Resistance to soil-borne diseases of wheat: Contributions from the wheatgrasses *Thinopyrum intermedium* and *Th. ponticum*

Hongjie Li[1,4], R. L. Conner[2], and T. D. Murray[3]

[1]National Key Facility of Crop Gene Resources and Genetic Improvement (NFCRI), Institute of Crop Sciences, Chinese Academy of Agricultural Sciences, Beijing 100081, China; [2]Agriculture and Agri-Food Canada, Morden Research Station, Unit 100-101 Route 100, Morden, Manitoba, Canada R6M 1Y5; and [3]Department of Plant Pathology, Washington State University, Pullman, Washington 99164-6430, USA. Received 5 January 2007, accepted 7 September 2007.

Li, H. J., Conner, R. L. and Murray, T. D. 2008. **Resistance to soil-borne diseases of wheat: Contributions from the wheatgrasses *Thinopyrum intermedium* and *Th. ponticum*.** Can. J. Plant Sci. **88**: 195–205. Eyespot, Cephalosporium stripe, and common root rot are soil-borne diseases that damage the stem bases, vascular system, subcrown internodes, and roots of wheat. Resistance in wheat to these diseases is insufficient to prevent significant yield loss when disease is severe. The wheatgrasses *Thinopyrum intermedium* and *Th. ponticum* are highly resistant to these diseases. Identification of disease-resistant wheat-*Thinopyrum* partial amphiploids, chromosome addition, substitution, and translocation lines makes them a valuable source of resistance genes for wheat breeding programs. Single chromosomes or chromosome segments containing resistance genes can be transferred into wheat to produce genetic stocks that afford a better understanding of the genetic control of resistance in wheatgrasses and new genetic resources for wheat improvement. Resistance to eyespot in *Th. intermedium* and *Th. ponticum* was associated with the homoeologous group 4 chromosomes, whereas resistance to Cephalosporium stripe was controlled by genes located on chromosomes 3 and 6 of *Th. ponticum*. Despite the fact that some eyespot- and common root rot-resistant wheat-*Thinopyrum* lines have blue kernels, resistance is not tightly linked to the blue aleurone trait.

Key words: *Thinopyrum intermedium*, *Th. ponticum*, eyespot, Cephalosporium stripe, common root rot, *Oculimacula yallundae, O. acuformis, Cephalosporium gramineum, Bipolaris sorokiniana*

Li, H. J., Conner, R. L. et Murray, T. D. 2008. **Résistance du blé aux maladies véhiculées dans le sol : bienfaits des agropyres *Thinopyrum intermedium* et *Th. ponticum*.** Can. J. Plant Sci. **88**: 195–205. Le piétin-verse, la strie céphalosporienne et le pourridié commun sont des maladies véhiculées dans le sol. Elles endommagent la tige du blé à sa base, son système vasculaire, les entrenæuds sous le collet et les racines. Les variétés de blé ne résistent pas assez à ces maladies pour empêcher une forte diminution du rendement dans les cas les plus graves. Les agropyres *Thinopyrum intermedium* et *Th. ponticum* résistent très bien à ces maladies. Les lignées blé-*Thinopyrum* résistantes partiellement amphiploïdes obtenues par addition, substitution ou translocation de chromosomes pourraient devenir un réservoir utile de gènes codant la résistance pour les programmes d'hybridation. On pourrait transférer des chromosomes ou des fragments de chromosome renfermant les gènes de résistance au blé pour obtenir du matériel génétique qui nous aidera à mieux comprendre la régulation génétique de cette résistance chez l'agropyre et nous donnera de nouvelles ressources phytogénétiques. Chez *Th. intermedium* et *Th. ponticum*, on associe la résistance au piétin-verse aux chromosomes homologues du groupe 4, alors que la résistance à la strie céphalosporienne résulte des gènes situés sur les chromosomes 3 et 6 de *Th. ponticum*. Bien que certaines lignées de blé-*Thinopyrum* résistantes au piétin-verse et au pourridié commun présentent des grains bleus, la résistance à ces maladies ne présente pas d'étroite corrélation avec le caractère de l'aleurone bleue.

Mots clés: *Thinopyrum intermedium, Th. ponticum*, piétin-verse, strie céphalosporienne, pourridié commun, *Oculimacula yallundae, O. acuformis, Cephalosporium gramineum, Bipolaris sorokiniana.*

Soil-borne fungal pathogens of wheat (*Triticum aestivum* L. em. Thell.) infect and destroy the stem bases, vascular system, subcrown internodes, and roots, resulting in discoloration and destruction of the infected parts (Wiese 1991; Bailey et al. 2003). Infection often causes a reduction in growth of the root system and lodging of stems when these diseases are severe. Infected plants can exhibit seedling blight, reduced tiller number, stunting, premature ripening of stems (white heads), smaller kernels, poor quality seeds, and reduced yield (Mathre et al. 1977; Wiese 1991; Martens et al. 1994). However, above-ground symptoms are often not evident, especially at early developmental stages, which make some soil-borne diseases difficult to recognize.

Eyespot is caused by *Oculimacula yallundae* (Wallwork & Spooner) Crous & W. Gams (syn. *Tapesia*

[4] *To whom correspondence should be addressed (e-mail: hongjie2000@caas.net.cn).*

yallundae Wallwork & Spooner) and *O. acuformis* (Boerema, R. Pieters & Hamers) Crous & W. Gams [syn. *T. acuformis* (Boerma, Pieters & Hamers) Crous.], which are the teleomorphs of *Helgardia herpotrichoides* (Fron) Crous & W. Gams [syn. *Pseudocercosporella herpotrichoides* (Fron.) Deighton var. *herpotrichoides*] and *H. acuformis* (Nirenberg) Crous & W. Gams (syn. *P. herpotrichoides* var. *acuformis*). Eyespot is prevalent in winter-wheat-producing areas in the Pacific Northwest US (PNW), the Great Lakes regions, and parts of northern Europe, South America, New Zealand and Australia (Wiese 1991). Cephalosporium stripe was first described in Japan in 1930 and occurs across a wide range of winter-wheat-producing areas of North America and Britain. This disease is caused by *Cephalosporium gramineum* Nisikado & Ikata in Nisikado et al. (syn. *Hymenula cerealis* Ellis & Everh.) (Wiese 1991). Common root rot is incited by *Bipolaris sorokiniana* (Sacc. in Sorok.) Shoem. [teleomorph *Cochliobolus sativus* (Ito and Kurib) Drechs. ex Dastur] and is a major soil-borne disease of spring wheat in the Canadian Prairies (Ledingham et al. 1973). The impact of these diseases on yield varies from year to year and ranges from minor to major, depending on the climatic conditions that favor each disease. Cool and moist weather in the autumn favors the occurrence of eyespot and Cephalosporium stripe in winter wheat, and warm weather and drought favors common root rot in winter and spring wheat (Wiese 1991). Yield loss due to all three diseases is most severe when wheat is subjected to moisture stress during grain fill. Resistant cultivars are generally considered to be a cost-effective method of reducing yield losses caused by soil-borne diseases (Scott et al. 1989; Tinline et al. 1989; Jones et al. 1995). However, resistance to these soil-borne pathogens is rare or absent in the common wheat gene pool. Thus, development of resistant cultivars relies on genetic resistance from other species related to wheat.

Intermediate wheatgrass, *Thinopyrum intermedium* (Host) Barkworth and D. R. Dewey ($2n = 6x = 42$) [syn. *Agropyron intermedium* (Host) Beauvois, *Elytrigia intermedia* (Host) Nevski], and tall wheatgrass, *Th. ponticum* (Podp.) Barkworth and D. R. Dewey ($2n = 10x = 70$) [syn. *A. elongatum* (Host) Beauvois and *Lophopyrum ponticum* (Podp.) Löve, *E. elongata* (Host) Nevski], have attracted much interest because they are sources of genes for improving agronomically important traits and resistance to biotic and abiotic stresses in wheat (Friebe et al. 1996; Fedak 1999). Attempts to transfer useful traits from wheatgrasses into wheat can be traced back to the 1930s, 1940s, and 1950s in the former USSR, USA, Canada, and China (Armstrong 1936; Peto 1936; Smith 1942, 1943; Tsitsin 1965; Sun 1981; Li et al. 1985). Early wheat-*Thinopyrum* hybridization was aimed at producing perennial wheat (Tsitsin 1965), which resulted in the development of numerous wheat-*Thinopyrum* hybrids and/or partial amphiploids that are still used as intermediates in the production of chromosome addition, substitution, and translocation lines.

Although none of the wheat-*Thinopyrum* hybridization projects were originally intended to transfer resistance to the soil-borne pathogens discussed in this review, resistance is present in *Th. intermedium* and *Th. ponticum* and some of their partial amphiploids (Conner et al. 1989; Cai et al. 1996; Cox et al. 2002). The identification of specific chromosome addition or substitution lines provides an opportunity to better understand the genetic control of resistance to soil-borne diseases in the wheatgrasses (Conner et al. 1989; Cai et al. 1996; Li et al. 2004a, 2005). This review summarizes the contributions of intermediate and tall wheatgrasses to the improvement of resistance in wheat against eyespot, Cephalosporium stripe, and common root rot.

CONTROLLING SOIL-BORNE PATHOGENS

Management of soil-borne pathogens has relied on reducing inoculum in the soil via cultural controls such as crop rotation and management of crop residues, and application of fungicides; however, these practices are only partially effective in reducing the incidence and severity of disease. Rotation of winter wheat with spring cereals or non-host crops such as corn (*Zea mays* L.) and legumes helps reduce damage to winter wheat by preventing inoculum build-up in soil, and summer fallow provides a crop-free interval during which pathogen populations in soil decrease (Diehl et al. 1982; Wiese 1991). The shortage of profitable alternative crops in areas such as the prairie drylands makes crop rotation less feasible (Conner and Atkinson 1989; Conner et al. 1996). Common root rot is most severe on barley (*Hordeum vulgare* L.), so spring wheat and barley should not be grown together in a crop rotation; however, the inclusion of barley in a rotation can diminish root rot severity in comparison to continuous planting of spring wheat (Conner et al. 1996). Late autumn planting reduces autumn root growth, which allows winter wheat to escape infection by *C. gramineum* and *B. sorokiniana*; however, it does not prevent infection at later growth stages (Wiese 1991). Minimum tillage and adequate fertilization (Verma et al. 1975; Conner et al. 1987) decrease the severity of common root rot, but will not eliminate symptoms of this disease.

Fungicide seed treatments are registered for the control of seedling blight, but they may not provide long-term control of root rot (Bailey et al. 2003). Benzimidazole fungicides applied to foliage have been used effectively in controlling eyespot of winter wheat in Europe and the US PNW beginning in the 1970s; however, concerns arose after the discovery of pathogen isolates with resistance to the benzimidazoles and their use has decreased (Rashid and Schlösser 1977; Brown et al. 1984; Murray 1996). No fungicides are available for the control of Cephalosporium stripe.

Development of eyespot-resistant wheat cultivars has been an important objective of winter wheat breeding programs for many years in the US PNW and Europe. Three resistance genes have been identified. Gene *Pch1*, derived from *T. ventricosum* Ces. (Doussinault et al. 1983), is located on chromosome 7DL (Gale et al. 1984) and is the primary gene used in eyespot-resistant cultivars in the US PNW. Gene *Pch2*, present in the French cultivar Cappelle-Desprez, is located on chromosome 7AL (Law et al. 1976; de la Peña et al. 1996), but is seldom used in US PNW cultivars. Although durable in Europe (Muranty et al. 2002), *Pch2* is less effective than *Pch1* and it is difficult to recover full resistance. Muranty et al. (2002) reported that gene *Pch2* was effective only at the seedling stage and that adult-plant resistance to eyespot in Cappelle-Desprez is controlled by another major gene on chromosome 5A. Gene *Pch3*, which is located on chromosome 4V of *Dasypyrum villosum* L. Candargy (syn. *Haydaldia villosa* L.), is highly effective (Murray et al. 1994; Yildirim et al. 1998), but needs to be transferred into adapted cultivars before it can be used in breeding programs. Uslu et al. (1998) confirmed that chromosome 4V controlled resistance to *O. yallundae*, but that chromosomes 1V, 2V, and 3V contributed to resistance to *O. acuformis*.

Resistance to common root rot is more complicated than eyespot. A monosomic analysis of moderate resistance to root rot concluded that resistance in the hard red spring wheat cultivars Apex and Cadet is controlled by a single recessive gene (Larson and Atkinson 1970), which was located on chromosome 5BL and designated *Crr* (Larson and Atkinson 1981, 1982). Minor genes for root rot resistance were identified on chromosomes 2B and 2D in Apex (Larson and Atkinson 1970). Savel'eva and Maistrenko (1983) identified major root rot resistance genes on chromosomes 2B and 2D, as well as minor genes on chromosome 6A in cultivar Skala. Bailey et al. (1988) determined that resistance to common root rot was a partially dominant trait and quantitatively inherited in progeny derived from the cross Willet/McMurachy//Manitou/Pitic and Carazinho/CT763//Atlas/CT 263/3/PI 266896.

Commercial cultivars of hard red spring and durum wheat [*T. turgidum* (L.) ssp. *durum* (Desf.) Husn.; syn. *T. durum* Desf.] are at best only moderately resistant to common root rot (Harding 1972; Tinline et al. 1989). Currently, most spring wheat cultivars available in western Canada are moderately resistant (Campbell 1970; Harding 1972; McKenzie 1976; Tinline et al. 1989), but do not have adequate resistance to protect wheat from the disease (Harding 1972).

Although variation in reaction to Cephalosporium stripe occurs in winter wheat, genotypes with highly effective resistance to *C. gramineum* have not been found and most cultivars are moderately to highly susceptible (Mathre et al. 1977; Jones et al. 1995). Consequently, wild wheats were evaluated for resistance to Cephalosporium stripe.

Wild species can provide effective sources of resistance to soil-borne diseases, but it requires methods that allow the identification of resistant genotypes and for manipulating the chromosomes carrying resistance genes with the goal of eventually recombining these genes into cultivated varieties. Resistance to eyespot, Cephalosporium stripe, and common root rot was found in *Th. intermedium* and *Th. ponticum* and wheat-*Thinopyrum* derivatives (Mathre et al. 1985; Conner et al. 1989; Cox et al. 2002). The identification and transfer of resistance to the soil-borne pathogens in the wheat-wheatgrass lines were thus carried out (Conner et al. 1989; Cai et al. 1996; Li et al. 2004a,b, 2005).

METHODS OF EVALUATING WHEAT FOR RESISTANCE TO SOIL-BORNE PATHOGENS

Reliable methods for the identification of resistant genotypes at both the seedling and adult plant stages are needed, since above-ground symptoms are usually not obvious until later stages of plant development. The visual evaluation of symptoms is routinely used to identify resistant and susceptible phenotypes in breeding programs because of its simplicity, ease of application, high throughput, and relative accuracy. Root rot severity can vary considerably among single plants even in plots containing genetically identical plants (Bailey et al. 1988). The possibility of escape from infection (Sallans and Tinline 1964) and the influence of environment on the reaction of individual plants (Bailey et al. 1988) make determination of root rot severity based on single plants unreliable. For these reasons, root rot reactions are not determined on a single plant basis, but rather by rating either the progeny of a particular plant or a representative sample of plants from a cultivar, cytogenetic line, or advanced breeding line. Disease severity is usually determined by categorizing plants based on discoloration of the infected plant parts. For common root rot, the most obvious symptom is a brown discoloration of the subcrown internode, which is the underground stem between the seed and the crown (Bailey et al. 2003) and disease severity is based on the extent of lesion development on the subcrown internode. Ledingham et al. (1973) developed a system that separates plants into severe, moderate, slight, and nil categories, which is widely used to determine the severity of common root rot. Severity of eyespot is based on the number and size of lesions on the stem base, whereas Cephalosporium stripe severity is based on colonization of successive leaves up to and including the ear; for both diseases, stems are evaluated and grouped into one of the four categories (Bockus and Sim 1982; Specht and Murray 1990; de la Peña and Murray 1994). Rating scores often are summarized as a percentage for common root rot (Burrage and Tinline 1960) or an index for eyespot (Strausbaugh and Murray 1989) and usually are associated with yield loss (Ledingham et al. 1973).

Because of the inconsistencies associated with visual rating systems, many attempts have been made to

improve the accuracy of disease severity classification. Although different rating scales have been used to differentiate resistance responses to pathogens (Sallans and Tinline 1964; Tinline et al. 1994), improvement is still needed to reduce subjective errors inherent in visual rating systems. Kokko et al. (1993) developed a method for quantifying discoloration of the subcrown internode using digital image analysis that makes assessment of root rot severity more objective and precise than visual ratings. Theoretically, image analysis of tissue discoloration can be extended to a wide range of specimens and used as a complement to visual ratings or as a stand alone system. However, this method is influenced by lighting, the three-dimensional shape of the specimen, and the hardware and software used by different laboratories.

Immunological and molecular approaches also have been used to assess disease severity. A serological test based on the enzyme-linked immunosorbent assay (ELISA) was developed to detect the eyespot pathogen *O. yallundae* and has been used to differentiate resistant and susceptible wheat cultivars (Lind 1988; Unger and Wolf 1988). Lind (1992) concluded that ELISA reliably identified eyespot-resistant genotypes only from anthesis to maturity. Although the pathogen could be detected in seedling plants, resistance to eyespot was poorly correlated with the ELISA readings. At later growth stages, however, the serological reaction was more accurate than the visual scoring method in differentiating resistant plants from susceptible plants (Lind 1992).

The *gusA* construct from *Escherichia coli* (Migula 1895) Castellani and Chalmers 1919, which encodes β-glucuronidase (GUS), is used as a reporter gene in studies of gene expression. Bunkers (1991) transformed *O. yallundae* with the GUS and reported no deleterious effects on growth in culture or virulence of the transformants. de la Peña and Murray (1994) used the GUS-transformed *O. yallundae* to develop the GUS seedling test for eyespot resistance. Because the GUS gene is under control of a constitutive promoter, GUS activity in infected plants is proportional to growth of the pathogen. Activity of GUS is measured fluorimetrically after extracting the sap from infected stem bases and resistance is associated with reduced colonization (lower GUS activity). The GUS seedling test has been used to identify resistance in several wild wheat species including the wheatgrasses (Murray et al. 1994; Yildirim et al. 1995; Cadle et al. 1997; Cox et al. 2002; Li et al. 2004a, 2005). *Bipolaris sorokiniana* and *C. gramineum* were transformed with the GUS gene to monitor their development in plant tissues (Liljeroth et al. 1993; Qi and Murray 1994; Douhan and Murray 2001). Green fluorescent protein also was used as a reporter gene to observe the growth of *O. yallundae* in plants, but has not been used in studies of disease resistance (Bowyer et al. 2000).

Amplification of pathogen DNA with the polymerase chain reaction (PCR) has been used to measure colonization of plants by pathogens. Competitive PCR was used to quantify the amount of pathogen DNA in plant tissues for detection of *O. yallundae* and *O. acuformis* (Nicholson et al. 1997). Difficulties have occurred in PCR-based diagnoses of *Oculimacula* spp. in young plants relative to older plants, especially for *O. acuformis*, which usually does not cause obvious symptoms in seedlings (Nicholson et al. 2002). Quantification of DNA was not always associated with visual ratings in the assessment of resistance of *D. villosum* to *O. yallundae* and *O. acuformis* (Uslu et al. 1998). Visual rating scores were determined by counting the number of leaf sheaths penetrated, whereas quantification of pathogen DNA reflected the extent of colonization of the pathogen in the entire stem base (Uslu et al. 1998).

Real-time PCR is another option for quantifying the amount of pathogen DNA in plants. Pathogen-specific sequences are amplified with a fluorescence-labeled dye and measured following each cycle of amplification using an integrated thermocycler-fluorimeter. Compared with competitive PCR, real-time PCR has a greater dynamic range, does not need post-reaction processing and there is no risk of carryover contamination (McCartney et al. 2003). Real-time PCR offers a fast, high-throughput method for quantitatively measuring the growth of pathogens in plant tissues. Walsh et al. (2005) developed a real-time PCR assay to discriminate and quantify *O. yallundae* and *O. acuformis* in plants. Gedye and Murray (unpublished data) have recently developed a real-time PCR and used it to evaluate colonization of seedlings inoculated with *O. yallundae* and *O. acuformis* as an alternative to the GUS seedling test.

Methods for the quantitative evaluation of disease severity based on serological methods, GUS activity, and DNA analysis are more sensitive than visual ratings and allow examination of a large number of samples at different growth stages. Such procedures are especially useful for evaluating the resistance of genotypes in multiple experiments carried out over locations or at different times. These tests do not replace field evaluations because the ultimate objective is to incorporate effective disease resistance that will reduce or prevent yield losses in wheat under natural conditions. However, the most susceptible individuals in a population can be eliminated in greenhouse or growth chamber experiments, so that only the most resistant individuals are included in field tests, which should increase the efficiency of field screening.

TRANSFERRING RESISTANCE TO SOIL-BORNE PATHOGENS FROM WHEATGRASSES TO WHEAT

Their ease of crossing with wheat makes the wheatgrasses attractive to breeders and pathologists. Attempts to cross wheat with the wheatgrasses have been initiated in several countries and partial amphiploids were developed through repeated backcrossing of wheat-*Thinopyrum* hybrids to adapted wheat cultivars. These partial amphiploids usually contain seven pairs of

Thinopyrum chromosomes, some of which carry resistance genes to various diseases. Because most of the wheat-*Thinopyrum* crosses were made decades ago, pedigree information is not always available. Understanding the genomic origin and composition of these partial amphiploids is therefore necessary for manipulation of specific chromosomes. Such an understanding can be accomplished by conducting molecular cytogenetic analyses using chromosome banding techniques, in situ hybridization and DNA-based detection systems.

Characterization of wheat-*Thinopyrum* partial amphiploids and detection of *Thinopyrum* chromosomes were recently reviewed by Chen (2005) and Fedak and Han (2005). Chen et al. (1998b) outlined a strategy using the St genomic DNA from *Pseudoroegneria strigosa* (M. Bieb) Á. Löve as a probe to differentiate chromosomes from the various genomes of *Thinopyrum*. Using a repetitive DNA sequence specific for genus *Thinopyrum* (Wang and Wei 1995), PCR was used to detect the presence of alien chromatin in wheat-*Thinopyrum* derivatives (Li et al. 2004a, 2005).

Most partial amphiploids contain no more than 56 chromosomes, even though *Th. intermedium* and *Th. ponticum* are hexaploid and decaploid species, respectively. Considerable sterility and meiotic instability is encountered in F_1 hybrids between different partial amphiploids. Initially, it was thought that partial amphiploids were composed of all the wheat genomes and one complete *Thinopyrum* genome (Li et al. 1985). However, recent molecular cytogenetic analyses have demonstrated that most of the alien genomes in the partial amphiploids examined consist of chromosomes from different genomes of *Thinopyrum* with a few exceptions (Banks et al. 1993; Zhang et al. 1996a,b; Chen et al. 1998a, 2003a,b; Fedak et al. 2000; Fedak and Han 2005). This conclusion was reinforced by analyzing chromosome addition lines derived from the same partial amphiploids (Chen et al. 1999; Tang et al. 2000). Chromosome exchanges between wheat and *Thinopyrum* or between the alien chromosomes occurred in some partial amphiploids (Cai et al. 1998; Zhang et al. 1996a,b; Chen et al. 2003b). The heterogeneous genomes of *Thinopyrum* in partial amphiploids are usually regular in their meiotic behavior, which ensures stable transmission of the alien chromosomes to the progeny (Banks et al. 1993).

Since partial amphiploids are readily accessible intermediates for the transfer of useful genes from wheatgrasses to wheat, some have received extensive study to better understand their genomic composition and reactions to various diseases. Although none of the original hybridizations were aimed at transferring resistance to eyespot, Cephalosporium stripe or common root rot, resistance to these diseases was detected in some wheat-*Thinopyrum* partial amphiploids, and some were resistant to more than one disease (Table 1; Cox et al. 2002). For example, the wheat-*Th. ponticum* partial amphiploid AT3425 ($2n = 56$) was identified as a promising source of resistance to Cephalosporium stripe, eyespot, and *Wheat streak mosaic virus* (WSMV) (Mathre et al. 1985; Cox et al. 2002; Li et al. 2004a). Using *Th. ponticum* genomic DNA as a probe, GISH analysis detected seven pairs of *Thinopyrum* chromosomes and three pairs of wheat-*Thinopyrum* translocated chromosomes in addition to 18 pairs of wheat chromosomes in AT3425 (Cai et al. 1998). This partial amphiploid was believed to have originated from a cross between common wheat and the diploid species *A. elongatum* (Mathre et al. 1985). However, further analyses based on GISH and chromosome pairing demonstrated that AT3425 was most likely derived from a wheat × *Th. ponticum* cross (Cai et al. 2001).

Agrotana is a wheat-alien partial amphiploid of unknown origin, which is resistant to common root rot, stem rust (*Puccinia graminis* Pers.:Pers. f. sp. *tritici* Eriks. & E. Henn.), WSMV and its vector, the wheat curl mite (WCM) (*Aceria tosichella* Keifer) (Whelan 1983; Conner et al. 1989). Initially, Agrotana was incorrectly described as a *T. turgidum* ssp. *durum-A. trichophorum* (Link) Richt. [syn. *Th. intermedium* ssp. *trichophorum* (Link) A. & Gr.] partial amphiploid (Bequette et al. 1961). Cytogenetic studies showed that Agrotana contained the A, B, and D genomes of wheat (Whelan 1983; Conner et al. 1989). Subsequent C-banding and GISH analysis demonstrated that this partial amphiploid was composed of 16 alien and 40 wheat chromosomes (Xu et al. 1994; Chen et al. 1995). Using an St genomic probe, GISH analysis determined that Agrotana contained 8 E- or J- and 8 J^s-genome-chromosomes derived from *Th. ponticum* (Chen et al. 1998a). By crossing Chinese Spring with Agrotana, a large number of progeny lines were developed, which permitted the transfer of resistance to common root rot into wheat (Li et al. 2004b).

Dr. W. J. Sando of the USDA made interspecific and intergeneric crosses between 1930 and 1940 and produced over 490 entries in the Sando selections that are maintained at the USDA National Small Grains Germplasm Research Facility in Aberdeen, ID. Cytological analysis demonstrated that these accessions have chromosome numbers ranging from $2n = 42$ to 56 (Hang et al. 2005). Some of the wheat-*Thinopyrum* collections were resistant to eyespot, Cephalosporium stripe, and WSMV (Table 1; Cox et al. 2002). Most of the Sando selections with multiple disease resistance were partial amphiploids with chromosome numbers $2n = 54$ or 56. Like other partial amphiploids, the Sando lines also contained a synthetic genome derived from *Thinopyrum* as revealed by GISH using St genomic DNA as a probe (Table 1). Some of them also had a perennial growth habit.

Many wheat-*Th. intermedium* partial amphiploids possess resistance to viral and fungal diseases. For example, the Zhong lines are partial amphiploids derived from the same *Th. intermedium* accession (Sun 1981) and are resistant to Barley yellow dwarf virus and WSMV (Zhang et al. 1996b; Chen et al. 2003b). Based on inoculation with GUS-transformed pathogens, Zhong 2, 3, 4, and 5 were resistant to *O. acuformis*,

Table 1. Reaction of wheat-*Thinopyrum* hybrids to eyespot, Cephalosporium stripe, common root rot, and Wheat streak mosaic virus (WSMV) and their chromosome constitutions

Line	Origin	Eyespot	Cephalosporium stripe	Common root rot	WSMV	$2n$ =	Chromosome constitution
Thinopyrum intermedium							
MT-2 (PI 505820)	USA	R	R	ND	R	52–56	23–24 W+8–10 St+8 J^s+13 J
Zhong 2	China	RR	ND	ND	R	53–56	42 W+2 St+4 J^s+4 J+2 St-J^s
Zhong 3	China	RS	ND	ND	R	56	42 W+4 St+4 J^s+2 St-St-J^s+2 St-J+2 J^s-J^s-St
Zhong 4	China	RS	ND	ND	R	54, 56	42 W+4 St+4 J^s+2 St-J^s+2 St-St-J^s +2 St-J
Zhong 5	China	RS	ND	ND	R	56	40 W+4 St+2 J^s+2 J^s-W+2 St-J^s+2 St-St-J^s+2 St-J+2 W-J^s
CI 15092	USA	SR	ND	ND	R	42	40 W+2 J^s
CI 17881	USA	RR	ND	ND	R	44	40 W+2 J^s+2 WL-WS-T
CI 17885	USA	RR	ND	ND	R	42	40 W+2 J^s
CI 17884	USA	RR	ND	ND	R	42	40 W+2 WL-J^sS
4161R	USA	R	ND	ND	R	42	T4DL·4Ai#2S
4165R	USA	R	ND	ND	R	42	T4DL·4Ai#2S
4266R	USA	R	ND	ND	R	42	T4DL·4Ai#2S
4274R	USA	R	ND	ND	R	42	T4DL·4Ai#2S
4292R	USA	R	ND	ND	R	42	T4DL·4Ai#2S
Th. ponticum							
PI 550713	USA	R[z]	R	ND	R	56	36 W+14 T+6 W-T[x]
PI 550715	USA	R	R	ND	R	56	ND
AT3425 (NSL 91403)	USA	R	R	ND	S	56	36 W+14 T+6 W-T
SS103 (PI 611891)	USA	R	R	ND	R	56	40 W+ 12 T +2 W-W-T+2 T-T-W
SS191	USA	S	R	ND	S	56	ND
SS237 (PI 611899)	USA	R	R	ND	R	56	42 W+ 14 T
SS259 (PI 611904)	USA	R	R	ND	S	54, 56	42–44 W+12 T
SS364 (PI 611911)	USA	S	R	ND	S	56	ND
SS365 (PI 611912)	USA	S	R	ND	S	56	ND
SS524 (PI 611923)	USA	R	R	ND	S	56	ND
SS679	USA	S	R	ND	S	56	ND
Agrotana (PI 550715)	USA	ND	ND	R	R	56	40 W+8 J+8 J^s
SS767 (PI 611939)	USA	RR[y]	S	ND	S	42	DS 4J(4D)
54-40-2-5-11	Canada	ND	ND	R	S	44	40 W+2 J-J^s+2 J^s-J
54-40-2-5-28	Canada	ND	ND	R	S	44	40 W+2 J-J^s+2 J^s-J
J99C009	USA	ND	R	ND	ND	42	3DS?3DL–3TL
J99C010	USA	ND	R	ND	ND	42	3DS?3DL–3TL
CI 13113	USA	ND	R	ND	ND	42	6Ae#(6A)
REA 9232	USA	ND	R	ND	ND	42	6Ae#(6A)
PI 561033	USA	ND	R	ND	ND	42	6Ae#(6A)
T-Ae	Canada	SS	ND	S	ND	42	4Ae, 5Ae, 6Ae(4D, 5D, 6D)
R-Ae 4D	Canada	SS	ND	ND	ND	42	4Ae(4D)
Blue Baart	USA	SS	ND	ND	ND	42	
Wheat+4J	Mexico	SS	ND	ND	ND	44	42 W+2 J

[z]R, resistant; S, susceptible; ND, not determined.
[y]RR, resistant to *Oculimacula acuformis* and *O. yallundae*; RS, resistant to *O. acuformis* but susceptible to *O. yallundae*; SR, susceptible to *O. acuformis* but resistant to *O. yallundae*.
[x]W, wheat chromosome; T, *Thinopyrum* chromosome; Ai, *Th. intermedium* chromosome; Ae, *Th. ponticum* chromosome; St, J, and J^s, St, J, and J^s genome chromosomes, respectively; L, the long arm of chromosome; S, the short arm of chromosome.

and Zhong 2 and 5 were also resistant to *O. yallundae* (Table 1). Using St genomic DNA as a probe, GISH analysis demonstrated that these partial amphiploids contained synthetic genomes that were composed of different combinations of chromosomes from St, J, and J^s genomes of *Th. intermedium* (Chen et al. 2003b). Montana-2 (MT-2) also was derived from a cross between durum wheat and *Th. intermedium* and released as a perennial forage wheat (Schulz-Schaeffer and Haller 1987). Chromosome number in this line varied from 24 to 28 wheat chromosomes and 18 to 36 *Thinopyrum* chromosomes (Jones et al. 1999). Chen et al. (2003a) determined that the alien genome of MT-2 consisted of 8 to 10 St-, 8 J^s-, and 13 J- genome chromosomes. The partial amphiploid MT-2 is resistant to *O. yallundae*, *C. graminum*, WSMV, and the WCM (Cox et al. 2002; Chen et al. 2003a).

GENETIC CONTROL OF RESISTANCE TO SOIL-BORNE PATHOGENS IN THE WHEATGRASSES

In addition to the chromosomes responsible for the traits of interest, partial amphiploids usually contain other wheatgrass chromosomes with undesirable traits, which make them unsuitable for direct use in wheat improvement. The alien chromosomes that are not associated with disease resistance must be removed to eliminate the deleterious traits of the wheatgrasses. Single alien chromosomes can be added to wheat or substituted for the homoeologous wheat chromosomes by crossing wheat with partial amphiploids. Furthermore, chromosome segments conferring disease resistance can be transferred to wheat chromosomes to produce translocation lines, which are more desirable than partial amphiploids in cultivar development. These genetic stocks not only allow the study of the genetic control of resistance to the soil-borne diseases, but they also provide resources for wheat improvement.

Eyespot

Resistance to eyespot was recently identified in *Th. intermedium* and *Th. ponticum* and several wheat-*Thinopyrum* lines (Cox et al. 2002). Among them, SS767 (PI 611939), a W. J. Sando selection, was as resistant to *O. yallundae* as the winter wheat cultivar Madsen, which carries *Pch1*. Based on GISH and C-banding analyses, SS767 was shown to be a 4J(4D) chromosome substitution line and the eyespot resistance of SS767 was therefore associated with chromosome 4J (Li et al. 2004a). Seeds of SS767 have a blue aleurone, which is controlled by gene(s) on *Th. ponticum* chromosome 4J. Several wheat-*Thinopyrum* lines have blue kernels, including T-Ae (*Triticum-A. elongatum*) and R-Ae (Rescue-*A. elongatum*) lines from Canada (Larson and Atkinson 1970), Blue Baart from the US (Morrison et al. 2004), and the wheat-*Th. bessarabicum* (Savul. & Rayss) Á. Löve chromosome 4J addition line from Mexico (Mujeeb-Kazi, personal communication to T. D. Murray). These wheat-alien entries were all screened for their reactions to *O. yallundae* and *O. acuformis*; however, none was as resistant as SS767, which indicates that eyespot resistance is not tightly linked to the blue aleurone trait (Table 1).

In a subsequent study, resistance to *O. yallundae* was detected in wheat-*Th. intermedium* chromosome addition, substitution, and translocation lines, which carry the *Th. intermedium* chromosome $4J^s$ (= 4Ai#2) or the short chromosome arm $4J^sS$ (= 4Ai#2S). Analysis of wheat lines with or without the alien chromosome arm further confirmed the association of eyespot resistance with chromosome arm $4J^sS$ (Li et al. 2005). Chromosome 4E of the diploid *Th. elongatum* (Host) D. R. Dewey [syn. *Lophopyrum elongatum* (Host) Á Löve] and chromosome 4J of *Th. bessarabicum* were not responsible for eyespot-resistance (Li et al. 2004a, 2005). Wheat-*Th. intermedium* lines with chromosome 4Ai#2 or chromosome arm 4Ai#2S also carry gene *Wsm1* for resistance to WSMV (Friebe et al. 1991, 1996; Sharp et al. 2002).

Cephalosporium Stripe

Mathre et al. (1985) determined that wheat-*Th. ponticum* partial amphiploid AT3425 was resistant to *C. gramineum* and its resistance was subsequently transferred to winter wheat (S. S. Jones, X. Cai, and T. D. Murray, unpublished data). The partial amphiploid AT3425 was crossed to Madsen winter wheat and the F_1 was backcrossed to Madsen and increased in the greenhouse. BC_1F_2 plants were grown in field plots inoculated with *C. gramineum* and resistant individuals were selected based on the visual rating of disease severity. Head rows of resistant BC_1F_3 plants were sown in field plots inoculated with the pathogen and resistant individuals were again selected and replanted as head rows. Uniform and resistant BC_1F_4 head rows were harvested and planted in a replicated field trial for comparison with resistant and susceptible control varieties. Ultimately, two lines, J99C009 and J99C010, were selected based on their highly effective resistance and limited yield loss when Cephalosporium stripe was severe. Both J99C009 and J99C010 have 42 chromosomes that pair as 21 bivalents during meiosis. A small terminal wheat-*Th. ponticum* translocation was detected in both lines using fluorescent in situ hybridization. Based on C-banding, chromosome 3D in J99C009 exhibited a similar pattern to the translocated chromosome 3DS?3DL-3TL in AT3425 and Cai et al. (1998) concluded that the translocated chromosome contained a gene(s) that conferred resistance to Cephalosporium stripe. Jones and Murray (unpublished data) screened a full set of wheat-*L. elongatum* disomic substitution lines for resistance to Cephalosporium stripe in a growth chamber study and concluded that chromosomes 2E and 3E had significant positive effects on resistance. Cai et al. (1996) subsequently identified a resistant breeding line, REA 9232, derived from a population in which CI 13113 was in the pedigree. Based on GISH, C-banding, and test crosses of CI 13113 with a full set of Chinese Spring

double-ditelosomic lines, they determined that REA 9232 and CI 13113 are disomic substitution lines in which wheat chromosome 6A has been substituted by chromosome 6Ae from *A. elongatum* [6Ae#2(6A)]. Sib line, REA 9257, which does not contain the substituted chromosome, was susceptible to Cephalosporium stripe based on field tests, which led the authors to conclude that gene(s) on the substituted wheatgrass chromosome are conferring resistance.

Common Root Rot

As a follow up to the research on gene *Crr* by Larson and Atkinson (1981, 1982), the effect of substituting specific group 5 chromosomes in wheat with the homoeologus chromosome 5Ag from *Th. ponticum* or its addition line was studied in the moderately resistant cultivar Cadet and the susceptible cultivar Rescue (Conner et al. 1993). Substitution of chromosome 5B with *Th. ponticum* chromosome 5Ag resulted in an increase in root rot resistance in the susceptible cultivar Rescue, but had no effect on the resistance of Cadet. Substituting either chromosome 5A or 5D with 5Ag or the addition of 5Ag had no impact on the resistance of either cultivar. If resistance genes were present on 5Ag, then the Rescue 5Ag addition line should have been resistant, but this line was as susceptible as Rescue. These results indicate that the root rot reaction of these wheat cultivars is primarily determined by the presence or absence of the dominant allele of gene *Crr*, which conditions a susceptible reaction (Larson and Atkinson 1970). These results are similar to those reported for other diseases, such as Victoria blight (*Cochliobolus victoriae* Nelson) in oats (*Avena sativa* L.) in which susceptibility is the result of sensitivity to a fungal toxin (Markham and Hille 2001). The results of the study by Conner et al. (1993) suggest that the replacement of chromosome 5B in Rescue by 5Ag might have resulted in the loss of toxin receptor sites.

Conner et al. (1989) evaluated a diverse collection of wheat-alien amphiploid and chromosome substitution lines for their reactions to common root rot and reported that the wheat-*Th. ponticum* partial amphiploid Agrotana expressed a high level of resistance to common root rot (Chen et al. 1995). Except for a winter wheat-*Aegilops tauschii* (Coss.) [syn *T. tauschii* (Coss.) Schmal. and *Ae. squarrossa* L.] chromosome 6D substitution line, the other wheat-alien chromosome substitution lines carrying chromosomes from *Ae. tauschii*, *Th. intermedium* and *Th. ponticum* were susceptible to common root rot. The high level of root rot resistance in Agrotana appeared to involve a different defense mechanism from that in the moderately resistant wheat cultivars. *Cochliobolus sativus* was isolated at very low frequencies from the subcrown internodes of Agrotana, but was isolated from most of the subcrown internodes of the moderately resistant, hard red spring wheat cultivar Neepawa, which suggests that resistance in Agrotana inhibits the growth of *C. sativus* at an earlier stage in the infection process than it does in Neepawa.

Despite its excellent disease resistance, Agrotana is not suitable for commercial production because of its poor seedling vigour, small kernel size, blue kernel color, and inferior agronomic performance. Crosses were made with the cultivar Chinese Spring to transfer root rot resistance from Agrotana into a wheat background with improved agronomic traits and to identify the chromosomal location(s) of the resistance gene(s) (Li et al. 2004b). Advanced generation lines (F_8 and F_{11}) with red and blue kernel color from the cross were evaluated for their root rot reactions and resistance. A cytogenetic examination of advanced breeding lines determined that two alien translocated chromosomes were present in all of the root rot resistant, blue kernel-colored lines. Using genomic DNA from *Ps. strigosa* as the probe and wheat genomic DNA as the block, GISH revealed that the root rot resistant lines carried a J-J^s and a J^s-J translocation. The subtelocentric J-J^s translocated chromosome consisted of the long arm of J chromosome and the short arm of a J^s chromosome. The J^s-J translocated chromosome was subcentromeric and composed of an arm of a J^s chromosome and a similar sized arm of a J chromosome; blue kernel color was always associated with this translocation. The two translocated chromosomes appeared to be independent rather than reciprocal translocations. The presence of only the J-J^s chromosome in red kernel-colored lines appeared to increase root rot resistance in comparison to other red kernel-colored lines that lacked alien translocations. However, the highest level of root rot resistance in the advanced generation lines was associated with the presence of both translocated chromosomes. Only a few of the advanced breeding lines expressed the same level of root rot resistance as that observed in Agrotana and none surpassed it (Li et al. 2004b).

Although superior resistance was observed in derivatives of Agrotana (Li et al. 2004b), linkage of the alien chromosomes with adverse quality and agronomic traits restricts their use in wheat improvement programs. Conner et al. (1989) demonstrated that blue kernel color is not always associated with root rot resistance, which suggests that red kernel-colored lines with high levels of root rot resistance might be obtained. Although crossing-over occurs between wheat and alien chromosomes (Cai et al. 1998; Zhang et al. 1996a,b; Chen et al. 2003b), the opportunity of such chromosomal exchange is limited. New approaches are required to stimulate the breakage of alien linkage groups before these resistance sources can be fully utilized.

CONCLUDING REMARKS

Soil-borne diseases such as eyespot, Cephalosporium stripe, and common root rot are important diseases of winter and spring wheat. Since resistance is generally unavailable in common wheat, *Th. intermedium* and *Th. ponticum* are important sources of resistance genes

against these diseases. Wheat-*Thinopyrum* partial amphiploids contain resistance genes to the soil-borne pathogens that have been used to develop wheat lines with resistance based on whole chromosome additions or substitutions and/or chromosome translocations. However, the yield or quality penalty of the wheat-*Thinopyrum* derivatives that results from deleterious wheatgrass genes prevents their extensive use in wheat breeding programs. Additional effort is required to transfer disease resistance from wheat-wheatgrass hybrids into well-adapted commercial varieties. Further manipulation of wheat-*Thinopyrum* translocations to remove the deleterious traits associated with the target traits is feasible with molecular cytogenetic approaches and effective methods for assessing disease severity.

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