USE OF ALIEN GENES FOR THE DEVELOPMENT OF DISEASE RESISTANCE IN WHEAT

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ABSTRACT

The genus Triticum contains three ploidy levels and about 30 species. Most of these species have been investigated as sources of disease-resistance genes and several have been used in successful transfers of resistance to domestic wheat (T. aestivum, genomes AABBDD). In addition, at least six genera from the tribe Triticeae have been used successfully as donors of disease-resistance genes for domestic wheat. The amount of alien chromatin involved in these transfers varies from a single gene to chromosome arms or entire chromosomes. No attempt was made in this review to describe all alien resistance gene transfers in wheat or to outline the various techniques involved. Alien disease resistance genes covered in detail are those that confer resistance to barley yellow dwarf virus, wheat streak mosaic virus, Cephalosporium stripe (caused by Cephalosporium gramineum) and eyespot (caused by Pseudocercosporella herpotrichoides).

INTRODUCTION

No other cultivated plant is equal to wheat (Triticum spp.) in the breadth of our knowledge of its genomic structures and relationships, availability of wild

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germplasm, and global importance. The genus *Triticum* contains three ploidy levels and about 30 species (24). Most of these species have been investigated as sources of disease-resistance genes, and several have been used in successful transfers of resistance to domestic wheat (24, 35, 40, 58). In addition, at least six genera from the tribe Triticeae have been used successfully as donors of disease-resistance genes for domestic wheat (24, 35, 58). The amount of alien chromatin involved in these transfers varies from a single gene (3) to chromosome arms or entire chromosomes (27). Ignoring selection, the main factor determining the amount of alien chromatin carried through generations of recombination is chromosome pairing. Recombination frequencies involving homologous chromosomes such as from *T. tauschii* (genome DD) and D genome chromosomes of wheat (*T. aestivum*, genomes AABBDD) can be very close to those observed for wheat-chromosome to wheat-chromosome exchanges. Jones et al (36, 37) recombined two different 1D chromosomes from *T. tauschii* with the 1D chromosome of wheat cultivar Chinese Spring to map the alien leaf rust (caused by *Puccinia recondita*) resistance gene *Lr21*, and stem rust (caused by *P. graminis* f. sp. *tritici*) resistance gene *Sr33* relative to other markers. They found that the recombination frequencies among previously mapped markers in these crosses were consistent with those from other wheat-by-wheat crosses. Conversely, transfers between chromosomes that do not normally pair are much more complex and usually result in translocations and a large amount of alien chromatin introgressed into the wheat genomes. The use of induced homoeologous pairing, spontaneous translocations, and irradiation to achieve gene transfer has been described by several authors (listed in Reference 35).

Recent reviews have addressed the transfer of disease-resistance genes from wild wheats to domestic wheat (24, 35, 40, 41, 58). Similarly, most of these reviews outline the techniques and complexities involved in alien transfers. Therefore, no attempt was made in this review to describe all alien resistance-gene transfers in wheat or to outline the various techniques involved. Rather, we sought to describe economically important examples of disease resistance that cover a range of diseases for which there is little or no genetic variation in domestic wheat.

**THE NEED FOR AND VALUE OF ALIEN SOURCES OF DISEASE RESISTANCE**

The power of alien sources of resistance is not only to expand existing genetic variation in the crop but, more importantly, to introduce truly novel variation. Although adult plant resistance (race-nonspecific) genes have been described in alien sources (25, 40), the majority have been race-specific genes for foliar fungal pathogens, especially the obligately parasitic rust fungi (58). The greater
need for alien disease-resistance genes, though, is against pathogens for which there is limited or no genetic variation in domestic wheat. Examples of the latter include viruses and the facultative saprophytic and facultative parasitic fungal pathogens of the foliage and roots. Indeed, this review addresses two virus-caused diseases, barley yellow dwarf and wheat streak mosaic, and two fungal diseases, eyespot and *Cephalosporium* stripe, each of which is caused by a facultative saprophyte. Each of these diseases is economically important and widespread in various wheat-growing areas of the world. Other diseases that are not discussed, but for which resistance is inadequate or does not exist in wheat include: scab (caused by *Fusarium* sp.), Take-all (caused by *Gaeumannomyces graminis var. tritici*), *Septoria/Stagonospora* blotch (caused by *Septoria tritici* and *Stagonospora nodorum*), and sharp eyespot (caused by *Rhizoctonia cerealis*).

**Barley Yellow Dwarf**

Barley yellow dwarf disease (BYDV) was first described by Oswald & Huston (68) in 1951. The disease is caused by any one of several closely related luteoviruses (48) that are transmitted by several aphid species (11, 29). It is currently the most economically important virus disease of small grains worldwide (14). Bruehl (11) lists 97 grasses that can serve as a host for BYDV. Only low levels of resistance or tolerance have been reported for barley yellow dwarf in wheat (15, 70). Bruehl & Toko (13) were the first to show that *Agropyron* spp. (syn. *Lophopyrum* and *Thinopyrum*) were highly resistant to BYDV and, noting a lack of resistance in wheat, listed several wheat relatives in addition to *Agropyron* spp. that had immune reactions to the virus. Although single genes for resistance to BYDV have not been described in wheat genetically, chromosome and genome manipulations have provided valuable insight into the number and location of the genes controlling resistance. Transfers of genes involving *Agropyron* chromosomes are complex because they do not pair well, or at all, with their wheat homoeologues.

Surveys of *Agropyron* spp. and amphiploids derived from common wheat × *Agropyron* spp. have shown high levels of resistance to BYDV (30, 57, 75, 76, 91). Using field disease reactions McGuire et al (57) found *Lophopyrum elongatum* (Host) A. Love (2n=2X=14) and *L. ponticum* (Podp.) A. Love (2n=10X=70) were both highly resistant. In the same study McGuire et al (57) used a Chinese Spring wheat/L. elongatum amphiploid (genomes AABBDDEEE) and disomic substitution lines (L. elongatum chromosomes substituted for wheat homoeologues) to determine the chromosomal location of the resistance genes. They found that although all of the alien chromosomes had an effect on resistance, major genes for BYDV resistance were located on chromosomes 2E and 5E.

Zhong et al (95) characterized a *L. ponticum* × common wheat backcross
line, SW35, highly resistant to BYDV under field conditions. Based on in situ hybridization, meiotic chromosome pairing, and isozyme analysis, this 46-chromosome line had chromosomes 3E and 6E in addition, and 5E was substituted for wheat chromosome 5D. Line SW35 is very wheat-like and will be important in the eventual gene transfer because of its low number of chromosomes. Manipulations with this line to reduce the chromosome number even further are under way (G. Zhong, personal communication).

Xin et al (91) characterized Zhong 4, a wheat × Thinopyrum intermedium (syn. A. intermedium) amphiploid (2n=8X=56), which was shown by Zhang et al (94) to be highly resistant to BYDV under field conditions. Based on enzyme-linked immunosorbent assay (ELISA) readings, F1 plants with 49 chromosomes derived from Zhong 4 × several common wheats were shown to have disease reactions intermediate to the resistant and susceptible parents: none of the F1 plants were as resistant as Zhong 4. Although the authors were not able to reconstitute the resistance of the amphiploid in lines with fewer chromosomes, they were confident that the lines will have value in future transfers of resistance. Banks et al (8) are using this material in further attempts to reduce the alien chromatin while retaining the resistance.

Both cDNA dot blotting and ELISA were used by Goulart et al (30) to study the BYDV resistance in BC2 lines derived from common wheat × L. ponticum. The backcross lines were segregating for resistance and chromosome number, but the number of chromosomes for individual lines was not reported. Chromosome numbers ranged from 44 to 55, and they reported that the lines with higher chromosome number were, in general, more resistant, including several lines that were as resistant as the L. ponticum parent. Although field testing was not performed on these lines, the results agree with those of McGuire et al (57) and suggest that inheritance of resistance to BYDV is complex and controlled by many genes.

Wheat Streak Mosaic

Wheat streak mosaic (WSMV) is a disease primarily of winter wheat but which can also cause damage to spring wheat (7). The vector for WSMV is the wheat curl mite (Eriophyes tulipae Keifer, syn. Aceria tulipae) (6). Although slight levels of tolerance have been observed to WSMV, there is no resistance in common wheat (26, 53). As with BYDV resistance, Agropyron spp. exhibit very high levels of resistance to WSMV (47, 59, 73, 75, 79, 87, 88). Strategies for developing resistant cultivars include transfer of genes for resistance to the virus, the vector, or both.

Jiang et al (34) characterized wheat lines CI 15321 and CI 15322 (74), which are highly resistant to both WSMV and the wheat curl mite (53). These lines were derived from a progeny with high chromosome number from common wheat × L. ponticum that was resistant to WSMV (73). Using wheat lines that
were either chromosome substitutions or translocations, Jiang et al (34) determined that chromosome 1E confers high resistance to WSMV in these lines.

Chromosome 4 from *Th. intermedium* has also been found to confer resistance to WSMV in a wheat background (85). Fribe et al (27) designated this chromosome 4Ai-2 and, using translocated chromosomes, determined that the short arm conferred resistance. They identified line CI 17884 as a 4DL/4AiS substitution for chromosome 4D and line CI 17766 as the same translocated chromosome substituted for chromosome 4A.

Successful resistance to the mite vector is also an effective method of controlling disease. Resistance to the wheat curl mite was derived from lines immune to WSMV that were triple substitutions of *L. ponticum* chromosomes for wheat homoeologs (43). Larson & Atkinson (44) identified chromosome 6Ag as controlling resistance to the mite in a wheat background. Working with different material, Whelan & Hart (89) reported a wheat/*L. ponticum* translocation involving chromosome 6Ag that also confers resistance to establishment of the mite.

Although there appear to be several diverse sources of resistance available to WSMV, there are no commercial cultivars carrying any of these genes. Fribe et al (27) point out that all of the genetic stocks carrying resistance have undesirable agronomic characteristics associated with resistance.

**Cephalosporium Stripe**

*Cephalosporium* stripe, caused by *Cephalosporium gramineum* Nis. & Ika. (syn. *Hymenula cerealis* Ell. & Ev.), is a vascular wilt disease of wheat and other small grains and grasses. The disease was first described from Japan in 1933 (65, 66) and later in North America (10) and the United Kingdom (31). Early workers recognized that the pathogen had a wide host range among the small grains and grasses (10, 66). Nisikado et al (66) found *Cephalosporium* stripe on wheat, barley (*Hordeum vulgare* L.), *Avena fatua* L. and later (65) on *Alopecurus agrestis* L. Bruehl (10) found the disease occurring naturally on wheat, rye (*Secale cereale* L.), *Agropyron repens* (L.) Beauv., *Bromus marginatus* Nees., *Dactylis glomerata* L., and *Elymus glaucus* Buckl. Bruehl (10) also demonstrated that 11 other genera of grasses were hosts when greenhouse-grown plants were inoculated with conidia of the pathogen.

Nisikado et al (66) were the first to suggest control of *Cephalosporium* stripe by selection and breeding of resistant cultivars. Several different groups have screened wheat for resistance to *Cephalosporium* stripe (10, 12, 51, 54, 56, 66, 93), and most have arrived at the conclusions that immunity to *C. gramineum* does not exist in wheat, and wheat cultivars vary in the amount of disease and yield loss they sustain, with most considered moderately to highly susceptible. In 1985, however, Mathre et al (55) reported that *A. elongatum*, *A. intermedium*, and *Agrotriticum* #3525 (2n=56, AABBDDDee) were all highly
resistant to *C. gramineum* in growth chamber and field screening studies. Other wheat relatives including *T. monococcum*, *T. dicoccum*, *T. timopheevii*, and *T. durum*, were susceptible. Some accessions of *T. tauschii* were moderately resistant, however.

Allan (4) demonstrated that *A. elongatum* chromatin has considerable value for resistance to *Cephalosporium* stripe and tolerance to eyespot (caused by *Pseudocercosporella herpotrichoides*). The hard red winter wheat CI 13113 (Chinese Spring 2*/A. elongatum/Pawnee) was initially used as a parent because it expressed resistance to leaf rust (*Puccinia recondita*), stripe rust (*P. striiformis*) and stem rust (*P. graminis*). Subsequent progeny derived from crosses involving CI 13113 were found to be highly resistant to *Cephalosporium* stripe and have tolerance to eyespot. One line, PI 561033 (WA7437), proved to have combined resistances to the three rusts and both soilborne pathogens (4). This line was derived from a complex cross involving CI 13113 and three club wheat parents (Paha/CI 13645/ Chinese Spring 2*/A. elongatum/Pawnee/3/2*Omar). Cytological characterization of PI 13113 and PI 561033 confirmed that both lines contain *A. elongatum* chromatin. Using in situ hybridization with total genomic *A. elongatum* DNA as a probe, it was determined that both lines are euploid and have chromosome 6Ag substituted for chromosome 6A (X Cai & SS Jones, submitted). Further improvement is needed in agronomic and quality characteristics of club wheat germplasm containing *A. elongatum* source resistance. PI 561033 and other sib lines have adequate baking and milling properties but cookie diameter (an important trait for predicting end-use quality) is marginal, and the maturity of plants in the field is often delayed compared to other related lines lacking *A. elongatum* chromatin (4).

Jones et al (38) reported that an amphiploid of Chinese Spring wheat and *L. elongatum* (AABBDDDEE) produced by BC Jenkins (20) was resistant to *Cephalosporium* stripe. They also used a complete set of *L. elongatum* substitution lines derived from this amphiploid (21, 23, 82) to identify chromosomes that carry the major genes for resistance. Using plants inoculated in the growth chamber (38), they showed that chromosomes 2E and 3E have a significant effect on resistance in a Chinese Spring wheat background. For both critical chromosomes, substitutions for the A and B genome homoeologues had a greater effect than substitutions for the D genome homoeologues. Working with the same substitution lines, McGuire et al (57) observed a similar phenomenon: when chromosome 2E was substituted for chromosome 2A it had higher BYDV disease scores than the 2E for 2B or 2D substitutions. They suggested that the absence of chromosome 2A increases susceptibility to BYDV. An alternate, although not contrary, explanation is that chromosome 2A carries a resistance gene(s) that interacts with the chromosome 2E gene(s) to increase resistance. Differential effects of homoeologue substitution have
been demonstrated in these same substitution lines for resistance to soil salinity (67). Clearly, the choice of chromosome targeted for transfer can have an effect on the ultimate level of expression.

Development of commercially acceptable cultivars with resistance to *Cephalosporium* stripe has been slow, and cultivars with high levels of resistance adapted to the Pacific Northwest are not currently available. Disease development in field plots varies among years and locations (12, 51, 54, 56), making selection of resistant genotypes difficult. Such variation may be due to differences in density of pathogen inoculum and techniques (51, 52, 54, 56). In addition, the disease is strongly influenced by environment, especially soil freezing during the winter, which may also interact with genotype (12, 50, 56). Anderegg & Murray (5) developed a growth chamber–greenhouse method for studying *Cephalosporium* stripe that has been used to screen wheat germplasm and relatives for disease resistance (38). Environmental variation is reduced and results are available in 6 months compared with 11 months for field tests. Identification and selection of resistant genotypes may be more precise if pathogen colonization of plants is measured rather than estimating disease severity. Qi & Murray (69) transformed *C. gramineum* with the β-glucuronidase (GUS) reporter gene from *Escherichia coli* and were able to measure GUS activity in plants as soon as 10 days after inoculation. Preliminary studies have indicated that colonization of resistant genotypes, reflected by GUS activity, is less than in susceptible genotypes (TD Murray & CA Blank, unpublished).

**Eyespot**

Eyespot, caused by the soilborne pathogen *Pseudocercosporella herpotrichoides* (Fron.) Deighton (teleomorph=*Tapesia yallundae* Wallwork and Spooner), is a stem base disease of wheat grown in cool, temperate climates. The discovery of strains of the pathogen that are resistant to benzimidazole-type fungicides (39, 64) has led to increased efforts to produce resistant cultivars. Only three genes have been described for eyespot resistance: cultivar Cappelle-Desprez (84) carries a gene of unknown source on chromosome 7A (45); breeding line VPM-1 (49) has a gene (*PchI*) derived from *T. ventricosum* on chromosome 7D (33); and recently, Murray et al (63) mapped a new resistance gene(s) to chromosome 4V in *Dasypyrum villosum*.

Resistance to eyespot was reported in *T. ventricosum* and other wild wheats nearly 60 years ago by Sprague (78). He predicted that resistant wheat cultivars might eventually be developed from wild relatives of wheat. Simonet (77) used a bridging species, *T. persicum*, in the cross *T. persicum*/*T. ventricosum*/*T. aestivum* Mame to transfer eyespot resistance to hexaploid wheat. Maia (49) then used these lines to transfer resistance into the cultivated wheat line
VPM-1. The first commercial wheat to use the eyespot resistance of VPM-1 was the French cultivar Roazon (33). However, it was never widely grown.

The eyespot resistance of VPM-1 is believed to be controlled by a single locus (3, 17, 80). Using monosomic analysis, Jahier et al (33) showed that resistance is located on chromosome 7D. Doussinault et al (19) showed that an independently derived line, H-93-70, also having eyespot resistance from *T. ventricosum*, has a single dominant resistance gene designated *Pchl*. Worland et al (90) believed that the *Pchl* gene of H-93-70 was allelic with the gene of VPM-1, and this was subsequently confirmed by Mena et al (61).

The use of *Pchl* in cultivar development in the US Pacific Northwest is a success story on the use of wild gene resources for improvement of disease resistance. The USDA-ARS winter wheat breeding program at Washington State University initiated crosses with VPM-1 in 1975. Although some progeny of these crosses had eyespot resistance, they lacked other important traits such as commercially acceptable end-use quality, yield potential, and cold-hardiness. Yield potential of VPM-1 was 30% lower than the long-term check Nugaines based on 16 site-years of tests in Washington State. In 1974 though, eyespot resistant selections 951 and 421 of VPM/Moisson from the National Institute of Agronomic Research of LaRheu, France, were obtained. These two lines were agronomically superior to VPM-1, had yields 14 to 24% less than Nugaines in 16 site-years of tests, and had better milling quality than VPM-1. Both lines were used extensively as parents in an attempt to develop a resistant cultivar that was acceptable to the growers.

From 1978 to 1983, several thousand progeny from over 100 populations involving VPM-1/Moisson 421 and VPM-1/Moisson 951 were evaluated in head-row nurseries. Lines with promising agronomic type and resistance to endemic foliar diseases were placed in preliminary yield tests. Approximately 80 club-headed and common-headed wheat lines were selected each year from preliminary tests and placed in advanced yield tests.

Disease trials consisted of replicated inoculated and uninoculated (control) paired plots. The inoculated plots were sprayed with conidia of *P. herpotrichoides* in November each year, and the control plots were sprayed with a benzimidazole fungicide for protection. Comparisons were made between the paired plots for grain yield and symptoms of eyespot including lodged tillers and white heads (prematurely dead spikes). Lines with eyespot resistance sustained minimal grain yield loss and had low incidences of lodging and white heads.

In 1984, two promising lines, WA7163 and WA7166, with eyespot resistance were identified. In addition to eyespot resistance, both expressed resistance to stripe rust, leaf rust, and stem rust (9). These two lines had high grain yield potential, adequate cold-hardiness, excellent milling quality, and fair-to-
good soft wheat flour quality. In 1988, WA7163, a soft white winter wheat with a common spike, was named Madsen and released to growers (1). A year later WA7166, a club type soft white winter wheat, was released and named Hyak (2). Both cultivars have been readily accepted by growers in the Pacific Northwest primarily because of their resistance to eyespot. In 1994, the two cultivars were grown on over 500,000 ha in the region, and use of fungicides to control eyespot has been reduced significantly. Estimates indicate over 250,000 ha planted to Madsen and Hyak did not require fungicide treatment; this reduced growers production costs by ca $40/ha (TD Murray, unpublished). In 1994, Madsen was the mostly widely grown cultivar in the Pacific Northwest (BC Miller, personal communication).

The *Pch1* resistance gene has had only limited use in Europe. According to Law et al (46), substitution of the 7D chromosome of VPM1 into several adapted United Kingdom wheats depresses yield by about 6%. These authors indicated that it should be possible to break the deleterious linkages between genes for low yield and the *Pch1* gene, as has been done in the US wheats.

Other genes may interact with *Pch1* to increase or decrease eyespot resistance. Hollins et al (32) concluded the potent eyespot resistance of Rendezvous was due to the combined resistance of Cappelle-Desprez and VPM-1. Similarly, Allan & Roberts (3) identified progeny with mean lesion indices that transgressed both resistant parents for higher resistance in a cross between VPM/Moisson 951 (resistant) and Cerco (moderately resistant).

The *Pch1* gene does not provide complete resistance to eyespot or the subsequent losses in grain yield associated with the disease. Madsen sustained significant yield losses (average 15%) in 5 of 13 tests when inoculated and control plots were compared (RE Allan, unpublished). Murray & Bruehl (62) observed similar results with VPM-1, which sustained significant loss in grain yield one year in four under favorable disease conditions. Additional sources of eyespot resistance are needed because the resistance conferred by *Pch1* is not complete and may not be durable. Increases in yield loss of Madsen relative to susceptible cultivars have been observed over a 12-year period in eyespot field evaluation tests (RE Allan, unpublished). In a search for new sources of resistance genes for eyespot, hundreds of accessions of *T. tauschii* (DD genome) have been screened and were shown to carry a high frequency of resistance (92). Work is in progress to transfer these new source genes, as well as the recently described gene from *D. villosum* (63), into Madsen, Hyak, and other adapted backgrounds (TD Murray & SS Jones, unpublished).

Recently, de la Peña & Murray (16) described an improved method for evaluating resistance to eyespot of wheat genotypes. They used a GUS-transformed eyespot strain to measure differences in disease development of 4- to 8-week-old wheat seedlings. This strain has the β-glucuronidase (GUS) re-
porter gene, and production of the enzyme is highly correlated with growth of the pathogen. de la Peña & Murray (16) showed that differences in GUS activity in seedling tissue of wheat genotypes was closely correlated with differences in resistance to eyespot. The method differentiated among highly resistant, resistant, and susceptible genotypes. Evaluation of resistance with this technique is reduced from about 11 months to 2 months.

Selecting for resistance to the eyespot pathogen using field or greenhouse screening procedures is labor intensive, sometimes inaccurate, and in the case of field tests, slow, taking up to 11 months. Breeding for eyespot resistance in Madsen and Hyak was greatly enhanced with the discovery of a marker for the Pchl gene of VPM-1 and its derivatives. Gale et al (28) suggested that the eyespot resistant gene was on 7DL and probably at the distal end because it segregated independently of an isozyme marker for alpha-amylase located near the centromere. The 7DL endopeptidase locus was shown to be about 42 map units from the centromere (60). Several progeny from a cross between Sel. 421 (eyespot resistant) to Sel. 101 (eyespot susceptible) were classified for their endopeptidase allele frequencies and eyespot resistance (60), based on lesion index scores of mature straw. A close association between the VPM-1 Pchl gene and the endopeptidase allele EP-VI (Ep-DIb) was detected. Others subsequently verified this close linkage using different parental lines (42, 83, 90). Summers et al (81) compared the association between the Pchl gene and EP-DIb endopeptidase allele among large numbers of progeny and confirmed the two genes were tightly linked in coupling. Worland et al (90) suggested that the Ep-DIb gene may confer eyespot resistance. Mena et al (61) proved that Ep-DIb and Pchl genes are only closely linked and can be separated from each other; they also showed Pchl was transferred from chromosome 7DV of T. ventricosum. Traditional evaluation of eyespot disease is difficult, time consuming, and not always successful.

**DISCUSSION**

Except for a few cases reported in other reviews (35, 58) and the eyespot resistance transfers discussed here, most alien gene transfers for disease resistance are still not used in commercial wheats. The resistance levels achieved in adapted lines are in some examples equal to that of the wild species, although linked with these genes come undesirable traits that affect yield, end-use quality, and other agronomic traits. Continued rounds of radiation or homoeologous pairing to promote recombination are still required to remove unwanted alien chromatin before breeders can incorporate these valuable genes into commercially acceptable cultivars.

New approaches are also needed for the identification of resistant genotypes,
especially when dealing with race-nonspecific resistance and pathogens that are facultative parasites. In these cases, potentially valuable genotypes can be lost owing to an inability to select resistant individuals because resistance is not complete and environmental effects may be large.

Marker-based selection, such as the Ep-D1b marker for the Pch1 eyespot resistance gene, enables selection of resistant individuals with certainty and eliminates undesirable genes. Developing markers still requires the identification of resistant genotypes based on a phenotypic response following inoculation with a pathogen. Use of a GUS-transformed strain of *P. herpotrichoides* (16) along with chromosome addition lines enabled Murray et al (63) to map a new gene for resistance in *D. villosum*, which was not possible with visual disease evaluations. The relative ease of transformation of most fungi should allow the use of reporter genes for detection of resistance in other host-pathogen systems.

Chromosome manipulation or, more specifically, chromosome engineering, which has been practiced for at least 50 years (71), is the main tool used by wheat researchers for introgression of alien genes. Not only has this work led to hundreds of well-characterized alien transfers but it has also set a standard for gene transformation systems to try to equal. Wheat is unique among plants in that it allows geneticists to target gene transfers to specific chromosomes through the use of aneuploid stocks and the induction of homoeologous pairing and recombination (72). Although transformation systems have recently been successful in wheat (86), chromosome engineering still has substantial advantages and potential that should not be abandoned. When transferring alien resistance genes, wheat cytogeneticists deal with the classical definition of the gene, a unit of inheritance. Thus, not only is the coding sequence of the gene transferred but so is everything else needed to confer resistance. What the “everything else” is at this point is not clear. It certainly involves regulatory sequences but may also include positional and other undescribed effects. Genetic transformation will certainly have an impact on the introduction of novel disease resistances into wheat, but this impact will not be realized until resistance genes are cloned, characterized, and their expression limited to the correct timing and tissue. In the meantime, chromosome engineering, aided by the latest techniques of in situ hybridization, chromosome banding, and DNA manipulation, will continue to contribute significantly to our understanding and to solve the very real problems involved in wheat-pathogen interactions.
Literature Cited

4. Allan RE, Rubenthaler GL, Moris CF, Line RF. 1993. Registration of three soft white winter wheat germplasm lines resistant or tolerant to strawbreaker foot rot. Crop Sci. 33:1111-12
25. Dyck PL, Lukow OM. 1988. The genetic analysis of two interspecific sources of


of host resistance to *Pseudocercosporella herpotrichoides* on yield and yield components in winter wheat. *Plant Dis.* 70:851–56


75. Sharma HC, Gill BS, Uyemoto JK. 1984. High level of resistance in *Agropyron* species to barley yellow dwarf and