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Resistance of the Race-Specific Type

P. L. Dyck
E. R. Kerber
Agriculture Canada Research Station, Winnipeg, Manitoba, Canada

I. Introduction

The terms race-specific and non-race-specific have received much attention in discussions on breeding for disease resistance (Van-

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derplank, 1968; Nelson, 1978; Ellingboe, 1975, 1978). Race-specific or vertical resistance implies resistance to some pathogen isolates and not to others and is relatively simply inherited. Nonspecific or horizontal resistance implies resistance to all isolates of the disease organism and is often polygenically determined. With race specificity as the basis for distinction between the two types of resistance, once a pathogen isolate has overcome nonspecific horizontal resistance it must be reclassified as specific or vertical. Specific resistance has also been defined as resistance to infection or hypersensitive resistance, while nonspecific or horizontal resistance permits infection but reduces colonization or spread of the disease. This implies that intermediate levels of resistance must be horizontal; generally, however, such resistance is race-specific. Nelson (1978) has suggested that the two kinds of resistance are conditioned by the same genes. A host gene may give hypersensitive resistance to some isolates and a rate-reducing resistance to others. Ellingboe (1975) has concluded that "nonspecific resistance is that resistance which has not yet been shown to be specific."

Race-specific resistance is conditioned by the interaction of specific genes in the host with those in the pathogen. The genetic principles underlying this interaction were established by Flor (1955), who, working with flax and its rust (*Melampsora lini: Linum usitatissimum* host–parasite system), showed that whether a cultivar is resistant or susceptible to a physiologic race of the pathogen depends on the genotype for resistance or susceptibility of the host and the genotype for virulence or avirulence of the race of the pathogen. A similar system has been shown to exist for most of the cereal crops and their rust pathogens. Such a gene-for-gene system seems a logical consequence of the coevolution of a host and its obligate parasite in nature, and also in "man-guided" evolution, which has seen the pathogen adapt repeatedly to overcome the resistance of new host cultivars. This ability of the pathogen to generate new virulent forms necessitates an ongoing search for new sources and types of resistance that can be utilized in breeding for disease resistance.

The different types of race-specific resistance, their sources, and their use in developing rust resistant cultivars is discussed. The discussion of race-specific resistance includes hypersensitive and intermediate or moderate seedling resistance, mature- or adult-plant resistance, and resistance due to genes with an additive or cumulative effect. There is an increasing consensus that all types of resistance must be utilized in the development of a breeding strategy to produce cultivars with stable rust resistance.

II. History of Race-Specific Resistance

In North America a severe wheat stem rust epidemic in 1904 stimulated the study of the rust disease and methods of control (Stakman, 1955). In 1905, a breeding program to develop rust resistant spring wheats was initiated in the United States. Also in that year, Biffen (1905) first reported on the Mendelian inheritance of stripe rust resistance in wheat. Another severe North American epidemic in 1916 developed early in the season and attacked susceptible cultivars, including the early-maturing cultivar Marquis. This epidemic further stimulated various aspects of rust research and resulted in the initiation of numerous breeding programs to develop resistant cultivars. Ceres, the first cultivar bred for rust resistance, was distributed in North Dakota in 1926 (Stakman, 1955). Because of its rust resistance and good quality, it was soon grown over a large area. By 1928 a race virulent on Ceres was found. This became the most prevalent race and caused severe epidemics in 1934 and in 1937. This probably was the beginning of the so-called "boom and bust" cycle of resistant cultivars succumbing to new, virulent biotypes of the pathogen.

Several new stem-rust-resistant cultivars were released in the 1930s, including Thatcher, which had resistance derived from Iumillo durum wheat. In subsequent years Thatcher was grown over a large spring wheat area and remained resistant until the early 1950s, when it was damaged by race 15B. However, prior to 1950 it was being replaced by other cultivars that had the Hope or H-44 type of resistance to stem rust and resistance to leaf rust. These cultivars were also damaged by race 15B. In 1954 Selkirk, which has gene *Sr6* plus the H-44 type of resistance, was released. The cultivars released since the 15B epidemic, including Manitou, which has the Thatcher type of resistance plus *Sr6*, have provided good resistance to the North American population of stem rust (Green and Campbell, 1979). Since the release in 1930 of stem rust resistant cultivars in Canada, losses due to stem rust have occurred only during the race 15B epidemics of the early 1950s when a partial breakdown of the Hope and H-44 types of resistance occurred.

In Australia (Watson, 1981), from 1920 to 1950, cultivars with single genes for stem rust resistance were released. The single genes used were *Sr6*, *Sr9b*, *Sr11*, *Sr17* or *Sr36(Tt-1)*. These cultivars had apparent good resistance conferred in each by a single gene and were well received by the farmer. However, the rust population adapted rapidly and became virulent on the newly released cultivars. After 1950 cultivars were released with various combinations of these and several other

genes. Since then the fungus has not been able to combine the matching virulence genes required to overcome the host gene combinations.

According to Roelfs (1978), the reduced frequency of stem rust epidemics in the United States during the past 25 years resulted from the use of resistant cultivars and the removal of the barberry. In the *CIMMYT Review* (International Maize and Wheat Improvement Center, 1981), the comment is made that "stem rust resistance in CIMMYT bread wheats was stabilized in the late 1950s and has been retained." It appears that the pyramiding of primarily specific genes in breeding for resistance to stem rust of wheat has been reasonably successful in several countries. On the other hand, breeding of cultivars with stable resistance to leaf rust has not been as successful. In Canada the first leaf-rust-resistant cultivars (with Hope resistance-*Lr14a*) released in the late 1930s became susceptible by 1945 (Johnson and Newton, 1946). Selkirk, with genes *Lr10* and *Lr16* (the only effective gene), was released in 1953 and was resistant to the prevalent races until approximately 1962. Manitou, released in 1965, and several related cultivars released since then, all with gene *Lr13* for adult-plant resistance, are now almost fully susceptible. Each of these Canadian cultivars had only one effective gene for resistance to leaf rust at the time of release. To further quote from the *CIMMYT Review* (International Maize and Wheat Improvement Center, 1981), cultivars resistant to leaf rust at the time of release "usually become susceptible after two or three years of commercial productions."

In barley a single dominant gene *T* (the Peatland gene) controls resistance to stem rust in both the seedling and adult-plant stages (Andrews, 1956). Although this is the only important source of resistance in barley and is still highly effective in many countries, some virulent strains of rust have been found (Johnson, 1961).

Major specific genes controlling resistance to stripe rust in wheat have been easily overcome by the pathogen. In the United States breeders are using minor recessive genes (Sharp, 1973), while in the United Kingdom "durable resistance," that is, resistance that has remained effective over time, is being used (Johnson, 1978).

III. Types of Specific Resistance

A. HYPERSENSITIVITY

Probably the most common race-specific resistance that has been used in breeding programs is the hypersensitive type. It is charac-

terized by macroscopic lesions at the infection sites. The early collapse and death of the host cells at these sites prevents the further growth of the fungal hyphae. This definition implies "an active resistance mechanism in which the rapid death of the host cells around the point of infection prevents colonization" (Robinson, 1976). However, it has been suggested that the necrotic hypersensitive response does not determine the incompatible reaction but that it is only an incidental stress symptom to the disease (Mayama *et al.*, 1975).

Many of the genes for leaf rust resistance in wheat confer a hypersensitive response, including *Lr1*, the *Lr2* alleles, *Lr3*, *Lr10*, and many of the genes transferred from related species (Browder, 1980). The best known example for wheat stem rust is *Sr6*. Genes for a hypersensitive resistance to crown rust in oats are common, including many of the those found in *Avena sterilis*.

B. IMMUNITY

According to the classification of the types of rust infection (Stakman *et al.*, 1962), an immune response is indicated by the absence of visible lesions on the host plant. It is generally agreed that plants immune to diseases are immune to infection by the pathogen. "An immune plant is a non-host" (Robinson, 1976). Gene *Sr5* has been considered a classical example of a gene for immunity. However, Rohringer *et al.* (1979) showed that when gene *Sr5* is in a Chinese Spring background, macroscopically visible lesions are produced. Furthermore, many of the genes for hypersensitive reaction can produce an immune response to some rust cultures and visible fleck infection types to other cultures, as for example, the *Lr2* alleles (Dyck and Samborski, 1974). Since an immune or hypersensitive reaction depends on host genetic background and/or the rust culture used, the presence or absence of visible lesions does not imply two different types of specific genes. The identified specific genes for immunity may all be hypersensitive.

C. MODERATE

Many of the genes for specific rust resistance give a moderate or intermediate level of resistance. In the seedling stage the resulting infection types can range from type 1 to type 3. With this type of resistance, the pathogen penetrates the host and some rust development occurs before an incompatible reaction becomes apparent. Vary-

ing amounts of urediospores are produced. These genes do not prevent colonization but reduce the rate of spread of the pathogen.

The seedling resistant reaction associated with wheat leaf rust genes *Lr11, Lr16, Lr17, Lr18,* and *Lr30* is of an intermediate type. With wheat stem rust, genes *Sr8,* the *Sr9* alleles, *Sr22, Sr24,* and *Sr33* are of this type. Usually genes that give moderate resistance in the seedling stage give the same type of resistance in the adult stage.

D. ADULT-PLANT RESISTANCE

Resistance that is first apparent in older plants is termed adult- or mature-plant or postseedling resistance. In early literature it was frequently referred to as field resistance. The onset of adult-plant resistance can vary. According to Anderson (1966), the common wheat cultivars Frontana and Klein Aniversario have a postseedling type of leaf rust resistance that appears as early as the third leaf stage. The adult-plant resistance of the cultivar Exchange is not fully expressed until after the emergence of the flag leaf (Samborski and Ostapyk, 1959). Genes for adult-plant resistance may be effective against a wide spectrum of rust races. In fact, Robinson (1976) states that all adult-plant resistance is of the horizontal type. However, race specificity has been found for several of the adult-plant genes for resistance in wheat including *Lr12* and *Lr13.* Gene *Lr22b,* an adult-plant gene in the cultivar Thatcher, gives resistance to only one known North American race. Some of the genes for adult-plant resistance in common wheat are of interspecific origin, including *Sr2* originally transferred from tetraploid Yaroslav emmer (McFadden, 1930) and *Lr22a* from *Aegilops squarrosa* (Dyck and Kerber, 1970).

Based on studies with stripe rust of wheat, Johnson (1978, 1981a) introduced the term "durable resistance" to refer to resistance that has remained effective in cultivars widely grown for many years. This type of resistance, the durability of which can be judged only in retrospect, can be either complexly or simply inherited. A cultivar with durable resistance to stripe rust is Cappelle-Desprez, which has several specific genes for seedling resistance and several for adult-plant resistance. Several, but not all, of the adult-plant factors appear to be responsible for the durable resistance. These factors appear to be associated with chromosome 5BS-7BS. However, recent evidence (Johnson, 1981b) indicates that the genetic background is important to the expression of durability. Hare and McIntosh (1979) have suggested that the Hope-derived adult-plant gene *Sr2* for stem rust resistance may be durable.

IV. Expression of Specific Resistance

A. TEMPERATURE SENSITIVITY

Temperature affects the expression of many genes for disease resistance. Some genes become ineffective at high temperatures, while others become ineffective under low temperatures. A classical example of high-temperature breakdown is that of *Sr6*, a gene for resistance to stem rust in wheat. Many workers have found *Sr6* to be inactivated by high temperatures. Green *et al.* (1960) tested an isogenic line of *Sr6* in Marquis wheat to a large number of races of stem rust under high and low temperatures and observed that resistance was inhibited at high temperature. Loegering and Harman (1969) showed that this breakdown occurs in the range of 20°–23°C. *Lr20*, a gene for leaf rust resistance, has also been shown to be temperature sensitive (Jones and Deverall, 1977). It is partially effective at 26°C and completely ineffective at 30.5°C. Most of the genes for resistance to stem rust in oats are temperature-sensitive (Martens, Chapter 4). The use of some of the genes sensitive to high temperature may be limited to cultivars grown in temperate climates. Gassner and Straib (1931) showed that some cultivars become less resistant to disease under low temperature. Wheat cultivars Malakof, Norka, and Democrat were resistant at 18.7°C but became susceptible at a lower temperature.

Some of the minor additive genes for resistance to stripe rust from wheat cultivar PI 178383 express the greatest resistance at high temperatures, while other minor genes confer more resistance at a lower temperature (Lewellen and Sharp, 1968). By selecting among infected plant populations grown at different temperatures, Sharp *et al.* (1976) obtained plants that were resistant over a wide range of temperatures. Presumably in this way they were able to combine different genes with additive effects.

Temperature sensitivity may assist in identifying certain genes that are masked when in combination with other genes. Thus, temperature sensitivity may be a tool that can be used in combining or pyramiding several resistance genes into one cultivar.

B. GENE INTERACTIONS

When a cultivar has several genes for resistance to the same disease, it is generally assumed that the genes act independently. A cultivar with two genes, each determining a different level of resistance, usu-

ally exhibits the rust reaction phenotype of the most effective gene; the gene conferring the least resistance is masked. The most effective gene is epistatic to those that condition a less resistant reaction. Furthermore, a cultivar with two or more genes will be resistant to all of the rust races to which the genes are effective separately.

However, genes for disease resistance do not invariably act independently. The gene action may be complementary; genes at different loci, or their products, may interact to give higher levels of resistance. Extreme forms of complementary resistance require the presence of two or more genes for the resistance to be expressed. In the oat cultivar Bond, two such complementary genes give resistance to crown rust (Simons *et al.*, 1978). Hosts with either gene alone are susceptible while plants with both genes are resistant to crown rust (Baker, 1966). Martens *et al.* (1981) reported on resistance to stem rust of oats that was conferred by complementary genes involving *Pg-12* and a second gene or a suppressor. This type of complementary action has usually been between recessive genes.

There are numerous examples of genes for disease resistance that interact to give an enhanced level of resistance (Schafer *et al.*, 1963; Knott, 1957; Voronkova, 1980). This complementary interaction, which may be additive, results in a higher level of resistance than that conferred by the genes singly. Dyck (1977) found that PI 58548 has two genes for seedling resistance to leaf rust, one giving a 1+ infection type and the second a 2+. When combined the two genes interact to produce a ;1 infection type. They also interact to produce superior adult-plant or field resistance. More recent studies (Dyck and Samborski, 1982; Samborski and Dyck, 1982) have shown additional interactions between each of two different pairs of genes for seedling resistance, between a pair of adult-plant genes, and between a pair of seedling and adult-plant genes. In general, leaf rust cultures avirulent on both of the combined genes showed the interaction for enhanced resistance, although there were several combinations that showed an interaction with races avirulent on one of the genes and virulent on the other. It should be noted that not all genes that result in intermediate levels of resistance will, when in combinations with other genes, interact to give superior resistance.

Clifford (1975) suggested that specific resistance genes may give a form of residual resistance to pathogen strains possessing the corresponding virulence genes. Such a residual or "ghost" effect has been described for the *Triticum: Erysiphe graminis* system by Martin and Ellingboe (1976) and by Nass *et al.* (1981). Nelson (1981) combined a number of these genes having a residual effect, which he termed "de-

feated" genes, and obtained effective reduction of disease development. Samborski and Dyck (1982) noted that seedlings of the cultivar Columbus with gene *Lr16* gave an incompatible reaction to several cultures of leaf rust that are virulent on *Lr16*. Presumably, an interaction between *Lr16* and *Lr13*, a gene for adult-plant resistance also present in Columbus, resulted in an incompatible phenotype similar to that produced by *Lr16* with avirulent cultures. It is possible that some of the interactions observed may be similar to the "ghost" effect of ineffective genes. It appears that some resistance genes having very little individual effect can interact with or modify other genes to condition a more genetically complex resistance.

There are also examples of nonallelic additive interactions in stem rust of wheat. Knott (1957) noted that resistance genes *Sr10*, *Sr11*, *Sr12*, and particularly *Sr9* were important modifiers of gene *Sr7*. Luig and Rajaram (1972) studied the stem rust reaction of homozygous and heterozygous combinations of *Sr5* and *Sr9b*, *Sr5* and *Sr13*, *Sr6* and *Sr8*, and *Sr8* and *Sr9b*. Additive gene interactions were observed especially when *Sr6* was involved. It would appear that some genes are more sensitive to nonallelic interaction than others.

Samborski and Dyck (1982) made a four-way cross between four backcross lines, each with a single gene resulting in a low level of resistance to leaf rust. Highly resistant lines were obtained from selections made in the F_2 and F_3 generations. These highly resistant selections probably had an accumulation of the genes derived from the four parents.

Sharp and co-workers state that resistance to stripe rust is controlled by two different types of resistance genes (reviewed by Robellen and Sharp, 1978). They found that each of the two cultivars PI 178383 and Chinese 166 had a different dominant major gene. Each gene gave a high level of resistance that was largely unaffected by the environment. However, F_2 plants lacking the major genes segregated for additional genes that gave some resistance themselves or acted as modifiers of the heterozygous major genes. Up to three minor genes were accumulated in lines with good levels of resistance. These and other minor genes for resistance to stripe rust are generally sensitive to environmental influence. There is controversy as to whether these minor genes for resistance to stripe rust are true resistance genes or modifiers in a gene complex that interacts to determine the rust reaction of the host plant. We consider them as resistance genes that are important in the development of cultivars with a genetically complex type of disease resistance.

Since combined genes for disease resistance do not necessarily act

independently but may interact to give a quantitative improvement in resistance, the following should be noted:

(1) The value of combining a number of genes for resistance into one cultivar is emphasized. Complex resistances can be much more effective than anticipated and may be long-lasting.
(2) Attempts to assign genotypes to cultivars and introductions on the basis of testing them with a series of rust cultures may be misleading. Interactions may mask the presence of individual genes.

C. INHIBITORY EFFECTS

Genes conditioning host resistance can also be inhibited or suppressed by nonallelic genes. The resistance to Canadian leaf rust cultures conferred by gene *Lr23* is suppressed by a gene in Thatcher, but this suppression is only partially effective with Australian cultures (McIntosh and Dyck, 1975). Kerber and Green (1980) observed that Canthatch nullisomic 7D is much more resistant to several cultures of stem rust than normal disomic Canthatch. They concluded that chromosome 7DL carries a gene that inhibits the expression of one or more genes for rust resistance present on other chromosomes of Canthatch.

D. BACKGROUND EFFECTS

The genetic background can affect the expression of specific genes for resistance. Several such genes, particularly those conferring resistance to stem rust and leaf rust of wheat, have been backcrossed into different cultivars. Alleles *Lr2a*, *Lr2b*, and *Lr2c* were backcrossed into the cultivars Thatcher, Prelude, and Red Bobs; they were most effective in the Thatcher background, intermediate in Prelude, and least effective in Red Bobs (Dyck and Samborski, 1974).

A gene for resistance may be dominant in one genetic background and recessive in another. Consequently, the susceptible parent in a cross can influence the degree of dominance of a gene as has been shown by Anderson (1966) for gene *Lr2*.

The reaction conferred by a gene may be dominant relative to one race of a pathogen and recessive to another (Knott and Anderson, 1956; Lupton and Macer, 1962). It has been suggested that this phenomenon may be due to two closely linked genes, the expression of one being dominant and the other recessive. Hooker and Saxena (1971) tested

this with gene *Rp3* in maize. By using a linked marker they were able to screen large testcross populations for crossovers. Since none were detected, the two supposed component genes of *Rp3*, one dominant and one recessive, would have to be less than 0.02 map units apart.

E. ALLELISM

Most genes for disease resistance are inherited independently of each other. When two or more genes are on the same chromosome, they may show varying degrees of linkage. In some cases the genes are either tightly linked or they are alleles, that is, they are at the same locus on a chromosome. Such tight linkage, or multiple allelism may restrict the number of genes that can be combined into one cultivar. In theory, a self-pollinated crop can be homozygous for only one gene at a locus. However, at several loci that were assumed to be multiple alleles for disease resistance, two or more of the alleles were recombined in coupling linkage, and they then behaved as one gene. In oats, stem rust resistance genes *Pg-3* and *Pg-9*, assumed to be alleles, have been combined (Koo *et al.*, 1955). Similarly, in wheat the two alleles at the *Lr14* locus have been combined (Dyck and Samborski, 1970).

Saxena and Hooker (1968) suggest that the *Rp1* locus in maize, which may have as many as 14 different alleles for resistance to *P. sorghi*, consists of a series of tandem duplications of the original gene. These duplications have gradually differentiated to give resistance to different races of the rust. They suggest that the different alleles may consist of one or more combinations of the original gene and/or its modified duplicates. They also suggest the possibility of synthesizing a gene at one of these complex loci (e.g., the *Rp1* complex has a large number of alleles with crossover values ranging from 0.10 to 0.37%) that would confer resistance to many cultures by systematically recombining several of the alleles.

Mayo and Shepherd (1980), using a modified cis–trans test for functional allelism, found that several of the *M* alleles for resistance to flax rust were in fact separate, closely linked loci. They combined two of the *M* genes in the coupling phase where each of the genes functioned independently. Thus, it may be possible to combine three or more of the *M* genes in coupling to construct a complex resistance genotype.

Some alleles at a locus, or closely linked genes, appear to be functionally related as they exhibit a similar phenotype. In wheat, each of the two alleles at the *Lr14* locus for resistance to leaf rust gives a mesothetic infection type but to different races (Dyck and Samborski,

1970). Also in wheat, each of the different alleles at the *Sr9* locus for resistance to stem rust conditions a type 2 infection (Roelfs and McVey, 1979). In oats the alleles or functionally related genes for resistance to stem rust, *Pg-3* and *Pg-9*, also give resistance to crown rust (McKenzie *et al.*, 1968).

Allelism, together with a scarcity of resistance genes, has been a particular problem in the development of stem rust resistant oat cultivars. Until recently it was assumed that there were only seven genes for resistance at three loci. It was suggested that these might involve three chromosomes belonging to a homoeologous series (McKenzie *et al.*, 1970). Two of the alleles at one locus were combined by Koo *et al.* (1955), who suggested that this was a complex locus consisting of pseudoalleles. Several additional genes at different loci have more recently been found (Martens *et al.*, 1980).

V. Sources of Specific-Type Resistance

Due to the continual evolution of rust pathogens to form new and virulent biotypes, there must be a constant search for germ plasm possessing resistance to the various cereal rusts. Three natural sources of the specific type of resistance are available: (1) cultivars that have been produced since the advent of modern plant breeding; (2) land races or primitive cultivars that predate the advanced cultivars; and (3) the relatives—both cultivated and wild—of the crop species under consideration. Although listings of germ-plasm collections maintained at various institutions are available (Ayad *et al.*, 1980; Creech and Reitz, 1971), relatively few details are known of specific accessions in regard to rust resistance, geographic origin and availability of seed. A fourth potential source of disease resistance is through the induction of mutations by various mutagenic agents.

A. WITHIN HOST SPECIES

The most obvious and immediate source of resistance would be local and international collections of old and of contemporary cultivars and breeding stocks of the crop to be improved. This source would be particularly appropriate for new breeding programs. Should suitable resistance not be identified in such material, the search can then be extended to primitive cultivars or so-called land races. This should

include a thorough search of comprehensive collections derived from the geographic centers of diversity (Leppik, 1970; Zohary, 1970). Because of their great genetic diversity, primitive cultivars and land races are more likely sources of new resistance genes than material derived from breeding programs. The known genes for rust resistance in most of the cereal crops are now being utilized on an international scale; consequently, few new genes for resistance can be expected from advanced breeding stocks.

B. RELATED CULTIVATED SPECIES AND WILD SPECIES

Plant breeders and pathologists are increasingly aware of the necessity to broaden the genetic pool from which effective sources of resistance can be drawn (Hooker, 1977; Knott, 1979; Krull and Borlaug, 1970; Moseman *et al.*, 1979; Watson, 1970). Ample evidence indicates a significant reservoir of resistance is available among the relatives of cultivated cereal crops (Dinoor, 1977; Gerechter-Amitai and Loegering, 1977; Kerber and Dyck, 1979; Pasquini, 1980; The, 1976). These have usually been the last resort because of problems in transferring such resistance, which is generally of the race-specific type, to commercially acceptable cultivars.

The earliest examples of the transfer of rust resistance to a cereal crop from related species trace to the investigations of American workers in the 1920s and 1930s. Hayes *et al.* (1920) produced the stem-rust-resistant common wheat cultivar Marquillo from a cross between susceptible Marquis common wheat and resistant Iumillo durum wheat. Later, McFadden (1930) crossed the highly disease-resistant tetraploid wheat Yaroslav emmer with Marquis, from which the two selections Hope and H-44 were developed, which at that time were highly resistant to stem rust and leaf rust. These two strains appear in the pedigree of numerous contemporary common wheat cultivars. Following these pioneering successes, this field of investigation was largely neglected.

In the past two to three decades, attention has again been directed toward the exploitation of the relatives of the cereal crops as sources of disease resistance. This renewed interest may be attributed to (1) the acquisition of much significant knowledge on the phylogenetic and cytogenetic relationships between cultivated cereal crops and their related species, (2) the development of techniques such as embryo culturing and the postpollination application of growth regulators to enhance seed development of interspecific crosses, and (3) the develop-

ment of cytogenetic stocks and methodology that permit the transfer of alien genetic material between normally nonhomologous or between homoeologous chromosomes of the donor and recipient crop species (Feldman, 1979; Knott, 1971; Knott and Dvorak, 1976; Riley and Kimber, 1966; Riley *et al.*, 1968; Sears, 1956, 1972, 1981; Stalker, 1980).

The effective utilization of the cultivated and wild relatives of the cereal crops must begin with the acquisition of a substantial collection of the related species to be surveyed. It should be emphasized that the stocks or accessions of the species to be evaluated should be representative of the geographic regions from which maximum variation for resistance can be expected—the centers of genetic host diversity (Harlan and Zohary, 1966; Rajhathy and Thomas, 1974; Zohary, 1970).

In the following discussion on the transfer of rust resistance to cultivated cereal crops from their relatives, the methodology and examples are taken primarily from wheat (*Triticum*). Most of the significant advances have been made within this genus, although some of the techniques are also applicable to barley, rye, and particularly oats, which, like common wheat and durum wheat, belong to a polyploid complex of species.

The relatives of common wheat ($2n = 42$ = AABBDD) that have been utilized to a limited extent as sources of disease resistance include the immediate tetraploid ($2n = 28$ = AABB) and diploid ($2n = 14$ = AA) progenitors within the wheat genus, the closely related genus *Aegilops*, *Secale*, and some species of *Agropyron*. The cytogenetic procedure to be used for the transfer to common wheat or durum wheat of genetic material from these related species is primarily dictated by their phylogenetic affinity and genomic constitution (Feldman, 1979; Knott and Dvořak, 1976; Riley and Kimber, 1966; Sears, 1981). The procedure most likely to prove successful depends on whether the chromosomes (genomes) of the donor species possessing the resistance are homologous or nonhomologous with those of the recipient wheat (Feldman, 1979; Knott and Dvořak, 1976; Sears, 1972, 1981).

1. *Transfer of Resistance Involving Homologous Chromosomes*

When the various related species have a genome(s) that is homologous with at least one of the genomes of cultivated common wheat, transfer of resistance is relatively simple. These species include the immediate tetraploid (AABB) and diploid (AA and DD) progenitors of the cultivated wheat. Because normal chromosome pairing and genetic

recombination occurs between homologous genomes in hybrids produced from crosses between wheat and these species, the transfer of genes is possible providing crossability and F_1 sterility barriers are not a hindrance.

a. Direct Crosses. Where the parents have a genome in common, the transfer of disease resistance to cultivated wheat from a related species by conventional crossing and selection usually presents little difficulty. Examples of this procedure were the transfer of the stem rust resistance gene *Sr22* from diploid wheat to both tetraploid (Kerber and Dyck, 1973) and hexaploid wheat (The, 1973). Similarly, stem rust resistance was transferred directly from tetraploid to hexaploid wheat (Hayes *et al.*, 1920; Kerber and Dyck, 1973; McFadden, 1930; Knott, 1979). In these hybrids, pairing between the common genome(s) of the donor species and recipient wheat is usually complete or nearly so. Partial or complete sterility of the tetraploid × diploid and hexaploid × diploid hybrids can be overcome by backcrossing to the respective tetraploid and hexaploid parental cultivars.

b. Bridge Crosses. Bridge crosses may be used where the transfer of genetic material, usually between different levels of ploidy, is difficult or impossible by direct hybridization. Kerber and Dyck (1973), for example, transferred the gene *Sr22* first from diploid (AA) to tetraploid (AABB) and then to hexaploid wheat (AABBDD). Although the direct cross between some genotypes of hexaploid and diploid wheats can be made, it is often difficult and the hybrid is sterile.

Another bridging method applicable to the transfer of genetic material to both tetraploid and hexaploid wheats is with natural amphiploids that have the A genome in common with these two wheats or the D genome in common with hexaploid wheat. The amphiploid selected for resistance is crossed to wheat, and the partially fertile hybrid is then backcrossed several times to the wheat cultivar to obtain meiotically stable, fertile plants. Natural amphiploids that could be used in this manner include *T. timopheevii* (AAGG), *Ae. cylindrica* (CCDD), *Ae. ventricosa* (DDMvMv), and others (Feldman, 1979). Similar procedures can be employed to transfer genes to the polyploid wheats from synthetically produced amphiploids having one or more genomes in common with wheat. Kerber and Dyck (1969) and Dyck and Kerber (1970) transferred two genes for leaf rust resistance, *Lr21* and *Lr22a*, and a gene for stem rust resistance, *Sr33* (Kerber and Dyck, 1979), from *Ae. squarrosa* ($2n = 14 =$ DD) to common wheat by first producing synthetic hexaploids ($2n = 42 =$ AABBDD) from the hybrid

between tetraploid wheat and the resistant *Ae. squarrosa* strains. The resistant synthetic hexaploid was then crossed and backcrossed several times to a common wheat cultivar to incorporate the resistance genes into a suitable genotypic background.

2. *Transfer of Resistance Involving Nonhomologous Chromosomes*

The transfer of genetic material to cultivated wheat from more distantly related species poses considerable difficulty because their chromosomes have differentiated from those of wheat to the extent that no pairing and genetic recombination between them normally occurs. Nevertheless, cytogenetic procedures are available by which genetic exchange can be induced between wheat chromosomes and those of related species.

a. Transfer of Resistance by Induction of Homoeologous Chromosome Pairing. Numerous species related to wheat have a genome(s) that is homoeologous (genetically and structurally similar) with those of common wheat. The three genomes of wheat themselves are homoeologous, having presumably descended from a common evolutionary ancestor. The genetic control or suppression of homoeologous chromosome pairing is largely due to a gene, *Ph*, on chromosome 5B (Wall *et al.*, 1971b). This gene normally prevents homoeologous pairing not only within wheat but also between homoeologues of wheat and of related or alien species when combined in hybrids. Three cytogenetic procedures have been developed by which the 5B effect can be nullified, thereby inducing pairing and recombination between homoeologues of wheat and related species in hybrid material.

Pairing between an alien chromosome and a homoeologue of wheat can be induced by crossing monosomic 5B of wheat with the species from which resistance is to be transferred. Pairing between the alien and wheat homoeologues will occur in F_1 plants that are deficient for chromosome 5B. However, the hybrids produced are highly sterile and the chance of inducing the desired gene transfer is very low when only a few seeds are produced on 5B-deficient plants. To overcome the high sterility to some extent, nullisomic-5B–tetrasomic-5D can be used in place of monosomic 5B. The use of the 5B-deficient method can be simplified and made more efficient by first producing alien substitution or alien addition lines in which the alien chromosome bears the gene(s) for resistance. The utilization and variations of the chromosome 5B-deficient procedure have been given by Riley and Kimber

(1966) and Sears (1972, 1981). This method was successfully used in the transfer to common wheat of rust resistance from rye (Joshi and Singh, 1979) and leaf rust resistance from *Agropyron elongatum* (Sears, 1972, 1973).

Another approach to the induction of homoeologous pairing is to suppress, rather than delete, the activity of *Ph* on chromosome 5B by adding the genome of certain forms of *Ae. speltoides* or *Ae. mutica*. Riley *et al.* (1968) applied this technique for the transfer to common wheat of stripe rust resistance from *Ae. comosa*. They produced a wheat stock with a disomic addition of the *comosa* chromosome bearing the gene for resistance which was crossed to *Ae. speltoides* to induce pairing between the *comosa* chromosome and its wheat homoeologue. Several backcrosses to common wheat eventually resulted in the cultivar Compair in which chromosome 2D carries a segment from the *comosa* chromosome that conditions resistance. Dvořak (1977) directly transferred genes for leaf rust resistance from *Ae. speltoides* to common wheat by taking advantage of the ability of this diploid to suppress *Ph* and thereby permit homoeologous pairing in the wheat–*speltoides* hybrid.

The most desirable situation for the induction of homoeologous pairing is the use of mutants of the *Ph* locus such as *Ph1a* and *Ph1b* obtained by Wall *et al.* (1971a) and Sears (1977, 1981), respectively. When used in crosses, these mutants have the advantage over the nullisomic 5B procedures by decreasing the amount of aneuploidy in the progeny of the hybrids, increasing fertility, and allowing recombination to occur between 5B and alien homoeologues.

b. Transfer of Resistance by Induced Chromosome Translocations. The use of irradiation to induce translocations for the transfer of disease resistance to a crop species from a related species involving nonhomologous chromosomes has been applied with considerable success since the initial procedure developed by Sears (1956) was employed to transfer leaf rust resistance from *Ae. umbellulata* to wheat. Briefly, Sears produced a line in which an isochromosome of *umbellulata*, bearing the resistance gene, was added to the wheat complement. Irradiation of this line and pollination of the cultivar Chinese Spring with pollen from the irradiated plants resulted in a 42-chromosome derivative in which a segment of chromatin bearing the resistance gene *Lr9* was translocated to chromosome 6B. Since then this procedure or modifications of it (Knott, 1971; Riley and Kimber, 1966; Sears, 1972) have been employed to induce transfers to wheat chromosomes of genetic material from nonhomologous chromosomes of related spe-

cies, particularly from *Agropyron* and *Secale*. Transfers of resistance to the rusts so induced include, in addition to *Lr9*, leaf rust resistance genes *Lr19*, *Lr24*, and *Lr25* and the stem rust resistance genes *Sr24*, *Sr25*, *Sr26*, and *Sr27* (McIntosh, 1973, 1979).

Now that various cytogenetic procedures have been devised by which the chromosome 5B pairing activity can be nullified, so inducing pairing and genetic recombination between homoeologous chromosomes of wheat and those of related species, it is likely that the use of irradiation will be limited to crosses in which homoeologous association does not occur. Transferring genetic material by induction of homoeologous pairing has the advantage of restricting the size of the alien chromosomal segment and reducing the possibility of introducing undesirable linkages that usually accompany most irradiation-induced translocations.

A review of the literature on the transfer of rust resistance to cereal cultivars from related species reveals several problems and difficulties may be encountered. Some of these with related aspects are as follows.

1. Resistance transferred to crop cultivars from related species may be linked with undesirable agronomic or quality characters (Knott, 1971; Knott and Dvořak, 1976, 1981).
2. Resistance transferred from a lower to a higher level of ploidy is often decreased or "diluted," as expressed by infection type (Dyck and Kerber, 1970; Kerber and Dyck, 1969, 1973, 1979), and in some cases may be completely suppressed (Kerber and Green, 1980; The and Baker, 1975).
3. Resistance of the race-specific type derived from alien species probably will be no more durable than that available within the cultivated crop. Virulent strains of the pathogen have been known to overcome this type of resistance (Knott, 1971; Johnson and Gilmore, 1980; Parlevliet, 1981).
4. Race-specific genes for resistance identified in the relatives of cereal crops are likely to be different from those known in cultivars of the crop (Kerber and Dyck, 1969, 1973, 1979; Knott, 1979).

C. INDUCED MUTATIONS

The interest shown 15–25 years ago in the use of mutagenic agents for the induction of rust resistance in cereal crops has waned in recent

years. This can be attributed in part to the meager improvements obtained for the substantial efforts used to test and screen large populations. This applies particularly to polyploid crops such as wheat and oats in which the observable mutation rate is substantially reduced by the buffering effect of duplicated genetic material. Nevertheless, some successes have been reported. In oats, Simons and Frey (1977) detected mutants having greater tolerance to crown rust as a result of ethyl methanesulfonate treatment. Skorda (1977) obtained wheat mutants induced by irradiation that were resistant or partially resistant to stem rust and stripe rust. Similarly, Borojevic (1979) observed increased resistance to leaf rust among mutant lines derived from irradiation of susceptible wheat cultivars. It is noteworthy that in wheat none of the race-specific genes for stem rust resistance (*Sr* genes) or leaf rust resistance (*Lr* genes) catalogued by McIntosh (1973, 1979) have originated from mutagenic treatments. This observation also applies to the major genes for stem rust resistance (*Pg* genes) and crown rust (*Pc* genes) resistance in oats (Simons *et al.*, 1978).

D. DETECTION AND EVALUATION OF SOURCES OF RESISTANCE

The evaluation of a large and diverse collection of cereal-crop germ plasm may be divided into two phases: initial screening of accessions for resistance to prevalent races or biotypes of the pathogen, and the identification of resistance genes and their relationships to other genes of the same source species and to known resistance genes of the host crop. Effective procedures have been developed for detecting resistance of the race-specific type (Dinoor, 1977; Parlevliet, 1981). Preliminary screening and selection can be done in field nurseries, where large populations can be tested for resistance to natural occurring inoculum, or under an artificially created epidemic to a race or to a composite of biotypes. More reliable information can be obtained by controlled testing of plants to specific pathogen cultures under greenhouse conditions. Some information of genetic variability among accessions of a crop or related species can be gained by noting the differential reactions expressed when tested to a series of critical cultures of the pathogen. This will often permit the classification of the material into groupings based on similarities and differences in reaction (phenotypes) to the rust cultures. These differential reactions can be related to those expressed by host cultivars with known race-specific genes,

as, for example, the leaf rust (*Lr*) and stem rust (*Sr*) resistance genes in wheat. This information may indicate whether the resistance being evaluated is different from that already available.

Computerized methods have been devised in which data on host reaction to specific pathogen variants are processed to provide an indication of the host genotype (Dinoor, 1977; Gerechter-Amitai and Loegering, 1977). This information is useful for categorizing new sources of resistance from which selections can then be made for detailed genetic study. The precise identification of genes for race-specific resistance in the host involves crosses and genetic analysis based on the classification of segregating generations into phenotypes as determined from the reaction of plants (F_2 backcross or F_3 families, for example) to specific pathogen cultures. Although the genetic identification of a source of resistance is not essential prior to its use, this information is important for the strategic employment of race-specific genes. In addition, comparative genetic analysis involving known genes for resistance avoids wasteful duplication in the utilization of identical types of resistance.

VI. Use of Specific-Type Resistance

A. BREEDING STRATEGY

The early years of breeding for resistance to the cereal rusts were filled with great hopes; cultivars with single genes for resistance were released, but virulent strains of the rust organism present in low frequency would then increase rapidly and spread over an entire area devoted to the new cultivar. Thus, cultivars with single-gene specific resistance were in most instances short-lived.

Gradually it became apparent that a crop required greater diversity in genetic resistance if more stability to disease resistance was to be achieved. Watson and Singh (1952) were among the first to propose the use of multiple-gene resistance to control stem rust. They suggested the development of cultivars with pairs of genes, each giving resistance to all of the prevalent races. If the origin of new pathogen races is by mutation only, which they indicated is the situation in Australia, new virulent strains can arise only through simultaneous or stepwise mutations at all the corresponding loci in the pathogen. The probability of this occurrence would be much less than that of single-gene mutations.

Multigene resistance cannot be expected to last when the genes are also exposed individually as single-gene resistance in other cultivars, due to the selection for strains virulent on the single-gene cultivars. In addition to the rate of mutation toward virulence, other factors that influence the duration of effectiveness of genetically complex resistance are the size of the rust population, during both the growing and the overwintering seasons, and the degree of selection pressure exerted. Multigene resistance may also be overcome more readily when genetic variability in the rust fungus originates through either sexual or somatic recombination of several mutant loci present in different rust strains. However, wheat cultivars with a more complex resistance appear to have stabilized the stem rust population in several regions including North America and Australia.

Multigenic resistance should not be considered permanent; eventually new rust strains can be expected to appear. Reference is frequently made to Caldwell's observation (1968) that polygenic mature-plant resistance derived from Chinese Spring was gradually overcome by the leaf rust organism over a 5-year period. Polygenic resistance was inferred from an observation of "a continuous array of infection severities from 0 to 100%" in F_3 populations. As few as two interacting adult-plant genes could give such a distribution (P.L. Dyck and D. J. Samborski, unpublished observations). Chinese Spring is reported to have gene *Lr12* for mature-plant resistance on chromosome 4A (McIntosh and Baker, 1966; Dyck and Kerber, 1971) plus one or two modifiers or additional genes for adult-plant resistance (Dyck and Kerber, 1971). Piech and Supryn (1978) found a second gene for adult-plant resistance on chromosome 7D. Failure of resistance due to only two genes is not a good example of breakdown of polygenic resistance.

The breeder can usually combine or pyramid several specific resistance genes into a cultivar. In wheat, most identified genes for resistance have been located on specific chromosomes and assigned gene symbols (McIntosh, 1973). Their typical phenotype and effectiveness is known (Browder, 1980; Roelfs and McVey, 1979). Such information permits planning as to which genes can be combined without the interference of linkage problems. Allelism may limit the number of host genes that can be combined; however, in the parasite there is no clear evidence for allelism of virulence genes, although linkage of virulence genes has been reported (Samborski and Dyck, 1976; Statler, 1979; Lawrence *et al.*, 1981).

If the use of specific genes in a breeding program is to be successful, the rust population must be surveyed regularly so that changes in virulence can be detected. A thorough survey should detect the ap-

pearance in the rust population of genes for virulence corresponding to the specific host genes combined in a cultivar. Also, surveys may detect new virulent strains several years before they reach epidemic proportions and so allow time for cultivar replacement. In such a case the cultivar should be replaced by one with a different combination of resistance genes before the pathogen can cause appreciable damage.

Combinations of specific genes permits the exploitation of additive or complementary interallelic interactions that have in some cases been shown to enhance the level of resistance. It also allows the utilization of any existing residual or ghost effect of specific genes. A cultivar with a combination of several effective and ineffective specific genes may result in complex and stable resistance. Leaving the ineffective specific genes in a commercial cultivar is contrary to the suggestion (Person *et al.*, 1976) that they should be removed from exposure to the rust population. Consequently, if stabilizing exists (Vanderplank, 1968), the corresponding virulence gene would disappear from the rust population, and the host gene could then be recycled.

B. BREEDING METHODS

When using the pedigree method of breeding, single crosses usually consist of a well adapted but rust-susceptible cultivar and a rust-resistant but frequently poorly adapted cultivar. The genetic diversity for rust resistance is then limited to that available from the one parent. To obtain greater genetic diversity, a double cross can be made to combine resistance from different sources. However, the likelihood of obtaining well adapted and highly rust resistant selections from a double cross of an adapted and three unadapted rust-resistant parents is poor. To increase the probability of obtaining desirable, highly resistant selections, double crosses can be made between advanced lines derived from other crosses, each with different types of rust resistance. These lines may have compensating weaknesses and strengths in other important characters. The breeding strategy employed at the International Maize and Wheat Improvement Centre (CIMMYT) emphasizes pedigree breeding with multiple or double crosses that lead to a rapid increase in genetic diversity (Dubin and Rajaram, 1981).

In a pedigree breeding program where rust resistance is a major concern, the parents should be carefully chosen. Ideally they have different types of genetic resistance that can be combined under the selection

procedures available. The potential parents should be evaluated for resistance under field conditions and, if possible, to specific races in the greenhouse. If the genetics of resistance is not known, parents with different ancestry should be chosen.

Rust resistance is a highly heritable character that is usually selected for in early generations. However, since resistance is frequently dominant or partially dominant, further selections must be made in later generations to ensure that homozygous lines are obtained. If the genes for resistance being used are additive or interacting, selection for resistance should also be made in later generations when more genes are homozygous and interactions are more apparent. In the wheat breeding program at the Winnipeg Research Station (Green and Campbell, 1979), F_2, F_4, and F_6 generations and all lines being yield-tested are grown in the rust nursery. Thus, lines are selected over several years and subjected to repeated artificial rust epidemics under different environments.

The bulk method of breeding differs from the pedigree in that segregating generations, usually F_2 to F_6, are grown in bulk and exposed to a disease epidemic. Mechanical separation of seed according to size eliminates the smaller, shrunken seeds of susceptible plants, aiding in the selection for rust resistance. With this method, many crosses can be handled with a minimum of labor. Selection over several years can result in highly resistant cultivars.

The backcross method is frequently used to improve or correct a defect, such as susceptibility to rust, in an otherwise well adapted and high-yielding cultivar. With this method the choice of the recurrent parent is very important, since an increase in the inherent yielding ability of the cultivar is not expected. The superior adaptibility of some cultivars may be due to genetic heterogeneity, which is not always recovered with backcrossing. Thus, it is important to use a number of plants of the recurrent parent in each backcross, particularly in the final one. To develop a cultivar with multigene resistance, several different known genes must be transferred into the recurrent parent in separate backcross series. When completed, the backcross lines are intercrossed and selections made for lines with various gene combinations.

Mac Key (1959) has described a modified convergent backcrossing scheme in which an adapted cultivar is crossed with four different resistant sources and the F_1 of each combination is backcrossed to the adapted cultivar. Double crosses are then made between resistant selections from the four backcrosses. In subsequent segregating genera-

tions, selections for maximum resistance and adaptibility are made. Since many breeding programs must meet some specific requirements—i.e., milling and baking or malting quality or adaptation—additional backcrosses can be made to the adapted cultivar before the double crosses are made. If selection methods are available that ensure the selection of lines with a number of genes for resistance, modified backcrossing may permit selection for increased yield and a complex resistance.

The use of male-sterile facilitated recurrent selection is a way of combining several different additive or complementary genes from a large number of potential sources (Driscoll, 1981; Ramage, 1977; Sharp, 1979). A backcrossing scheme using male sterility can be used to transfer these additive genes into an adapted cultivar.

Regardless of the breeding method used, the segregating populations should be subjected to a timely rust epidemic. Reliance on naturally occurring epidemics is usually not adequate. The breeding material is interspersed with spreader rows of highly rust-susceptible plants that are inoculated with a mixture of rust races. The rust nursery should be planted at a time and place so that the appropriate stages of plant development will coincide with conditions most favorable for rust development. If natural dew formation is not adequate, sprinkler irrigation may facilitate infection and uniform spread of the disease. The inoculum used should be representative of the indigenous rust population. Green and Campbell (1979) used "the most prevalent and widely virulent races available."

The development of a cultivar with complex genetic resistance may be obtained to some extent by combining different types of resistance. For example, specific genes, either the hypersensitive or rate-reducing type, could be combined with genes for adult-plant resistance or with additive, interacting genes. If resistance considered as durable exists, it could perhaps be combined with specific resistance genes. Johnson (1981a) suggests that to be certain that the final selections possess durable resistance, all specific gene sources present in the breeding program should be susceptible to the rust culture used to test for the presence of durable resistance.

Selection for seedling resistance can also be done in the greenhouse, particularly during the off season or winter, or on subsamples of lines grown in field nurseries. Preferably, rust races with known genes for virulence should be used in seedling tests so that lines can be selected for combinations of known specific genes. In Australia, a National Rust Control Program has been established (Watson, 1977) whereby a

central laboratory screens wheat lines submitted by participating breeders from throughout the country. On the basis of seedling and adult-plant tests, selections are made and breeders are advised on the types of resistances that are present.

Various methods have been proposed to verify or detect the presence of specific genes when they are combined in advanced lines derived from a pedigree breeding program or from different backcross lines.

1. Differential rust cultures obtained from disease surveys, or through mutagenic and genetic studies can be used; however, care should be taken so that virulent cultures do not escape into commerical fields. The establishment of an international exchange would be desirable whereby testing for combinations of genes in breeder's lines could be done in another country with isolates from a different rust population.
2. Enhanced resistance resulting from gene interactions may make it possible to detect various gene combinations. Since additive genes do not generally show epistasis, several of them could be accumulated in a cultivar by merely selecting for the most resistant lines.
3. Genes conferring different infection types can be combined by selecting plants with the lower infection type from lines that are segregating for low infection type and are homozygous for the higher infection type.
4. If one of the genes is temperature-sensitive, manipulating the temperature may make it possible to detect a gene(s) hidden by a temperature-sensitive gene.
5. Seedling resistance genes can be backcrossed into recurrent parents with adult-plant resistance, thus combining genes for both seedling and adult resistance.
6. A member of a pair of linked specific genes may be used to select for the other; i.e., since *Sr24* and *Lr24* are inherited as a unit, backcrossing *Lr24* into a line already possessing *Sr26* will simultaneously result in combining *Sr24* and *Sr26*.
7. Flor and Comstock (1971) developed three-gene lines in flax by first developing two-gene lines with one gene in common, for example, *LLm3m3N1N1* and *llM3M3N1N1*. From an intercross of these two lines, selection would be made only for genes *L* and *M3*.
8. If none of the above options is available, resistant F_3 lines from intercrosses of several backcross lines or breeding lines, each with

different genes, may have to be test-crossed to lines with the genes being used or to a susceptible tester, as outlined by Johnson and Gilmore (1980).

VII. Conclusions

The development of cultivars with race-specific rust resistance usually involves the application of routine plant-breeding methods. However, because of the continual evolution of new races or biotypes of the pathogen, the strategy of utilizing this type of resistance is not routine or straightforward. Although it is generally agreed that the use of cultivars with single-gene, race-specific resistance should be avoided, the total abandonment of major resistance genes, as proposed by some pathologists and breeders, is unwarranted. The key to the development of cultivars with long-lasting resistance is diversity—genetic diversity in the types of resistance, and diversity in their strategic deployment. This would include a combination of race-specific with non-race-specific resistance. Strategies to increase the durability or longevity of resistance include the pyramiding of genes into a cultivar, multiline cultivars, regional deployment of different resistance genes, and the diversification of types of resistance among cultivars. No single overall strategy can be recommended. The primary objective of the various approaches and strategies should be to reduce the selection pressure for virulence in the pathogen.

Although a sufficient number of race-specific genes appears to be available at present for effective control of the rusts, for the future, greater emphasis will have to be placed on the exploitation of resistance known to exist among the relatives of the cereal crops.

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