THE CEREAL RUSTS: AN OVERVIEW

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ABSTRACT

For centuries cereal rusts have been threatening mankind imposing severe yield losses on crops, economic and social impacts and sometimes even famine. By far, they are the most damaging diseases of cereals. The pathogens are obligate parasites that co-evolved with their hosts in a system greatly influenced by the environment. This review presents a historical retrospective of cereal rusts as well as aspects of their biology, such as taxonomy, life cycle, physiological specialization and the process of infection. In addition, more specific details are presented for each of the main rusts of wheat, barley, oats and rye. Methods of controlling them, particularly genetic, chemical and through cultural practices are also presented.

Keywords: barley, crown rust, leaf rust, oat, Puccinia, rye, stem rust, stripe rust, wheat

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THE CEREAL RUSTS IN HISTORY

Rusts are known as the most destructive of the cereal diseases and since Ancient times have had a great influence on human civilizations. The first knowledge of famine that can be attributed to rust is in the Old Testament, in the story of Joseph in Egypt, 1,800 BC. Joseph interpreted two dreams of the Pharaoh as one and the same: The seven fat flashed cows and the good ears of grain meant seven good years when the crops of Egypt would be plentiful, and the seven lean fleshed cows and the ears blasted by east wind would be seven years of famine when the crops of Egypt would

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fail. The analysis of the climate, of the vegetation and of the patterns of the predominant winds in the area takes to the current interpretation that the warm and wet winds of great cyclonic storms would have swept eastward over the Mediterranean and spread trillions of rust spores from wild wheat and barley bushes, the other hosts of rusts, which blasted wheat crops in Egypt (Carefoot and Sprott 1967). A scientific evidence of this occurrence in Ancient times was provided for by Homer in the Iliad. In a passage from the Bronze Age (1300 BC) two ancient lemma fragments of wheat with charred, but well preserved spores and hyphae of the stem rust fungus were found (Kislev 1982).

In Greece, centuries before the Christian era, Aristotle (384–322 BC) wrote that the farmers of the time thought that the rusts were produced from warm vapors and he noted that their severity fluctuated from year to year. Theophrastus, pupil and heir of Aristotle, regarded as the “Father of Botany”, in his Historia Plantarum, written twenty two centuries ago, stated that cereals were more liable to rust than pulses and that periods of warm sunshine following the heavy dews of Greek spring seemed to bring rust to the wheat crops. Although the philosophers understood the weather conditions that promoted rust attacks on the wheat they could not tell how to combat the diseases, so the peasants lived from hand to mouth. When the time of Christ’s birth, wrote about Greek farmers still praying to Apollo, the sun God from relief from the rusting of their wheat (Carefoot and Sprott 1967).

The Roman wheat farmers were also plagued by rusts. As was the case in many unexplained events that occurred at that time, the Romans created a god, Robigus, the god of rust, who was honored in Robigalia, a religious ceremony practiced since 700 BC until the decline of the Roman Empire. Every Spring, on April 25th, a ceremony which involved sacrificing reddish colored animals, such dogs or cows to appease Robigus and induce him to hold back his vengeances. Historical writings and the study of the growth rings of the great old trees from the Mediterranean area, along both the European and African shores, show that the climate in the three first centuries after Christ’s birth was unusually wet, leading to conditions favorable for the spread of wheat rust. This weather was undoubtedly responsible for heavy losses due to wheat rusts. With these losses came hunger, discontent, famine and diseases, as typhus, plague and other decimating scourges of mankind, which helped to destroy the Roman civilization. Weakened from within by the destruction of their food supplies and the resulting human diseases, and beset from without by the barbarian hordes, the Roman Empire suffered a steady deterioration into subspecies, until the 20th century that understanding the life history is mostly wrong ones – before the main facts of the life history of these most destructive of all plant diseases, the rusts, were fully understood. Plant pathologists found out long before the 20th century that understanding the life history is not enough, but that the cultigens must be studied through all its phases, too. Understanding all aspects involved in the host-parasite relationship between small grain cereals and their rusts is a unique way to provide methods for managing these diseases and minimizing their impact on cereal production (Carefoot and Sprott 1967).

**TAXONOMY AND LIFE CYCLE**

The cereal rusts are diseases characterized by pustules resulting from uredial development of fungi classified into the phylum Basidiomycota, class Urediniomycetes, order Uredinales and family Pucciniaceae, which contains 17 genera and approximately 4121 species, of which the majority are in the genus Puccinia (Kirk et al. 2001). Rusts are functionally obligate biotrophs and, with the exception of stripe rust, are exclusively heteroblastic. The dikaryotic nature of the rust fungus, which is divided into distinct spore stages that occur during asexual reproduction on its graminaceous hosts and sexual reproduction that begins in the resting spore stage and culminates on the alternate host. The five spore stages can differ from each other in appearance, ploidy, pathogenicity, virulence, structures formed, and mechanism of penetration (Heath 1997; Staples 2000).

Based on spore size and host range, various subdivisions into subspecies, varieties and formae speciales have been proposed for the majority of the rust fungus species attacking cereal grasses (Dinoor et al. 1988; Chen 2005; Leonard and Szabo 2005).

In temperate climates, with the senescence of the graminaceous host and unfavorable environment conditions near the end of growing season, the cereal rust fungus produce under the leaf or stem epidermis thick-walled, two-celled teliospores (Fig. 11 and 1J). Each teliospore cell is dikaryotic when first formed, but dikaryogamy occurs early in teliospore maturation. Teliospores remain dormant in the infected straw until spring, when they germinate in synchrony with the new leaf growth in the alternate, non-graminaceous host. With spring rains and favorable temperatures, one or both cells of the teliospores germinates, undergoes meiosis and produces a four-celled hyphal protrusion called a promycelium or basidium. When meiosis is completed, the resulting haploid cells are separated from each other by three transverse septa. A projecting sterigma forms on each promycelium cell, and the haploid nucleus migrates through the sterigma into newly forming basidiospore as it expands at the tip of sterigma. Mitosis results in two identical haploid nuclei per mature basidiospore (Roelfs 1985; Roelfs et al. 1992; Leonard and Szabo 2005).

Mature hyaline and light- to dark-volute basidiospores are ejected from the sterigmata and carried by air currents to probably limited distances, to infect the alternate host. Basidiospores germinate and penetrate directly. Infection results in the production of flask-shaped pycnia, usually in the upper leaf surface. The pycnia produces receptive hyphae and small, thin-walled pycniospores which exude from the tip of the pycnium in a drop of pycnial nectar. The nectar is attractive to insects that, along with rain splashing, brushing of leaves, and activities of animals, serve to disseminate pycniospores among pycnia. Pycniospores serve as the male gametes and consist mainly of a single haploid nucleus with little surrounding cytoplasm. Flexuous hyphae extend out of the top of the pycnia and serves as the female gametes. Two mating types, commonly designated + and –, have been identified and appear to be under monogenic control (Roelfs 1985; Roelfs et al. 1992; Leonard and Szabo 2005).

When a pycniospore of one mating type contacts flexuous hyphae in a pycnium of the other mating type, fusion...
occurs. The dikaryotic stage is established with nuclear division and paired association of + and – mating type nuclei. The next spore stage starts with the growth of a cup-shaped, dikaryotic aecium formed below the pycnium 7 to 10 days after fertilization, frequently rupturing the lower epidermis of the alternate host’s leaves (Fig. 1A, 1B). Chains of single-celled, dikaryotic aeciospores (Fig. 1C) are produced, which can infect gramineous hosts but not the alternate host. As the aeciospores are the product of genetic recombination, they may differ in virulence and aggressiveness, and the extent of variation depends on the differences between the parental isolates (Roelfs 1985; Roelfs et al. 1992; Leonard and Szabo 2005).

Aeciospores are hydroscopically released from the aecium and are airborne to the gramineous host over long distances. Infection by aeciospores results in the production of a dense mat of hyphae beneath the host epidermis, where sporophores grow and produce masses of single-celled dikaryotic urediniospores (Fig. 1F) that rupture the host epidermis producing a pustule known as a uredinium (Fig. 1D, 1E). Urediniospores are dispersed by wind and can reinfect the cereal host, repeating asexual cycle within each 7 to 14 days, depending on the rust species and environment conditions. Under field conditions where temperatures vary greatly the cycle can be either lengthened or shortened. Generally, lower temperatures in the field, at least at the
early stages of the crop cycle tend to lengthen the latent period. On maturing hosts, uredinia eventually cease production of urediniospores and begin to produce two-celled teliospores (Fig. 11, 1J). At that stage the infection structure is called a telium (Fig. 1G). Teliospore stalks remain intact and the spores are not dispersed from the telial pustule (Fig. 1H; Roelfs 1985; Roelfs et al. 1992; Leonard and Szabo 2005).

The alternate host currently provides little direct inoculum of cereal rusts, but may be a mechanism for genetic exchange between races and perhaps populations. In most areas of the world, the life cycle of cereal rusts consists of continual uredinal generations. The fungi spread by airborne urediniospores from one gramineous plant to another and from field to field. Primary inoculum may originate locally from volunteer plants, which serve as a ‘green bridge’ to sustain the uredinal stage, or be carried long distances by wind and deposited by rain (Singh et al. 2002).

An alternate host is not known for the stripe rust pathogen, *P. striiformis*. This is most likely a hemiromust in that the life cycle seems only to consist of the uredinal and telial stages, and so, the urediniospores are the only known source of inoculum (Singh et al. 2002). Teliospores have a very short dormancy and basidiospore production is rapid and survival short (Wright and Lennox 1978). On the basis of cytological information, Goddard (1976) believes that this rust would be macrocyclic if an alternate host were found.

**PHYSIOLOGIC SPECIALIZATION**

The causal agents of cereal rusts are obligate parasites that co-evolved with their hosts as components of a system very influenced by the ecological conditions, in other words, any change in the predominant population of the host results in subsequent changes in the population of the pathogen, in way that the balance be reestablished (Wahl et al. 1984). Stakman and Piemeisel provided the first understanding that the *formae speciales* could be subdivided into races. They found that varieties of *P. graminis* f. sp. *tritici* were not homogenous but consisted instead of physiological forms, strains or races, separated from one another by their effects on different varieties of the host plant (Roelfs 1984). Stakman and Levine (1922) then developed a standard set of differential varieties of wheat to identify races of *P. graminis* *tritici* within field collections. This set consisted originally of 12 varieties that consistently ensured uniformity of race identity. Mains and Jackson (1926) used a set of 11 varieties of the single species *T. aestivum* to identify pathotypes of wheat leaf rust (*Puccinia triticina*). Mains and Jackson (1926) also termed ‘differential variety’, but apparently they preferred the term ‘differential strain’ because many wheat varieties at that time were not genetically homogeneous. Over time with greater emphasis on varietal purity in crops, the term ‘differential variety’ became generally accepted and ‘differential strain’ disappeared from the literature.

Differential varieties also made possible the study of genetics of resistance in flax and virulence in flax rust, *Melampsora lini*, by Flor (1956) from 1935 to 1956. In a series of studies during this period he elucidated the genetics of host-parasite relations and formulated the gene-for-gene hypothesis, one of the most important paradigms in plant pathology. The basis for Flor’s hypothesis was founded on the work of Eriksson and Henning who proposed the new *formae speciales* as a subspecies of *P. graminis* (Shafer et al. 1984) and his theory continues as the dominant force in understanding the cereal rusts. Flor’s most important conclusion was that suppression of *M. lini* in resistant flax plants was due not just to the resistance gene in flax, but rather to the interaction between the resistance gene in flax and the corresponding avirulence gene in the flax rust fungus. Person (1959) expanded on Flor’s analysis by demonstrating that maximum numbers of pathotypes that can be distinguished when each differential variety has a different single gene for resistance.

As first elucidated by Flor (1956), races themselves are now known to be of heterogenous genotypes, which can be distinguished from other genotypes of the same race by virulence patterns on supplemental differentials added to the standard set of differential varieties of the host plant used to define the races (Stakman and Harrar 1957). Although any number of differentials may be used, standard sets generally include about 8 to 16 differential varieties. Larger sets become unwieldy for routine use. The sets of differential varieties have differentials with different single resistance genes backcrossed into a common genetic background. Near-isogenic differentials eliminate confounding effects of modifier genes acting on resistance in different host backgrounds. Currently, the sets of differential hosts used to identify races of the main cereal rusts are that described by Long and Kolmer (1989), for *P. tritici* (*wheat leaf rust*), by Roelfs and Martens (1988) for *P. graminis* f. sp. *tritici* (*wheat stem rust*); by Chen (2005) for *P. striiformis* f. sp. *tritici* (*wheat stripe rust*); by Chong et al. (2000) for *P. coronata* f. sp. *avenae* (*oat crown rust*); by Fetch and Jin (2007) for *P. graminis* f. sp. *avenae* (*oat stem rust*); by Franckowiak et al. (1997) for *P. hordei* (*barley leaf rust*) and by Chen et al. (1995) and Chen and Line (2003) for *P. striiformis* f. sp. *hordei* (*barley stripe rust*).

Rusts population dynamics in plant pathology (Roelfs et al. 1992) in advances in the basic studies of epidemiology and population dynamics in plant pathology (Roelfs et al. 1992). Laboratories throughout the world have been conducting virulence surveys and molecular characterization of major cereal rusts because of the usefulness of these data for both, breeding resistant varieties and epidemiological studies (Singh 1991; Roelfs et al. 1992; Kolmer 1999; Kolmer 2001; Kolmer and Liu 2000; Hovmoller 2001; Park et al. 2001; Chen 2005; Leonard and Martinelli 2005; Chaves et al. 2005; Martinez et al. 2005; Chaves and Barcellos 2006; Manninger 2006; Mebrate et al. 2006; Woldeab et al. 2006; Kolmer et al. 2007; Lind and Gultyaeva 2007; Jiráková and Hanzalová 2008; Kolmer et al. 2008).

**INFECTION PROCESS**

The development of rusts on cereal hosts occurs during the asexual phase of the fungus. After urediniospore deposition on the host and its germination, a series of essential structures for the establishment of a successful parasitic relationship are formed (Leonard and Szabo 2005). The germinated spore forms a germ tube which differentiates called the appressorium when it recognizes the stomata. Therefore after, inside the plant tissues, a substomatal vesicle, the infective hyphae, the haustorium mother cells and the haustorium are formed. The presence of free water on the plant surface is the principal factor stimulating the process of spore germination, but there is evidence that the presence of some ions and changes in foliar pH can also stimulate spore germination (Cowey and Sreenivasan 1961). Independently of the rust species, the germ tube growth becomes oriented perpendicularly to the venation of the leaf, which is the first indication of fungal response to the host (Roderick and Thomas 1997). Vaz Pato and Niks (2001) observed that initially the germ tube grows in a random direction, and after contact with the epidermal cells junction directional growth perpendicular to the venation of the leaf occurs. This growth orientation is maintained through the growth of small germ tube ramifications on cell junctions. This
growth pattern reflects an evolutionary advantage, because it increases the probability of finding some stomata.

Very little, if any, change in the total volume of cytoplasm occurs during spore germination, so the cytoplasm in the spore migrates through the germ tube until the appressorium and other infective structures. During growth ofurediospore germ tube, the basipetal-most region of the cytoplasm forms long, trailing processes that extend back into the host cell cytoplasm. The layer of the hypha that occurs in this region of the hypha is due to a continued development and enlargement of vacuoles that probably requires new membrane synthesis. These vacuoles are surrounded by vesicles that secrete, probably inside of the vacuoles, substances that increase the water absorption, and consequently increase the hydrostatic pressure, allowing germination and appressorium penetration through closed stomata and the fast expansion of substomatal vesicle (Littlefield and Heath 1979).

Appressorium differentiation occurs after the germ tube development on leaf surface, and it is induced by specific topographical signs of the host leaf surface (thigmotropism responses), as well as by the emission of volatile compounds in the region around the stomata (chemotropic responses). Although mechanisms involved in signal recognition by the germ tubes are not clear, there are evidences that the angle and the distance of the germ tube growth on cells are crucial for appressorium differentiation. This hypothesis partially explains why the germ tube grows in different sizes before differentiating an appressorium (Collins and Read 1997). The spatial requirements for appressorium induction varies greatly among rust species, however, they usually correspond to the dimensions of the host stomata (Staples 2000). Some compounds present in the epicuticular layer or volatilized through the stomata induce the appressorium formation in vitro, and when the topographical and chemical stimuli are applied together, the appressorium differentiation is very similar of that observed on plants. These compounds can also induce differentiation of substomatal vesicles and haustorium mother cells (Marte and Montalbini 1999; Wiethölter et al. 2003; Reisige et al. 2006).

In rust species P. graminis and P. hordei, 91 to 99% of the germ tubes form appressoria when they reach the plant stomata. Using artificial substrates, Read et al. (1997) showed that 83 to 86% of the germ tubes formed by this rusts species are induced to differentiate appressoria only by topographic signs. These signs induce a sequence of unique biochemical and morphological events that lead to the differentiation of a set of infection structures: appressorium, penetration peg, substomatal vesicle, infective hyphae, and germ tube. Penetration peg penetration occurs by the emission of volatile compounds that trigger the resistance mechanisms in surrounding cells, and the fungus hyphae are postulated to enter the host cytoplasm by the plant (Dodds et al. 2007). After forma- tion of the first haustoria, infective hyphae form other haustorium mother cells. The energy to form the first haustoria is provided by reserves from urediniospores. Subsequent haustorium formation will be limited by nutrient reduction that will depend on the size of the haustorium and the lesion establishment. Very long or very ramified germ tubes seem to dispose less energy to progress with the plant infection (Niki 1990). Although appressorium formation is critical for the infection process, there are exceptions such as the species P. striiformis, whose germ tube can penetrate directly through stomata (Staples 2000).

Urediospore germination and appressorium formation usually occur at night in the presence of a film of water on the foliar surface. The development of P. graminis is paralysed after appressorium formation until the next morn- ning, when its growth is reestablished through light stimu- lus. Light stimulates the photosynthetic process, which reduces the internal rate of CO₂ in stomata, allowing the appressorium to develop. Appressorium penetration of P. grami-

minis was not observed in non photosynthetic or etiolated wheat plants submitted to CO₂, environmental rates less than 1% (Yigou and Caldwell 1963; Leonard and Szabo 2005). High luminous intensity increases the rate of penetration of Puccinia striiformis, however, the penetration through stomata also occurs in dark periods, which suggests that other phenomena controlled by light are involved in this process (Vallavieille-Pope et al. 2002).

After fungal penetration, haustorium mother cells, substomatal vesicle and infective hyphae are formed. In wheat, this process occurs in about 6 h after inoculation of the leaf rust pathogen, P. triticina (Hu and Rijsenberk 1998). Infective hyphae differentiate in a haustorium mother cell in about 12 h after inoculation. An infection peg penetrates the cellular wall, probably through enzymatic dissolution and mechanical pressure. Inside of the cellular wall, a specialized cell expands in the periplasmatic space, forming a haustorium, about 24 h after inoculation. Intercellular hyphae grow inside host tissues forming a fungal colony (Harder and Haber 1992; Heath 1997; Hu and Rijsenberk 1998; Leonard and Szabo 2005). During haustorium development, the fungus penetrates the plant cell wall and invaginates the host plasma membrane. The plant plasma membrane remains intact but becomes specialized in the region surrounding the haustorium. There is evidence that morphological and functional changes occur in the haustorial membrane. The region between the haustorial cell wall and the extrahaustorial membrane is the extrahaustorial matrix. This double membrane interface between the host cell and the parasite fungus seems to be the primary site of absorbance of nutrients from the host (Kneale and Farrar 1985; Mendgen et al. 2000; Perfect and Green 2001; Szabo and Bushnell 2001; Panstruga 2003; Leonard and Szabo 2005; O’Connell and Panstruga 2006; Dedos et al. 2007). Voegle et al. (2001) localized a hexose transporter in Uromyces fabae expressed only in haustoria. This transporter is homologous to that found in other Basidiomycetes and Ascomycetes fungi, what indicates that it can be present with a similar function in species of the genus Puccinia.

The haustorium formation is now recognized as a key element in maintenance of biotrophic organisms, facilitating their interaction with the host cell. In addition to their role in nutrient acquisition, haustoria also appear to manipulate host metabolism and defence responses (Voegle and Mendgen 2003), as well as inducing re-organisation of the host cell cytoskeleton and nuclear DNA (Heath 1997). Thus, the haustorium-host cell interface appears to mediate a dynamic interaction involving extensive trafficking of nutrients and signalling molecules. Effector proteins are secreted by the pathogen and are postulated to enter the host cytoplasm interacting for differentiation of haustoria, host metabolism and defence responses. When recognised by a corresponding resistance protein (R) the effector proteins are referred to as avirulence (Avr) proteins (Dedos et al. 2007). After forma-
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2001; Panstruga 2003). Once a compatible relationship has been established in a rust-infected host, the normal direction of phloem transport is altered to divert nutrients to the infected tissue at the expense of actively growing plant tissue. This change is characterized by massive increases of respiration and accumulation of cytokinins in the infected area. Sugars accumulate in the lesion area, and an invertebrate provides hexose sugars used by the fungus for growth and sporulation (Leonard and Szabó 2005).

**WHEAT RUSTS**

**Distribution and economic importance**

Modern agriculture feeds 6.7 billion people today (United Nations 2007), and 30% of food supply is provided by wheat (Triticum aestivum L.), which is the most important cereal in terms of tonnage and financial value (FAO 2008). Rust diseases are the most significant constraint to wheat production because they can reduce grain yields substantially or even totally (Roelfs et al. 1992; Chen 2005). Leaf rust, caused by *Puccinia triticina* Eriks., is a wheat disease of major historical and economical importance worldwide (Saari and Prescott 1985; Samborski 1985; Roelfs et al. 1992). It is the most widespread of the three types of wheat rusts. The other two are stem rust, caused by *Puccinia graminis* Pers.:Pers. f. sp. *tritici* Eriks. & Henning, and stripe rust, caused by *Puccinia striiformis* West. f. sp. *tritici* Eriks. & E. Henning (Singh et al. 2002; Chen 2005).

Historically, cereal rust diseases were clearly of major significance but estimation of yield losses received attention only in the 20th century due to a better understanding of disease biology and increasing need to appraise economically the financial investment in control programs (McIntosh et al. 1995).

In the USA, in epidemic years from 1918 to 1976, yield reductions due to wheat stem rust were of 50% or more. Wheat stripe rust was more restricted in distribution, even though losses up to 70% in commercial fields were recorded (Roelfs 1978). Wiese (1977) reported that during the 1960's the rusts were estimated to have reduced North American wheat yields by over 1 million tones (2%) annually.

In Australia, estimation of crop losses due to wheat stem and leaf rusts varied from 30% in leaf rust-susceptible cultivars (Rees and Platz 1975) to 55% in wheat susceptible to both stem and leaf rust (Keed and White 1971). A widespread leaf rust epidemic in Western Australia in 1992 caused yield losses of up to 37% in susceptible cultivars with average losses of 15% across many fields. Economic evaluations of financial losses have also ranged from A$ 100-200 million due to the 1973 stem rust epidemic to an estimated A$ 8 million cost of chemical application for disease control during an epidemic of stripe rust in 1983 (Wellings and Luig 1984; McIntosh et al. 1995). An economic analysis of losses due to a range of wheat diseases in Australia (Brennan and Muray 1988) estimated that the annual value of control strategies for stem, stripe and leaf rust was A$ 124, 139 and 26 million, respectively.

In South America, leaf rust is currently the most prevalent and severe wheat disease. A very high proportion of the wheat area is planted with susceptible or moderately susceptible cultivars, allowing widespread local oversummering and early onset of epidemics in the growing season. Losses caused by leaf rust can be over 50% in severe epidemics if fungicides are not used. Losses estimated at US$ 50 million (Germán et al. 2007). Stripe rust is more important in the wheat area in southern Chile, where a severe epidemic occurred in 1940. During 1976-1988 stripe rust caused economic losses at least once every 2 years (Andrade Vilaro 1990). Although stripe rust infections have not caused major concern over the past few years, an early epidemic in 2001 affected several spring cultivars (Germán et al. 2007). Incidence of stem rust historically has been more sporadic, but it caused higher levels of damage during severe epidemics. It was considered the most destructive wheat rust in Brazil, Paraguay, Uruguay, the northern wheat growing area of Chile and the northern and central north wheat area of Argentina. A very severe epidemic occurred in Argentina and other southern American countries in 1950 (Antonelli 2000). During 1975-1976 widespread epidemics occurred in Brazil, Argentina and Uruguay, even under unusually unfavorable environmental conditions. For over 2 decades wheat stem rust has not been severely epidemic, but during 1975-2003, 2 stem rust epidemics were observed in Brazil and localized epidemic outbreaks occurred during the 1990’s on some widely grown cultivars in Paraguay (Germán et al. 2007).

Severe epidemics of cereal rusts have been recorded in India since the early 1800’s (Joshi 1976). In Egypt, crop losses due to leaf rust infection were as high as 50% (Abdel Hak et al. 1980). In Europe, stripe rust and leaf rust are mostly associated with cereal rust losses. Priestley and Bayles (1988) estimated that losses in susceptible winter wheat varieties due to stripe and leaf rust were of £83 million in 1984 in the USA, £130 million in the UK and £175 million in Australia. In China, winter wheat production also is affected by recurrent epidemics of stripe rust. Epidemics in 1950, 1964 and 1990 were estimated to have caused losses of 6, 3 and 2.5 tones, respectively (McIntosh et al. 1995).

In the late 1980s, a new race of stripe rust evolved in eastern Africa and migrated to South Asia through Middle East and West Asia in about 10 years, causing severe epidemics and crop losses over US$ 1 billion in its migration path (Singh et al. 2004). In 2007 and beyond, the world could be facing an even more devastating situation with the outbreak of stem rust race Ug99 in eastern Africa. Race Ug99 was initially detected in Uganda in 1999, then in Kenya in 2002-2003 and soon after in Ethiopia in 2003-2004. Because most of leading cultivars and lines tested in Kenya and Ethiopia are susceptible severe epidemics have been reported (Figs. 2, 4). The migration of this new race to neighboring areas and beyond has been motive of great concern (Expert Panel on Stem Rust Outbreak in Eastern Africa 2005). Damages could total US 1-2 billion in Asia alone, based on a 10% yield loss estimate and depending on market price. In response to this new threat to food security, a Global Rust Initiative was launched in 2005 led by CIMMYT (International Wheat and Maize Improvement Center) in partnership with ICARDA (Center for Agricultural Research in the Dry Areas) and various National and Advanced Research Institutions (Singh et al. 2006; Duveiller et al. 2007).

**Symptoms and epidemiology**

**Leaf rust**

The wheat leaf rust is characterized by uredia containing masses of urediospores of brown-orange color, which appear at the rupture of the leaf epidermis and usually have an elliptic format, and, in some cases are surrounded by a chlorotic halo (Fig. 3). The symptoms are randomly distributed on both parts leaf and leaf sheet mainly, although in years of severe epidemics they can be found on any green part of the plant. In the end of the crop cycle, when the plant reaches maturity and the environment becomes more unfavorable, the pustules start to produce teliospores in replacement of the urediospores. These spores are two-celled, have thicker walls and darker color (Samborski 1985).

The main source of initial inoculum of *P. triticina* in each crop season is wheat plants close to the area or volunteers plants, which form the so-called “green bridge”. The primary alternative host (Thalictrum speciosissimum) does not have an important role in the production of initial
inoculum per se, but in generating genetic variability in the population of races, because the sexual stage of the pathogen occurs on it. However, in South and North America as well as in Australia, there are not reports that the alternative host has an important epidemic function (Singh et al. 2002).

Epidemics of wheat rusts, as well as other diseases, are affected by the three components of the “triangle of the epidemic”: pathogen, host and environment. In areas where the rust inoculum is always present and susceptible cultivars are planted, the environment is the key factor to influence the occurrence and the severity of epidemics (Chen 2005).

The environmental conditions for the survival of P. triticina are the same as those required for the survival of the host leaves. The fungus can cause infection within a period of three hours of foliar wetness or less, at temperatures of about 20°C, but most of the infections require longer periods of wetness. At lower temperatures, the period of wetness is even longer. For instance, at 10°C 12 h of surface wetness is necessary for infection to take place. At temperatures higher than 32°C or lower than 2°C little or no infection occurs (Stubbs et al. 1986). The urediniospores germinate after 30 min of contact with free water, at temperatures between 15 and 25°C. At higher and constant temperatures (25°C), sporulation can occur within 7-10 days after urediniospore germination and penetration of the fungus, which takes place through the stomata. At low temperatures (10-15°C) or with varying diurnal temperatures, longer periods are necessary. The maximum sporulation occurs about four days after the initial sporulation, at temperatures around 20°C. A single uredium can produce up to 3000 spores a day, and it can continue to produce them for 3 weeks or more, according to the leaf condition (Singh et al. 2002). The urediniospores can be disseminated by wind over long distances (Hirst and Hirst 1967). Under unfavorable conditions or when the plant starts to senesce, teliospores are formed under the epidermis, which stay adhered to plant tissues and can be disseminated to great distances by the wind, animals or human action (Roelfs et al. 1992). In South America teliospores do not have an epidemiological function, since they do not find the alternate host and are not able to re-infect wheat.

**Stem rust**

Stem rust attacks mainly the stems and leaf sheets (Fig. 4A), but, like leaf rust, it can infect other parts of the plant, such as leaves and glumes (Fig. 4C, 4D), in years of severe epidemics. The epidermis is drastically broken by the uredia, giving a rough aspect to the surface of the affected tissue. The uredia produce urediniospores with brown-red color, and usually are more prolonged and larger than the leaf rust,
reaching up to 10 mm in length (Fig. 4B). As the wheat matures, the uredospores are replaced by teliospores (Leonard and Szabo 2005).

The epidemiology of *P. graminis* f. sp. *tritici* is similar to that of *P. triticina*. However, the range of temperatures low, optimum and maximum requested for the germination of the spores (2, 15-24 and 30°C, respectively) and sporulation (5, 30 and 40°C, respectively) are higher. This characteristic makes the stem rust more important at the end of the crop season, for varieties with longer cycles or in late-sowing areas. Wheat stem rust also differs from leaf rust for requesting a larger wetness period of the tissue (6 to 8 h). Besides, high light intensity for a period of three hours is essential for penetration, and maximum infection is obtained with 8 to 12 h of wetness at 18°C followed by light intensity equal or superior to 10,000 lux and 30°C (Rowel 1984). A single uredinium of stem rust can produce 10,000 spores a day (Katsuya and Green 1967). Although the productivity of spores for pustules is larger than leaf rust, the infectivity is smaller, since only 1 out of 10 spores germinate and have success in the infection. The uredia usually survive for longer periods than leaf rust, however, the rate of development of both diseases is similar. The urediniospores of *P. g. f. sp. tritici* are very resistant and can be disseminated by the wind to distances up to 8000 km (Singh et al 2002). The main alternative host of *P. g. f. sp. tritici* is the bush *Berberis* spp. This host is the largest source of primary inoculum in North America being also responsible for the generation of genetic variability in the population of races of the pathogen in that area (Roelfs 1982; Peterson et al. 2005). In South America there are no reports of its occurrence.

**Stripe rust**

Stripe or yellow rust is characterized by uredia distributed in lines or stripes that extend along the leaf between the vascular bundles (Fig. 5). Besides the non random distribution pattern, the uredia of stripe rust also differ from other wheat rusts for being smaller and for producing yellow-clear urediniospores. Similar to leaf and stem rusts, in years of severe epidemics stripe rust uredia can also affect other green parts of the plant. As the plants mature the production of urediniospores ceases and is substituted by teliospores (Chen 2005).

Of the three rusts that attack wheat, *P. striiformis* is the one that requires lower temperatures, and, for this reason, it occurs in areas of temperate climate and in high lands in areas of tropical climate. Due to this characteristic, the disease can begin very early in the crop season, and in these cases, can cause more severe damage than leaf or stem rusts, which require higher temperatures for their development (Chen 2005). The low, optimum and maximum temperatures for pathogen infection are 0, 11 and 23°C, respectively (Roelfs et al. 1992; Singh et al. 2002). Similarly to leaf rust, the urediniospores of stripe rust need a wetness period of 3 h for the germination and infection to take place (Chen 2005). Although the urediniospores of stripe rust are three times more sensitive to ultraviolet radiation than those of stem rust (Singh et al. 2002), their dissemination by the wind at a distance of 2000 km, from Australia to New Zealand, was reported (Beresford 1982). The urediniospores are the only source of inoculum and, in most areas of the world where the disease occurs, the initial inoculum seems to be originated from volunteer wheat plants in the same or in neighboring areas (Line 1976; Stubbs 1985; Zadoks and Bowman 1985).

**BARLEY RUSTS**

**Distribution and economic importance**

Barley (*Hordeum vulgare* L.) is the world’s fourth most important cereal crop after wheat, maize and rice. Barley has the widest geographical range of any cultivated crop plant – from the equator to the Arctic Circle – and is grown over a broader environmental range than any other cereal. It has persisted as a major cereal crop for many centuries because of three unique characteristics: broad ecological adaptation, utility as a feed and food grain, and superior malt for brewing beer. The main producing countries are Canada, Spain, Turkey, United States, Germany, Morocco, France, Ukraine, Algeria, Ethiopia and Tunisia (Brown Jr. et al. 2001; Wodde et al. 2007).


The disease caused on barley depends on the developmental stage of the plant and is at the moment of infection. The epidemics that happen before or during the flowering period cause the largest reductions in yield. The rusts increase the respiration and transpiration of the plant, reducing photosynthesis, vigor and growth of roots, besides the production of withered grains (Mathre 1985). In some areas, a leaf rust pathogen (*P. triticina*) can infect barley too, but it is considered a weak pathogen (Wahl et al. 1984; Mathre 1985).

Barley leaf rust is a widespread disease responsible for great yield losses (Whelan et al. 1997; Kicherer et al. 2000; Das et al. 2007). Griffey et al. (1994) reported damage in yield up to 30-40%, depending on the resistance level of the cultivated variety and Das et al. (2007) observed yield reductions of 33% in plants not treated with fungicides, for the susceptible cultivar ‘Wysoy’. The disease results in smaller yield of grains because it reduces the number of tillers in 31% and weight of grains in 21% (Whelan et al. 1997).

Crown rust is considered a recent disease in barley. The pathogen is a variant of *P. coronata* Corda. However, the uredial and telial stages are morphologically distinct from the rust caused by this pathogen on *Hordeum* spp. and *Festuca* spp. The exact taxonomic position of the pathogen is under investigation (Jin et al. 1992; Jin and Steffenson 1993). The first damages caused by the disease were reported in Nebraska (USA) in 1991. Currently, it is widespread in many regions of North America, mainly in the mid-west. The alternative host is *Rhamnus cathartica*, a bush that grows abundantly in the areas where this pathogen occurs (Jin et al. 1992; Jin and Steffenson 1997; Jin and Steffenson 1999; USDA-ARS 2008).
Since the first report of the disease in 1991, it has occurred every year with different intensities. In some years, there were only traces of the disease, while in others, severities of up to 60% have been reported (Long et al. 1996; Jin and Steffenson 2002). The damages caused by *P. coronata* var. *hordei* in barley have not yet been estimated (Jin and Steffenson 2002). However, there is the possibility that it causes large yield losses, since it can start very early in the crop season due to the presence of the alternate host and consequently the initial inoculum, damaging the quality of fodder and/or grain filling.

Barley stem rust is a disease of sporadic occurrence but in years with favorable weather conditions can cause large yield decreases, as the infected plants produce withered grains. The intensity of the damage is proportional to the yield decrease, as the infected plants produce withered grains. The time of occurrence of the disease. The earlier the disease occurs, the greater the severity will be and consequently damage to the seeds. Generally when environmental conditions are not favorable to disease development, control is relatively easy. The fungus causing barley stem rust is *Puccinia graminis* and there are few specialized forms for barley, which is infected by forms that also attack wheat, rye, triticale and timothy-grass. The most common forms are *P. graminis* f. sp. *tritici* and *P. graminis* f. sp. *secalis* (the rust species attacking wheat and rye). Their occurrence depends on the region, although the *formae speciales* of wheat seems to be more important on barley. Epidemics of stem rust on barley are often associated with epidemics of stem rust on wheat or rye nearby (Matthe 1985; Martens et al. 1988). The alternate hosts are *Berberis vulgaris*, *B. canadensis* and *B. fendleri*, as well as some species of *Mahonia* spp. (USDA-ARS 2008).

Barley stripe rust, also known as yellow rust, can cause yield damage of up to 50% and is common under favorable conditions and lack of chemical treatment. Marshall and Sutton (1995) reported 72% in yield losses in a susceptible cultivar in the USA. The quality of malt is impaired under conditions of severe infection (Line 2002). There are no reports of the occurrence of alternate hosts and sexual stage. However, the pathogen’s host range is much broader than the crop can reduce its occurrence. Low altitude (<2500 m) is important because they contribute to increase the inoculum of the host. The disease develops quickly at temperatures above 30°C (USDA-ARS 2008).

### Symptoms and epidemiology

#### Leaf rust

The symptoms of barley leaf rust are characterized by the presence of circular pustules that produce a mass of orange spores, the urediniospores, predominantly in the upper surface of leaves (Fig. 6). It can occur on both leaves and sheaths. The penetration of the stomata occurs via an apressorium and penetration peg. The formation of substomatal vesicles, hyphae and intercellular haustoria, with a knob or globular form, complete the process of infection. This process takes around 6-8 h and the production of secondary inoculum from the first infection occurs in about 10 days (Matthe 1985). The telia produced at the end of the season are less abundant that the uredia, possess a circular or oblong format, dark color and remain covered by the epidermis of the host. In countries where the alternate host is not present their germination, if occurs, is not a source for new infections (Matthe 1985; Hollaway and Horsham 2008a).

The life cycle is complex and involves the alternate host *Ornithogalum umbellatum* L., known at the Star of Be host. Other species of *Ornithogalum* can also be alternate hosts, allowing the formation of picnia and aecia. However, aecial infection has only reported in some parts of Asia and around the Mediterranean Sea (Matthe 1985).

The main sources of initial inoculum in regions of barley production are urediniospores. These are spread to long distances and can remain from one season to another on voluntary plants. The disease develops quickly at temperatures between 15 and 22°C, when the humidity is not a limiting factor. The optimum temperature is around 18 and 20°C and lower temperatures to 18°C reduce the chances of occurrence of epidemics (Matthe 1985; Woldeab et al. 2007).

The system of production affects the occurrence, development and percentage of damage caused by leaf rust on barley. The fertilization with nitrogen can increase the occurrence of rust as the absence of rains at the end of the crop can reduce its occurrence. Low altitude (<2500 m) may increase the disease intensity, while altitudes over 2500 m are associated with its reduction. Thus, the effect of the disease may be influenced by cultural practices and the environment in which the barley is grown (Fekadu 1995; Woldeab et al. 2007).

#### Crown rust

Crown rust are characterized by orange linear pustules, usually associated with chlorosis, occurring mostly on the blade of leaves and occasionally on sheaths, stems and own. The name “crown rust” is due to the appearance of teliospores, which have appendices on the apex conferring a crown aspect. The telia are linear, dark and covered by the epidermis. The remaining teliospores on barley debris and other susceptible cereals germinate and produce basidiospores that infect the alternate host, *Rhamnus cathartica*, where pyenial and aecial stages occur. The aeciospores produced on *R. cathartica* are the primary source of inoculum for barley and the infection can occur before the extrusion of the third leaf. The spores are disseminated by wind over long distances, from both *R. cathartica* or from barley (Jin et al. 1992; Szabo 2006; USDA-ARS 2008).

Crown rust develops at temperatures around 20 to 25°C during the daytime and between 15 to 20°C at night, associated with high humidity. The urediniospores need a film of free water on the surface of the leaves to germinate. Penetration occurs via stomata, through apressoria at temperatures between 10 and 25°C and the infection is inhibited at temperatures above 30°C (USDA-ARS 2008).

In addition to barley, *P. coronata* var. *hordei* can infect rye, wheat and other wild grasses, like *Elytrigia repens*, *Elymus trachycaulus*, *Pascopyrum smithii*, *Hordeum jubatum*, *Elytrigia spp.*, *Elymus spp.* and *Leymus spp.* Also, it infects some species of *Brachypodium*, *Bromus*, *Festuca*, and * Lolium*, from the tribe *Poaeae*, and *Phalaris* from the tribe *Aveneae*. All these species are epidemiologically important because they contribute to increase the inoculum of *P. coronata* (Jin and Steffenson 1999, 2002; USDA-ARS 2008).

Fig. 6 Symptoms of *Puccinia hordei* (leaf rust) on barley first leaves.
Stem rust

The pustules are large, with an oval format, red and usually surrounded by a broken epidermis and occur on stems and on leaf sheaths, but can also attack leaves, glumes and awns. At the end of the season pustules produce teliospores of dark color, which remain on the dead tissues. The alternate hosts, where the aecial and pycnial stages occur, are species of Berberis (B. vulgaris, B. canadensis Mill. and B. fendleri) and some species of Mahonia. Conditions of high temperatures and humidity favor the occurrence of the disease. The optimum temperature for development is 20°C, temperatures below 15 and above 40°C are limiting to the occurrence of the disease. Thus, the disease is of greater economic importance in areas where such conditions are present throughout the crop season (Mathre 1985; Hollaway and Horsham 2008a, 2008b).

Stripe rust

Symptoms of stripe rust usually occur earlier than leaf and stem rust in the crop season. The pustules are yellow and occur mainly on leaves and spikes, often linearly and parallel to the vascular bundles, except at the seedling stage, where the pustules occur randomly. The pustules quickly cover the leaves and often the spike, leading the plant to become dry. The germ tubes can penetrate directly into the stomata, without needing to form appressoria and a penetration peg. At the end of the season, dark teliospores, possessing a short period of dormancy, are produced. The basidia are not functional, since no alternative host is known (Mathre 1985; Brown Jr. et al. 2001). The inoculum can be successfully disseminated by more than 800 km from the point of origin (Zadoks 1965).

The mycelium remains viable at temperatures of up to -5°C, and the optimum temperature for germination of urediniospores is from 5 to 15°C, with limits between 0 and 21°C. The development of the disease is faster between 10 and 15°C with moisture available. The moisture is the limiting factor for occurrence of the disease since the urediniospores need at least 3 h of humidity close to saturation in order to induce germination and infection in barley. In the presence of dew and temperatures between 2 and 15°C germination and formation of appressorium occur. Germination is hampered by periods of dissection occurs. The latent period of pustules is also dependent on temperature, where temperatures between 0 and 10°C inhibit it but do not stop the development of the fungus. Since the fungus is established in the plant, it can withstand high or low temperatures without being killed (Mathre 1985; Adams and Line 1997; Brown Jr. et al. 2001; Line 2002; Yan and Chen 2006).

OAT RUSTS

Distribution and economic importance

Oat (Avena sativa L.) is an important grain crop in most countries where small grain cereals are cultivated such as Australia, Europe, North and South America. In the subtropical to temperate climate of South Hemisphere, oat is grown in the winter and is used as a winter cover crop, food for both livestock and humans, and as raw material for industrial uses. It is the third most important winter crop in southern Brazil (Leonard and Martineili 2005). In all these regions, two rust diseases are of major concern for oat crop management: crown rust, which is caused by Puccinia coronata f. sp. avenae P. Syd. & Syd., a heterogeneous macrocyclic rust (Dinnor et al. 1988) and stem rust, caused by Puccinia graminis Pers. f. sp. avenae Eriks. & Henning. Both attack all species of oats, including wild oats (Wallwork 1992) and are well recognized diseases, occurring almost everywhere these crops are grown (Zillinsky 1983).

Crown rust is the most harmful oat disease and is distributed worldwide. Its occurrence is common in all areas where these crops are cultivated (Simons 1985; Malvick 1989; Martineilli 2004). As a consequence of the induced physiological stress, grain yields are negatively correlated with crown rust severity. Even small amounts of disease can reduce yield significantly, close to 100 kg per percentage unit of severity at the tillering stage (Martineilli et al. 1994). The damage is more pronounced at the earliest stages of crop development. The economic threshold of the disease is very low, around 15-20% of foliar incidence or 0.2 to 0.3% of plant severity. Martineilli et al. (1994) observed on susceptible cultivars grown under field conditions up to 90% of disease severity and yield reductions from 24 to 50%. Chaves et al. (2002) also observed reductions close to 80% in some cultivars in South Brazil, but the largest reduction of 95% was registered by Martineilli and Buss (1999) on cv. ‘UPF16’. These losses are consequence of the aggressive nature of the pathogen population, the lack of effective resistance genes and the favorable environmental conditions for long periods.

Crown rust occurs almost every year in some provinces in Canada, such as Ontario, Quebec, Manitoba and eastern Saskatchewan, causing severe crop losses (Martens et al. 1988). In Australia it can be devastating, reaching crop losses up to 100% (Wallwork 1992). In South America, stem rust epidemics are typically at the end of the crop season, from the booting stage onwards, when the temperature is warmer. In Argentina, where Avena sativa is also used as forage, stem rust is particularly important because its cycle is longer than that in Brazil, where the disease has its importance diminished because oats are harvested earlier (authors’ pers. obs.).

Symptoms and epidemiology

Crown rust

Oat crown rust symptoms appear as yellow pustules containing masses of urediniospores, which are exposed after the rupture of the epidermis (Fig. 7). These lesions are circular or oblong and they occur on both surfaces of the foliar sheet, and can reach other green parts of the plant, when the epidemic becomes more severe. After some weeks, the borders of the uredia can turn black, with the formation of teliospores. When the infected plants reach maturity, the production of urediniospores ceases and are replaced by the teliospores (Browning 1973; Simons 1985; Harder and Haber 1992).

The primary infections are caused by urediniospores or aciospores. In areas of subtropical and temperate climate, where oats grow during winter, the urediniospores from volunteer plants that survive summer are usually responsible for the primary infections of plants sown in autumn. The urediniospores and aciospores of P. c. f. sp. avenae are spread widely by wind and can reach long distances. Their germination needs free water on the leaf surface and the infection occurs through the stomata. These two processes are favoured by temperatures between 10 and 25°C. Temperatures above 30°C inhibit the infection process (Simons 1985).

Prevailing wind patterns usually distribute urediniospores of P. coronata in a cyclical pattern throughout the oat-growing regions of Brazil, Argentina, and Uruguay as a shared epidemiological system. Epidemics typically start early; sometimes while Avena spp. is still at the tillering stage of development. Avena strigosa is less susceptible than A. sativa, but rust severities of 5-10% are common during the crop season (Leonard and Martineilli 2005). In Europe and in North America, the alternate hosts, Rhamnus spp. are an important source of inoculum for the oats, since they contribute to the great variability observed in the pathogen population through sexual recombination. The telio-
spores on the infected straw that remain from the previous summer germinate in the spring producing basidiospores, which in turn infect young leaves of Rhamnus. These infections produce aecidia from which aeciospores arise and then infect the oats. On the other hand, in South America teliospores of *P. coronata* have no known survival function (Martinelli 2000).

### Stem rust

Oat stem rust symptoms most commonly appear on the stems and leaf sheaths, but leaf blades and spikes may also become infected. Urediniospores develop in pustules (uredia) that rupture the epidermis and expose masses of reddish brown spores (Fig. 8). The pustules are larger than those of crown rust, oval shaped or elongated, with loose or torn epidermal tissue along the margins. They may appear on both surfaces of the leaf. They continue to be produced until the plants approach maturity. After that, teliospores develop, either in the same uredia or in different fruiting structures called telia (Martinelli 2004).

Epidemics are more probable to occur when temperatures are warm (15-30°C) and conditions moist (Wallwork 1992). Stem rust develops its sexual stage on Berberis vulgaris L. In North America the disease is usually not as widespread as crown rust, probably because the alternate host is less common than *Rhamnus cathartica* (Harder and Haber 1992). In the absence of the alternate host (South America and Australia) its distribution and epidemiology follows the same pattern as that of crown rust (Leonard and Martinelli 2005).

### RYE RUSTS

Rye (*Secale cereale* L.) is second only to wheat among the grains most commonly used in the production of bread. Rye is also a highly versatile crop. As a green plant, it is used as livestock pasture and as green manure in crop rotations; as grain, it is used for livestock feed and as feedstock in alcohol distilling; and as flour, it is used in breads and many other baked products. Rye flour can be used alone to produce “black” bread, which is consumed extensively in Eastern Europe and parts of Asia. Rye is the acknowledged trademark of Canadian whiskey. In Argentina, it is an important pasture crop, and in southern Australia, it is planted to prevent wind erosion. Rye is also a particularly important crop in Poland, Germany, and three republics of the former Soviet Union. Although rye is inferior in several ways to the predominant cereal crops (wheat, rice, and maize), it will continue to be an important crop for farmers in many countries because of its winter hardiness and ability to grow in poor soils and because of consumer demand for baked products with rye flour (Bushuk 2001). Rye can be infected by three different types of rusts: leaf rust, caused by *P. recondita* f. sp. *secalis* Robereger, crown rust, caused by *P. coronata* var. *hordei* Jin & Steff. and stem rust, caused by *Puccinia graminis* f. sp. *secalis* Erikss. & Henning.

### Leaf rust

Rye leaf rust is widely distributed in the regions where rye is grown. The pustules are small, circular, and orange-colored, and are distributed on the leaves and sheaths. As the plants mature, the pustules become dark in color due to the development of telia. The alternate host is *Lycopsis arvensis* (Wehling et al. 2003; USDA-ARS 2008). Yield losses can reach up to 40% under natural conditions of infection. Reductions on the 1000-seed weight can reach up to 27% (Miedaner and Sperling 1995; Wehling et al. 2003).

### Crown rust

*P. coronata* f. sp. *hordei* is responsible for crown rust on barley and rye, but differs from other reported forms of *P. coronata*, in both morphological and pathological terms. The most marked characteristics are the teliospores with elongated and branched appendages, in addition to the broad pathogenicity to the tribe Triticeae. The pustules of crown rust on rye are similar to those found in barley. They are elongated, bright orange and often associated with chlorosis. They occur mainly in the leaf blades and can appear on sheaths, stems and own. The telia are brown and remain covered by the epidermis. The alternate host is *Rhamnus cathartica* (Jin and Steffenson 1999; USDA-ARS 2008).

### Stem rust

Rye stem rust, *Puccinia graminis* f. sp. *secalis*, in addition to rye infects barley and *Agropyron repens* (Wahl et al. 1984; Martens et al. 1988). It is considered the most important disease of the crop in Brazil. Its cycle is comparable to wheat stem rust. The plants initially show discoloration on the infected tissues. With the progress of the disease, pustules appear brown with a format that varies from oval to elongated, alone or in clusters, on stem, leaf sheaths and leaves. The spores of the fungus are spread mainly by wind and survive on volunteer plants (Wahl et al. 1984; Martens et al. 1988; Nascimento Jr. et al. 2005). The alternate hosts are *Berberis vulgaris* and *B. canadensis* (USDA-ARS 2008).

### CONTROL

Understanding the epidemiology of a disease is essential before starting any control strategy, especially one invol-
vancing cultural and chemical measures. The most effective means of controlling the cereal rusts is the combination of cultural control practices with disease resistance and perhaps fungicide applications. No single practice is effective under all conditions, but using a series of cultural practices greatly enhances the existing resistances. Because of the airborne nature of the inoculum of cereal rusts, quarantine methods against the pathogen only delay, and do not prevent entry of the diseases and/or specific virulence combinations. However, care must be taken not to permiturediospores 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 (Singh et al. 2002).

Genetic resistance

One of the first demonstrations of the possible genetic manipulation of plant disease resistance occurred in 1905, when Biffen (1905) showed that resistance in wheat cultivars to stripe rust was simply inherited. Since then, host resistance (Fig. 9) has become one of the primary control methods of plant diseases and a priority objective of plant breeding (Byerlee 1996). This form of control is relatively inexpensive for plant producers to implement and is reported to be more environmentally friendly than any other control strategies (Bockus et al. 2001).

Varieties can carry different types and levels of leaf rust resistance. With the discovery of the genetic basis of resistance (Biffen 1905), physiological specialization in rusts (Stakman and Levine 1922), and the gene-for-gene hypothesis (Flor 1956) the utilization of race-specific resistance has dominated in wheat improvement. Particularly for the cereal rusts, race-specific resistance genes often break down with exposure to the rust pathogen, contributing to a “boom-bust” cycle of resistance and associated genetic vulnerability because of shifts in the pathogen population (Smale et al. 1998). The likelihood of resistance breakdown increases considerably if these race-specific resistance genes are the only source of resistance in cultivars released to farmers. Race-specific resistance genes typically breakdown within 5 years of deployment (Singh and Huerta-Spino 2001).

Race-nonspecific, also known as slow rusting resistance, proposed by Vanderplank (1963) and applied to leaf rust resistance by Caldwell (1968) has been the dominant method used by CIMMYT’s wheat-breeding programs. Genes conferring slow rusting resistance to leaf rust in wheat have partial and additive effects, and although the response to infection is essentially susceptible, the rate of disease progress is slowed. Geneticists and pathologists at CIMMYT believe that adequate levels of nonspecific resistance can limit disease losses to insignificant levels in farm fields and is more likely to endure many crop seasons (Johnson 1984; Smale et al. 1998). This long-term resistance is extremely relevant to the developing world, where many countries do not yet have efficient seed production and variety replacement capacity (Trenbath et al. 2005). Research at CIMMYT indicates that over the past few decades the impact of breeding for genetic resistance has generated a large proportion of the global economic return to investment in international wheat research (Byerlee and Traxler 1995; Reynolds and Bolarga 2006). For example, the benefits of incorporating nonspecific resistance to leaf rust caused by Puccinia triticina into modern bread wheat have been estimated using data on resistance genes identified in cultivars, trial data and area sown to cultivar in Yaqui Valley, Sonora State, Mexico. In the most pessimistic scenario, the gross benefits generated from 1970 to 1990 were US$ 17 million (in 1994 real terms). Even when costs were overstated and benefits were understated, the internal rate of return on capital invested was 13%, well within the range recommended for use in project evaluations by the World Bank (Smale et al. 1998). The economic benefits of maintenance research are also reported as large by Marasas et al. (2003). The authors indicate that the internal rate of return on CIMMYT’s research investments in breeding for leaf rust resistance spring bread wheat was estimated at 41% and the net present value (discounted at 5%) at US$ 5.36 billion (at 1990 dollars), with a benefit-cost ratio of 27:1.

Genetic resistance to wheat rusts

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. Today, official (Sr followed by a number) or provisional (Sr followed by letters) symbols have been described for about seventy wheat stem rust genes (McIntosh et al. 2003; USDA-ARS 2008). Green and Campbell (1979) summarized the success of the Canadian stem rust programme. In Australia, a series of cultivars with Sr26 have been released since 1971 and have grown on nearly 1 million ha without stem rust losses (Luig and Rajaram 1972). The adult plant resistance gene Sr2 derived from Hope results in an absence of uredia in the internode tissues (Hare and McIntosh 1979; Sunder- wax and Roelfs 1980). This has been probably the most commonly used Sr resistance gene worldwide since the 1940s. The 1BL.1RS wheat-rye translocation is associated with Sr31, Lr26 and Yr9. Gene Sr31 provides a highly to moderately effective resistance to stem rust and was effective worldwide until 1999, with the outbreak of stem rust race Ug99 in eastern Africa, which overcome the resistance provided by this gene. The strategies to reduce the possibilities of major epidemics include monitoring the spread of race Ug99 beyond eastern Africa, massive testing of advanced lines in East Africa accompanied by an emergency crossing program to achieve satisfactory levels of resistance. Up to now, a few wheat genotypes that combine resistance and high yield potential have been identified but need rigorous field testing to determine their adaptation in target areas (Singh et al. 2006; Duveiller et al. 2007).

The genes for wheat stem rust resistance have been obtained primarily from cultivars of T. aestivum, but some are from other Triticum spp. as well as from Triticeum (Aegilops), Secale (rye) and Agropyron, but the usefulness or durability of resistance does not seem to be associated with the donor genera or species (Singh et al. 2002). Up to now, about 70 official (Lr followed by a number) or provisional (Lr followed by letters) gene symbols have been catalogued for resistance to wheat leaf rust (McIntosh et al. 2003; USDA-ARS 2008). Although virulence occurs for a majo-

Fig. 9 Wheat genotypes showing susceptibility (left) and resistance (right) to Puccinia triticina (leaf rust).
rity of race-specific resistance genes, a few historical cultivars, such as ‘Americano 25’, ‘Americano 44d’, ‘Surpreza’, ‘Frontana’ and ‘Fronteira’ (Roelfs 1988; Perez and Roelfs 1989) have maintained some resistance for many years. Several spring wheat cultivars developed by CIMMYT have shown a slow rusting type of resistance (Rajaram et al. 1996). Gene Lr34 together with other unnamed slow rusting genes is believed to be involved in the durable resistance of ‘Frontana’ and other wheats (Singh and Rajaram 1992). Slow rusting resistance of ‘Pavon 76’ involves Lr46 in combination with two unnamed slow rusting genes (Singh et al. 1998; William et al. 2005). Slow rusting to wheat leaf rust may also be based in the Lr22b, Lr35, Lr37 resistance genes and in the combination of Lr12 + Lr 34 (Dyck 1987; Roelfs 1988; Kerber and Dyck 1990; German and Kolmer 1999).

Biffen (1905) first demonstrated that resistance to stripe rust in wheat follows Mendel’s laws. Seventy genes with official (Yr followed by a number) or provisional (Yr followed by letters) symbols have been reported (Chen 2005). Most of the known genes confer all-stage resistance, but many of them have been described as temperature sensitive and/or adult plant types (Lewellen et al. 1967; Sharp and Volin 1970; Robbelen and Sharp 1978; Wallwork and John-son 1984; Singh and Rajaram 1994). Some of these resistances are considered non-specific and multiple resistance alleles have been reported for the Yr3 and Yr4 loci (Lupton and Mader 1962; Chen and Line 1993; Chen et al. 1996).

In Europe, the most durable resistance has been that of ‘Cappelle-Desprez’ (Yr3a, Yr4a, Yr6) (Johnson 1981), ‘Juliana’ (Yr14+, Yr15) ‘Carstens VI’ (Yr12+, Yr1) and ‘Arminda’ (Yr13, Yr14+) (Stubbs 1985). In the United States, the cultivars ‘Gaines’ and ‘Nugaines’ have provided resistance on a long-term scale (Line et al. 1983). Some CIMMYT germplasm-derived cultivars, such as ‘Anza’ (Yr18) and ‘Pavon 76’, also have durable resistance (Singh and Rajaram 1994). Slow rusting gene Yr18, in combination with other unnamed slow rusting genes, is currently believed to be involved in durable resistance of several spring and winter wheats (Mcintosh 1992; Singh 1992).

Genetic resistance to barley rusts

The genes conferring resistance to barley leaf rust are designated by the Rph symbol and the majority of these genes acts in a gene-for-gene system, resulting in hypersensitivity reaction. Nineteen Rph genes have been described and mapped in H. vulgare and H. spontaneum (Martens et al. 1988; Weerasesa et al. 2004) but virulence is reported for almost all of them (Roelfs et al. 1990; Jin et al. 1993; Sief- fenson et al. 1993; Kicherer et al. 2000; Shtaya et al. 2006). Some barley lines carry unknown hypersensitivity genes effective to many European isolates (Niks et al. 2000), and many of them also carry some levels of slow rusting resistance (Shtaya et al. 2006).

Durable resistance to barley stem rust has been conferred by the Rpg1 gene for over 60 years (Brueggeman et al. 2002; Horvath et al. 2003; Zhang et al. 2006). The efficiency of this gene seems to be increased by the presence of other genes such as Rpg3, Rpg4 and RpgU (Jedel et al. 1989; Harder and Leggo 2000).

At least 26 different resistance genes were described for barley stripe rust in 18 barley lines (Chen and Line 2003). Among these lines, ‘Grannenlose Zweizeilige’, a cultivar from Ethiopia, has a recessive gene, temporarily designated as rpgGZ, conferring resistance to all races of P. striiformis identified in the USA (Yan and Chen 2006).

Genetic resistance to oat rusts

The sources of resistance to oat crown rust are found in diploid, tetraploid and hexaploid oat species, being A. sterilis one of the major sources of genes (Harder and Haber 1992). Resistance genes from two distant graenecious species (Avena sp. and Lolium sp.) and effective against two different formae speciales of P. coronata seem to be orthologous, what indicates that sources of resistance to crown rust can be identified by colinearity (Sim et al. 2007). Most of the race-specific resistance genes to crown rust were transferred to cultivated oats from A. sterilis, from accesses collected in Israel and other Countries in the Mediterranean region during the 1960’s and 1970’s (Leonard et al. 2004). However, the genetic base for rust resistance is very narrow and there is no single gene effective to all races. Amongst the crown rust resistance genes known, 97 are race-specific, named Pc genes (Pc1 to Pc96 and PcX).

Besides Pc genes, other genes conferring oat leaf rust are also described, although not completed characterized or named as yet. Plants with genotypes Pc frequently respond to P. coronata through the development of a hypersensitivity reaction (HR), with death of cells at the local infection site. Two loci seem to control cellular death: Rds suppress the HR reaction, but not resistance, mediated by the resistance gene Pc82. In contrast, locus Rih confers HR to resistant as well as to susceptible plants. Data showed that the locus Rds is not linked to the cluster Pca, while the Rih locus is intimately linked to it. This indicates that many variables affect the development of HR suggesting that HR may not be essential to crown rust resistance. Rds and Rih moderate HR-independent gene-for-gene resistance (Yao et al. 1998; Xin You et al. 2001).

Despite the vast number of known race-specific genes, only a few of them have good potential to be used in breeding programs. Among the qualitative genes that still show good potential for use in breeding programs some have been brought to attention, such as P.c30, P.c91, (Park 1999), P.c94 (Chong and Aung 1998; van Niekert et al. 2001) and P.c68 (Park 1999; van Niekert et al. 2001; Leon-ard and Martinelli 2005). Their indication as genes of potential use is not related to a complete immunity to the pathogen, but to the consistent low frequency of virulence of races to these genes in the pathogen population. Efforts are being made on the search for a more stable, quantitative, non-specific resistance to crown rust, such as the one provided by the A. sativa genotype ‘MN841801’ from the Univer- sity of Minnesota breeding program. In Brazil, Chaves et al. (2004a, 2004b) were able to identify a number of promising genotypes as sources of quantitative resistance.

Regarding oat stem rust, there is a much more limited reservoir of resistance against, but known genes can provide effective and long term resistance when used in appropriate combinations. In Argentina the only resistant oat variety to stem rust in use is the genotype ‘UFRGS-16’, bred by the oat breeding program from the Brazilian Federal University of Rio Grande do Sul State (LC Federizzi, pers. comm.).

Genetic resistance to rye rusts

Compared with other cereals such as wheat and barley, little information is available about the inheritance of resistance to rye leaf rust. Resistance genes conferring specific resistance are more studied, although slow rusting resistance has also been reported. As for the wheat leaf rust, the Lr symbol is used to designate the resistance genes against rye leaf rust, although there is a proposal that this symbol changes to Pr. Examples of specific resistance genes to rye leaf rust are Lr4, Lr5, Lr6, Lr7, Lr25, Lr26, LrSatu, Pr1, Pr2, Pr3, Pr4 and Pr5 and some of them also confer resistance in wheat and in triticale (Wehling et al. 2003; Roux et al. 2004).

The rye gene resistance SrR seems to be a promising source of resistance to stem rust, and, just like the Sec31, will be able to be transferred to wheat. However, the simulta-neous transfer of genes in the Sec-1 locus, coding for secalin, can decrease flour quality of commercial varieties (Lee et al. 1995; Sce et al. 1995). Attempts to break the link-age between SrR and Sec-1 have been made, but no suc-cess was obtained up to now (Mago et al. 2004; Anugra-hwati et al. 2008).
Fungicides (chemical control)

When genetic resistance is overcome or when its level is insufficient to control cereal rusts, chemical control is needed. Chemical control has been successfully used in Europe, permitting high yields (6 to 7 t/ha) and where prices for wheat are warranted (Stubbs and de Bruin 1970; Buchenauer 1982). Chemicals were also used to control a leaf rust epidemic in the irrigated Yaqui and Mayo Valleys of Mexico (Dubin and Torres 1981). 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 USA has been practiced when expected yields exceed 2 t/ha (Roelfs et al. 1992).

In North America the first large-scale, successful use of fungicides to control stripe rust occurred in 1981, when Line and his associates had demonstrated the effectiveness of triadimefon and developed guidelines for the timely application for economical control. Fungicide use prevented multimillion dollar losses, and in recent years, use of fungicides has successfully reduced yield damage caused by stripe rust. Nevertheless, the use of fungicides adds a huge cost to wheat production, which is a burden for many growers, especially in developing countries. The use of fungicides may also create health problems for users, adversely affect the environment, and result in the selection of fungicide-resistant strains of the pathogen (Chen 2005).

In South America, fungicides are currently used to control wheat and oat rusts, because the most popular varieties are susceptible or moderately susceptible. The use of fungicides to control wheat rust differs among countries. Fungicides have been recommended since 1977 in Brazil, have been used widely in Paraguay since 1976, and have been more commonly used in Uruguay since the 1990s. In normal epidemic conditions at least 2 applications of fungicides are required to control the disease on highly susceptible cultivars. In Argentina, one fungicide application to control leaf rust is used on about 35% of the wheat area. In Chile, the use of fungicides to control leaf rust is not as common, but in the last 5 years the use of chemical control has increased with the increased importance of the disease. Considering the large areas sown to cultivars that require chemical control in an average epidemic, the total annual cost of fungicide applications to control wheat rusts in the region is about US$ 50 million (Germán et al. 2007). In Brazil, the chemical protection of oat plants for a 35-day period, after the first symptoms have started, is enough to prevent significant yield losses (Martinelli et al. 1994).

Cultural practices

Some cultural practices can also help, at least partially, in the control of cereal rusts. The use of early maturing cultivars in Australia and the early sowing in Mexico are examples of cultural practices that marked initial success in controlling wheat stem rust even before the use of resistant cultivars (Borlaug 1954; McIntosh 1976). The “green-bridge” formed by volunteer cereals plants guarantees the maintenance and dissemination of inoculum between crop seasons or between areas with different sowing dates. Removing this “green-bridge” with tillage of herbicides is an effective control measure for rust epidemics that would result from endogenous inoculum. The vulnerability of commercial cultivars to rust epidemics can be reduced through broadening the genetic basis in breeding programs. Some of the benefits of gene deployment can also be obtained by the growers if cultivars with different levels or types of resistance are grown within the farm and/or in immediate neighbors. In some areas, control of timing, 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 downwind from late maturing cultivars. In any situation, each cultural practice must be tested against the anticipated types of epidemic that occur in the area (Roelfs et al. 1992; Singh et al. 2002).

An alternate host eradication program for stem rust was successful in northern Europe (Hermansen 1968) and in the north-central states of the USA from 1918 until the 1980s, when over 500 million bushes were destroyed nationally, approximately 1 million in Minnesota (Roelfs 1978; Peterson et al. 2005). However, the reemergence of barley in some sites in Minnesota where the bushes were destroyed as a source of stem rust inoculum 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 would probably not be feasible (Singh et al. 2002).

The eradication of the alternate Rhamnus and Berberis hosts has been an important factor in reducing oat crown and stem rust epidemics in areas where these hosts play a chief effect as sources of inoculum and genetic variability of the pathogen. The use of oat rust as a model role to control stem races of North America. Rhamnus is difficult to control and extensive infestations still remain particularly in Ontario, Canada (Harder and Haber 1992).

CONCLUDING REMARKS

The cereal rusts are the most striking and fascinating of the plant pathogens. Obviously, the devastating and even catastrophic effect of rust epidemics on cereal host populations was, since early records, the first factor of notoriety for these pathogens. In addition to the food issue, its influence on early civilizations also had religious and political connotation, but, at that time, the most striking features of the cereal rusts had not yet been unveiled. Knowledge about the biology, ecology and epidemiology of such diseases has been built since the Renaissance period due to the need to achieve effective methods to control and minimize the impacts of the rust epidemics. The biotrophic nature of the parasitism, the life cycle with 5 spore stages on 2 different, genetically distant hosts and the physiologic specialization on host species and genotypes were the first revelations about the cereal rusts, which showed how unique these pathosystems are. The findings of Billen on the inheritance of resistance, and of Flor on the gene-to-gene relationship between plant and pathogen on the establishment of diseases provided a great progress in controlling cereal rusts through genetic resistance. However, cereal rusts once again showed that their millenary coevolution with cereals had taught them to survive. The cycles of boom-and-bust, where resistant cultivars were released and quickly overcome by rust pathogens were another striking feature of the cereal rusts.

Fortunately, durable forms of resistance to cereals rusts were detected and their genetic control has been comprehensively studied. Advances in knowledge about the physiology of parasitism and how physical and chemical stimuli positively and negatively affect each stage of the infective process also provided new perspectives for the understanding and use of genetic resistance. The current paradigm regarding the durability of resistance associates its expression to the mechanism involved in pathogen recognition by the host and to the triggered defense mechanisms that prevent or limit infection. Differences in resistance levels appear to be related to signaling and to the intensity at which these mechanisms are expressed. However, despite the progress in understanding durable resistance, much remains to be learned, especially about the development of the specialized feeding structure (the haustorium) in the periplasmic space of the host; about the establishment of the obligate-parasite relationship; when and where the
recognition occurs, and what genes control the events that prevent or delay the development of the infection structures of rust fungi.

Among the challenges for the future are the unveiling of the signal transduction pathways triggered during the resistance process; the spatial and temporal characterization of the wheat response to the metabolic process involved in plant defense reaction, and the characterization of genes differentially expressed in partially resistant and susceptible genotypes. Thus, efficient genetic markers associated with the genes involved in the defense process can be obtained. Genomic studies of the fungi-causing cereal rusts and the discovery of mechanisms involved in non-host interactions, such as rice and cereal rusts, are also great challenges for the future. Exploitation of the enormous information about the mechanisms involved in resistance to rusts will require a multidisciplinary collaborative work of research programs in a way to converge the specific knowledge of plant pathology, genetics, biochemistry, physiology, histology, structural and functional genomics, proteomics and bioinformatics to give valuable answers to sustain grain production for the 9,000 million or so people likely to inhabit on Earth in the coming 50 years.

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REFERENCES


Adams EB, Line RF (1997) Plant pathogenesis, development, and microbial interaction. Phytopathology 87, 320-329


Antonelli EF (2000) La roya negra del tallo (Puccinia graminis f. sp. tritici). Una ilustre ausente de los campos de trigo. ¿Por cuánto tiempo más? Necio, Argentina, 43 pp


Collins RM, Read ND (1990) Pustules of rust fungi: penetrating the host–haustorium barrier. Advances in Plant Pathology 6, 35-41


Davies JR, Knight ID (1982) Stem rust resistance in wheat cultivars having resistance genes at Yr3 and Yr4 loci. Phytopathology 82, 1228-1233

Chester KS (1946) The nature and prevention of the cereal rusts as exemplified in the leaf rust of wheat. In: Chronica Botanica, Waltham, MA, USA, p 269


Chen XM, Line RF (1993) Inheritance of stripe rust resistance in wheat cultivars postulated to have resistance genes at Yr5 and Yr4 loci. Physiopathology 83, 382-388

Chen XM, Line RF (2003) Identification of genes for resistance to Puccinia striiformis f. sp. hordei in 18 barley genotypes. Euphytica 129, 127-146

Chen XM, Line RF, Leung H (1995) Virulence and polymorphic DNA relationships of Puccinia striformis f. sp. hordei to other rusts. Phytopathology 85, 1335-1342

Chen XM, Jones SS, Line RF (1996) Chromosomal location of genes for resistance to Puccinia striiformis in seven wheat cultivars having resistance genes at Yr3 and Yr4 loci. Phytopathology 86, 1228-1233

Chester KS (1946) The nature and prevention of the cereal rusts as exemplified in the leaf rust of wheat. In: Chronica Botanica, Waltham, MA, USA, p 269


Collins RM, Read ND (1990) Pustules of rust fungi: penetrating the host–haustorium barrier. Advances in Plant Pathology 6, 35-41


De Candolle A (1865-1866) New Discoveries in plants and crops. The New Phytologist, 1, 36-41


Cox DL (1985) Genetic variation between and within Ethiopian barley landraces with emphasis on durable disease resistance. PhD thesis, Wageningen Agricultural University, The Netherlands, 56 pp

Cereal rusts – history, biology and control. Chaves et al.


Agricultural Economics 29, 253-263


McIntosh RA (1992) Genetic linkage of genes conferring adult-plant resistance to leaf rust and stripe rust in wheat. Plant Pathology 41, 523-527


Rees RG, Platz GJ (1975) Control of wheat leaf rust with 4-n-butil-1,2,4-triazole. Australian Journal of Experimental Agriculture and Animal Husbandr 15, 276-280


Reynolds MP, Borlaug NE (2006) Impacts of breeding on international collaboration in wheat improvement. Journal of Applied Genetics 144, 3-17


Singh RP (1992) Genetic association of leaf rust resistance gene Lr34 with...
adult plant resistance to stripe rust in bread wheat. Phytopathology 82, 835-838
Singh RP, Rajaram S (1992) Genetics of adult-plant leaf rust resistant to "Frontona" and three CIMMYT wheats. Genome 35, 24-31
Singh RP, Rajaram S (1994) Genetics of adult leaf rust resistance to stripe rust in ten spring bread wheats. Euphytica 72, 1-7
Stakman EC, Levine MN (1922) The determination of biologic forms of Puccinia graminis on Triticum spp. Minnesota Agricultural Experimental Station Technical Bulletin 8, 1-10
Stakman EC, Harrar JG (1957) Principles of Plant Pathology, New York, 581 pp
Stubbbs RW, de Bruin T (1970) Bestrijding van geel roest met het systemische fungicide oxycaroxin 'Plantvax'. Biological Research Information Service (TRI), 89-104
Tzouetti GT (1952) V. Almirurgia: True nature, causes and sad effects of the rusts, the bunts, the smuts, and other maladies of wheat and oats in the field. In: Tehon LR (translation) Phytopathological Classics (No 9) American Phytopathological Society, St Paul, MN, USA, (Originally published 1976) 139 pp
Vaz Pinto MC, Nkis RT (2001) Leaf wax layer may prevent appressorium differentiation but does not influence orientation of the leaf rust fungus Puccinia hordei on Hordeum chilense leaves. European Journal of Plant Pathology 107, 795-803
Wiese MV (1977) Compendium of Wheat Diseases, American Phytopathological Society, St. Paul, Minnesota, USA, 112 pp