Rust Diseases of Wheat

Concepts and Methods of Disease Management

RUST DISEASES OF WHEAT

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CONCEPTS AND METHODS OF DISEASE MANAGEMENT

CIMMYT is an internationally funded, nonprofit scientific research and training organization. Headquartered in Mexico, the Center is engaged in a worldwide research program for maize, wheat, and triticale, with emphasis on improving the productivity of agricultural resources in developing countries. It is one of 16 nonprofit international agricultural research and training centers supported by the Consultative Group on International Agricultural Research (CGIAR), which is sponsored by the Food and Agriculture Organization (FAO) of the United Nations, the International Bank for Reconstruction and Development (World Bank), and the United Nations Development Programme (UNDP). The CGIAR consists of a combination of 40 donor countries, international and regional organizations, and private foundations.

CIMMYT receives core support through the CGIAR from a number of sources, including the international aid agencies of Australia, Austria, Belgium, Brazil, Canada, China, Denmark, Finland, France, Germany, India, Italy, Iran, Japan, Mexico, the Netherlands, Norway, the Philippines, the Republic of Korea, Spain, Switzerland, the United Kingdom, and the USA, and from the European Economic Commission, Ford Foundation, Inter-American Development Bank, OPEC Fund for International Development, UNDP, and World Bank. CIMMYT also receives non-CGIAR extra-core support from the International Development Research Centre (IDRC) of Canada, the Rockefeller Foundation, and many of the core donors listed above.

Correct Citation: Roelfs, A.P., R.P. Singh, and E.E. Saari. 1992. Rust Diseases of Wheat: Concepts and methods of disease management. Mexico, D.F.: CIMMYT. 81 pages.

ISBN: 968-6127-47-X

Printed in Mexico

AGROVOC descriptors: Disease control, plant diseases, rusts, *Triticum* spp., wheat.

AGRIS category codes: H20, F01.
Dewey decimal classification: 632.425.

Design: Miguel Mellado Enciso

Design assistants: Eliot Sánchez P., Ma. Concepción Castro A., J. Manuel Fouilloux, and J. Luis Delgado.

Cover photos: Sergio Pastén and Gene Hettel.

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ABSTRACT

Roelfs, A.P., R.P. Singh, and E.E. Saari. 1992. Rust Diseases of Wheat: Concepts and methods of disease management. Mexico, D.F.: CIMMYT. 81 pages.

The rust diseases of wheat are among the most studied of the plant diseases. Since Tozzetti and Fontana in 1767, there has been almost an endless list of scientific publications concerning the rust pathogens, the rust diseases, and rust resistance.

This publication reviews some of the more recent scientific literature concerning the pathogens *Puccinia recondita* f.sp. *tritici*, *P. graminis* f.sp. *tritici*, and *P. striiformis* f.sp. *tritici*; the diseases leaf rust, stem rust, and stripe rust; and the resistance to the pathogens. The goal is to provide a single source of information for isolated scientists and new students.

A brief history and general description of the wheat rusts are followed by a detailed summary of each of the rust diseases, their epidemiology, their hosts (including resistance), and their pathogens (including virulences). Methods for disease control through resistance, chemicals, and cultural methods are discussed. Techniques are presented for inoculum production, collection, and storage; inoculation methods; disease scoring; testing for resistance; epidemiology, yield loss, and physiologic race studies; isolation of resistance genes; and utilization of resistance.

Other Manuals in this Series

Stubbs, R.W., J.M. Prescott, E.E. Saari, and H.J. Dubin. 1986. Cereal Disease Methodology Manual. Mexico, D.F.: CIMMYT.

Eyal, Z., A.L. Scharen, J.M. Prescott, and M. van Ginkel. 1987. The Septoria Diseases of Wheat: Concepts and methods of disease management. Mexico, D.F.: CIMMYT.

PREFACE

This publication is a much needed reference concerning the three rust diseases of wheat (leaf, stem, and stripe). It represents one of a series of wheat disease technical manuals being developed by CIMMYT.

In 1976, a "Cereal Disease Methodology Manual" was contemplated and it finally materialized in 1986. Shortly after its publication, a group of individuals conceived the idea of a series of more focused manuals devoted to specific diseases and aimed at a developing country audience. Drs. H.J. Dubin, A.R. Klatt, J.M. Prescott, E.E. Saari, R.W. Stubbs, and E. Torres should be especially acknowledged for getting this series of manuals on track. In 1987, the award-winning "Septoria Diseases of Wheat" was published. Now, in 1992, the rust manual becomes the third contribution to the series. A manual devoted to the smuts and bunts of wheat is in the early development stages and scheduled for publishing in 1993.

The wheat rusts, historically, have been diseases of great importance. The losses caused by these three diseases worldwide over the centuries have been substantial. In the developing world, they are still considered the diseases of major significance. Unfortunately, in many instances, recording and quantification of the rust diseases and the losses attributed to them have not been adequate. All too often, references to their occurrence appear in obscure publications and only when an epidemic is unusually severe.

Over the past 20 to 30 years, much progress has been made in relation to breeding for resistance to the three diseases. However, it should be emphasized that because of ever evolving virulence in these pathogens, the rust diseases remain ones of concern and importance. If continuous monitoring and an active effort to maintain resistance level and improve its durability are not ongoing, the resurgence of the diseases is virtually assured.

This manual provides an extensive literature review of the subjects (more than 400 citations) and a summation of practical information available. There are a number of publications and reviews available, but no single reference summarizes the concepts and how to apply them to the management of the disease(s). This publication also brings together the principles and methods used by wheat rust workers. It deals with the important elements required to understand the complexities of the diseases and provides instructions and examples on how to deal with them. The information here is intended for rust workers in developing countries, but I am certain it will be equally well received in the developed countries. This manual will serve as a valuable guide in the efforts to manage these "shifty enemies."

R.A. Fischer
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The Wheat Rusts

History

Early records indicate that wheat was affected by blight, blasting, and mildew, which are now assumed to be at least, in part, due to the rust fungi. Aristotle (384-322 B.C.) writes of rust being produced by the "warm vapors" and mentions the devastation of rust and years when rust epidemics took place. Theophrastus reported that rust was more severe on cereals than legumes. Excavations in Israel have revealed urediniospores of stem rust that have been dated at about 1300 B.C. (177). "Stern Robigo, spare the herbage of the cereals, ...withhold we pray thy roughening hand..." was part of the official prayer at a Robigala ceremony as given by Ovid (43 B.C.-17 A.D.) and gives the impression that stem rust was a serious disease in Italy during that time.

The Italians Fontana and Tozzetti independently provided the first unequivocal and detailed reports of wheat stem rust in 1767 (106, 382). In 1797 Persoon named the causal organism of wheat stem rust *Puccinia graminis*. In 1946 Chester (67) provided one of the first detailed histories of published literature on the rusts.

In the early records, wheat leaf rust is not distinguished from stem rust (67). However, by 1815 de Candolle (79) had shown that wheat leaf rust was caused by a distinct fungus and described it as *Uredo rubigo-vera*. The pathogen underwent a number of name changes until 1956 when Cummins and Caldwell (74) suggested *P. recondita*, which is the generally used nomenclature today.

Although Gadd first described stripe rust of wheat in 1777, it wasn't until 1896 that Eriksson and Henning (99) showed that stripe rust resulted from a separate pathogen, which they named *P. glumarum*. In 1953 Hylander et al. (154) revived the name *P. striiformis*.

The Diseases

Of the three rust diseases of wheat, the most common is called leaf or brown rust. It occurs on the leaf blades, although leaf sheaths can also be infected under favorable conditions, high inoculum densities, and extremely susceptible cultivars. It frequently lacks the abundant teliospore production of stem rust at the end of the season, resulting in a brown leaf lesion rather than a black stem lesion that occurs with stem rust. When leaf rust teliospores are produced, they usually emanate from telia on the lower leaf surfaces, which remain covered by the epidermal cells. The disease develops rapidly at temperatures between 10 and 30°C. Leaf rust occurs to some extent wherever wheat is grown. Losses in grain yield are primarily attributed to reduced floret set. In severe epidemics with moisture stress, shriveling of the grain occurs. In rare genotypes, florets, tillers, and plants can be killed by early (pre-heading) epidemics. Losses due to leaf rust are usually small (< 10%), but can be severe (30% or more).

Stem rust is also known as black rust or summer rust due to the abundant production of shiny black teliospores, which form in the uredinium at the end of the season or with unfavorable conditions. Stem rust is favored by humid conditions and warmer temperatures of 15 to 35°C. It is the most devastating of the rust diseases and can cause losses of 50% in 1 month when conditions for its development are favorable. Losses of 100% can occur with susceptible cultivars.

Stripe or yellow rust is principally a disease of wheat grown in cooler climates (2-15°C), which are generally associated with higher elevations, northern latitudes or cooler years. It takes its name from the characteristic stripe of uredinia that produce yellow colored urediniospores. Because of the disease's early attack, stunted and weakened plants often occur. Losses can be severe (50%) due to shriveled grain and damaged tillers. In extreme situations, stripe rust can cause 100% losses.

Tables 1 and 2 summarize primary hosts, alternate hosts, symptoms, and generally accepted environmental conditions needed by the three rust diseases.

Table 1. The rust diseases of wheat, their primary and alternate hosts, and symptoms.

Disease	Pathogen	Primary hosts	Alternate hosts	Symptoms
Leaf rust	*Puccinia recondita* f.sp. *tritici*	Bread & durum wheats and triticale	*Thalictrum, Anchusa Isopyrum*, and *Clematis*	Isolated uredinia on upper leaf surface and rarely on leaf sheaths
Stem rust	*Puccinia graminis* f.sp. *tritici*	Bread & durum wheats, barley, and triticale	*Berberis vulgaris*	Isolated uredinia on upper and lower leaf surfaces, stem, and spikes
Stripe rust	*Puccinia striiformis* f.sp. *tritici*	Bread & durum wheats, triticale, and a few barley cultivars	unknown	Systemic uredinia on leaves and spikes and rarely on leaf sheaths

Epidemiology

There are several areas worldwide in which each of the rusts can cause severe losses (326). In other areas the environment is marginally suited for the diseases. In such areas, the disease is severe only in years when:

- Conditions are unusually favorable.
- Susceptible cultivars are grown.
- Cultural practices are altered.
- The above factors occur in combination.

Table 3 provides a general summary of the current and historical importance of the rust diseases worldwide.

Urediniospores of the wheat rusts initiate germination within 1 to 3 hours of contact with free moisture over a range of temperatures depending on the rust. Urediniospores are produced in large numbers and can be blown considerable distances by the wind (149, 392). However, most urediniospores are deposited close to their source (309) under the influence of

Table 2. Environmental conditions required for the wheat rusts.

	Temperature (°C)				
Stage	Minimum	Optimum	Maximum	Light	Free water
Leaf rust					
Germination	2	20	30	Low	Essential
Germling	5	15-20	30	Low	Essential
Appressorium		15-20		None	Essential
Penetration	10	20	30	No effect	Essential
Growth	2	25	35	High	None
Sporulation	10	25	35	High	None
Stem rust					
Germination	2	15-24	30	Low	Essential
Germling		20		Low	Essential
Appressorium		16-27		None	Essential
Penetration	15	29	35	High	Essential
Growth	5	30	40	High	None
Sporulation	15	30	40	High	None
Stripe rust					
Germination	0	9-13	23	Low	Essential
Germling		10-15		Low	Essential
Appressorium			(not formed)		
Penetration	2	8-13	23	Low	Essential
Growth	3	12-15	20	High	None
Sporulation	5	12-15	20	High	None

gravity. The terminal velocity of urediniospores in still air is approximately 1 cm/sec (385). It takes a spore about 8 hours and 20 minutes to fall 300 m. For spores that escape the crop canopy, only about 10% are still airborne in that plane after 100 m (294). Gregory's (124) equations probably adequately describe the depletion rate of spore concentrations between 1 and 100 m from the source. Spore impaction is probably an important mechanism of deposition at these distances. At greater distances from the source, most urediniospores will remain airborne until scrubbed from the air by rain (124, 259, 314, 323).

Table 3. Current (C) and historical (H) importance of wheat leaf, stem, and stripe rusts for the epidemiological zones of Saari and Prescott (326).

	Leaf Rust		Stem rust		Stripe Rust	
Zone	**C**	**H**	**C**	**H**	**C**	**H**
Africa						
North	Major	Major	Local	Major	Local	Local
East	Local	Local	Major	Major	Major	Major
Southern	Local	Local	Local	Major	Rare	Rare
Asia						
Far East	Local	Local	Local	Major	Major	Major
Central	Major	Major	Minor	Minor	Local	Local
South	Local	Major	Minor	Major	Local	Local
Southeast	Major	Major	Minor	Minor	Rare	Rare
West	Local	Local	Local	Major	Major	Major
Australia, New Zealand	Local	Local	Local	Major	Local	Rare
Europe						
East	Major	Major	Minor	Major	Local	Local
West	Local	Major	Minor	Major	Major	Major
North America	Major	Major	Minor	Major	Local	Local
South America	Major	Major	Local	Major	Local	Local

Major = severe losses without the cultivation of resistant varieties; minor = usually occurs, but of little significance; local = only occurs in a small part of the region, losses in these areas may be occasionally severe if susceptible cultivars are grown; rare = not present, rarely seen, or as in Australia and New Zealand recently introduced.

Urediniospores are relatively long-lived and can survive in the field away from host plants for periods of several weeks (139, 140, 254, 272, 366). Urediniospores can withstand freezing if their moisture content is lowered to 20 to 30%. Viability rapidly decreases at moisture contents of more than 50%.

Long-distance spread of urediniospores is influenced by latitude and the respective wind patterns. In general, spores move west to east due to the winds resulting from the rotation of the earth. At progressively higher latitudes winds tend to take a more southerly component in the Northern Hemisphere and a northerly component in the Southern Hemisphere. Studies in the USA (299) show spore movements to be from the southwest to northeast, north of 30° latitude. In the Southern Hemisphere, because most of the wheat areas and land masses, in general, are north of 30°S latitude, the movement is more west to east (211). However, over a period of years, barley stripe rust moved south and eastward across South America (84). In India spores move southward probably as a result of katabatic wind flows from the mountains into the plains (261). In most areas studied, spores produced in the upper levels of the crop canopy move into a geographical area where the crop phenology is less advanced. Long distance transport is often supposed for the initiation of rust diseases, but the critical evidence is often lacking to separate endemic inoculum from exodemic sources (404).

Under favorable conditions, urediniospores are most likely to be present above the crop canopy in high numbers. For example, 10,000 urediniospores/cm^2 were caught in 5-mm diameter rod impacting traps when a particular day was clear, hot (>25°C), and dry (relative humidity <30%) with moderate winds (5 m/sec) and no rainfall in the previous 24-48 hours. Spore numbers trapped the previous day were

moderate (500 to 1000/cm^2) indicating that 2 days of high urediniospore production seldom occur in sequence (305).

Hot days cause the air to rise from inside the canopy. When the humidity is high, fewer spores leave the uredinia. Low wind velocities dry the canopy, agitate the leaves, and free the spores from the uredinia. High wind velocities may result in the release of more spores, but such winds rapidly dilute the concentration above the canopy and may be more important in generating long-distance transport than in local spread. Rain favors disease by scrubbing spores from the air, depositing them on the plants, and increasing the humidity. However, rain can also wash spores from the plant surfaces and high humidity restricts spore movement. The change in temperature due to rain will influence disease progress.

Host-Pathogen Interactions

Host-pathogen interactions can be divided into at least two categories: specific and nonspecific. Specific interactions occur when a single pathogen isolate interacts with a single host genotype to produce a different disease response than another isolate with the same host in the same environment. Nonspecific interactions occur when all isolates result in a similar response on a given host genotype. Nonspecific resistance is theorized to be the better resistance to use in a breeding program. However, to prove nonspecificity, every member of the pathogen population would need to be evaluated, which, of course, is impossible.

Specific interactions

The specific-type interactions provide the basis for the gene-for-gene theory (104). Readers interested in more detail than presented here should see references 48, 201, 247, 279, 303, and 307. In these gene-for-gene relationships, three assumptions have often been made; none of these are always true. The first is that specific resistance is due to dominant genes in the host; *Sr12* and *Sr17* are exceptions (243, 346). The second is that dominance is complete; this has not been true for many stem rust resistances (307). The third is that avirulence is dominant; in limited studies exceptions are common where avirulence is recessive (307). However, for ease in explaining specific interactions, the example used (Figure 1) has dominant resistance (RR) and avirulence (AA).

Resistance of a cultivar to an isolate is a genetic character. Therefore, a cultivar never loses its resistance to that isolate. With some temperatures, inoculum densities, light intensities, host nutrition levels, host growth stages or tissues, the resistance may be ineffective or not expressed, but the resistance gene remains. A cultivar may be resistant to one isolate and susceptible to another, and conversely an isolate may be virulent on one cultivar and avirulent on another (Figure 1).

Figure 1. The gene-for-gene interaction expressed as infection types between a single host resistance gene and a single pathogen virulence gene.

	Host		
Pathogen	**RR**	**Rr**	**rr**
AA	Low	Low	High
Aa	Low	Low	High
aa	High	High	High

Infection types are the visible response of the interaction of the host, the pathogen, and the environment. Seedling infection types are generally scored on a 0 to 4 scale for leaf and stem rusts (297) (see Table 21, page 43) and a 0 to 9 for stripe rust (248) (see Table 22, page 43). In selecting useful resistances, infection types 3 and 4 (on the 0 to 4 scale) and 7, 8, and 9 (on the 0 to 9 scale) are often considered to indicate a compatible host-pathogen interaction. The compatible interaction is considered inadequate for commercial use. However, in genetic studies any low infection type, even a 3 in the case of leaf or stem rust, indicates some level of resistance when the host line without the gene results in an infection type 4. The lower infection type reflects the degree of incompatibility between the host and pathogen in that environment.

The expression of incompatibility can occur early in the disease process and may result in an immune response, or incompatibility may be expressed slowly at the end of the process causing only a slight reduction in sporulation. The lower infection types are generally quite characteristic for a particular host-pathogen-environment-time interaction.

If two specific resistance genes are present in the same host line, the infection type produced by an isolate avirulent on both genes is, generally, that of the most effective gene. Thus, if a line with *Sr6* (low infection type 0;) and *Sr8a* (low infection type 2) is inoculated with an isolate avirulent on both genes, the infection type observed is a "0;" conditioned by *Sr6*.

Figure 1 is a simplification because any host with one gene pair for resistance must have many gene pairs for susceptibility. In

Notes:

the case of stem rust, there are more than 50 resistance genes and likewise the pathogen must have more than 50 avirulence/virulence gene pairs. Note that in this example, the host genotype (rr) is susceptible (high infection type) to all three of the possible pathogen genotypes specific for that host gene, even for the so-called 'avirulent' isolate (aa). Thus, none of the host genotypes are resistant to all of the corresponding pathogen genotypes. Quite frequently the trained observer can distinguish between the four low infection types in Figure 1 if complete dominance is lacking in the pathogen and intermediate infection types occur (307). Preliminary, certainly not conclusive, evidence indicates there may be some differences between some of the high infection types (41). The resistance genes that have been matched by virulence factors in the pathogen may have a residual expression by reducing the pustule size and sporulation compared to the control line.

Recent evidence indicates some specific and nonspecific resistance genes in combinations have an additive (117, 332, 344) or complementary effect (351). Additionally, the host genome seems to affect in some way the expression of this specific interaction (95, 311) or one host gene may interact with other host resistance genes (302).

Nonspecific interactions

Resistances that have sometimes been characterized as adult plant, horizontal, generalized, slow-rusting, partial, minor gene, etc. are placed in this group. Proving these resistances are nonspecific is limited to the pathogen isolates at hand. It is not surprising that additional studies have shown some of these resistances to be race-specific. Nevertheless, it is important to keep searching for resistance that is totally nonspecific or if not universal at least nonspecific in the area used.

The difference in disease severity between similar host genotypes may be due to differences in host growth stage. Susceptibility and resistance are often highly correlated with host growth stage even for race-specific resistances. Plant vigor is also closely correlated with differences in susceptibility/resistance, even among plants of the same cultivar. These relationships are further complicated by daily changes in the environment. A host line may be subjected to a heavy inoculum density with a favorable infection period at a critical growth stage, while another line may not be confronted with similar circumstances when it is at the critical stage a few days earlier or later. Experiments are lacking for controlling inoculum density during cycles of reinfection in the field. Additionally, control over the favorability of infection periods is not only lacking, but description of favorability of an infection period is currently not possible without actually counting the number of resultant uredinia or emptied appressoria.

The pathogens may also vary in aggressiveness. This is particularly true if the pathogen occasionally goes through the sexual reproductive cycle. Continual asexual reproduction tends to favor selection for similar growth rates or aggressiveness. This discourse is not to indicate that nonspecific resistance does not exist. It does suggest that many of the resistances initially reported to be nonspecific are later shown to be specific. It takes only two isolates to show specificity, while all pathogen genotypes must be evaluated to prove nonspecificity. The latter, of course, is an impossibility.

Leaf Rust

Leaf or brown rust of wheat caused by *Puccinia recondita* Rob. ex Desm. f.sp. *tritici* is a major disease of wheat worldwide. The map in Figure 2 shows regions of the world where leaf rust has historically been a major or local problem (as delineated in Table 3). Early historical centers for leaf rust work were at Purdue and Kansas State Universities in cooperation with the U.S. Department of Agriculture and in Winnipeg with Agriculture Canada. Important work has been done in Argentina, Australia, Brazil, Egypt, Germany, Italy, India, Mexico, Portugal, the USSR, and Yugoslavia. In addition to these countries, work is currently underway in China, Iran, Israel, Morocco, The Netherlands, Pakistan, and South Africa.

Epidemiology

P. recondita can survive the same environmental conditions that the wheat leaf survives, provided infection but no sporulation has occurred. The fungus can infect with dew periods of 3 hours or less at temperatures of about 20°C, however, more infections occur with longer dew periods. At cooler temperatures, longer dew periods are required, for example at 10°C a 12-hour dew period is necessary. Few if any infections occur where dew period temperatures are above 32°C (376) or below 2°C.

Most of the severe epidemics occur when uredinia and/or latent infections survive the winter at some threshold level on the wheat crop, or where spring-sown wheat is the recipient of exogenous inoculum at an early date usually before heading. Severe epidemics and losses can occur when the flag leaf is infected before anthesis (67). Occasionally, autumn-sown wheat can be severely infected in the autumn, resulting in reduced root growth, tillering, and winter survival and even plant death before anthesis (166). Often disease development in late autumn and early winter is terminated when the older infected leaves die and a combination of unfavorable moisture and temperature limits disease spread to the newer leaves. A similar phenomenon occurs in the spring when day temperatures are warm enough for plant growth (10°C average daily temperature), rain is absent, and night time conditions either do not favor dew formation or temperatures result in frost. When rain occurs during the day, some infections will occur but often low night temperatures will limit the number of infections during the night. The critical month system of leaf rust disease forecasting (66, 67) is based on determining severities at the end of an unfavorable dew and temperature period and assumes that disease increases will be at a uniform rate after this period.

Leaf rust uredinia developing in the spring from infections occurring in the autumn or winter (endogenous inoculum) are usually low in the canopy with the oldest infections on the lowest leaves. Leaf rust developing from airborne (exogenous) inoculum generally occurs high in the canopy with the upper leaves being infected. Inoculum from a local or endogenous source and that from long-distance transportation or exogenous inoculum can usually be distinguished on this basis. Spread from a single uredinium low in the canopy frequently results in a focus of heavily infected tissue within a radius equal to the height to which it has spread in the canopy. Such foci are generally 1 m in diameter when the disease reaches the flag leaf. Infections high in the canopy usually result in a rapid horizontal spread across the crop (309). The horizontal spread of inoculum often results in heavily infected flag leaves, but little or no rust infection on the lower leaves of the wheat plants.

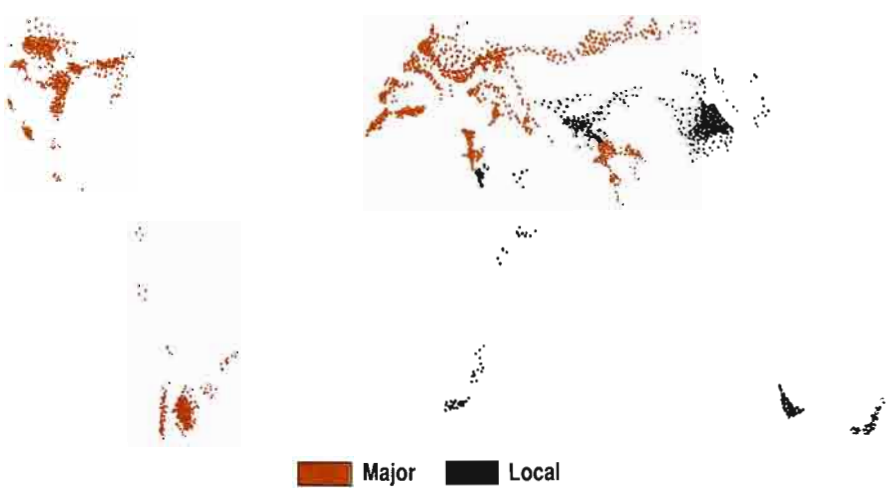

Figure 2. Wheat areas of the world where leaf rust has historically been a major or local problem.

Disease spread can be very rapid under favorable environmental conditions. A single uredinium can produce about 3000 spores per day over a 20-day period (67, 376), following the initial 7- to 10-day latent period. About 33% of the urediniospores that germinate on a susceptible host tissue will result in an infection if there is a favorable infection period. If one assumes no loss in spore numbers during transport to a nearby site and a 10-day period from infection to sporulation, a uredinium could then have 1000 daughter lesions 10 days later, 2000 after 11 days, 1,010,000 after 21 days, and 2,010,000 after 22 days. This explains the explosive nature of the disease when conditions are favorable.

Hosts

P. recondita attacks a wide number of grasses; however, there seems to be a strict specialization of the host range of the various formae speciales. *P. recondita* f.sp. *tritici* is primarily a pathogen of wheat, its immediate ancestors, and the man-made crop triticale. The f.sp. *secalis* on rye does not attack wheat. Recent evidence (152) indicates that populations of leaf rust exist in Europe, Asia, and Africa that are primarily pathogens of durum wheat. They are all distinct from the population that exists worldwide on bread wheat.

Table 4. Alternate hosts reported to have a role in the development of *Puccinia recondita* f.sp. *tritici* on wheat.

Host	Reference
Anchusa italica Retz.	d'Oliveira and Samborski (77)
Clematis mandshurica Rupr.	Azbukina (15)
Isopyrum fumarioides L.	Brizgalova (39, 40)
Thalictrum flavum L.	Jackson and Mains (156)
Thalictrum foetidum L.	Tommasi et al. (381)
Thalictrum japonicum Thunb.	Tommasi et al. (381)
Thalictrum speciosissimum Loefl.	d'Oliveira (76)

Alternate hosts

The fungus produces its sexual gametes (pycniospores and receptive hyphae) on the alternate host. The alternate hosts for *P. recondita* are in the Ranunculaceae and Boraginaceae families. Several species of *Thalictrum*, *Anchusa*, and *Clematis* and *Isopyrum fumarioides* can serve as alternate hosts (Table 4). Most workers assume that *Thalictrum speciosissimum* is the primary alternate host for *P recondita* f.sp. *tritici* in Europe. Alternate hosts probably seldom, if ever, function in North America (327), South America, and Australia. *Clematis* spp. were reported to be infected by *P. recondita* in the Soviet Far East (15) and *Isopyrum fumarioides* was reported as the primary source of inoculum for wheat in Siberia (39, 40). Infection of *T. thunbergii* D.C. was found near wheat fields in Japan, but probably is not the primary source of inoculum for wheat (401). The alternate host is considered important at least for recombining virulence factors in part of the Mediterranean area (9, 77, 364, 381). There is some evidence for specialization on the alternate host on the Iberian Peninsula (402). *Thalictrum*- and *Anchusa*-wheat forms of the rust fungus may have specialized virulence factors relating to the alternate host species involved.

Alternate host studies must involve not only the taxonomy of the pathogen, but also virulence studies under natural conditions. Additionally, epidemiological studies must show a relation between the disease on the alternate and primary hosts.

The alternate host is infected when the teliospores germinate in the presence of free moisture. Basidiospores (1N) are produced that are capable of being carried short distances (a few meters) to infect the alternate host. Approximately 7 to 10 days following infection, pycnia with pycniospores and receptive hyphae

appear. These serve as the gametes and fertilization occurs when the nectar containing the pycniospores is carried to receptive hyphae of the other mating type by insects, splashing rain, or cohesion. The aecial cups appear 7 to 10 days later on the lower leaf surface producing aeciospores that are windborne and cause infection by penetrating the stomata of wheat leaves. The distances travelled by aeciospores appear to be relatively short.

The primary importance of the sexual stage is the recombination of the various virulence and avirulence factors as well as all other genetic characters into new combinations. The alternate hosts may also serve as a source of inoculum for the wheat crop before exogenous urediniospores arrive. The importance of the alternate host in generating changes in the pathogen population for virulence combinations and other factors is unknown. The areas where the alternate host has been reported to be functional seem to have no more virulence combinations nor more severe epidemics than areas without the alternate hosts.

Accessory hosts

P. recondita attacks many species of grasses, but which ones serve as functional hosts in nature for the forma specialis *tritici* is unclear. With artificial inoculation many grasses can be infected, however, this may not occur in the field. Potential hosts for wheat leaf rusts could be wild or weedy species of the genera *Triticum* and *Aegilops* (now classified as *Triticum*) and the related species of *Agropyron* and *Secalis*. *Agropyron repens* L. has been reported as a host for *P. recondita* [*P. persistent* subsp. *persistens* f. *agropyrina* (Eriks.) Urban et Markova], which easily transfers from and to wheat (15). In southern Italy, *Agropyron* sp. is also reported to be infected by a wheat- and *Thalictrum*-infecting rust (62). In North America, *T.* (*Aegilops*) *cylindrica* L. is a host for wheat leaf rust, but the races are different from those that attack the nearby wheat (208). The most common noncrop host for wheat leaf rust is volunteer or self-sown wheat. These plants may be in fallow fields, along the edges of fields and roads, as weeds in a second crop, and as a cover crop under orchards, along irrigation canals, etc. This is the major source of inoculum throughout much of the world where wheat is autumn- or winter-sown.

Primary hosts

The primary host of wheat leaf rust is *Triticum aestivum* L. em. Thell; it has generally been of lesser importance on *T. turgidum* L., except in the Mediterranean and Middle East and Ethiopia and India where durum wheats are more extensively cultivated. It is of minor importance on *T. monococcum* L., *T. dicoccum,* and *T. speltoides* (Tausch) Gren. ex Richter. Wheat leaf rust would appear also to be a major threat to triticale (X *Triticosecale* Wittmack), the crop derived from the man-made cross between wheat and rye (358). In Table 5 named resistance genes are described. The genes for resistance have been obtained primarily from cultivars of *T. aestivum*, but some are from other *Triticum* spp. as well as from *Triticum* (*Aegilops*), *Secalis* (rye), and *Agropyron*. The usefulness or durability of resistance does not seem to be associated with the donor genera or species.

Of the group of race-specific resistances, *Lr19* from *Agropyron elongatum* is still effective worldwide, but it has been used commercially only on a limited area. Unfortunately, this gene is linked to a factor that produces yellow flour color, an undesirable trait in some areas. This problem has now been at least partially solved (183, D.V. McVey, per. comm.). Resistance genes *Lr22a* (adult plant), *25*, *29*, *32*, and *33* are effective, but few cultivars with these resistances have been widely grown. The resistance genes *Lr24* and *Lr9* were used in the United States and virulent isolates of leaf rust appeared after a relatively short period, but yield losses were generally light. Virulence for *Lr24* also occurred in Argentina, Brazil, and South Africa. After the cultivars with *Lr24* were removed from production, the corresponding virulence factors in the leaf rust population quickly decreased. In contrast the virulence genes in the rust population for other resistance genes, such as *Lr3* and *Lr10*, have remained at a high frequency in the rust population even though these genes for resistance are no longer present in the host population. Virulence for *Lr20* may be universal in the North American leaf rust population, although the gene for resistance was never used in the North American Great Plains. Therefore, each resistance gene should be evaluated against the local pathogen population before incorporation into a cultivar. The reasons are unknown why some virulence genes occur in high frequencies and others disappear when no selection pressures are exerted by the host population.

In the case of leaf rust, the best hope for control lies in the use of combinations of genes, irrespective of whether they are major or minor. The Canadian cultivar, Columbus, has *Lr13* and *Lr16*, which has more resistance than just the sum of the effect of the genes independently (332). The combination of the adult plant genes *Lr13* and *Lr34* also has proved very effective (302). *Lr2* alleles when placed in various susceptible background cultivars

Table 5. Named genes for leaf rust resistance, source, genome location, low infection type to an avirulent culture(s), and tester lines.

Lr gene	Genome location	Source	Response to avirulent culture: Seedling[a]	Response to avirulent culture: Adult[b]	Tester	Remarks	References
1	5DL	Malakof	0;	I	RL6003		Ausemus et al. (12)
2a	2DS	Webster	0;,;1	I,MR	RL6016		Dyck and Samborski (94)
2b	2DS	Carina	;1,;1+	R,MR	RL6019		Dyck and Samborski (94)
2c	2DS	Brevit	;1N,23	MR-R	RL6047		Dyck and Samborski (94)
3	6BL	Democrat	;C,2	R,MR	RL6002		Haggag and Dyck (128)
3bg	6BL	Bage	;C,23	MR-MS	RL6042		Haggag and Dyck (128)
3ka	6BL	Klein Aniversario	;C,12C	MR-MS	RL6007		Haggag and Dyck (128)
9	6BL	*Triticum umbellulatum*	0;	I	RL6010		Soliman et al. (360)
10	1AS	Lee	;,2	R-MS	RL6004		Choudhuri (68)
11	2A	Hussar	Y	MR	RL6053	Test at 18°C	Soliman et al. (361)
12	4A	Exchange	-	R	RL6011	Adult plant resistance	Dyck et al. (96)
13	2BS	Frontana	;	R	Manitou	Test at 30°C,	Dyck et al. (96)
14a	7BL	Hope	X	MS	RL6013	Test at 18°C	Dyck and Samborski (93)
14b	7BL	Bowie	X	MS	RL6006		Dyck and Samborski (93)
15	2DS	Kenya 1-12 E-19-J	;C	R	RL6052		Luig and McIntosh (212)
16	2BS	Exchange	;1N	MS-MR	RL6005		Dyck and Samborski (92)
17	2AS	Klein Lucero	;1+,0;	MR-MS	RL6008		Dyck and Samborski (92)
18	5BL	Africa 43	2+2-	MS	RL6009	Test at 18°C	Dyck and Samborski (92)
19	7DL	*Agropyron elongatum*	0	I	RL6040	Linked to *Sr25*	Sharma and Knott (341)
20	7AL	Thew	0;	R	Thew	Linked to *Sr15*	Browder (50)
21	1DL	*T. tauschii*	0;,12-	I	RL6043		Rowland and Kerber (324)
22a	2DS	*T. tauschii*	-	MR	RL6044	Adult plant resistance	Rowland and Kerber (324)
22b	2DS	Thatcher	-	R	Thatcher	Adult plant resistance	Dyck (86)
23	2BS	Gabo	1;,23	MR,MS	RL6012	Test at 25°C	McIntosh and Dyck (237)
24	3DL	*A. elongatum*	0;	R	RL6064	Linked to *Sr24*	Browder (51)
25	4Aß	Rosen rye	;N	I	Transec		Driscoll and Anderson (83)
26	1BL-1RS	Imperial rye	0;,;1	I	RL6078	Linked to *Sr31* & *Yr9*	Singh et al. (348)
27	3BS	Gatcher	X-	MR	Gatcher	Functional only with *Lr31*, linked with *Sr2*	Singh and McIntosh (352)
28	4BL	*T. speltoides*	0;	I	RL6079		McIntosh et al. (246)
29	7DS	*A. elongatum*	;1N	R	RL6080		Sears (337)
30	4BL	Terenzio	;1,23	R	RL6049		Dyck and Kerber (89)
31	4Aß	Gatcher	X-	MR	Gatcher	Functional only with *Lr27*	Singh and McIntosh (352)
32	3D	*T. tauschii*	;1+	MR	RL5497-1		Kerber (171)
33	1BL	PI58458	1+,22+	MR	RL6057		Dyck et al. (91)
34	7D	Terenizo	12C	MR-MS	RL6058	Test at 10°C, linked to *Yr18*	Dyck (87)
35	2B	*T. speltoides*	-	-	RL5711	Adult plant resistance, linked with resistance to stem rust	Unpublished
36	6BS	*T. speltoides*	-	-	E84018		Unpublished
37	2AS	*T. ventricosa*	-	-	RL6081	Linked to *Sr38* and *Yr17*, test at 18°C	Unpublished
38	2AL	*A. intermedium*	-	-	-		Unpublished
39	2DS	*T. tauschii*	-	-	KS86NGRC02		Unpublished
40	1D	*T. tauschii*	-	-	KS89WGRC07		Unpublished
41	1D	*T. tauschii*	-	-	KS90WGRC10		Unpublished
T3		Terenizo	-	S-MS	TcLrT3		Unpublished
Exch		Exchange		?	RL6014		Unpublished
B		Brevit	2,;	?	RL6051		Unpublished

[a] Tests unless otherwise indicated were at 20°C and 10,000 lux of light in the greenhouse.
[b] I = immune, R = resistant, MR= moderately resistant, MS = moderately susceptible, S = susceptible.

Table 6. Wheat cultivars that remained leaf rust resistant for a number of years.

Name	Growth habit	Source	Released	Probable source of resistance	*Lr* gene(s)
Americano 44d	spring	Uruguay	1918	Land variety	?
Atlas 66	winter	USA	1948	Frondosa	13,+
Chris	spring	USA	1965	Frontana	13,34,+
Centenario	spring	Uruguay	1933	Americano 44d	1,+
Ciano F67	spring	CIMMYT	1967	Chris	13,+
Era	spring	USA	1970	Frontana	10,13,34,+
Frondosa	spring	Brazil	1934	Alfredo Chaves (land variety)	13,+
Frontana	spring	Brazil	1943	Frondosa	13,34,T3,+
Fronteira	spring	Brazil	1934	Alfredo Chaves (land variety)	13,+
Gage	winter	USA	1963	?	3,+
Klein Aniversario	spring	Argentina	1945	Americano 44d	13,3ka,+
Klein Cometa	spring	Argentina	1942	Americano 44d	13,+
Klein Lucero	spring	Argentina	1950	Americano 44d	17,+
Klein Progreso	spring	Argentina	1937	Americano 44d	13,+
Klein Rendidor	spring	Argentina	1954	Americano 44d	13,+
Klein Titan	spring	Argentina	1925	Americano 44d	13,3ka,+
Klein Vencedor	spring	Argentina	1925	Americano 44d	13,+
La Prevision 3	spring	Argentina	1935	Americano 44d	13,34,+
La Prevision 25	spring	Argentina	1937	Americano 44d	13,34,+
La Prevision 32	spring	Argentina	1935	Americano 44d	13,34,+
Minter	winter	USA	1949	?	
Pavon F76	spring	CIMMYT	1976	Ciano F67'S'	1,10,13,+
Redcoat	winter	USA	1960	Surpreza	13,+
Sinvalocho MA	spring	Argentina	1936	Americano 44d	13,+
Sturdy	winter	USA	1960	Sinvalocho MA	12,34
Surpreza	spring	Brazil	1934	Alfredo Chaves (land variety)	13,+

Table 7. Cultivars susceptible to wheat leaf rust and some of their important characteristics.

Cultivar	Wheat type	Growth habit	Day length requirement	*Lr* gene(s)[a]	*Sr* gene(s)[b]	*Yr* gene(s)[c]
Agra Local	bread	spring	short	?		
Baart	bread	spring	short	10	LC	
Berkmen	durum	spring	long			
Cheyenne	bread	winter	long	?	5	
Fertas	bread	spring	short	?	none	none
Glossy Hugenot	durum	spring	short	?		
Lebanon	bread	spring	short	?		
Line E	bread	spring	short	?	none	none
Little Club	club	spring	long	?	LC	none
Local Red	durum	spring	short	?		
Morocco	bread	spring	short	?	none	none
Monon	bread	winter	long	?		
Pima 1	bread	indeterminant	short	?		
Thatcher	bread	spring	long	22b,+	5,9g, 12,16,+	7
Tachung 32	bread	spring	?	?	?	none
Triumph 64	bread	winter	long	?	TMP	unknown

[a,b,c] See Tables 5,10, and 14, respectively.

resulted in different levels of resistance (94), indicating some interaction of resistance gene and background genotype.

Table 6 lists a number of cultivars that have shown a long period of usefulness in areas where leaf rust is important. Nearly all these cultivars have a combination of genes for leaf rust resistance. It is likely that many more such cultivars exist. However, resistance in some of these cultivars may fail if used outside of the area in which they were tested.

In most studies of resistance and epidemiology, susceptible hosts or checks are required. A number of such cultivars have been identified and a few have been cultivated at least regionally. Table 7 provides characteristics of the selected susceptible cultivars.

Many cultivars have been reported to have nonspecific resistance to wheat leaf rust (Table 8). The extent and method of evaluation vary greatly between cultivars, thus a reference is provided for each cultivar. Some of these cultivars have race-specific genes for resistance as well. Recognition of nonspecific resistance in the field often involves a comparison between the test line and a standard or check. A line may be slow-rusting with a long latent period when compared to one check or environment, but not in another (397).

Pathogen

De Candolle (79) separated wheat leaf rust from other rusts of wheat and called it *Uredo rubigo-vera in* 1815. Eriksson and Henning (98) described the causal organisms of both wheat and rye leaf rust as *P. dispersa*. Eriksson (97) separated the

wheat and rye leaf rust fungi and the causal organism of wheat leaf rust became *P. triticina*, a name still used in parts of eastern Europe. Mains (221) placed the causal organism of wheat leaf rust in *P. rubigo-vera* and established a complex group of 52 formae speciales for the fungi causing leaf rust. In 1984, Savile (334) stated that *P. triticina* should be the binomial for wheat rust and *P. recondita* for rye leaf rust. The current binomial used by most pathologists is *P. recondita* recommended by Cummins and Caldwell (74) and *P. recondita* f.sp. *tritici* is used by most if not all leaf rust workers (330). Virulence and disease development indicate that a taxonomic study of this complex species should be undertaken.

Life cycle

Figure 3 shows the life cycle for *P. recondita* f.sp. *tritici* and the disease cycle for wheat leaf rust. The time for each event and frequency of some events (sexual cycle, wheat cropping season, and "green bridge") may vary among areas and regions of the world.

The alternate host currently provides little direct inoculum to wheat (see section on alternate hosts), but may be a mechanism for genetic exchanges between races and perhaps

Table 8. Cultivars given in the literature as having nonspecific resistance to wheat leaf rust, named resistance genes as known, type of nonspecific resistance, and source of information.

Cultivar	*Lr* gene(s)	Type of nonspecific resistance	Reference	Remarks
Akabozu		latent period	Broers and Jacobs (42)	2 genes
BH 1146	13, 34	latent period	Broers and Jacobs (42)	2-3 genes
Ble Tendre			Caldwell et al. (60)	
Borah		latent period, uredinia size	Bjarko and Line (32)	
Bulgaria 88	11		Caldwell et al. (60)	
Choti Lerma	13, 34		Singh and Satyavir (354)	
Dual			Caldwell et al. (60)	
Fairfield			Shaner and Finney (340)	
Gros Bleu			Miller and Line (253)	
INIA 66	13,17		Caldwell et al. (60)	
Kalyansona		uredinia number	Kapoor and Joshi (169)	
Kharchia		uredinia number	Kapoor and Joshi (169)	
La Porte			Caldwell et al. (60)	
Lee	10		Wilcoxson (397)	
Lerma 50			Caldwell et al. (60)	
Lerma 52			Caldwell et al. (60)	
Lerma Rojo 64A	14a,17, 34		Singh and Satyavir (354)	
Menkemen			Caldwell et al. (60)	
Mentana	3bg?		Caldwell et al. (60)	
Milyang 8-6			Lee and Shaner (193)	2 recessive genes
Purkof			Caldwell et al. (60)	
Sonalika	13	latent period, uredinia size, and number	Singh and Satyavir (354)	
Suwon 85		latent period, uredinia size	Kuhn et al. (189)	
Thatcher	22b,+		Gavinlertvatana and Wilcoxson (115)	
Vigo			Shaner and Finney (340)	
Wampum			Bjarko and Line (32)	recessive gene
Westphal 12A		latent period	Broers and Jacobs (42)	3 genes

populations. The pathogen survives the period between wheat crops in many areas on a green bridge of volunteer (self-sown) wheat (see section on epidemiology). Inoculum in the form of urediniospores can be blown by winds from one region to another. This is the case in North America where leaf rust is introduced annually in the northern spring-sown wheat area from urediniospores produced on the southern autumn-sown wheat area, where it is warmer and the wheat is earlier maturing.

Urediniospores initiate germination 30 minutes after contact with free water at temperatures of from 15 to 25°C. The germ tube grows along the leaf surface until it reaches a stoma; an appressorium is then formed, followed immediately by the development of a penetration peg and a substomatal vesicle from which primary hyphae develop. A haustorial mother cell develops against the mesophyll cell and direct penetration occurs. The haustorium is formed inside the living host cell in a compatible host-pathogen interaction. Secondary hyphae develop resulting in additional haustorial mother cells and haustoria. In an incompatible host-pathogen response, haustoria fail to develop or develop at a slower rate. When the host cell dies, the fungus haustorium dies. Depending upon when or how many cells are involved, the host-pathogen interaction will result in a visible resistance response (316, 317).

Spore germination to sporulation can occur within a 7- to 10-day period at optimum and constant temperatures. At low temperatures (10-15°C) or diurnal fluctuations, longer periods are necessary. The fungus may survive as insipid mycelia for a month or more when temperatures are near or below freezing. Maximum sporulation is reached about 4 days following initial sporulation (at about 20°C). Although the number can vary greatly, about 3000 spores are produced per uredinium per day. This level of production may continue for 3 or more weeks if the wheat leaf remains alive that long (67, 376).

The teliospores are formed under the epidermis with unfavorable conditions or senescence and remain with the leaves. Leaf tissues can be dispersed or moved by wind, animals, or man considerable distances. Basidiospores are formed and released under humid conditions, which limit their spread. Basidiospores are also hyaline and sensitive to light, further limiting travel to probably tens of meters. Aeciospores are more like urediniospores in their ability to be transported by wind currents, but long distance transport has not been noted for some reason.

Vickie Brewster

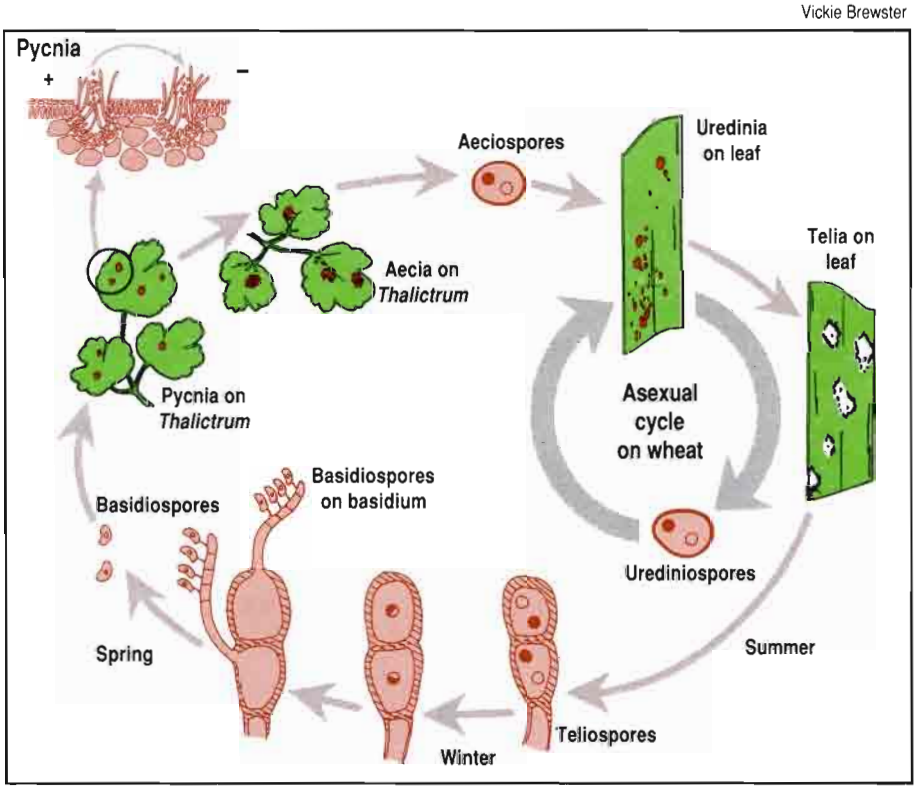

Figure 3. Life and disease cycles for *Puccinia recondita* f.sp. *tritici* (wheat leaf rust).

Virulence

Virulence is the ability of a pathogen to overcome a specific gene for resistance. On a worldwide basis, virulence probably exists for all numbered *Lr* genes except *Lr19*. Virulence has been reported for *Lr19*, but confirmation has not been done and isolates are unavailable. Virulence for *Lr9* and *Lr24* is absent in many parts of the world and no isolate has been reported to be virulent when the two are combined. Because virulence exists for most of the resistance genes singly and on various combinations of two or more genes, it is essential to know what combination of virulence exists in the pathogen population before spending time in combining resistances in a host cultivar. This requires a systematic pathogen survey from which samples are obtained from different

cultivars and different geographical and ecological areas throughout the season. In most areas the rust (thus virulences) can survive the entire year in the asexual cycle.

Laboratories conduct virulence surveys in different manners. Few comparisons have been made between the virulences on the various continents (36, 151). Table 9 lists the latest race surveys published for each country or region. Some laboratories may no longer publish survey results in international journals and others may have discontinued surveys altogether. Because of the usefulness of the virulence frequency and virulence combination data, both in breeding resistant wheats and in epidemiology studies on an international basis, it is essential that such data be made available wherever possible. The Proceedings of the European and Mediterranean Cereal Rusts Conferences and the Cereal Rusts Bulletin have been the vehicles for interchange throughout much of the world.

Table 9. Recent virulence surveys of *Puccinia recondita* f.sp. *tritici* on regional or national basis published in international journals.

Country	Period	Reference
Afghanistan	1963-64	Hassan (136)
Argentina	1956	Cenoz and Vallega (64)
Australia		Annual reports, Plant Breed. Inst.
Brazil	1987-88	Barcellos (22)
Bulgaria	1983-87	Donchev (82)
Canada	1985	Samborski (331)
Canada	1988	Kolmer (188)
Chile	1940	Vallega (386)
China (PRC)	1986	Hu and Roelfs (151)
Czechoslovakia	1981-83	Bartos et al. (24)
Denmark	1960-63	Hermansen (147)
Egypt	1971	Abdel-Hak and Kamel (1)
Ethiopia	1979-81	Dmitriyev (80)
Hungary	1969-72	Bocsa (33)
India	1982-83	Nagarajan et al. (262)
Iran	1968-72	Bamdadian (20)
Iraq	1967-68	Natour et al. (264)
Italy	1982-83	Casulli and Sinigalco (63)
Kenya	1968-69	Harder (131)
Mexico	1988-89	Singh (350)
Nepal	1982-83	Nagarajan et al. (262)
Pakistan	1983	Rizvi et al. (292)
Peru	1956	Postigo et al. (281)
Poland	1975-76	Rysz (325)
Portugal	1972-81	Freitas (108)
Romania	1968-70	Negulescu and Ionescu-Cojocaru (267)
South Africa	1983-85	Pretorius et al. (282)
Spain	1972-75	Salazar et al. (329)
Sweden	1960-63	Hermansen (147)
USA	1987	Long et al. (209)
USSR	1982-83	Bazhenova (27)
Yugoslavia	1963-67	Boskovic (35)
Yugoslavia	1982-83	Pavlova et al. (276)

Aggressiveness

Not all isolates have the same ability to cause epidemics even when they possess all the necessary genes for virulence. This difference in aggressiveness may relate to unmeasured differences in spore production, environmental fitness, survivability or infectibility of spores, and latent or sporulating periods. With the cereal rusts, it is difficult to determine to what extent the difference in latent period is due to pathogen aggressiveness, environmental conditions their interactions, and to what extent it is due to nonspecific resistance.

Stem Rust

Stem or black rust of wheat is caused by *Puccinia graminis* Pers. f.sp. *tritici*. The map in Figure 4 shows that, at one time, it was a feared disease in most wheat regions of the world. In part, this was due to its seriousness globally and the amount of published information from Europe, North America, and Australia. The fear of stem rust was understandable because an apparently healthy crop 3 weeks before harvest could be reduced to a black tangle of broken stems and shriveled grain by harvest. In Europe and North America, the removal of the alternate host reduced the number of combinations of virulence and the amount of locally produced inoculum (aeciospores). In addition, in some areas early maturing cultivars were introduced to permit a second crop or to avoid flowering and grain filling during hot weather. Early maturing cultivars escape much of the damage caused

by stem rust by avoiding the growth period of the fungus. The widespread use of resistant cultivars worldwide has reduced the disease as a significant factor in production (Table 3). Although changes in pathogen virulence have rendered some resistances ineffective, resistant cultivars have generally been developed ahead of the pathogen. The spectacular epidemics that developed on Eureka (*Sr6* in Australia) in the 1940s and on Lee (*Sr9g*, *Sr11*, *Sr16*), Langdon (*Sr9e*, +), and Yuma (*Sr9e*, +) in the United States in the mid-1950s really have been the exceptions in the past. The experience in other parts of the world have been similar (211, 301, 326). Today, stem rust is largely under control worldwide (Table 3).

Epidemiology

The epidemiology of *P. graminis* is similar to *P. recondita*. The minimum, optimum, and maximum temperatures for spore germination are 2, 15-24, and 30°C (150); for sporulation, 5, 30, and 40°C—about 5.5°C higher in each category than for *P. recondita*. Stem rust is more important late in the growing period, on late-sown and maturing wheat cultivars, and at lower altitudes. Spring-sown wheat is particularly vulnerable in the higher latitudes if sources of inoculum are located down wind. Large areas of autumn-sown wheat occur in the southern North American Great Plains, providing inoculum for the northern spring-sown wheat crop. In warm humid climates, stem rust can be especially severe due to the long period of favorable conditions for disease development when a local inoculum source is available. Under such conditions, some of the specific resistances (*Sr6*, *Sr10*, *Sr17*, etc.) are ineffective due to temperature and some of the nonspecific resistances (e.g., Thatcher) are inadequate due to inoculum densities.

Stem rust differs from leaf rust in requiring a longer dew period (6 to 8 hours are necessary). In addition many penetration pegs fail to develop from the appressorium unless stimulated by at least 10,000 lux of light for a 3-hour period while the plant slowly dries after the dew period. Maximum infection is obtained with 8 to 12 hours of dew at 18°C followed by 10,000+ lux of light while the dew slowly dries and the temperature rises to 30°C (318). Light is seldom limiting in the field as dews often occur in the morning. However, little infection results when evening dews and/or rains are followed by winds causing a dry-off prior to sunrise. In the greenhouse, reduced light is often the reason for poor infection rates. The effect of light probably is an effect on the plant rather than the fungus system as urediniospores injected inside the leaf whorl result in successful fungal penetrations without light striking the fungus. Stem rust uredinia occur on both leaf and stem surfaces as well as on the leaf sheaths, spikes, glumes, awns, and even grains.

A stem rust pustule can produce 10,000 urediniospores per day (170, 255). This is more than leaf rust, but the infectibility is lower with only about one germling in 10 resulting in a successful infection. Stem rust uredinia, being mostly on stem and leaf sheath tissues, often survive longer than those of leaf rust, which are confined more often to the leaf blades. The rate of disease increase for the two diseases is very similar.

Stem rust urediniospores are rather resistant to atmospheric conditions if their moisture content is moderate (20-30%). Long distance transport occurs annually (800 km) across the North American Great Plains (299), nearly annually (2000 km) from Australia to New Zealand (211), and at least three times in the past 75 years (8000 km) from East Africa to Australia (392).

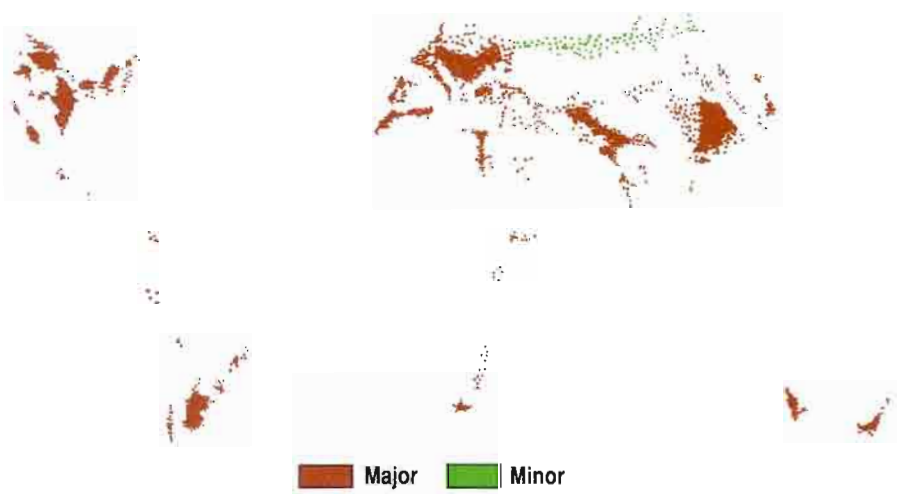

Figure 4. Wheat areas of the world where stem rust was historically considered a problem and would probably be a major disease if resistant cultivars were not grown.

Notes:

Aeciospores can also be a source of inoculum of wheat stem rust. Historically, this was important in North America and northern and eastern Europe. This source of inoculum has generally been eliminated or greatly reduced by removal of the common or European barberry (*Berberis vulgaris*) from the proximity of wheat fields. Aeciospores infect wheat similarly to urediniospores.

Hosts

Wheat, barley, triticale, and a few related species are the primary hosts for *P. graminis* f.sp. *tritici*. However, the closely related pathogen, *P. graminis* f.sp. *secalis*, is virulent on most barleys and some wheats (e.g., Line E). *P. graminis* f.sp. *secalis* can attack *Sr6* and *Sr11* in a Line E host background (211). The primary alternate host in nature has been *Berberis vulgaris* L., a species native to Europe, although other species have been susceptible in greenhouse tests. The alternate hosts are usually susceptible to all or none of the formae speciales of *P. graminis*.

Alternate hosts

The main alternate host for *P. graminis* is *Berberis vulgaris*, which was spread by man across the northern latitudes of the Northern Hemisphere. Because of its upright, bushy growth with many sharp thorns, it made an excellent hedge along field borders. The wood was useful for making tool handles, the bark provided a dye, and the fruit was used for making jams. Settlers coming to North America from Europe brought the barberry with them. The barberry spread westward with man and became established as a naturalized plant from Pennsylvania through the eastern Dakotas and southward into northeastern Kansas. Many species of *Berberis*, *Mahonia*, and *Mahoberberis* are susceptible to *P. graminis* (298). The Canadian or Allegheny barberry, *B. canadensis*, should be added to this list.

The alternate host was a major source of new combinations of genes for virulence and aggressiveness in the pathogen (126). The amount of variation in the pathogen made breeding for resistance difficult, if not impossible. Of the virulence combinations present one year, many would not reoccur the following year, but many new ones would appear (296). The barberry was the source of inoculum (aeciospores) early in the season. Generally, infected bushes were close to cereal fields of the previous season, so inoculum traveled short distances, without the loss in numbers and viability associated with long distance transport. A single large barberry bush can produce about 64 x 10^9 aeciospores in a few weeks (365). This is the equivalent of the daily output of 20 million uredinia, in an area of 400 m^2.

Barberry was a major source of stem rust inoculum in Denmark (148) and North America (296). The success of reducing stem rust epidemics in northern Europe and North America following removal of barberry near wheat fields has probably led to an over emphasis of the role of this alternate host in generating annual epidemics elsewhere.

Resistance to *P. graminis* in *Berberis* is reported to be due to the inability of the pathogen to directly penetrate the tough cuticle (250). *B. vulgaris* becomes resistant to infection about 14 days after the leaves unfold. However, infections occur on the berries, thorns, and stems, which suggests the toughening of the cuticle may not be as important as originally thought. In recent testing of alternate host cultivars, a hypersensitive response has been observed particularly with *Mahonia* spp.

Accessory hosts

It is necessary to separate the accessory hosts for *P. graminis* f.sp. *tritici* from those of the other formae speciales, especially those of *P. graminis* f.sp. *secalis*. Additionally, many other grasses can be infected as seedlings in the greenhouse or as adult plants when spores are directly injected into the leaf whorl, but they are rust free under field conditions.

Barley, triticale, and an occasional rye plant are infected by wheat stem rust. Wild *Hordeum* spp., such as *H. jubatum* L. and rarely *H. pusillum* Nutt., and *Triticum* (*Aegilops*) *cylindrica* Host. are sometimes infected in the United States (301); however, it is thought that the inoculum generally comes from wheat to these grasses rather than vice versa in North America.

In the center of origin for both the primary host and the pathogen, the accessory host may play a more important role. Basile (26), Arthaud et al. (10), Gerechter-Amitai and Wahl (116), and Skorda (356) described accessory hosts in Italy, Morocco, Israel, and Greece, respectively.

Primary hosts

Triticum aestivum L. and *T. turgidum* L. as cultivated wheats are the primary hosts of wheat stem rust. Within these species, and in the closely related grasses, there exists a wide range of specific and nonspecific resistances. The resistance is expressed as 1) a reduction in number of lesions, 2) a reduction in the size of the sporulating area, 3) an increase in the length of the latent period, and 4) a reduction in the length of the sporulating period. Some of the resistances function throughout the life span of the host, others only at certain growth stages and in certain tissues, and still others only under certain environmental conditions.

The adult plant resistance *Sr2* derived from Hope results in an absence of uredinia pustules in the internode tissues (133, 377). This has been probably the most commonly used *Sr* resistance gene worldwide since the 1940s. The *Sr13* gene provides a reduction in pustule size, but appears not to affect the number of uredinia. It is more effective at high temperatures (25°C) and in tetraploid wheats. This gene is probably present in most North American durum wheats. *Sr22* provides a high to moderate level of resistance, but has not been used extensively in commercial cultivars. In the USA, Argentina, Brazil, and South Africa, *Sr24* is present in some cultivars and results in moderate size uredinia. *Sr25* is an effective seedling resistance gene but is often moderately susceptible in the adult plant stage. *Sr26*, present in the Australian cultivars Eagle, Kite, Jabiru, Avocet, Harrier, and Hybrid Titan, has been used extensively in Australia for more than a decade (211). *Sr27* is a highly effective gene from rye and probably is in some commercial rye cultivars and triticales (215). *Sr29*, resulting in small to moderately large uredinia, provides inadequate resistance in nurseries, but may be adequate in large fields.

The 1B/1R wheat-rye translocation is associated with *Sr31*, *Lr26*, and *Yr9*. It provides a highly to moderately effective resistance to stem rust worldwide. Currently, it is common in many high yielding wheats including Aurora, Kavkaz, Burgus II, Lovrin 10, Riebesel, Siouxland, Alondra, Weique, Salzmuendu Bartweizen, Nautica, Clement, Pak 81, Faisalabad 85, and the Veery and Bobwhite crosses from CIMMYT. *Sr32* and *Sr33* provide a high level of resistance, but they have not been used commercially. *Sr37* provides a high level of resistance, but it is difficult to maintain in a homozygous resistant line and has not been used commercially. *SrGt* provides a moderate level of resistance in Gamut and *SrWld-1* provides a moderate level of resistance in Waldron, Ellar, Olaf, and probably ND 81. This resistance along with *SrGt* may be overcome by high inoculum densities.

Table 10 lists named resistance genes. The low infection types are generally those at 18°C and 10,000+ lux on seedlings when North American and other selected isolates are used (298, 311).

Much has been written about nonspecific resistance to wheat stem rust; however, there are few critical studies. Rowell and McVey (320) evaluated the receptivity of a series of winter wheats of diverse origin, using cultivars that produce a susceptible infection type to the isolate used. There was a wide range of differences in rust severity when the cultivars were uniformly inoculated on three consecutive nights and then observed for the severity of disease 14 days later. The inheritance of this type of resistance and its effect at other growth stages and environments are unknown.

Several examples of nonspecific resistance have been found to be associated with or in part due to the specific resistance in the cultivar. The nonspecific resistance of the Hope cultivars seems to be due mostly to the effect of *Sr2* (133, 377). The slow-rusting of some cultivars derived from *T. timopheevii* was shown by Rowell (316, 317) to be due to the specific gene *Sr36* (*SrTt-1*).

Table 10. Named genes for stem rust resistance, source, genome location, low infection type to an avirulent culture(s), and tester lines.

Sr gene	Genome location	Source	Response to avirulent culture: Seedling[a]	Response to avirulent culture: Adult[b]	Tester	Remarks	Reference
1						See *Sr9d*	
2	3BS	Yaroslav emmer	-	S	CS (Hope 3B)	Few uredinia, adult plant resistance	Knott (181)
5	6DS	Reliance	0;	I	ISr5-Ra	Test at 18°C	Sears et al. (338)
6	2DS	Red Egyptian	0;,X	R	ISr6-Ra		Knott and Anderson (185)
7a	4BL	Kenya 117A	2C	MR	Line G sel		Knott and Anderson (185)
7b	4BL	Marquis	2+-	MS	ISr7b-Ra		Loegering and Sears (203)
8a	6AS	Red Egyptian	2+-	MS	ISr8-Ra		Knott and Anderson (185)
8b	6AS	Barleta Benvenuto	X	MR	Barleta Benvenuto		Singh and McIntosh (353)
9a	2BL	Red Egyptian	2-,2+3	MR,MS	ISr9a-Ra		Knott (180)
9b	2BL	Kenya 117A	2,23	MR	W2691Sr9b		Green et al. (123)
9d	2BL	Hope	;2-	MR	ISr9d-Ra		Loegering and Sears (204)
9e	2BL	Vernstein	;;;1+	R	Vernstein		McIntosh and Luig (242)
9f	2BL	Chinese Spring	2	?	Chinese Spring		Loegering (200)
9g	2BL	Lee	2-	MR	CnSSr9g	Linked to *Yr7*	McIntosh et al. (244)
10		Egypt NA95	X-N	MR	W2691Sr10		Knott and Anderson (185)
11	6BL	Lee	;2	R-MR	ISr11-Ra		Green et al. (123)
12	3BS	Thatcher	;1+,X	I-R	BtSr12Tc	Test at 18°C	Sheen and Snyder (346)
13	6AL	Khapstein	2-2	MR-MS	W2691Sr13	Test at 25°C	Knott (179)
14	1BL	Khapstein	1+3-CN	MS	Line A sel		Knott (179)
15	7AL	Norka	X-CN	MS-S	W2691Sr15	Test at 18°C	Watson and Luig (394)
16	2BL	Thatcher	2	MS	ISr16-Ra		Loegering and Sears (203)
17	7BL	Renown	;1-N	R	CS (Hope7B)	Test at 18°C	McIntosh et al. (243)
18	1D	Marquis	;	I	LCSr18Mq		Baker et al. (19)
19	2BS	Marquis	1	R	LCSr19Mq		Anderson et al. (7)
20	2BL	Marquis	2	MS	LC		Anderson et al. (7)
21	2AL	*Tricticum monococcum*	0;	R	Einkorn		The (379)
22	7AL	*T. monococcum*	22-	MR	SwSr22T.B.		The (379)
23	2BS	Exchange	23C	MS	Exchange	Linked to *Lr16*	McIntosh and Luig (241)
24	3DL	*Agropyron elongatum*	2+-	MR-MS	BtSr24Ag	Linked to *Lr24*	McIntosh et al. (238)
25	7DL	*A. elongatum*	2	MS-S	LCSr25Ars	Linked to *Lr19*	McIntosh et al. (238)
26	6AL	*A. elongatum*	;2-	MR	Eagle		Knott (178)
27	3A	Imperial rye	0;	I	W2691Sr27		Acosta (3)
28	2BL	Kota	0;	I	W2691Sr28Kt		McIntosh (233)
29	6DL	Etiole de Choisy	2-	MS	PusaSr29Edch		Dyck and Kerber (88)
30	5DL	Webster	2	MS	BtSr30Wst		Knott and McIntosh (187)
31	1BL-1RS	Petkus rye	2-	R	LineESr31Kvz	Linked to *Lr26* and *Yr9*	Singh et al. (348)
32	2A,2B	*T. speltoides*	2-	MR	ER 5155		McIntosh (235)
33	1DL	*T. tauschii*	2-	MR	TetraCanthatch/ *T. tauschii*		Kerber and Dyck (172)
34	2A,2B	*T. comosa*	23CN	MR	Compair	Linked to *Yr8*	McIntosh et al. (246)
35	3AL	*T. monococcum*	0;	I	Mq(2)5xG2919		McIntosh et al. (239)
36	2BS	*T. timopheevi*	0;,X-	I,TrS	W2691SrTt-1		McIntosh and Gyarfas (240)
37	4AL	*T. timopheevi*	0;	I	W2691SrTt-2	Off-type plants	McIntosh and Gyarfas (240)
38	2AS	*T. ventricosa*	-	-	VPM1	Linked to *Lr37* and *Yr17*	Unpublished
39	2B	*T. speltoides*	2-	-	RL5711		Unpublished
40	2BS	*T. araraticum*	-	-	RL6087		Unpublished
Tt-3		*T. timopheevi*	1+C	I-R	Fed*2/SrTt-3		Unpublished
Tmp	4B	Triumph 64	2-	MS	Triumph 64		Unpublished
LC		Little Club	;1+	-	Little Club		Unpublished
McN		McNair 701	;2-	-	McNair 701		Unpublished
Gt		Gamut	2+	MS	BtSrGtGt		Unpublished
dp-2		Golden Ball	2	MR	Media Ap9d		Unpublished
X		Marquis	23	MS	PdSrXMq		Unpublished
Kt'2'	2BL	Kota	2	MS	Line AE sel		Unpublished
Wld-1		Waldron	2	R-MS	BtSrWldWld		Unpublished
U	2D	Red Egyptian	X-CN	-	CnSSrURE		Unpublished
H		H-44	23C	MS	H44 deriv.		Unpublished
Pl		Peliss	;1	-	Peliss		Unpublished
Pt		Petterson ML68-14	2-	-	Petterson ML68-14		Unpublished
M		Maruccos 623	X	-	Maruccos 623		Unpublished
Agi		*A. intermedium*	;2	R	*A. intermedium* deriv.		Unpublished
;		Frn//Ky58/Nth	;2	R,MS	8N122		Unpublished
Wst-2		Webster	2	MR	LCSrWst2Wst		Unpublished

[a] Low infection types at 18°C, may vary at other temperatures (213).
[b] I = immune, R = resistant, MR = moderately resistant, MS = moderately susceptible, S = susceptible, TrS = trace susceptible.

Thatcher (*Sr5*, *9g*, *12*, *16*), derived from the cross Marquis/Imuillo durum//Marquis/Kanred, also has been thought to have some form of slow-rusting resistance. In a series of Baart/Thatcher lines evaluated under a severe and moderate epidemic, differences in disease severity were independent of *Sr9g* and *Sr16*, but were associated with lines having *Sr5* and an undescribed gene (265). Brennan (38) assigned the resistance of Thatcher to two recessive genes.

Using the F_5 derivatives from the crosses of Lee, Idaed 59, Kenya 58, Marquis, and Thatcher with the susceptible Baart and Prelude, resistance measured as area under the disease progress curve (AUDPC) was hypothesized as the result of interaction between 6 to 14 genes that were different from the specific genes known in these cultivars (359). Further genetic work combining these genes independently of the specific genes has not been done. The morphological resistance reported by Hart (134) has been re-evaluated (273) and it is now thought that the resistance may have been due to *Sr30* (187).

Much of the resistance thought to be race-nonspecific is combined in cultivars with specific resistance that is sensitive to inoculum density (265, 312) and effective at only certain growth stages and temperatures. Certain *Sr* genes, e.g., *Sr2*, *Sr6* (72), and *Sr36* (316), are often associated with slow-rusting to stem rust. Table 11 lists cultivars mentioned in the literature as having nonspecific resistance.

Various susceptible hosts have been used worldwide for wheat stem rust. Historically, Little Club has been widely used, but it has a gene for resistance (*SrLC*) to isolates isolated from barberry and perhaps even from wheat (369). Another disadvantage of Little Club is its high susceptibility to leaf rust and powdery mildew. At the USDA Cereal Rust Laboratory, the winter wheat cultivar McNair 701 has been used as a susceptible seedling host due to its leaf rust (*Lr9*) and powdery mildew resistance. It is not satisfactory in most tests with adult plants because it has a vernalization requirement. It also has specific resistance (*SrMcN*) to a few isolates obtained from barberry. Baart (*SrLC*), which is tall, weak-strawed, and late in maturity in many areas, has also been used. CIMMYT has used the cultivar Morocco due to its short straw and good agronomic type; however, it often succumbs to other diseases in the field before stem rust appears.

The principal susceptible cultivars used in the early genetic studies were Chinese Spring, Marquis, and a durum wheat—Maruccos 623 (PI 192334). Unfortunately, Marquis has specific genes (*Sr7b*, *18*, *19*, *20*, *X*) for resistance to many isolates and Chinese Spring has *Sr9f*. PI 192334 has a gene for resistance to some of the North American stem rust population. Chinese Spring (*Sr9f*) is an excellent parent in genetic crosses, but it fails to produce seed in the field north of 45°N latitude, probably due to its photoperiod requirement.

The Australian program at the Plant Breeding Institute (PBI) developed the susceptible line W3498 from the cross Little Club//Gabo*3/Charter. This line has no known race-specific resistance to *P. graminis* f.sp. *tritici* and is susceptible to many isolates of *P. graminis* f.sp. *secalis* worldwide. Its disadvantages are susceptibility to leaf rust and powdery mildew and poor agronomic type. Another line specifically developed as a stem rust susceptible host is Purdue 5481-C, which has good leaf rust resistance (at least in North America), but it is tall and has *Sr7b*

Table 11. Cultivars given in the literature as having nonspecific resistance to wheat stem rust, their specific resistance as known, type of nonspecific resistance, and source of information.

Cultivar	*Sr* gene(s)	Type of nonspecific resistance	References
Agatha	5,9g,12,16,25	low receptivity	Martin et al. (229)
Era	5,6,8a,9a,10, 11,12,16,17,+		Martínez-Gonzalez et al. (230)
Exchange	23,McN	slow-rusting	Southern (363)
FKN[a]	5,6,7a,8a,9b,;	slow-rusting	Ayers et al. (13)
Hope	2,7b,9d,17		McIntosh (234)
Idaed 59	36	low receptivity	Rowell (316)
Kenya 58	6,7a	slow-rusting	Skovmand, et al. (359)
Kalyansona	?	low receptivity	Kapoor and Joshi (169)
Lee	9g,11,16	slow-rusting	Skovmand, et al. (359)
Redman	2,7b,9d,17	slow-rusting	Southern (363)
Sentry	+		Mont (255)
Sonalika	2,+	latent period, low receptivity	Kapoor and Joshi (169)
Thatcher[b]	5,9g,12,16,+	slow-rusting	Brennan (38)
Webster	30,Wst-2	morphological	Hart (134)

[a] Frontana//Kenya 58/Newthatch.
[b] Two recessive genes.

and *10*—both effective against many North American stem rust races. Elsewhere, its height would be its main disadvantage. Table 12 lists the major susceptible cultivars and their important characteristics.

Pathogen

Fontana (106) made the first known detailed study, including precise drawings, of *P. graminis* in 1767. Persoon named the fungus on barberry *Aecidium berberidis* in 1791 and the form on wheat *Puccinia graminis* in 1794. DeBary (78) showed that the two fungi were different stages of a single species. Craigie (73) made the first controlled crosses between strains of *P. graminis*.

Life cycle

In most areas of the world, the life cycle (Figure 5) of *P. graminis* f.sp. *tritici* consists of continual uredinial generations. The fungus spreads by airborne urediniospores from one wheat plant to another and from field to field. Primary inoculum may originate locally (endemic) from volunteer plants or be carried long distances (exodemic) by wind and deposited by rain. In North America, *P. graminis* annually moves 2000 km from the southern winter wheats to the most northern spring wheats in 90 days or less and in the uredinial cycle can survive the winter at sea level to at least 35°N. Snow can provide cover that occasionally permits *P. graminis* to survive as infections on winter wheat even at severe subfreezing temperatures experienced at 45°N (308). The sexual cycle seldom occurs except in the Pacific Northwest of the United States (306) and in local areas of Europe (364, 404). Although the sexual cycle produces a great genetic diversity (306), it also produces a large number of individuals that are less fit due to frequent recessive virulence genes (307) and to reassortment of genes for aggressiveness. *P. graminis* has successfully developed an asexual reproduction strategy that apparently allows the fungus to maintain necessary genes in blocks that are occasionally modified by mutation and selection.

Urediniospore germination starts in 1 to 3 hours at optimum temperatures (Table 2) in the presence of free water. The moisture or dew period must last 6 to 8 hours at favorable temperatures for the spores to germinate and produce a germ tube and an appressorium. Visible development will stop at the appressorium stage until at least 10,000 lux (16,000 being optimum) of light are provided. Light stimulates the formation of a penetration peg that enters a closed stoma. If the germling dries out during the germination period, the process is irreversibly stopped. The penetration process takes about 3 hours as the temperature rises from 18 to 30°C (318). The light requirement for infection makes *P. graminis* much more difficult to work with in the greenhouse than *P. recondita*. Most likely, light seldom has an effect in the field except when dew periods dissipate before daybreak.

Table 12. Cultivars susceptible to wheat stem rust and some of their important characteristics.

Cultivar	Wheat type	Growth habit	Day length requirement	*Sr* gene(s)[a]	*Lr* gene(s)[b]	*Yr* gene(s)
Agra Local	bread	spring	short	?		
Baart	bread	spring	short	LC	10	
Chinese Spring	bread	spring	short	9f	12,27,34	
Fertas	bread	spring	none		?	
Glossy Hugenot	durum	spring	none		?	
Line E[c]	bread	spring	short		?	
Little Club	club	spring	long	LC	?	
Local Red	durum	spring	short		?	
Marquis	bread	spring	long	7b,18,19,20,X	22b	
Maruccos 623	durum	spring	long	M	?	
McNair 701	bread	winter	none	McN	9	?
Morocco	bread	spring	none		?	
Prelude	bread	spring	short	16?	?	
Purdue 5481-C	bread	spring	long	7b,10	res.	
Red Bobs	bread	spring	long	7b,10		

[a] See Table 10.
[b] See Table 5.
[c] Susceptible to some isolates of *P. graminis* f.sp. *secalis*.

As the host matures, telia are produced directly from urediniospore infections or teliospores can be produced in a mature uredinial pustule. The teliospores are dicaryotic (N + N) and remain with the straw until spring. During this time, karyogamy occurs and the teliospores become diploid (2N). With spring rains and favorable temperatures, the teliospore germinates, undergoes meiosis, and produces a four-celled basidium. Each cell produces a stigma with a single haploid basidiospore (1N). The hyline basidiospore is windborne short distances (meters) to the barberry bush. Basidiospores germinate and penetrate directly. For maximum infection, the barberry leaf tissue should be less than 2 weeks old. Infection by a basidiospore results in the production of a pycnium (1N). The pycnium produces receptive hyphae and pycniospores of a single mating type (+ or -) that serve as female and male gametes for the fungus. Pycniospores of one mating type must be transferred to the receptive hyphae of the opposite mating type to initiate aeciospore development. The transfer of pycniospores is frequently done by insects, which are attracted to the oozing pycnial nectar produced by the pycnium. Mating of + and - types can also be facilitated by splashing rain, brushing of leaves, larger animals, and neighboring infections that coalesce. Aeciospores are dicaryotic (N + N) and are produced in aecia generally on the lower surface of the barberry leaves 7 to 10 days following fertilization. The aeciospores are the products of genetic recombination and may differ in their virulence and aggressiveness. The extent of variation depends on the differences between the parental isolates. *P. graminis* f.sp. *tritici* has been crossed with other formae speciales and crosses with *P. graminis* f.sp. *secalis* were relatively fertile (163). In Australia evidence points to recombination of wheat stem rust and the scabrum rust (*P. graminis* f.sp. *secalis*) (56, 214).

Aeciospores are hydroscopically released from the aecium and are airborne to wheat over distances of meters to perhaps a few kilometers. Aeciospores require similar conditions for infection to that of urediniospores. Infection by aeciospores results in the production of dicaryotic (N + N) uredinia with urediniospores. The repeating asexual cycle then involves urediniospores producing uredinia in about a 14-day cycle with optimum conditions. Under field conditions where temperatures vary greatly, the cycle can be either lengthened or shortened. Generally, lower temperatures in the field, at least in the early stages of the crop cycle, tend to lengthen the latent period. In northern India, a latent period of 31 days was recorded for stem rust (167).

Urediniospores are relatively resistant to light and temperatures at humidities of 20 to 30%. Wind frequently transports urediniospores 100 km in a viable condition and sometimes up to 2000 km (211). It is postulated that they have even been transported 8000 km from East Africa to Australia (392) at least three times this century (211).

Virulence

Worldwide virulence for *Sr2*, *13*, *22*, *24*, *25*, *26*, *27*, *29*, *31*, *32*, *33*, *34*, *37*, *Gt*, and *Wld-1* is limited. *Sr13* is ineffective at low temperatures, 18-20°C; *Sr29* and *34* may

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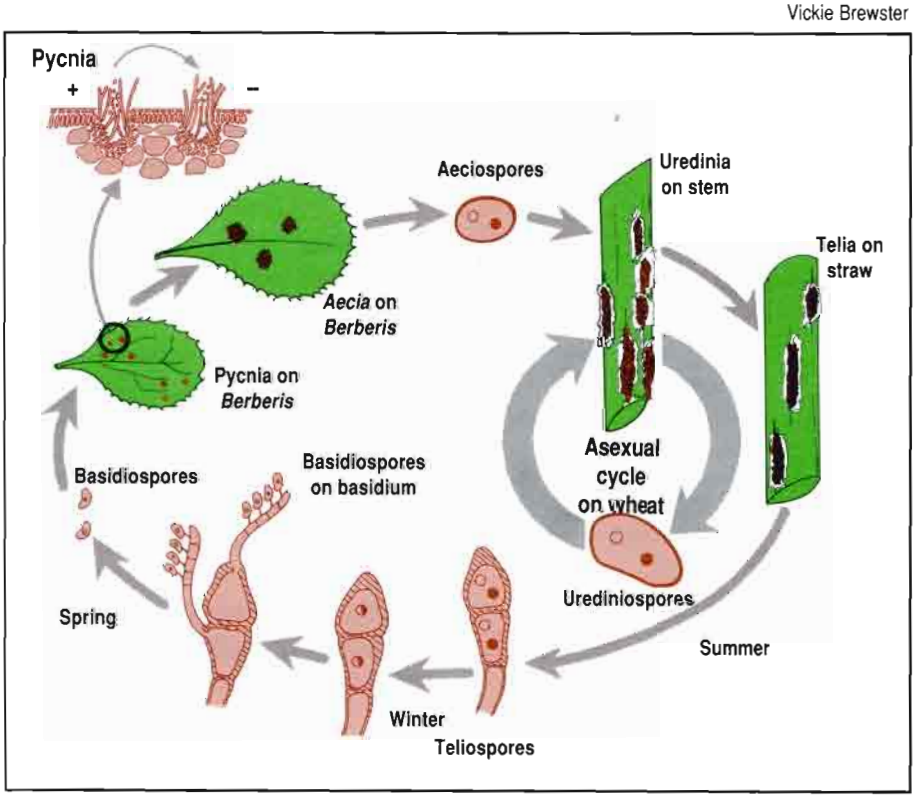


Figure 5. Life and disease cycles for *Puccinia graminis* f.sp. *tritici* (wheat stem rust).

be ineffective under high inoculum densities. Virulence for *Sr24* exists in South Africa (191) and Madagascar, for *Sr25* in India, and for *Sr27* in Australia (245). Isolates often appear to be virulent on *Sr24*, *29*, *34*, *Gt*, and *Wld* in the field due to the low level of effectiveness under high inoculum densities. *Sr37* virulent isolates have so far been unconfirmed and perhaps were obtained from the off-type plants. Virulence for *Sr26* has been undetected despite the widespread use of the cultivar Eagle and its derivatives in Australia. Likewise, the wide use of *Sr31* in Kavkaz and similar wheats with the 1B/1R translocation has not revealed virulence for *Sr31*.

Virulence for *Sr6*, *11*, and *17* is common wherever these resistances have been used. It would be expected that virulence to these genes could develop rapidly wherever they are extensively used. Virulence for *Sr5*, *9e*, and *21* seems to be common in some areas, but remains low or absent in other areas. Virulence is common for *Sr8b* (except in southern Africa and Australia-New Zealand); *Sr9a*, *Sr9d*, and *Sr14* (except North America); *Sr12* (except North America and Australia-New Zealand); *Sr15* (except Africa, North America, and Australia-New Zealand); *Sr16*; *Sr18*; *Sr19*; *Sr20*; and *Sr28* (except in China, India, Nepal, Pakistan, and Ethiopia). Avirulence on *Sr18*, *19*, *20*, or similar genes may explain the avirulence of rye stem rust (*P. graminis* f.sp. *secalis*) to wheat. Table 13 shows recent virulence surveys. Hamilton (129) and Luig (210) summarized the distribution of wheat stem rust races for the years 1955 through 1966 and on a worldwide basis, respectively. Green (120) described the evolution of virulence combinations in Canada.

Table 13. Recent virulence surveys of *Puccinia graminis* f.sp. *tritici* that are generally available in international literature.

Country	Year	Reference
Brazil	1982-85	Coelho and Sartori (70)
Bulgaria	1974-78	Kurjin (190)
Canada	1985	Martens (227)
Canada	1988	Martens et al. (228)
Czechoslovakia	1981-83	Bartos et al. (23)
Egypt	1974-76	Nazim et al. (266)
Ethiopia	1979-81	Dmitriyev (80)
Ethiopia	1982-83	Solomatin and Hussein (362)
France	1977	Massenot (231)
Germany (FDR)	1965-66	Hassebrauk (142)
Greece	1963-69	Skorda (357)
Hungary	1969-72	Bocsa (33)
India	1980-82	Bahadur et al. (17)
India	1983-86	Bahadur et al. (18)
India	1984-86	Mutkekar et al. (258)
Iraq	1967-69	Natour et al. (264)
Italy	1982-83	Siniscalco and Casulli (355)
Italy	1984	Corraza (71)
Kenya	1969-70	Harder et al. (132)
Korea	1971-72	Chung and Lee (69)
Mexico	1988-89	Singh (350)
Mozambique	1971	Fonseca (105)
Pakistan	1961-64	Hassan et al. (137)
Pakistan	1976	Hassan et al. (138)
Portugal	1980	Freitas (107)
Romania	1968-70	Negulescu and Ionescu -Cojocaru (267)
South Africa	1985	Le Roux and Rijkenberg (191)
Spain	1968-71	Salazar and Branas (328)
USA	1987	Roelfs et al. (304)
USSR	1968-75	Novokhatka and Kryzhanouskaya (269)
USSR	1969-71	Babajants (16)
USSR	1971	Azbukina (14)
Uruguay	1968	Bettucci et al. (30)
Yugoslavia	1976-83	Vlahovic (388)

Aggressiveness

The major factor in the survival of the pathogen is virulence to the common commercial wheat cultivars. However, there are many other factors required for a pathogen to successfully compete. The measurement of this complex set of characteristics has been difficult, especially using isolates obtained from natural epidemics where the most unfit individuals are rapidly outnumbered by the fit. For example, all isolates obtained from nature have approximately a 7-day latent period; however,

in an F_2 progeny from the cross of races 111 and 36 made by Loegering and Powers (202), individual isolates varied in latent period from 7 to 16 days. In North America, race Pgt-TPM has been the most commonly identified race for more than 15 years. Race Pgt-QTH has occurred as a small part of the population since at least 1968. In the past 10 years, these two races have been used to inoculate a number of nurseries of varying wheat genotypes, with many more lines susceptible to Pgt-QTH than Pgt-TPM. In all but 2 years, race Pgt-TPM has been the most commonly identified race from hosts susceptible to both races, while in the 2 warmer years Pgt-QTH was the most commonly identified. Race Pgt-QTH has always been a more important component of the pathogen population late in the season in Texas and Mexico when temperatures are usually higher. Perhaps race Pgt-QTH is more adapted to warm temperatures than Pgt-TPM.

Katsuya and Green (170) and Browder (44) studied the reproduction potential of races 15 (Pgt-TMM) and 56 (Pgt-MCC) and found that race 15 was more aggressive than race 56.

Stripe Rust

Stripe or yellow rust of wheat caused by *Puccinia striiformis* f.sp. *tritici* can be as damaging as stem rust. However, stripe rust has a lower optimum temperature for development that limits it as a major disease in many areas of the world. Stripe rust is principally an important disease of wheat during the winter or early spring or at high elevations. The map in Figure 6 shows regions of the world where stripe rust has been a major or local problem (as delineated in Table 3).

Stripe rust of wheat may be the cause of stripe rust on barley (372). In Europe a forma specialis of *P. striiformis* has evolved that is commonly found on barley and seldom on any but the most susceptible wheats (403). *P. striiformis* f.sp. *hordei* was introduced into South America where it spread across the continent (84) and is now in North America (CIMMYT, unpublished).

Hassebrauk (141, 143) and Hassebrauk and Robbelen (144, 145) have compiled a four-part monograph on stripe rust. Robbelen and Sharp (293) translated into English the sections that deal with breeding for disease resistance and genetics of the host-pathogen interaction. In 1988 Manners (226) reviewed the genetics of virulence and resistance of cereals and grasses. Chapters on stripe rust by Stubbs (372, 373) summarize much of the early work on stripe rust and provides more recent previously unpublished information on virulences of the pathogen worldwide. The evolution of the pathogen with the introduction of resistant cultivars in the Netherlands is also outlined.

Epidemiology

P. striiformis has the lowest temperature requirements of the three wheat rust pathogens. Minimum, optimum, and maximum temperatures for stripe rust infection are 0, 11, and 23°C, respectively (150). *P. striiformis* frequently can actively overwinter on autumn-sown wheat (Figure 7). Most of the epidemiology work has been done in Europe and was recently reviewed by Zadoks and Bouwman (404) and Rapilly (287).

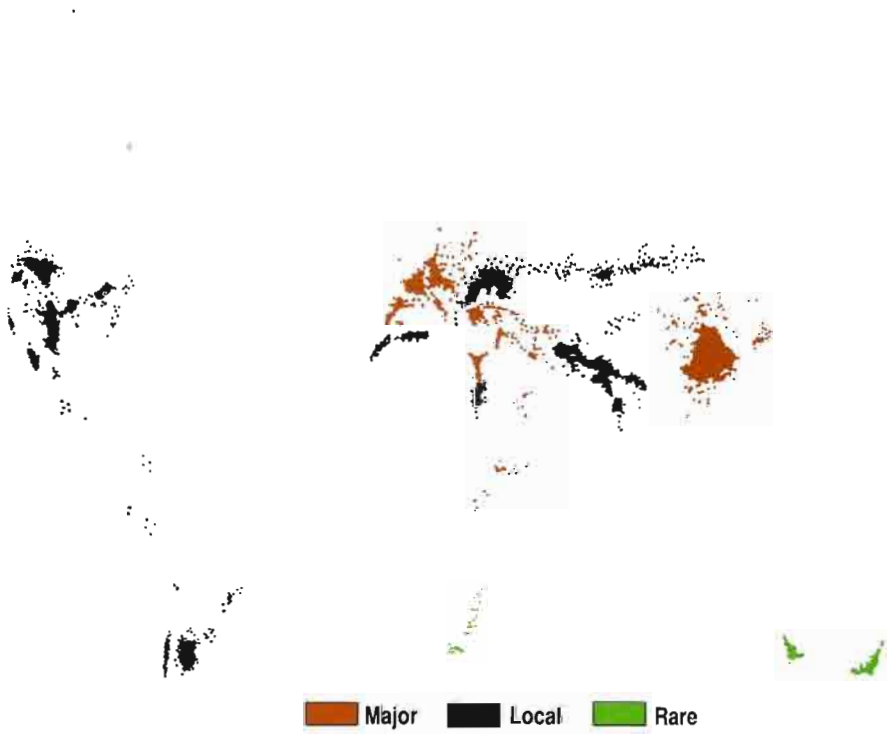


Figure 6. Wheat areas of the world where stripe rust has been a major or local problem.

Notes:

In Europe *P. striiformis* oversummers on wheat (403). The amount of oversummering rust depends on the amount of volunteer wheat which, in turn, is a function of moisture in the off season. The urediniospores are then blown to autumn-sown wheat. In northwestern Europe, overwintering is limited to urediniomycelia in living leaf tissues as temperatures of -4°C will kill exposed sporulating lesions. Latent lesions can survive if the leaf survives. In other areas of the world, snow can insulate the sporulating lesions from the cold temperatures so air temperatures below -4°C fail to eliminate the rust lesions. The latent period for stripe rust during the winter can be up to 118 days and is suspected to be as many as 150 days under a snow cover (403).

In areas near the equator, stripe rust tends to cycle endemically from lower to higher altitudes and return following the crop phenology (326). In more northern latitudes, the cycle becomes longer in distance with stripe rust moving from mountain areas to the foothills and plains.

Due to their susceptibility to ultraviolet light, urediniospores of stripe rust probably are not transported in a viable state as far as those of leaf and stem rusts. Maddison and Manners (220) found stripe rust urediniospores three times more sensitive to ultraviolet light than those of stem rust. Still Zadoks (403) reports stripe rust was wind-transported in a viable state more than 800 km. The recent introductions of wheat stripe rust into Australia and barley stripe rust into Colombia were probably aided by man through jet travel (84, 271). However, the spread of stripe rust from Australia to New Zealand, a distance of 2000 km, was probably through airborne urediniospores (29). Perhaps an average spore of stripe rust has a lower likelihood of being airborne in a viable state over long distances than of the other wheat rusts, but certainly some spores must be able to survive long distance transport under special and favorable conditions. There are several examples of the sequential migration of stripe rust. The 8156 virulence (cultivars: Siete Cerros, Kalyansona, Mexipak) was first recorded in Turkey and over a period of time was traced to the Subcontinent of India and Pakistan (326) and may be associated with the weather systems called the "Western Disturbance". As mentioned, barley stripe rust in South America migrated from its introduction point in Colombia to Chile over a period of a few years (84).

Most areas of the world studied seem to have a local or nearby source of inoculum from volunteer wheat (197, 372, 404). However, some evidence points to inoculum coming from noncereal grasses (146, 380). Future studies of stripe rust epidemiology need to take into account not only the presence of rust on nearby grasses, but also the fact that the rust must occur on the grasses prior to its appearance on cereals. The virulence phenotype must be shown to be the same on both hosts and that it moves from the grass to wheat during the crop season.

Stripe rust epidemics in the Netherlands can be generated by just a single uredinium per hectare surviving the winter if the spring season is favorable for rust development (404). Visual detection of a single uredinium per hectare is unlikely, however, as foci develop around the initial uredinium, it becomes progressively easier to detect.

Hosts

Puccinia striiformis is a pathogen of grasses and cereal crops: wheat, barley, triticale, and rye. Stripe rust is the only rust of wheat that

consistently spreads beyond the initial infection point. Resistance to stripe rust that results in a reduction in the number of infections or fewer spores per uredinium may be overcome by the pathogen's ability to spread without additional spores or infection periods.

Alternate hosts

Only the telial and uredinial stages of stripe rust are known. Eriksson and Henning (98) looked for the alternate host among species of the Boraginaceae. Tranzschel (383) suggested that *Aecidium valerianella*, a rust of Valerianella, might be related to *P. striiformis*. Mains (222) thought that *P. koeleriae* Arth., *P. arrhenatheri* Eriks., and *P. montanensis* Ellis, which have aecidial states on *Berberis* and *Mahonia* spp., might be related to *P. striiformis*.

Straib (370) and Hart and Becker (135) were unsuccessful in attempts to infect *Berberis*, *Mahonia*, and *Valerianella* spp. The alternate host of the rust, *P. agropyri* Ell. and Ev., is *Clematis vitalba*. This rust closely resembles *P. striiformis* so Viennot-Bourgin (387) suggested that the alternate host of stripe rust might occur in the Clematis family. Teliospores readily germinate immediately to produce basidiospores (400) and the teliospores probably do not assist the fungus as a winter survival mechanism. An epidemiological factor to consider is the possibility of infection of the alternate host late in the summer so aeciospores could infect the newly sown wheat or late cool season grasses. In some high altitude areas of West Asia, the wheat crop may take 13 months to mature. In such cases, early spring season infections of the alternate host would be possible.

Accessory hosts

P. striiformis seems to lack the clearly defined formae speciales that occur with *P. graminis*, and isolates of stripe rust seem to have a wider host range than those of *P. recondita*. Sufficient evidence exists for the separation of the primary wheat attacking form from the barley attacking form (372, 403).

Puccinia striiformis attacks members of the subfamily Festucoideae and Eragrostoideae with the principle hosts in the genera *Aegilops* (*Triticum* to some taxonomists), *Agropyron*, *Bromus*, *Elymus*, *Hordeum*, *Secale*, and of course *Triticum* (372). The assumption that stripe rust, which occurs on various grass species, has a similar virulence to that which is attacking wheat is probably not justified (225, 380). Likewise, the ability to produce a few uredinia on some plants of a species in greenhouse tests does not prove that species is a host under field conditions. Furthermore, there is no reason to expect that race-specific resistance does not occur in accessory hosts. Many of the existing race-specific genes for resistance have been transferred from species that are accessory hosts.

Primary hosts

Triticum spp. are a major host for stripe rust. Stripe rust on barley in Tibet has historically been an important disease where wheat is a minor crop. Comparisons between Tibetan and European stripe rust remain to be done. Rye was often reported as a host of stripe rust in the last century, but in more recent times rye is seldom seen to be infected by stripe rust (372).

Biffen (31) did the first resistance studies for wheat stripe rust. For several reasons, less is known about the resistance to this disease than the other wheat rust diseases. The disease requires somewhat more specialized controls in the greenhouse due to its sensitivity and the facts that: 1) infection types are less discrete, 2) there are numerous recessive resistance host genes (194), 3) many resistance genes have additive effects (344), 4) there are temperature-sensitive genes, and 5) many genes function only in the adult plant stages (285). Table 14 shows the current status of stripe rust resistance.

Many of the resistances against stripe rust have been of the additive temperature-sensitive and/or the adult plant types (195, 293, 344, 390). Some of these resistances are considered nonspecific (Table 15). It must be noted that changes in the pathogen races have resulted in the failure of many resistances to stripe rust suggesting specificity (198, 372). Still, most of these cultivars are less susceptible than Michigan Amber, *Triticum spelta saharense*, and Taichung 29 (372). Some resistances have been long lasting. In Europe the most durable resistance has been that of Capelle-Desprez (*Yr3a*, *Yr4a*, *Yr16*) (159), Juliana (*Yr14*, +), Carstens VI (*Yr12*, +), and Arminda (*Yr13*, +) (372). In the United States, the cultivars Gaines and Nugaines have provided resistance on a long-term scale (199). Some wheats developed by CIMMYT, such as Anza, also have had long-term resistance (160, 286). Table 16 lists susceptible hosts for stripe rust.

Pathogen

Gadd and Bjerkander first described stripe rust in 1777. It was reported to have caused an epidemic on rye in Sweden in 1794 (99). Schmidt designated the pathogen as *Uredo glumarum* in 1827; Westendorp designated

the stripe rust pathogen of rye as *Puccinia striaeformis* in 1854. Eriksson and Henning (99) chose the name *P. glumarium* in their comprehensive taxonomic work. Hylander et al. (154) and Cummins and Stevenson (75) revived the name currently in use, *P. striiformis* West. It probably is desirable to add the forma specialis if it has been determined.

Life cycle

P. striiformis is most likely a hemiform rust in that the life cycle seems to consist only of the uredinial and telial stages (Figure 7). Stripe rust populations can exist, change in virulence, and result in epidemics independent of an alternate host. Urediniospores are the only known source of inoculum for wheat and they germinate and infect at cooler temperatures

Table 14. Named genes for stripe rust resistance, source, genome location, low infection type to an avirulent culture(s), and tester lines (186, 372).

Yr gene	Genome location	Source	Response to avirulent culture: Seedling[a]	Response to avirulent culture: Adult[a]	Tester	Remarks	Reference
1	2A	Chinese 166	1	1	Chinese 166		Lupton and Macer (216)
2	7B	Heines VII	4	4	Heines VII	With *Yr*?	Lupton and Macer (216)
3a		Vilmorin 23	2	2	Wilmorin 23		Lupton and Macer (216)
3b		Hybrid 46	2	2	Hybrid 46	With Yr4b	Lupton and Macer (216)
3c		Minister	2	2	Minister		Lupton and Macer (216)
4a		Capelle-Desprez	2	2	Capelle-Desprez	With *Yr3a,16*	Lupton and Macer (216)
4b		Hybrid 46	2	1	Hybrid 46	With *Yr3b*	Lupton and Macer (216)
5	2BL	*Triticum spelta album*	1	1	*T. spelta album*		Macer (218)
6	7BS	Heines Kolben	4	4	Heines Kolben	With *Yr2*	Macer (218)
7	2BL	Lumillo durum	2	2	Lee	Linked to *Sr9g*	Macer (218)
8	2D	*T. comosa*	1	1	Compair	Linked to Sr34	Riley et al. (291)
9	1BL-1RS	Imperial rye	1	1	Riebesel 47/51, Clement, Fed/Kavkaz	Linked to *Sr31*, *Lr26*	Macer (219)
10	1BS	Moro	1	1	Moro		Macer (219)
11		Joss Chambier	-	2	Joss Chambier	Adult plant resistance	Priestley (283)
12		Caribo	-	2	Mega	Adult plant resistance	Priestley (283)
13		Ibis	-	2	Maris Huntsman	Adult plant resistance	Priestley (283)
14		Falco	-	2	Maris Bilbo	Adult plant resistance	Priestley (283)
15	1B	Dippes Triumph	1	1	*T. dicoccoides* G-25	With *Yr*?	Amitai et al. (6)
16	2DS	Capelle-Desprez	-	3	Capelle-Desprez	Adult plant resistance with *Yr3a,4a*	Worland and Law (399)
17	2AS	*T. ventricosa*	-	-	VPM1	Linked to *Lr37* and *Sr38*	Unpublished
18	7D	Anza, Condor	-	4 to 7	Anza, Condor	Adult plant resistance, linked to *Lr34*	Unpublished
A		Avocet	5	5	Avocet		Unpublished

[a] McNeal et al. (248)—see Table 22, Stubbs (372), and Knott and Johnson (186).

Table 15. Cultivars given in the literature as having nonspecific resistance to wheat stripe rust, their specific resistance as known, type of nonspecific resistance, and source of information.

Cultivar	*Yr* gene(s)	Type of nonspecific resistance	Reference	Remarks
Anza	A, 18	durable	Johnson (160)	
Arminda	13,+		Stubbs (372)	
Atou	3a,4a,16	durable	Johnson (160)	
Bon Fermier	3a		Stubbs (372)	
Bouquet	3a,4a,14,16?	durable	Johnson (160)	
Cappelle-Desprez	3a,4a,16		Lupton et al. (217)	
Carstens VI	12		Stubbs (372)	
Champlein	3a,4a,16		Johnson (160)	
Elite Lepeuple	2		Johnson (160)	
Flanders	1,3a,4a,16?		Johnson (160)	
Flinor			Johnson (160)	
Gaines			Line et al. (199)	
Heines VII	2		Stubbs (371)	
Holdfast			Johnson (160)	
Hybrid 46	3b, 4b		Johnson (160)	
Hybride de Bersee	3a,4a,16?	durable	Johnson and Law (161)	5BS-7BS chromosome
Ibis	1,2,13	temperature sensitive?	Stubbs (371)	
Itana		additive	Sharp and Volin (344)	
Joss Chambier	2,3a,11		Lupton et al. (217)	
Jubilar			Johnson (160)	
Juliana	14,+		Stubbs (372)	
Karamu	A	durable	Johnson (160)	
Little Joss			Lupton et al. (217)	
Luke			Line et al. (199)	
Manella	2,14		Stubbs (372)	
Maris Huntsman	2,3a,4a,13,16?		Johnson (160)	
Maris Widgeon	3a,4a,8,16?		Lupton et al. (217)	
Norda			Robbelen and Sharp (293)	
Nugaines			Line et al. (199)	
PI 178383	10	high-temperature	Sharp and Volin (344)	1 major,3 minor genes
Starke II			Johnson (160)	
Vilmorin 27	3a,4a,16?		Johnson (160)	
Wanser		field	Sharp et al. (345)	
Wilhelmina			Stubbs (372)	
Yeoman	13		Johnson (160)	

Table 16. Cultivars susceptible to wheat stripe rust and some of their important characteristics.

Cultivar	Wheat type	Growth habit	Day length requirement	*Yr* gene(s)	*Lr* gene(s)	*Sr* gene(s)[a]
Desprez 80	bread	winter				
Fertas	bread	spring	short			
Lemhi	bread	spring				10
Little Club	club	spring	long		?	LC
Local Red	durum	spring	short			
Michigan Amber	bread	winter				
Morocco	bread	spring	short			
Omar	bread	spring				
Strubes Dickkopf	bread	winter				
Taichung 29	bread	spring				
Triticum spelta saharense	spelt	spring	long			

[a] See Table 10

with the optimum reported at 9-13°C (Table 2). These temperatures, on average, are about 10°C below those for leaf rust; thus, stripe rust is a disease of more northern or southern latitudes and high elevations.

Sporulating uredinia survive to a temperature of -4°C and incipient infections can survive as long as the host leaf survives. Infections may occur at temperatures near or just below freezing (150). Latent periods of more than 188 days during the winter occur in Europe (403). Sporulation and infection can occur when daytime temperatures reach 5°C (404).

P. striiformis seems to be more sensitive to ultraviolet light and air pollution than the other rusts (252, 342, 372). This may affect the pathogen's survival in long distance transport and in highly polluted areas. Note, however, that Stubbs (372) feels isolates in northwestern Europe have a tolerance to local pollutants.

Virulence

Hungerford and Owens (153) reported the occurrence of strains of stripe rust on wheat and Allison and Isenbeck (5) showed the existence of races. Extensive studies were done in Germany in the 1930s, and again after 1955 (109). Stubbs (372) summarized this work (see Table 17). Currently, virulence studies of stripe rust are being done in the Netherlands (375), USSR (2), Peoples Republic of China (196), USA (198), India and Nepal (262), United Kingdom (284), and Australia (396). Findings of most of these surveys are not published except periodically for the international audience.

Aggressiveness

Little is known about differences in aggressiveness among isolates of *P. striiformis*. Differences in aggressiveness probably exist, but they are obscured by the variability in the resistance response. Additionally, differences in relative humidity, light, temperature, and pollutants combined with adult plant resistance have made studies of differences in pathogen aggressiveness difficult.

Vickie Brewster

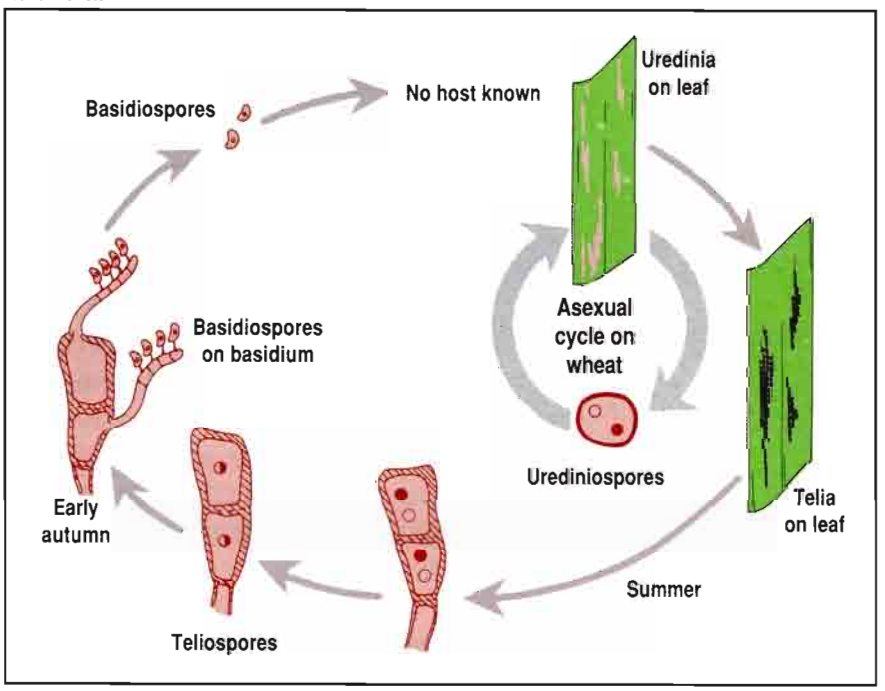


Figure 7. Life and disease cycles for *Puccinia striiformis* (wheat stripe rust).

Table 17. Zonal distribution and frequency of wheat stripe rust virulence in populations of *Puccinia striiformis* f.sp. *tritici*.

	Virulence for *Yr* genes[a]									
Zone	**1**	**2**	**3a**	**4b**	**5**	**6**	**7**	**8**	**9**	**10**
West Europe and North Africa	3	4	4	3	0	3	2	1	1	0
East Europe and West Asia	1	2	3	1	0	3	3	3	1	2
South Asia	2	1	1	0	1	2	4	4	0	0
Far East	4	2	3	0	0	3	3	2	1	0
North America	2	4	3	0	0	2	1	0	0	1
South America	1	3	4	3	0	2	2	0	1	0
Australia and New Zealand	0	4	4	4	0	3	2	0	0	0

[a] Frequency in percentage virulence in the race population: 0 = not known, 1 = less than 10%, 2 = 11-25%, 3 = 26-50%, and 4 = over 50%; after Stubbs (372).

Disease Control

It cannot be over emphasized that it is essential to understand the epidemiology of a disease before starting any control strategy, especially one involving cultural or chemical control measures. Without a doubt a combination of cultural control practices with disease resistance and perhaps fungicide applications will be the most effective means of controlling the cereal rust diseases. Because of the airborne nature of the inoculum of the cereal rusts, quarantine measures against the pathogen only delay, and do not prevent entry of the disease and/or specific virulence combinations. However, one should take care not to unknowingly transport or permit urediniospores of the cereal rusts to escape outside their epidemiological areas. Important differences in virulence, aggressiveness, and adaptation exist in the different pathogen populations of these fungi worldwide.

Table 18 summarizes the various control methods discussed in the following sections.

Genetic Resistance

The principle mechanism of control of the cereal rusts has been through the use of resistant cultivars (159). A few cultivars, such as Thatcher and Hope (133) for stem rust; Americano 25, Americano 44d, Surpreza,

Frontana, and Fronteira (278, 303) for leaf rust; and Wilhelmina, Cappelle-Desprez, Manella, Juliana, and Carsten's VI (372) for stripe rust have maintained some resistance for many years. Most cultivars have remained resistant for 5 years or more, which is about the agronomic lifespan of a cultivar where an active breeding program exists. However, some cultivars have rusted before they were grown on more than a fraction of the cultivated acreage. In most, if not all the cases, the failures have been due to inadequate knowledge of the virulences present in the pathogen population. In other cases, mutations or perhaps recombinations of existing virulence combinations occurred and rendered the host susceptible. In some instances, the disease screening protocol is inadequate to identify and select the resistant wheat lines.

The failure of resistance over the short term has led to a boom-and-bust syndrome (173). However, among the breeding programs for rust resistance, some have been successful for a number of years. The greatest successes have been against stem rust, perhaps because of the nature of the pathogen, and perhaps due to the greater number of scientific years of study and work. Green and Campbell (122) have summarized the success of the Canadian stem rust program. In Australia a series of cultivars with *Sr26* have been released since 1971 and are now grown on nearly 1 million hectares without stem rust losses (211). Maintenance of leaf rust resistance has been more difficult, but the series of diverse cultivars used in North America has been undamaged by rust for more than 30 years (295). The bread wheat, Era, was released in Minnesota in 1972 and rapidly replaced other cultivars; by 1980 it was grown on nearly 1.5 million hectares annually. Recently, it has been replaced by other cultivars with similar resistance; Era and its derivatives still remain rust resistant on some 1 million hectares.

Many reviews of resistance to the cereal rusts exist. A 1987 CIMMYT symposium entitled "Breeding Strategies for Resistance to the Rusts of Wheat" (347) provides a good summary of recent work. Other recent sources are chapters on resistance of the race-specific type (90) and resistance of the race-nonspecific type (274) and the book by Knott (184).

Advantages

- May reduce or eliminate the need for chemical control.
- Requires no action by farmers after cultivar selection.
- Cost spread to all users of the cultivar.
- Control can be maintained through seed supply.
- No known environmental impact.

Disadvantages

- Resistance may become ineffective after a period.
- Diverts effort from breeding for yield.
- No change possible after planting.
- Requires knowledge of pathogen virulence and evolution.

Chemical (Fungicide) Control

Chemical control has been successfully used in Europe permitting high yields (6-7 t/ha) and where prices for wheat are supported (55, 374). Chemicals have also been used to control a leaf rust epidemic in 1977 in the irrigated Yaqui and Mayo Valleys of Mexico (85). Elsewhere, chemicals have had limited use on high yielding wheats in the Pacific Northwest of the USA for stripe and leaf rust control. Chemical control of leaf rust in the eastern and southern United States has been practiced when expected yields exceed 2 t/ha. In Brazil and Paraguay, chemicals are used on wheat with expected yields of 1 t/ha and above to control an array of other diseases.

Table 18. Methods of controlling the rust diseases.

Control method	Controlled by	Cost to	Cost	Effectiveness
Resistance				
Gene pyramids	Breeder	Taxpayer/seed buyer	Low	Good
Gene deployment	Breeder group	Taxpayer/seed buyer	Moderate	Good
Multilines	Breeder	Taxpayer/seed buyer	High	Fair to good
Cultivar mixtures	Seed producer	Seed buyer	Moderate	Fair to good
Chemical	Grower	Grower	High	Good
Cultural	Grower	Grower	Low	Fair
Eradication of alternate hosts	Legal system	Taxpayer	High	Fair to good

Notes:

Advantages

- Chemicals can be applied when needed.
- Little monitoring of pathogen populations is required except for appearance.
- Breeding efforts and funding can be concentrated on increasing wheat yields and improving quality.

Disadvantages

- Farmers assume direct material and application costs.
- Large stores of chemicals need to be maintained—some of which have limited shelf life. Adequate amounts of chemicals must be stored to spray large areas (hundreds of thousands of hectares) in a few days.
- Known or unknown environmental hazards are connected with continual use of fungicides over a large area.
- Many other fungal pathogens have developed resistance to chemicals and this may occur with the rusts.
- Most available fungicides provide inadequate control on susceptible cultivars when environmental conditions are favorable for disease development.

Cultural Methods

Cultural practices provide another method for at least partial control of wheat rust epidemics. No single practice is effective under all conditions, but using a series of cultural practices greatly enhances the existing resistances. Farrer's development and use of early maturing cultivars marked the initial successes in controlling stem rust in Australia (232). Mexican farmers had learned to sow early to avoid stem rust prior to the use of resistant cultivars (34).

Zadoks and Bouwman (404) emphasized the importance of the green bridge in carrying the disease from one crop to the next. The green bridge can be lengthened when some growers plant early and others late. Removing the green bridge with tillage or herbicides is an effective control measure for epidemics that would result from endogenous inoculum. In some areas volunteer plants must be controlled several times during the season when wheat is not grown.

Some of the benefits of gene deployment can be obtained by a grower if more than one cultivar are used that differ in resistance and from those grown by immediate neighbors. In some areas, control of timing and frequency and amount of irrigation and fertilization applications can aid in disease control. On large farms, it may help if fields are arranged so that the early maturing cultivars are down wind from late maturing cultivars. Late planting may avoid autumn infections, but late planting may increase the chance of spring infection by exogenous inoculum. As a disease control measure, autumn- and spring-sown wheats probably should not be grown in the same area. Whatever the situation, each cultural practice must be tested against the anticipated types of epidemic that occur in the area.

Advantages

- Reduce environmental pollution.
- Enhance effectiveness of chemicals when used.
- Enhance effectiveness of resistance.
- Delay disease onset and thereby severity.

Disadvantages

- Farmers may lack knowledge and resources to use these methods properly.
- Require the cooperation of most or all farmers in an area.
- Can be rendered useless by large inputs of exogenous inoculum.

Eradication of the Alternate Host

An alternate host eradication program for stem rust was successful in northern Europe (148) and the North Central States of the USA (295). Except for eastern Europe and the northwestern USA, no other areas of the world are known where alternate hosts play any role in stem rust epidemiology. Eradication efforts by individual growers probably would not result in visible gains immediately in stem rust control due to large amounts of asexual inoculum. The alternate host for leaf rust may function more as a source of sexual reproduction than a source of epidemic-generating inoculum. For southern Europe eradication of *Thalictrum* or *Anchusa* would probably not be feasible.

Advantages

- Increases the durability of resistance genes.
- Can delay disease onset and initial disease severities.
- May reduce need for chemical and/or cultural control measures.

Disadvantage

- Eradication of alternate host often not economically feasible.

Techniques for Study of Rust Diseases

Many techniques have been developed for studying the rust diseases. To describe them all would require a series of volumes. Thus, we describe techniques in this manual that we believe are generally the most useful and practical. Those germane to specific purposes are mentioned briefly. References are provided for generally available literature. Browder (48), Joshi et al. (168), Rowell (318), and Stubbs et al. (376) have written recent reviews.

Inoculum Production

Studies of the cereal rusts require the increase and preservation of inoculum, which, in most cases, involve urediniospores. For many experiments, inoculum of a particular pathogen phenotype or a particular isolate is needed. In such situations, it is essential to be able to purify and maintain isolates over a period of years. In other cases, larger quantities of inoculum for field inoculation may require multiplication, collection, and storage for various periods of time.

Spore increase

The usual procedure is to select a susceptible host (Tables 7, 12, and 16, for leaf, stem, and stripe rusts, respectively). A local host line can be used if it is susceptible to the isolate to be increased. Sometimes it is possible to select a host that is susceptible to the isolate to be increased, but resistant to other isolates, eliminating some of the contamination problems. It is desirable, but usually impossible, to find and use a host that is resistant to other common greenhouse diseases, such as powdery mildew (*Erysiphe graminis*).

Urediniospores are easily airborne and may be present outside the laboratory, representing a potential source of isolate contamination. Infected plants growing in the greenhouse and plants not immediately discarded after use are common sources of contaminating spores. Place waste plants in a covered barrel for several days of composting before sending them for disposal. To reduce contamination in work areas where rusted plants were grown, wash the area with water before bringing in new plants. To reduce contamination to a minimum, keep the greenhouse clean.

Several situations may cause problems in obtaining adequate infection (318). When spores have been stored dry, a slow rehydration process may be required. If seedlings or adult plants are sprayed with water to simulate dew formation, mineral or other contaminants in the water may inhibit spore germination. Pollutants in the air have been reported to reduce infection as well (252, 342, 372).

Inoculum can be increased either on seedlings or adult plants. The choice is based primarily on personal preference and local conditions.

Seedling plants. Generally, inoculate seedlings at 7 to 9 days of age when the primary or seedling leaf is fully expanded. Following incubation in a dew chamber, move the seedlings to a greenhouse or growth chamber. To prevent contamination by spores from other isolates and urediniospores from outside, isolation of the seedlings is desirable. Isolates maintained on seedlings in single, small pots can be covered with a glass lamp chimney, which has the top covered with a fine mesh cloth to allow heat exchange, but minimizes spore movement. Browder (48) designed an isolation chamber composed of a chimney and a cap with a space for air exchange.

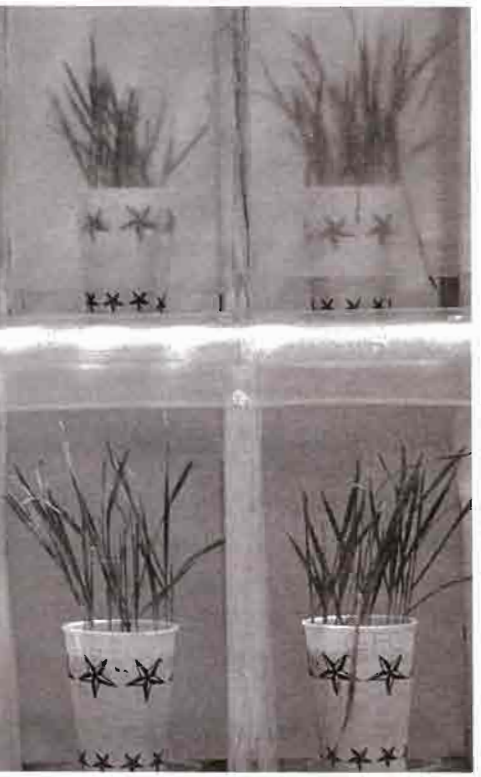


Figure 8. Example of easy-to-construct cages for maintaining isolates of the cereal rusts on seedling host plants.

Small plastic-covered cages, 30 x 25 x 20 cm, can be constructed in which single pots or cups can be placed (Figure 8). Some cage designs have the front flap hinged at the top and about 3 cm short of reaching the cage floor to allow air to enter with minimal spore exchange and to allow access for watering and spore collection. It is important to have cages large enough so the plastic does not come into contact with the inoculated part of the seedling, as some plastics are coated with a phytotoxic substance. If the humidity in the cage is very high, spore viability is affected. If dew formation or guttation drops are present for long periods of time, reinfections may occur. Best results are normally obtained when spores are collected in the afternoon. Plants may be treated with maleic hydrazide at a rate of 5 to 10 mg with 50 ml of water per pot (10-cm diameter) at emergence to reduce plant growth and enhance spore production (318). Flats of any size, thickly sown with wheat (page 39), can be used for large increases of inoculum. A 30- x 25-cm flat can produce 5 g of urediniospores which may be collected with a large cyclone collector (65).

Advantages

- Less space and time required.
- Easier to move plants in the greenhouse.
- Fewer problems with greenhouse pests.
- Minimizes the risk of contamination by other rust isolates.

Disadvantages

- Spores produced in small quantities.
- Frequent need to repeat the process.

Adult plants. Adult plants are also used as hosts for inoculum increase. Inoculum can be collected directly from the field, but it is often a mixture of pathotypes, which may or may not be desirable. Field-collected inoculum is often contaminated with spores of other fungi that can affect subsequent experiments. To increase inoculum on adult plants in the greenhouse, it is important to select a susceptible host, maintain isolation, avoid dew formation in the isolation chamber if possible, and have high sanitation standards to prevent infection or infestation of the host with undesirable diseases or insects. High levels of humidity often result in the development of hyper-parasitism of the rust and lower spore viability of the rust fungi. Contamination is most likely to take place before or during the inoculation and incubation processes. Contamination can also occur when isolates are watered or collected. This is particularly important where isolates are maintained for long periods of time on adult plants.

Advantages

- Isolates can be maintained for 30 to 60 days without reinoculation.
- By using injection techniques for inoculation, no special incubation equipment or procedures are required.

Disadvantages

- Larger cages required.
- Insect and disease control problems before and after inoculation, particularly spider mites, aphids, and powdery mildew.

Spore collection

Tapping. Urediniospores can be collected in large numbers by tapping a rusted plant over a piece of smooth dry paper or aluminum foil (48). Plastic is unsatisfactory because static electricity usually results in the clinging of spores to the plastic's surface. After collections are dried (20-30% relative humidity), the spores can then be stored in a container. A modification of this method involves tapping the plant directly over a funnel or container. It is essential to clean and dry the funnel between collections. Take precautions not to tap soil debris or water drops into the spore collection and avoid collecting aphids. Aphids

and other particles can be removed by screening, but a considerable number of spores will be lost in the process. Any aphids or water drops remaining in the spore collection will raise the moisture content of the mass, resulting in reduced spore viability. To remove spores in the immediate area and air after collection, use a fine spray mist or fog.

Advantages

- Inexpensive.
- Easy procedure.

Disadvantages

- After each collection, many spores are dispersed into the air, which can serve as a source of contamination.
- Quantities collected are relatively small.

Cyclone collectors. Different types of cyclone collectors (for example see Figure 9) now exist. They require a power source for creating a vacuum and centripetal force (43, 47, 65, 192, 378). These devices greatly enhance collecting both small amounts (mg) of spores from a single uredinium to large amounts (kg) from field plots.

Advantages

- Rapid process.
- Easy to use.
- Smaller amounts of urediniospores are released into the air.

Disadvantages

- Need a power source.
- General unavailability of these collectors in regular commercial channels.

Collecting small samples. The most common way of collecting a small sample of spores from the field is to gather 4 to 10 rusted stem or leaf sections (75-100 mm long). Fold leaves in half across the mid-rib to prevent rolling as they dry. Remove the nodes from stem sections so that they will dry better. Place the plant material immediately in a glycine (pollination bag) or thin paper envelope. Collections should never be put in plastic, waterproof, or other heavy bags as they will remain moist and rot. Avoid collecting excessive unrusted plant material because it often results in residual moisture that will rot the collection. If a wheat spike is included for identification purposes, attach it in a separate bag or envelope. It is best to make collections from dry plants when they are free of moisture. If collections must be made from wet plants, either place the collected plant material between blotters, which are changed every few hours, or lay the collection envelopes out individually at room temperature (18-25°C) until the material dries, usually 1 or 2 days. Never leave a collection in direct sunlight or in a closed room or container. Avoid heaters, closed cars, and external mailboxes. The spores can be easily removed from the plant surfaces with a scalpel or small cyclone collector.

Single spore technique. The collection of one spore for making a single spore isolate can be done easily under 50x magnification of a dissecting scope (127). Dust loose urediniospores onto a microscope slide or other similar surface. Attach a short (2 cm), stiff hair to the end of a wooden stick with a drop of glue. Rubbing the hair between clean fingers will impart a slight electrical charge to the hair, which can then be used to approach a spore under the dissecting scope. The electrical charge will cause the spore to attach itself to the hair. Check visually to make sure a single spore is indeed attached to the hair; then transfer the spore to a plant surface by rubbing the hair against the plant tissue. Check again under the microscope to ensure that the spore actually separated from the hair (127). An experienced technician can do this several times per minute.

Figure 9. The components of a typical cyclone collector.

Spore storage

There are different methods of spore storage depending on the length of storage time required and the amount of spores involved (summarized in Table 19).

Room temperature. Urediniospores can be stored at room temperatures for short periods of days (stripe rust), weeks (stem rust), and months (leaf rust) depending on moisture. The storage time can be increased by drying and maintaining the spores at 20-30% relative humidity over a desiccant.

Advantages

- Inexpensive.
- Easy to do.

Disadvantage

- Spores remain viable for a relatively short period.

Refrigeration. After drying the urediniospores, they can be stored at 5-8°C for variable periods of weeks or months depending upon the rust and basic conditions. They must be sealed in an airtight container or kept in a desiccator. This period can be perhaps doubled by storing the spores in a nontoxic isoparaffinic oil or in a partial vacuum desiccator. Urediniospores on dried stem or leaf pieces can be stored for several weeks in a refrigerator. Refrigeration of masses of wet spores is not recommended.

Advantages

- Relatively inexpensive.
- Easy to do.
- Longer spore viability.

Disadvantage

- Power source required for refrigerator.

Vacuum drying. Vacuum drying (Figure 10) of urediniospores in vials makes storage possible for up to 10 years (343). Dry the spores under reduced pressure (40 to 50 Torres). At this reduced pressure, use a flame to seal the open end of the vial containing the spores. This is the most critical part of the operation because, if the vial is pulled during this process, a small hole might develop through which moisture will return—causing the spores to lose viability. Vials are generally stored at 5-8°C for long-term storage (longer than 1 year). They can be stored at room temperature for periods of 1 year or less. After removing the spores from storage, slowly rehydrate them over a period of about 3 hours at 50% relative humidity (315). However, in cases where dew forms slowly on the plants after inoculation, this extra rehydration step may be unnecessary.

Advantages

- Long-term storage up to 10 years.
- Large quantities can be stored.

Disadvantages

- Difficulty in sealing the vials.
- Special equipment and training are required.

Liquid nitrogen. Most major laboratories worldwide use a method where urediniospores can be stored for long periods in liquid nitrogen at -196°C (205, 206). The spores are dried to 20-30% relative humidity and then sealed in

Table 19. Approximate storage life of dry (20-30% relative humidity) urediniospores for five methods.

	Length of storage		
Conditions	**Leaf rust**	**Stem rust**	**Stripe rust**
Room temperature	Months	Weeks	Days
Refrigeration	6 months	Month	Weeks
Vacuum drying	Years[a]	Years[a]	Years[a]
Liquid nitrogen	Indefinite[ab]	Indefinite[ab]	Indefinite
Ultra-low refrigeration	Years[ab]	Years[ab]	Years

[a] Rehydration recommended for about 3 hours at 50% relative humidity.
[b] Heat shock, 40°C for 5-7 minutes, recommended when removed from storage.

glass vials or aluminum packets. Stem and leaf rust urediniospores require a heat treatment in a 40°C water bath for 5 to 7 minutes to break cold-induced dormancy upon removal from storage (206). Rehydration of the urediniospores is desirable. Usually, the heat shock treatment and rehydration are not necessary for stripe rust urediniospores. Because of the extremely cold temperatures, special procedures are required to identify and locate the isolates (192). If glass vials are improperly sealed, they may explode after removal from the liquid nitrogen. The use of polyethylene-coated aluminum bags avoids this hazard. Observe standard precautions for handling liquid nitrogen.

Advantage

- Spore viability remains unchanged over many years.

Disadvantages

- Operator risks injury due to the extreme cold.
- Chance that poorly sealed vials will explode when they are removed from liquid nitrogen.
- Liquid nitrogen and the special container required are expensive items.
- Liquid nitrogen must be added to the refrigerator about every 2 weeks, requiring a nearby source of liquid nitrogen.

Ultra-low refrigeration. More recently, it has been found that the longevity of spores stored at any temperature below -50°C is similar to those stored in liquid nitrogen. Commercial companies now sell such ultra-low refrigerators. Dry the spores to 20-30% relative humidity and then seal them in plastic bags, glass, or plastic vials. We have also stored dried leaves with uredinia on them in unsealed glycine bags with good recovery for up to 18 months. It is important to cool the material rapidly, so with large quantities some workers use liquid nitrogen as a cooling agent. The Cereal Rust Laboratory freezes gram-sized lots in the ultra-low refrigerator by placing them in a sealed plastic bag and laying them flat on the refrigerator floor. The urediniospores of *P. recondita* and *P. graminis* require the same heat shock treatment on removal as spores removed from liquid nitrogen storage.

In the event of a power failure, availability of a backup storage system for important isolates is essential. Thawing due to power outages of 24 hours can result in a relatively high loss of spore viability, but few isolates have ever been lost. Some decrease in spore viability occurs with time compared to liquid nitrogen, but isolates have been recovered after 10 years of storage.

Figure 10. Apparatus for drying urediniospores in a vacuum prior to long-term storage.

Advantages

- Easy operation.
- Easy recovery of spores.
- No special hazards.

Disadvantages

- Dependence on uninterrupted electrical power.
- Equipment relatively expensive.

Inoculation Methods

Spores can be placed on the plants in a number of ways (summarized in Table 20). The method selected depends on the purpose of the inoculation, the number of plants to be inoculated, the amount of inoculum available, and the occurrence of a favorable dew or wet period during the inoculation process. A short dew period can result in spore germination, but no infection. If the spores are placed on wet plants in the morning, they may germinate but will fail to establish infection before the dew evaporates.

Dusting

This method employs the principle of dispersing dry spores over plants either with or without a carrier. A small mechanical duster, aspirator or even a cloth bag can be used to aid dispersion. If more than a single isolate is involved, the use of an uninoculated check is desirable to detect contamination levels. A recent improvement involves placing plants on revolving tables (48, 251) in a released cloud of urediniospores.

Advantages

- Inexpensive and simple.

Disadvantages

- Little control of inoculum density.
- Requires large amounts of spores.
- Results in a large number of spores being dispersed into the air, which contaminates equipment and clothing.

Brushing

This method, in which infected host plants are rubbed over the plants to be inoculated (48, 103, 369), provides fairly uniform inoculation. Brushing was the major inoculation method in early greenhouse experiments and has been used to start small infection centers in the field. One procedure is to place plants in a closed inoculation chamber. Infected plants (inoculum source) are carried to the inoculation chamber in a closed container. After brushing, thoroughly mist the chamber and surrounding

Continued on page 41

Table 20. Four methods of rust inoculation and three carriers for the inoculum.

	Control of inoculum	Risk of contamination	Spores needed	Equipment required	Cost	Labor	Other needs
Inoculation methods							
Dusting	Limited	High	Many	Duster	Low	Low	Dew (Moisture)
Brushing	Poor	High	Many	None	Low	Moderate	Dew
Injection	Excellent	Low	Few	Syringe	Low	Intensive	None
Spraying	Excellent	Moderate	Few-many	Sprayer	Low	Low	Dew
Carriers for inoculum							
Talcum powder	Fair	High	Moderate	Duster	Low	Low	Dew
Mineral oil	Good	Moderate	Few-many	Sprayer	High	Low	Dew after oil evaporates
Water	Fair	Moderate	Moderate	Sprayer	Low	Low	Dew before water evaporates

Adult Plant Host Responses to the Rust Diseases

Leaf Rust

R

MR

MS

S

Stem Rust

R

MR

MS

S

Stripe Rust

R (2)

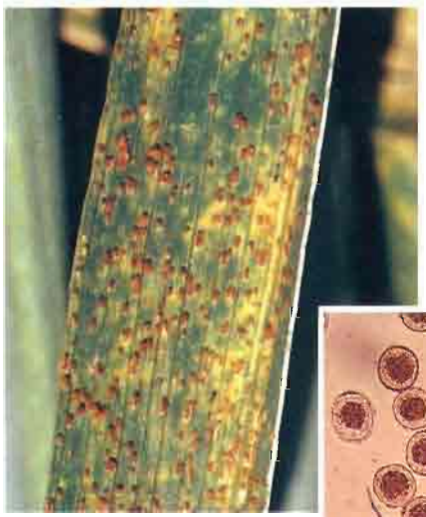
MR (4)

MS (7)

S (9)

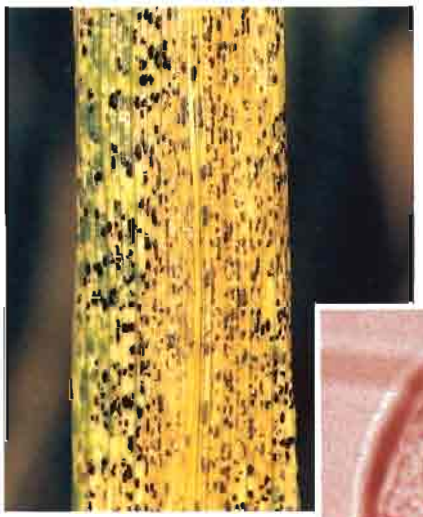
Symptoms and Spore Morphology of the Rust Diseases

Leaf Rust

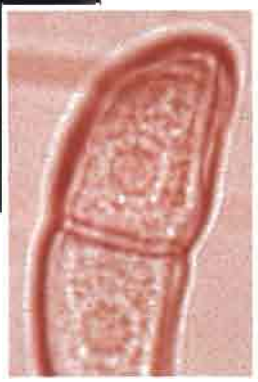


Uredinia

Urediniospores (400x)



Telia

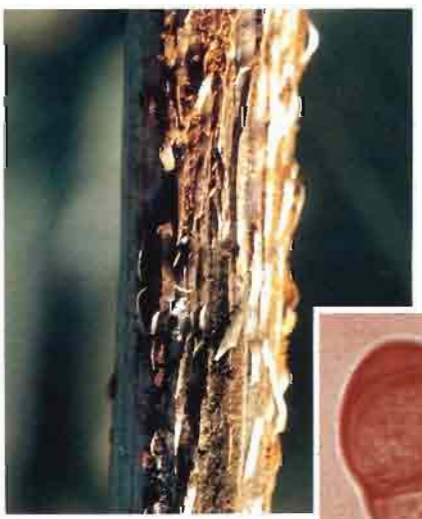


Teliospores (400x)

Stem Rust

Uredinia

Urediniospores (400x)

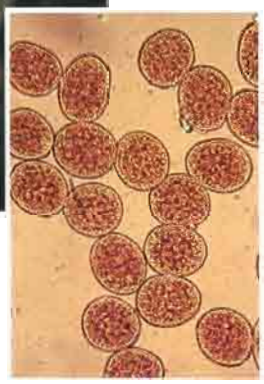


Telia

Teliospores (400x)

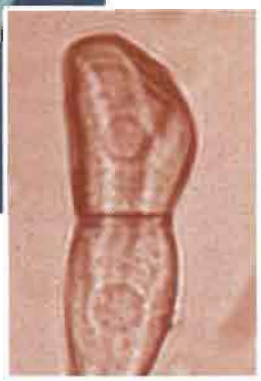
Stripe Rust

Uredinia



Urediniospores (400x)

Uredinia in the spikelets

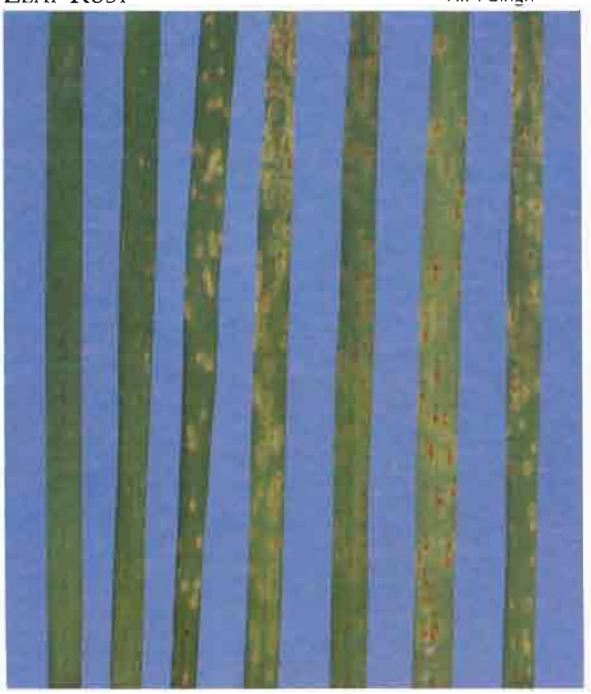


Teliospores (400x)

Example of a flat thickly sown with wheat for large increase of inoculum (see page 32).

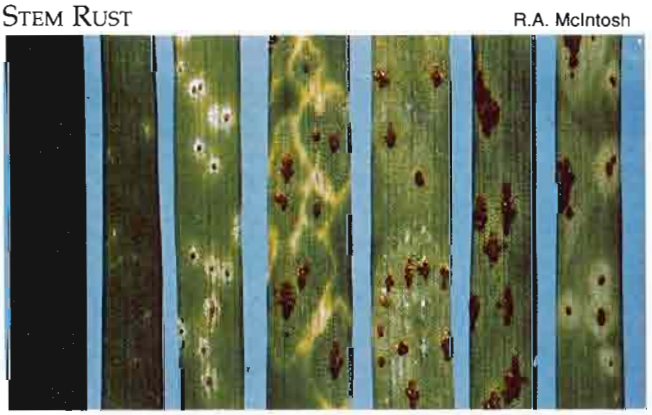
Seedling Infection Types of the Rust Diseases

Leaf Rust R.P. Singh



0 ; 1 2 3 4 X

Stem Rust R.A. McIntosh



0 ; 1 2 3 4 X

Stripe Rust R.W Stubbs

0 1 3 5 7 9

area with water so that the spores are washed to the floor.

Advantages of brushing

- Rapid and easy.

Disadvantages of brushing

- Difficult to control infection density.
- Contamination of facilities with spores.

Direct brushing is not used in the field. Instead, the rusted plants are placed or transplanted in the field at regular intervals (like 1 m) within the susceptible borders as soon as conditions become favorable for disease development. For best results, the spreader plants should be about the same size as the plants to be inoculated.

Advantages of spreader plants in the field

- Spores are released for several days to perhaps 2 weeks.
- The daily production of inoculum reduces the necessity of a favorable incubation period on a given day.

Disadvantages of spreader plants in the field

- A large number of infected plants are required.
- Detrimental effects of transplanting.
- Intensive labor required.
- Time required for disease spread.
- Rain can wash the spores from the source plants limiting disease spread.

Injection

Spores in a water suspension made with a small amount of wetting agent (such as a mild soap or Tween 20) can be injected into adult plants when the tillers are large enough for needle injection. Place the needle above the last existing plant node and inject the spore-water suspension upward until a drop of the spore suspension appears at the top of the leaf whorl. For leaf rust inoculations, do this before flag leaf emergence. Stem and stripe rusts can be inoculated as late as the early boot stage; however, earlier inoculations are much more desirable to get even distribution of inoculum through the plot area. Generally, inoculate one to three tillers/meter.

Advantages

- The leaf whorl or boot is the dew chamber.
- Less dependent on environmental conditions.
- Risk of contamination is low.

Disadvantages

- Time consuming and labor intensive.
- Can result in uneven disease severity.

Spraying

Spores in a water suspension made with a small amount of wetting agent, prepared in same way as for injection, can be sprayed on seedlings or plants of any growth stage. Nonphytotoxic isoparaffinic oils (8, 315, 321) can also be used as carriers and are recommended especially for greenhouse inoculation where more uniformity is desirable (see section on mineral oils below). An atomizer or sprayer with a fine nozzle can be used for spraying. If water is used as a carrier, the recommended time for spraying in the field is late in the evening just before or after dew formation. For large areas power sprayers can be used.

Advantages

- Easy.
- Inexpensive if water is the carrier.
- Large areas can be inoculated.

Disadvantages

- Oil may be expensive.
- Excessive oil is phytotoxic.
- Depends on dew formation.

Spore carriers

The principal carriers for spores are talcum powder, light weight mineral oils, and water (Table 20). Some other powders such as flour and chalk can also be used.

Talcum powder. With this carrier, inoculum density is controlled by the amount of spores mixed with the talc and the quantity dusted on to the plants. A skillful operator and the quality of the mechanical applicator can control to some extent the uniformity of inoculum application. Generally, the spores should be released up wind and allowed to drift over the plot. Power dusters work best under still conditions. Inoculum can be applied at any time during the day, but it must be followed by an adequate dew period.

Advantages

- Spores are applied dry.
- Cheap and easy to use in the field and greenhouse.

Disadvantages

- Urediniospore contamination of the air.
- Air movement important with passive application.

Mineral oils. Many rust workers currently use nonphytotoxic isoparaffinic oils as spore carriers in the field and greenhouse (315). Oil inoculation has led to many techniques to provide for a more uniform inoculum distribution (8, 321). Browder (45, 48) has developed inoculators for use in the greenhouse. Backpack mist blowers are excellent for field use, but any sprayer with a fine nozzle will work. The oil must

Notes:

evaporate prior to dew formation or misting of plants. Water drops can form over the oil and prevent it from evaporating, which means water cannot reach the spores to initiate germination. In the field, do not begin inoculation after dew is starting to form or if rain is expected in less than 1 hour. In the greenhouse, allow approximately 30 minutes for the oil to evaporate before placing plants in the dew chamber. If room humidity is low, less time is required; if relative humidity is 90% or above, it might be a good idea to wait 60 minutes before placing inoculated plants in the dew chamber.

Most oils kill conidiospores of powdery mildew, so if mildew is a contaminant in the rust inoculum, add the oil to the spores to be used as inoculum about 1 hour before inoculation to allow the oil time to reduce the number of viable mildew spores.

Advantages

- Low number of urediniospores required.
- Spores settle rapidly from the air due to weight of the oil drop.
- Uniformity of inoculation.

Disadvantages

- Excessive oil can be phytotoxic.
- Oils are expensive.
- Availability of oil is limited.

Water. Water should be as pure as possible and free of chlorine. The urediniospores settle out rapidly even with agitation—when placed in a drop of water, spores travel rapidly to the edge. To help maintain the spores in suspension, add a nonphytotoxic wetting agent, such as Tween 20 or mild soap. Once the spore suspension is made, use it within 1 to 3 hours, otherwise the spores may germinate resulting in low infectivity.

A number of application methods can be used with a water carrier. One method is to float urediniospores in a large beaker or pail. The plants to be inoculated are inverted, dipped, and slowly extracted. Syringe injection and spraying are other inoculation methods.

Water can be used to transfer spores from a single lesion to a small number (6-12) of seedlings. First prepare the plants to be inoculated by rubbing them lightly between moistened fingers to remove the wax bloom. Then use a smooth spatula to remove spores from the uredinium and place them directly on the plant or in a drop of water on a microscope slide. If employing the latter option, gently wipe the water drop containing the urediniospores from the slide to the seedling (48). Cotton, sponges, swabs, or pads may be used but many spores will fail to be transferred.

Advantages

- Easy and inexpensive.

Disadvantages

- Water is a poor spore carrier.
- Inoculum density is erratic.

Disease Scoring

The standard scoring systems for the rust diseases are summarized in Tables 21 and 22. These scoring systems vary to a certain extent for each investigator. Roelfs (298) proposed that resistance can also be measured in four ways that are not entirely independent:

- The number of uredinia per unit of inoculum. This is expressed as receptivity of the host or infectibility of the pathogen. If the reduction in the size of the uredinium is complete, it is called immunity and is also scored as a low infection type.
- The size of the uredinia produced. This is reflected in a lower infection type and a more resistant host response.

Table 21. Host response and infection type descriptions used in wheat stem and leaf rust systems.

Host response (class)	Infection type[a]	Disease symptoms
Immune	0	No uredinia or other macroscopic sign of infection
Nearly immune	;	No uredinia, but hypersensitive necrotic or chlorotic flecks present
Very resistant	1	Small uredinia surrounded by necrosis
Moderately resistant	2	Small to medium uredinia often surrounded by chlorosis or necrosis; green island may be surrounded by chlorotic or necrotic border
Heterogeneous	X	Random distribution of variable-sized uredinia on single leaf
Heterogeneous	Y	Ordered distribution of variable-sized uredinia, with larger uredinia at leaf tip
Heterogeneous	Z	Ordered distribution of variable-sized uredinia, with larger uredinia at leaf base
Moderately susceptible	3	Medium-sized uredinia that may be associated with chlorosis
Susceptible	4	Large uredinia without chlorosis

[a] The infection types are often refined by modifying characters as follows: =, uredinia at lower size limit for the infection type; -, uredinia somewhat smaller than normal for the infection type; +, uredinia somewhat larger than normal for the infection type; ++, uredinia at the upper size limit for the infection type; C, more chlorosis than normal for the infection type; and N, more necrosis than normal for the infection type. Discrete infection types on a single leaf when infected with a single biotype are separated by a comma (e.g., 4,; or 2=, 2+ or 1,3C). A range of variation between infection types is recorded by indicating the range, with the most prevalent infection type listed first (e.g., 23 or ;1C or 31N); after Roelfs (297).

Table 22. Host response and infection type descriptions used in the wheat stripe rust system.

	Infection Type[a]		
Host response (class)	McNeal	Gassner[b]	Disease symptoms
Immune	0	i	No visible infection
Very resistant	1	00	Necrotic/chlorotic flecks, without sporulation
Resistant	2	0	Necrotic/chlorotic stripes, without sporulation
Moderately resistant	3	I	Trace sporulation, necrotic/chlorotic stripes
Light moderate	4	I	Light sporulation, necrotic/chlorotic stripes
Moderate	5		Intermediate sporulation, necrotic/chlorotic stripes
High moderate	6	II	Moderate sporulation, necrotic/chlorotic stripes
Moderate susceptible	7	II	Abundant sporulation, necrotic/chlorotic stripes
Susceptible	8	III	Abundant sporulation, with chlorosis
Very susceptible	9	IV	Abundant sporulation, without chlorosis

[a] McNeal = McNeal et al. (248), Gassner = Gassner and Straib (112).
[b] This scale used for descriptions of seedling infection types only.

- Resistance is also expressed as length of latent period (period from inoculation to 50% of uredinia eruption). This may result in a lower infection type if the notes are taken on a given day after inoculation.
- The length of time a uredinium sporulates. Probably not a major factor of resistance, this is reflected by lesions associated with chlorosis and necrosis. In respect to yield, chlorosis or necrosis may do as much or more damage to the plant than an unrestricted uredinium (285), however in terms of epidemic development, it may reduce the rate of disease spread. Early telia formation due to host resistance also reduces the length of the sporulation period.

Seedling studies

Seedling host responses are normally scored as susceptible or resistant depending on the infection type produced with a designated isolate in a particular environment. The infection type produced will often change if the environment is altered.

Infection type. For leaf and stem rusts, a relatively uniform set of infection type symbols has been developed over the years (Table 21). Stripe rust workers use the scales in Table 22. Selected seedling infection types for the three diseases are shown in the color photos on page 40.

Infection types for a particular host-pathogen interaction are modified by environmental conditions, host age, host nutrition, host tissue, inoculum density, and time. Thus, for scoring infection type, standard conditions must be developed and used. Known gene-carrying checks should

also be included. An international standard would be useful, but, because of the different isolates and hosts used, it is not practical. For example, *Sr6* should be studied below 20°C, while *Sr13* is more effectively studied at 30°C. In published studies, the conditions under which the infection type was observed should be given.

Infection types are relatively easy to score, however, all useful resistances are not expressed in the seedling leaf. In addition, low receptivity (reduced number of infections) and latent period duration are not measured by infection type scoring. No exact relationship exists between infection type and usefulness of the resistance in a breeding program. Most believe that infection types 3 and 4 (8 and 9 for stripe rust) are too susceptible to use. In areas favorable for rust development, infection types 2, X, Y, and Z (5 to 7 for stripe rust) may provide inadequate levels of resistance.

Latent period. Studies of latent period have been used as a measure of resistance. These studies require precise inoculum densities and environmental control during the incubation period. The scoring involves the number of days (hours would be more appropriate if notes were taken that often) from the time of inoculation until 50% of the uredinia have erupted. Because the total number of uredinia to erupt must be known to determine when 50% had erupted, these studies require many hours of counting lesions. The period from inoculation to sporulation must be identical for direct comparison of results of different experiments. Use the same isolate in the study because isolates also influence latent period. Therefore, before making generalizations about latent periods, evaluate a number of isolates. Inoculum or infection density also affects latent period. Areas of tissue with many infections generally have a shorter latent period than areas with fewer infections.

When evaluating the latent period for breeding purposes, use a check cultivar of a known acceptable latent period as a standard. The 50% uredinia eruption period for the check cultivar must be determined prior to the test. Take notes when 50% of the uredinia are expected to have erupted on the check cultivar. Discard all test lines with more uredinia than the check (short latent period) and save those with the same number or fewer uredinia than the check (long latent period).

Caution: Race-specific resistance and low receptivity will cause lines to appear to have long latent periods in this test.

Receptivity. Receptivity is the measure of the number of lesions produced with a standard amount of inoculum in a defined environment for a specific host-pathogen interaction. Environmental controls are critical. Use a single isolate in testing host material. The isolate should be tested under a range of environmental conditions and compared with other isolates before making generalizations. An example of low receptivity resistance is that conditioned by *Sr36* (317).

Caution: Receptivity is affected by a number of environmental conditions as well as host growth stage and plant parts infected. Inoculum density, spore viability, and environmental conditions must be controlled.

When selecting for low receptivity of seedlings in a breeding program, make a comparison with a cultivar of acceptable low receptivity as a check for variation between tests. Uniformly inoculate the test material and check with the selected isolate. When the lesions on the check are fully developed, lines with fewer lesions than the low receptivity check are retained. Low receptivity is sensitive to environmental factors.

Adult plant studies

Evaluation of adult plant resistance is commonly done in the field where notes are taken on the disease intensity at the end of the season. Two types of scoring are normally combined:

- The modified Cobb scale (280) is used to determine the percentage of possible tissue (100%) rusted (Figure 11). Only about one third of the actual tissue can be affected by the disease. Other keys have been developed for scoring the percent of severity (157), but they have not been widely used for the rusts.
- The host response to infection in the field is scored using 'R' to indicate resistance or miniature uredinia; 'MR' to indicate moderate resistance, expressed as small uredinia; 'MS' to indicate moderately susceptible, expressed as moderate sized uredinia somewhat smaller than the fully compatible type; and 'S' to indicate full susceptibility (Table 21). See the color photos on page 37. Host response to stripe rust is also scored on McNeal's 0-to-9 scale (248, Table 22). Depending on the disease potential in a region, MS responses may be considered too susceptible to use or where the disease potential is much less, MS lines may be retained as useful resistance. Disease severity is affected by the inoculum density (312). A disease score of 5S in one nursery may be as susceptible as the check, while in another nursery, 5S may indicate a line with a long latent period or low receptivity and have useful resistance. 5S may also indicate the presence of a different virulence (race) at a low level.

Caution: When interpreting the results of severity response data, make comparisons with the check cultivars, the growth stages of the test material, and virulences of the pathogen population to designated resistances.

Rust development is closely correlated with host growth stage. Maturity differences of even a few days may expose the plant to a different inoculum density or environment. Low amounts of rust are generally indicated by 'T' (trace). However, pustules/culm can be converted to disease severity for leaf

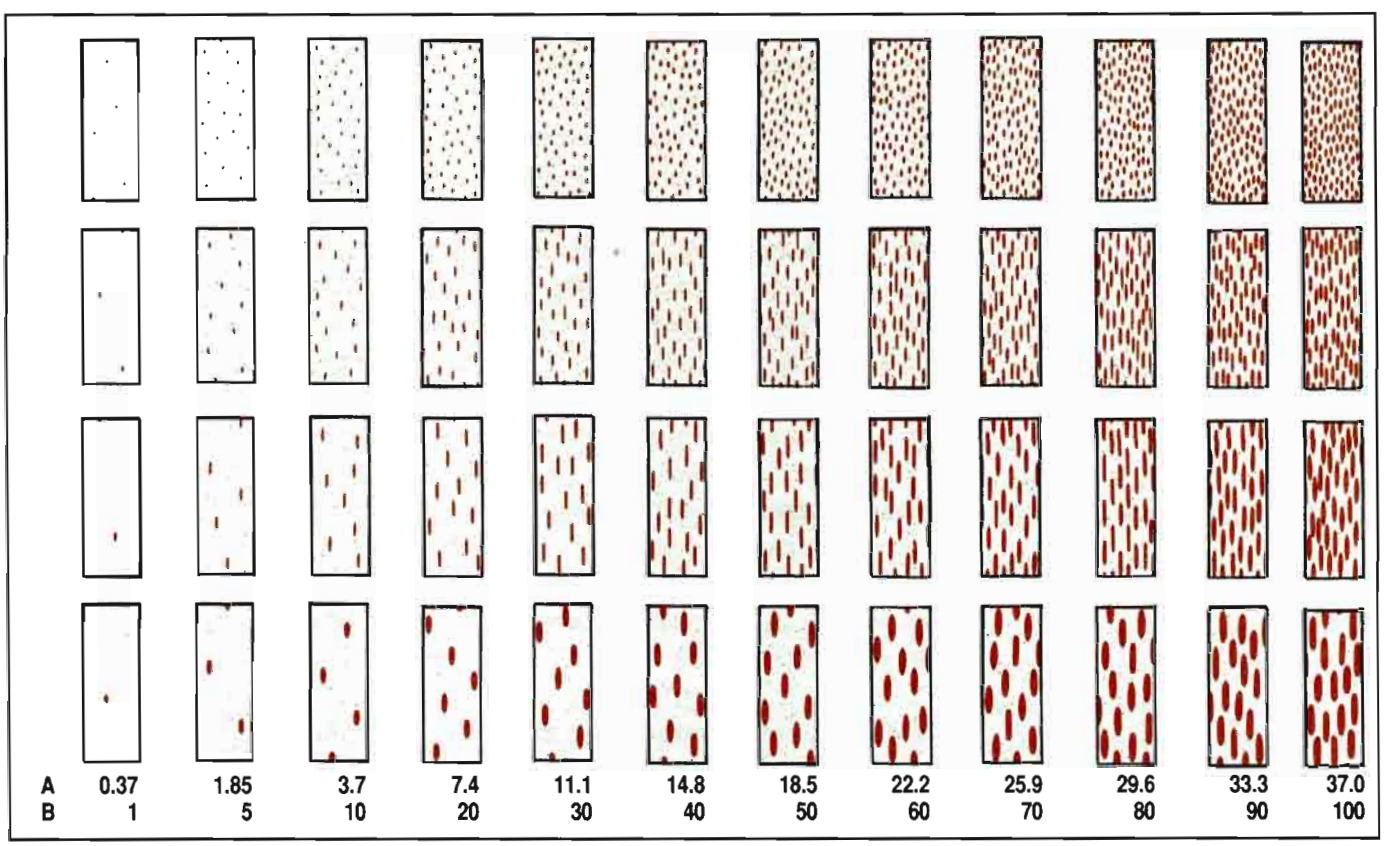


Figure 11. The modified Cobb scale: A, actual percentage occupied by rust uredinia; B, rust severities of the modified Cobb scale after Peterson *et al.* (280).

Notes:

rust where approximately 18 pustules/leaf equal 1% severity (58); and for stem rust where 10 pustules/tiller equal 1% severity (175). Due to the systemic nature of stripe rust, such comparisons are difficult to make.

Factors other than resistance or susceptibility of the host and virulence and avirulence of the pathogen affect severity and host response readings. Environment affects the pathogen, host, and their interactions. Inoculum density affects the expression of resistance in some cultivars. The response and severity can be influenced by adjacent lines (312). It is often difficult to tell if small pustules are due to resistance, crowding, or recent infections that have not reached their maximum size. In spite of these problems, field evaluation has been the mainstay of wheat breeding programs.

When making comparisons between hosts, there are hazards in assuming that each pathogen population and environment affect the host identically. For example, at one location a cultivar with *Sr15* may be resistant because the isolates are avirulent on *Sr15*; at another location, the same isolate may result in a susceptible response on *Sr15* due to higher temperatures. In another year, the same pathogen population may respond differently due to temperature changes. Lines with resistance such as *Sr36* or *Sr2* may be resistant (trace R) if their neighbors are resistant, but susceptible (trace S-10S) if their neighbors are susceptible—with the same environment and pathogen populations. However, another 10S response in the same nursery may be due to the virulence of a small portion of the population.

Disease severity and host response data are often combined into a single value called the coefficient of infection (C.I.). The C.I. is calculated by multiplying the severity times a constant for host response: where immune = 0.0, R = 0.2, MR = 0.4, MS = 0.8, and S = 1.0. For example, the disease score 60S becomes 60 (60 x 1.0) and the score 10MR becomes 4 (10 x 0.4). This makes it easy to rank or compare between nurseries. The adding of two separate factors into a single coefficient can result in nearly equal coefficients but from different disease scores. For example, a C.I. of 32 can result from many small uredinia (80MR), while a moderate severity of compatible uredinia (30S) has a C.I. of 30. Generally, low CIs reflect low disease severities.

Scoring of rust diseases can be on a designated leaf or the whole plant. The method employed depends on the objective of the experiment and to some extent on the researcher. Often, leaf rust is scored on a single observation using the flag leaf. Disease on the flag leaf is generally a reflection of earlier pathogen development and yield loss is most closely related to severity there (339). Stem rust is scored on the stem leaf sheath and true stem. Severity on the stem is closely related to yield loss. Notes on stripe rust can be taken on the entire plant, flag leaf only, and, in some cases, on spike infection. Spike infection can have a significant effect on yield.

Multiple readings are useful in detecting certain types of resistances. With multiple severity readings, the area under the disease progress curve (AUDPC, Figure 12) has been calculated as a measure of slow-rusting resistance (398). Johnson and Wilcoxson (158) have calculated a series of AUDPC tables for selected frequencies of notetaking and severities. The curved disease progress line is often replaced by a least squares regression line ($y = a + \hat{b}x$), and severity is usually transformed to logs or logits.

Caution: The AUDPC is cumulative, i.e., uredinia present early in the season will affect AUDPC throughout the season. The last few days of the epidemic often add most of the area to the AUDPC. This effect is critical when comparing cultivars or lines that differ in maturity. Each unit of the AUDPC is not equally related to yield.

Factors that affect the disease (i.e., inoculum density, host growth stage, environment, and pathogen isolate) also affect the disease progress curve. The $\hat{b}$ (slope) value from the linear regression equation, $Y = a + \hat{b}x$, also has been used to estimate resistance—this is Vanderplank's 'r'. Some resistances are less effective at senescence—this permits a rapid increase in severity due to inoculum levels in nurseries and gives high $\hat{b}$ and r values. The rate of disease increase might be most useful where disease is being measured in large plots (hectares) or where the inoculum density is uniform across the area. Parlevliet (274) has reviewed the use of rate of disease increase. Other workers have concluded that it is not the most useful measurement for cereal rust studies (288, 289, 340).

Testing for Resistance

Seedlings

Some resistances are expressed as a low infection type and others are expressed as a longer latent period. Some resistances result in fewer infections when inoculated with a specific isolate. Each different resistance studied may require specifically designed experiments.

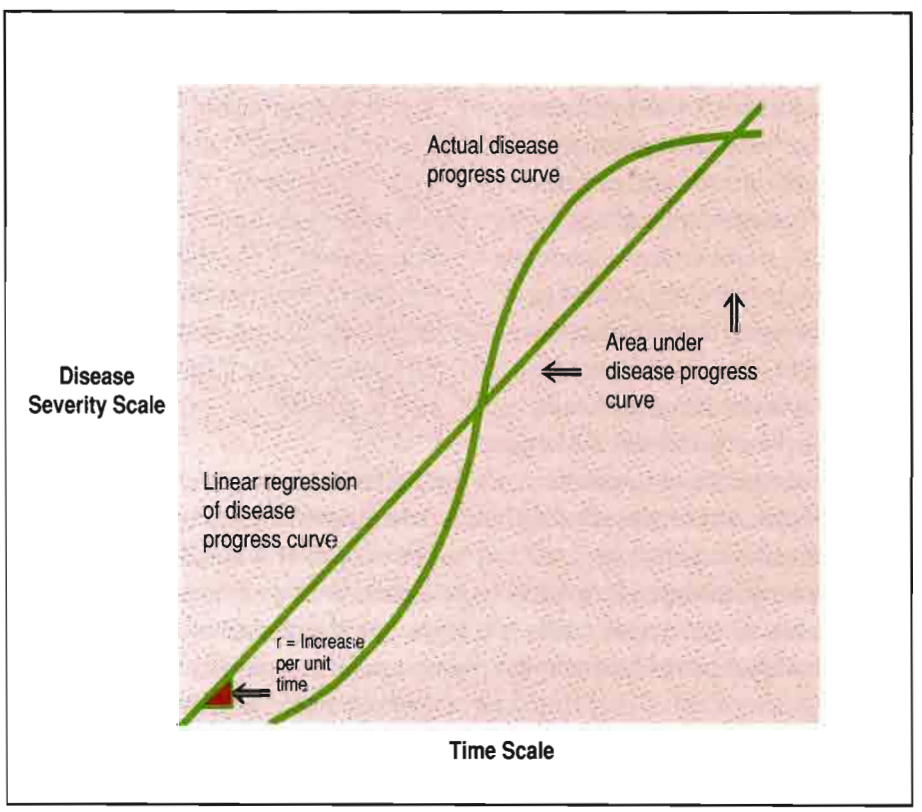


Figure 12. Area under the disease progress curve as a measure of slow-rusting.

Specific type resistance. Tests for specific type resistance are normally done on the primary leaf in the greenhouse. Use a single isolate in each test and include as checks a susceptible line and a selected series of lines with designated genes for resistance. The host population per line is generally 8 to 10 plants in advanced generations and entire populations can be used in the F_2. Inoculate plants about 7 days after planting and take notes 10 to 14 days later. Following a seedling test, the plants with desired resistance can be transplanted.

A modification of this method involves inoculating with a composite of many races. This is useful, but often results in mixed infection types. It is difficult to distinguish between the mesothetic response and the action of several resistance genes in a line to several isolates, or a single resistance gene to several races in the composite isolate. A further modification is to use an inoculum bulk that includes races each with a different urediniospore color. A major problem, however, is obtaining uniform infection with all races and finding color mutants with the desired combination of factors for virulence and avirulence.

A single leaf can be inoculated by two or more isolates simultaneously by placing the spores at intervals with a cotton swab or other device (49). Problems are often experienced with this technique in controlling inoculum density and identifying immune responses versus escapes. Placement of races is critical in host-pathogen interactions where a Y or Z infection type is common—as with the wheat leaf rust system. In cases where there are no differences in resistance

between the primary leaf and the next few leaves, inoculate each succeeding leaf a few days later. This requires care in handling plants so the first infections develop normally and inoculum from the second and subsequent inoculations do not affect the leaves inoculated previously. If only two races are to be used on the same plant, inoculate with the first race and after taking notes on the primary leaf, cut the plant off just above the soil (2 to 3 cm), wait for the emerging tissues, and then inoculate with the second race.

Caution: These techniques require precise timing and are recommended only for specific experiments.

Nonspecific type resistance. A wide range of experiments for selecting nonspecific resistance has been done on seedlings. Most latent period studies have been done on seedlings. Usually six to eight plants with four replicates are inoculated with a single isolate (389). Wilcoxson et al. (398) were able to measure slow-rusting on detached leaves, which was correlated with the field response of selected cultivars. These techniques require precise environment and inoculum controls. Plants requiring evaluation in a breeding population typically number in the hundreds and adequate numbers of check plants (1 for every 4 to 10 test plants) of known latent period are essential. If nonspecific resistance is multigenic in inheritance, then tests must be designed to detect small differences. Evidence indicates that the expression of nonspecific resistance often differs with host growth stage. Even if the resistance is thought to be nonspecific, it is a good procedure to repeat the experiments with several isolates. It will normally be necessary to do statistical analysis on the data.

Receptivity. Receptivity studies on seedlings may not correlate well with adult plant receptivity (317). In the case of *Sr36*, some of the causes for low receptivity are known. For example, lowered receptivity occurs if the first cells that the fungus attempts to penetrate collapse—resulting in the death of the fungus. If just one of the first cells penetrated survives to nourish the pathogen, the fungus will grow and survive the collapse of some cells infected later—resulting in a longer latent period, but with full sized uredinia (11, 316, 317). Similar studies are needed on more host and pathogen genotypes. Receptivity studies require precise inoculum densities and environmental control; checks are essential. The data are as difficult to obtain and analyze as with latent period studies. Host growth stage and environmental conditions are often critical for expression of resistance.

Advantages of seedling tests

- Permit the tracking of specific resistances.
- Fast and relatively inexpensive if greenhouse space is available.
- Specific host pathogen and environmental interactions can be studied.

Disadvantages of seedling tests

- Adult plant receptivity and latent period resistances usually cannot be predicted.
- Resistance observed in seedling tests may be ineffective under field conditions or with other rust isolates.

Adult plants

Much less work has been done on adult plant resistance because of the difficulties involved. Adult plants in the greenhouse require more space. Use of adult plants in the field eliminates the control of environment and contamination influences. Most adult plant studies involve inoculation at a specific growth stage using a single race or isolate. Usually several plants are inoculated with a

predetermined inoculum density. Older plants may require higher inoculum levels. The incubation period may increase for plants in the latter stages of development, i.e., boot through milk stages.
Environmental factors will not only affect plant growth but rust development and the resistance response. Consequently, adult plant studies should be replicated. Susceptible and resistant checks should be included. In the field, include checks every 10 to 20 rows. Depending upon the purpose of the study, notes can be taken for latent period, % infection, size of uredinia, infection types, etc.

Advantages of greenhouse inoculation

- Better control of the environment.
- Pathogen genotype and inoculum density can be controlled.
- Specific host growth stages can be selected for evaluation.

Disadvantages of greenhouse inoculation

- Special equipment is needed for uniform inoculation and incubation conditions.
- Large numbers of plants must be maintained free of insects and other diseases for several months.
- Does not measure the effect of resistance on multi-infection cycles.
- Ignores resistance in variable environments.

For resistances affecting latent period and receptivity, use the same procedures as for seedling tests. Plants must be maintained disease- and pest-free. Provide the plants with adequate light and nutrition for normal growth. Always use a wide range of isolates to minimize the probability of selecting race-specific resistances.

Caution: Select insecticides and fungicides carefully because many have effects on subsequent rust infections.

Rust tests in the field should be conducted using the recommended agronomic practices for cultivation and fertilization. Irrigation is not essential, but in some areas of the world it is the only way to assure adequate moisture and dew formation. Sprinkler irrigation is not satisfactory because it tends to cause plant lodging and the water drops dislodge and wash spores to the ground. A lush plant stand normally has a more severe disease development, so we endorse using the upper recommended levels of nitrogen fertilizer. Depending on which rust is being studied, it may be desirable to plant earlier or later than the recommended period to ensure more favorable conditions for disease development. Stem rust normally is more severe on late-planted material, while spike infection of stripe rust is more common in early-seeded plots. An off-season (summer) or a high elevation nursery can ensure severe disease development, based on the organism(s) under test and local environmental conditions.

An example of a disease screening nursery lay out is to plant test material between susceptible spreader rows. Sow 1- or 2-m long rows per test cultivar or line. Place susceptible lines every 20 rows to serve as checks and to assist in ensuring adequate inoculum development. Incorporate additional checks—parents of the crosses—after every 100 progeny rows and an occasional specific check to indicate an acceptable level of resistance or presence of a particular virulence(s). Inoculate the nursery as early as possible in terms of host development to ensure time for the epidemic to develop and spread. All virulence combinations (even those present in low frequencies) need to be used in the inoculum if resistance is to be incorporated. Take disease notes (severity response) at least once near the end of each disease season. If only a single set of notes is to be taken, the best time is from early to mid-dough. Such nurseries are the easiest and cheapest to operate and have permitted successful breeding for rust resistance for many years.

If a natural epidemic is occurring outside the nursery, it may make up a larger portion of the pathogen population in the nursery than the isolate included in the inoculation. Under severe disease epidemic conditions, minor differences in disease resistance can be obscured and rejected. Some authorities feel that selection under such conditions eliminates important sources of resistance. It should be noted that many breeding programs have produced a succession of highly resistant cultivars under intense disease pressure. A successful nursery has a severe epidemic annually with pathogen types representing the range of virulence combinations existing in the epidemiologic area. If the cultivar is to be used outside the area, evaluation for rust resistance in that area is essential. To make sure all important virulence combinations were successfully established in the nursery, samples should be collected from the susceptible checks and lines used for detecting specific virulences. The pathotypes of the samples should then be determined. If variations in resistance are small, separate each test row with a buffer row. This may vary from a susceptible buffer—if high inoculum density is desired—to a resistant buffer—if low inoculum levels are desired.

Caution: Resistant lines may have only a single gene. The epidemic may be dominated by one race.

Low receptivity. Rowell and McVey (320) evaluated low receptivity for a series of lines susceptible to the isolates used. At the specific host growth stage, they inoculated a pure isolate at a uniform rate on three consecutive nights. This procedure ensured a heavy infection of stem rust on at least one night in this environment. The area had no endogenous inoculum present. Exogenous inoculum generally arrived later in the season. They used two races, but more can be used. Host lines were planted in a long row with 0.3 m between lines within the row. Every tenth line was a susceptible check. To obtain a uniform inoculum density, the spores were placed in an oil carrier and applied with a backpack mist blower. An equal amount of inoculum per meter of row was applied by walking beside each row and directly spraying the test material. A stop watch was used to assure a constant speed of travel along each of the test rows. Fourteen days after the last inoculation, notes were taken on disease severity for those infections resulting from the 3-day inoculation period.

Latent period. Adult plants in the greenhouse or growth chamber are generally used for latent period testing because field experimental techniques usually cannot separate the various components of resistance (274). The method described by Rowell and McVey (320) for receptivity can also work in the field for latent period studies. When the long latent-period check reaches 50% of the expected sporulation, the test lines with a lower uredinia number or disease severity than the check are saved. Generally, there is no way to evaluate latent periods if the lines also have other effective resistances.

Slow-rusting. Slow-rusting in the broadest sense is currently considered as a reduction in the severity of an epidemic on one cultivar compared with another. Although slow-rusting has been assumed to be polygenic in inheritance, race-nonspecific, and durable, none of these are necessarily true. Small plots, even hill plots, have been used to evaluate slow-rusting, but this normally results in the selection of resistance genes that have a major effect. Therefore, it is recommended that slow rusting be evaluated in large plots (3 x 5 m), so that the epidemic develops more normally with an inoculum density that approaches the level of a farmer's field. Slow-rusting may be the result of:

- Fewer uredinia.
- Smaller uredinia.
- Longer latent periods.
- Resistances that function only at certain growth stages.
- Any environment-resistance interaction.

Often a reduction in terminal disease severity has been used in selecting for slow rusting resistance. A better measurement probably is AUDPC or the number of spores trapped above the canopy. Slow-rusting is often a useful resistance. Assumptions about its genetic nature, nonspecificity, and durability should not be made without careful study of the host-pathogen/environment interactions.

Results of slow-rusting testing depend on the host and isolates tested. For example with stem rust, Marquis is a slow ruster compared to Morocco and a fast ruster compared to Lee. All three are fast rusters compared to Thatcher.

Caution: Environmental effects on the pathogen and host resistance can result in slow-rusting. Slow-rusting is a relative measurement against a specific check and can also be due to a nonaggressive pathogen.

Epidemiology Studies

This section provides basic information on: 1) determining the favorability of the environment for a rust disease, 2) determining the source of inoculum, and 3) annual monitoring of the disease's progress.

Favorability of environment for an epidemic

Where long-term records have been kept, the favorability of an area for epidemic development is most likely available in the literature (290). In all cases, the literature is a good first source of information. However, changing cultivars and cultural practices can change greatly the area and degree of epidemic risk in a country over a period of time.

If local cultivars are resistant, a set of experiments to determine the effect of environment on rust development can be conducted. Plant a series of isolated plots of a susceptible cultivar at times that approximate the range of commercial seeding dates and inoculate the plots by syringe to ensure disease establishment. Terminal severity will be a measure of the disease potential under existing environmental conditions. The plots should be at least 2 x 2 m to permit near normal disease development. The host cultivar should be susceptible—avoid cultivars that may have low receptivity, long latent period, or that are unadapted. Select a pathogen isolate that is locally adapted and unique.

High severities at harvest do not always indicate a severe yield loss. To determine yield loss, maintain a disease-free plot and compare grain yields and 1000-kernel weights. Use at least 1 m^2 from near the center of each plot. If it is impossible to maintain a rust-free check, use Table 23 (176) and Table 24 (67) to obtain a rough estimate of the percent yield loss for stem rust and leaf rust, respectively. For stripe rust, use the equation of Doling and Doodson (81) (see the section on yield loss studies, p. 56).

After a number of years, the frequency of damaging disease development can be determined in early, optimum, and late plantings when inoculum is present. The acceptable level of yield loss is left to the investigator. Accuracy of yield loss estimations is poor for losses of less than 10%. The information on frequency of disease occurrence and loss can be adjusted for the amount of commercial wheat planted during each period. A risk level is then available for each susceptible

Table 23. Relation between wheat stem rust severity, wheat growth stage, and percentage yield loss (176).

Percent disease severity[a]						
Boot[b]	Flower	Milk	Early dough	Late dough	Ripe	Percent yield loss
-	-	-	-	trace	5	0.0
-	-	-	trace	5	10	0.5
-	-	trace	5	10	25	5
-	trace	5	10	25	40	15
trace	5	10	25	40	65	50
5	10	25	40	65	100	75
10	25	40	65	100	100	100

[a] Modified Cobb scale (280).
[b] Growth stage, see Figure 13.

Table 24. Relation between wheat leaf rust severity, wheat growth stage, and percentage yield loss (67).

Percent disease severity[a]						
Pre-tillering[b]	Jointing	Boot to heading	Flowering	Milk	Early dough	Percent yield loss
-	-	trace	10	25	40	1
-	trace	10	25	40	65	3
trace	10	25	40	65	100	10
10	25	40	65	100	100	20
25	40	65	100	100	100	35
40	65	100	100	100	100	50
65	100	100	100	100	100	70
100	100	100	100	100	100	95

[a] Modified Cobb scale (280).
[b] Growth stage, see Figure 13.

Notes:

cultivar and planting date—if inoculum is present. Resistant cultivars have no risk. If resistant cultivars become susceptible to new pathogen virulence combinations, the risk changes. Perhaps over time, a relationship between each disease and rainfall or weather patterns (261) can be developed.

Sources of inoculum

Essentially, there are three sources of inoculum: 1) the alternate host (except for stripe rust), 2) exogenous urediniospores, and 3) endogenous urediniospores. It would be advantageous if visual phenotypic differences existed among rust genotypes; however, since none exist, virulence differences are the best and easiest way to distinguish differences among races—this measurement requires a virulence/avirulence (or race) survey.

Alternate host. In general, the importance of the alternate host as an inoculum source has been overestimated. The major importances of aeciospores are their early appearance in the spring, the range of virulence/avirulence combinations present, and the large numbers of spores produced (296). Inoculum from the alternate host is relatively easy to detect due to the great diversity of pathogen phenotypes near the source (126, 306). The number of phenotypes in an alternate host is much like the number of phenotypes in an F_2 host population. Additionally, a disease gradient is visible from the source (alternate host) to the wheat (28). This is particularly true of stem rust where the alternate host is a large perennial plant that grows along the field edges.

Exogenous inoculum. Exogenous inoculum is a major source of inoculum in areas where a host is absent or where the environment during part of the year is too severe for pathogen survival. In general, the ability of the pathogen to withstand adverse environmental conditions has been underestimated. This is demonstrated by the ability of stripe rust to survive hot dry summers in Australia (211), leaf rust to survive the hot summers of Morocco (102), and stem rust to survive the dry summers of Pakistan and Kansas (21, 59) and the cold winters of Wisconsin and North Dakota (277, 308).

Exogenous inoculum results in a characteristic pattern of disease spread that helps in its recognition. The oldest infections are at a standard height on the plant (313). Generally, the initial inoculum is deposited on the newly emerged tissue. The first uredinia appear within the canopy—due to the growth of the plant during the latent period. Secondary infections tend to be at the same height or just above or below the initial uredinia. Generally, the initial infections are randomly distributed over a large area (322)—except when the environment is so marginal for initial infection that it occurs only in certain ecological niches. Still, infections are random within these niches. The random distribution pattern of exogenous inoculum results from the dispersal of the spores throughout the air during transport and their deposition by rain scrubbing (323). The initial infections that are high in the canopy when sporulation occurs result in a rapid horizontal spread of the disease (309).

To prove long-distance transport that occurs with exogenous inoculum, 10 criteria must be studied and shown to be compatible (404):

- Crop phenology in the source area.
- Rust phenology in the source area.
- Weather conditions in the source area.
- Air trajectories from source to target area.
- Spore content of the air between source and target area.

- Spore trapping data in the target area.
- Weather conditions in the target area.
- Crop phenology in the target area.
- Rust phenology in the target area.
- Matching of phenotypes in source and target areas.

It is difficult to prove that sources of inoculum are exogenous. Circumstantial evidence is good in Australia (211), China (326), Egypt (326), India (261), North America (299, 301), and New Zealand (29, 211).

Larger trap plots will be required to detect exogenous inoculum if arrival is low and the environment is less favorable. For detecting stem rust in Minnesota, the plot size is fifteen 1-m rows, five 3-m rows, or six 2.5-m rows. A plot four times larger may not be adequate in Oklahoma. The cultivar used in the trap plot should be susceptible and well adapted to the area. It should be planted early, about the time the first commercial fields are planted, so that adequate foliage is available to trap the first spores. Row spacing of at least 30 cm will permit frequent, careful examination without causing damage to the plants. If the initial infections are missed and the secondary spread is assumed to be the primary infection, the date of long distance transport may be incorrectly estimated by 2 weeks or more. This could result in a misinterpretation of the source of inoculum. Secondary infections often occur in clusters of three or four uredinia within a few centimeters. Such clusters can be a clue that an older infection exists.

When examining trap plots for uredinia, remember that the presence of uredinia represent infections that occurred 2 or more weeks earlier, and the amount of host and pathogen growth that occurred depends on temperature. Search for uredinia on tissues that would have been exposed at that earlier date. The rain-deposited nature of exogenous inoculum makes it possible to concentrate observations at a time equal to one latent period. The duration of the latent period will depend on the rust and the environment following each rain. As little as 2 mm of rain can be effective in removing spores from the air (125).

Spore traps can also be used to detect the arrival of exogenous inoculum, but they present two problems:

- The inability to tell where the trapped spores originated—especially with air sampling spore traps. Often, spores assumed to be transported long distances are probably produced locally.
- The difficulty of identification of the rust species—not to mention the forma specialis.

There seems to be a clear relationship, however, between spores trapped in rain samples and disease development in the northern Great Plains of North America (314) and in central India (261, 263). Shorter movements of exogenous inoculum also occur. The shorter the distance of transport the harder it generally is to distinguish it from endogenous inoculum.

Exogenous inoculum follows the movement of air masses. In the higher latitudes of North America and Europe, the general spread of inoculum is from southwest to northeast during the wheat growing season (301). Individual storms may carry inoculum, at least short distances, in any direction. In other areas of the Northern Hemisphere, the recorded movement of inoculum has been from the west to east and southeasterly (261, 326). In the Southern Hemisphere, the general direction is from west to southeast, but is affected by geographical features and season (211, 326).

Endogenous inoculum. Most epidemics are the result of endogenous inoculum. The presence of low levels of local inoculum is difficult to detect. Zadoks and Bouwman (404) state that in the Netherlands a single overwintering uredinium of stripe rust per hectare is adequate to cause an epidemic.

The characteristic of endogenous inoculum spread is that the oldest infections are generally low in the canopy (within 2-3 cm of the ground). In date of planting studies, the earlier planted plots generally have the most disease and foci. The horizontal and vertical spread of disease is nearly of equal distance until the disease reaches the top of the canopy. Foci are normally found in a nonrandom pattern and different foci are often caused by different pathogen genotypes. The erratic pattern of the foci may be predictable (299). For example, foci may occur only near volunteer wheat plants, on plants protected by snow cover along a tree row (100), near snow fences, or near an accessory host. Generally, the endogenous inoculum present is measured directly by disease occurrence on susceptible plots. Impaction-type spore traps also are useful in detecting endogenous inoculum (305).

Disease control is difficult in areas where endogenous inoculum exists. Generally, the green bridge consists of volunteer plants of the same genotype that are being planted the following year. For control to be

possible, the green bridge of susceptible host plants must be broken. Resistance has been generally of short duration, probably due to the constant host-pathogen association. Fungicides provide inadequate protection due to the high inoculum density and the length of time control is required.

Monitoring disease development

Regardless of the source of the inoculum, it is important to monitor disease development annually. Such surveillance programs or surveys are conducted in the United States by the Cereal Rust Laboratory based in Minnesota for the eastern and central areas and by the Cereal Disease Laboratory based in Washington for the Pacific Northwest. Agriculture Canada does annual surveys in Canada; the Indian Agriculture Research Institute and the Plant Protection Directorate do systematic surveys in India; and surveillance is done during annual traveling wheat seminars in Pakistan. These surveys attempt to determine the extent of the disease, its incidence (% of tillers infected), and severity (modified Cobb scale). Cultivars are evaluated and rust collections are made for virulence determinations. The procedures used vary to fit local situations. The following illustrates how the Cereal Rust Laboratory (300) carries out its survey work.

Because of the large area sown to wheat in the central U.S. (25 million hectares), several trips are required. Each trip is planned to coincide with the heading growth stage of wheat. A preselected route is chosen using all-weather roads that go through the major wheat production regions and areas where rusts have historically been a problem. The first field after 10 km on the car odometer starts the survey and then stops are made every 40 km thereafter. The observer walks into the field beyond the border and then examines approximately 33 m of row for rust. The observer selects places in the field where rust should be favored to make a rust collection. Incidences and severities are recorded for the field as an average. The cultivar, growth stage, crop condition, and other diseases or stresses are noted. Along the survey route, visits are made to experimental and demonstration plots where information is obtained as to whether or not rust is associated with planting dates, certain cultivars, and/or certain cultural practices. Data from experimental plots are not included with the data from farmers' fields. Every 2 weeks, the Laboratory issues a report to inform wheat breeders, pathologists, and industry and extension personnel of the disease situation. Observers collect rust from every field where it is present and from each plot location (generally from susceptible and common commercial cultivars). Collections are also made from lines or cultivars that were previously resistant.

The epidemiology information obtained is used to make decisions on where resistant cultivars are needed and the level of resistance required. Once sources of inoculum are identified, this information can be used to help reduce or eliminate these sources. Examples are the eradication of the barberry in north central North America and the ban on growing spring wheat cultivars in Denmark (148).

Fungicide Evaluation

Fungicides currently available for rust control are too expensive for routine use except in the most productive wheat areas. Environmental concerns and the effectiveness of resistance have generally reduced the need for

fungicides. Occasionally, however, the need arises to evaluate fungicides.

Initial tests can be done *in vitro*. One method is to deposit urediniospores uniformly on a pure polyethylene film at a density of 25 spores/mm^2. Place 2 ml of the test chemical in a pyrex glass dish (2.5-cm diameter x 0.6-cm) and float the film on the surface of the chemical. Incubate the dish in the dark at 18°C for 24 hours and then count the number of germinated spores in five microscope fields (100x). It is standard to use three replicates of the plates at five concentrations of the test compound along with a distilled water check (319).

The final determination of the usefulness of a fungicide is a field test. However, seedling plants can be used to define the chemical's activity as a protectant and an eradicant, its uptake by foliage and roots, activity after soil application, and phytotoxicity. Such tests greatly enhance an optimal performance test in the field.

Seed treatment

A maximum of 400 g of dry material/100 kg wheat seed can be retained. Larger doses of chemical require pelleting of the seed. Place a 10-g lot of seed in a vial with 40 mg of dry material for each dosage. Use Diatomaceous earth for the control and to dilute the chemical to the desired concentration. Shake the vials for 6 minutes using a reciprocal shaker. Some chemical sticks to the vial, so discard the first seed lot per concentration because the vial must be precoated. For liquid formulations, use a pipette to place the aqueous dilution onto the side of the vial and then rotate the vial to spread it evenly before adding the 10-g lot of seed. Potentially useful compounds will have a low dosage response and negligible phytotoxicity.

Rowell (319) recommends the following procedure for evaluating seed treatment fungicides for rust control. Five uniform 7-day-old seedlings are chosen per pot and inoculated uniformly. Ten days later, count the number of uredinia per leaf and note any phytotoxic effects. Re-examine plants for changes after an additional 4 days. Use four replicated pots per dose and evaluate five dosages at tenfold intervals. Generally, a narrow concentration range exists in which the pathogen responds differentially to the dosage. Subsequent tests are selected that most likely fall within the range of 10 to 90% control. Trials often vary ±50% of the mean.

Soil treatment

These tests detect compounds that have systemic activity. Rowell (319) recommends mixing the test chemical with 50 ml of distilled water and adding it to the surface of a 10-cm^3 plastic pot. Avoid leaching by adding increments sufficient to wet the soil without drainage from the pot. Use a dose of 660 mg/pot, which can be extrapolated to 1 kg/ha. Assay the protective activity by applying the test chemical at planting and inoculate 7 days later. Assay eradicant activity by applying the test solution to seedlings 3 days after inoculation and compare with control pots.

Foliar treatment

Use a spray chamber in which pots rotate to provide a uniform spray coverage. Calibrate the process so that the equivalent of 1.3 kg fungicide/ha of mature wheat is evenly distributed on the plant surface without runoff. Evaluate fungicides as eradicants by spraying wheat seedlings 3 days after inoculation.

Field experiments

In field experiments, use spray equipment and the amount of carrier per hectare that will be similar to commercial usage. Spraying should be done uniformly and runoff avoided. Use replicated, randomized small plots arranged in rows separated by wide rust-free borders and alleys to reduce interplot interference and improve the discrimination of small differences between treatments. The optimum timing of applications of a systemic fungicide is governed by the stage of the epidemic and the fungicide's properties. Highly effective protectants, but poor eradicants, are most effectively applied before the rust is present. Highly effective eradicants are best applied at about 10% severity. The nature of the disease also affects optimum timing of systemic fungicides. Leaf rust is easier to control than stem rust because fungicide deposits are greater on the upper surface of the leaf where this pathogen occurs. Rowell (319) is an excellent review of evaluating fungicides for rust control.

Caution: The susceptible check cultivar used in pathology experiments often has high receptivity. This can give an unrepresentative evaluation of a chemical's efficacy. This can be avoided by making the test cultivar(s) the same as the one(s) to be sprayed in the field.

Notes:

Yield Loss Studies

Measurement of yield loss can present a number of problems. Yields may be difficult to measure if experimental plots have cultivars that appear homozygous but are actually heterogeneous. Yield is the expression of the individual plant genotype against the environmental conditions it faces. Agronomists usually recommend at least four-row plots 5 m long, replicated four times, from which only the center two rows are harvested after 0.3 m have been removed from each end. Even so, differences of less than 10% are generally not significant. Under these circumstances, imposing a disease that also affects the host genotype and the environment complicates the situation. An additional complication is that the airborne rust diseases move rapidly from plot to plot. Even resistant plants can be affected by the amount of disease on adjacent plants (333). Susceptible plants may be affected by a lack of inoculum being produced on nearby resistant plants (37). To obtain a relatively uniform inoculum density, Roelfs (294) recommends plot sizes of 10 x 10 m. By taking samples from the center of such a plot, it should be representative of a commercial field. Whether this truly represents areas as large as 1000 ha is questionable. Attempts have also been made to study losses on an individual plant basis (174).

Losses due to rust in areas where wheat is economically grown vary from a trace to no more than 10% (295). This is a level below which most yield loss studies are difficult to measure statistically. To overcome this problem, research has been done on large losses and then smaller losses are estimated using a straight line relationship. Most of these models have been developed for wheat growing in the northern latitudes of the world. How well they apply elsewhere is unknown. Table 23 (176) and Table 24 (67) have been widely used for determining yield loses due to stem rust and leaf rust, respectively.

Greaney (119) found the average loss in spring-sown wheat for stem rust to be 5.4% (range 3.1 to 9.7%) for each 10% of terminal rust severity (modified Cobb scale). A 1% terminal severity is equivalent to a 0.54% loss in yield so a 10% severity is equivalent to a 5.4% loss. Note, this would transcribe a 100% terminal severity as equal to a 54% loss, which is probably an underestimation for severe epidemics. Kingsolver et al. (175) related losses to 100% disease severity at different growth stages (Table 25, Figure 13). Buchenau (53, 54) obtained a relationship between the AUDPC curve and loss caused by leaf and stem rusts on autumn-sown wheat. Buchenau (53) described this relationship in the form of a chart for growers to use (Figure 14). Burleigh et al. (57) were able to predict leaf rust losses using three observations of disease severity. Calpouzos et al. (61) expressed these data in tabular form (Table 26). They also developed a model to predict stem rust losses on spring-sown wheat (Figure 15). The generalized model shown in Figure 15 was developed by

Continued on page 58

Table 25. Loss in yield caused by stem rust associated with a 100% severity at various stages of crop growth; from Kingsolver et al. (175).

Growth stage[a]	Loss from stem rust (%)
Boot	98
Boot-heading	90
Heading-flowering	82
Flowering-milk	80
Milk-dough	73

[a] See Figure 13.

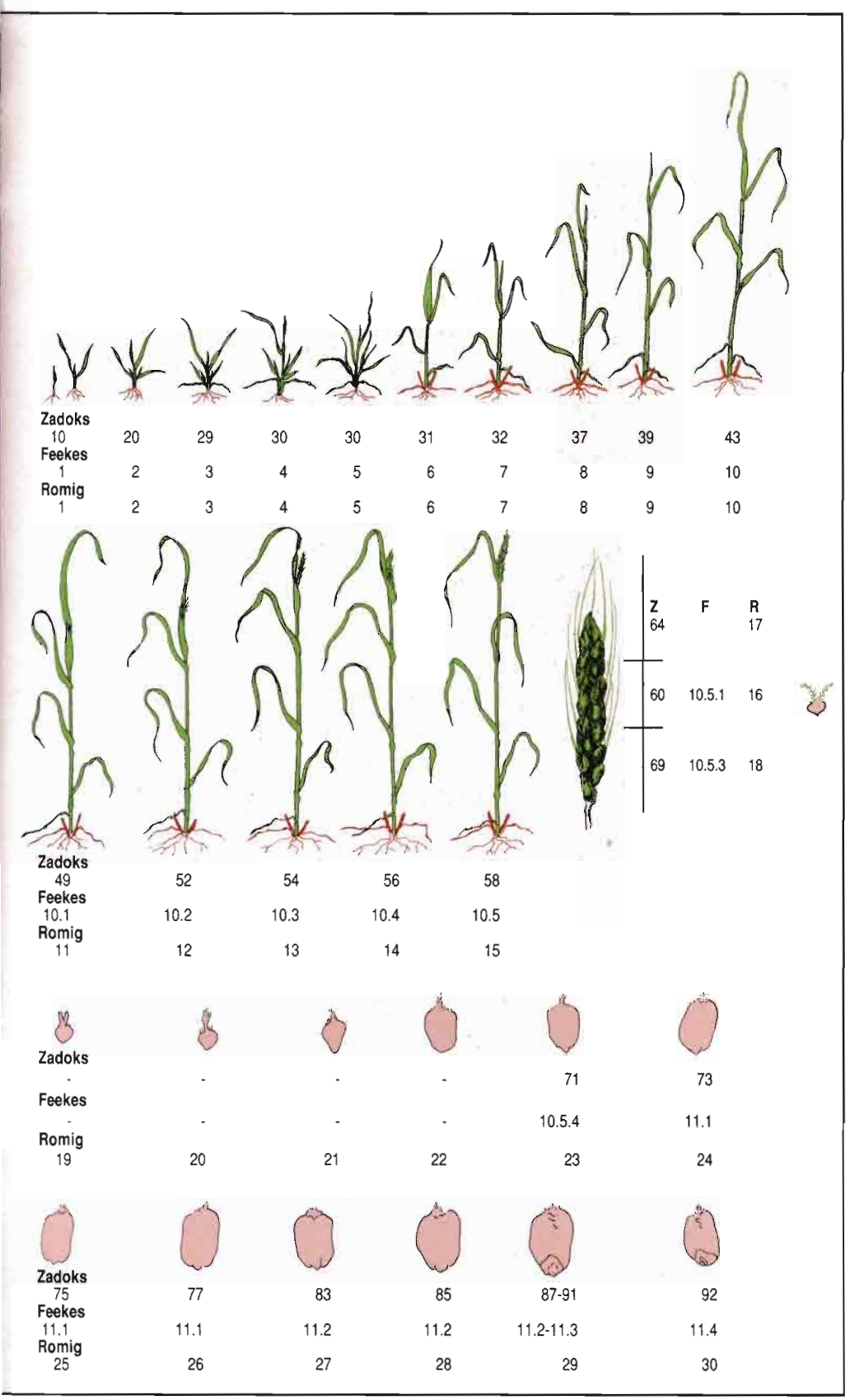
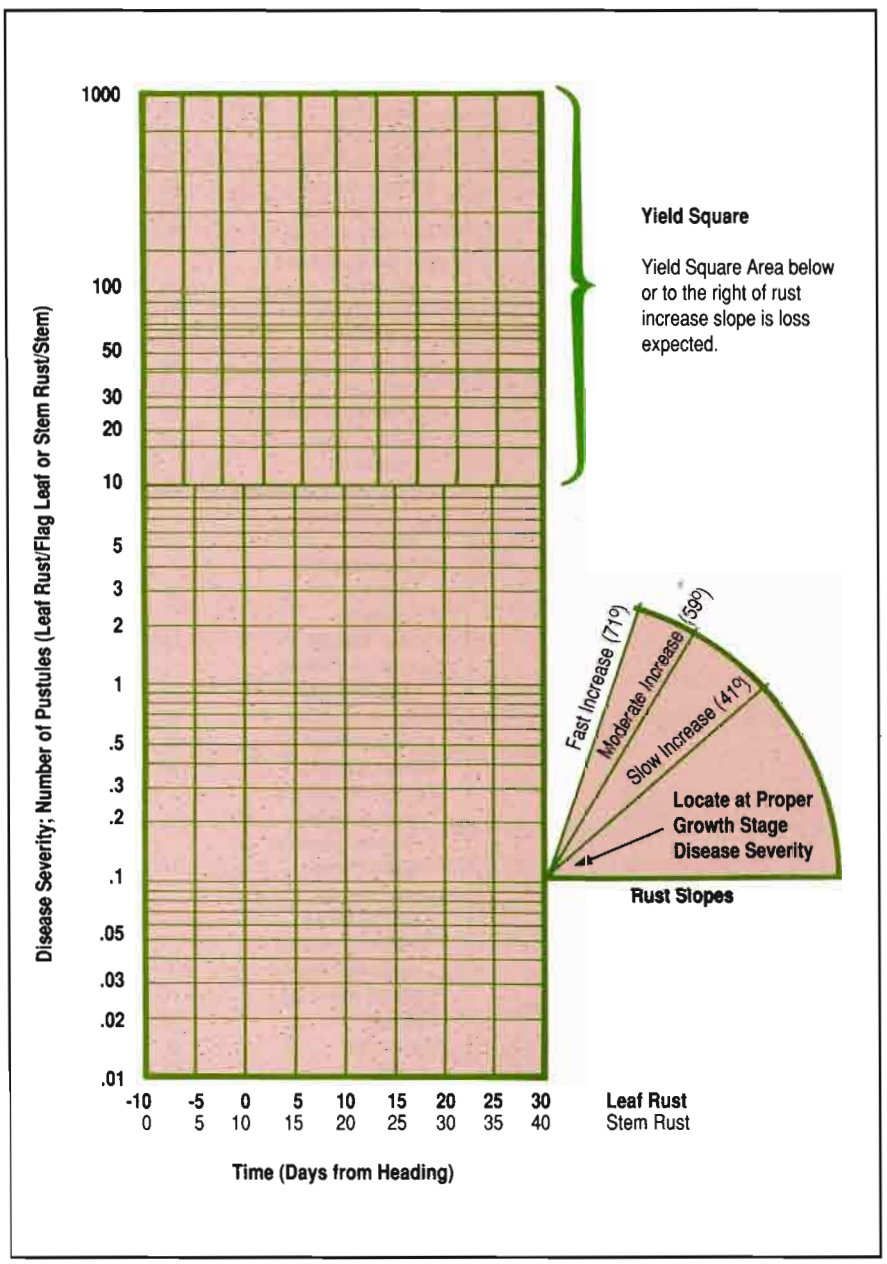


Figure 13. Descriptions of the growth stages for wheat using the Zadoks, Feekes, and Romig Scales.

Description	Growth scale		
	Zadoks	Feekes	Romig
Dry seed	00	—	—
Start of imbibition	01	—	—
Leaf just at coleoptile	09	1	—
First leaf through coleoptile	10	1	1
First leaf unfolded	11	—	1
Two leaves unfolded	12	—	—
One or more leaves unfolded	19	—	—
Main shoot only	20	2	—
Main shoot and 1 tiller	21	3	2
Main shoot and 2 tillers	22	3	2
Main shoot and 9 or more tillers	29	3	3
Pseudo-stem	30	4-5	4-5
1st node detectable	31	6	6
2nd node detectable	32	7	7
6th node detectable	36	—	—
Flag leaf just visible	37	8	8
Flag leal ligule/collar just visible	39	9	9
Flag leaf sheath extending	41	10	—
Boot just visibly swollen	43	10	10
Boot swollen	45	10	10
Flag leaf sheath opening	47	10.1	—
First awns visible	49	10.1	11
1st spikelet of inflorescence just visible	50	10.1	—
1/4 of inflorescence emerged	52	10.2	12
1/2 of inflorescence emerged	54	10.3	13
3/4 of inflores-cence emerged	56	10.4	14
Emergence of inflores-cence completed	58	10.5	15
Beginning of anthesis	60	10.5.1	16
Anthesis half-way	64	—	17
Anthesis complete	69	10.5.3	18
Kernels near middle of head 1/8 formed	—	—	19
Kernels near middle of head 1/4 formed	—	—	20
Kernels near middle of head 1/2 formed	—	—	21
Kernels near middle of head 3/4 formed	—	—	22
Caryopsis watery ripe	71	10.5.4	23
Early milk	73	11.1	24
Medium milk	75	11.1	25
Late milk	77	11.1	26
Early dough	83	11.2	27
Soft dough	85	11.2	28
Hard dough	87	11.2	29
Caryopsis hard, 16% water	91	11.3	29
Caryopsis hard	92	11.4	30

combining the data on five susceptible cultivars in 374 epidemics. The r^2 value of 0.69 indicates that the generalized model may have sufficient predictive power for practical use in various geographical areas where susceptible cultivars are prevalent.

The Calpouzos et al. model can be used to predict yield loss if values for the calculated onset and the rate of epidemic development (slope) have been estimated. These values can be obtained in the following manner. Estimate the onset of the epidemic by making two sequential observations on stem rust severity as the epidemic is increasing linearly, i.e., when the rust severities are between 5 and 95%. Plot these two observations in Figure 16a and draw a line to intercept the observations and the X-axis. Read the onset of the epidemic from the X-axis. Determine the approximate slope of the epidemic by superimposing the information developed in Figure 16a onto Figure 16b. Finally, determine the yield loss by locating the values for epidemic onset and epidemic slope on Figure 16c. The point where the two values meet indicates the yield loss by linear interpolation between the 95 and 5% loss contours.



The multipoint models require more data and are more accurate in experimental plots. With the large variations in environment, host, and perhaps the pathogen in commercial production fields, accuracy may not be as important as most pathologists strive to obtain. Seck et al. (339) found that 26, 12, and 3% of the yield of wheat growing in the greenhouse were contributed by the flag, penultimate, and antepenultimate leaves, respectively. Most models must, consequently, be altered to account for the rust's location on the plant. The relationship between yield and leaf position may well change with the cultivar used.

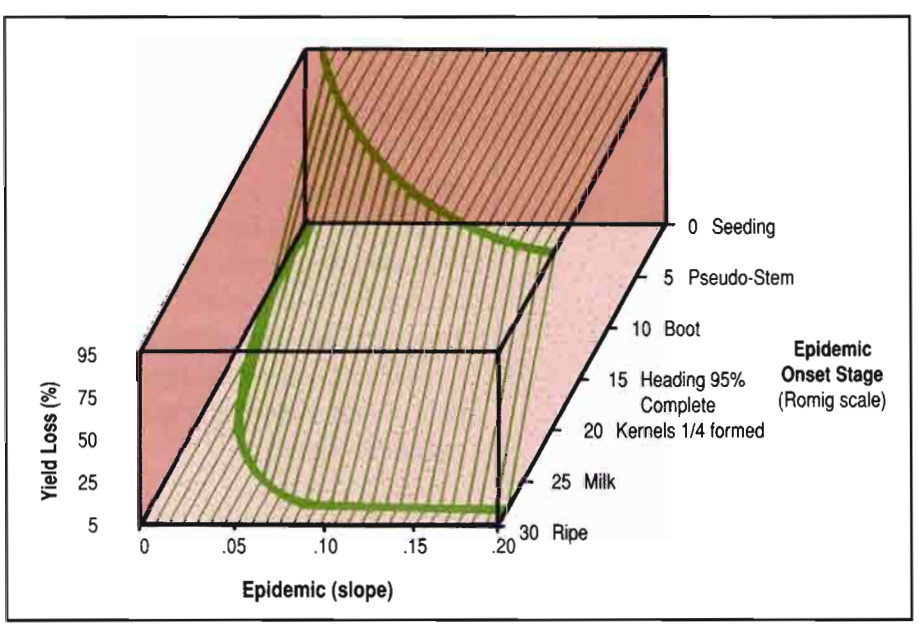
In England, Doling and Doodson (81) predicted losses due to stripe rust using a set of two equations they developed for autumn-sown wheat. They found losses from stripe rust to be equal to 3 times the square root of the disease

Figure 14. Diagram for estimating losses resulting from wheat leaf and stem rusts based on disease severity, stage of crop maturity, and anticipated rate of disease development; from Buchenau (53).

Table 26. For selected leaf rust severities (modified Cobb scale[a]), values of the X_2, X_5, and X_7 terms to be substituted into the yield loss formula of Burleigh et al. (57). Percent yield loss = 5.3783 + 5.5260X_2 - .3308X_5 + .5019X_7.

Leaf rust severity[a]	Uredinia/ leaf	Values for the terms		
		5.5260x_2[b]	.3308X_5[c]	.5019X_7[d]
.001	1.8/100	0	No loss [f]	No loss
.01	1.8/10	0	No loss [f]	No loss
.1	1.8/1	0	No loss [f]	No loss
1	18/1	6	No loss [f]	No loss
2		11	1	1
3		16	1	2
4		22	1	2
5		28	2	2
10		55	3	5
15		83	5	8
20		100% loss [e]	7	10
25		100% loss [e]	3	12
30		100% loss [e]	10	15
35		100% loss [e]	11	18
40		100% loss [e]	13	20
45		100% loss [e]	14	22
50		100% loss [e]	16	25
60		100% loss [e]	20	30
70		100% loss [e]	23	35
80		100% loss [e]	26	40
90		100% loss [e]	30	45
100		100% loss [e]	33	50

[a] Modified Cobb scale, see Figure 11.
[b] Leaf rust severity per culm at boot stage (see Figure 13 for growth stages).
[c] Leaf rust severity per flag leaf at early berry.
[d] Leaf rust severity per flag leaf at early dough.
[e] This level of severity at boot results in a 100% loss.
[f] No losses expected at this severity level.



severity at flowering and reported a linear relationship between yield loss and disease severity. The two equations are:

Loss = (0.268 x disease severity) + 3.9 or
Loss = (3.01 x the square root of disease severity) - 3.6.

For practical purposes, the formula relating the percentage of yield loss to 3 times the square root of disease severity is recommended, except when spike infection is involved. Doling and Doodson expected that spike infection by the disease would cause the yield loss to be underestimated. Mundy (257) found that 3 times the square root of disease severity at flowering underestimated the yield loss. Mundy's data show that:

Loss = (0.442 x disease severity) + 13.18 or
Loss = (4.87 x the square root of disease severity) - 0.13.

However, equations based on a later growth stage are:

Loss = (0.44 x disease severity) + 3.15 or
Loss = (5.06 x the square root of disease severity) - 17.15.

Severe amounts of rust can halt plant growth or even kill the plant by reducing the photosynthetic area and causing losses of nutrients and water. The restriction in photosynthesis results in a weakened root system and shriveled grain. With stem rust, lodging and stem breakage are common with early disease onset (prior to heading).

Figure 15. Yield loss as related to epidemic slope and onset stage of stem rust. The three-dimensional illustration shows the response surface of yield loss due to two parameters for 374 epidemics (61).

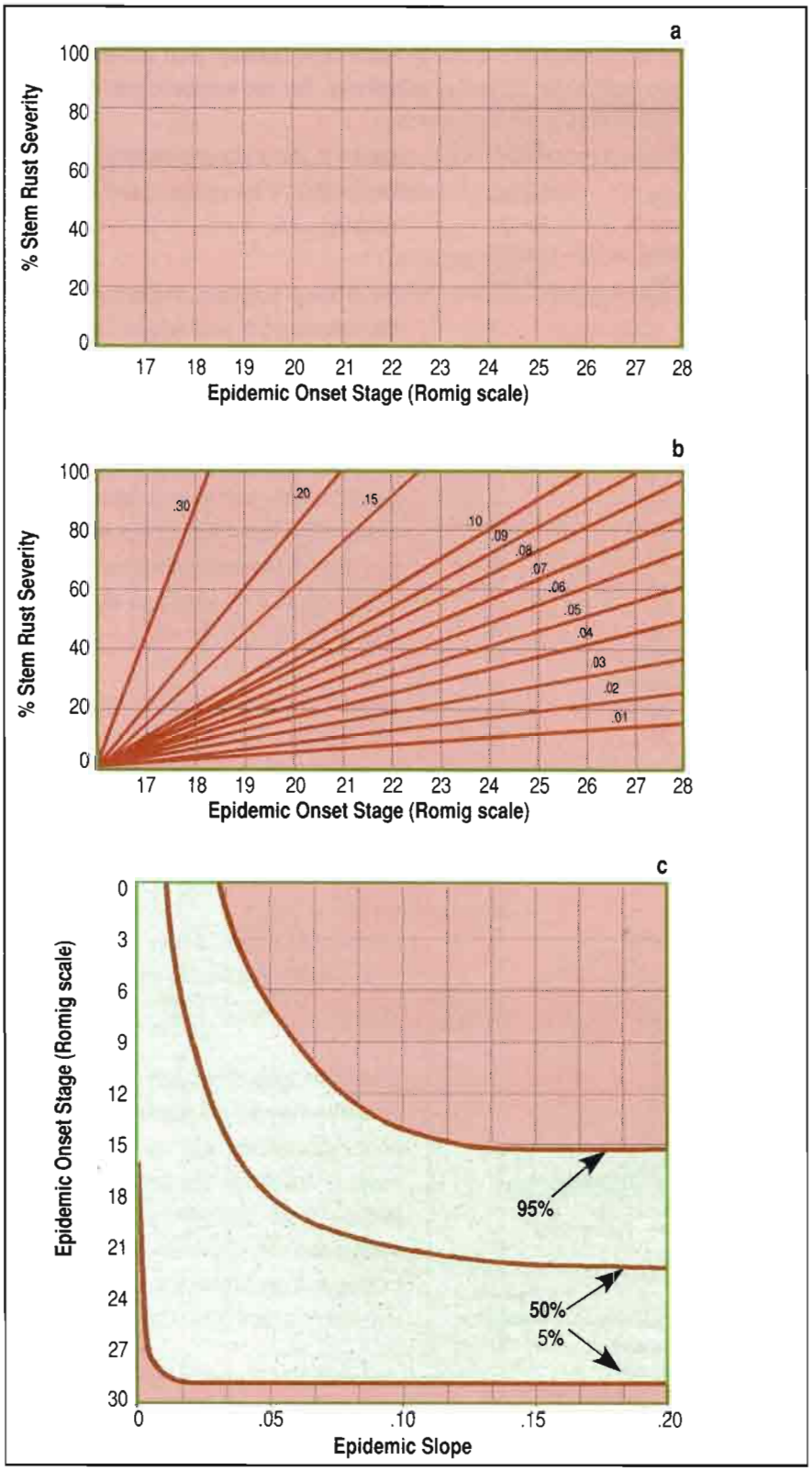


Figure 16. Diagram for estimating yield loss due to stem rust using the generalized model (Figure 15): a) estimation of epidemic onset, b) estimation of epidemic slopes, and c) model to estimate losses from disease onset and epidemic slope.

Losses can be divided into direct losses caused by the disease and indirect losses that occur during harvesting. The direct loss is usually seen in the shriveled grain. A reduction in the number of tillers is less frequent with rust diseases except when the disease becomes severe before jointing. This would be most typical of stripe rust on autumn-sown wheat in areas with mild winters or for leaf rust on either autumn- or spring-sown wheats where initial inoculum is heavy and from a local source.

A reduction in root growth occurs as the disease kills leaves that provide the photosynthate for root development. Cold weather, drought, heat, and other stresses can add to the damage caused by reduced root growth. Losses can also occur in forage production and straw yield, which are economically important in some wheat growing areas. An indirect loss occurs due to an inability, especially with mechanical harvesting equipment, to save the shriveled seed. The ineffective pick-up of grain from lodged plants and stunted tillers is another source of indirect loss. Grain from severely rusted wheat is often of poor quality, which results in a lower market price for the farmer.

Physiologic Race Surveys

Race surveys that monitor virulence changes in the pathogen population have been conducted to aid in the development of wheat cultivars resistant to cereal rusts. However, the long record of changes in the pathogen populations has also resulted in advances in the basic studies of epidemiology and population dynamics in plant pathology. Roelfs (249) recently reviewed race specificity and methods of study.

Wheat leaf rust

Mains and Jackson (223, 224) established the physiologic races in *Puccinia recondita* f.sp. *tritici*. In the latter report, they established a set of 11 differential hosts, i.e., Malakof, Turkey, Norka, Brevit, Webster, Carina, CI 3747, Loros, Mediterranean, Hussar, and Democrat. Turkey and Norka were similar to Malakof in North America and CI 3747 was similar to Webster and only Malakof and Webster were retained.

Waterhouse (391) showed that variation for virulence in *P. recondita* f.sp. *tritici* existed outside that detected by the eight standard differential hosts and was differentiated by the cultivar Thew. Environmental variation caused a great deal of variation in infection types on the differential host cultivars Brevit, Carina, and Hussar (67). To eliminate this variation, these differential hosts were dropped and the races grouped on a unified scheme including evaluation of Malakof, Webster, Loros, Mediterranean, and Democrat (25). The resistance genes currently known to be present in these cultivars are *Lr1* in Malakof, *Lr2*a in Webster, *Lr2c* in Loros, and *Lr3* in Mediterranean and Democrat (52). Johnston and Browder (165) published the last international key in 1966 using these eight differential hosts. Since then the trend has been toward the use of single gene differential hosts developed in Winnipeg by Agriculture Canada.

The original leaf rust differentials had a single effective resistance gene unlike the other two rusts where the differentials had resistance genes in combination. The placing of the *Lr* resistance genes in a uniform genetic background improved consistency in scoring. Most of the important host resistance genes currently being used were not included in the original differential set. Although no standard set of differentials is in use worldwide, Table 27 lists those being used in selected countries. In 1989, the North American leaf rust workers chose to use a set of 12 differentials, which includes: *Lr1*, *2a*, *2c*, *3*, *9*, *16*, *24*, *26*, *3ka*, *11*, *17*, and *30* (207).

Because of the large number of adult plant resistances used in commercial cultivars, any system of evaluating virulence only in the seedling stage is bound to give an incomplete picture of virulence in the pathogen population. Those interested in virulence analysis of *P. recondita* f.sp. *tritici* should review the work of Chester (67), Browder (46, 48), and Samborksi (330).

Table 27. Leaf rust resistance genes commonly used by physiologic race surveys in various countries.

	Lr genes evaluated																				
Country	**1**	**2a**	**2b**	**2c**	**3**	**3ka**	**3bg**	**9**	**10**	**11**	**13**	**14a**	**14b**	**15**	**16**	**17**	**20**	**23**	**24**	**26**	**27+ 31**	**30**
Argentina	X	X	X	X	X	X		X	X				X		X	X			X			
Australia	X	X	X	X	X	X			X	X	X			X	X	X	X	X	X	X	X	
Brazil	X	X	X	X	X	X		X	X				X		X	X			X			
China	X	X	X	X	X	X		X	X			X	X	X	X	X						
Czechoslovakia	X	X	X	X						X												
Hungary	X	X	X	X						X												
India	X	X	X	X				X	X			X		X		X	X	X	X	X		
Iran	X	X	X	X						X												
Italy	X	X	X	X	X	X		X	X	X		X	X		X	X			X			X
Mexico (CIMMYT)	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X
Morocco	X	X	X	X	X	X		X							X	X						
Nepal	X	X	X	X				X	X								X		X	X		
North America (Canada and USA)	X	X		X	X	X		X		X					X	X			X	X		X
Pakistan	X	X	X	X				X	X	X		X				X			X	X		X
Portugal	X	X	X	X	X	X		X	X	X	X	X		X	X	X	X	X	X			
Romania	X	X	X	X	X					X												
Spain	X	X	X	X	X					X												
USSR	X	X	X	X	X			X	X	X		X	X		X							
Yugoslavia	X	X	X	X	X					X												

Wheat stem rust

Stakman and Piemeisal (368) first described races of wheat stem rust in 1917. Stakman and Levine (367) published the first key in 1922 using 12 host cultivars: Little Club (*SrLC*), Marquis (*Sr7b, 18, 19, 20, X*), Reliance (*Sr5, 6, 18, 20*), Kota (*Sr7b, 18, 28, Kt2*), Arnautka (*Sr9d*), Mindum (*Sr9d*), Spelmar (*Sr9d*), Kubanka (*Sr9g*), Acme (*Sr9g*), Einkorn (*Sr21*), Vernal (*Sr9e*), and Khapli (*Sr13, 14*). Stakman et al. (369) published the last key to the standard 12 differential hosts in 1962. Gradually, a shift has occurred to single gene lines possessing known *Sr* genes. Separate systems have evolved in Australia (393), Canada (121), and the United States (298). Roelfs (298) lists the *Sr* genes in each of the cultivars in these sets of differential hosts. In 1988, Roelfs and Martens (310) proposed a new set of international differentials (Table 28).

Table 28. International differential lines and cultivars of known resistance to wheat stem rust and their genes for resistance to *Puccinia graminis* f.sp. *tritici* (310).

Sr gene	Differential line	Winter habit cultivars	Spring habit cultivars
5	ISr5-Ra	Cheyenne	Summit
21	*Triticum monococcum* derivative		Einkorn
9e	Vernstein		Vernal
7b	ISr7b-Ra	Hart	Red Fife
11	ISr11-Ra		Gabo[a]
6	ISr6-Ra		McMurachy
8a	ISr8-Ra	Flavio	Mentana
9g	CSSr9g		Kubanka[b]
36(Tt-1)	W2691SrTt-1	Kenosha	Idaed 59
9b	W2691Sr9b		Gamenya
30	BtSr30Wst		Festiguay
17	Combination VII[c]	Scout 66[d]	Regent[e]

[a] Additional gene for resistance to some North American isolates.
[b] Additional gene(s).
[c] *Sr13* also present, however, the 0;1N low infection type for *Sr17* is epistatic to the 2+ low infection type of *Sr13* at 18°C.
[d] *Sr9d* also present.
[e] *Sr7b* and *9d* also present.

Wheat stripe rust

Hungerford and Owens (153) first reported physiologic races within *P. striiformis* f.sp. *tritici* in 1923, which were confirmed by Gassner and Straib (110, 111) in 1929-30 and Allison and Isenbeck (5) in 1930. Gassner and Straib (112, 113, 114) established a differential set of 12 wheats, 2 barleys, and 1 rye as hosts. Johnson et al. (162) developed the most commonly used nomenclature system in 1972.

Stubbs (372) has reviewed the various difficulties of reproducing results between laboratories. For reproducibility of results, evaluate cultivars under controlled temperatures (18°C day and 11°C night) with at least 10,000 lux for a 16-hour day (162). At lower latitudes, it may be necessary to use less than a 16-hour day to correlate seedling resistance with field experiments. However, for international communication, a standard environment for evaluation of pathogen virulence is necessary.

Stubbs (372) gives a summary (Table 29) of the race patterns based in part on the International Virulence Gene Survey. The most recent surveys have not been published in internationally available journals. Work is concentrated in northwestern Europe, southern USSR (2), USA (198), Peoples Republic of China (196), India (260), Romania (268), and Australia (395).

A yet unaddressed problem worldwide is the specialization of *P. striiformis* on the important adult plant resistances that are so widely used. Zadoks (403) and Priestley and Doodson (285) have studied the specificity of isolates for these adult plants. Priestley and Doodson used polyethylene houses that provided the necessary isolation and some environmental control. A single race based on seedling tests

can be composed of several different virulence combinations for adult plant resistances (384, 403).

Genetic Basis of Resistance

A number of different host genes or their combinations confer resistance to rusts. Table 30 shows the genome locations of these genes for the three rusts. These genes are not expressed if virulence or virulence combinations occur in the rust population evaluated. Furthermore, a rust race can possess virulences to several resistance genes. Hence, it is extremely important to use races of known avirulence/virulence combinations in genetic studies. Genetic studies should be conducted with pure races to avoid confusion in scoring infection types. Genetic studies in the field should use single races if possible.

Gene postulation and genetic and cytogenetic analyses are the commonly used methods for identifying rust resistance genes.

Gene postulation

For gene postulation, the seedlings of the cultivar with unknown genes for resistance along with host lines possessing designated resistance genes are individually tested to pathogen isolates with a wide array of avirulence/virulence genes. To postulate a resistance gene(s), infection types and response array of the test cultivar with the designated genotypes are compared across all isolates (249). A high infection type on a test line indicates that the line does not have any of the resistance genes for which the test isolate is avirulent. A test isolate avirulent on *Sr5* and producing a high infection on the test line(s) would indicate *Sr5* is not in that line(s). If the resistance gene were present, the line would be resistant with a low infection. When test lines have the same low infection type (range) and the same pattern of low and high infection types as a known gene line, then that gene is postulated to be present (see Table 31). The pedigree of the test cultivar can also aid in making a particular postulation if the parents' resistance is known.

Table 32 (349) shows the responses of five wheat cultivars along with some selected control genotypes to seven isolates of *P. recondita* from Australia. It was easy to

Table 29. The wheat stripe rust differential hosts used in various areas of the world with their *Yr* genes if known (372).

Europe		India		United States		China	
Host	Yr gene	Host	Yr gene	Host	Yr gene	Host	Yr gene
Carstens V		Chinese 166	1	Chinese 166	1	Abbondanza	
Chinese 166	1	Compair	8	Durchamp	3a,+	Beijing 8	
Clement	2,9,+	Heines Kolben	6	Heines VII	2	Bima 1	
Compair	8	Heines VII	2	Lee	7	Danish 1	
Heines Kolben	6	Hybrid 46	3b,4b	Lemhi		Early Premium	
Heines VII	2	Kalyansona	2	Moro	10	Feng Chan 3	
Heines Peko	2,6,+	Lee	7	Paha	12,+	Fulhard	
Hybrid 46	3b,4b	Moro	10	Produra		Funo	
Lee	7	Riebesel 47/51	9,+	Riebesel 47/51	9,+	Jubilejna 2	
Moro	10	Strubes Dickkopf	2,3a,4a	Stephens	3a,+	Kangyin 655	
Nord Desprez	3a,4a	Suwon 92/Omar		Tadorna		Kansu 96	
Reichersberg 42	7,+	Sonalika	2,A,+	Yamhill	2,4a,+	Lovrin 13	9
Riebesel 47/51	9,+	*Triticum spelta album*	5			Lutescens 128	
Spaldings Prolific		Vilmorin 23	3a,4a,+			Mentana	
Strubes Dickkopf	2,3a,4a					Quality	
Suwon 92/Omar						Shuiyun 11	
Vilmorin 23	3a,4a,+					Strubes Dickkopf	2,3a,4a
						Tanshan 1	
						Trigo Eureka	
						Virgilio	
						Xibei 54	
						Xibei Fenshou	
						Zun 4	

postulate the resistance genes in wheat cultivars W3750, W3752, W3753, and W3761 because of infection type variation or the expression of a characteristic mesothetic infection type comparable to that of the check lines. The response of cultivar W3760 indicated that any of three genes (*Lr9*, *19*, or *24*) could be present. If the parentage of W3760 were known, one or more of these genes might be eliminated. If additional isolates that vary in virulence to *Lr9*, *19*, and *24* are available, they could be used in additional postulation tests. Otherwise, the further study of resistance of W3760 would require genetic studies.

Table 30. Genome location for resistance to the cereal rust diseases.

	Genome		
Chromosome	**A**	**B**	**D**
	Leaf rust resistance genes		
1	10	26,33	21,40,41
2	11,17,37,38	13,16,23,35	2*,15,22*,39
3		27	24,32
4	12,25,31	28,30	
5		18	1
6		3*,9,36	
7	20	14*	19,29,34
	Stem rust resistance genes		
1		14,31	18,33
2	21,32,34,38	9*,16,19,20,23,28 32,34,39,40,Kt2	6,U
3	27,35	2,12	24
4	37	7*,Tmp	
5			30
6	8*,13,26	11	5,29
7	15,22	17	25
	Stripe rust resistance genes		
1		9,10,15	
2	1,17	5,7	8,16
3			
4			
5			
6			
7		2,6	18

* Multiple alleles at these loci.

Once a postulation is made, genetic analysis can be used to confirm the postulation. Gene postulation should at least indicate when further study is not warranted.

Advantages

- Analysis can be done in several weeks.
- Fairly accurate and easy when only a few genes are present and necessary variation exists in the pathogen population.
- The absence of a resistance gene is conclusively demonstrated when a high infection type occurs on the test line with a pathogen avirulent on the resistance gene.

Disadvantages

- A collection of isolates differing in virulence is required.
- The presence of a gene is indicated but not proven.

Genetic analysis

Studies may involve crossing resistant and susceptible cultivars, or crossing various parents with one or more known gene(s) for resistance ('allelism test'). The inheritance of resistance in various filial generations can be used to estimate the number of genes segregating for resistance in the cross.

Figure 17 illustrates the pattern of monogenic inheritance. The first observation is recorded on the response of F_1 hybrids. A resistant response similar to the parent indicates dominance of resistance. Partial dominance is characterized by an intermediate response. Harvest the F_1 hybrids, preferably as single plants, which will give rise to the F_2 population. The ratio of resistant versus susceptible F_2 plants indicates the number of resistance genes segregating in the cross. Following Mendel's law of segregation and in the

Table 31. A demonstration of gene postulation using a wheat stem rust model.

Host line	*Sr* gene	Isolates 1	2	3	4
		Check lines			
ISr6-Ra	6	;	;	4	4
ISr8-Ra	8a	2	4	2	4
Line E	none	4	4	4	4
		Test lines			
Line 1	?	;	;1-	4	4
Line 2	?	2	4	2-	4
Line 3	?	4	4	4	4
Line 4	?	;	;	2	4
Line 5	?	0	3+	1N	4

Conclusions drawn from the table:

Line 1 is postulated to have *Sr6*, infection pattern is similar to ISR6-Ra.

Line 2 is postulated to have *Sr8a*, infection pattern is similar to ISr8-Ra.

Line 3 *does not* have either *Sr6* or *8a*.

Line 4 is postulated to have *Sr6* (cultures 1 and 2) and *Sr8a* (culture 3). Note due to epistasis the low infection type to culture 1 is that of *Sr6*.

Line 5 has resistance, but neither the pattern of low infection types nor the low infection types expressed are related to *Sr6* or *8a*. With these cultures no postulation can be made even though perhaps two resistance genes are present.

absence of linkage, the expected F_2 segregation ratios and interpretations are:

3 resistant: 1 susceptible = 1 dominant gene;

1 resistant: 3 susceptible = 1 recessive gene;

13 resistant: 3 susceptible = 1 dominant + 1 recessive gene;

7 resistant: 9 susceptible = 2 recessive genes;

15 resistant: 1 susceptible = 2 dominant genes;

9 resistant: 7 susceptible = 2 complementary genes.

More complex ratios can also be interpreted, but a larger population size is required. Hanson (130) described the minimum family size requirement for different segregation patterns.

Some ratios are difficult to establish even though large populations are tested. One such example is in distinguishing the 3:1 ratio from the 13:3 ratio. If segregation occurs for distinctly different low infection types (e.g., ; and 2), it is advisable to classify seedlings in all possible infection type classes to break the ratio into components. The 13 resistant: 3 susceptible ratio expected for the segregation of 1 dominant gene + 1 recessive gene can be further divided into: 12 resistant with infection type ";" , 1 resistant with infection type 2, and 3 susceptible responses for a more critical statistical analysis.

The F_2 plants should be harvested individually to obtain F_3 lines. Segregation in the F_3 lines provides the genotypic classification of individual F_2 plants based on the response of the progeny. The F_3 line ratio permits the number of genes to be more accurately estimated. For example, a distribution of 1 homozygous resistant, 2 segregating, and 1 homozygous susceptible indicates segregation at a single locus. A distribution of 7 homozygous resistant, 8 segregating, and 1 homozygous susceptible indicates two genetically independent loci.

A matrix of data can be generated, which can be used to identify and estimate the number of genes segregating against each isolate, by inoculating F_3 lines (all or selected) with isolates differing in virulence. F_3 data are superior to F_2 data because their analysis provides simultaneous genotypic classifications of each host line with each isolate.

Backcrosses of the F_1 plants with the susceptible (dominant resistant) or resistant (recessive resistant) parents can be evaluated along with those of the F_2s. When a susceptible parent is used in

Table 32. Gene postulations using seedling infection type data from five wheat cultivars and seven leaf rust isolates (349).

Wheat cultivars	Isolates 72469	67028	63666	76694	76348	81501	64-L-3	Postulated *Lr* gene(s)
W3750	X=	X=	X-	X-	X-	X-	X	13
W3752	X-	0;	0;	X=	X-	X=	0;	1,13
W3753	;	0;	3+	3	3+	3	0;	3
W3760	0;	0;	;	;	0;	;	0;	9/19/24[a]
W3761	X	0;	0;	3	X-	3+	0;	1,17
				Checks				
Lr1	3+	0;	0;	3+	3+	3+	0;	
Lr3	;	0;	3+	3+	3+	3+	0;	
Lr13	X=	X=	X-	X-	X=	X-	X-	
Lr17	X	X-	X-	3	X-	3+	X	
Lr9	0;	0;	0;	0;	0;	0;	0;	
Lr19	0;	0;	0;	0;	0;	0;	0;	
Lr24	0;	0;	0;	0;	0;	0;	0;	

[a] Response data suggest that one of these genes is involved.

backcrossing resistant F_1s, the expected backcross segregation ratios and their interpretations are:

1 resistant: 1 susceptible = 1 dominant gene;
3 resistant: 1 susceptible = 2 dominant genes;
7 resistant: 1 susceptible = 3 dominant genes.

Disadvantages of backcross ratios

- If the resistant parent possesses two genes, one dominant and one recessive, the segregation in the BC_1 generation will be the same as that expected for one dominant gene.
- Production of large backcross hybrid populations is fairly costly.

The F_2 generation is a better option than the BC_1 for estimating the number of resistance genes. If necessary, the F_2 segregation ratio should be verified by evaluating the F_3 lines.

The F_2 and F_3 ratios are altered if the segregating genes are not independent. The amount of alterations depends on the percentage of crossing over between the two loci. Some genes, e.g., *Sr36* for stem rust resistance (270), are known to give distorted segregation ratios. Such distorted segregation patterns require genetic analysis beyond the scope of this manual.

When studying F_3 lines, small seed samples can simultaneously be evaluated with other less important races that carry different virulence combinations. Some genes for resistance to one rust are known to be linked with genes for resistance to other rust diseases (see Tables 5, 10, and 14). The F_3 lines would be useful for testing resistances to more than one rust—providing that the susceptible parent was also susceptible to the other rust.

Sometimes, it is difficult to identify the resistance gene due to the limited variation in available pathogen isolates. In such cases, the resistant parent can be crossed with tester lines possessing designated genes. The parentage of the resistant line may aid in choosing which lines to cross. The absence of segregation in the F_2 indicates that both parents probably have the same gene or another allele at the same locus. Exceptions usually involve close linkages between resistance genes linked in repulsion. Segregation indicates that the parents do not have the same gene(s). Occasionally, a complexity may arise if one of the parents used in the cross has a translocation involving the chromosome segment with the resistance gene.

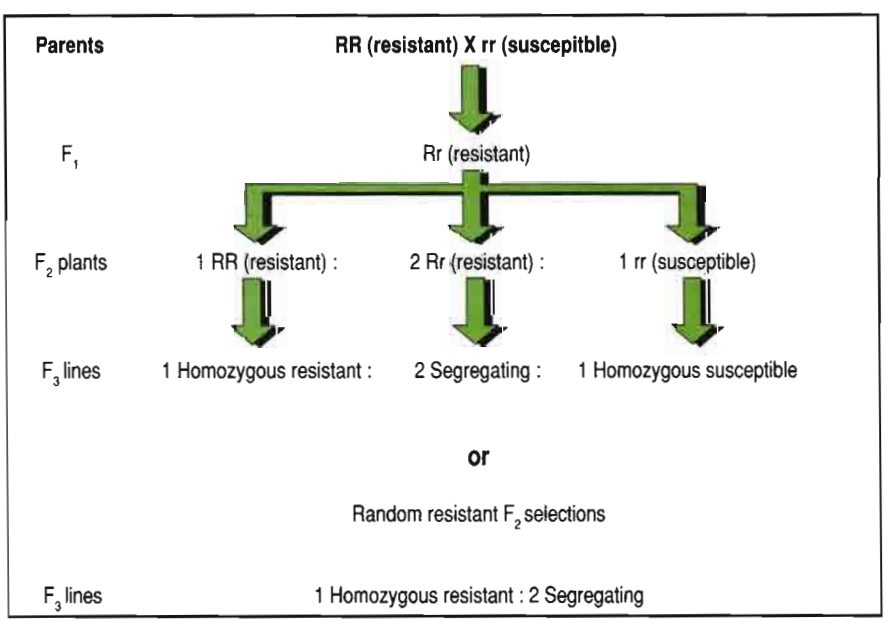


Figure 17. The pattern of monogenetic inheritance for a dominant rust resistance gene in a cross between a susceptible cultivar and a resistant cultivar.

Cytogenetic analysis

Cytogenetic analysis involves using monsomic (one missing chromosome) and telosomic (one missing chromosome arm) plants. Studies can be made of segregating generations from

crosses of the selected resistant cultivars with standard susceptible monosomics for the 21 chromosomes to identify a gene's location on the chromosome (335). Use susceptible monosomic plants for each chromosome (as the female parents) in crosses with resistant cultivars (as male parents) (Figure 18). Select and self the monosomic F_1 plants and determine the segregation ratios for the F_2 generation. The segregation is normal for the resistant x monosomic cross (i.e., 3 resistant:1 susceptible) if resistance is dominant and the gene is not located on that chromosome. A ratio of approximately 97 resistant:3 susceptible indicates the resistance gene is located on that chromosome. Only one out of the 21 crosses (21 monosomics) will segregate in an abnormal manner. The ratio may vary slightly depending on the chromosome involved and the environment where the plants were grown. Susceptible plants occur at a very low frequency (approximately 3%) in the F_2 population of the critical cross because male gametes with 20 chromosomes have approximately a 3% fertilization frequency and gametes with 21 chromosomes have a frequency of 97%.

The F_2 plants can be advanced to obtain F_3 lines, which is more desirable when the resistance is recessive. The response of the F_3 lines can be correlated with the chromosome constitution of the parental F_2 plants. For example, in a 'critical' cross, disomic (2N = 42) F_2 plants will produce homozygous resistant progenies and monosomic (2N = 41) F_2 plants will again segregate with abnormal ratios. Susceptible plants in these progenies will usually be nullisomic (2N = 40). Other low frequency chromosome arrangements in each of the response classes are possible—for example, spontaneous monosomics for other chromosomes and secondary aneuploids.

Monotelosomic (deficient in one entire chromosome and one arm of the homolog) plants, if available, are preferred to monosomic plants because they minimize the chances of monosomic shift. In addition, the F_1 hybrids with 2N = 41 + a telosome can be used to locate the gene in a chromosome arm and to estimate the distance of the gene from the centromere (336).

A new resistance gene must be located on the chromosome before allocating a permanent designation. Although the individual plants in a line may be homozygous, an unselected parent line may be genetically heterogenous. For valid comparisons, it is essential to use 'pure' lines that can be obtained by selecting a single plant or single spike progeny for crossing. Keep selfed seed of the parent plant for comparison with segregating progenies and with the original unspecified bulk line. It often is desirable to inoculate parents with rust before making the cross. Bag F_1 spikes to avoid outcrossing. Keep the number of races low (ideally one) in the mixture to be used for field inoculations. It is often useful to determine if the rust in the nursery has the same virulence as the isolates used to inoculate the nursery. This contrasts with a breeder's selection nursery where a mixture of races may be preferred when the objective is to select a progeny with resistance to the widest possible array of races.

Disadvantages

- Cytogenetic methods are labor intensive and time consuming because aneuploids must be maintained, the test lines need to be crossed with various aneuploids, and cytology is required at various stages.

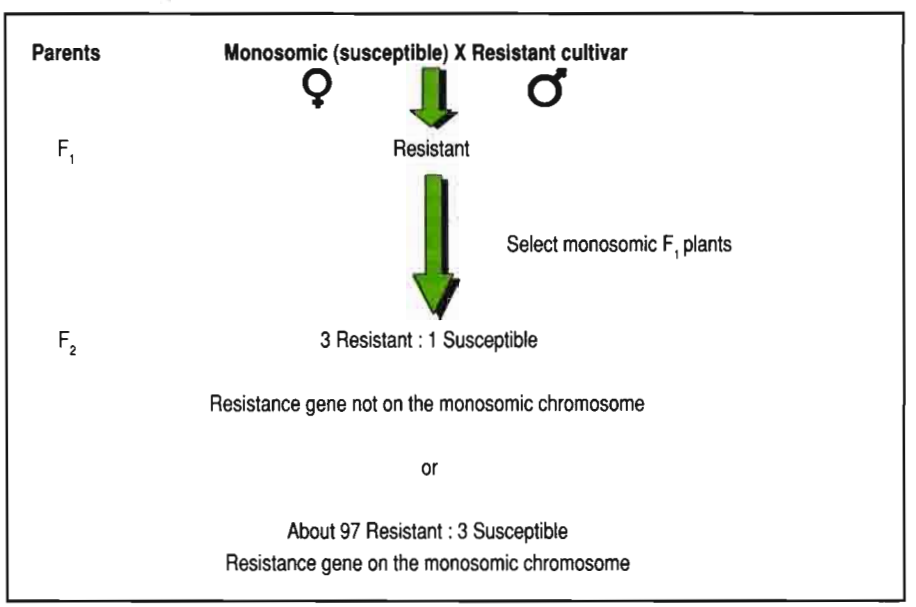


Figure 18. The pattern for inheritance of resistance for monosomic wheats crossed with a resistant cultivar.

Notes:

Strategies for Using Resistance

Several methods of using host resistance more effectively have been proposed, including:

- Pyramiding genes for resistance.
- Deploying new genes for resistance.
- Using multilines or cultivar mixtures.

Gene pyramids

A gene pyramid involves the use of several genes in a single cultivar to provide a wider base of disease resistance. Most breeders worldwide use this approach for the three rusts. Many gene pyramids have been successful, although some have quickly been rendered ineffective. At least in a few cases, *Lr13* and *16* (332), *Lr2a* and *16* (117), *Lr13* and *34* (101), *Lr27* and *31* (351), and undesignated genes for stripe rust resistance (118, 344) seem to have an additive effect in combination.

Some resistance gene combinations, such as the '*Sr2* complex' for stem rust resistance (236, 286), the 'Frontana complex' for leaf rust resistance (302), and the resistance of Anza and Little Joss for stripe rust (160), have shown long-term durability. These complexes provide the basic resistance in the emerging bread wheat germplasm at CIMMYT (286). Such durable resistance can be combined with other genes to provide some diversity.

The breeding methodology for developing gene pyramids involves the identification of genetically different sources of resistance, followed by the incorporation of these resistances into a high yielding and adapted background. This can be accomplished by any selection methodology (pedigree or bulk breeding) following simple, top (three-way), or double crosses. CIMMYT breeders use the modified bulk method of selection:

- *F_1 simple crosses.* These are evaluated on the basis of disease resistance, agronomic character, and hybrid vigor and harvested in bulk for the F_2.
- *F_1 three-way, double cross, and backcross populations.* These materials are handled on an individual plant basis. Materials are space-planted; outstanding plants with resistance are identified, selected, and advanced to the F_2.
- *F_2.* Two to three thousand plants are space-grown in well watered, high fertility conditions under rust epidemics. Individual plants are identified on the basis of rust resistance and agronomic suitability (i.e., tillering, lodging, fertility, maturity, and grain type).
- *F_3.* Progeny of selected F_2 plants are grown in solid stands in 2-m, two or three-row plots. Rust epidemics are again created. Progeny of each plant is treated as a separate population. Selection is based on the plot and then individual selected spikes in the plot are bulked.
- *F_4.* Bulk F_4 is planted in solid stands and the selection criteria are the same as for the F_3.
- *F_5.* Approximately 100 plants/plot selected in the F_4 are space-planted. Individual plants are selected on the basis of rust resistance, acceptable agronomic characters, and spike fertility.
- *F_6.* Each plant selected in the F_5 is grown individually as F_6 in plots of two or three rows, 2 m long. Agronomically outstanding lines acceptable in rust resistance are bulked and advanced to yield evaluations.

If the objective is to develop pyramids of minor genes, their accumulation and combination with other agronomic and yield characters are difficult, but not impossible to achieve. Parlevliet (275) has recently reviewed strategies for the utilization of partial resistance for the control of cereal rusts. This type of resistance is often more vulnerable to environmental effects. In a severe epidemic, partial resistance seems to be less effective and hence selecting for this character by creating severe epidemics is a doubtful procedure. The best breeding strategy to combine polygenes for partial resistance is recurrent selection in which improvement of resistance is a continuous process. When partial resistance is controlled by a few genes with additive effects, accumulation of such genes can be achieved by carefully choosing the parents for crossing and through any breeding methodology.

Advantages

- Residual effect of defeated genes (41).
- Reduction in the fitness of the pathogen.

Disadvantage

- Many genes are exposed to the pathogen at one time.

Gene deployment

The use of different combination(s) of resistance genes in different areas of an epidemiological zone (164, 182, 256) is a promising strategy. However, in practice, it is difficult unless all breeding programs, farmers, and government agencies in a zone agree to cooperate. In addition, seed distribution must be controlled and adequate, and good resistances must be available. It is essential that the resistances used are known to be different. For effective gene deployment, the inoculum source for the area where the disease control is targeted must be exogenous. Endogenous inoculum would be virulent on locally grown cultivars.

Cultivar mixtures (mulitivars) and multilines

A certain diversity already exists in agriculture due to farmer use of several cultivars and the intervening space between wheat fields due to roads, canals, towns, and fields of other crops. However, cultivar mixtures and multilines have not been used commercially for wheat to any great extent (256). Multivars can be developed by selecting a high yielding, well adapted cultivar and by incorporating the genes from various genetically different sources through backcrossing.

Advantages

- A more natural system where host plants are not genetically identical and pathogen levels are maintained at a nonepidemic level (256).
- Host diversity might result in the stabilization of the pathogen population at intermediate levels of aggressiveness and with intermediate numbers of virulence genes.

Disadvantages

- A considerable amount of field testing must be done to test the above theoretical advantages using a range of host and pathogen types.
- A conservative approach where backcrossing or selecting aim to obtain similar agronomic types.
- Many genes are individually exposed to the pathogen population, which probably increases the risk of selecting a super virulent race—one that attacks all the resistance genes used.
- The time required to breed each multiline component approaches the time required to develop a pure line cultivar.
- Great difficulties are encountered when breeding for resistance to several diseases at the same time.
- A larger seed increase, maintenance, and distribution facility is generally required because, unlike pure line cultivars, the farmer's seed may shift from the initial desired frequency of components.
- Competition between multiline components may reduce the benefits of controlling the disease (4).

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GLOSSARY

Accessory host—A host other than the primary host on which uredinia are produced.

Aecium—The structure on the alternate host in which aeciospores are produced.

Aeciospore—The dikaryotic spore (N+N), produced on the alternate host, capable of attacking the cereals.

Alternate host—The rust host on which pycnia and aecia are produced.

Aneuploid—Having a chromosome number that is not an exact multiple of the haploid number.

Appressorium—The structure formed at the end of the germ tube above a stoma from which the infection peg develops.

AUDPC (ADPC)—Area under the disease progress curve used as a measure of slow rusting.

Avirulence—The specific inability of the pathogen to overcome a host gene for resistance.

Basidiospore—The haploid spore (N), produced after meiosis from a basidium, which infects the alternate host.

Basidium—The structure produced by a germinating teliospore on which basidiospores are produced.

Coefficient of infection—The product of the percent disease severity (modified Cobb scale, Fig. 11) and a constant; immune = 0; resistant = 0.2; moderately resistant = 0.4; moderately susceptible = 0.8; and susceptible = 1; used to give a single value for disease resistance in field evaluations.

Culture—A clone of a urediniospore that is maintained in the laboratory. See isolate.

Cultivar—A cultivated variety as opposed to a botanical (taxonomic) variety.

Dicaryotic—A tissue or spore in which plasmogamy has occurred but karyogamy has not occurred.

Differential host—A wheat line that is susceptible to some isolates and resistant to others.

Disease onset—The day disease first appeared in a field or plot which may not be the day disease was first observed.

Disease progress curve—Amount of disease (or its transformation) plotted against time.

Disomic—A plant with all homologlous pairs. In hexaploid wheat, a plant with 21 homologous bivalents.

Endogenous—From inside the area under consideration.

Epidemiology—The study of how disease increases and spreads.

Epistasis—The suppression or modification (interallelic interactions) of the effect of a gene by a nonallelic gene.

Exogenous—From outside the area under consideration.

Fleck—Necrotic or chlorotic spot due to the resistance that results in no sporulation, often assigned the ';' symbol.

Forma specialis (f. sp.)—Form within a pathogen species that refers to the primary host species attacked.

Gene-for-gene theory—The specific interaction between a host gene for susceptibility or resistance and the corresponding pathogen gene for virulence/avirulence.

Gene pyramid—Accumulation of several genes for resistance to a single disease in a cultivar or line. Addition of new genes for resistance to those existing in previous cultivars.

Green bridge—Presence of green host plants during the crop's off season.

Heterogeneous—A mixture of genetically different individuals. A wheat cultivar is often heterogeneous.

Infectibility—Number of infections per unit of inoculum on a specific host (N/N).

Infection peg—The structure that develops from the appressorium and penetrates between guard cells of the host epidermis into the substomatal cavity.

Infection type—The visible symptoms of disease produced by the interaction of the host and pathogen in a specific environment.

Infectivity—The difference in a number of infections per unit of inoculum between pathotypes. This is a characteristic of the pathogen. See receptivity.

Inoculum—Propagule by which pathogen is spread, in the case of wheat and the cereal rusts urediniospores or perhaps aeciospores.

Inoculum density—Number of inoculum propagules per unit area or volume.

Isolate—A clone derived from a single uredinium. See culture.

Karyogamy—The fusion of nuclei in sexual mating.

Latent period—Time, usually in days, from spore germination until 50% of the uredinia are producing spores.

Lesion—A uredinium in the case of the cereal rusts.

Mesothetic—An infection type produce by a single isolate consisting of a range of uredinial sizes, i.e. the X, Y, or Z responses (see Table 21).

Misting—Creation of fine droplets of water to form an artificial dew on plants.

Monosomic—A plant with one missing chromosome. In hexaploid wheat a plant with 20 homologous bivalents and one univalent for a complete chromosome.

Monotelosomic—A plant deficient in one entire chromosome and one arm of the homolog. In hexaploid wheat, a plant with 20 homologous bivalents and one arm of the missing chromosome pair.

Multiline—A cultivar composed of several agronomically similar lines that differ in resistance to a disease.

Nullisomic—A plant with one missing chromosome pair. In hexaploid wheat, a plant with 20 homologous bivalents.

Pathotype—A phenotypic description of the host-parasite response.

Physiologic race—Virulence/avirulence pattern grouping for isolates evaluated on a specified set of differential hosts.

Plasmogamy—The fusion of the cytoplasm in sexual mating.

Primary host—The cereal host for the rusts on which urediniospores are produced.

Pustule—A uredinium in the case of the cereal rusts.

Pycniospore (spermatium)—The haploid spore (N) that serves as the male gamete, normally moved by insects or water.

Pycnium (spermagonium)—Structure (N) resulting from basidiospore infection that bears pycniospores and receptive hyphae on the alternate host.

r value—The rate of disease increase.

Race (physiological)—A nonrandom assemblage of virulences and avirulences as determined on a series of differential hosts.

Ratooning—The growth of new shoots after a crop has been cut.

Receptive hypha—The haploid mycelium (N) in a pycnium that serves as the female gamete.

Receptivity—The number of infections produced with a standard amount of inoculum in a specific environment for a specific host. Receptivity is a characteristic of the host. See infectivity.

Resistance—The genetic character of the host that prevents avirulent isolates from attacking it.

Resistance, types of:

Adult plant—Resistance expressed near or after heading.

Durable—Resistance that has been effective for many years when in widespread use.

Field—Resistance that is observed in the field.

Generalized—Resistance that is effective against most isolates.

Horizontal—Resistance that is equally effective against all isolates evaluated.

Hypersensitive—Resistance that is characterized by a chlorotic or necrotic spot (fleck) where a few host cells died near the point of infection. No sporulation occurs.

Immunity—Resistance that results in no visible symptoms to the unaided eye.

Major gene—Resistance that is easy to measure and due to a single host gene.

Minor gene—Resistance that is difficult to measure and usually thought to be due to several host genes.

Monogenic—Resistance due to a single gene.

Multigenic—Resistance due to several genes.

Oligogenic—Resistance due to a few genes.

Partial—Resistance that permits some sporulation.

Polygenic—Resistance due to several genes often less effective singly.

Qualitative—Resistance that appears to be in distinct classes.

Quantitative—Resistance that is difficult to classify in discrete classes.

Race-nonspecific—Resistance that is equally effective against all isolates evaluated.

Race-specific—Resistance that is effective against only some isolates.

Seedling—Resistance expressed in the primary leaf often effective through the plant's life.

Slow-rusting—Occurs when a cultivar displays a susceptible response, but with slower disease progress than in a susceptible check cultivar.

Vertical—Resistance that is effective against only some isolates.

Self-sown—Plants that germinate in the field, along the roadside, or elsewhere from seeds that are dropped during harvesting or transportation.

Substomatal vesicle—Fungal structure from in the intercellular space beneath the guard cells from which primary hyphae develops.

Susceptibility—The inability of the host to prevent a pathogen from attacking it.

Teliospore—A dikaryotic black resting spore (N+N) which becomes diploid (2N) before germinating.

Telosomic—In bread wheat a hexaploid missing the same arm for one chromosome pair.

Tolerance—The theoretical ability of a plant to yield in spite of a high level of disease.

Translocation—A plant where a segment or an arm of a chromosome is exchanged with or transferred to a nonhomologous chromosome.

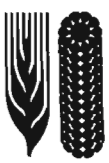
Uredinium (uredium)—The lesion on cereals that produces urediniospores, also called a pustule or lesion.

Urediniospore (uredospore, urediospore)—The asexual dikaryotic repeating spore (N+N) of the rusts. Often airborne over great distances.

Virulence—The specific ability of the pathogen to overcome the host gene for resistance.

Volunteer—See self-sown.

ISBN: 968-6127-47-X



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