

## Genetics of Slow Rusting in Cereals

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Slow rusting in cereals has been known for many years (5), but its value was not fully appreciated until the great epidemics of stem rust devastated the bread wheat and durum wheat crops during 1953–1954 (33). During the last 10 yr, interest in slow rusting has increased greatly because plant breeders and pathologists are beginning to realize that it may be a more stable type of resistance than that characterized by different infection types.

**Methods of Recognition.** Slow rusting is recognized by observing rust development during an epidemic. In individual plants, the uredia may or may not be associated with chlorosis, and the infection types indicate that the plants are susceptible in all stages of growth. Slow rusting is determined by comparing disease progress curves (31) or rust severity data collected during the logarithmic stage of development of an epidemic (1,6,12,13,16,18, 29). The comparison of disease progress curves may be facilitated by calculating the area under the curve (31), the rate of disease development (26), or the logit line intercept (26). Loegering et al (12) developed an indexing method for estimating slow rusting. Most studies are done with a limited diversity of inoculum in natural epidemics or by inoculating plants with a few races, but Moore and his associates (17) have selected slow-rusting lines of oats in a buckthorn nursery where many races of the crown rust pathogen were present each year.

Slow rusting has been reported in: *Avena byzantina* (13); *A. sativa* (17); *Dactylis glomerata* (12); *Hordeum vulgare* (8); *Lolium multiflorum* (36); *Panicum virgatum* (3); *Triticum aestivum* (1,4,34); and *Zea mays* (7,11,29).

**Components.** There is a relationship between slow rusting and resistance components that operate after penetration of the host plants (24). The postinfection components most frequently studied are: length of the latent period; frequency of infection; size of uredia; duration of sporulation; and quantity of spores produced

(1,6,16,19,20,32). Obviously, these are the components that can be easily studied; they could be subdivided and others could be suggested. For example, Moore (17) noted that in some lines of oats, telia formed early, which reduced the length of the period of urediospore production.

**Stability.** Slow rusting has been a stable trait over relatively long periods of time; however, stability in the presence of many races of pathogen is a better test of stability. Thatcher and Lee spring wheats have been known to rust slowly with *P. graminis* f.sp. *tritici* for 55 (5) and 30 yr, respectively. The oat cultivar Red Rustproof has been known to rust slowly with *P. coronata* f.sp. *avenae* for more than 100 yr (13). In Africa, slow rusting of corn with *P. sorghi* has been known for at least 80 yr and with *P. polysora* for about 30 yr (27). In the USA there has never been a severe epidemic of *P. sorghi* in field corn although seedlings of most hybrids are fully susceptible (7). Since the mid-1960s Knox winter wheat has been known to rust slowly with *P. recondita* f.sp. *tritici* (20).

Experimental evidence on the stability of slow rusting also has been developed. Beginning in 1971, and annually for 8 yr, the wheat cultivars Baart, Prelude, and Marquis (fast rusting); Lee and McMurachy (moderately slow rusting); and Kenya 58, Exchange, Thatcher, and Idaed 59 (slow rusting) were evaluated at Rosemount, MN, in nurseries inoculated with race 15-TLM and naturally occurring races and at the stem rust nursery in St. Paul where test cultivars were inoculated with at least six different races of the stem rust pathogen. Each year the ranking of the cultivars for the slow rusting trait was as indicated above (*unpublished*).

In 1977, cooperative tests on the stability of slow rusting with *P. graminis* f.sp. *tritici* were made in South Africa, Mexico, and Minnesota (R. D. Wilcoxson, J. N. W. de Jager, and H. J. Dubin, *unpublished*). The spring wheats studied were: fast-rusting Baart, Prelude, Marquis, and line 430-9 (Thatcher/Kenya 58); moderately slow-rusting Lee; and slow-rusting Kenya 58, Thatcher, Idaed 59, line 124-2, line 123-7 (both Baart/Idaed 59), and line 434-8 (Thatcher/Kenya 58). In South African trials Kenya 58, Baart,

Prelude, Marquis, and line 430-9 rusted rapidly; line 434-8 rusted moderately rapidly; and Lee, Thatcher, and line 124-2 rusted slowly. Idaed 59 and Line 123-7 were not infected. In Mexico, Baart, Prelude, and line 430-9 rusted rapidly, and the other lines and cultivars rusted slowly although some low infection types (indicating specific resistance) were observed in Lee and Marquis. In Minnesota, the cultivars and lines rusted as they had in previous trials. Results of the test clearly indicated that a cultivar may rust slowly in one area and rapidly in another.

Applications of  $\text{NH}_4\text{NO}_3$ ,  $\text{P}_2\text{O}_5$ , and  $\text{K}_2\text{O}$  in a factorial experiment over a 6-yr period at Rosemount, MN, did not alter the slow or fast rusting characteristics of cultivar Lee, which rusted moderately slowly with *P. graminis* f.sp. *tritici* and moderately slowly with *P. recondita* f.sp. *tritici*; Marquis, which rusted rapidly with both pathogens; and Thatcher, which rusted rapidly with the leaf rust pathogen and slowly with the stem rust pathogen. The interaction between fertilizers and cultivars was statistically nonsignificant (35).

The possible effects of the amount of inoculum used to start an epidemic was investigated (J. E. Ayers, unpublished). The slow rusting wheats Thatcher, Redman, Exchange, Idaed 59, Marquillo, Frontana, McMurachy, and Kenya 58; moderately slow rusting Lee, and fast rusting Baart, Prelude, and Marquis were uniformly sprayed with 0.10, 0.033, and 0.011 g of urediospores of race 113-RTQS of *P. graminis* f.sp. *tritici* in 300 ml of light mineral oil. Except for cultivars McMurachy and Kenya 58, which rusted rapidly in all plots, the ranking of the cultivars for slow rusting was as indicated above regardless of the concentration of the inoculum, although stem rust was more severe in plots that had received the larger amounts of inoculum. These conclusions were confirmed by Nazareno (18) with Baart, Prelude, and Thatcher wheats in stem rust epidemics.

Slow rusting varied with the planting dates of spring wheats at Rosemount, MN, during 1975 and 1976 when races 15-TLM and 151-QSH of *P. graminis* f.sp. *tritici* were the prevalent races (32). In nine wheat cultivars planted in late April or the first week of May (normal date for the area) stem rust developed rapidly or slowly according to the cultivars tested but, when the wheats were planted 1 mo later, stem rust developed two to six times more rapidly than usual.

The slow rusting of corn due to *P. sorghi* was a stable trait in India although there was some small variation due to the location of the tests (29). Kim and Brewbaker (9) demonstrated that most of the hybrids and lines of corn from the midwestern USA rusted rapidly with *P. sorghi* in Hawaii. However, 40 lines rusted slowly and five were outstanding in this respect. Line CM-105 has rusted slowly in the midwestern USA, and in Hawaii, India, and Taiwan (29).

**Effect of Races on Slow Rusting.** Whether races of the pathogens will limit the usefulness of slow rusting remains to be learned. For many years, as indicated above, wheat, oats, and corn have been known to rust slowly. Recently, the winter wheats Pb 028 and Suwon 85 were shown to rust slowly with 22 isolates of the leaf rust pathogen (10). More recent evidence now indicates that slow rusting wheat cultivars will rust rapidly when exposed to appropriate races of the stem rust pathogen (32). Other evidence indicates that components of slow rusting may be affected by races of the wheat and barley leaf rust pathogens (2,16,23).

Southern (32) tested extensively the effect of races on slow rusting utilizing nine spring wheats and five races of *P. graminis* f.sp. *tritici* in isolated field plots at Rosemount, Lamberton, Crookston, and Grand Rapids, MN. In 1975 race 15-TNMH predominated in all plots except at Grand Rapids where races 113-RKQ, 151-QSH, and 31-RSH predominated. In each of the plots, except those at Grand Rapids, the ranking of the cultivars, from those that rusted rapidly to those that rusted slowly was: Prelude, Marquis, Lee, Thatcher, Redman, McMurachy, Kenya 58, Exchange and Idaed 59, as had been observed many times previously. At Grand Rapids, cultivars McMurachy, Kenya 58, and Redman rusted as rapidly as Prelude and Marquis while the others rusted slowly. In 1976, the tests were repeated. In all plots Prelude and Marquis ranked among the cultivars that rusted

rapidly, Lee, Thatcher, Exchange, and Idaed 59 rusted slowly. McMurachy and Kenya 58 rusted as rapidly as Prelude in plots inoculated with races 151-QSH and 113-RKQ, but as slowly as Thatcher in plots inoculated with the other races. Redman rusted as rapidly as Prelude with races 31-RSH and 15-TMR and slowly with the other races.

The effect of races on components of slow rusting also has been tested. Clifford and Clothier (2) noted that isolates of *P. hordei* from a particular cultivar tended to be most vigorous, in terms of the numbers of infections produced and in the production of urediospores, on the cultivar from which they originated than on other cultivars.

Brennan (1) reported that in lines containing the Hope chromosome 3B, the frequency of infection with races of *P. graminis* f.sp. *tritici* influenced the rate of infection of wheat lines. Lines containing the Hope chromosome 3B reduced more effectively the frequency of infection by race 56 than by race 15B-1. In addition, lines 502 and 517 reduced more effectively the numbers of infections by race 17 than by a mixture of races 15B-1 and 15B-4.

In the glasshouse, Parlevliet (22) investigated latent periods (days from inoculation to sporulation by the pathogen) in six barley cultivars inoculated with five isolates of the leaf rust pathogen during the seedling and flag leaf growth stages. The latent period with isolate 11-1 was about one half day shorter than that with the other four isolates and the difference was more pronounced in slow rusting cultivars than in those that rusted rapidly.

Milus and Line (16) reported that in seedlings of wheat cultivars Wampum and Borah infected with culture 2 of *P. recondita* f.sp. *tritici* the latent period (the percentage of sporulating uredia on various days after inoculation) was shorter than when infected with culture 1, whereas in the cultivar Wandell the opposite was true. In flag leaves of Wampum infected with culture 1, the latent period was shorter than when infected with culture 2, but again, the opposite was true for the cultivar Wandell. Differences of this type were also noted for frequency of infection and for sporulation on the cultivars.

**Specificity of Slow Rusting.** Thatcher spring wheat rusts slowly with *P. graminis* f.sp. *tritici* and rapidly with *P. recondita* f.sp. *tritici*. Lee rusts moderately with both pathogens. Lines that rusted slowly with both pathogens were obtained from crosses of these two wheats (4). In Africa in 1949, corn rusted slowly with *P. sorghi* but not with *P. polysora*, but over the years lines were developed that rusted slowly with both pathogens (27).

**Genes for Race Specific Resistance and Slow Rusting.** The slow rusting characteristics of wheat cultivars or lines have been determined in the field and then the *Sr* genes that they possess have been identified in the glasshouse with appropriate cultures of *P. graminis* f.sp. *tritici*. Slow rusting in wheat has been shown to be independent of *Sr* genes 5, 6, 7a, 7b, 8, 9b, 11, 12, 16, and Tc (15,18,30) and J. E. Ayers and J. W. Southern (unpublished) found no relationship between slow rusting and various combinations of the genes *Sr* 5, 6, 8, and 9b.

Brennan (1) demonstrated that *Sr* genes that no longer condition race specific resistance to wheat stem rust did not provide slow rusting resistance. He found that all lines of Marquis isogenic for *Sr*5, 9d, and 11 were as severely rusted as Marquis itself.

**Combination of Slow Rusting and Race Specific Resistance.** The genetic factors that condition slow rusting and those that condition race specific resistance can be combined into single lines or cultivars (31). Martinez-Gonzalez (15) crossed Era spring wheat, which is resistant to most races of *P. graminis* f.sp. *tritici*, with Prelude and Morocco, cultivars that rust rapidly. In both populations, there were many  $F_2$  lines that rusted with *P. graminis* f.sp. *tritici* as slowly as Thatcher or Lee and some rusted even more slowly. Similar conclusions were made by J. E. Ayers and J. W. Southern (unpublished) with crosses of FKN (Frontana/Kenya 58/Newthatch), a line widely used as a source of generalized resistance against wheat stem rust, and two fast-rusting wheats.

The fact that cultivars may carry genes that condition both kinds of resistance also was demonstrated in barley infected with *P. hordei* by Parlevliet and Kuiper (25) who crossed Cebada Capa, Gondar, and LeEstanzuela, cultivars that carry gene *Pa* 7, with line



L94, a line with no known genes for resistance to *P. hordei*. The F<sub>2</sub> populations showed continuous segregation for latent period, from lines that had latent periods as short as L94 (about 8 days) to those that had latent periods as long as Vada (about 16 days).

**Number of Genes Conditioning Slow Rusting.** Because the distribution of variation among the progenies of crosses between slow and fast rusting cultivars is continuous and because transgressive segregation is common, it has been assumed that many genes condition slow rusting. While this assumption may be true, it is now known that some genes have a greater effect than others and that single genes may condition slow rusting. It is also likely that differing methods of study may detect differing numbers of genes that condition slow rusting.

It was suggested (5) that two pairs of genes conditioned the slow rusting of Thatcher wheat and that they were inherited independently of the factors that conditioned the race specific resistance of the cultivar. Hooker (7) concluded that slow rusting of corn infected with *P. sorghi* was probably conditioned by relatively few genes. Kim and Brewbaker (9) concluded that two partially dominant genes conditioned the slow rusting of the corn line OH 545 infected with *P. sorghi*. Luke et al (14) reported that two or three genes conditioned the slow rusting of *Avena byzantina* infected with *P. coronata*. Between 2 and 12 genes were estimated to condition slow rusting in wheat infected with *P. graminis* f.sp. *tritici* (29), and between 4 and 14 genes in wheat infected with *P. recondita* f.sp. *tritici* (4). In the latter two studies, the number of genes involved with slow rusting varied with the crosses studied.

Slow rusting of wheat with *P. graminis* f.sp. *tritici* was associated with *SrTt1* and by *Sr6* acting alone or in concert with unidentified genes (30). Apparently *Sr6* and *SrTt1* are more effective in certain backgrounds than in others. *Sr6* is especially interesting because at about 18C it conditions race-specific resistance but is ineffective at about 25 C (31). Thus, when the daily temperatures in the field exceed 25 C, the pathogen grows and when they fall below 18 C, the pathogen ceases to grow. In Minnesota, wheats with *Sr6* rust more slowly than those that lack the gene. It is likely that any *Sr* gene that is noneffective at certain temperatures may condition slow rusting.

Slow rusting with *P. graminis* f.sp. *tritici* in wheat may be conditioned by *Sr2* (34). Seedling plants that possessed this gene were susceptible, but older plants manifested varying degrees of resistance when inoculated with appropriate races. In the field, Hopps and Hopps/Federation, wheats that both possess *Sr2*, were much less severely rusted than were Gabo, McNair 701, Baart, or Line W3498, wheats that lack genes for resistance to the races used. Sunderwirth and Roelfs (34) considered *Sr2* to be a universally effective gene for adult plant resistance to stem rust and suggested that much more work should be done with it.

A number of genes also control the components of slow rusting. Brennan (1) suggested that at least six genes affected the frequency of infection by *P. graminis* f.sp. *tritici* in substitution lines of Chinese Spring that carried chromosomes from Hope or Thatcher. Rowell and McVey (28) found that low receptivity to infection by *P. graminis* f.sp. *tritici* in Idaed 59 was probably due to *SrTt1*, that in Thatcher to two recessive complementary genes, and that in Lee to a recessive gene. Parlevliet (23) estimated that one-to-five genes conditioned the latent period in barley infection with *P. hordei* as follows: in line L94 (latent period, 8 days) there were no genes for slow rusting; in Sultan (latent period, 10.5 days), three genes; in Volla (latent period, 10.5 days), three genes but with at least one of them different from those in Sultan; in Julia (latent period, 13 days), five genes; and in Vada (latent period, 15.5 days), six genes. One of the genes in Vada appeared to have a large effect and was recessive, whereas the other five genes had minor effects and were additive.

**Inheritance of Slow Rusting.** During the past 5 yr, slow rusting has been reported to be an heritable trait in wheat (1,4,31), oats (14), barley (8), corn (7,9,29), and ryegrass (36). Parlevliet (21) studied the inheritance of latent period in barley and Brennan (1) studied the inheritance of the frequency of infection in wheat.

In a study of inheritance of slow rusting in wheat infected with *P. graminis* f.sp. *tritici*, Skovmand et al (31) crossed seven cultivars in all combinations without reciprocals. The parent cultivars were the

fast rusting Baart and the slow rusting Idaed 59 and five intermediate cultivars: Lee, Kenya 58, Marquis, Prelude, and Thatcher. Stem rust severity was estimated during the development of epidemics in plants and the slow-rusting trait was estimated from area under the disease progress curve. The mean area under the curve varied among the 80 lines of each of the 21 crosses studied. The range for the area under the curve varied from 0.9 to 1,147 in the progenies of Thatcher/Idaed 59 and was similar in the other crosses. The distribution of the progenies in each cross, except those involving Kenya 58 and Idaed 59, resembled normal distributions. Transgressive segregation occurred in each cross. The genetic control of slow rusting was predominantly additive though epistatic gene action was also detected. Narrow-sense heritability was 82%. The data suggested that slow rusting is a quantitatively inherited character.

The inheritance of slow rusting in spring wheat infected with *P. recondita* f.sp. *tritici* was studied by Gavilertvatana and Wilcoxson (4) in 80 F<sub>3</sub> lines of crosses with Thatcher, Marquis, and Lee. The development of leaf rust was continuously distributed from the slow to fast rusting categories and transgressive segregation was common. Heritability ranged from 55 to 87%.

Wilkins (36) found in 12 ryegrass lines infected with *P. coronata* f.sp. *lolii* that a single dominant gene conditioned the resistance of two of 12 parents. The 10 other parents lacked the specific resistance gene and there was continuous distribution of the progenies from slow to fast rusting without dominance or epistasis. The genetic control of slow rusting was largely additive and the narrow-sense heritability was calculated to be 22%. He concluded that slow rusting was conditioned by a number of minor genes.

Brennan (1) studied the genetics of slow rusting with *P. graminis* f.sp. *tritici* using progenies of four slow rusting lines crossed with Opal, a fast rusting wheat. The slow rusting lines were 437 (Hope-Marquillo-Bonza-Hope), 553 (Thatcher-Bonza-Hopps-Hope), 790 and 875 (both from Marquillo-Renown-Frontana-Yaqui-Minn II-50-17 [FKN]). In the field, in an epidemic started with race 15B-1, the parents, populations of F<sub>1</sub>, F<sub>2</sub>, and backcrosses to both parents were evaluated for stem rust severity and frequency of uredia in five tillers. The populations were small, and there was considerable variability in the data, but slow rusting was a heritable trait and could not be attributed to simple genetic effects. A study of the distributions in the F<sub>1</sub>, F<sub>2</sub>, and the backcross populations and of the generation means indicated that dominance, epistasis, additive effects, and interactions among them were the types of gene action that conditioned slow rusting.

Johnson and Wilcoxson (8) studied slow rusting in nine crosses of seven barleys infected with *P. hordei* and found that differences in the speed of rusting were continuously distributed among the progenies in which transgressive segregation was common. Estimates of heritability varied from 13 to 65%. The heritabilities were higher in crosses between slow and fast rusting cultivars than between slow or fast rusting cultivars. Johnson and Wilcoxson (8) also studied selection of latent period and infection frequency using crosses of barley that rusted rapidly or slowly.

Luke, Barnett and Pfahler (14) found that progenies of oat crosses infected with *P. coronata* were continuously distributed between slow and fast rusting types. The gene action indicated some partial dominance for fast rusting and broad-sense heritability was 87%.

Kim and Brewbaker (9) studied the inheritance of slow rusting in corn by means of diallel and generation mean analysis of crosses among 11 inbred corn lines in severe epidemics of *P. sorghi* in Hawaii. The results of diallel analysis indicated the following conclusions: the nine parents showed significant general combining ability for slow rusting; heterosis was not apparent; specific combining ability effects were small, though significant; and the generation mean analysis indicated that broad-sense heritability was 83% and narrow-sense heritability was 47% and higher for the slowest rusting lines.

Sharma (29) who studied slow rusting in corn infected with *P. sorghi* in India, used six slow rusting and two fast rusting inbred lines, the 28 single crosses of these lines, the F<sub>2</sub>, and backcrosses to the F<sub>1</sub> parents. Among the progenies the data varied continuously

from slow to fast rusting without fitting simple genetic models. Transgressive segregation was common in each cross when the parents varied in slow rusting. This suggested that the nature of the resistance was quantitative and that it was multigenically inherited. Heterosis over the slower rusting parent was apparent in a few crosses. Inbreeding depression was indicated by the fact that the  $F_1$  rusted more slowly than the  $F_2$ . General and specific combining abilities were both significant, but general combining ability was considered to be more important because its variance was three times greater than that of specific combining ability. Both additive and nonadditive gene effects influenced slow rusting, but the additive effects predominated. Epistatic gene action was absent. Graphic analysis of the data suggested the presence of overdominance.

The inheritance of latent period in barley infected with *P. hordei* was studied by Parlevliet (21) on flag leaves of cultivars L94 and L92 (latent periods, 8 and 8.6 days, respectively) and Minerva and Vada (latent periods 16.9 and 17.1 days, respectively). The mean latent periods of the  $F_1$  and the  $F_2$  populations were intermediate between those of the parents or tended toward that of the susceptible parent. L94 and L92 and their progeny and the  $F_1$  populations of crosses between susceptible and resistant parents were homogenous, but not for resistant parents and their  $F_1$  progenies. The  $F_2$  progenies of the resistant parents were somewhat more variable than the parents and the  $F_1$ . In the cross of the susceptible and the resistant parents, the  $F_2$  showed considerable variation and the distribution was positively skewed. The distribution of most  $F_2$ s was bimodal with a peak at the value about equal to the  $F_1$  and a second peak at a higher value. Transgressive segregation was not observed nor were parental types recovered. The above study was repeated with cultivars L94, Sultan, Volla, Julia, and Vada with similar results (23).

**Studies with *P. striiformis*.** Studies by Sharp and his associates at Montana State University (11) appear to be related to the topic of slow rusting although the resistance is recognized by differences in infection types and is more readily discerned at higher temperatures. The infection types produced in wheat infected with *P. striiformis* form a continuously intergrading series from immune to large uredia. Tests were conducted in a growth chamber at 15/24 C (night/day temperatures) or at 2/28 C, photoperiods of about 12 hr with light intensities gradually increasing during the first half from 32.3 to 37,660 lux (3 to 3,500 ft-c) and then gradually decreasing, and relative humidities of 95% in darkness and 65% in the light.

Crosses of PI 178383, Chinese 166, and Lemhi were studied by Lewellen et al (11). PI 178383 was immune to prevalent races of *P. striiformis*, Chinese 166 was resistant to some races, and Lemhi was susceptible. With the cross of PI 178383 and Lemhi, all of the  $F_1$  progeny were resistant. The  $F_2$  progenies tested at 2/18 C were all resistant, but when tested at 15/24 C, some were susceptible. Tests with  $F_3$  progeny confirmed the results. The results were also confirmed by testing progenies of the crosses Chinese 166 / Lemhi and Chinese 166 / PI 178383.

PI 178383 and Chinese 166 were each considered to each carry one incompletely dominant major gene for resistance to the isolates studied as well as a number of minor genes that conferred resistance intermediate to that of the parents. At least three minor genes came from PI 178383 and one from Chinese 166. The major and minor genes acted independently of each other, but the effect of the major genes was modified by the minor genes. The minor genes gave better resistance at the higher temperatures than at the lower.

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