Mini Review

Physiological Aspects of Resistance to *Botrytis cinerea*

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Accepted for publication 3 April 1995.

Botrytis cinerea Pers.:Fr., the causal agent of gray mold, inflicts serious food and ornamental crop losses, particularly after harvest (57). Disease control is difficult, because the pathogen can attack crops at any stage of growth and can infect all plant parts. *B. cinerea* is especially important as a postharvest pathogen, because environmental conditions and the susceptibility of the crop tend to favor its development. Many of the strategies currently used to control gray mold, particularly the use of fungicides, kill or inhibit growth of the pathogen. As the application of fungicides becomes more restricted and fungicide resistance in pathogen populations becomes more widespread, the identification and manipulation of host disease-resistance mechanisms will become increasingly important in minimizing losses.

The natural defenses that protect plants from *B. cinerea* are many and diverse. These defenses can be strengthened by cultural practices and management of preharvest and postharvest environments. Genetic variation for resistance to *B. cinerea* has been observed within species, but no gene-for-gene resistance has been identified. Development of resistant genotypes will depend on successful identification, characterization, and transfer of resistance traits.

DELAYING SENESCENCE INCREASES RESISTANCE

B. cinerea is an opportunistic pathogen that easily invades weak, damaged, or senescent tissues. Resistance to gray mold declines with aging or ripening, particularly in fruit and flowers (57). Senescence is composed of many processes, and it is unknown which of these are important for *B. cinerea* resistance. Here we will consider how several changes associated with senescence may be related to host plant resistance.

Membrane Changes During Senescence

Activated oxygen species arising from deteriorating membranes may be involved in infection of plant tissues by *B. cinerea*. Activated oxygen species and free radicals are produced by both the host and pathogen during infection (28,31,99). In some host-pathogen systems, these compounds serve as part of the signal transduction mechanism for resistance responses (18,53). When inducible defense mechanisms are not present, activated oxygen species most often assist the development of the pathogen. These species react with cell components, initiating a self-propagating series of free-radical reactions. Free radical-induced lipid peroxidation is considered an important mechanism of membrane deterioration during aging (25,29). In solution cultures of *B. cinerea*, the ability of cultures grown on different substrates to generate H_2O_2 is correlated with their ability to infect leaves (31).

Additional keywords: calcium, ethylene, pathogenesis, senescence

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Activity of the free-radical scavenging enzyme peroxidase is higher in infected than in uninfected rose petals (O. Shaul and Y. Elad, *unpublished data*). In old, injured, or senescing plant parts, membrane deterioration may already occur (29,74). The free radicals generated during this process may damage membranes and increase the susceptibility of the plant to the pathogen. Membrane damage increases leakage of nutrients to the petal surface, where they support growth and penetration of the fungus, and into the apoplast, where postpenetration growth occurs. Changes in the composition of the apoplast solution may contribute to cell wall hydrolysis and the penetration of toxins into host cells, causing more rapid cell death (24). The deteriorating membrane itself releases compounds, such as hydrogen peroxide or other products of lipid peroxidation, that increase the susceptibility of the host to disease development.

It is apparent that free-radical scavenging and antioxidant systems in the infected tissue are insufficient to prevent an increase in free radicals, whether they are generated by the pathogen or produced by the host cells. External scavengers of free radicals reduce gray mold in several hosts, and antioxidants reverse the promotive effect of hydrogen peroxide on gray mold (35). It is possible that strengthening the antioxidant systems in plants could improve resistance to *B. cinerea*. However, these systems are complex, involving many enzymes, each with several possible functions in plant metabolism. Likewise, little is known about the causes of membrane oxidation during senescence. Overexpression or gene-disruption techniques could be used to determine the importance of particular antioxidant enzymes in host-pathogen interactions.

Ethylene and Host Susceptibility to *B. cinerea*

Ethylene is often produced in plant tissues in response to pathogenic attack (1). Although some pathogenic fungi can produce ethylene when grown on defined media, ethylene production during *B. cinerea* infection is most often attributed to the host plant (5,55). The evidence for this is mostly circumstantial. *B. cinerea* produces no detectable ethylene when grown on agar or on autoclaved flower tissue supplemented with methionine (32). Aminoxyacetic acid, an inhibitor of ethylene production by plants, but not fungi (1), inhibits ethylene production during *B. cinerea* infection of tomato fruits (5) and rose petals (32).

Fungal infections, like other biotic and abiotic stresses, stimulate ethylene production in plants (1). For example, healthy rose flowers produce very little ethylene, whereas *B. cinerea*-infected flowers show a dramatic increase in ethylene production. The amount of ethylene generated is correlated with the development of necrosis (32). Ethylene treatment increases the susceptibility of a variety of fruits, vegetables, and flowers to *B. cinerea* (5,32).

How does ethylene production promote the development of gray mold? It is possible that *B. cinerea*-induced production of ethylene accelerates senescence processes that favor disease development. Rose flowers become more susceptible to gray mold with aging (32). Ethylene-accelerated senescence in many flowers,

including roses, is associated with increased membrane permeability (43). Treatment with antioxidants reduces ethylene production and reverses the promotion of gray mold caused by ethephon (an ethylene-releasing compound) (35), suggesting that ethylene promotes oxidative reactions in membranes and that membrane oxidation enhances ethylene production and action.

Ethylene also communicates information directly to the pathogen. Ethylene production related to ripening serves as a signal for germination and multiple appressorium formation for *Colletotrichum* sp. strains capable of invading climacteric fruit (44). Ethylene stimulates germination and hyphal growth of *B. cinerea* and other fungal pathogens (63,64), sometimes by interaction with other plant volatiles (30).

This suggests that one strategy to improve host resistance would be to reduce the ethylene response. Ethylene scrubbing and inhibitors of ethylene action or production inhibit gray mold development (32,36). Species and cultivars vary in the degree and type of ethylene response (111). Carnation cultivars with reduced ethylene production or reduced ethylene sensitivity have been identified (110,112), indicating that genetic variability for ethylene response traits is available. However, these plants have not been tested for their resistance to *B. cinerea*. Inhibition of ethylene biosynthesis in tomato plants, using antisense technology, results in delayed senescence and fruit ripening (75,78). Such inhibition would be expected to maintain resistance to *B. cinerea*.

Effects of Other Hormones Correlate with Effects on Senescence

Gibberellic acid (GA_3) inhibits development of gray mold in rose flowers (90). Since GA_3 inhibits the senescence-related increase in permeability of the petal cell membranes (85) and delays senescence of roses when applied as a postharvest treatment (47), it is likely that the effect on disease resistance is based on delaying senescence. Auxins and cytokinins also increase resistance to gray mold in roses at concentrations that do not affect ethylene production (Y. Elad, *unpublished data*).

Abscisic acid (ABA), a plant hormone associated with dormancy and stress responses, accelerates petal senescence and increases ethylene sensitivity (12). Endogenous ABA levels increase in rose flowers during postharvest aging (9,12). ABA is produced in culture by *B. cinerea* (52,76) and may be produced by the pathogen during infection as well. ABA may act by accelerating senescence, and it reverses the disease-suppressing action of GA_3 (91).

BARRIERS TO PATHOGEN GROWTH

The Cuticle as a Barrier

Germination of *B. cinerea* conidia depends on the microenvironmental conditions of the phylloplane, particularly water and nutrient availability. Since free water is required for germination (16,56), one of the primary strategies for controlling postharvest infections is to avoid condensation. An intact cuticle prevents diffusion of cellular solutions, limiting water and nutrient availability on the plant surface. The hydrophobicity of cuticular waxes reduces the likelihood of rain, irrigation water, or condensation accumulation on plant surfaces.

The cuticle provides a significant barrier to penetration by *B. cinerea*. Wounding and treatments that disrupt or dissolve the cuticle result in more rapid infection by *B. cinerea* and other phytopathogenic fungi (10,51,70,98). The cuticle may function as a chemical, as well as a physical, barrier since it reportedly contains substances antagonistic to fungi (73). Cuticle thickness has been correlated with resistance to gray mold in tomato fruits (83) and roses (49). Thicker cuticles might impart resistance in several ways: by providing mechanical resistance to penetration (54), by presenting a greater quantity of substrate to be degraded by cutinase and other enzymes, and by reduced permeability resulting in less diffusion of nutrients to the phylloplane (73). Nutrients influence germination, surface growth, and infection processes in *B. cinerea* (19).

Approaches to increasing resistance to *B. cinerea* might be targeted at increasing the effectiveness of the cuticle as a physical and chemical barrier. Evidence accumulated so far indicates that *B. cinerea*-penetration and -postpenetration growth rates, but not germination rates, are lower on resistant cultivars (19,49,79), suggesting the importance of the epidermal (including the cuticle) barrier. Cuticles that are thicker, that are more resistant to cracking, and that lack pores or other areas more easily accessed by pathogens would certainly improve resistance. For example, raspberry cultivars resistant to *B. cinerea* have hairy, waxy canes, and their resistance is attributed to greater runoff of water (57).

Fungal pathogens are believed to penetrate the cuticle through a combination of enzymatic and mechanical forces. Cutinase, which hydrolyzes the primary alcohol ester linkages of the cutin polymer (65), has been found in many plant-pathogenic fungi, including *B. cinerea* (92). The importance of cutinase activity for penetration of the cuticle is still a matter of debate (26,27,69, 88,96). Studies of the importance of cutinase are complicated by the likelihood that cuticle penetration is more important for some host-pathogen systems than for others. In a study of *B. cinerea* cutinase inhibition, treatment of inoculated gerbera flowers with a monoclonal antibody against cutinase from *B. cinerea* reduced lesion formation by up to 80% (86). Whether plants have any natural defenses conferring resistance to disease by specific inhibition of cutinases is unknown. It is possible that compounds are present in the cuticle that inhibit fungal cutinase activity. Natural or artificial inhibition of cutinase might be useful in control of gray mold and other diseases (69).

The Cell Wall as a Barrier

The frequency of latent (quiescent) infections in young fruits and flowers indicates that defenses beyond the cuticle are very important. In one study, more than 50% of symptomless roses collected from commercial greenhouses in Israel showed the presence of latent infections (33). These developed into active disease spread when the flowers were incubated under favorable conditions. In latent infections, the fungus has penetrated the cuticle but failed to produce disease (spreading necrosis). Although the environment is important in determining whether latent infections will develop further (33), resistance mechanisms also must be present to account for the development of disease in some cases but not in others. Some of these resistance mechanisms may be encountered when *B. cinerea* invades the host cell wall.

Once the plant cuticle has been breached, or shortly after germination and germ tube extension in wounded or senescent tissues, the fungus secretes enzymes that degrade the cell walls of the host. Since the development of the spreading necrosis that characterizes gray mold may be delayed in the case of quiescent infections, the plant cell wall should be considered the second barrier to infection of intact tissue. In some host and nonhost species, papillae (host cell wall thickenings that may include callose, lignin, and tannins) are formed in the cell walls at the site of penetration, restricting growth and further penetration of *B. cinerea* hyphae (71,83). A more typical response is that of rose petals, in which the fungal hyphae penetrate the cuticle, the cuticle separates from the cell walls, and degradation of the plant cell walls occurs in advance of the growing hyphal tips (49,80).

Cell wall-degrading enzymes have been demonstrated in *B. cinerea*-infected tissues from many plants and in cultures of *Botrytis* spp. on media containing plant cell walls (19). These include pectin methyl esterase, endo-polygalacturonase (PG), exo-PG, cellulase, pectin lyase, xylanases, arabinase, β-glucosidase, β-galactosidase, β-mannosidase, and α-galactosidase (4,61,101,106). The role of cell wall hydrolases in plant disease is well established, and some of the genes encoding these enzymes in other plant pathogens (including several bacteria and a few fungi) have been cloned (3,20,23). The evidence for the importance of hydro-

lases in gray mold disease is mostly correlative (67,87) or analogous with other plant pathogens (20).

The ability of *B. cinerea* mycelia grown in liquid culture to produce the cell wall-hydrolyzing enzymes found in infected plant tissue suggests that the fungus is the primary source of these enzymes. In fruit, it is possible that gray mold accelerates the production of host hydrolytic enzymes associated with ripening, perhaps via induction of ethylene synthesis. Likewise, the action of plant hydrolases may induce production of fungal hydrolases, because production of these enzymes in fungi is stimulated by the presence of galactose and other soluble materials released from the plant cell wall (104).

The most obvious role of the cell wall-hydrolyzing enzymes is to degrade cell walls and release substrates that serve as nutrients for this necrotrophic fungus. There is evidence that cell wall hydrolysis creates osmotic stress on the protoplasts, resulting in cell death (6,97). However, it also is possible that cell death results from the release of toxins or signals from the cell wall.

Susceptibility of the cell wall to degradation by cell wall hydrolases is probably of major importance in determining severity of gray mold. Abundant calcium reduces gray mold on several crops (39,42,108). The ability of *B. cinerea* to produce PG and thrive on Na pectate as a sole carbon source is reduced if $CaCl_2$ is added to liquid cultures (108). Similarly, decay of apple cell walls by PG isolated from *Penicillium expansum* or *B. cinerea* is retarded in fruits treated with calcium (21,22). During development of gray mold, slower cell wall hydrolysis would presumably reduce the rate of cell death and the development of lesions. Selecting plants whose cell walls are more resistant to enzymatic degradation, either because the calcium content is higher or because the physico-chemical structure presents a more challenging substrate, would probably improve resistance to *B. cinerea*.

Another factor determining the resistance of plant cell walls to degradation by fungal hydrolases is the presence of proteins that specifically inhibit endo-PGs secreted by fungi. A polygalacturonase-inhibiting protein (PGIP) in raspberry fruit declines with fruit ripening, correlating with the increase in susceptibility to fungal decay (60). Cervone et al. (17) suggested that the reduced rate of depolymerization by endo-PG in the presence of PGIPs results in accumulation of greater quantities of oligogalacturonides with a degree of polymerization that activates plant defense responses. However, recent work has shown that PGIP from pear cells does not prevent the hydrolysis of pear cell wall polyuronides to chain lengths below those that elicit ethylene production and defense responses, apparently because PGIP inhibits some but not all *B. cinerea* PGs (89). Although the role of PGIPs in defense may be limited to retarding the action of certain fungal PGs, they could still be useful in breeding for improved resistance.

CONSTITUTIVE AND INDUCIBLE DEFENSES

Limiting the Spread of *B. cinerea* Within Host Tissue

Plant tissues can form papillae and accumulate lignin, suberin, and callose to limit the growth of *Botrytis* in host tissue (71). Papillae formation is a nonspecific response and may be important in nonhost resistance. Cell wall thickening may be followed by other chemical changes in tissues around the infection site, increasing tissue resistance to maceration and penetration (19). For example, a factor from *B. cinerea* cultures can induce defense responses, including suberization, lignification, and accumulation of phytoalexins in carrot (50). These responses are similar to those occurring after mechanical wounding, which also confer resistance to opportunistic pathogens such as *B. cinerea* (13). The degree of disease resistance is related to both the magnitude and the speed of these wound-healing responses (13). During postharvest handling of fruits and vegetables, temperature and humidity are manipulated to encourage wound-healing reactions and reduce storage rots. Genetic variation for traits related to wound responses has been related to disease resistance in some species (13). Wound response rate could be used as a selection criterion in breeding programs.

In pears, the pedicel abscission layer creates a barrier to the spread of *B. cinerea* from the flower into the fruit (57). However, senescing flower parts are important sources of latent infections on raspberries and strawberries (104). This suggests a strategy for reducing latent infections in some fruit crops, i.e., production of plants that promptly abscise their flower parts after fruit set. This would replace the saphrophytic base from which young fruits are infected through an abscission scar, which develops a wound-healing response similar to that described above. Indeed, plum and nectarine flowers, which do not remain attached to young developing fruit, are not a source of latent infections (45).

Latent infections may be held in check by preformed or active physical and chemical defenses, but these are typically overcome when conditions become more favorable to the pathogen. Most latent infections in fruit become active during ripening, but ghost spot disease in tomato fruit is an interesting exception. Ghost spot occurs when *B. cinerea* successfully penetrates young fruit and creates a visible lesion, but further spread is prevented, even during fruit ripening (19). This limitation of tissue maceration has been linked to the accumulation of lignin, phenols, and glycoalkaloids. Tomato fruit is unusual in that phenols increase during ripening (105).

Prolonging quiescence of latent infections would reduce losses to gray mold, especially after harvest. Cultural and storage practices are crucial, since reactivation of latent infections has been linked to temperature, moisture levels, and nutrition (58). There are undoubtedly genetic determinants of quiescence as well, some of which might be responsible for observed differences in disease resistance among cultivars.

Pathogenesis-Related and Other Defense Proteins

Plants may respond to pathogen invasion by deploying several inducible defenses that slow disease spread. Some of these defenses may involve production of a group a proteins, collectively called pathogenesis-related (PR) proteins, that are induced in plants upon attack by pathogens and that are associated with necrotic reactions (103). Some of the PR proteins have been identified and characterized, including chitinases and 1,3-β-glucanases, which are thought to function by attacking fungal and bacterial cell walls (82,94). (*B. cinerea* has chitin and 1,3-β-glucan in the cell walls [46]). Chitinase increases during *B. cinerea* infection as well as in response to other fungal pathogens, bacteria, viruses, nematode feeding, ethylene, biotic and abiotic elicitors, and a variety of other treatments (82). Soluble fragments released from the fungal walls as a result of these activities can further enhance host defense responses (82,94).

There are other proteins that may be important in host resistance to *B. cinerea* (review in [14]). Protease inhibitors are widely distributed in plants and probably have a function in plant protection against herbivores, fungi, and bacteria (84). They have antinutrient effects on the digestive systems of animals and inhibit germination and growth of several fungi, including *B. cinerea* (68,84). Hydroxyproline-rich glycoproteins (HGRP) increase in plants in response to stress and during fungal infections. HGRP accumulation has been linked to host resistance to several fungal diseases (93). They may function by assisting in the formation of a protective barrier or by immobilizing pathogens (93). There are several groups of small, cysteine-rich proteins in plants that play a role in fungal pathogenicity and specificity (100). If these proteins are shown to be important for resistance to *B. cinerea*, then it may be possible to develop or genetically engineer plants with enhanced expression of the genes encoding these proteins.

Production and Detoxification of Chemical Defenses

Plants contain many secondary metabolites with antifungal activity, including glucosinolates, nonprotein amino acids, phenolic compounds, and phytoalexins. These compounds are likely to be important for both constitutive and inducible defense (recently reviewed by Bennett and Wallsgrove [8]). Their effectiveness in containing infection depends on several factors, including concentration, speed of induction, contact between pathogen and chemical, and whether the pathogen can tolerate, detoxify, or prevent synthesis of defense chemicals (8,102). For example, the phytolalexin lettucenin A accumulates in lettuce after infection with several fungal pathogens, including *B. cinerea*. When lettuce leaves are inoculated with *B. cinerea* conidia, lettucenin A accumulation is associated with limited lesions restricting the spread of disease, but when leaves are inoculated with mycelial suspensions, lettucenin A accumulation appears to be overcome by degradation processes, and the disease spreads (7).

A laccase (a polyphenol oxidase) produced by *B. cinerea* may be involved in inactivating host defenses. Some cucurbits are capable of producing compounds called cucurbitacins, which inhibit fungal laccase and result in decreased infection of cucumber fruits (107). Although the substrates of this laccase are unknown, it may be involved in disrupting lignin synthesis (a barrier to pathogen penetration) by oxidizing its substrates or changing its metabolism (107). When cucurbitacins are absent, the host may be unable to form a protective barrier in time to stop pathogen spread (107).

It is well-known that fruit become more susceptible to gray mold as they ripen, resulting in new infections or in the activation of latent infections. Part of the increased susceptibility undoubtedly results from the senescence changes discussed previously and from cell wall degradation associated with fruit softening. However, the loss of polyphenolic compounds such as tannins and proanthocyanidins (59) also may be important. In grapes, proanthocyanidins competitively inhibit activity of a laccase produced by *B. cinerea*, but only in the presence of tannins, which decline during ripening (77). Inducible defenses may operate differently in ripening fruits. Inoculation of freshly harvested (unripe) avocado fruit with a nonpathogenic mutant strain of *Colletotrichum magna, path-1*, inhibited subsequent decay development by the pathogen *C. gloeosporioides. Path-1* induced higher levels of epicatechin that persisted in ripening fruits, whereas epicatechin levels in fruits inoculated with *C. gloeosporioides* persisted for only 1 day (81). Epicatechin inhibits the activity of fungal cell wall-macerating enzymes (109) and plant lipoxygenase, which degrades a preformed antifungal diene found in unripe avocado fruit (81).

The observation that pathogens are often susceptible to phytoalexins from plants other than their hosts suggests that one approach to disease control might be to develop plants that produce different kinds of phytoalexins (102). Another possible approach would be to produce plants with the ability to disrupt the detoxification mechanisms employed by phytopathogenic fungi. The limitations of these approaches are discussed by VanEtten et al. (102).

Induction of Generalized Defenses

The induction of general defense responses by nonpathogens or abiotic stress improves resistance to disease. Limited infection or treatment with certain chemical agents increases resistance to several fungal, bacterial, and viral diseases (66). This resistance is associated with the deployment of generalized resistance mechanisms, such as accumulation of phytoalexins and PR proteins, resulting in systemic resistance to a variety of pathogens (66). A key feature of this resistance is the rapid activation of existing resistance mechanisms at the onset of infection.

Research on systemic responses and induced immunity has been conducted with a variety of host-pathogen combinations, but only a few studies have investigated the use of induced resistance to control gray mold. Heat treatments of flowers in warm water (20 to 40 s at 50°C) reduce postharvest *B. cinerea* blight of roses (40). Unlike longer exposures used for gray mold control in fruits and bulbs, the treatment induces host resistance rather than inhibiting the pathogen directly. Moderate heat treatments increase the activity of superoxide dismutase and peroxidase in tomato fruit (S. Lurie, *personal communication*). Recently, several saphrophytic microorganisms were identified that reduce the severity of gray mold, at least in part, by inducing local resistance (37,38). More research should be done to determine whether nonpathogens or abiotic stresses could be used to improve resistance to *B. cinerea*.

GENETIC AND ENVIRONMENTAL EFFECTS

Genetic Resistance

Genetic resistance to *B. cinerea* has been introduced into several crops, including strawberry and grape. However, the mechanisms of resistance are diverse. In strawberry, the anthers provide a major route for infection, so pistillate flowers produce fewer infected fruit (95). Resistant cultivars of grape accumulate phytoalexins, and their cuticles have fewer and smaller pores (2). Some resistant lines of kenaf (*Hibiscus cannabinis*) have a hypersensitive response to infection (15). Faba bean breeders have found that resistance to chocolate spot (caused by *B. cinerea* and *B. fabae*) is complex, and it is difficult to transfer the resistance to another genetic background. However, breeding for early flowering (disease escape), slow leaf senescence, and open canopy (reduced humidity) leads to a reduction in disease incidence (11).

Breeding for resistance to *B. cinerea* is difficult because different resistance mechanisms are involved at different developmental stages. Each stage of development must be assessed separately to identify and transfer resistance traits (11). The resistance response is influenced by plant genetic and environment backgrounds; resistance expression is not stable across genotypes and environments (11). To succeed in breeding for resistance, identification of traits highly correlated with the resistance response is essential. Traits such as the ability to accumulate certain phytoalexins, the rate of suberin deposition after wounding, or the rate of senescence of leaves or flowers could be scored along with traditional measures of disease incidence and used to identify genetic markers closely associated with resistance by quantitative trait loci analysis.

Environmental Conditions

One of the most important means of reducing losses due to *B. cinerea* is control of the environment in the field and greenhouse and after harvest. Like other fungal pathogens, *B. cinerea* is slowed by low temperature and humidity. During greenhouse crop production, temperature must be kept high during the day to support crop productivity, but managing night temperatures in the greenhouse can reduce *B. cinerea* infections (113). During the postharvest period, temperature management is crucial to delay senescence (and, therefore, increased host susceptibility) as well as development of the pathogen.

In the field or greenhouse, humidity is controlled by plant spacing, management of canopy architecture, heating, ventilation, and irrigation practices. It is particularly important to avoid periods of free water on plant surfaces, because this allows *B. cinerea* spores to germinate (34,113). Periods of high humidity coincident with suitable temperatures favor the development of gray mold outbreaks (113). After harvest, high humidity is usually provided to prevent excess water loss from the crop, and this also favors the development of gray mold. Curiously, a high humidity environment favors the reactivation of quiescent infections, even though the fungus is already inside the host tissue, where the microenvironment is presumably very humid at all times. There is some evidence that the water content of the host tissue can affect infection or development of latent infections (57).

It has been suggested that very humid conditions favor active spread of lesions by facilitating the diffusion of toxins or enzymes within the host tissue (57,62). If this is true, then high external humidity should be more important for rapidly transpiring tissues like leaves and flowers than for fruits and vegetables with a low transpiration rate.

Microclimate conditions can affect not only the development of gray mold after infection, but also the resistance of the host. Susceptibility of rose flowers to gray mold has been correlated with vapor-pressure deficit during production (72), nonoptimal production temperatures (41), and air movement in the greenhouse (48). The mechanisms by which these environmental factors influence resistance is unknown. They may affect the production of phytotoxic compounds, the diffusion of defense or toxic compounds in the host tissue, or the ability of the plant to rapidly deploy active defenses.

CONCLUSIONS

Resistance to infection by *B. cinerea* can be manipulated by modifying the genetic and environmental factors that affect the physiological status of the host tissue. In general, treatments that advance senescence of the host tissue make it more susceptible to *B. cinerea*, whereas those that delay senescence have the opposite effect. The influence of plant hormones and compounds affecting the oxidative status of the tissue can be explained mostly via their effects on aging and senescence-related processes. Plants employ a variety of physical and chemical barriers to invasion and spread of *B. cinerea*, some of which are altered during senescence. The environment affects the balance between the host's defenses and the pathogen's ability to overcome them. However, traits such as the rate of senescence, magnitude of chemical and structural defenses, and cell wall structure are regulated genetically. None of these factors manipulated individually will provide complete resistance to *B. cinerea*. Horticulturists and plant breeders interested in reducing the losses of crop plants to gray mold disease, therefore, must employ a variety of strategies, each of which tips the balance in favor of host plant resistance.

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