Chapter 7

RUST

J. R. Stavely and M. A. Pastor-Corrales*

Introduction

Bean rust is caused by *Uromyces appendiculatus* (Pers.) Unger var. *appendiculatus* (syn. *U. phaseoli* (Reben) Wint.). The disease has a worldwide distribution (Zaumeyer and Thomas, 1957). It consistently causes major production problems in humid tropical and subtropical areas and periodic severe epidemics in humid temperate regions (Ballantyne, 1974; Vargas-G., 1980; Zaumeyer and Thomas, 1957).

In Latin America, major losses occur in Argentina, Bolivia, Brazil, Colombia, Costa Rica, Cuba, Dominican Republic, Ecuador, El Salvador, the Chimaltenango district of Guatemala, Haiti, Honduras, Jamaica, Mexico, northern Nicaragua, and coastal Peru (Crispín-Medina et al., 1976; Dongo-D., 1971; González-Avila, 1976; Guerra and Dongo-D., 1973; López-G., 1976; Rodríguez-Alvarado, 1976; Shaik, 1985b; Vargas-G., 1970, 1971, and 1980).

Major losses occur in Burundi, Ethiopia, Kenya, Malawi, Rwanda, South Africa, Tanzania, Uganda, and Zimbabwe (Assefa, 1985; CIAT, 1981). Severe epidemics occur in Australia, China, United States, and some areas of Europe (Ballantyne, 1978; Kelly, 1982; Teng, 1932; Yeh, 1983; Zaumeyer and Thomas, 1957). A major rust epidemic occurs in many areas of Mexico every four to

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* Research plant pathologist, Microbiology and Plant Pathology Laboratory, Plant Protection Institute, Agriculture Research Service, United States Department of Agriculture (ARS/USDA), Beltsville, MD, USA; and Bean Program pathologist, Centro Internacional de Agricultura Tropical (CIAT), Cali, Colombia, respectively.

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five years, although in the valley of Mexico, other valleys, and some Gulf states rust is endemic and causes substantial losses every year (Crispín-Medina et al., 1976).

Yield losses are most severe when plants are infected during the preflowering and flowering stages of development (Almeida et al., 1977a; Costa, 1972; Crispín-Medina et al., 1976; Nasser, 1976; Wimalajeewa and Thavam, 1973; Yoshii and Gálvez, 1975). Disease loss estimates in the greenhouse and field include 40%-50% plant dry weight reduction (Almeida et al., 1977a). Yield losses are estimated at 18%-28% (Dongo-D., 1971; Venette and Jones, 1982b; Wimalajeewa and Thavam, 1973; Zulu and Wheeler, 1982), 36%-45% (Kelly, 1982; Nasser, 1976; Venette and Jones, 1982b), and 40%-100% (Hilty and Mullins, 1975; Kelly 1982; Schwartz, 1984; Solís, 1977; Venette and Jones, 1982b; Zaumeyer and Thomas, 1957).

Uromyces appendiculatus infects many species of *Phaseolus*, including tepary bean (*P. acutifolius* A. Gray var. *acutifolius*), scarlet runner bean (*P. coccineus* L.), lima bean (*P. lunatus* L.), *P. coccineus* subsp. *obvallatus* (Schlecht.) M.M.S., *P. polystachyus* (L.) B.S.P., *P. maculatus* Scheele, *P. polystachyus* var. *sinuatus* (Nutt) M.M.S., and common bean (*P. vulgaris* L.). It also infects siratro (*Macroptilium atropurpureum* (DC.) Urb.), cowpea (*Vigna unguiculata* (L.) Walp. ssp. *unguiculata*), (Arthur, 1915; Rey-G. and Lozano-T., 1961; Zaumeyer and Thomas, 1957), *V. luteola* (Jacq.) Bentham, *V. adenantha* (G. F. Meyer) M.M.S., and *V. vexillata* (L.). A. Rich. (Almeida et al., 1977c). The prevalent host is *P. vulgaris*. Its natural occurrence on *P. lunatus* in United States is rare, and differs from the primary rust pathogen of *Vigna* species which is the cowpea rust fungus (*Uromyces vignae*) (Cummins, 1978).

Common names frequently used for rust in Latin America include "roya" and "chahuixtle" in Spanish and "ferrugem" in Portuguese.

Almeida (R. T. Almeida, 1977) reported the existence of a variety of bean rust collected in 1945 from *Macroptilium longepedunculatum* (Benth.) Urban (then known as *Phaseolus longepedunculatus* ex Benth.) by Viegas, who named the rust *Uromyces* *phaseoli longepedunculati* Viegas. Almeida studied herbarium samples of the original collection, confirmed that it differs morphologically from *U. appendiculatus* var. *appendiculatus*, and, according to current nomenclature rules, named it *Uromyces appendiculatus* (Pers.) Ung. var. *brasiliensis* R. Almeida var. nov.

Phaseolus vulgaris, although susceptible to the soybean rust fungus (*Phakopsora pachyrhizi* Sydow), is, apparently, an uncommon host of that pathogen (Cummins, 1978; Stavely et al., 1985; Vakili and Bromfield, 1976). This fungus is not known to produce pycnia or aecia and produces uredia and teliosori very different from those of *U. appendiculatus* (Cummins, 1978; Stavely et al., 1985). Several uredia, each less than 0.3 mm in diameter, are produced in a necrotic lesion 0.2 to 4 mm in diameter. Uredia and spores are lighter in color and spores are smaller than those of *U. appendiculatus*. In Popayán, Colombia, *Phakopsora pachyrhizi* occurs on *Phaseolus lunatus*, and *P. lunatus* x *P. vulgaris* hybrids, but not on *P. vulgaris* (M. A. Pastor-Corrales, unpublished data).

Etiology

Uromyces appendiculatus is an obligate parasite which belongs to the Basidiomycotina subdivision of fungi. It has an autoecious, macrocyclic life cycle which is completed entirely on the bean host (Andrus, 1931; Cummins, 1978). Overwintering, resting teliospores germinate to produce basidia and basidiospores that infect the host leaf, producing pycnia. Upon cross fertilization with pycniospores, an aecium is produced and aeciospores develop, infecting the leaf and producing uredia pustules. The uredia in turn, produce uredospores that infect the plant, producing more uredia and giving rise to repeated infections over most of the growing season. As uredia age, if conditions are appropriate, they produce thick-walled teliospores.

Pycnia and aecia are rarely observed under field conditions although aecia have been found in regions of Oregon (Zaumeyer and Thomas, 1957), New York (Jones, 1960), North Dakota (Venette et al., 1978), and southern Germany (Heinze, 1974). In North Dakota, the aecia were observed on volunteer bean plants within a canopy of wheat in a field that had contained rusted beans the previous year. Aecia have been studied in detail in the greenhouse by Andrus (1931) and, more recently, by Groth and Mogen (1978).

When the basidiospores infect bean leaves, it takes about six days at 22-26 °C for a small chlorotic fleck containing the pycnium to develop (Figure 31). About seven days later, the pycnium produces droplets of cloudy white nectar containing spermatia (+ or - mating type) and receptive hyphae (Andrus, 1931; Gold and Mendgen 1984a; Groth and Mogen, 1978). Cross fertilization of a pycnium by pycniospores of the opposite mating type will begin aecium formation, usually on the lower leaf surface (Figure 32), within 9-12 days at 22-26 °C. Aecia may form occasionally on the upper leaf surface also. Aeciospores form in the white aecium and, upon their release, are able to infect bean plants. Eight to ten days later each aeciospore infection produces a uredium with uredospores (Andrus, 1931; Groth and Mogen, 1978).

Subsequent cycles of infection rely solely upon the uredospore stage. These uredospores are capable of germinating to provide infectious hyphae that infect the plant and form new uredia in which new uredospores, and eventually teliospores, will develop (Andrus, 1931). Fusion of dikaryotic nuclei occurs in the teliospores immediately after they are formed (Gold and Mendgen, 1984b). Teliospores are produced by many but not all races (Groth and Mogen, 1978; Groth and Shrum, 1977; Harter and Zaumeyer, 1941; Stavely, 1984b).

Teliospores of *U. appendiculatus* require a dormant period before they will germinate (Gold, 1983; Gold and Mendgen, 1983a; Harter et al., 1935). Gold and Mendgen (1983a) found that teliospores, removed from bean leaves, will germinate after 9-48 months of storage in a refrigerator at 4 °C and 70% relative humidity (r.h.) upon incubation in the proper environment. Storage at the extremes of -18 °C or 20 °C severely reduces germinability (Gold, 1983; Groth and Mogen, 1978). For teliospores exposed to the winter environment in Germany, the dormant period lasts three or four months and maximum germination occurs in seven to eight months. High summer temperatures kill ungerminated teliospores. When teliospores are exposed to favorable conditions following sufficient overwintering outdoors or proper storage indoors, a three- to four-day lag precedes germination (Gold, 1983; Gold and Mendgen, 1983a; Groth and Mogen, 1978). Optimal laboratory temperature and light intensity for teliospore germination on 2%-distilled-water agar in a petri dish are 18 °C and 17,000 lux (Gold, 1983; Gold and Mendgen, 1984a and 1984c).

Alternating light and dark periods are essential for teliospore germination and release of the basidiospores. Peak basidiospore release occurs after about 7 hours of dark (Gold, 1983; Gold and Mendgen, 1984a). The minimal dark period is three to four hours and the minimal light period is 0.5 hr with 1000 lux. Groth and Mogen (1978) found that prewashing teliospores in cold running water for three hours to eight days had no noticeable effect on teliospore germination. However, some teliospores germinated on water agar three to four weeks after a brief washing. Exposing teliospores to unidentified volatile substances from germinating bean seedlings for 8-12 days stimulates germination in the presence of alternating light and dark periods and also overcomes the requirement for dormancy (Gold, 1983; Gold and Mendgen, 1983b).

The teliospore germinates to produce a basidium in which meiosis occurs and on which haploid basidiospores develop (Gold and Mendgen, 1984b). Mature basidiospores are reniform to ovate-elliptical in shape, smooth surfaced, and measure 9 µm by 16 µm. If supplied with 100% r.h. in darkness, basidiospores begin to germinate on agar or bean leaves in about two hours (Gold, 1983; Gold and Mendgen, 1984a). On a susceptible cultivar, an appressorium is formed, penetration is direct (Gold, 1983), and inter- and intracellular hyphae develop (Gold and Mendgen, 1984c). Pycnial formation is favored by temperatures of 22-26 °C (Gold and Mendgen, 1984c; Groth and Mogen, 1978).

The most commonly observed spore forms are the uredospore (summer or vegetative spore) and teliospore (winter or resting spore). Uredospores are produced in rows within the cinnamon-brown uredium (sorus, pustule) on the upper or lower leaf surface. Uredospores are light brown, one celled, spiny, thin walled, and globoid to ellipsoid in shape. They may have two equatorial or superequatorial pores and measure 20-27 µm by 24-30 µm (Cummins, 1978; Zaumeyer and Thomas, 1957). Near the end of the growing season, teliospores may form within the pustule in response to changes in light intensity, temperature, moisture, cultivar response, leaf age, or plant maturity. Teliospores have a hyaline pedicel and are blackish brown, one celled, have few to numerous verrucae (wart-like projections), are rarely smooth, thick walled, and are globoid to broadly ellipsoid in shape. They may have a hyaline papilla over the pore and measure 24 µm by 30 µm. Some races of *U. appendiculatus* do not produce teliospores (Groth and Mogen, 1978; Groth and Shrum, 1977; Harter and Zaumeyer, 1941; Stavely, 1984a), apparently surviving solely by uredospores.

Although *U. appendiculatus* does not grow in culture, viable spores can be preserved for varying time periods in the laboratory. Uredia and uredospores on dried leaves on dried leaves have been successfully stored at -20 °C for two years (Harter and Zaumeyer, 1941). Dundas (1948) reported that storage at -18 °C for five to seven months could reduce spore germination markedly and induce pathogenic mutations. Uredospore germinability is higher if spores are collected from young, rather than old, uredia and leaves, and if they are produced at 16-21 °C rather than at 24-27 °C (Imhoff et al., 1981). Uredospores can be conveniently stored at -18 °C for about one to three years if removed from uredia, placed in a vial, and dried over a desiccant for a few hours to remove excess moisture before freezing (Bromfield, 1964; Davison and Vaughan, 1963b; Stavely, 1983). Uredospores stored at 7 °C for 26 weeks were still capable of infecting plants in the greenhouse (Harter and Zaumeyer, 1941). Viable spores (40% germination) have been recovered after storage for nearly two years in a special freezer at -60 °C (Schein, 1962) and after storage for at least seven years in liquid nitrogen (Cunningham, 1973). Frozen uredospores of some rust fungi are dormant upon thawing, but not those of *U. appendiculatus* (Bromfield, 1964).

Epidemiology

Infection by *Uromyces appendiculatus* uredospores is favored by prolonged periods (10-18 hours) of moisture, greater than 95% r.h., and moderate temperatures between 17-27 °C (Augustin et al., 1972; González-Avila, 1976; Harter et al., 1935; Schein, 1961a; Zaumeyer and Thomas, 1957). The optimal temperature for uredospore germination is 16-24 °C. Germination occurs in the first six to eight hours in the presence of moisture (Imhoff et al., 1981; von Alten, 1983). Temperatures greater than 32 °C may kill the fungus (Crispín-Medina et al., 1976; Imhoff et al., 1982; Schein, 1961a and 1961b; Zaumeyer and Thomas, 1957). Temperatures less than 15 °C retard fungal development (Crispín-Medina et al., 1976; Imhoff et al., 1981 and 1982; Zaumeyer and Thomas, 1957). Daylength and light intensity are important factors (Harter and Zaumeyer, 1941). Augustin et al. (1972) reported that infection is favored by incubation in low light intensity (2 x 10^{-5} μE cm^{-2} s^{-1}) for 18 hours.

The latent period for uredium development (measured as time from inoculation until 50% of the uredia on the adaxial leaf surface open), varies from seven days at 24 °C to nine days at 16 °C constant canopy-level air temperatures (Imhoff et al., 1982). Leaf temperatures in this study were 1-3 °C higher than air temperatures. At 27 °C constant air temperature, lesions do not develop to the sporulation stage.

Uredospore production and release also are influenced by moisture and temperature. Spore production increases when infected plants are exposed to high humidity conditions for limited or prolonged periods (Imhoff et al., 1982; Yarwood, 1961). Cohen and Rotem (1970) reported that sporulation increased when infected plants received at least a 12-hour photoperiod. *Uromyces appendiculatus* can produce one million uredospores per square centimeter on leaves bearing two to 100 uredia per square centimeter (Yarwood, 1961). This spore production occurs in waves, peaking every three to four days. Efficiency of sporulation per unit of leaf area varies inversely with uredium density (Imhoff et al., 1982). Dense infection also reduces uredium size (Harter and Zaumeyer, 1941; Stavely, 1984c). Nasser (1976) reported that the largest number of spores are released during temperate (higher than 21 °C), dry (less than 60% r.h.) days which are preceded by a long dew period or rain the previous night. Uredospores can survive nearly 60 days under field conditions (Zambolim and Chaves, 1974). They contain a water-soluble germination self-inhibitor, methyl cis-3,4 dimethoxycinnamate (Allen, 1972; Macko et al., 1970 and 1976). This inhibitor is removed by washing spores with water and is counteracted by a water-soluble substance in bean leaves (Thomas and

Meiners, 1977), as well as by several defined compounds (Macko et al., 1976).

Uredospores and teliospores can overwinter in bean debris and on wooden supports used for climbing beans (Davison and Vaughan, 1963b). Uredospores can be transported long distances by wind currents. They may provide primary, as well as secondary, inoculum during epidemics in Latin America, Africa, and other places where multiple cropping and/or staggered planting dates provide a continuum of susceptible host tissue during favorable environmental conditions.

Bean rust incidence may be influenced by different cropping systems. For example, in one study, rust incidence was lower when beans were grown in monoculture than in association with maize (GLP, 1976). However, in another study, rust incidence was significantly higher under monoculture than in multiple cropping of beans with maize (Moreno and Mora, 1984). Apparently several factors such as resistance induced by incomplete infection of the beans by pathogens of the companion crop and microclimatic effects, may influence such situations (Allen, 1976; Moreno and Mora, 1984).

Infection by Uredospores

Uromyces appendiculatus uredospores will germinate in the absence of the host if the germination inhibitor is removed by washing with water (Macko et al., 1970). Germination is enhanced by supplying certain divalent cations (Baker et al., 1983a). The appressorium is induced by certain contact stimuli such as the stomatal outer lip (Wynn, 1976) or a scratch on a hydrophobic membrane (Staples et al., 1985). Under artificial conditions, this signal may be replaced by potassium (Staples et al., 1983), glucose and sucrose (Kaminskyj and Day, 1984), or inhibitors of cyclic nucleotide phosphodiesterase (Hoch and Staples, 1984).

The infection process for a uredospore begins as a germ tube develops an appressorium upon physical contact with the edges of a stoma (Pring, 1980; Wynn, 1976). Infection is most efficient on young leaves which are less than 70% of their final size (Groth and

Urs, 1982; Harter and Zaumeyer, 1941; Schein, 1965; Stavely, 1983; von Alten, 1983). In contrast, on older leaves, fewer appressoria (von Alten, 1983), less necrosis in the necrotic small-uredium reaction (Shaik and Steadman, 1986), and fewer and smaller uredia occur (Kolmer et al., 1984; von Alten, 1983; Zulu and Wheeler, 1982). An infection peg develops from the appressorium and pushes between the guard cells until the fungal cytoplasm is transferred into the substomatal vesicle. The substomatal vesicle contains numerous glyoxysomes, lipid bodies, and glycogen particles (Mendgen, 1973). In most instances, only one infection hypha emerges from the substomatal vesicle. At the tip of the infection hypha, haustorial mother cell development is induced upon contact with a parenchymatous cell (Mendgen, 1978a). The host cell is penetrated, a haustorium differentiates, and nutrients are transferred from the host to the haustorium and intercellular hypha (Mendgen, 1979). Intercellular ramification proceeds throughout the host tissue, eventually forming a young uredium (Pring, 1980; Sziráki et al., 1984).

Host physiology and biochemistry are affected during the infection and sporulation processes. Respiration increases and photosynthesis decreases during infection, especially after the sixth day (Raggi, 1980). Initially, reducing sugars, sucrose, starches, and free amino acids increase in infected tissue. Later, certain amino acids and sugars decrease as sporulation begins (Inman, 1962; Raggi, 1974). Various enzymes such as peroxidase, catecholoxidase, glycolate-oxidase, and glyoxalate reductase, increase their activity during infection (Montalbini and Cappelli, 1973; Raggi, 1974; Sempio et al., 1975). Quinones such as vitamin K, plastoquinones A, C, and O, and ubiquinone, also increase during rust infection and development (Montalbini, 1973). In hypersensitive, necrotic-resistant reactions, deposition of tannins and death of affected host cells occur soon after infection (de la Torre-Almaraz et al., 1985).

Infection reduces the transfer of metabolic byproducts from leaves to roots and developing seeds (Zaki and Durbin, 1965). Stomatal transpiration decreases two days after infection (Duniway and Durbin, 1971b; Sempio et al., 1966) because stomatal opening is inhibited (Duniway and Durbin, 1971b). Transpiration and water vapor loss through the damaged cuticle then increases as infection proceeds (Duniway and Durbin, 1971a; Sempio et al., 1966). Infected plants become more sensitive to moisture stress as sporulation occurs (Duniway and Durbin, 1971a).

Symptomatology

Uromyces appendiculatus may infect leaves (Figure 33), pods (Figure 34), and, rarely, stems and branches (Figure 35). Initial infection may occur on the upper or lower leaf surface. However, symptoms usually appear first on the lower surface as minute, whitish, slightly raised spots (Figure 36) about five or six days after inoculation. These spots enlarge to form mature reddish brown uredial pustules which rupture the epidermis about two days later. Sporulation begins and the uredium may attain a diameter of 1-2 mm within 10-12 days after inoculation. Secondary and tertiary uredia may develop around the perimeter of this primary uredium (Harter and Zaumeyer, 1941; Zaumeyer and Thomas, 1957). The entire infection cycle occurs within 10-15 days. Uredospores are released passively from open uredia and scattered by farm implements, insects, animals, and wind currents (Yarwood, 1961; Zaumeyer and Thomas, 1957). Later, black teliospores may form in the uredium. The teliosori become dark brown to black as teliospores replace uredospores (Figure 37). The bean rust fungus is not seed transmitted (Zaumeyer and Thomas, 1957).

Various interactions have been observed between infections by *Uromyces appendiculatus* and other bean pathogens or nonpathogens, usually under controlled conditions. Rust infection may predispose plants to subsequent infection by bean pathogens such as the halo blight bacterium (*Pseudomonas syringae* pv. *phaseolicola* (Burk.) Young et al.), anthracnose fungus (*Colletotrichum lindemuthianum* (Saccardo *et* Magnus) Briosi *et* Cavara) (Figure 38), and the root-rot fungus (*Thielaviopsis basicola* (Berkely *et* Broome) Ferraris), and by nonpathogens such as cucumber powdery mildew (*Sphaerotheca fuligena*) and tobacco mosaic virus (TMV)(Yarwood, 1969 and 1977).

A high incidence of rust infection may suppress the appearance of halo blight symptoms (Yarwood, 1969). Necrotic rings can occur on the perimeter of rust uredia when rust-infected plants are inoculated with TMV (Gill, 1965; Wilson, 1958), and possibly other viruses (Figure 39), or with cucumber downy mildew (*Pseudoperonospora cubensis* (Berk. *et* Curtis) Rostovzev) (Yarwood, 1977). Heavily rusted sections of leaves were slowly killed during the interaction between bean rust and cucumber downy mildew. Rust spores may contain compounds which inhibit virus multiplication when rust and virus are inoculated simultaneously onto plants (Gill, 1965; Wilson, 1958).

Control by Cultural Practices

Cultural controls include crop rotation and removal of old plant debris which may bear viable uredospores and teliospores (Vieira, 1967; Zaumeyer and Thomas, 1957). However, such sanitation measures may have only limited value in controlling rust (Plaut and Berger, 1981). Reduced plant density also may decrease rust incidence. Planting dates may be adjusted in certain production areas to avoid or reduce the incidence of rust infection. Such adjustment will minimize exposure to moderate to cool temperatures and long dew periods during the critical preflowering to flowering stage of plant development.

Biological Control

Biological control is not intentionally used for bean rust, but it may have some potential for the future. The fungus (*Verticillium lecanii* (Zimm.) Viegas) penetrates, invades, and kills uredospores and teliospores, and colonizes uredia of *U. appendiculatus* (Allen, 1982; Grabski and Mendgen, 1986). This pathogen of the rust fungus is easily found in some seasons in the subtropics and tropics (R. T. McMillan, personal communication) and may have a role in the cyclic nature of rust epidemics. It has given 68% control of bean rust in the greenhouse, but gave little control in the field in Germany (Grabski and Mendgen, 1985). *Bacillus subtilis* (Ehrenberg) Cohn, and other *Bacillus* spp. to a lesser degree, gave excellent control of bean rust when applied before inoculation of plants with uredospores in the greenhouse (Baker et al., 1983b). When sprayed on field-grown beans three times per week, *B. subtilis* caused a 75% reduction in rust severity (Baker et al., 1985).

Results from recent experimental greenhouse and field tests suggest that inoculation of specific bean cultivars with specific races of *U. appendiculatus* to which they are not susceptible will protect against other races to which they are susceptible (M. A. Pastor-Corrales, unpublished data).

Control by Chemicals

Bean rust reduces yields more severely when infection occurs before, rather than after, flowering. Therefore, chemical control is most effective during early plant development (Yoshii and Gálvez, 1975). Bean rust has been controlled by dusting plants every 7-10 days with sulfur at a rate of 25-30 kg/ha (Crispín-Medina et al., 1976; Harter et al., 1935; Zaumeyer and Thomas, 1957), after uredia first appear. However, sulfur can cause leaf burning if applied at higher rates at temperatures above 30 ⁰C.

A seven- to fourteen-day spray schedule is recommended for other preventive chemicals such as chlorothalonil (225 g/100 L), or maneb (4 kg/ha), and/or mancozeb (3-4 kg/ha) (Costa, 1972; Crispín-Medina et al., 1976; Frenhani et al., 1971; González et al., 1977; Hilty and Mullins, 1975; Steadman and Lindgren, 1983; Tompkins et al., 1983; Venette and Jones, 1982a; Vieira, 1967; Wimalajeewa and Thavam, 1973).

Other effective chemicals but which have not yet been approved for use in the United States are bitertanol, triadimefon, and Propiconazole (Mullins and Hilty, 1985; Nieuwoudt, 1984; Venette and Jones, 1982a). Phytotoxicity can be a problem with this last group of fungicides (Mullins and Hilty, 1985).

Uredospores germinate on beans treated with triphenylphosphite, a chemical that is not commercially available as a fungicide. Although the uredospores infect the host plant and form haustorial mother cells, haustoria and uredia do not develop (Rusuku et al., 1984).

Oxycarboxin can be somewhat therapeutic. It is effective when sprayed at 1.8-2.5 kg/ha 20 and 40 days after planting or every two weeks until the end of flowering (Costa, 1972; Crispín-Medina et al., 1976; Frenhani et al., 1971; González et al., 1977; Yoshii and Granada, 1976). Dongo-D. (1971) reported that one preflower application of oxycarboxin (0.9 kg/ha) reduced rust infection by 40% and increased yields by 26%. However, seed treatment with oxycarboxin did not give satisfactory control (Frenhani et al., 1971). Oxycarboxin (4000 ppm) is therapeutic when applied up to three days after inoculation and preventive when applied less than seven days before inoculation (Almeida et al., 1977b and 1977c). Although Issa and de Arruda (1964) concluded that chemical control was not economically practical in parts of Brazil, this is not true in epidemic years in many other areas of the world.

In the absence of rust, yields of beans sprayed with some fungicides may still exceed that of unsprayed beans because of improved micronutrient nutrition or other benefits.

Pathogen Variation

Uromyces appendiculatus is among the most pathogenically variable of all plant pathogens. This variability was first reported by Harter et al. in 1935. The first 20 races were defined in United States in 1941 (Harter and Zaumeyer, 1941) by differential reactions (immune to susceptible) of seven bean cultivars after inoculation with different isolates of the fungus. Host cultivars or lines, the reactions of which are used to differentiate among pathogenic races, are called "differentials."

Variability in *U. appendiculatus* has occurred in many regions of the world, including Australia (Ballantyne, 1978; Ogle and Johnson, 1974), Brazil (Augustin and da Costa, 1971; Carrijo et al., 1980; Coelho and Chaves, 1975; Dias-F. and da Costa, 1968; Junqueira-Netto et al., 1969), Central America (Christen and Echandi, 1967; Vargas-G., 1970, 1971, and 1972), Colombia (Zúñiga de Rodríguez and Victoria-K., 1975), eastern Africa (Howland and Macartney, 1966), Mexico (Crispín-Medina and Dongo-D., 1962), New Zealand (Yen and Brien, 1960), Peru (Guerra and Dongo-D., 1973), Portugal (Rodríguez, 1955), and Taiwan (Yeh, 1983). Intensive studies have identified at least 80 races in Brazil (Augustin and da Costa, 1971; Carrijo et al., 1980; Coelho and Chaves, 1975; Dias-F. and da Costa, 1968; Junqueira-Netto et al., 1969; Vieira, 1983), 65 in United States (Fisher, 1952; Groth and Shrum, 1977; Harter and Zaumeyer, 1941; Stavely, 1984c; Zúñiga de Rodríguez and Victoria-K., 1975), 31 in Mexico (Crispín-Medina and Dongo-D., 1962), 25 in Australia (Ballantyne, 1978; Ogle and Johnson, 1974), 21 in Jamaica (Shaik, 1985b), 18 in Puerto Rico (López-G., 1976; Ruiz et al., 1982), 15 in Taiwan (Yeh, 1983), and 2-8 in other countries (Christen and Echandi, 1967; Guerra and Dongo-D., 1973; Howland and Macartney, 1966; Rodríguez, 1955; Vargas-G., 1970 and 1971). Two to eight races are frequently found in single field collections from a susceptible cultivar (Ballantyne, 1978; Coelho and Chaves, 1975; Groth and Roelfs, 1982b; Stavely, 1984c). Isolation and increase of spores from a single uredosorus is usually necessary to obtain a pure culture that will give a uniform reaction on each differential. Sometimes several successive such isolations are required to achieve purity.

Most authors have assigned successive numbers to each new race. Thus, races 1-57 are now identified in the first series (Fisher, 1952; Harter and Zaumeyer, 1941; Stavely, 1984c; Zúñiga de Rodríguez and Victoria-K., 1975), of which 55 are from United States and two are from Colombia (Zúñiga de Rodríguez and Victoria-K., 1975). In Brazil, race numbers are preceded by capital letters that symbolize the place of origin. Thus, there are 16 B races from Rio Grande do Sul (Augustin and da Costa, 1971; Dias-F. and da Costa, 1968), 26 FM (Ferrugem, Minas Gerais) races (Junqueira-Netto et al., 1969), and 39 V races from Viçosa (Carrijo et al., 1980; Coelho and Chaves, 1975). In Australia, Ballantyne (1978) assigned lower case letters, a through i, to each of ten differentials and named races by letters of the differentials upon which they were virulent. Her race designations are therefore abbreviated virulence/avirulence formulae. Because of the occurrence of intermediate host reactions with bean rust, an arbitrarily assigned level must be used to separate virulence from avirulence. Otherwise an additional designation has to be used for the intermediate reaction. Differential lines, containing one of each of a number of single resistance genes backcrossed separately into a single recurrent parent to create a nearly isogenic set of differentials, are used for some cereal rust fungi. However, much more genetic research is needed before such a set can be developed for bean rust.

An International Bean Rust Workshop, held in Puerto Rico in 1983, developed a standard list of 20 differential cultivars (Table 1) and adopted a standard grading scale for rating host reaction (Table 2). Such standardization aimed to overcome the inconsistencies that had developed over the years in the differentials and grading scales that were employed (Stavely et al., 1983). Most race identifications from 1941 to 1983 used most of the original Harter and Zaumeyer (1941) differentials, but often some were deleted and other cultivars added (Augustin and da Costa, 1971; Ballantyne, 1978; Dias-F. and da Costa, 1968; Fisher, 1952; Pereira and Chaves, 1977). A unique set of differential cultivars was used in Mexico (Crispín-Medina and Dongo-D., 1962). Some cultivars used as differentials from 1941 to 1983 were or had become genetically mixed or heterozygous (segregating for reaction to some races). Hence, the new international set of 20, which has now been reduced to 19 (Stavely, 1984c), has been single-plant selected for several generations to obtain homozygosity (Stavely, 1984c; Stavely et al., 1983). Limited quantities of seed of these differentials are available from the authors of this chapter. Most of the other differential cultivars used from 1941 to 1983 are available in the International Bean Rust Nursery, distributed by the Centro Internacional de Agricultura Tropical (CIAT), Colombia (CIAT, 1979; CIAT, 1985).

Table 1. Cultivars adopted at the 1983 International Bean Rust Workshop, USA, as standard differentials for defining races of *Uromyces appendiculatus*.[a]

U.S. 3	Mexico 235
California Small White 643	Mexico 309
Pinto 650	Brown Beauty
Kennedy Wonder 765	Olathe Pinto
Kennedy Wonder 780	AXS 37
Kennedy Wonder 814	NEP 2
Golden Gate Wax	Aurora
Early Gallatin	51051
Redlands Pioneer	Compuesto Negro Chimaltenango
Ecuador 299	

a. Mountaineer White Half Runner was in the original list but has been deleted because of its similarity to Kennedy Wonder 780 (Stavely, 1984c).

SOURCE: Stavely et al., 1983.

Table 2. The uniform bean rust grading scale adopted at the 1983 International Bean Rust Workshop, USA, with the addition of interpretative symbols for degree of resistance or susceptibility suggested by these reaction grades.

Grade[a]	Definition	Symbol[b]
1	Immune, having no visible symptoms	I
2	Necrotic or chlorotic spots, without sporulation, and less than 0.3 mm in diameter	HR
2+	Spots, without sporulation, 0.3-1.0 mm diameter	HR
2++	Spots, without sporulation, 1.0-3.0 mm diameter	HR
2+++	Spots, without sporulation, greater than 3.0 mm diameter	HR
3	Uredia less than 0.3 mm diameter	R
4	Uredia 0.3-0.5 mm diameter	MR
5	Uredia 0.5-0.8 mm diameter	MS
6	Uredia larger than 0.8 mm diameter	S
2+, 2++, etc. -3, -4, etc.	Necrotic spot of appropriate size surrounding uredosori of appropriate size	R, MR[c]

a. When several reaction grades are present, they are recorded in order of predominance, the most prevalent being listed first and least prevalent, last. Intensity is recorded separately, using the modified Cobb Scale (Stavely, 1985).

b. These symbols have been used at Beltsville for at least 15 years (J.P. Meiners and J.R. Stavely, unpublished data) and the categories resemble Ballantyne's categories (Ballantyne, 1978). Their precise definitions are: I = immune; HR = hypersensitive or highly resistant; R = resistant, reactions having any of the grades 2 with grade 3 present or predominant with some grade 4; MR = moderately resistant, grade 4 predominant and no grade 5 uredia; MS = moderately susceptible, uredia larger than grade 4, but none larger than grade 5; S = susceptible, grade 6 uredia. Another category is VS = very susceptible, grade 6 uredia predominant.

c. This reaction first described by Harter and Zaumeyer (1941) occurs on Kentucky Wonder 780 with many races. It is characterized by a uredium in the center of a necrotic spot. Whether R, MR, or other is determined by the size of uredium as described in footnote b.

SOURCE: Stavely et al., 1983.

By using appropriate inoculation methods (Ballantyne, 1978; Coelho and Chaves, 1975; Davison and Vaughan, 1964; Stavely, 1983 and 1984b) and grading scales, it is possible to determine whether an isolate is already a described race or unique by comparing it with reported races (Stavely, 1984c). When making comparisons with earlier race descriptions, care must be taken since several changes were made in the grading scale from 1941 to 1983

(Ballantyne, 1978; Crispín-Medina and Dongo-D., 1962; Davison and Vaughan, 1963a; Harter and Zaumeyer, 1941). However, these scales have been well enough defined to often permit separation of new isolates from previously described races (Stavely, 1984a).

Control by Plant Resistance

Resistance to bean rust is expressed in many ways (Figure 40). Resistant reactions range from immunity, through various consistent types of hypersensitive, nonsporulating, or sporulating necrotic reactions (necrotic spot with a small, central uredium), to very small, small, or intermediate uredia (Table 2) (Ballantyne, 1978; Harter and Zaumeyer, 1941; Stavely et al., 1983). Different types of cell reactions also occur within the leaf (Mendgen, 1978b). Smaller uredia produce fewer uredospores and, if sufficiently small, have no effect on host yield (Pastor-Corrales and Correa-Victoria, 1983). Genetic studies require use of pathogenically uniform, single uredium isolates (cultures) of defined races (Ballantyne, 1978; Stavely, 1984b and 1984c).

Genetic studies of resistance have shown that reaction grade is controlled by single dominant genes and that there are many such genes in beans (Ballantyne, 1978; Christ and Groth, 1982a; de Carvalho et al., 1978; Grafton et al., 1985; Kolmer and Groth, 1984; Meiners, 1981; Stavely, 1984a and 1984b; Stavely and Grafton, 1985; Zaumeyer and Harter, 1941). *P. vulgaris* has only $n=11$ chromosomes and *U. appendiculatus*, if it is similar to cereal rust fungus, *Puccinia graminis* (McGinnis, 1953), has only about $n=6$ chromosomes. The gene-for-gene relationship has been shown to occur in the *U. appendiculatus-P. vulgaris* host-pathogen interaction (Christ and Groth, 1982a and 1982b). Monogenic, dominant resistance-genes have been identified that are effective against multiple pathogen races (Kardin and Groth, 1985; Stavely and Grafton, 1985). They occur in linkage groups (complex loci) in which there is a single gene for each of many races (Stavely, 1984a and 1984b; Stavely and Grafton, 1985). Some genes are epistatic to other single resistance genes (Kolmer and Groth, 1984; Stavely 1984a and 1984b).

In pedigree and backcross breeding resistance is screened by using several appropriate individual races simultaneously on single plants (Stavely, 1983). In this way, it is possible to "pyramid" two, three, or more such genes or complex loci that are effective against multiple races. Thus, it is possible, by identifying and carefully deploying resistance genes, to develop cultivars with several known genes for resistance to available races and significantly reduce the likelihood of resistance-breaking races developing (Coyne and Schuster, 1975; Schafer and Roelfs, 1985). If virulence and avirulence genes be tightly linked in the pathogen, then resistance may be stabilized by combining as few as two appropriate host resistance genes or linkage groups of such genes (Van der Plank, 1968). However, this is not yet a useful hypothesis, because among the avirulence/virulence genes that have so far been identified in rust fungi no such linkages have been found. A multiline, in which each component line contains a different broadly effective gene or linkage group backcrossed into the same recurrent parent, may also stabilize resistance (Coyne and Schuster, 1975; Van der Plank, 1968).

Should virulence in basidiospores and uredospores be under independent genetic control in *U. appendiculatus*, pathogen variability may be reduced and resistance better stabilized by separately breeding for resistance to the basidiospore stage (Groth and Roelfs, 1982a). However, the same pathogen genes appear to condition virulence or avirulence in both basidiospores and uredospores (Kolmer et al., 1984).

Nearly 70 years ago, a reduced intensity of uredia per unit of leaf area and decreased spore production were recognized as potentially useful forms of resistance to bean rust (Fromme and Wingard, 1921). Of course, if a line has a necrotic, nonsporulating reaction or immunity to a portion of the races present in an area, the uredium intensity will also be reduced. So, a critical first step in assessing any suspected reduced intensity-type resistance is to determine the line's reaction to each race. Some cultivars such as Royal Red Kidney (Groth and Urs, 1982) and Jamaica Red (Shaik, 1985a), have a kind of resistance in which uredial intensity has been reduced with all races tested thus far. This is called "low receptivity" and can be assessed under carefully measured and controlled inoculum concentration, host growth rate, and leaf age (Groth and Urs, 1982). Stomatal density is directly proportional to the number of uredia that develop. However, the sparseness of stomata is apparently not the only cause of low receptivity (Groth and Urs, 1982; Shaik, 1985a). Recent evidence suggests that increased leaf-hair density also reduces the number of uredia by preventing a portion of the uredospores from reaching the leaf surface (Shaik, 1985a). Analysis of the genetic control of stomatal and leaf-hair density may reveal a polygenic mechanism and it may be possible to enhance low receptivity through intensive, careful selection for transgressive segregants.

A longer latent period from infection to sporulation, an important component of so-called "slow rusting," may not be associated with the reduced uredium-intensity type of resistance (Shaik, 1985a), although it is associated with monogenic, small-uredium resistance (Stavely, 1984b). Certain Cuban cultivars are apparently late or slow rusting (González-Avila, 1974).

Vieira (1972) has suggested that in Brazil, where diverse cultivars have been developed locally, there is substantial "horizontal" resistance (equally effective against all races). Eight Brazilian bean lines varied in incubation period, latent period, infection frequency, infection type, and infection intensity against different isolates of *U. appendiculatus*. This suggests that so-called "vertical" (probably single) resistance genes play at least some role in expression of these reactions (Menten and Bergamin-Filho, 1981).

There are several other potentially useful types of resistance to bean rust. Germplasm may vary in length of dew or drying periods and increase in resistance with plant development (Ballantyne, 1974; Berger, 1977). Some cultivars are more heavily infected in lower than upper foliage (Canessa-Mora and Vargas-G., 1977). Rodríguez-Medina (1976) reported that Mexico 309, which has a series of linked monogenic factors for resistance to many races (Stavely, 1984b), is susceptible to race CR-29, but yields as well as cultivars resistant to this race. Tolerance, in which fully susceptible type uredosori occur, but yield is not reduced, would be a most desirable character if methods were found to identify it in the process of developing new cultivars.

Alexander et al. (1985) measured virulence changes in a polymorphic *U. appendiculatus* population over five asexual generations. He found that changes in virulence may be independent of pathogen exposure to host resistance. *U. appendiculatus* frequently carries virulence at a level much higher than the minimum needed for pathogenicity.

Many bean cultivars and lines have been bred for resistance to rust (CIAT, 1979 and 1985; Stavely and Steinke, 1985; Wood and Keenan, 1982; Zaumeyer and Thomas, 1957); for example, such popular cultivars as Olathe, Fleetwood, Aurora, and the CIAT cultivars BAT 48, 73, 76, 93, 308, and 520. Although these cultivars are not resistant to all races of rust, they comprise a significant factor in reducing yield losses from rust.

Table 3. The most rust-resistant cultivars in the International Bean Rust Nurseries from 1975 to 1984; and the percentage of their reactions, according to reaction class across all locations and years.

Cultivar tested in years	Reaction and percentage of occurrence[a]			
	I	HR	R	MR-S
1975-1984				
Redlands Greenleaf B	19.6	45.8	30.8	3.7
Redlands Greenleaf C	16.1	40.4	39.4	4.0
Cocacho	15.9	44.7	33.0	6.4
Mexico 309	40.2	41.1	12.1	6.5
Cuilapa 72-1	29.9	37.4	25.2	7.5[b]
Ecuador 299	18.7	37.4	35.5	8.4[b]
Mexico 235	26.8	35.0	28.9	9.3[b]
Turrialba 4	29.6	27.8	31.5	11.1
Puerto Rico 5	23.4	38.3	26.2	12.1
Compuesto Chimaltenango 3	22.3	50.5	22.3	11.6
Compuesto Chimaltenango 2	31.1	32.0	22.3	14.6[b]
Redlands Autumn Crop	10.3	39.2	35.0	15.5
Turrialba 1	17.9	29.2	34.9	17.9[b]
1976-1984				
Redlands Pioneer	13.0	54.3	29.3	3.3
Mexico 6	11.9	34.5	41.7	11.9[b]

a. Reactions are described in Table 2, footnote b. Percentages are calculated by using only those locations where readings were obtained.

b. Uredia larger than 0.5 mm at one or more locations in 1981 to 1984.

SOURCE: CIAT, 1985.

The International Bean Rust Nursery was established in 1974 and is coordinated by CIAT pathologists (G. E. Gálvez, H. F. Schwartz, and M. A. Pastor-Corrales). It has tested differential cultivars and resistant germplasm worldwide since 1975 (CIAT, 1979 and 1985; Meiners, 1974). No cultivar or line has yet been resistant for all years at all locations in this nursery. The most resistant of the standard entries are listed in Table 3. The most resistant CIAT lines have been the BAT cultivars listed above, which have been tested continuously since 1979. As more is learned about pathogen virulence, pathogen race dynamics, and genetics of host resistance, the potential for developing effective deployment strategies for resistances will lead to more effective control of bean rust.

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