was present since 1926, WPR-2 was discovered on Norco in 1976 at Pullman, WPR-3 was found at Mt. Vernon in 1977, and WPR-4 was isolated as a component of WPR-2 in greenhouse studies in 1978, making four races up to that time, based on extensive collections and studies. In contrast, C. O. Johnston, 1959, reported 10 races from seven collections from Washington, and in 1968 Johnston, et al stated that race UN-11 was prevalent 1936-1960. These discrepancies are doubtless the result of changing differentials and means of identifying races.

In 1978 Daws, Hyslop, Luke, and McDermid, important cultivars at that time, were resistant to WPR-1. These all inherited the *Lr-1* gene for resistance from Sel. 101, an important semi-dwarf breeding line. There was little protection in 1978 to WPR-2, because 98% of the wheat acreage was in cultivars susceptible to it. This race was present east of the Cascade Mountains where it was easily the most important race. Line concludes that as more emphasis in the breeding program is placed on specific resistance to leaf rust, the number of races will increase, the reason breeders and pathologists alike are stressing nonspecific forms of resistance. By 1989 Line had identified eight races, but most were new combinations of virulence genes rather than new virulences.

## Foot Rot

The only true "foot rot" of wheat of consequence is caused by *Pseudocercospora herpotrichoides*. McKinney (1925) discussed the use of the term foot-rot, but the origin of its application to rots of the culm base at or near the soil line is not known. Atanasoff (1920) used it in reference to *Fusarium graminearum*, but it, like *F. culmorum*, attacks both the roots and the base of the stem. *Gaeumannomyces graminis* blackens the stem base, but it is probably more of a root rot than a foot rot. *Pseudocercospora herpotrichoides* does not attack roots, making it a true foot rot.

Strawbreaker foot rot. *Pseudocercospora herpotrichoides*. Called eye-spot in Europe because of the elliptical lesions on basal leaf sheaths and culms.

The first verified specimens of strawbreaker foot rot in the U.S. were those of Heald, 1919, collected northeast of Hillyard (near Spokane). Heald consistently isolated a dark fungus from diseased stems, but he could not identify it because it did not sporulate in culture. In France a fungus called X, because they too couldn't identify it, produced symptoms of foot rot (Foex and Rosella, 1929). Sprague found elongate, septate conidia in X cultures grown outdoors at Corvallis, Oregon in the cool part of the year. The spores were like those of *Cercospora herpotrichoides* (= *Pseudocercospora herpotrichoides*), described from wheat in France by Fron in 1912. At ordinary laboratory temperatures in the dark *Pseudocercospora herpotrichoides* does not sporulate, the reason French workers and Heald were unable to identify it. The mysterious X- fungus was now identified. Before Sprague identified the pathogen the disease was locally called Columbia Basin foot rot because it was not known to be similar to the disease in France.

Pioneer farmers near Hillyard in the Peone Prairie believed that before 1910 footrot was limited to the base of north slopes (Sprague, 1936). Foot rot attracted attention because it was locally destructive, causing white heads and lodging. Between 1919 and 1933 foot rot was known in northern Idaho, north central Oregon, and in limited parts of Asotin, Columbia, Klickitat, Spokane, Walla Walla, and Whitman Counties (Sprague, 1939). The 1933-1934 season favored foot rot and Sprague estimated the loss for Washington at 285,000 bushels. In 1948 he added sites in Yakima and Lincoln Counties.

Sprague (1937) was a competent agrostologist and ecologist, and he associated foot rot with a *Festuca-Agropyron* consociation. The association was so strong he believed foot rot would be limited to these areas. He also suspected that the pathogen was native, largely on *Agropyron* spp. In 1948 he still limited it to areas that were lush sod-grass in the days of the Indians, areas with 14-25 inches of rainfall.

By 1968 foot rot was found essentially wherever wheat was grown in eastern Washington, except in rotation under irrigation (Bruehl et al., 1968, Bull. 694). Sprague was a thorough collector and there is no doubt that the discrepancy between the distribution of foot rot he described and that in 1968 was real.

Foot rot is associated with cool, moist climates, especially in northwestern Europe, yet it occurred in our true dryland, even in the driest wheat areas of Washington, such as in the Horse Heaven Hills in Benton County. This was so unexpected Bruehl and Juju Manandhar (1972) grew isolates from differing rainfall areas on media adjusted to differing water potentials. There was no evidence of dryland ecotypes of *P. herpotrichoides*. The conclusion was reached (1968, Bruehl, Walt Nelson, Fred Koehler, Orville Vogel) that with continued winter wheat-fallow-winter wheat, as farmers improved their summer fallow, seeded earlier, fertilized more and doubled their yield, the fungus expanded its geographic range. The fungus hadn't changed, conditions had. Better moisture conservation, earlier seeding, and improved soil fertility changed the microclimate and host physiology at the soil line during late fall, winter and early spring, the time when the fungus sporulates and invades the host. We are fortunate when a skilled observer like Sprague leaves detailed records so that subsequent scientists can plot changes in disease distribution.

The use of benomyl to control foot rot documents the profound changes in the distribution and severity of foot rot. In an unusually severe season, 1933-1934, Sprague estimated the loss in Washington at

285,000 bushels. In contrast, in some years in the 1970s as many as 500,000 acres of wheat were sprayed with benomyl at a cost of at least \$5,000,000. The farmers did not believe they wasted their money. It would take an increase of 3 bushels per acre to pay for the fungicide x 500,000 acres = 1,500,000 bushels of wheat, probably a great underestimation. The disease increased greatly since the surveys of Sprague. Murray is improving recommendations for the use of fungicides, as to when needed and when to apply them. Fungicide resistant strains of *P. herpotrichoides* are appearing. Murray is documenting their distribution and is actively seeking fungicides to replace Benlate, Topsin, and Merteck, all of which are similar. Bruehl advocated the use of Benlate for years before the DuPont Company decided its use was beneficial. This company did not jump at a new market. Ironically, the first year they tried to sell Benlate for footrot control, no one used it.

#### *Resistance to Strawbreaker Foot Rot*

Roderick Sprague found *Aegilops ventricosa* highly resistant to *Pseudocercospora herpotrichoides* (1936). European breeders, about 1953, crossed *A. ventricosa* (genome DDMM) with *Triticum persicum* (AABB) and obtained fertile amphidiploids by doubling the chromosomes with colchicine. This wheat was crossed with a common wheat, Marne, to form VPM-1 (= Ventricosa x Persicum x Marne), transferring the resistance of *Aegilops ventricosa* to common wheat. Robert Allan, USDA at Pullman, has released two varieties, Madsen and Hyak, with the resistance of VPM-1 for local use. The resistance was discovered by Sprague, its use was made possible by French cytogeneticists, and a local breeder now has the resistance of *A. ventricosa* in local wheats, a long, round-trip back home.

Palani Chidambaram (PhD 1976) found lignitubers in host tissues. Murray (1981) found that the hypodermis of VPM-1, and of Cappelle-Desprez, another resistant wheat, was highly developed. Murray and Ye (1986) found marked papillae and cell-wall thickening in epidermal cell walls of resistant wheat seedlings. Carl Strausbaugh (1988) found that resistance in some crosses with Cappelle-Desprez was governed by one semi-dominant gene and in VPM-1 by one dominant gene.

I think the lignified hypodermis important, that resistance to *P. herpotrichoides* is not totally governed by one or two genes, unless these genes have multiple effects.

#### *Mt. Saint Helen's Ash*

*Pseudocercospora herpotrichoides* infects the plant at or slightly below the soil line and, under favorable conditions, it girdles the stem, resulting in lodging (= strawbreaker). A field about 5 miles north of Lind, was damaged to an unusual extent in the dry environment (annual precipitation about 10 inches) of that location. Wheat is seeded in summer fallow with a deep-furrow drill in a dust mulch, leaving ridges 14-16 inches apart. A farmer did not like driving trucks across the ridges at harvest, so in early spring, when the soil was moist and mellow, he habitually harrowed the fields to partially smooth the soil surface. In so doing, friable soil covered the base of the plants in the furrow (in March). After several years the result was severe foot rot and a 50% loss. Anhydrous ammonia was commonly injected into fields of growing winter wheat in early spring in the dryland area, and even this slight "tillage" sometimes increased the severity of foot rot in friable soils (Bruehl, Nelson, Koehler, Vogel, 1968). Any operation that placed soil about the crowns of winter wheat (= dirting) in early spring is potentially dangerous.

On May 18, 1980, Mt. Saint Helens deposited over 2 cm of ash on the soil surface at Lind. We feared that the ash mulch would greatly increase foot rot. Instead, yields were higher than normal. Nevertheless, knowing the effect of harrowing wheat in the spring in the dryland region on foot rot, in 1980-1981 and 1981-1982 the bases of wheat plants in deep-furrow drill rows were covered with ash or with soil to see if foot rot would be increased (Bruehl and Machtmes, 1984). When the wheat was 'dirted' with ash or soil April 13-20, disease was increased and yields reduced. When dirted May 18, the date of the volcanic eruption, disease was not increased. Apparently 'dirting' must occur early in the spring to

have a significant deleterious effect. Maybe the next time a volcano covers wheat with volcanic ash someone can predict the result better than we could.

### *Erosion Control in the Palouse*

The Palouse Hills are covered by deep, moisture-retaining soil subject to severe erosion. Until some time in the 1970s, much winter wheat was seeded on summer fallow. If winter wheat is seeded in mid-September it makes much growth before fall and forms a protective ground cover, controlling erosion. In favorable growing seasons standard height winter wheats seeded this early lodge severely before harvest. In an effort to make early seeding practical, Orville Vogel, USDA wheat breeder at WSU, developed semi-dwarf wheats. Reducing plant height reduced the danger of lodging, making earlier seeding possible. The first semi-dwarf wheat, Gaines, was named in honor of E. F. Gaines, the early breeder of bunt-resistant wheats at WSU. The semi-dwarf Gaines not only enabled farmers to seed early and avoid severe lodging, but yields were increased 25% over the best standard-height wheats. This was the second great achievement in wheat breeding here (the first being the development of Hybrid 128 by Spillman).

Early seeding favors barley yellow dwarf, stripe rust, and strawbreaker foot rot, especially the latter. We now had wheats adapted to early seeding, but they were subject to several problems. Foliar applications of benzimidazole fungicides controlled foot rot on early-seeded wheat, making early seeding economical. Murray, about 1988, began to find resistance to the fungicide, but as of 1990 resistance is not general enough to render these fungicides ineffective.

Idaho, Oregon, and Washington formulated a federally supported project, called STEEP (Solutions to Environmental and Economic Problems), that in Washington was largely aimed at sustained wheat production and minimizing soil erosion and water run-off. STEEP started in 1976 and it is still in progress (1989). STEEP funds support efforts to farm with as little soil tillage as possible, including seeding wheat into standing stubble. Agricultural engineers, soil scientists, breeders, economists and plant pathologists became involved. Bruehl (foot rot), Murray (foot rot, cephalosporium), and Duran (common bunt) received STEEP funds.

A recent development is reduced use of summer fallow in the higher rainfall areas of eastern Washington, in favor of a crop produced every year, locally called recropping. Each crop exhausts the soil moisture of the entire root zone, increasing the capacity of the soil to absorb fall and winter rains, helping to reduce soil erosion. In dry summers and falls in recropped soil there is insufficient moisture to germinate seed until rains come in the fall, resulting in later seeding which decreases strawbreaker foot rot and Cephalosporium stripe.

Efforts to control erosion stimulated changes in farm practices that changed disease problems, especially diseases caused by soil-borne pathogens.

Sharp eye-spot. *Rhizoctonia cerealis*. Sharp eye-spot, like eye-spot (*P. herpotrichoides*), causes elliptical lesions on basal leaf sheaths and stem bases, but the margins of the lesions are dark brown, giving the lesions distinct, sharp borders. In eye-spot (strawbreaker foot rot) the lesion margins blend in color into that of healthy tissues, making less distinct borders. In eastern Washington sharp eye-spot has no economic importance, with lesions being few per stem and essentially superficial. In dense stands in humid western Washington *R. cerealis* spreads 8-10 inches up the stems, and minute sclerotia may form within the elliptical lesions (Sprague's book, p. 133 for an illustration). Jim Patras, a former graduate student, found the sexual state, a *Ceratobasidium*, on material of this type at Puyallup.

Diseased wheat collected by County Agents in Thurston, Cowlitz, Snohomish Counties in 1918 and in Clark, Grays Harbor, Klickitat, Lewis, Pierce and Whatcom Counties in 1919 was studied by Dana, Heald, and Zundel. Their descriptions are confusing but they include symptoms caused both by *R. cerealis* and

by *Gaeumannomyces graminis* var. *tritici*. Dana identified what he called *R. solani* on some of these plants. Sprague isolated what he also thought a distinct strain of *R. solani* from wheat in Oregon. The fungus was a weak pathogen of alfalfa.

Sprague (Popular Bull. 192, 1948) reported sharp eye-spot important on poor soils in Mason and Thurston Counties. He recommended lime and rotation with legumes. Patras and Bruehl observed extensive stem colonization in a foot rot (*Pseudocercospora*) nursery, and wheats resistant to foot rot were susceptible to *R. cerealis*. The same relationship has been reported in western Europe.

So far as I know this disease is relatively insignificant, even in Western Washington.

### **Root Rots**

Take-all, *Gaeumannomyces graminis* var. *tritici*. Dana, Heald and Zundel, 1918, 1919, described symptoms on winter wheat we now know were caused by *Ceratobasidium cerealis* and *Gaeumannomyces graminis* var. *tritici* on specimens sent by county agents in Clark, Cowlitz, Grays Harbor, Lewis, Pierce, Snohomish, Thurston and Whatcom Counties. Dana isolated *Rhizoctonia* from some of the specimens, but Sprague, who found some of the wheat in the herbarium, identified take-all as the main disease. The years of collection suggest to me that the plea for greater wheat production during World War I caused some farmers to grow wheat without rotation. Take-all was obviously widespread in Western Washington.

Take-all, world-wide, is favored by cool, wet weather and by light, neutral or alkaline soils, but, even though soils of Western Washington are mostly acidic, take-all without rotation is devastating. I twice forgot about take-all while working with yellow dwarf at the Vancouver station, and take-all destroyed my winter wheat when it followed winter wheat, once on a clay soil and once on a sandy soil. Rotation is essential in this region. Weller and Cook test candidate bacterial isolates for control of take-all by seed bacterization at Mt. Vernon, taking advantage of this situation.

Take-all was unimportant in Eastern Washington (Sprague, 1948), except in seasons with unusually mild winters and prolonged wet springs. Sprague did not stress take-all under irrigation. Take-all is serious under irrigation when rotation is ignored, leading me to conclude that farmers in the older irrigation projects of that time had learned to rotate long ago, so Sprague had no reason to stress the disease. Bruehl, who came in 1954, considered take-all very low priority because farmers in Western Washington and irrigation farmers had the option of several crops, making rotation feasible.

Chuck Gould studied *G. graminis* var. *avenae*, on turf grasses, the only state pathologist to work extensively with this species. Ophiobolus patch was serious on putting greens, and Gould and Roy Goss, Extension Agronomist, learned to control it with ammonium sulfate fertilizer or other strong soil acidifiers which were at least as effective as fungicides.

Some farmers in the new Columbia Basin Project began to grow wheat in monoculture, and take-all became important, leading James Cook to study the disease. Yields at first were high, but as take-all increased, yields declined. Some farmers continued to grow wheat, take-all declined, and yields began to increase, usually after about 4 or 5 years of monoculture. This unusual, spontaneous decline in disease severity was called take-all decline in Europe. It is attributed to the increase of microorganisms favored by the diseased roots. Cook and Alan Rovira of Australia in 1976 attributed take-all decline mainly to special strains of *Pseudomonas fluorescens*. Peter Shipton, a visiting take-all decline expert from Britain, Cook and Jerry Sitton studied decline (1973), especially in a field near Quincy. This field is the source of many bacterial isolates highly antagonistic to *G. graminis*.

Late in the 1970's farmers in the Palouse learned that rainfall was adequate to grow a crop every year, that summer fallow was not really essential, leading to annual cropping (recropping). At about the same

time farmers began to try minimum or no-till farming in an effort to reduce erosion. Recropping and no-till increase take-all. Wheat and barley, especially the former, together occupy about 90% of the acreage, so recropping led to intensive monoculture so far as take-all is concerned. Wheat is more susceptible than barley, but both are hosts. The worst situation is following spring wheat or barley with winter wheat, no-till. Stubbles and roots have little time to deteriorate between crops, and the pathogen is at full strength.

Kevin Moore (1984), a student of Cook's, found that inoculum potential was greatest in entire stubbles and least in finely fragmented stubbles. In conventional tillage, especially with summer fallow as in the past, the stubbles are finely fragmented and much is decomposed by the time the new crop is seeded. In no-till, with recropping, wheat is seeded directly into fresh unfragmented stubble.

Don Huber, an Idaho cereal pathologist, observed that take-all was most serious on irrigated wheat when fertilized with nitrate, less serious when nitrogen was in the ammonium form. Dick Smiley, a student of Cook's, proved that the rhizospheres of wheat roots receiving ammonium nitrogen were about one pH unit more acid than when nitrate was available. Smiley attributed the effect of form of nitrogen to changes in rhizosphere pH.

Development of glyphosate herbicide (Roundup) led some farmers to substitute chemical fallow for tillage fallow, killing cheat grass, weeds, and volunteer grain with the systemic herbicide. Death of the plants is slow, and *R. cerealis*, *R. oryzae*, and *G. graminis* increase rapidly on the debilitated roots prior to death. If the next crop is seeded soon after glyphosate application disease increases dramatically. We learn of unknown beneficial effects of "conventional" tillage as farming methods change.

#### *Bacterization*

The addition of beneficial bacteria to seed or propagation materials to reduce disease (bacterization) has recently received much emphasis, stimulated by the desire to reduce the use of chemical pesticides. The USDA Biological Control and Root Disease Laboratory at Pullman (R. J. Cook, Dave Weller and Linda Thomashow) has tested rhizosphere *Pseudomonas fluorescens* isolates for their ability to control take-all, *Pythium* spp., and *Rhizoctonia* spp. on wheat. They have patented the use of bacteria to control take-all and are cooperating with industry in attempts at commercial application.

To be effective the bacteria must colonize the roots as they emerge from the treated seed and develop on the new root system. Ideally they should increase and remain a part of the soil microflora, leading to residual and cumulative benefit. To date there is no evidence of residual effects, so the treatment would have to be repeated each time wheat is sown. Up to the present, the most effective bacteria give only partial control, but efforts to isolate more effective strains continue. The most effective bacteria for take-all control produce a phenazine antibiotic antagonistic to many soilborne pathogens.

*Rhizoctonia solani* and *R. oryzae*. Bare patch or purple patch. Bare patch of wheat and barley, an important disease in Australia (bare patch, purple patch) and South Africa (crater disease), was unknown in the United States until farmers in the PNW began to plant directly into stubble, no-till. James Cook and David Weller in Washington and Dick Smiley in Oregon have studied bare patch in this region (1984 and later). The amazing thing is that tillage a few inches below seed level controls it, explaining its absence as a disease before no-till. Conventional tillage controlled it. Albert Rovira of Australia believes the fungus makes a mycelial web within the soil and that tillage disrupts the web, destroying the ability of *R. solani* to mobilize sufficient energy to quickly and vigorously attack root tips. Tillage reduces inoculum potential by disrupting the mycelium in the soil.

Roots of seedlings are blighted not far from the seed, resulting in "spear tips", brownish abstricted rotted root ends. When enough roots are affected, seedlings are so stunted they never fully recover. The disease occurs in patches, giving it its common names, bare patch or crater. Wheat or barley is not killed but the patches are practically bare because the diseased plants remain very small. In 1987 Akira

Ogoshi identified the pathogens of the PNW as anastomosis group 8 of *R. solani* and as *R. oryzae*. Both are true root pathogens and Cook and Weller state that local isolates of *R. oryzae* are more virulent than those of *R. solani* AG-8. *R. solani* AG-8 and *R. oryzae* have wide host ranges.

Some years ago *R. cerealis*, treated under foot rots (sharp eye spot), was not differentiated from *R. solani*. *R. cerealis* has a *Ceratobasidium* teleomorph, *R. solani*, *Thanatephorus*. Vegetative hyphae of *R. cerealis* are binucleate, those of *R. solani* have multinucleate hyphal tips. Sprague considered the "*R. solani*" of sharp eye spot atypical, and he called a fungus isolated from cheatgrass along the Lewiston grade *R. oryzae* (in his book, p. 138). Sprague had remarkable acuity as a mycologist.

Pythium spp. on wheat. Roderick Sprague, during his brief stay at Pullman, isolated *P. debaryanum*, *P. "graminicola"*, *P. tardicrescens*, and *P. ultimum* from wheat and grasses (see his book, 1950). Sprague included *P. aristosporum* and *P. arrhenomanes* within his "*P. graminicola*," so they are not specifically reported. Sprague noted that few grass pathologists report *P. debaryanum*, even though Hesse (1874) listed proso millet and corn among its hosts. *P. ultimum* was abundant in specimens from cool, wet soils and Sprague believed it had some importance in the Palouse. He isolated *P. tardicrescens* only once.

Bruehl, who had worked on *P. arrhenomanes* on spring wheat and barley in South Dakota, found no root rot of comparable severity in Washington, and did not study *Pythium* root rots here.

An entire nursery on low, wet land was destroyed by *Pythium* snow rot in the winter of 1968-69 on the Spillman Farm, Pullman. Deep snow covered the plot for over three months. Orville Vogel dug through the snow in March and water was running beneath the snow in the drill rows. The wheat couldn't have been killed by cold and it was not killed by *M. nivale* or *Typhula* spp. The plants were dirty brown and rotted. The Japanese had described a snow rot of winter wheat grown on paddy rice fields that was caused by *Pythium* spp. Bruehl found oospores in profusion in the rotted crowns and leaves. He isolated *Pythium* spp., mainly *P. iwayamai*, but failed to complete Koch's postulates using snow mold techniques. The disease was common around Pullman and in the wheat breeding plots at Moscow, Idaho, especially in those of Warren Pope.

Nothing succeeded until a graduate student, Patrick Lipps, produced zoospores to inoculate wheat. It is probable that oospores in Bruehl's inoculum, with standard snow mold techniques, never germinated. Lipps made a classic study of the effect of water relations, water quality, and temperature on zoospore production. Some wheats resistant to snow mold are fully susceptible to snow rot, and early-seeded wheat is more susceptible than late-seeded wheat.

While working on snow rot of wheat, Pat Lipps isolated *P. ultimum* and *P. aristosporum* during winter (1978). Under snow rot conditions, *P. aristosporum* was moderately virulent, *P. ultimum* was avirulent. Lipps confirmed Japanese work listing *P. iwayamai* as a true snow rot fungus. He described *P. okanoganense* from snow rot wheat but failed to complete Koch's postulates with it. Within a few years Japanese isolated *P. okanoganense* and proved it virulent. The amazing thing about the snow rot *Pythium* spp. is that their zoospores are attracted to stomata and host penetration is largely indirect, unique so far as I know within the genus *Pythium*. Snow rot develops only with standing or running water under snow, limiting it to drainage ways or relatively small areas. Most of the soils in snow areas of Washington are well drained, making snow rot of minor importance state-wide. The topography of fields in south central Okanogan County, with many depressions, gives the disease some importance there.

Working with snow rot fungi is as much art as science. The oospores in diseased tissues quickly became dormant if the plants are warmed prior to isolation attempts, leading to use of an ice chest on collection trips. When Pat Lipps removed petri dishes containing zoospores in water from refrigerators, if the dishes were agitated during handling the zoospores encysted when in contact with the glass. So much for the skill of Pat Lipps.



About the time Jim Cook became interested in *Pythium* spp. on wheat. Cook, Sitton and Waldher (1980) reported a large yield response in direct-seeded wheat following soil fumigation and Cook and Haglund (1982) confirmed the yield response. These losses were mainly attributed to *P. ultimum* attacks on seeds and seedlings. C. Chamswarng (1983), while a student of Cook's, identified *P. aristosporum*, *P. heterothallicum*, *P. irregulare*, *P. torulosum*, *P. volutum*, *P. ultimum* and *P. ultimum* var. *sporangiferum* as parasites of wheat in cool soil.

Tom Hering (1987), a visiting scientist from the University of Nottingham, Nottingham, England (Robin Hood country) found *P. irregulare* and *P. ultimum* var. *sporangiferum* in the scutellum of many wheat seeds sown in very wet soil (-0.1 bar) in two days at 20°C. If the seeds were pregerminated 48 hours, the seed-invading *Pythium* spp. did no harm. Old seed blighted much more severely than new seed. This is important because seed dealers often increase a wheat and sell its seed over a period of several years.

If wheat is seeded in aerated, moist soil at moderate temperatures, stand establishment and seedling vigor problems are avoided. *Pythium* seedling problems arise when winter wheat is seeded very late in cold, wet soil. Ole Becker (1988), working with Cook, found several bacteria that protected seed from *Pythium*, partly by producing siderophores limiting the availability of iron.

Dryland Root Rot of Winter Wheat. The Canadians found *Bipolaris sorokiniana* and *Fusarium culmorum* so general in the prairie provinces that they described "common" root rot as a complex of the two pathogens. They gradually attributed most "common" root rot to *B. sorokiniana*. Sprague (1948, Bull. 192) included both pathogens under common root rot and then attributed "dryland" root rot primarily to *B. sorokiniana* and brown root rot primarily to *F. culmorum*. These pathogens had similar distributions but they caused diseases sufficiently different to lead Sprague to avoid treating them as a "complex".

In June, 1964, on his way to a Lind field day, Orville Vogel found a devastated field of Burt winter wheat about 10 miles north of the dryland station at Lind. Whole plants were stunted with white heads and a significant yield loss was obvious. Stew Holton and I diagnosed the problem as caused by *F. culmorum*. The field had been in oats in 1962. Nothing further was done until James Cook arrived in August of 1964. The 1966 wheat crop (Gaines, the next crop because of the summer fallow system, was severely diseased. The disease was essentially limited to the land in oats 4 years earlier. Cook and Jerry Sitton sampled the problem area repeatedly and the number of propagules per gram of soil, about 3000, was uniform over the entire area and the propagule count remained remarkably constant for some years. Cook called it *Fusarium* foot rot because, when severe in Washington, the stem is discolored 1-3 inches above the soil. The discolored area blends gradually into normal straw color, so the disease is both a foot rot and a root rot.

The disease was important on early-seeded, over-fertilized winter wheat, and oats increased the pathogen. Cook and R. I. (Bob) Papendick established the critical role of water stress in predisposing wheat to attack by *F. culmorum*. Germ tubes from chlamydospores in the soil survived well in relatively dry soil but were lysed by bacteria in wet soil (1970). Hyphal growth along wheat straws in soil occurred down to at least -50 bars water potential (1972). Water potential within wheat leaves was about -22, -30, and -38 bars in wheat near Harrington fertilized with 0, 60, and 120 pounds of nitrogen, per acre, respectively. By 1974 Cook and Papendick had determined that wheat infected with *F. culmorum* survived at -30 to -33 bars, but died at -35 to -40 bars. Healthy wheat survived the high water stress. Their experiments conclusively proved the role of water stress in dryland root rot caused by *F. culmorum*. Losses from dryland root rot (*Fusarium* root rot) declined greatly when farmers adjusted nitrogen application to available water, avoiding severe water stress.

The Cambridge System of estimating competitive saprophytic ability lists *F. culmorum* as a good saprophytic competitor. Work by Cook, Bruehl and Sitton did not support results of the Cambridge System. *F. culmorum* is widespread in Central Washington, yet it is not abundant in most fields in spite of

a constant supply of wheat and barley refuse. It attains high populations only after a strong parasitic phase.

*F. graminearum* is prevalent in warmer areas, in south central Washington. It causes similar symptoms. Cook found, as in Australia, that dryland foot rot isolates of *F. graminearum* have become truly asexual, having lost the ability to form perithecia.

### **Snow Mold**

Knowledge of the snow mold fungi of the Pacific Northwest developed slowly. C. W. Hungerford (1923) reported a serious occurrence of *Sclerotium rhizodes* (= *Typhula idahoensis*) in southern Idaho and Heald reported it from near Chelan, WA in 1924. P. A. Young reported *Typhula graminum* (= *T. incarnata*) in Montana in 1929. Earle Blodgett reported *Fusarium nivale* (= *Microdochium nivale*) in Idaho in 1946. Sprague, Bill Fischer and Peggybeth Figaro reported *Sclerotinia borealis* in the Molson-Chesaw region of northern Okanogan County near the Canadian border in 1961. In 1975 *Typhula ishikariensis* was added to the complex (Bruehl, Rollin Machtmes and Dick Kiyomoto). In Washington *Typhula* spp. and *M. nivale* are the major pathogens. *Sclerotinia borealis* develops only on frozen ground and to date it has not been found outside the original discovery site near the Canadian border at about 4000 ft elevation. The limited distribution of *S. borealis* is very important because winter wheats resistant to *Typhula* spp. and *M. nivale* are not resistant to *S. borealis*.

The *Typhula* spp. increased slowly, becoming serious in parts of Douglas and Okanogan Counties in the 1940s. Roderick Sprague, while still at Pullman, became involved. The fall of 1947 was wetter than normal, snow fell early on unfrozen ground, and 30-40,000 acres were lost in Douglas County in 1948. The fall and winter of 1948-49 were also favorable and 20-30,000 acres were lost, requiring reseeding with spring wheat. C. S. Holton and Sprague collaborated in these years. Fungicides applied in the fall controlled the disease but they were too costly. Holton confirmed farm observations that wheat seeded in August survived better than wheat seeded in September. No local wheats had any real resistance. Sprague moved to Wenatchee as tree fruit pathologist in 1950, and from there he worked intensively trying to find economic fungicides effective against both *M. nivale* and *Typhula* spp. Organic mercuries were the best (thank goodness they were not used by farmers).

Sprague read of resistant wheats in Japan, and in the fall of 1960 he planted 5200 wheats from the world collection west of Mansfield in Douglas County. In the spring of 1961 he reported 24 with moderate resistance (none of which was from Japan). In the fall of 1961 he planted the best of the 1960-61 wheats and 4200 more. The winter favored mold and selection was severe. Sprague died without seeing the results of his last nursery and Bruehl inherited the project. Several wheats survived the severe test.

Walter Nelson, superintendent of the Dryland Research Unit at Lind, and Bruehl dug up survivors, transplanted them at Lind, and Dick Nagamitsu crossed them with Itana, Gaines, and Sel. 101. Subsequent testing identified the most resistant wheats and a selection of the cross, PI 181268/Gaines, was released to farmers in 1972 as 'Sprague', the first snow mold resistant wheat in the region. It is still (1989) the leading wheat where snow molds are serious. A second soft white common wheat, 'John', was released in 1986 and a hard red, 'Andrews', was released in 1987. Sprague was named in honor of Roderick Sprague, who planted the nurseries in which real resistance was found but didn't live to see real resistance. John was named to honor two Washington State Wheat Commission members who supported snow mold research, John Goldmark and John Thompsen. Andrews was named for a village (St. Andrews) in the hard red wheat area of Douglas County. The combination of resistance and seeding August 20-26 has controlled snow mold at no cost to growers. Clarence Peterson (1990) will release a soft white wheat, (P.I. 167822 x Sel. 101) x Luke, with good resistance to snow mold, high yield and good straw, the first mold resistant wheat with adequate straw strength.

Breeding for mold resistance in Scandinavia and Japan has been less successful. In those regions *Sclerotinia borealis* is important. Resistance to *Typhula* spp. and *M. nivale* is correlated; resistance to *S. borealis* is different, and no wheat has yet been developed that is resistant to all three pathogens.

Winter barley is far more susceptible than wheat to *Typhula* and to *M. nivale*, and efforts to develop resistance in barley, pursued vigorously by a graduate student, Douglas Gertenbach, 1978-1980, have been abandoned. Of over 1200 winter barleys tested near Harrington on the Bob Kramer ranch, Kamiak, a local winter barley, was about as good as any. Local breeders may have selected for resistance to *T. incarnata* and *M. nivale* in their routine winter survival trials. Winter barley cannot be grown where *Typhula* and *M. nivale* are serious.

Bill Fischer, son of George W. Fischer, was the technical aide on this project, about 1956 to 1964. He worked intensively to control mold by spreading blackeners on the snow to hasten melting and shorten the incubation period. Results were considered uneconomical in Washington. Snow-blackeners are used to some extent in northern Utah and southern Idaho.

Bruehl, to the irritation of Gardner Shaw, thought the sexual state unimportant, but changed his mind. Barry Cunfer (1973) proved that infection developed from basidiospores of *T. idahoensis*. Dick Kiyomoto selfed *T. idahoensis* and found great variations in pathogenicity among offspring, from avirulent to more virulent than the parent (1976). Alice Christen (1979) crossed *T. ishikariensis* and *T. idahoensis*, and found few hybrids that were sexually competent but some could survive asexually in nature and introgression of one species into the other is possible. Basidiospores are very important when sclerotial populations in the soil are low. They spread the fungi, giving them a chance to increase greatly. When sclerotial inoculum is abundant basidiospores are inconsequential because mycelium from sclerotia develops quicker and with greater vigor than from basidiospores, rendering the latter noncompetitive.

I regret not determining the longevity in soil of sclerotia of the three pathogenic *Typhula* spp., but field observations indicate they survive several years. Roy Davidson (1972) found that sclerotia of *T. idahoensis* within the top 1 cm of soil caused typical snow mold; sclerotia buried 2 cm were ineffective as inoculum. Darrel Jacobs (1986) screened sclerotia from field soils from Idaho and Washington and found natural infestations as high as 670 sclerotia per kilogram of soil. Average infestations were much lower. About half the sclerotia recovered from soil germinated. *T. incarnata* is both a true snow mold, attacking leaves, and it can attack roots and crowns below the soil surface in the absence of foliage attack.

Russian and Japanese scientists proved that wheat didn't suffocate under deep snow mold: it became weakened by depletion of carbohydrates, rendering it susceptible to *Typhula* and *Microdochium*. Cunfer (1971) and especially Kiyomoto (1977), confirmed and expanded our understanding of this relationship. Resistant wheats accumulated more carbohydrates before winter and they tended to utilize them more sparingly in the dark than susceptible wheats.

Most workers claim that *T. incarnata* has considerable saprophytic ability, that this contributes to its wide distribution. In my opinion the saprophytic abilities of the three *Typhula* spp. are minimal, to such an extent that I believe they could not survive without parasitism. Jacobs (1986) found that hyphae of *T. incarnata* obtained some nutrition from natural soil, that hyphae cut free of sclerotia made limited further growth on the surface of Ritzville silt loam but not on Palouse silt loam. Wheat is harvested in August, and vegetative parts are dead even before harvest. *Typhula* sclerotia do not become active until cool, damp conditions exist at the soil surface, usually about mid-October. The straw and stubble by that time is not "virgin", uncolonized substrate. I have found a few sclerotia that formed on refuse saprophytically. The virulent *Typhula* spp. do not compete well with true saprophytes already established in the refuse. After a good snow mold winter, wheat leaves are covered with sclerotia; refuse in adjacent fallow fields is not. *Typhula incarnata* starts on senescent leaves of live plants, but this is not true saprophytism. In my opinion laboratory experiments purporting to prove the saprophytic power of these fungi mostly confuse rather than elucidate reality in nature.

I have marvelled at the accomplishments of Roderick Sprague, and one might wonder how such a talented mycologist missed the sexual state of *Microdochium nivale* (= *Fusarium nivale*). Snow mold was devastating when he worked on it and control dominated his efforts. Perithecia form on leaf sheaths above the soil late in development of the host. After severe snow mold all growth of above ground vegetative structures occurs in the spring. All pre-winter leaf tissue is rotted. Perithecia of *Monographella nivalis* are rare in wheat in the snow mold region (Cook and Bruehl, 1968). They are common in leaf sheaths at or after host maturity in dense vigorous stands of wheat not damaged by snow mold. The possible role of ascospores in the life cycle in Eastern Washington is unknown, but I suspect it minor at best.

### ***Cephalosporium* Stripe**

*Cephalosporium* stripe (*Cephalosporium gramineum*) was described in Japan by Nisikado, Matsumoto and Yamauti in 1934 but it was not found in Washington until 1955. When first observed it was an oddity, with a few diseased plants here and there, usually in low spots or drainage ways in fields of winter wheat. In 1956 it was widespread on Elmar, Brevor and Golden (Bruehl, 1957). It is rarely seed-borne in wheat, but it is commonly present in seed of winter barley. The fungus is systemic and it invades the lemmas and paleas of barley kernels. It was found on several grasses. The sporodochial stage (*Hymenula cerealis*) was described by Ellis and Everhart from wheat straw collected in 1894 in West Virginia, so the fungus was present in the United States for many years prior to 1955.

The smut resistant variety Brevor dominated the high rainfall areas of eastern Washington, 1952-1962, and this variety is highly susceptible. In retrospect the fungus had probably been present for years without causing much damage, but repeated growth of Brevor led to its increase. Brevor, Elmar and Golden were replaced by Gaines in 1963. Gaines gave way to Nugaines in 1966. Gaines and Nugaines have moderate resistance. The disease subsided. When highly susceptible Hyslop, McDermid, and Stephens became important, starting in 1976, the disease again increased (Bruehl, Murray, and Allan, 1986). Varietal susceptibility plays a major role in epidemiology and breeders in the region now include *cephalosporium* stripe in the selection process. High susceptibility is eliminated.

The disease devastated wheat near Rockford, and a farmer (Bill Bell) and I teamed up. I surveyed all the winter wheat fields for disease severity. He determined the cropping history of the fields. In every severe case (up to 100% diseased plants), rotation was absent. In all lightly diseased fields, the farmer followed a 3-year rotation, with winter wheat being grown every third year (winter wheat, spring barley or peas, summer fallow, winter wheat). Rotation followed year-after-year is the best control. A survey of this type is cheap research. The results are still true.

Fertilization with ammonia-based materials increased when semi-dwarf wheats with the ability to remain erect under high rates of fertilization replaced standard height wheats. Mahler, Halvorson, and Koehler studied the trends of soil pH in Idaho and Washington and reported a general decline in soil pH in high moisture areas. They attributed the acidity to the effects of the dominant sources of nitrogen. Connie Love (1987) found that low soil pH favored *cephalosporium* stripe, as reported by Bockus and Claasen in Kansas (1985). In acid soils the level of resistance in cultivars should be higher than was formerly necessary. Murray (1988) found that the pathogen produced more dry weight in culture at low pH values. Larry Specht, a post-doctorate, worked with Tim Murray to further evaluate the effects of pH on the pathogen and on disease incidence. The antibiotics produced by *C. gramineum*, according to preliminary results (Bruehl, Lai, and Huisman, 1964), are ineffective at pHs above 6.0, a factor which could affect saprophytic survival when straw is within the soil. This relationship needs further study.

To date no chemical has been found, either a seed treatment or foliar spray, that controls *Cephalosporium* stripe.

When first studied, *Cephalosporium* stripe was found primarily around the periphery of the wheat region at higher elevations. It is now more widespread. Early seeding increases the disease. Infested straw on the soil surface favors disease more than buried straw, so minimum tillage tends to increase inoculum levels. The most severely damaged fields I ever saw, however, were all in summer-fallow-wheat-summer-fallow "rotations", with conventional tillage. Wheat is seeded earlier and develops more vigorously on summer fallow than in recropping or no-till situations, resulting in larger root systems before winter, exposing more host tissue to infection.

### ***Diseases of the Grain***

Scab. Wheat of Central and Eastern Washington has historically been free of fusarium head blight (scab), caused primarily by *Fusarium graminearum*, because of dry weather during late stages of wheat development. Moisture in the head is required for spore germination and infection. In recent years scab developed in fields irrigated with center pivot systems. Carl Strausbaugh and Otis Maloy sampled wheat in 132 irrigated and 35 nonirrigated fields in central Washington in 1983 and 1984. Scab was common near the center of the fields with center pivot systems. Wheat is sprinkled for long periods near the center and increasingly shorter periods toward the periphery of the circle. With wheel line irrigation the wet front advances with the wheel line and dries after passage of the system. In rill irrigation (small ditches) water does not reach the heads; only the humidity within the field is raised. Scab averaged 6.3% with center pivot, 0.8% with wheel line, and 0.4% with rill irrigation, and 0 without irrigation. Scab was greatest where water on the head was greatest. Strausbaugh and Maloy gave the relative prevalence of the scab fungi as *F. graminearum* 391, *F. nivale* 54, *F. culmorum* 49, and *F. avenaceum* 1.

In one center pivot field with cornstalks on the soil surface 89% of the heads contained scabby kernels. Experience in the corn belt long ago proved that corn is the most dangerous crop preceding wheat or barley if the corn stalks are not cleanly plowed under. The sexual state of *F. graminearum*, *Gibberella zeae*, forms in profusion on corn stalks, providing air-borne ascospores. This "cornbelt" research, demonstrating the effect of corn stalks on the soil surface on the incidence of scab, is another example of the effect of changes in farm practices (center pivot irrigation) upon plant diseases.

No wheat in Washington has yet graded scabby, inspite of these central pivot irrigation systems. Finding 89% of heads with scab does not mean 89% of the kernels were scabby. Many kernels on the head escape. Also, grain is bulked within market channels, so the scab of a few fields is diluted below detection. Nevertheless, farmers with central pivot systems should plow under corn stalks or not follow corn with wheat in the rotation. Freedom from scab is a valuable attribute of our wheat (and barley). Scabby grain causes vomiting and other disorders in man, swine and some other animals.

Fungi and insects in stored wheat. Certain xerophytic fungi digest wheat in storage, but in very dry grain the fungi do not develop. Some insects that invade wheat and flour obtain metabolic water during digestion of carbohydrates in the grain and they elevate the humidity to a point at which fungi can grow. Together the fungi and insects raise the temperature and moisture within the grain, forming hot-spots. Insects alone, fungi alone, and both in combination reduce the quality and salability of wheat. Because some *Aspergillus* spp. produce mycotoxins the Hay and Grain Fund of the Washington State Department of Agriculture supported the study of insects and fungi in grain by P. P. Sikorowski (PhD, 1964).

One of the fungi, *Aspergillus versicolor*, was food for the red flour beetle, flat grain beetle, and somewhat for the saw-tooth grain beetle. In contrast, *Aspergillus repens* was detrimental to all three insects, so generalizations about the interrelation of insects and fungi are not safe. *Aspergillus candidus* was a good source of food for the red flour beetle and the flat grain beetle, but not the saw-toothed grain beetle. Beetles given free choice of wheat or the fungi, preferred *A. versicolor* to the other fungi and they chose that fungus about as often as they chose wheat. Within grain beetles moved to places where *A. versicolor* was growing, and live spores were present in their feces, so they can carry the fungus with them, either internally or externally.

The flat grain beetle was strongly attracted to hot spots, the red flour beetle and saw-toothed grain beetle were less strongly attracted, but all three grain beetles moved toward hot spots from surrounding grain. Grain in the hot spots is cemented together by hyphae of *Penicillium* spp., *Aspergillus flavus*, and to a lesser degree, *A. versicolor*. Only the flat grain beetles penetrated into the core of the hot spot. The other insects accumulated around its periphery. The fungi produced enzymes that partially digest the wheat kernel, improving it as food for the insects. Starting with grain too dry for molds to grow; insects produce metabolic water, elevate humidity; molds start to grow, raising the temperatures and humidity; and both insects and fungi are favored.

### **Virus Diseases**

Wheat Streak Mosaic. Virus diseases of cereals received no attention in Washington until H. H. McKinney and Jack Meiners found wheat streak mosaic in experimental plots at Pullman (1954). Because it was found on the station, the alarming possibility was that the virus had somehow been introduced by experiment station personnel. Wheat mosaic is a major disease in the Great Plains, extending all the way to Alberta, Canada. Bruehl, who replaced Meiners in 1954, and after observing wheat streak mosaic, 1955-1957, concluded that under normal farming practices it posed no threat and that it was not a recent introduction. Severe WSM is limited to very early-seeded winter wheat and possibly very late-seeded spring wheat. Normal seeding dates control it completely. WSM is common in very early-seeded winter wheat in Douglas County where wheat is seeded early because of snow mold. It has not been a serious problem in wheat seeded August 20-26, early enough to develop resistance to snow mold. It destroyed some unusually early-seeded wheat in the Arbon Valley of Southern Idaho, a severe snow mold region.

The vector, *Aceria tulipae* (= *Eriophyes tulipae*), like aphids, depends upon green leaves for survival. It has no stage, including the eggs, that resists desiccation. Normal dry summers reduce the mite to ineffective numbers in eastern Washington, and my suspicion is that mites are not disseminated during cool weather. Dry, dead foliage in the summer is a natural, free control. I have not seen wheat streak mosaic in Western Washington and the mite may be subject to fungal attack during prolonged periods of sustained high humidity, or no winter wheat was seeded early enough to be infected.

Art Finley (1957) reported serious losses from wheat streak mosaic in sweet corn in southern Idaho. To my knowledge no such losses have occurred in Washington.

Clarence Peterson, who replaced Orville Vogel as the soft white wheat breeder at Pullman, completed a MS thesis on WSM and barley stripe mosaic at the University of Idaho (1959). He studied grass hosts of WSMV and of *Aceria tulipae*. The list of grasses susceptible to the mite and the virus is sufficiently long to explain the ever-present threat of WSM locally if wheat is planted so that green plants go through the warm months (July, August). A former county agent of Whitman County, Felix Entemann, did a date of seeding trial, and his results were typical of those of Slykhuis, Andrews, and Pittman in Alberta (1951). Seedings in July = 100% diseased, decreasing as the date became later, to zero at normal seeding dates.

I see no need for research on WSM in Washington, unless to determine the natural reservoirs of the virus.

I had the privilege of "escorting" H. H. McKinney around Washington looking for virus diseases in cereals. When away from Pullman I always worked long hours. McKinney refused to do this. He wanted to work only with good light: no early or late day searching when subtle symptoms could not be seen.

Rollie Line said, include the time wheat streak mosaic destroyed our wheat in the Plant Pathology plots at Pullman. Several years ago the Animal Science Department planted winter wheat in the spring as ground

cover and horse pasture adjacent to our experiments. The wheat became 100% infected, and the mites brought the mosaic virus to 100% of our wheat. We were sad and angry, but then hail fell on the same wheat, so we lost our experiments twice in one year.

Barley stripe mosaic caused by seed-borne virus, has at times been wide spread in barley in agronomy plots on Spillman Farm, but to my knowledge it has not been an economic problem on the farm, and WSU scientists have not studied it.

Barley Yellow Dwarf. In 1951 Oswald and Houston described a "new" disease at Davis, California. After a mild winter and a wet spring that delayed planting, scattered yellow spring barley plants occurred among healthy plants. Aphids were abundant and Oswald and Houston quickly proved the aphids were vectors of a virus that caused stunting and discoloration, not only of barley, but of oats and wheat as well. Most commercial barleys turned yellow, oats red or purplish, and wheat yellow, red or purple, depending on the variety. Pathologists around the world quickly realized they had seen this disease before. How could an important disease of wheat, barley and oats go unrecognized as such, until 1951, when the hosts are among the most widely studied plants in the world?

In the 1930s, Roderick Sprague, while at Corvallis, Oregon, wrote papers on red leaf of oats. He attributed most of the red leaf to environmental conditions--wet, cold, acid soil in particular. Sprague didn't have a chance. If 100% of the plants are discolored, it is logical to suspect soil and weather. At Davis, diseased and healthy plants were intermixed, ruling out soil and weather as direct causes, leading to the search for an infectious agent. Discovery in this case was favored by less rather than by more disease.

The conditions observed by Sprague in Western Oregon were common in Western Washington as well. Bruehl and Damsteegt established plots at Vancouver, of both winter and spring grains, and 100% infection was commonly achieved. Complete fertilizer (NPK) was added to reduce danger of confusing nutrient deficiency symptoms with those of yellow dwarf.

Toko and Bruehl (1957, 1959) found vector-specific isolates of BYDV, one transmitted by the English grain aphid and not by the bird-cherry-oat aphid, and one transmitted by the bird-cherry oat aphid and not by the English grain aphid. Rochow in New York quickly followed this report. In his early efforts Rochow found mostly vector-specific isolates. In Washington wheat, oats and barley vector-specific isolates were exceedingly rare (limited to the two above). A controversy with Rochow ensued. He won. Most virologists have concentrated on vector-specificity. It is still a mystery to me how vector-specific isolates can compete with isolates transmitted by three or more aphid species (nonspecific isolates).

Vernon Damsteegt, a graduate student, lived in summer in an apartment on the second floor of the main building at the Vancouver research station. During this period and later, we tested 3600 spring oats, several hundred winter oats and spring barleys, all the winter barleys, and many wheats for resistance to BYD, judged visually in preliminary trials, progressing to yield trials.

Among the spring oats, one of several Craig x Alamo lines from New York and Minnesota II-22-220 (C.I. 2874) were selected as outstanding. The Craig x Alamo selection, after extensive tests with Herman Austenson, agronomist at Puyallup, was released by Cal Konzak as Cayuse. The Minnesota oat was used in crosses with Cayuse by Konzak, WSU spring oat and spring wheat breeder.

Efforts to screen winter barleys at Vancouver for BYD resistance were abandoned. 1230 winter barleys were seeded September 15, 1959, with every eleventh row being local White Winter as a check. Scald (*Rhynchosporium*) was so severe BYD was incidental and obscured. To my amazement, every eleventh row, the local White Winter check, was resistant to scald, an example of the value of local "adaptation".

The winter wheat Sun, a wheat adapted to Western Washington, was used as the check for winter wheat trials. Of 3842 winter wheats few did better than Sun. According to J. Allen Clark, John Martin, and C. R. Ball, *Classification of American Wheat Varieties, 1922*, Sun (= Sol) originated at Svalof, Sweden. It was distributed as Sun by C. H. Lilly and Co., Seattle, WA. Sun was grown in Pierce and San Juan Counties, according to this bulletin. I remember some one (Herman Austenson?) telling me that Sun for many years held the Washington yield record of over 100 bu/acre in a field on Whidbey Island. Sun is no longer grown, but it may have value to breeders.

Charles Schaller at Davis, California, discovered several Ethiopian barleys with good resistance to yellow dwarf, and he and associates described the  $Yd_2$  gene for resistance. We found several other resistant barleys, also from Ethiopia. We thought we had several genes for resistance. Vernon Damsteegt (1962 PhD thesis) found, however, that all the barley accessions had the same gene for resistance. Fortunately the  $Yd_2$  gene has remained effective all over the world.

Walter Nelson, superintendent of the Dryland Research Unit at Lind, called my attention in the 1960s to fields of winter wheat heavily colonized by the bird cherry-oat aphid in the fall. Every plant had many aphids. I eagerly awaited to see the BYD epidemic, only to find the wheat healthy. That so many aphids could be free of BYDV was hard to believe. Sometime in the 1970's, BYD began to be common in early-seeded winter wheat in the dryland region, suggesting the possibility of a change in the virus. Judith Brown (1981) found that much of the corn grown in the Columbia Basin acted as a summer reservoir of BYDV. She and Wyatt found that the English grain aphid brought virus to the corn and that the bird cherry-oat aphid colonized the corn later, bringing BYDV to winter cereals as the corn matured. Donna Hazelwood (1983) found that BYDV from north of the Columbia Basin, out of the migration path of aphids from corn to winter wheat, did not infect corn. It appears that in the early years of the Columbia Basin irrigation project BYDV was not adapted to corn but in time strains that multiplied in corn were selected, making corn a widely grown summer host of BYDV. BYD is common in dryland wheat now, depending on severity of the winter, a marked change from earlier times.

Bruehl quit working on BYD because, except for occasional destructive epidemics on spring grain, it was not important enough to warrant breeding for resistance in winter wheat. The adaptation of the virus to corn has resulted in Wyatt and R. A. Allen, wheat geneticist, USDA at Pullman, working to develop resistant winter wheats.

Steve Wyatt is determining the genetic code of a local vector nonspecific strain of BYDV. Laura Seybert (1977-1978) found vector nonspecific virus predominant in Eastern Washington on cereals. Mark Kainz, working in root chambers with Walt Hendrix, demonstrated the severe stunting of roots caused by BYD.

Soilborne Oat Mosaic. A soilborne mosaic of winter oats with the spindle-shaped lesions similar to a type common in southeastern USA was found at Vancouver and in the Willamette Valley of Oregon. Winter oats are of no importance in Washington and this disease was not studied.

### ***Tribute to Orville Vogel***

Tribute to Orville Vogel, USDA wheat breeder at Pullman. This unselfish person supported the search for snow mold resistance and foot rot resistance by furnishing his machinery and crew to plant large nurseries for years. He used his combine to cut the early trials with fungicides to control foot rot. Vogel was an experienced, astute observer, and he brought departures from normal to the attention of plant pathologists. He supported their efforts in every way he could. I regret not giving Vogel more credit for his help through the years.



*Dedication*

I dedicate this book to Director Ira Cardiff and President Compton, two forceful leaders who tried to help agriculture at WSU and were fired by the regents.