Chapter 8

WEB BLIGHT

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Introduction

Web blight is caused by the fungus Rhizoctonia solani Kühn—the sclerotial, or asexual, stage of the basidiomycete fungus Thanatephorus cucumeris (Frank) Donk. Rhizoctonia solani is a soilborne fungus that is widely distributed throughout the world. Both the sclerotial and basidial stages can initiate the disease, although they cause different symptoms. In most areas of Latin America where blight occurs, the sclerotial stage is significant for the initiation and epidemiology of the disease (Galindo, 1982, Galindo et al., 1982c, 1983a, and 1983b).

Rhizoctonia solani is a pathogen of a large number of host species including bean, beet (Abawi and Martin, 1985), cabbage, carrot, cucumber, eggplant, melon, soybean (O'Neill et al., 1977), tobacco, tomato, watermelon, and many uncultivated plants (Daniels, 1963; Vargas-G., 1973). It also causes a diversity of diseases such as seed decay, root-and-hypocotyl rot, and foliar blight. Although diverse in host range and disease symptomatology, the isolates demonstrate specialization according to their mode of attack. Even though morphologically similar, some isolates cause aerial infection such as web blight of beans, while others attack only roots and hypocotyls (see Chapter 6, p. 107-114).

Web blight is a very important bean-production problem in the humid lowland tropics of Latin America and the Caribbean, where warm to high temperatures and abundant rainfall prevail. The disease also occurs, and can cause severe damage, in middle altitude areas (1200-1600 m.a.s.l.), particularly during rainy weather and

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high humidity. Under field conditions web blight can occur at any stage of the bean-crop cycle and cause severe blight, resulting in rapid defoliation and often complete crop failure (Crispín-Medina and Gallegos, 1963; Galindo, 1982). In the Guanacaste region of northern Costa Rica, a web blight epidemic caused up to 90% reduction of bean yields in 1980 (Se perdió la cosecha de frijol veranero en Guanacaste, 1980).

In Latin America, web blight occurs in the warm, humid, southern, bean-producing areas of Mexico (Crispín-Medina and Gallegos, 1963), all countries of Central America and the Caribbean (Echandi, 1966; Galindo, 1982; Manzano, 1973), and in South America in the Amazon region of Peru and Brazil (Deslandes, 1944; Müller, 1934), the coffee zone of Colombia, and the northwestern region of Argentina (Costa, 1972; Ploper, 1981). Web blight has also been reported in United States, Japan, Philippines, Burma, and Sri Lanka (Weber, 1939; Zaumeyer and Thomas, 1957). The lack of reports of web blight occurrence from African countries suggests that this disease is, currently, of minor importance (CIAT, 1981), although it has been reported from Kenya (Mukunya, 1974) and Malawi (Msuku and Edje, 1982).

Common names used for web blight in Latin America in Spanish include "mustia," "mustia hilachosa," "telaraña," "chasparria," "Rhizoctonia del follaje," and "pringue." In Portuguese, common names include "mela," "mela do feijoeiro," "murcha da teia micélica," and "podridão das vagens."

Etiology

The asexual stage of the web blight fungus, *Rhizoctonia solani*, is distributed worldwide (Baker et al., 1967; Hawn and Vanterpool, 1953; Papavizas and Davey, 1962). This pathogen was originally described as *R. microsclerotia* Matz, although this designation is no longer accepted (Parmeter et al., 1967; Zaumeyer and Thomas, 1957). The current accepted designation for the basidial stage is *Thanatephorus cucumeris* (Flentje et al., 1963b).

Isolates of *R. solani* are highly variable in cultural characteristics, response to environmental changes, and pathogenicity. However, 196



they can be classified into different groups, according to the anastomosis grouping (AG) concept: that is, hyphal fusion occurs only between isolates of the same AG. Earlier researchers showed that the majority of *R. solani* isolates fall into one of four anastomosis groups: AG-1, AG-2, AG-3, and AG-4 (Parmeter et al., 1969). Three more groups, AG-5, AG-6, and AGB1 have since been discovered and AG-2 was recently subdivided into AG2-1 and AG2-2 (Kuninaga et al., 1978).

Hyphal anastomosis groups are not, according to most authors, host specific, although some tendencies are evident (Bolkan and Ribeiro, 1985; Parmeter et al., 1967). Except for AGB1, the anastomosis groups are genetically unique and differ in pathological and cultural characteristics (Bolkan and Ribeiro, 1985; Kuninaga et al., 1978).

Galindo et al. (1982b) characterized 71 isolates of *R. solani* that were obtained from naturally infected bean leaves in different beangrowing areas of Costa Rica. All isolates were pathogenic to leaf and hypocotyl tissues of the bean cultivar Mexico 27, but varied significantly in virulence which was positively correlated to growth rate in culture. Twenty-six isolates belonged to AG-1, 38 to AG-2, and 9 did not anastomose with any of the four AG-4 testers used.

Similarly, Bolkan and Ribeiro (1985) reported that two Brazilian isolates of *R. solani*, obtained from kidney bean leaves, belonged to AG-1, while seven isolates from kidney bean hypocotyls belonged to AG-4. Most of the *R. solani* isolates associated with bean hypocotyls and soils in New York belonged to AG-4. However, some isolates belonged to AG-1 and AG-2, but not to AG-3 (Galindo et al., 1982a). All six aerial isolates of *R. solani* associated with web blight in Colombia were AG-1 (Galindo et al., 1982a).

In addition, the *R. solani* isolates associated with web blight are characteristically fast growing, produce abundant sclerotia, and are intolerant of carbon dioxide. Those associated with seed decay and root-and-hypocotyl rot are characteristically fast growing, produce fewer sclerotia, and are more tolerant of carbon dioxide (Flentje and Stretton, 1964). Parmeter et al. (1967) established that *Rhizoctonia* isolates which possess multimediate hyphae have *Thanatephorus cucumeris* as their perfect stage and those which possess binuclear hyphae have *Ceratobasidium* as the perfect stage.

The following description of Rhizoctonia solani is from Holliday (1980). Colonies on potato dextrose agar (PDA) are at first colorless, rapidly becoming brown. Aerial mycelium is variable. giving a felted or mealy surface on which long, sparsely branched hyphae are frequently present. Some isolates show diurnal zonation. Sclerotia develop as a crust, radiating out from the inoculum center or scattered over the colony surface. Hyphae are usually 5-12 µm wide and up to 250 µm long, with cells at the advancing edge of a colony. Branches form near the distal end of cells, are constricted at the point of origin, and are septate above this constriction. Phase contrast microscopy shows cells are multinucleate (2-25, mostly 4-8), with conspicuous dolipore septa. An older mycelium shows large variation in hyphal dimensions and has shorter cells because of the formation of secondary septa. The branching angle is nearly 90° and branches may arise at various points along the cell length. Some hyphae differentiate into swollen moniliform cells which are 30 µm or more in width. Small (0.2-0.5 mm diameter), immature, superficial, white sclerotia also form and become brown to dark brown, rough, and subglobose with maturity (Weber, 1939). Isolates grown in the laboratory on PDA may differ for growth rate, sclerotial production (Flentje and Stretton, 1964), mycelium color, amount of aerial mycelium, saprophytic behavior, and enzyme production (Papavizas, 1964 and 1965; Papavizas and Ayers, 1965).

The basidial stage, Thanatephorus cucumeris, was first discovered in beans in the USA by Weber (1939) who reported that mycelia and sclerotia from both asexual and sexual sources were indistinguishable. Basidial fructifications appear whitish and form on top of a hymenium which is a thin sheet or collar commonly found on stems or leaves just above the soil surface or on soil particles. It is discontinuous and composed of barrel-shaped subcylindrical basidia, 10-25 µm long x 16-19 µm wide, arranged in imperfect cymes or racemes. The short basidia bear stout, slightly divergent sterigmata, usually four in number, but can have two to seven per basidium. They are 5.5-36.5 µm long and occasionally have adventitious septa. Hyaline basidiospores, produced on the sterigmata, are oblong to broadly ellipsoid, unilaterally flattened, prominently apiculate, smooth, and thin walled. They measure 6-14 µm x 4-8 µm and germinate by repetition.

The fungus grows rapidly in continuous, indirect, or intermittent light. Within 24-36 hours it can cover the surface of a 9-cm petri dish containing artificial media incubated at 26-29 °C. Sclerotia form in culture but differ from those produced on host plants which are brown to dark brown, and more irregular in form and size (as large as 1 cm in diameter), and more or less flattened (Weber, 1939). Heterokaryosis occurs in *T. cucumeris* and may alter its ability to form sclerotia on minimal media or to form isolate pathogenicity and variants (Flentje and Saksensa, 1957; Flentje et al., 1963a and 1967; Gálvez and Cardona-Alvarez, 1960; McKenzie et al., 1969; Meyer and Parmeter, 1968).

The perfect stage of the web blight fungus can be induced in vitro (Flentje, 1956; Stretton et al., 1964; Tu and Kimbrough, 1975) with 12-16 hours of light (Flentje et al., 1963b; Stretton et al., 1964; Weber, 1939; Whitney, 1964), adequate aeration (Whitney, 1964), 20-30 °C, and 40%-60% relative humidity (Stretton et al., 1964; Weber, 1939). Self-sterile mutants frequently appear in progenies of basidiospores (Stretton et al., 1967; Whitney, 1964). Isolates of *Rhizoctonia solani* vary for their cultural characteristics and ability to fruit on artificial media or sterilized soil (Houston, 1945; Olsen et al., 1967; Stretton et al., 1964). For example, pathogenic isolates of *T. cucumeris* fruit only on sterilized soil, while nonpathogenic isolates fruit on either substrate (Stretton et al., 1964).

Epidemiology

Web blight epidemics are favored by rainy weather, high (30 °C) to moderate (20 °C) air temperature (average 25-26 °C), high to moderate soil temperature, and high relative humidity of at least 80% (Galindo, 1982; Galindo et al., 1983b; Weber, 1939; Zaumeyer and Thomas, 1957). The main sources of inocula that can initiate infection are sclerotia and mycelium fragments, either free in the soil or present on colonized debris. Bean plants are inoculated by the web blight pathogen when raindrops splash soil particles, infested with sclerotia or mycelium, onto plants (Galindo et al., 1983b; Prabhu et al., 1982).

Basidiospores can also cause infection (Echandi, 1965). However, in most locations with abundant rain and endemic web blight,



basidiospores do not contribute significantly to epidemic development, particularly when lesions from basidiospore infection appear late in the crop cycle (Galindo et al., 1983b). Infected bean seed can disseminate the pathogen over long distances, introduce it into new fields, or act as a source of primary inoculum. When rain-splashed sclerotia and mycelium are the main source of inoculum, initial symptoms of web blight always appear on primary leaves two weeks after planting.

The mycelium of the fungus first grows on the soil particles splashed onto bean leaves and then advances to adjacent healthy tissue, causing primary or initial infections. Trifoliolate leaves are usually infected by hyphal strands growing from infected primary leaves, but can also be infected by rain-splashed soil. Infected leaves rapidly become covered by small sclerotia of the fungus. New sclerotia also form, beneath the canopy, on fallen leaves and the soil surface within 24 hours. After trifoliolate leaves are infected, plant-to-plant infection occurs through direct hyphal growth from previously infected leaves (Galindo et al., 1983b).

Basidiospores are dispersed during the night (Echandi, 1965) and remain viable for only a few hours. Sclerotia can remain viable in soil for several years and can survive as vegetative mycelium within plant residue (Weber, 1939).

Symptomatology

Web blight symptoms initiated by rain-splashed sclerotia or mycelium fragments differ from those elicited by basidiospores. Sclerotia germinate during periods of favorable environmental conditions by producing hyphae, a few mm in length, that branch profusely until they reach host tissue. An infection cushion then develops and penetration occurs directly or through stomata (Dodman et al., 1968; Weber, 1939). Subepidermal hyphae develop inter- and intracellularly. Lesions first appear on the primary leaves as small necrotic spots (5-10 mm in diameter) with brown centers and olive-green margins. These lesions resemble hot-water scalds. Under favorable environmental conditions, high humidity, and warm temperature, they progress very rapidly but appear irregular and somewhat zonate (Figure 41). Under dry conditions, their

development stops. Often these lesions coalesce and affect the entire leaf. Infected leaves rapidly become covered by small sclerotia and mycelium.

The light-brown superficial hyphae spread in a fan-shaped manner on either leaf surface. Hyphae may grow rapidly over healthy leaves, petioles, flowers, and pods (Figure 42), eventually killing plant parts or covering the entire plant with a web of mvcelium (Figure 43). Small brown sclerotia (Figure 44) form three to six days after infection (Galindo, 1982; Weber, 1939; Zaumeyer and Thomas, 1957). The many lesions produced by basidiospores are distinct, small, necrotic, circular, and measure 2-3 mm in diameter (Figure 45). They are light brown or brick red with a lighter center. Under humid and rainy conditions, these round spots fall from the leaf surface, resulting in a symptom known as "cock's eye." These lesions usually do not enlarge much, nor coalesce to form large lesions, and seldom cause defoliation. Pod lesions caused by sclerotia, mycelium, or basidiospores are similar to foliage lesions. Pod lesions initiated by basidiospores are also small, circular, and have light-brown centers surrounded by a reddish brown darker border.

Bean pods may become infected during the grain-filling stage. Young pod infections appear as light-brown, irregular-shaped lesions which frequently coalesce and kill the pod.

Seeds can become infected in the endosperm and radicular end of the embryo and on the seed-coat surface (Baker, 1947; Cardoso et al., 1980; Leach and Pierpoint, 1956; Le Clerg, 1953).

Control by Cultural Practices

Control by cultural practices includes planting seed free of internal or external contamination, sanitation of infected crop debris, and crop rotation with nonhosts such as tobacco, maize, and grasses. A most effective cultural practice is mulching. Mulch forms a barrier and impedes the splashing of pathogen propagules from the soil to plant tissues. Under experimentation, effective mulches are rice husks, maize leaves, sugarcane leaves, or standing weeds killed by herbicides 15 days after planting (Galindo et al., 1982c and 1983b;

Rosado-May, 1982; Rosado-May and García-Espinosa, 1985). Examples of preemergent herbicides used to kill weeds for mulching are paraquat or glyphosate (1 kg/ha) (Galindo et al., 1983a). Postemergent herbicides such as fluazifop-butyl (1 kg/ha) and bentazone (0.75 kg/ha), can be used for broad-leaved weeds. Obando (1983) and Sancho (1984) established that, for an effective, integrated, control of the pathogen, preemergent applications of paraquat, pendimethalin, and glyphosate can be used in association with foliar applications of the fungicide benomyl.

Small subsistence bean farmers in Costa Rica and Nicaragua rely upon a similar practice known as "frijol tapado" (covered beans). This practice consists of broadcasting bean seeds into plots with established weeds and cutting the weeds down to cover the seeds as a plant mulch. By using herbicides, a standing weed mulch can be created (Galindo et al., 1982c). Indeterminate cultivars grow through the mulch and eventually cover it, effectively preventing new weed growth and conserving soil moisture. In addition, the mulch prevents the splashing of infected soil. This practice is effective, even in areas where the climate is optimal for web blight development (Galindo, 1982; Galindo et al., 1982a, 1982b, and 1982c, 1983a, and 1983b). However, mulches may create more favorable conditions for slug infestation and resulting crop loss in some production regions.

Where farmers have more resources, beans should be planted in spaced furrows (Corrêa, 1982; Weber, 1939, Zaumeyer and Thomas, 1957) which will maximize air circulation and improve microclimatic conditions. Intercropping beans in relay or in association with maize will also reduce disease severity (Msuku and Edje, 1982; Rosado-May, 1982).

Control by Chemicals

Benomyl (0.25-0.5 kg/ha) helps manage the pathogen when it is applied at first-symptom appearance and then every 15 days (Cardoso, 1980; Cardoso and de Oliveira, 1982; Manzano, 1973; Oliveira et al., 1983). The chemicals protect plant foliage from infection by inoculum from nearby infested soil. Fentin acetate (0.16 kg/ha) or fentin hydroxide (0.20 kg/ha) applied after benomyl 202

(Cardoso and de Oliveira, 1982), gives good control. Thiophanate-methyl (0.5 kg/ha), carbendazim (0.5-1.0 kg/ha), and captafol (1.0-3.5 kg/ha) (CIAT, 1975; Manzano, 1973) are also useful. The use of systemic fungicides is important where rains prevail. However, expense may limit their use, even though recent work has shown that two or three applications are sufficient to control mild infections (Villalobos-Pacheco, 1985).

Control by Plant Resistance

Cultivars differ in their reaction to the web blight pathogen under field conditions. Susceptible cultivars exude chemicals which stimulate the formation of infection cushions whereas resistant or tolerant cultivars do not exude these chemicals (Flentje et al., 1963a). Although various cultivars have low levels of resistance to the web blight pathogen (Manzano, 1973; Weber, 1939), there are no reports of cultivars with high resistance or immunity. The Centro Internacional de Agricultura Tropical (CIAT), in collaboration with the national bean programs of Colombia and Costa Rica, has identified bean cultivars with some resistance to web blight. These are: Turrialba 1, Porrillo 70, Porrillo Sintético, S-630-B, and Talamanca (Mora and Gálvez, 1979). Crosses with these cultivars have produced progenies exhibiting resistance such as Negro Huasteco 81, Huetar, HT 7716, and HT 7719, which are superior to the resistant parents.

Integrated Control

The most practical approach to manage this very serious and damaging disease is by using an integrated management strategy. Such strategy is based upon cultural practices, complemented by judicious use of chemicals, and, where possible, use of resistant cultivars. This involves using clean seed, eliminating pathogen-infested crop debris at harvest, wide row-spacing (Corrèa, 1982; Weber, 1939; Zaumeyer and Thomas, 1957), planting resistant cultivars with erect architecture to permit greater air circulation, mulching and minimum tillage, applying fungicides, and rotating with nonhost crops such as cereals and vegetables. Such practices can offer an economic, efficient, and practical control of web blight.

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