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Crown Rust

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I. Introduction

Crown rust of oats (*Avena sativa* L.), which is caused by *Puccinia coronata* Cda., is the most widespread and damaging disease of this cereal. The fungus has an almost innumerable array of pathogenic variants, and has repeatedly demonstrated its ability to adapt to constraints imposed by man as control measures.

This chapter will briefly review our knowledge of the disease, with emphasis on aspects having relevance to potential control measures.

II. Geographic Distribution

The crown rust fungus occurs nearly worldwide on oats. The distribution even includes islands far from any major land mass.

The aecial stage has been reported from all major oat-producing areas of the Northern Hemisphere where Rhamnaceous hosts occur in proximity to oats, including the Middle East where both hosts and the fungus presumably originated (Wahl, 1970). Susceptible species of *Rhamnus* are rare or nonexistent in South America and Australia, and thus the aecial stage does not occur there. It is also rare in some areas of relatively mild climate, such as the southern United States, even where susceptible hosts exist. This is due to the presumed requirement of low temperatures to break teliospore dormancy, although there are some unanswered questions on this point.

III. Economic Importance

A. HISTORY

It is likely that *P. coronata* has caused serious losses in oats ever since the crop was first cultivated. Over two centuries ago Tozzetti (1767) was aware of crown rust, as distinct from stem rust, and wrote that crown rust was very destructive to oats in some years (see Tozzetti, 1952). Generally, however, specific references to crown rust, as distinguished from stem rust and other diseases, are rare in the early literature. This is particularly true with regard to damage from the disease. It was not until late in the nineteenth century that Cornu (1880) wrote of severe damage from crown rust in Europe. At about the same time, Sivers (1887) reported three successive years of complete failure of the oat crop in the Baltic area because of crown rust.

In North America, Peck (1872) specifically listed *P. coronata* as a species new to New York in 1872, and it was collected in Iowa in 1874 (Arthur, 1924). Damage to oats in Connecticut was reported in 1890 (Thaxter, 1890).

B. LOSSES IN YIELD

Since 1900 there have been many reports of losses in yield of oats due to crown rust. Such reports are worldwide, and a representative sample follows.

In Uruguay, winter oats were sometimes killed by crown rust before they reached heading (Gassner, 1916). If flowering occurred, the resulting panicles were mostly barren. An epidemic of crown rust in 1940 in Portugal was so severe that barely enough seed for the next year's sowing was saved (d'Oliveira, 1942). In southeastern Europe, heavy attacks of crown rust were reported in 1959 (Kostic, 1959), and in Israel some cultivars were badly damaged (Wahl and Schreiter, 1953). Severe damage has occurred in coastal areas of Australia, with crops ruined and wild oats killed in years favorable to the disease (Waterhouse, 1952).

Severe losses of both grain yield and quality are not uncommon in both the northern and southern sections of the United States (Murphy *et al.*, 1942). A series of years in the 1940s and 1950s in which severe losses occurred in the United States were associated with the rapid increase in races of *P. coronata* attacking cultivars with the Bond resistance that occurred in conjunction with the increase in popularity of these cultivars. In the state of Iowa, losses were estimated at 12, 18, 20, 8, and 30% in 1949, 1950, 1951, 1952, and 1953, respectively (Sherf, 1954).

In recent years, losses due to crown rust have been light nationwide, but often are substantial over relatively large geographic areas and can be severe in individual fields. In 1979, losses were estimated to have reduced the potential crop by 2% in Minnesota, and by 3% in both North and South Dakota (D. L. Long, unpublished). Such losses in these major oat-producing states represent substantial dollar amounts.

C. METHODS OF ASSESSING LOSSES

There is no doubt that *P. coronata* significantly reduces potential oat yields. As with other economically important plant diseases, however, expressing such losses in terms of quantity or value, especially over

large areas, is a vexing problem. An early attempt to make a quantitative estimate of losses caused by *P. coronata* was made by Pammel, who estimated that a severe epidemic in 1907 had resulted in a loss of 50% of the crop (Melhus *et al.*, 1942).

Immer and Stevenson (1928) compared yields of a group of 200 strains of oats that ranged from 5 to 70% infected. Each 1% increase in disease reduced yield by about one-third of a bushel per acre after allowances were made for differences in maturity.

Murphy *et al.* (1940) found that later maturing oats were more severely damaged by *P. coronata*. Studies of the severe epidemic of 1938 showed that the coefficient of infection (an index that combines percentage of leaf area covered by uredia with the size and other characteristics of the uredia) was more highly correlated with damage from *P. coronata* than was percentage of leaf area covered alone.

Quantitative estimates of losses caused by *P. coronata* for the state of Illinois in the moderately severe crown rust year of 1957 were published by Endo and Boewe (1958). Since some of the oat cultivars being grown were resistant to the crown rust races then present, performance of susceptible and resistant cultivars could be compared in nurseries grown in different areas of the state. Data were adjusted to reflect differences in acreage of each major cultivar in each area. Adjustments for inherent differences in cultivar yields were calculated from earlier years in which crown rust had been insignificant. Total losses averaged over cultivars ranged from 10 to 40% among the areas, with a statewide loss of 20%. Grain quality (test weight) of susceptible cultivars was also greatly reduced. A study in the mild rust year of 1956 indicated that crown rust destroyed over 43,000 metric tons of grain, showing that significant losses can result from small amounts of disease.

Fungicides can be used in estimating losses from *P. coronata* (Martens *et al.*, 1972). In Manitoba, yields of control plots, maintained rust-free with a fungicide, were compared with yields of plots exposed to natural infection over a period of years. In 1968 and 1969, the two years in which *P. coronata* developed with some degree of severity and was the only disease of consequence, yields were reduced in the cultivar Kelsey by 26 and 31%, respectively. Seed quality was also significantly reduced.

D. OTHER LOSSES

Puccinia coronata can have other adverse effects on its host in addition to reducing grain yield and quality. One of these is reduction of

yield and quality of forage. Severe damage to oats being grown for forage has been reported from South Africa (Sawer, 1909), Germany (Straib, 1937b), and Australia (Miles and Rosser, 1954).

In two New Zealand locations, *P. coronata* reduced the dry-matter yield of forage oats during late winter and early spring from 13.6 to 11.4 and 6.66 to 2.85 tons per hectare (Eagles and Taylor, 1976). The grazing season is shortened by as much as 4–6 weeks by crown rust infection in Texas (Reyes and Futrell, 1962). In the first 2 years of a study of the effect of *P. coronata* on forage oats in Texas, the cultivar Moregrain outyielded Suregrain. Both cultivars were resistant to the prevalent forms of *P. coronata* present in these years. In the third year, forms of the fungus appeared that moderately infected Moregrain. Suregrain was resistant and it outyielded Moregrain significantly.

Oat straw has important economic value in many areas, and *P. coronata* can significantly reduce straw yield of susceptible cultivars (Simons, 1980). The use of controls maintained free of rust by a fungicide showed that straw yields are reduced by as much as half by severe epidemics. Lesser amounts of disease resulted in proportionally smaller losses.

The harvest index is the ratio of grain yield to total yield of grain and straw, and this ratio is sometimes an important consideration in oat breeding. It has been shown (Simons, 1980) that harvest indices of different cultivars can be differentially affected by *P. coronata*. Harvest index, or percentage of grain, of some cultivars was increased, while other cultivars showed a decrease in harvest index as a result of infection.

Winter hardiness is a concern in many areas where winter oats are grown. *Puccinia coronata* reduces the ability of young oat plants to harden at low temperatures, with the loss of cold resistance proportional to the severity and length of exposure to the disease (Murphy, 1939). In the field, greater winter injury was observed in sections where the disease had been heavy, and resistant oats had less winter injury than susceptible strains (Rosen *et al.*, 1942).

The relative protein percentage of oat grain is related to the feeding value of the grain, and attempts have been made to determine the effect of crown rust on protein percentage (Sebesta, 1974; Singleton *et al.*, 1979; Simons *et al.*, 1979b). No single answer has been found, but generally, moderately severe crown rust slightly reduces protein percentage. However infection has caused an increase in protein percentage of certain cultivars. Of greater importance is the significant reduction in total protein produced per acre due to the direct effect of a severe infection on reducing grain yield.

IV. Taxonomy

A. SPECIES LEVEL

The fungus responsible for crown rust of oats and other grasses was first described in the aecial stage by Persoon, as *Aecidium rhamni* (Gmelin, 1791). The telial stage was described by Corda (1837), who listed the rush *Luzula albida* as the host. He named it *Puccinia coronata* due to the projections on the apical end of the teliospore. Castagne (1845) was the first to recognize the fungus as a grass rust. He observed it on *Avena sativa*, *A. fatua*, and *Festuca arundinacea*, and named it *Solenodonta graminis*. Klebahn (1892) found, as had Nielsen (1875) earlier, that crown rust occurred in two forms, one that parasitized *Rhamnus frangula* and certain grasses, and one that parasitized *R. cathartica*, oats, and certain other grasses. He regarded the form on *R. cathartica* and oats as a separate species and designated it *P. coronifera*, while retaining the name *P. coronata* for the form on *R. frangula*. Later, others divided it into several species by the reactions of various species of *Rhamnus* and genera of grasses, resulting in some controversey regarding the number of species (Simons, 1970).

The controversy has continued, and the status of the specific names *P. coronifera* and *P. lolii* is still not settled. Azbukina (1976) contended that *P. coronifera* should be maintained as a species separate from *P. coronata*, noting they differ somewhat in morphology and markedly in aecial and uredial hosts, and that *P. coronata* and *P. coronifera* do not cross. Nevertheless, most researchers follow Cummins (1956) and Cunningham (1964), who regarded all species differentiated on the basis of pathogenicity as synonyms of *P. coronata*. They recognized, however, that forms of the fungus show considerable morphological diversity, which might indicate a need for subspecific taxa based on morphology.

B. SUBSPECIFIC

Eriksson and Henning (1894) subdivided *P. coronifera*, the species on *R. cathartica*, into two categories, which they designated "f." for form. The form on cultivated oats was designated f. *avenae* and the other, on the grass *Alopecurus pratensis*, f. *alopecuri*. Eriksson (1894) showed that urediospores from one grass genus did not infect species of other grass genera. The term *formae speciales* (f. sp.) was introduced to describe such pathogenic strains, and the term *P. coronata* f. sp. *avenae* became generally accepted for isolates of the fungus that parasitized

wild and cultivated oats. Many experiments were done in the next 20 years and were summarized by Muhlethaler (1911). A number of additional special forms were described and, recognizing some exceptions, it was felt that special forms were restricted in pathogenicity to a single grass genus or even to certain species in a genus. However, Melhus *et al.* (1922) felt obliged to stress that some of the forms induced infection in species of several genera in addition to the ones for which they were named.

Klebahn (1895) noted that specialization of the fungus aptly described the situation. Nevertheless, he disliked the term *forma specialis* as it added another unit to the concept of species and varieties and it had no recognized rank. Stakman (1929), aware of these shortcomings of the term *forma specialis*, suggested that the term variety be used to designate subdivisions of species based on pathogenic specialization at the level of host species and genera. Such varieties might differ somewhat in size and shape of spores but were mainly characterized by pathogenicity. Each variety could parasitize several species of one or more genera of grasses but could not parasitize members of other genera, which might, however, be susceptible to other varieties of the fungus.

Fraser and Ledingham (1933), working in Canada, followed Stakman in the use of variety to designate subdivisions of *P. coronata*. Morphological features and rhamnaceous hosts were important parts of their scheme. Isolates of *P. coronata* parasitizing oats were designated *P. coronata* var. *avenae*, and this term was subsequently recognized by many investigators.

Eshed and Dinoor (1980) compared pathogenicity of cultures of *P. coronata* isolated from the grass genera *Agrostis*, *Alopecurus*, *Arrhenatherum*, *Phalaris*, *Festuca*, and *Lolium*, and nine cultures from cultivated oats. Seedlings of 22 grass species in eight genera and one oat cultivar were inoculated with each culture. The results showed that differentiation of *P. coronata* into *formae speciales* is largely meaningless. The fact that a culture from one host infected a second host did not necessarily mean that another culture originally from the second host would infect the first one. The culture isolated from *Festuca* was infectious to hosts in six other genera, while *Festuca* itself was susceptible to only one additional culture. At the other extreme, the culture isolated from *Arrhenatherum* was innocuous to any of the other host genera while *Arrhenatherum* was susceptible to at least some extent to all the forms. The nine cultures originally isolated from oats should belong to form *avenae*. They were classified into seven

races on differential oat cultivars (see Section V,A), but reactions of the grass hosts grouped them into four different forms. There was no greater similarity between these four forms than between forms from the other host genera. Thus, it seemed that there was no essential difference between a race as defined on oat cultivars and a form as defined on grass species and genera. They concluded that forms and races are parallel taxonomic entities, and races are not subordinate to forms in a taxonomic hierarchy. Thus, I find it difficult to justify the use of Latin trinomials such as *P. coronata avenae* and follow the suggestion of Eshed and Dinoor (1980) that the subspecific divisions of the species *P. coronata* be abandoned.

V. Pathogenic Specialization

A. ON OATS

Pathogenic specialization of *P. coronata* at the cultivar level is also the basis of cultivar resistance. Surveys of pathogenicity, or virulence, have covered various areas over different periods of time. The data have been interpreted in relation to the presence or absence of the alternate host, the susceptibility or resistance of the cultivars grown, and the reactions of oats having potential value as sources of resistance. Until recently, most surveys have been reported in terms of races (defined below), as this summarized the data in a form that was readily understandable.

Proof of specialization within *P. coronata* at the cultivar level was first published by Hoerner (1919). Seven cultivars of oats were inoculated with 30 isolates of *P. coronata* collected in widely scattered locations in the U.S. and Canada. The reactions of two of the cultivars, Ruakura and Green Russian, clearly illustrated pathogenic specialization at the cultivar level. Isolates of *P. coronata* characterized by such specific patterns of pathogenicity on designated groups of oat cultivars are usually designated as races.

Murphy (1935) summarized the work of Hoerner and those who followed him in the area of pathogenic specialization of *P. coronata* and established a system of classification of infection types into reaction classes that is still in general use:

Immune (I): no macroscopic evidence of infection.
Nearly immune (O): no uredia; necrotic areas or chlorotic flecks present.
Highly resistant (1): uredia few, small; some necrotic areas without uredia.

Moderately resistant (2): plentiful small to medium-sized uredia; necrotic area seldom without uredia.
Mesothetic (M): a combination of two or more types in varying proportions.
Moderately susceptible (3): abundant medium-sized uredia in chlorotic areas; no necrosis.
Completely susceptible (4): abundant large uredia without necrosis or chlorosis.

Races were characterized on the basis of only three reaction classes: resistant (classes I, 0, 1, and 2), mesothetic, and susceptible.

After extensive testing, Murphy (1935) established a standard set of 11 different cultivars (later enlarged to 13) for purposes of race identification. Thirty-three races were identified among 533 isolates collected throughout the United States as well as from Mexico and Canada during the years 1927–1932. They varied in versatility from very aggressive to very restricted forms. Some were common and present annually, and others were rare and appeared occasionally. Certain races overwintered on fall-sown oats in the winter-oat region; others were dependent on the alternate host for initial dissemination in the spring. More races were identified from 100 isolates collected in the north than in the south, indicating that hybridization and segregation of races on *Rhamnus* in the north resulted in a more diversified *P. coronata* population than occurred in the asexual population in the south.

In Canada, Peturson (1935) identified 11 races among 544 isolates of *P. coronata*, most from the prairie provinces of Canada. Straib (1937b), working in Germany, established a set of 15 differential cultivars, including seven of those used in North America. He identified 142 races from 144 isolates, and concluded that specialization of the fungus in Germany was stronger than in North America. Since almost every isolate represented another race, he felt there was little point in additional testing. Straib used finer distinctions of infection types than had the North American workers, and he also noted, as had others, that different environmental conditions, particularly temperature during the incubation period, could make a difference in reaction class. Subsequent race surveys in Northern Europe have generally indicated that *P. coronata* in this area is markedly specialized. For example, Hermansen (1961) isolated five races from six samples.

In South America, Pessil (1960) and Vallega (1951) showed that North and South America had certain prevalent races in common, but some South American races parasitized the cultivars Landhafer and Trispernia, which were resistant to North American races. Races that parasitized the diploid cultivar Saia were rare in North America but were common in Brazil.

The most resistant oat cultivars in the Soviet Union were Victoria,

Landhafer, Santa Fe, and Bondvic (Zhemchuzhina, 1978), while in North America they were generally susceptible.

The 13 standard differentials were used worldwide from 1935 to the early 1950s. Descriptions of the 112 races identified were given by Simons and Murphy (1955). The cultivars of the standard set chosen in the mid-1930s had lost much of their usefulness by 1950 because new or formerly unimportant races of *P. coronata* had become prevalent. In 1950 and 1951, Simons and Murphy tested *P. coronata* isolates made in the United States on additional cultivars. As a result, a second set of 10 cultivars was established as a new standard for identification of races of *P. coronata*. Races identified on it by Simons and Murphy (1955) were combined with races identified in South America and Canada and 59 races were initially described. These were numbered starting with number 201, to prevent confusion with races identified on the old set of cultivars. The new set of cultivars was widely used for the next two decades, and a total of 294 races were identified worldwide by 1976 (Michel and Simons, 1977).

The current trend in the study of pathogenic specialization of *P. coronata* at the host cultivar level is away from the use of standard sets of differential cultivars. Individual investigators in different areas now commonly survey and evaluate the virulence of *P. coronata* in their area in relation to available sources of resistance or resistance found in commercial cultivars grown locally. Examples of such studies have been reported by Martens *et al.* (1977) in New Zealand and Harder (1980) in Canada.

B. ROLE OF *RHAMNUS*

The origin of pathogenic diversity of *P. coronata* at the host cultivar level has important practical implications in the development of methods to control the disease. One way by which new combinations of pathogenicity, or races, arise is by recombination of existing genes for pathogenicity in the sexual cycle. Populations of *P. coronata* originating on *Rhamnus* have been compared with populations that exist in the uredial stage where *Rhamnus* is absent or unimportant. In a study of *P. coronata* isolates from the northern and southern United States, Murphy (1935) showed that certain races seemed to overwinter in the south in the uredial stage, while others appeared to be dependent on spread from *R. cathartica*. A relatively large number of rather rare races were collected from *Rhamnus*, or from oats near *Rhamnus*, suggesting that *Rhamnus* functioned effectively to generate pathogenic diversity.

Surveys over many years show that more races of *P. coronata* occur in eastern Canada than in the west (Fleischmann *et al.*, 1963). There is also less tendency for a few races to predominate in the east. These differences were explained by the relatively frequent occurrence of the alternate host in the oat-growing areas of eastern Canada. It has also been shown in Canada that more races can be identified from *Rhamnus* than in equal numbers of collections made from oats (Fleischmann, 1965). The diversity of races of *P. coronata* in Yugoslavia was attributed to the abundance of aecia on the alternate host (Kostic, 1965).

In 28 isolates of *P. coronata* from a *Rhamnus* hedge in Minnesota, there were 13 known races and four new races (Saari and Moore, 1963). Some could be subdivided by the use of supplementary differential cultivars. It was suggested that planting resistant oat selections next to buckthorn would measure the pathogenic potential of the fungus, while providing a severe test of the value of the resistance being considered. Material from this same *Rhamnus* nursery was compared by Simons *et al.* (1979a) for pathogenicity on 24 differential oat cultivars with *P. coronata* isolates collected in Texas, where *Rhamnus* does not function as a host of the fungus. As expected, more virulence patterns (races) occurred in material from the buckthorn nursery. However, there was very little difference between the two populations in numbers of virulence genes. This emphasizes that *Rhamnus* serves only to segregate virulence genes, and plays no role in their origin. Such segregation conceivably could expedite the development of "super races" having a wide virulence but the data failed to support that possibility.

The general pattern of race succession in the United States in recent years substantiates the subordinate role of *Rhamnus* (Michel and Simons, 1977). For example, race 290 and similar races that dominated in the 1960s were supplanted in the 1970s by race 264B, with a much wider range of virulence. Race 290, however, remained an important component in collections from *Rhamnus*, suggesting that *P. coronata* on *Rhamnus* follows, rather than leads, the evolution of virulence of the fungus.

C. METHODOLOGY

Studies of the occurrence and distribution of virulence in populations of *P. coronata* have advanced our knowledge of the disease in the areas of epidemiology and fundamental genetics. Their primary objective, however, was to help in developing resistant oat cultivars. Information needed for this major objective included (1) year-to-year preva-

lence of the various races and (2) early detection of new and potentially dangerous races. Unbiassed data on the relative prevalence of common races can best be obtained from material collected on universally susceptible cultivars. This prevents the sampling bias resulting when material is collected from cultivars that have resistance that screens out part of the fungus population. The appearance of new and rare forms can best be detected in collections made from oats with the important resistance genes (Simons, 1955).

Browder (1969) considered the various objectives of surveying virulence of the rusts, and made the important point that all such work must ultimately be grounded in the gene-for-gene theory of parasitism (Flor, 1956). Browder felt that virulence data presented in terms of races tended to obscure important information. He suggested that it would be more meaningful if presented on the basis of virulence to single host cultivars or, better, as virulence corresponding to single genes for resistance. However, there should be some way of recognizing associations of virulence in reporting survey data.

As a compromise, Simons and Michel (1959) utilized temporary differential cultivars that were changed from year to year, or as the need arose, in addition to the standard differential cultivars. Virulence of the fungus isolates on the standard differentials was reported in terms of races, and on the temporary differentials, in terms of virulence on the individual cultivars. Some information is lost using this system because it is not possible to show the range of virulence of individual isolates on the temporary differentials. However, it makes the data understandable in terms of practical breeding. Fleischmann (1967) presented survey data both as races and as virulence for cultivars.

VI. Genetics of *Puccinia coronata*

A. MUTATION

Mutation is the only source of basic new virulence in the fungus. Mutation at a given locus is a rare event, and critical experimentation is limited. However, Zimmer *et al.* (1962) observed measurable rates of mutation of races 202 and 290 for virulence toward cultivars resistant to these races. The mutation rate of race 202 for virulence on Ascencao was estimated at one in 2200 infections; for virulence on Ukraine, one in 6450; and for virulence of race 290 on Ukraine, one in 7900.

The appearance of a new race of *P. coronata* in Canada appeared to exemplify the process of natural mutation in the origin of new virulent

races (Fleischmann, 1963b). This race, numbered 332, differed from the then very common race 216 only in its ability to parasitize the cultivar Saia. The appearance of the new race, and its increase to where it was common enough to be detected in the survey, occurred in the absence on any known Saia-type resistance in cultivars being grown commercially, suggesting the operation of selective forces other than host resistance.

B. SEGREGATION

The artificial hybridization of isolates of *P. coronata* on *Rhamnus* could throw light on the relative importance of *Rhamnus* in the appearance of new races. Unfortunately, the problems of producing telia in isolation and inducing telia to germinate have proven to be a deterent. However, some studies are available. Eriksson (1908) inoculated *Rhamnus* with spores from *Alopecurus* and found that the aeciospores parasitize cultivated oats that were not hosts for the original culture. Similarly, a culture from *Festuca* attacked *Lolium* after passage through *Rhamnus*.

Eshed and Dinoor (1976) noted that the overlapping host ranges of different forms of *P. coronata* facilitated genetic studies, because a common host could serve as a propagating host for the F_1 and F_2 generations. A selected group of 32 species of grasses, representing 18 genera, were inoculated with forms of *P. coronata* parasitic on *Avena* and *Phalaris* and with the F_1 and F_2 progeny cultures. Some of the F_2 isolates were unable to parasitize either *Avena* or *Phalaris*. Others had a widened host range to include additional virulence on oats, and included another *Phalaris* species that was resistant to the original cultures. The increased virulence on oat cultivars was assumed to be due to inhibitors. Interactions between parental, F_1, and F_2 cultures and oat cultivars could be explained by 30 loci, while 44 loci were needed to explain interactions on the wild-grass hosts. Segregation within hosts, as well as gradations in reactions to many cultures, pointed to the possibility that despite satisfactory classical genetic explanations of many of the results, pathogenicity might be polygenically controlled in some cases. Biali and Dinoor (1972) showed that the genetics of pathogenicity in *P. coronata* does not always follow the usual pattern with virulence recessive. Selfing an isolate of race 276, virulent on many oat cultivars, showed that virulence was dominant for some loci.

Murphy (1935) identified urediospores of races 1 and 3 from the cultivar Hawkeye and used telial material from the same plants to

inoculate *Rhamnus*. Most of the resulting cultures were identified as the previously unknown race 18, but a few were races 1 or 3 or another new race, 19. Hawkeye was resistant to races 18 and 19, and this indicated that races 1 and 3 were highly heterozygous for pathogenicity. Zimmer *et al.* (1965) selfed a relatively homogeneous natural population of *P. coronata* and obtained pathogenically diverse S_1 progenies. Segregation for pathogenicity to 10 of 16 oat cultivars occurred in 52 cultures derived from this selfed population. A high degree of heterozygosity was also found by Dinoor *et al.* (1968), who intercrossed nine pycnia representing five pathogenic races and identified at least 25 races among the progeny. Among the progeny was an array of recombinants ranging from those with limited virulence to those with wide virulence, including virulence that the parental cultures had not exhibited. Nof and Dinoor (1981) crossed two isolates of *P. coronata* and two oat cultivars, and studied segregating populations for virulence and resistance, respectively. They found the oat–*P. coronata* system is under gene-for-gene control, as had been demonstrated for other rust fungi earlier (Flor, 1956).

C. HETEROKARYOSIS VIA ANASTOMOSIS

Hyphal anastomosis of *P. coronata* may be a possible source of pathogenic variation. This was shown by Bartoš *et al.* (1967), who mixed urediospores from two single-spore isolates of *P. coronata* that differed in pathogenicity. The mixture was used to inoculate a susceptible oat cultivar, and single-pustule isolates resulting were identified. The majority of these were of the parental types. However, two new races were found. Reassociation of parental nuclei following germ-tube fusions or hyphal anastomoses apparently was responsible for the occurrence of the new races.

D. LINKAGE

Whether the virulence of isolates of *P. coronata* is linked to traits like aggressiveness is important both theoretically and practically. Brodny *et al.* (1979) showed that isolates of race 276 usually had greater infection ability and capacity to develop infection than did isolates of race 263. At or above 25°C, there was a greater tendency for early telia formation among isolates of race 263 than race 276.

In New Zealand, Martens *et al.* (1977) found that most isolates of *P. coronata* carried apparently unnecessary genes for virulence that

seemed to confer no competitive disadvantage. Such data do not support stabilizing selection as a force in *P. coronata*.

VII. Signs and Symptoms

The uredial stage of *P. coronata* occurs mainly on the leaf blades of the oat plant, but to some extent on the sheaths and floral structures. On susceptible cultivars, the uredia appear as bright orange-yellow, round-to-oblong pustules that are up to 5 mm or more in length when infection is light. Cultivars with different degrees and types of resistance may show reactions ranging from small, light-colored flecks through small to medium-sized pustules surrounded by generally well-defined chlorotic or necrotic areas. Telia usually appear after uredia are well established and sometimes form in rings around the uredia. They are black or dark brown and remain covered for some time by the epidermis. Pycnial and aecial stages occur mainly on the leaves, often with hypertrophy, but also to some extent on the petioles, young stems, and floral structures of susceptible species of *Rhamnus*. The pycnia appear early in the spring as small, round, orange-yellow, slightly raised structures, usually on the upper surface of the leaf. Aecia, which usually form on the underside of the leaf beneath the pycnia, follow the pycnia and appear as round or somewhat irregular, tightly packed clusters of small orange-yellow cups. Aecia may be up to 5 mm or larger in diameter.

VIII. Life History of *Puccinia coronata*

Puccinia coronata is a typical heteroecious, long-cycle rust, with its repeating dikaryotic uredial stage occurring on oats more or less throughout their active growing period (Simons, 1970). As the season advances and as the plants start to mature, telia are formed in and around the uredia, and these serve to overwinter the fungus. Meiotic reduction occurs in the teliospores, and germination of the teliospores results in haploid basidiospores. These infect young leaves of susceptible species of *Rhamnus*. DeBary (1867) first demonstrated this connection between *P. coronata* on grasses and on buckthorn when he used "sporidia" from an unspecified grass to inoculate *R. frangula*. Both "spermogonia" and aecia resulted, but attempts to inoculate oats, rye,

and wheat with the aeciospores were unsuccessful. The function of the haploid pycnia and pycniospores that result directly from infection by basidiospores was not discovered until many years later, when Craigie (1928) showed that the diploid condition was restored by the transfer of the self-incompatible pycniospores to flexuous hyphae of other pycnía. Dikaryotic aeciospores then appear in the aecia.

Heteroecism of *P. coronata* was proved by Nielsen (1875) when he obtained uredial infection on *Lolium perenne* from inoculation with aeciospores from *R. cathartica*. A few years later, Cornu (1880) used aeciospores from *R. cathartica* and *R. oleoides* to obtain heavy infection on young oat plants.

In climates where the winters are mild, the fungus may live indefinitely in the uredial stage on cultivated, volunteer or wild oats.

IX. Epidemiology

A. ROLE OF *RHAMNUS*

The relative importance of *Rhamnus* in areas of the world where it functions in the epidemiology of the disease varies greatly. *Puccinia coronata* on oats is invariably associated with *Rhamnus* in Siberia (Wahl *et al.*, 1960). Since the fungus can not overwinter in the uredial stage in the severe climate and there is no source of wind-borne urediospores, the disease is dependent on the presence of *Rhamnus*.

In northern Europe, *P. coronata* does not overwinter in the uredial stage, and *Rhamnus* is the primary source of inoculum (Straib, 1937b). However, wind-borne urediospores may move from the south.

Much of the oat acreage of the world is where *P. coronata* does not overwinter in the uredial stage and where both *Rhamnus* and wind-borne urediospores may be important. The relative importance of aeciospores and urediospores varies annually, and depends on the amount of disease on oats in the overwintering area, velocity and direction of the wind at critical times, progress of *Rhamnus* eradication programs, etc. The north central United States and prairie provinces of Canada exemplify such areas. The progress of a limited epidemic initiated by *Rhamnus* in Iowa was documented by Dietz (1923), who made detailed observations on the progress of several such epidemics. In one of these, the first pycnia appeared on May 13, aeciospores were mature on May 16, and uredia first appeared on nearby oats on May 22. He assumed that uredia found on oats during the next week resulted di-

rectly from aeciospore infection. Viable aeciospores were produced up to June 10 on *Rhamnus*, and resulted in no infection beyond 2.4 km from the *Rhamnus* plants. Thus, the direct spread of the disease from aeciospore infection covered 1,300 hectares. Urediospore infection had spread over 8,500 hectares by June 4, 32,600 by June 7, and 165,000 by June 10, at which time infection occurred at a maximum distance of 85 km from the hedge. No other *Rhamnus* bushes were found in the 32,600 hectares in which infection occurred by June 7. Oats in fields adjoining this hedge were a total loss, and yields were significantly lowered in all fields within the area of aeciospore infection.

Simons (1970) summarized work of Dietz (1926) and others that showed that a large number of the known species of *Rhamnus* (Wolf, 1938) are susceptible to the forms of *P. coronata* that parasitize oats. However, only a small number are of economic importance. In North America and northern Europe, *R. cathartica* is the most important species involved in the infection of oats. It was introduced into North America in pioneer times and was well adapted to northern United States and Canada where oats are grown. Because of its desirable qualities as a hedge plant and shrub, it still is widely grown, and has escaped cultivation in many places.

Rhamnus frangula, also native to Europe and common in the northern United States as an introduced shrub, apparently plays no significant role in the epidemiology of crown rust of oats on either continent, in spite of an occasional report of susceptibility to strains of *P. coronata* that parasitize oats. *Rhamnus lanceolata* is a common shrub native to much of the central United States and is susceptible to forms of *P. coronata* capable of infecting oats, but is of little importance as it occurs only infrequently or in wooded areas away from oats.

The situation elsewhere is poorly documented, but it appears likely that a relatively small number of *Rhamnus* species are of economic importance. In Israel, four *Rhamnus* species are present, but *P. coronata* occurs on only two, and only *R. palaestina*, appears to be economically important in the epidemiology of the disease.

B. CROWN RUST WITHOUT *RHAMNUS*

Rhamnus shrubs and hedges are commonly responsible for severe local epidemics of crown rust in northern climates, but general, widespread epidemics in the north central United States and Canada seem to result from infection from airborne urediospores from infected oats planted in the fall or earlier in the spring in milder southern climates.

In most years this spore movement occurs as a stepwise process in which spores are blown relatively short distances from field to field as the season advances. Occasionally, however, disease and climatic conditions are such that spores may be transported in large quantities over long distances and be deposited on oats growing far to the north at a relatively early stage of growth. The severe epidemic that occurred in Iowa and adjoining states in 1953 was good example (Sherf, 1954). In early May, 1953, *P. coronata* was common in maturing oat fields in Texas and Oklahoma. From May 12 to May 14, strong, steady south winds blew across Oklahoma and northward at 10,000 feet and deposited a relatively heavy shower of urediospores over much of Iowa and areas of adjoining states. Initial infection thus occurred on these plants at an early stage in their development, and the weather for the remainder of the season was near ideal for development of subsequent generations of the fungus. The resulting epidemic was one of the most destructive ever recorded, causing an estimated loss of 30% of the potential oat crop in Iowa.

Rhamnus generally does not function in the epidemiology of crown rust in areas where winters are relatively mild and where summers are long and hot. Oats are produced under such conditions in many places in the world, including the southern United States, South America, Australia, etc. The epidemiology of the disease in these situations has not been investigated as intensively as in the north, but it is known that the oats are rusted during at least part of the cool or winter season. It is not always clear how the fungus survives the long, hot summers in the uredial stage to provide initial inoculum in the fall or winter. Generally the fungus survives on volunteer or wild oats that grow through the summer and fall. Observations made by Forbes (1939) showed that this is not true in Louisiana. Urediospores did not survive over the summer on straw piles or on dead leaves, and no volunteer oats or susceptible grasses could be found during the summer. It was suggested that the uredial stage was maintained in the northern states during the fall on volunteer oats and that wind-borne urediospores from these plants gradually spread southward to reach Louisiana in the winter. Such southward movement of *P. coronata* has been reported by Atkins and McFadden (1947) in Texas. The fungus became established on volunteer oats in the fall in the northern part of the state, where winters are too severe for *P. coronata* to survive in the uredial stage, and then spread southward across the state to winter oat areas as the season advanced.

Migrating birds may be a factor in the epidemiology of *P. coronata*

and other cereal rusts. Warner and French (1970) noted that large flocks of blackbirds, as well as other species, regularly passed the winter in central and southern Mexico in areas where urediospores are continuously produced by several species of rust fungi on cultivated cereals. Here, they may become heavily contaminated with urediospores and carry these spores with them when they migrate to the northern United States and Canada in the spring. Urediospores could be carried to the south in the fall by the same process. Warner and French showed experimentally that starlings were quite efficient in picking up urediospores from infected oat plants and transferring them in a viable condition to healthy plants in another location.

X. Control

A. RESISTANCE

1. *History*

Of the measures that are available to control *P. coronata*, the use of resistant cultivars is the most important. In one of the earliest known tests of cultivar reaction, Sivers (1887), after three successive years of crop failure due to crown rust, planted 41 cultivars in an effort to find one that would be sufficiently resistant to permit cultivation of oats. Most of them were rated as moderately or strongly infected, but infection was rated as weak on eight and there was almost no infection on one, the Russian Orel oat.

Observations clearly aimed at distinguishing between different reactions to *P. coronata* were made by Norton in 1907 in the United States. At about the same time, Sawer (1909) reported *P. coronata* to be a serious problem on oats in South Africa. He noticed differences in susceptibility among cultivars, observing that three of the red oat group were the most resistant.

Another important milestone in the utilization of resistance was Parker's (1920) demonstration that resistance could be found in progeny from crosses between resistant and susceptible cultivars. Parker (1918) was also one of the first to carry out extensive comparisons of reactions of cultivars in the seedling and later growth stages. This set the stage for the division of resistance to *P. coronata* into two categories: (1) specific (roughly equivalent to seedling, vertical, oligogenic, etc.) and (2) general (adult plant, horizontal, polygenic, etc.).

2. *Specific Resistance*

a. Inheritance. The first clear-cut demonstration of Mendelian inheritance of resistance to *P. coronata* was done by Davies and Jones (1927) studying a cross between the susceptible Scotch Potato oat and the resistant Red Rustproof.

The introduction of the Victoria oat from South America in 1927 and its subsequent recognition as a valuable source of resistance prompted many studies. Murphy *et al.* (1937) showed that Victoria had a single partially dominant gene for resistance, but Smith (1934) found resistance to be dominant only in some crosses. Chang and Sadanaga (1964) found that the resistance of Victoria to race 290 was conditioned by two genes, only one of which conditioned resistance to race 203.

It appears that Victoria has several genes for crown rust resistance. One gene was probably the one reported by Chang and Sadanaga (1964) to condition resistance to race 203. This gene was used in developing the large number of Victoria-derived cultivars that were released in the 1940s that later proved to be susceptible to the destructive Victoria blight, caused by *Helminthosporium victoriae* (Murphy and Meehan, 1946). Victoria also has one or more additional genes conditioning a high degree of resistance to certain races of crown rust that were not associated with susceptibility to *H. victoriae.* Additional genes conditioning moderate or low levels of resistance to many races of crown rust are also present.

The cultivar Landhafer was recognized during the 1930s as having potentially valuable resistance to crown rust and was subsequently used in the development of many important cultivars. Litzenberger (1949) reported its resistance to be controlled by a single dominant gene, which was verified by most other studies.

In the late 1950s the discovery of the great potential value of wild *Avena sterilis* from Israel and other parts of the Mediterranean and Near East (Wahl, 1970) led to investigations of this resistance. An early study (McKenzie and Fleischmann, 1964) of crosses between such wild-oat strains and a susceptible cultivar showed that each strain carried a single gene for resistance. In addition to these major genes for seedling resistance, the strains of *Avena sterilis* each had one or more genes for field resistance. Such results are typical of studies by other investigators.

A synopsis of studies of inheritance of resistance in terms of genes described has been compiled by Simons *et al.* (1978). This catalog lists 61 genes for crown rust resistance. Most of those reported since the early 1960s have been genes carried by *A. sterilis.* Most of the genes

listed have, or had, some special characteristic that makes them of value in breeding resistant oat cultivars. It is now apparent that there is little point in studying the genetics of resistance to *P. coronata* unless the resistance has some potential practical usefulness.

The failure of oat cultivars with single genes for resistance to give lasting control of crown rust prompted combining resistance genes in a cultivar. This had the obvious advantage of providing combined resistance to a greater range of rust races than could be achieved with a single gene. Also, such "stacking" of genes in a single cultivar would force the pathogen to mutate simultaneously at two loci to overcome the resistance of the cultivar. There was also a possibility that combined genes might be additive in effect (Finkner, 1954). The significance of such transgressive segregation under field conditions has not been investigated.

A knowledge of dominance is useful in transferring resistance genes to cultivars that are superior agronomically. The majority of studies of inheritance of resistance to *P. coronata* have reported resistance to be dominant or partially dominant. A lack of dominance is not rare, and Simons and Murphy (1954) illustrated the complexity of the situation. The degree of dominance exhibited seems dependent on the genic background involved. Dominance also varies with different races (Sebesta, 1979).

The possibility of linkage between resistance genes and genes governing other traits was studied by Osler and Hayes (1953). They found no linkage between resistance genes and genes for traits such as stem rust resistance, date of heading, number and length of basal hairs, percentage of lower florets awned, strength of awns, or plumpness of seed.

Allelism of genes for resistance is of importance as the resistances conferred by allelic genes cannot be combined in the same plant. The majority of studies have shown that genes for resistance in unrelated oats are generally at different loci. Exceptions, however, have been found (Upadhyaya and Baker, 1965).

b. Breeding. The history of breeding for resistance to *P. coronata* was reviewed from its beginnings until about 1960 by Coffman *et al.* (1961). The first resistant cultivar developed through hybridization was started in 1919, when Dietz crossed the stem-rust-resistant Richland with Green Russian. The cultivar Hawkeye, moderately resistant to *P. coronata*, resulted from this cross. A cross made in 1928 by Coffman between the susceptible Markton and the moderately resistant Rainbow resulted in Marion, which was the first oat cultivar to

combine resistance to *P. coronata* with resistance to other major pathogens.

The introduction of Victoria from Uruguay in 1927 provided a much better resistance than had been available to American breeders previously. By 1945, about 90% of the oat acreage of the Corn Belt and 75% of the acreage of the entire United States was planted to Victoria derived cultivars (Murphy, 1952). Then, the sudden appearance and devastating impact of *Helminthosporium victoriae* (susceptibility to which is linked to the *P. coronata* resistance of Victoria) rendered cultivars with the Victoria resistance virtually worthless in large areas of the country. Their decline in popularity was almost as rapid and complete as had been their original acceptance. They had, nevertheless, yielded large benefits during their brief period of popularity (Murphy, 1946) because of their resistance to *P. coronata.*

Races parasitizing Victoria were rare components of the fungus population in the 1930s (Murphy and Levine, 1936), and they did not dramatically increase in prevalence when the Victoria cultivars were being widely grown. Possibly they lacked fitness. They increased to dominate the population in the 1950s. This may have been associated with the growing of cultivars in the South that were derived from Victoria and that still made up an appreciable part of the acreage (Simons and Michel, 1958). The increase in prevalence of the Victoria races in the 1950s also was evident in Canada, where it was correlated with widespread commercial production of the cultivars Garry and Rodney, which were resistant to the older races but susceptible to the Victoria races (Fleischmann, 1963a). Some Victoria-derived cultivars had other Victoria genes. Some of these cultivars were widely grown after the Victoria cultivars susceptible to *H. victoriae* had disappeared. This was particularly true in Canada (Welsh *et al.*, 1953), but important cultivars of this type were also developed in the United States (Poehlman and Kingsolver, 1950). In general, these cultivars had less resistance than the *H. victoriae*-susceptible cultivars, but under field conditions they held up well where susceptible cultivars were severely damaged by *P. coronata.*

The cultivar Bond was introduced from Australia at about the same time as Victoria. The potential of its resistance to *P. coronata* was not recognized until later, but development of cultivars adapted to all the oat-growing areas of the United States carrying the Bond resistance were available to replace the Victoria cultivars. In 1950, the single Bond cultivar Clinton was grown on 75% of the oat acreage of the United States, and other cultivars with the same resistance made up much of the remaining acreage (Murphy, 1965). This very rapid in-

crease of the Bond cultivars provided a striking example of the effect of resistant cultivars on the *P. coronata* population. Races capable of parasitizing Bond were discovered in the 1930s, but remained very rare until the Bond cultivars appeared in commercial fields. At that point the increase in races attacking Bond and the consequent decrease in avirulent races paralleled the increase in acreage of Bond cultivars. The Bond-virulent races made up a trace of the population in 1943 when Bond cultivars were grown only in nurseries, but comprised over 90% of the fungus population in 1949 and 98% in 1950. A similar change was observed in Canada (Peturson, 1951).

Landhafer, an important source of resistance that was used in the United States in the 1950s because of its resistance to the Bond races, was found in Uruguay by Gassner (1916). It was a native strain grown in Uruguay for many decades, during which time it had become adapted to conditions of that region, including crown rust. Other cultivars were also known that were resistant to the races that attacked the Bond derivatives (Coffman *et al.*, 1961). Santa Fe, an introduction from South America, was the source of Clintafe, the first cultivar resistant to *P. coronata* to be developed by systematic backcrossing with the objective of developing a cultivar identical with an existing one, Clinton, except for resistance. Landhafer was used in the same way to develop the popular Clintland and related cultivars.

The appearance and subsequent spread of race 290 and similar races, which effectively parasitized both Landhafer and Santa Fe, largely nullified the value of the Landhafer and Santa Fe derivatives as resistant cultivars (Michel and Simons, 1966; Fleischmann *et al.*, 1963). Since then, no one source of resistance has dominated in plant breeding. This is partly because of the recognition of the futility of using a single resistance gene over a large geographic area, and because of the discovery that the wild *A. sterilis* of the Mediterranean and Near East comprised a rich and diverse source of genes for resistance. With many genes to choose from, there was no need to concentrate on a single source of resistance (Dinoor and Wahl, 1963), and strains of the wild *A. sterilis* have served as the primary sources of resistance in breeding programs since the late 1950s. In the relatively small area of the country of Israel alone, both seedling and adult plant resistance to races with wide virulence were prevalent and widespread (Wahl, 1970). Some form of resistance was found in over 30% of the wild plants collected in 446 locations. Resistance was much more common in the northern areas of the country, where *P. coronata* is common, than in the more arid southern areas where the fungus is rare.

Resistance of wild oat populations from other Mediterranean and

Near Eastern countries was also assayed. Martens *et al.* (1980) screened over 1400 oat accessions from Iran, Iraq and Turkey. Plants of the eight *Avena* species represented were tested as both seedlings and adult plants. Resistance was common in *A. barbata* from Turkey and in *A. sterilis* from all three countries.

Unfortunately, a linkage between resistance genes and yield may occur. Simons (1979) crossed a strain of *A. sterilis* known to carry a gene for resistance to *P. coronata* with a susceptible cultivated oat. In the disease-free plots, the mean yield of resistant selections was 23% less than the susceptible lines, suggesting a linkage of resistance and low yield. Individual lines combining resistance and high yield, however, were found. Frey and Browning (1971), in the process of developing isolines to be used as components of multiline cultivars, found resistance genes from two strains of *A. sterilis* to be associated with significant yield increases in the absence of *P. coronata*.

3. *Diploid and Tetraploid Oats*

Strains of diploid and tetraploid oats with potentially valuable resistance to *P. coronata* are easy to find (Simons, 1959). Such resistance has been used to a very limited extent due to the difficulty of transferring it to cultivated oats.

Simons *et al.* (1959) found each of three diploid strains to have a different single dominant gene. The resistance of a tetraploid oat was conditioned by a single gene without dominance. Another tetraploid strain derived from crossing a resistant diploid with a susceptible tetraploid (Zillinsky *et al.*, 1959) carried a single dominant gene. Other investigators (Marshall and Myers, 1961) found the resistance of diploid and tetraploid strains of oats generally conditioned by single genes.

One diploid line in particular, Saia, has been the subject of intensive study in an effort to transfer genes for resistance from diploid to cultivated oats. Zillinsky *et al.* (1959) crossed Saia with a susceptible tetraploid. Continued backcrossing to cultivated oats led to true-breeding hexaploid oats with the resistance of the diploid (Sadanaga and Simons, 1960). In the Iowa multiline program, lines with this resistance yielded slightly less than the recurrent parents, and they were dropped (Frey and Browning, 1971). Cytogenetic studies showed the presence of 21 pairs of chromosomes plus two fragments with the gene for resistance located on the fragments. Transmission of the fragments was somewhat irregular, and lines without them were susceptible and

yielded as well as the recurrent parent (Dherawattana and Sadanaga, 1973; Brinkman *et al.*, 1978).

By using thermal neutron radiation, Sharma and Forsberg (1977) were able to transfer resistance from Zillinsky's tetraploid to cultivated oats. The resistance gene was transferred from an alien chromosome to one in the hexaploid complement, and showed normal transmission.

4. General Resistance

a. Inheritance. The conspicuous failure of specific resistance stimulated interest in the use of general resistance. A knowledge of the inheritance of general resistance would be useful, but a very limited amount of information is available.

In segregating progenies from a cross between a resistant and a susceptible oat cultivar, Parker (1920) found that multiple factors were responsible for the resistance. Red Rustproof is typical of a group of cultivars having what we call general resistance. Luke *et al.* (1975) found that the resistance of Red Rustproof was controlled by a small number of genes showing slight partial dominance for susceptibility. Heritability was high, with a broad sense value of 87% making selection for this resistance possible.

Simons (1975) found heritability values of the resistance of four unadapted oat strains to range from 46 to 86% when measured in terms of yield reduction attributable to *P. coronata*, and 65 to 92% in terms of reduction in seed weight. The relationship of yield to resistance in the absence of rust was generally negative, and none of the lines combined maximum yield with maximum resistance. Thus manipulation of general resistance in breeding programs will require large populations in spite of the heritability values.

Kiehn *et al.* (1976) showed that the resistance of two strains of *A. sterilis* was controlled by a number of minor recessive genes having additive effects, a type of inheritance commonly associated with general resistance.

b. Pathogenic Specialization. General resistance to *P. coronata* is thought to be less subject to the vagaries of pathogenic specialization than is specific resistance. Because of the difficulty of testing this hypothesis, few studies have been done. Peturson (1944) found that five oat cultivars, susceptible in the seedling stage, differed in resistance in the adult stage. Some were resistant to all races tested.

Others were resistant to some and susceptible or moderately susceptible to others. Simons (1961) found that cultivars having useful sources of general resistance are clearly susceptible to certain races. It now appears unlikely that a source of general resistance will be discovered that will be effective against all forms of *P. coronata.*

c. Manner of Expression. In contrast to specific resistance, general resistance to *P. coronata* is expressed in many different ways. Durrell and Parker (1920) observed that resistance was expressed as a low percentage of infection from a given quantity of spores, and by a long incubation period. Differences among cultivars for receptivity—that is, the relative numbers of uredia resulting from equal amounts of inoculum—can be striking. In Australia, the number of pustules produced on the cultivars Algerian, Garry, and a strain of *A. sterilis* were 124, 76, and 9, respectively (Kochman and Brown, 1975). Luke *et al.* (1981) regarded low receptivity as a major component of slow rusting, and showed that low receptivity was expressed at low but not at higher levels of infection.

Heagle and Moore (1968) compared the highly susceptible Coachman cultivar with Portage, which has some degree of general resistance. Isolates of *P. coronata* virulent on seedlings of both cultivars did not produce epidemics in pure stands of Portage, but did in Coachman. When the infection processes on adult plants were compared, there were fewer penetrations, hyphal growth rates were slower, onset of sporulation was delayed, and fewer spores were produced per pustule in Portage than in Coachman.

Adult plants of cultivars having general resistance exhibit variation in reaction to *P. coronata;* the younger tissues and the younger plants appear most susceptible, and the older tissues and older plants most resistant (Murphy, 1935; Newton and Brown, 1934).

The epidemiological significance of general resistance was shown by Berger and Luke (1979). They compared the cultivars Fulghum and Burt (susceptible) with Red Rustproof-14 (slow-rusting). The average apparent infection rates on Fulghum, Burt, and Rustproof-14 were 0.4, 0.35, and 0.2 units/day, respectively. When progress of the disease was measured in terms of isopath movement, the rates for Fulghum, Burt, and Red Rustproof-14 were 0.9, 0.4, and 0.35 m/day, respectively.

d. Use in Breeding. General resistance is of potential value in the control of *P. coronata,* but the complexity of its inheritance coupled with difficulties associated with evaluating it delay practical application. To determine the feasibility of manipulating it in breeding pro-

grams, Simons (1981b) crossed, and backcrossed, 14 lines of *A. sterilis* known to have general resistance with the susceptible cultivar Clinton. Lines derived from F_2, Bc_1, and Bc_2 plants were selected for cultivated plant type. In terms of reduction in grain weight attributable to disease, 71 of the lines derived from F_1 plants, 52 from Bc_1 plants, and 27 from Bc_2 plants were siginificantly more resistant to *P. coronata* than was the parental Clinton. Lines combining the yield of the cultivated Clinton with a statistically significant improvement in resistance appeared in all but one population.

5. *Multilines*

There is considerable interest in the use of multiline cultivars of various crops to achieve diversity and thereby longer lasting genetic protection from various plant diseases. Much of the work has been done with the crown rust disease. Jensen (1952) noted that a risk of serious loss from *P. coronata* had been created because the fungus could move freely from oat field to oat field without being checked by genetic barriers in the form of resistant cultivars. He suggested a form of intravarietal diversity in which a cultivar would consist of a blend of lines having different resistance genes. The component lines could then be changed from year to year to meet changes in the *P. coronata* population. Borlaug (1965) proposed that such composite cultivars be produced by backcross methods. The component lines would be developed by backcrossing a current commercial cultivar to a number of different types of resistance. The resulting lines would be increased separately, and then mixed to form the cultivar. Such a multiline cultivar would be morophologically uniform, but as the rust races change, individual lines could be changed.

Cournoyer (1970) tested the multiline hypothesis experimentally by using large plots planted to oats consisting of various mixtures of resistant near-isogenic lines. Spores were trapped daily adjacent to the plots, and the final cumulative spore count showed that incorporation of resistant plants decreased the number of spores produced. A greater proportion of resistant plants resulted in fewer spores being produced. Since spore production and disease severity are directly correlated, Cournoyer concluded that her results supported the multiline hypothesis that mixtures of near-isogenic lines effectively buffer the oat population against the pathogen population.

The first commercial multiline cultivars were developed in Iowa and released in 1968 (Browning and Frey, 1981). In the years following, a total of eight early and five midseason multiline cultivars were re-

leased. They were well received by farmers and were widely grown for several years before declining in popularity due to the scarcity of *P. coronata* during the period, and to the appearance of new cultivars agronomically superior to the multilines. As this is written, a new multiline is in the final stages of development in Iowa. The cultivar Lang, which is high-yielding and resistant to barley yellow dwarf virus, is the recurrent parent.

Politowski and Browning (1978) found that multiline cultivars developed much less disease than susceptible pureline cultivars. There was only slightly more disease on the multiline cultivars than on cultivars that were resistant to the *P. coronata* isolates that were present. The amount of reduction in yield and seed weight attributable to infection corroborated the observations on incidence of disease.

The early multiline studies were done in Iowa, where the disease season is severe but short. Oats are grown over a much longer disease season in the southern United States. A study under long disease seasons compared the performance of multiline and pureline cultivars at two locations on the southern coastal plain of Texas, Browning and Frey (1981). There, *P. coronata* overwinters in the uredial stage on oats, and severe epidemics developed in the large isolated plots that were used. Relative yields of spores from multilines and from susceptible isoline checks were the same in Texas as in the North, showing that multilines will protect oats from *P. coronata* in long as well as short disease seasons. Disease was less severe at one of the locations, but even there, susceptible pureline check cultivars were killed prematurely by the fungus. Both the multiline and an artificial cultivar mixture in the ratio of one resistant to two susceptible developed no more rust than did the fungicide-sprayed control plots.

Indirect evidence substantiating the value of a relatively small proportion of resistance in a multiline comes from a study of mass selection for resistance (Tiyawalee and Frey, 1970). The frequency of resistance genes was increased from 0.21 in the F_3 to about 0.35 in the F_{10}. Most of the increase occurred in the first three cycles of selection, suggesting that damage from *P. coronata* was negligible when about one-third of the plants carried resistance genes.

The mechanism by which multilines are protected from damage is not completely understood, but it is assumed to result from any plant being resistant to some of the spores that land on it. Thus a portion of the inoculum that would contribute to build-up of the epidemic in a pureline cultivar is removed from contention, resulting in slower development of disease. A given plant in the multiline also serves as a mechanical barrier to the spread of spores to which it is resistant. Tani

et al. (1980) demonstrated a cross-protection effect from inoculation of oat cultivars with spores of incompatible isolates of *P. coronata*. Since plants in a multiline cultivar are regularly exposed to inoculum of incompatible isolates, they may benefit from this cross-protection effect.

6. *Gene Deployment*

The geographical deployment of genes for resistance has been considered as a means of controlling *P. coronata*. Jensen and Kent (1963) noted that fields of oat cultivars susceptible to *P. graminis* were not severely damaged by this pathogen when they were interspersed among more numerous fields of resistant cultivars. They suggested that a series of cultivars representing a diversity of genes for resistance to *P. coronata* be developed and used. These cultivars would be randomly planted in different fields. Most fields in a given area would thus contain plants resistant to the majority of the spores that might fall on them.

Browning *et al.* (1969) noted that with the eradication of buckthorn in the north central United States, *P. coronata* was initiated mainly by wind-borne urediospores from the South. *Puccinia coronata* in the South, in turn, is initiated, they assumed, by spores produced further north during the summer. They suggested that available genes for resistance be deployed so that those genes used in cultivars in the northern states and Canada would differ from those used in the southern states and Mexico. Thus when spores were blown from one region to the other they would encounter only cultivars on which they would be avirulent, and the cycle would be broken. The geographical deployment of resistance genes also figured importantly in a comprehensive management scheme to control *P. coronata* in the United States and Canada that was described by Frey *et al.* (1980).

7. *Pyramiding of Resistance Genes*

The combination of two or more resistance genes in the same cultivar, sometimes referred to as stacking or pyramiding resistance genes, to control *P. coronata* is theoretically attractive. Mutations in the fungus population for virulence toward a resistance gene are rare events, and the occurrence of two simultaneous mutations necessary to overcome the resistance of such a cultivar would be unlikely. An examination of races that have appeared in the past, however, suggests that pyramiding genes for resistance in the same cultivar may not guarantee long-lasting protection (Simons *et al.*, 1957). During the ear-

ly 1950s resistance genes carried by Landhafer and Santa Fe were widely used in breeding programs. At that time, no race was known that could parasitize either of them. In the mid-1950s races appeared that were virulent on the combination, but no race was ever found that could parasitize only one. Thus, combination of genes for resistance would not have prevented the breakdown of the resistance.

8. *Telia and Resistance*

In areas where the fungus is dependent on the alternate host, telia are obviously essential to survival. In many of the major oat-growing regions of the world, the alternate host has either little or no role in the epidemiology of the disease. The production of telia under such circumstances merely ends the repeating uredial phase of the disease. Thus plant pathologists tend to regard the formation of telia as an indication of cultivar resistance.

Parker (1918) believed that telia were a sign of resistance as they did not form on highly susceptible cultivars. In Murphy's (1935) more detailed observations, the early development of telia was positively correlated with host resistance. Races showing restricted virulence generally develop telia more readily.

Zimmer and Schafer (1960), however, found that rapidity of telia formation was not correlated with range of virulence, specific virulence of the pathogen, or with maturity of the host. They believed telia formation was a manifestation of a specific relationship between a cultivar and an isolate of the fungus. They noted that some cultures did not produce telia under any of the experimental conditions they worked with, and suggested that some isolates of the rust may have lost the capacity to produce telia under any conditions.

9. *Tolerance*

Tolerance to *P. coronata* was defined by Caldwell *et al.* (1958) as that capacity of a susceptible plant that enabled it to endure severe attack without sustaining severe losses in yield or quality. A clear distinction was made between tolerance and intermediate or lesser degrees of resistance. Theoretically, tolerance should be more stable than resistance. If a pathogenic race of the fungus arises to which the previously tolerant cultivar was not tolerant, the result would be greater injury to the host, but with no consequent increase in the relative rate of increase of the new race. This eliminates the screening mechanism whereby the new race gains an advantage over the established races. Caldwell *et al.* (1958) used two pairs of oat cultivars (Clinton-59 and

Clintland, and Benton and Bentland) identical except that Clintland and Bentland had a resistance gene. In the severe epidemic of 1957, infection reached 100% on the susceptible Benton and Clinton-59, while only a trace of *P. coronata* developed on Bentland and Clintland. Under these conditions Benton yielded 14% less than Bentland, and Clinton-59 yielded 54% less than Clintland. Since both Benton and Clinton-59 were equally infected, the difference between the 14 and 54% losses reflected the greater relative tolerance of Benton.

Simons (1966a) discussed the difficulties involved in the use of tolerance in breeding programs and concluded that the reduction in seed weight attributable to *P. coronata* was the best measure of tolerance, with reduction in yield a useful adjunct. To separate inherent differences in seed weight and yield from differences due to tolerance, field tests were planted in a split-plot design in which half the plot was infected and the other half protected by a fungicide. Data were expressed as ratios of diseased to nondiseased plot pairs. This system, even with hill plots, was effective for measuring small differences in tolerance among unrelated cultivars, and it was equally useful for measuring intermediate or lesser degrees of resistance that are difficult to evaluate visually.

Wahl (1958) observed that although *A. sterilis* was widespread and abundant in Israel and was often heavily infected by *P. coronata*, it did not seem to suffer from the disease. Simons (1972) crossed three strains of *A. sterilis* that were susceptible with the highly susceptible cultivars Richland and Clinton. Cultivated-type, seemingly rust-susceptible segregates selected from the resulting populations were evaluated for tolerance. The amount of tolerance transmitted by the different *A. sterilis* parents varied significantly as estimated by the mean tolerance values of the lines derived from each parent. Most lines derived were more tolerant than the cultivated parent. Heritability of tolerance was estimated to be 76%, and most lines were lower in yield, under rust-free conditions, than the cultivated parents.

Politowski and Browning (1978) further refined the concept of tolerance and showed that dilatory resistance—i.e., resistance that delays the progress of an epidemic—may not be visually apparent. To differentiate between dilatory resistance and tolerance, they compared final cumulative spore counts of *P. coronata* from large plots with host yield and seed weight data from hill plots. Yield and seed weight of Otter and Cherokee were depressed by about the same amount. Otter, however, produced more spores than did Cherokee, and thus was regarded as more tolerant. Both Otter and Cherokee had a degree of dilatory resistance when compared with the susceptible check, which had higher

spore counts and whose yield and kernel weight were reduced more than were those of Otter or Cherokee. Singleton *et al.* (1982) found that the yields of some moderately resistant cultivars equalled or exceeded the yields of highly resistant cultivars under severe epidemic conditions, even though the moderately resistant cultivars were more heavily infected.

Genetic studies (Simons, 1969) showed that tolerance was inherited as a complex quantitative trait in two crosses. Heritability of yield and seed weight response to infection was sufficiently high to permit effective selection in breeding programs.

10. Mutation

All host genes for resistance to *P. coronata* presumably originated as mutations. Rosen (1955) found a single disease-free plant in a field of oats that was otherwise heavily infected with *P. coronata*. Circumstances and the characteristics of the resistance of this plant were such that it appears mutation is its likely source.

Most of what is known about mutation in oats for resistance to *P. cornoata* was discovered as a result of applying artificial mutagenic agents to susceptible oat cultivars. Frey (1954) X-rayed the susceptible cultivar Huron and obtained a few strains with increased field resistance to *P. coronata*. Luke *et al.* (1958) radiated Floriland with thermal neutrons and isolated lines with field resistance to the virulent race 264. These lines were used in developing the cultivars Florad and Florida 500 (Sechler and Chapman, 1965).

Atkins *et al.* (1964) used both neutrons and X-rays in varying dosages to induce mutations for resistance to *P. coronata*, and one cultivar, Alamo-X, resulted.

Ethyl methane sulfonate (EMS) was used to induce tolerance in susceptible cultivars by Simons (1971). A few lines were obtained that were superior to the control in both yield and seed weight response to infection, but in the absence of *P. coronata* these lines yielded less than the control. Seeds derived from EMS-treated seed of Clintland-60 were retreated with EMS (Simons, 1981a). Lines from this treatment were retested for tolerance. Data suggested that tolerance could be obtained with high yield.

B. ERADICATION OF *RHAMNUS*

The role of the alternate host in the initiation of infection by *P. coronata* on oats early in the spring and its role in the origin of new

races through genetic recombination are well known. Thus, eradication of *Rhamnus* is a measure to help control the disease. Historically, an early record of such a specific recommendation was published by von Thumen (1886). He believed that eradication of *Rhamnus* would be an effective but incomplete control measure. Sivers (1887) stated that eradication of *Rhamnus* probably would not be carried out, and indeed in some areas would not even be feasible.

Dietz (1930) surveyed species of *Rhamnus* in the north central United States. He estimated that there were several hundred thousand bushes growing in the upper Mississippi valley, and obviously, the simple physical problem of locating and destroying them would be expensive. A limited goal of eradicating buckthorn hedges adjacent to fields in which oats might be planted is often economically feasible. Several states of the United States and a number of other countries have enacted legislation aimed at promoting eradication of *Rhamnus*. Exact figures are not available, but the combination of legislation and educational programs to make farmers aware of the benefits of eradication has resulted in a great reduction in *Rhamnus* in areas such as Iowa.

C. ERADICATION OF OTHER GRAMINEOUS HOSTS

The susceptibility of wild and volunteer oats and certain grass species to strains of *P. coronata* that infect cultivated oats make them a frequent source of inoculum. Straib (1937a) believed that such infected plants were reservoirs of infection and acted as the primary source of inoculum in certain situations in Germany, and there are other areas in the world where eradication of such plants would be beneficial.

Grasses that are susceptible to *P. coronata* are rarely important in the initiation of *P. coronata* on oats, and therefore eradication of them normally would not be recommended.

D. DATES OF PLANTING AND MATURITY

Early-planted oats often ripen before the fungus has time to affect them seriously. Sivers (1887) was clearly aware of the value of early seeding, and states that early seeding was the best means available of avoiding damage from *P. coronata*. Simons (1966b) showed that the effect of *P. coronata* on yield and grain quality increased significantly as planting data was delayed. This effect was most pronounced with highly susceptible oat cultivars.

Fleischmann and McKenzie (1965) found that under conditions of artificially initiated crown rust, early- and late-seeded plantings of the commercially important cultivar Garry suffered 28 and 50% yield losses, respectively.

Early-maturing oat cultivars tend to be less damaged by *P. coronata* than do later cultivars. This is probably because the early cultivar ripens before the disease has had time to severely damage it. This correlation of relative maturity to damage caused by *P. coronata* was demonstrated by Simons and Michel (1968).

E. FERTILIZATION

Pammel (1892) noted that where oats were rank and thick, rust was severe, but where thin the rust was less severe. Gassner and Hassebrauk (1934) showed that nitrogen tends to favor crown rust whereas potassium reduces it. At present, the feeling is that any kind of a fertilization program resulting in vigorously growing high-yielding oats will permit severe damage to susceptible cultivars when other conditions favor the disease.

F. FUNGICIDES

Puccinia coronata on oats has been used as the experimental system in several studies of the potential usefulness of modern fungicides (Chin *et al.*, 1975; Prusky *et al.*, 1981). Currently, the relatively low acre value of oats, coupled with the infrequent occurrence of severe damage from *P. coronata*, makes the use of fungicides on commercial fields generally impractical. Fungicides may be used effectively in special situations such as experimental work and production of seed.

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