9C. Effects of Microbial Pathogens

1. Introduction

Plants frequently encounter potentially pathogenic fungi, bacteria, and viruses, yet disease results from relatively few of these exposures. In many cases there is no obvious trace of its occurrence, and the microorganism fails to establish itself due to a low pathogenicity or highly effective plant defense mechanisms. Other encounters leave evidence of an intense plant–microbe interaction, which results in the arrest of pathogen development after attempted colonization. In these cases plant tissues often display activated defense functions that produce antimicrobial compounds (phytoalexins), enzymes, and structural reinforcement that may limit pathogen growth (Delaney 1997). Plant defense responses against **pathogens** have much in common with responses following **herbivore** attack (Chapter 9B on ecological biochemistry), in terms of both signaling and final outcome, as will be explored below.

Because of the marked differences between their cellular structures and modes of life, one might expect very different strategies for attack, defense, and counterattack to have evolved in plants and animals and their respective pathogens. However, current evidence suggests that plants and animals share several individual components of host–pathogen interactions, either conceptually or mechanistically (Taylor 1998).

2. Constitutive Antimicrobial Defense Compounds

Host resistance to microbial pathogens may be based on the constitutive accumulation of inorganic compounds, e.g., **silicon** in *Oryza sativa* (rice), a typical Si-accumulating species (Sect. 4.1 of Chapter 6 on mineral nutrition). Silicon may act as a **physical barrier**, when it is deposited beneath the cuticle to form a cuticle–Si double layer. This layer can mechanically impede penetration by fungi. An alternative mechanism is that soluble Si acts as a **modulator of host resistance** to pathogens. Several studies in both monocots and dicots have shown that plants supplied with Si can produce phenolics and phytoalexins (Sect. 3) in response to fungal infection, such as those causing rice blast and powdery mildew. Si may also activate some defense mechanisms. For example, in roots of *Cucumis sativus* (cucumber) that are infected and colonized by *Pythium*, Si enhances the activity of chitinases, peroxidases, and polyphenoloxydases. Unlike rice, many plants do not accumulate Si at high enough levels to be beneficial; genetically manipulating the Si uptake capacity of the root might help plants to accumulate more Si and improve their ability to overcome biotic and abiotic stresses (Ma & Yamaji 2006).

Other inorganic compounds that may confer resistance against microbial pathogens include **heavy metals**, which offer an attractive explanation

for the existence of **hyperaccumulators** (Sect. 3.3 of Chapter 6 on mineral nutrition). The hypothesis that hyperaccumulation confers resistance against biotic stress was initially formulated based on an observation that fewer insects feed on Ni hyperaccumulators. Further investigations have subsequently shown that high levels of Ni, Zn, Cd, or Se can provide effective protection against fungi, or even viruses (Poschenrieder et al. 2006). Hyperaccumulation might therefore offer **cross-resistance** against microbial pathogens and herbivores (Sect. 3.1 of Chapter 9A on ecological biochemistry). Organic compounds, rather than inorganic nutrients or heavy metals, are the most common molecules involved in plant defense. Some of these compounds have multiple defense functions, acting against both microbial pathogens and herbivores (**cross-resistance**). For example, aucubin and catalpol, two iridoid glycosides present in *Plantago lanceolata* (snake plantain), confer in vivo resistance to both the generalist insect herbivore *Spodoptera exigua* (beet armyworm) and the biotrophic fungal pathogen *Diaporthe adunca*. The bitter taste of iridoid glycosides probably deters feeding by *Spodoptera exigua*, whereas the hydrolysis products formed after tissue damage following fungal infection likely mediate pathogen resistance (Biere et al. 2004).

Plants produce a wide range of compounds with an **antimicrobial** effect (**phytoanticipins**) (VanEtten et al. 1994). Some of these have already been discussed in Sect. 2.1 of Chapter 9B on ecological biochemistry (e.g., alkaloids, flavonoids, and lignin). **Saponins** are plant glycosides that derive their name from their soap-like properties. A common species that contains saponins is *Saponaria officinalis* (soapwort), which used to be grown near wool mills; the soapy extracts from its leaves and roots were used for washing wool. Saponins consist of triterpenoid, steroid, or steroidal glyco-alkaloid molecules that bear one or more sugar chains (Fig. 1). Saponins have been implicated as preformed determinants of resistance to fungal attack. For example, wounding of *Avena strigosa* (lopsided oat) plant tissue which results from pathogen attack causes a breakdown of compartmentalization, which allows an enzyme to contact the saponin avenacoside B, yielding a fungitoxic compound that causes loss of membrane integrity (Osbourn 1996).

Lipid-transfer proteins, which we discussed in Sect. 3.5 of Chapter 4B on effects of radiation and temperature, may also be active in plant defense. Lipid-transfer proteins from, e.g., *Raphanus sativus* (radish), *Hordeum vulgare* (barley), and *Spinacia oleracea* (spinach) are active against several pathogens, with varying degrees of specificity (Kader 1996). In addition, in *Allium cepa* (onion) some of the genes encoding antimicrobial proteins with lipid-transfer activity are up-regulated in response to infection by fungal pathogens (Kader 1997). An additional onion (*Allium cepa*) seed protein with homology to lipid-transfer proteins has a strong antimicrobial activity, without being able to transfer lipids. The name lipid-transfer protein is unfortunate in that the transfer of lipids is unlikely to be the (sole) role of these proteins in vivo (Cammue et al. 1995). It is as yet unknown how lipid-transfer proteins inhibit the growth of pathogens.

Lectins (defense compounds against herbivores; Sects. 3.4 and 3.5 of Chapter 9B on ecological biochemistry) are also effective against pathogens. For example, the lectin in rhizomes of *Urtica dioica* (stinging nettle) hydrolyzes fungal cell walls (Raikhel et al. 1993). **Thionins**, which are cysteine-rich proteins, represent another group of antimicrobial proteins that are involved in plant defenses (Epple et al. 1997).

The constitutive defense against microorganisms obviously incurs **costs** for synthesis and storage. When a range of cultivars of *Raphanus sativus* (radish) that differ widely in their sensitivity to *Fusarium oxysporum* (fungal wilt disease) are compared, the most resistant ones have the lowest relative growth rate and vice versa (Fig. 2). The exact nature of the constitutive defense is unknown, but it is probably not based solely on the presence of glucosinolates, which tend to be present only in low amounts (Sect. 3.1 of Chapter 9B on ecological biochemistry). Slow-growing, resistant radish cultivars contain more cell-wall material in leaves, but their roots have a high biomass density due to more cytoplasmic elements (proteins) rather than large amounts of cell-wall material. It has been speculated that this higher protein concentration accounts for the rapid and adequate resistance reaction, although at the expense of greater construction and turnover costs (Hoffland et al. 1996a).

In some phytopathogenic fungi, **detoxifying enzymes** have evolved that break through the plant's constitutive or induced defense against fungal attack (VanEtten et al. 1994). A number of fungi avoid the toxicity of plant saponins. Some do so by growing only in extracellular plant compartments. Some fungi that infect *Solanum lycopersicum* (tomato) **lower the pH** at the infection sites to levels at which the saponin in tomato (α-tomatin; Fig. 1) has no effect on membrane integrity. More important mechanisms involve a change in **membrane composition** of the fungus and the production of **saponin-detoxifying enzymes** (Osbourn 1996).

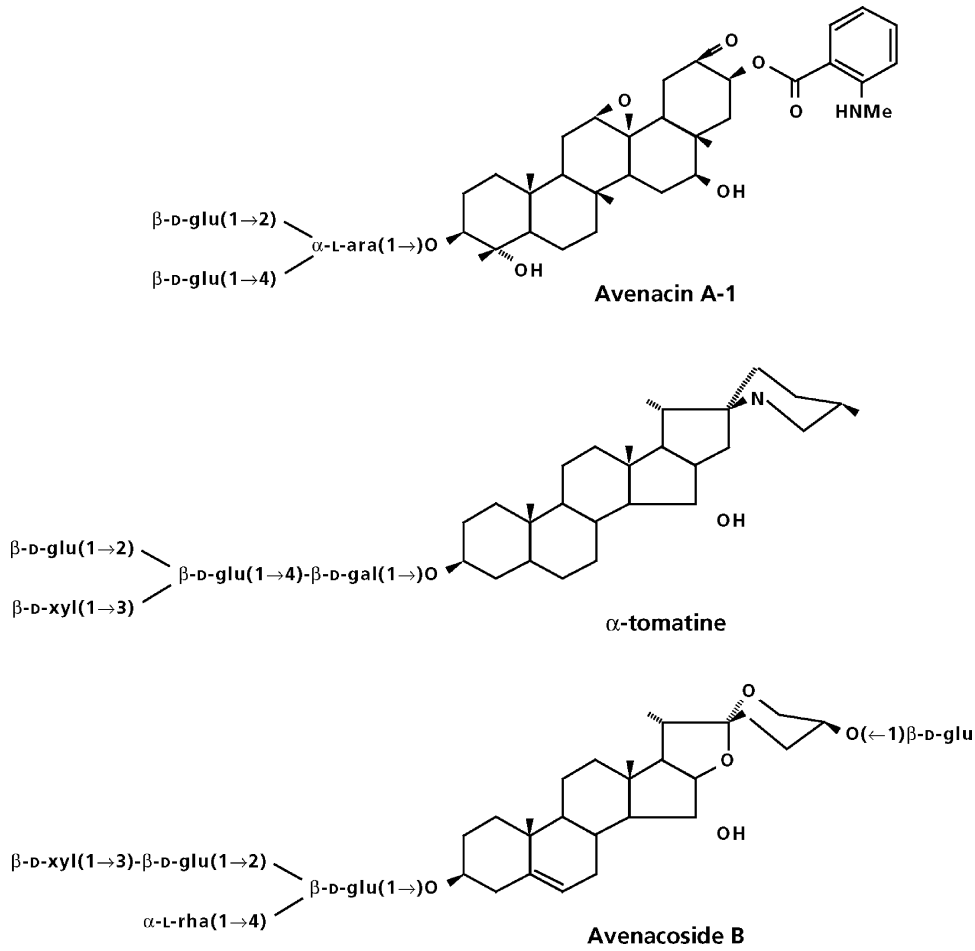

FIGURE 1. Structures of saponins from *Solanum lycopersicum* (tomato) and *Avena strigosa* (oat) (Osbourn 1996).

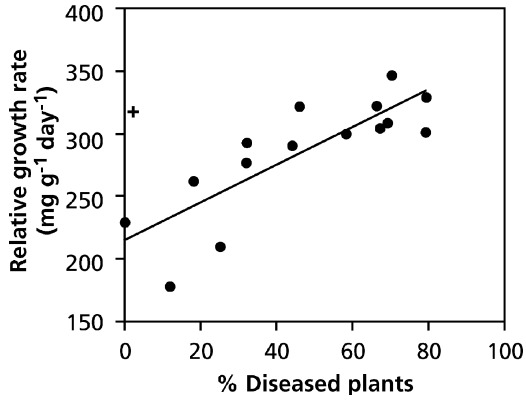

FIGURE 2. Correlation between the relative growth rate (RGR) of *Raphanus sativus* (radish) grown in the absence of pathogens and resistance level to *Fusarium oxysporum* in 15 cultivars. Each symbol refers to a different cultivar (after Hoffland et al. 1996a).

3. The Plant's Response to Attack by Microorganisms

Highly sophisticated defense strategies have evolved in plants to counteract pathogenic microorganisms. The primary immune response in plants has evolved to recognize common features of the microbial pathogens that are referred to as **pathogen-associated molecular patterns** (PAMPs) (Chrisholm et al. 2006). PAMP-triggered immunity is part of the first line of induced defense, and results in a basal level of resistance. During the evolutionary "arms race", pathogens acquired the ability to suppress this first line of induced defense via the delivery of effector proteins. In turn, plants acquired R proteins that are able to recognize these

attacker-specific effector proteins, resulting in a highly effective second line of defense called **effector-triggered immunity**. Effector-triggered immunity is also known as the gene-for-gene relationship, which was first identified in the 1940s in *Linum usitatissimum* (flax) and its fungal pathogen *Melampsora lini* (flax rust) (Flor 1971). Many different plant genes that encode disease resistance occur in clusters, either as single genes with multiple alleles that encode different resistance specificities or as a series of tightly linked genes forming complex loci (Pryor & Ellis 1993). Resistance to pathogens is thought to involve a specific recognition between a resistant plant and the pathogen. This interaction triggers a set of responses that act to confine the pathogen. If the specific gene is absent in the plant or in the pathogen (or in both), then there is no concerted defense response and disease generally ensues. There are many examples of gene-for-gene interactions of plants with fungi, bacteria, and nematodes (Taylor 1997); however, there are also more general defenses in plants.

Plants react to pathogen attack by activating an elaborate defense system that acts both locally and systemically which is in many ways similar to the kind of response we discussed in Sect. 3 of Chapter 9B on ecological biochemistry. In many cases, local resistance is manifested as a **hypersensitive response** (Stakman 1915): membrane damage, necrosis, and collapse of cells. This "suicidal" response is often confined to individual penetrated cells. It is considered as a sacrifice of locally infected tissue (sometimes only one or a few cells) to protect against the spread of the pathogen into healthy tissue. Mutants that spontaneously form patches of dead tissue (necrosis) occur in many plant species. Further analysis of these mutants shows that the hypersensitive response is caused by the production of toxic compounds by the plant or pathogen and also results, partly, from genetically programmed cell death (Jones & Dangl 1996). The hypersensitive response differs from cell death that spreads beyond the point of infection which follows from the interaction of a susceptible plant and a virulent pathogen. In this interaction, cell death does not effectively prevent pathogen multiplication or spread (He 1996).

The hypersensitive response often enhances the production of **reactive oxygen species** (O_2^-, H_2O_2), which are generated by a signaling pathway similar to that employed by mammalian neutrophils during immune responses (Mehdy et al. 1996). Reactive oxygen species are involved in cross-linking of cell-wall proteins, rendering these more resistant to attack by enzymes from the pathogen. The reactive oxygen species are also thought to be toxic for pathogens. In addition they may act as "second messengers" in the induction of defense genes (Boller 1995). These reactive oxygen species might be the cause of up-regulation of the gene encoding the **alternative oxidase** (Fig. 3). Up-regulation of this gene greatly enhances the capacity for cyanide-resistant respiration of the infected plant tissue (Sect. 4.8 of Chapter 2B on plant respiration). Increased activity of the alternative path presumably allows a rapid flux through the oxidative pentose phosphate pathway and NADP-malic enzyme, thus producing carbon skeletons and NADPH that are required in the defense reaction (Fig. 4; Simons & Lambers 1998).

In the hypersensitive response of a resistant plant to an avirulent pathogen ("incompatible interaction") specific molecules from the pathogen physically interact with specific molecules from the host which causes an array of defense responses (De Wit 1997). These responses include an **oxidative burst**, which can lead to cell death; thus, the pathogen may be "trapped" in dead cells and be prevented from

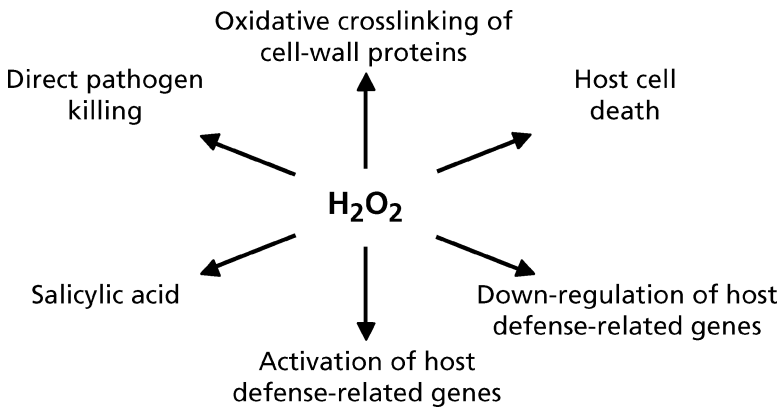

FIGURE 3. A central role for hydrogen peroxide in defense responses of plants to microbial pathogens infection. The responses shown occur in different plant species and may not occur within a given species (Mehdy et al. 1996). Copyright Physiologia Plantarum.

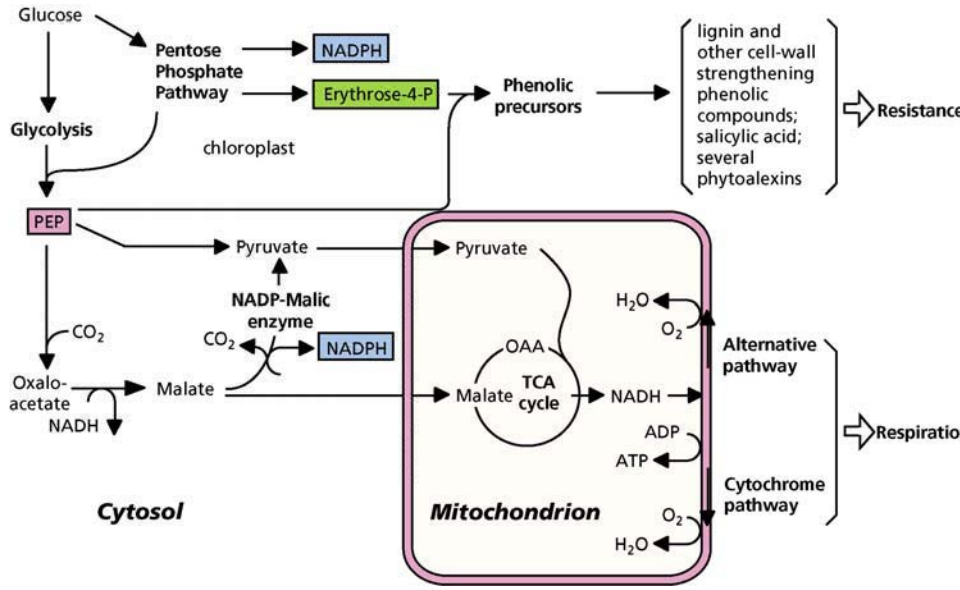

FIGURE 4. Major metabolic pathways involved in the plant's resistance response to pathogens and its association with respiration. Plant defense requires an increased production of erythrose-4-phosphate for numerous phenolic precursors. Erythrose-4-phosphate is produced in the oxidative pentose pathway, which also generates the NADPH that is required for the biosynthesis of, e.g., lignin and some phytoalexins. Additional NADPH is produced by NADP-malic enzyme, which decarboxylates malate to pyruvate. Increased activity of these reactions enhances the production of pyruvate, which is postulated to require an increased activity of the alternative path. Up-regulation of the gene encoding the alternative oxidase may be triggered by accumulation of reactive oxygen species (after Simons & Lambers 1998).

spreading from the site of infection. The cells surrounding the site of entry modify their cell walls so that they can inhibit penetration by the pathogen (Heil & Bostock 2002). They also produce antimicrobial compounds, such as **phytoalexins** (i.e., low-molecular-mass antibiotics that are not found in uninfected plants). The chemical nature of phytoalexins is extremely variable (Fig. 5); closely related species often have phytoalexins with a similar structure. Microorganisms, or components thereof (**elicitors**), induce the formation of phytoalexins (Boller 1995). Numerous other compounds in microbial pathogens (e.g., carbohydrates and lipids) may also cause nonspecific production of phytoalexins. In addition, cell-wall components (e.g., glucans or glucomannans) may elicit rapid synthesis of phytoalexins in resistant cultivars.

Now that phytoalexins have been introduced, we stress two points. First, accumulation of a specific compound upon attack does not prove that this compound is involved in resistance. Rather, accumulation may be a side reaction that has nothing to do with the actual resistance mechanism. To prove that a compound is involved in a resistance mechanism may require mutants that are unable to make the putative defense compound. Some 40 years ago, Chamberlain & Paxton (1968) demonstrated that the stems of a cultivar of *Glycine max* (soybean), which is susceptible for the fungus *Phytophthora megasperma*, can become resistant upon addition of a phytoalexin isolated from a resistant cultivar. Inhibition of phenylalanine **ammonia lyase**, which is a key enzyme in the synthesis of isoflavanoids, decreases the concentration of phytoalexins and increases the growth of the infecting fungus. Similar to what we discussed in Sect. 3.3 of Chapter 9B on ecological biochemistry, there is also an **arms race** between plants and microbial pathogens, which have evolved to detoxify phytoalexins. For example, isolates of the phytopathogenic fungus *Nectria haematococca* produce pisatine demethylase that detoxifies the toxic phytoalexin pisatine in *Pisum sativum* (pea) (VanEtten et al. 1995). The second point we stress is that synthesis of phytoalexins is only one of a range of mechanisms involved in combating the pathogen. Production of hydrolytic enzymes (e.g., chitinases and glucanases) is also part of the defense response (Vierheilig et al. 1993). The

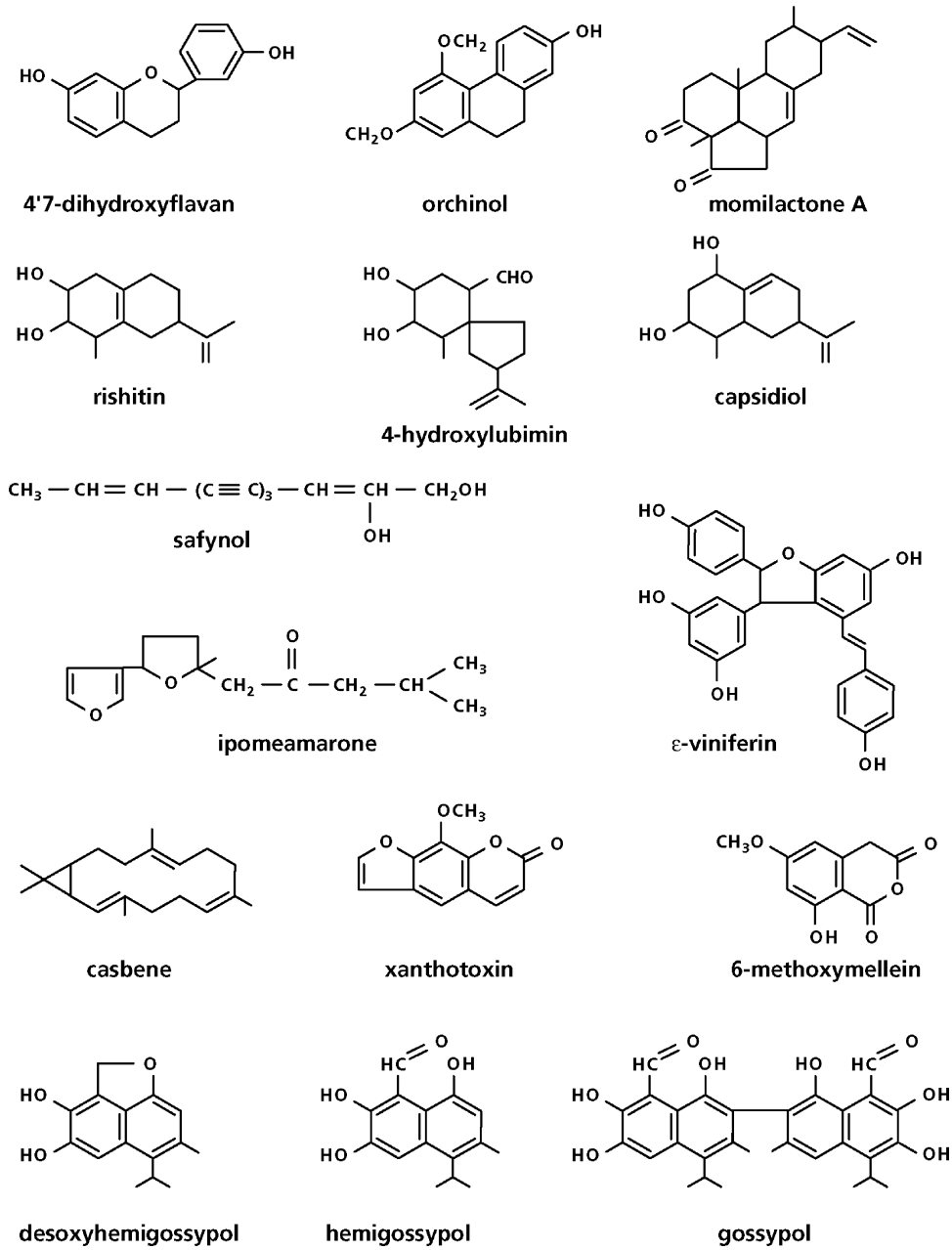

FIGURE 5. Some examples of phytoalexins in different plant species (Bell 1981). With permission, from the Annual Review of Plant Physiology, Vol. 32, copyright 1981, by Annual Reviews Inc.

proteins induced upon pathogen attack are referred to as **pathogenesis-related proteins** (**PRs**); they accumulate either in intercellular spaces or intracellularly in the vacuole (Heil & Bostock 2002, Van Loon et al. 2006). Some of these PRs confer disease resistance (Broglie et al. 1991) and inhibit fungal growth in vitro as well as in vivo (Boller 1995). Whilst phytoalexins typically accumulate at the site of attack, PR proteins also accumulate **systemically** (Van Loon et al. 2006).

After attack of a resistant host by an avirulent pathogen, the enzymes required for the synthesis of phytoalexins are first produced de novo, and then the phytoalexins accumulate. The chemicals are

produced in living plant cells, and may well lead to the death of these cells, due to their toxicity for the host plant as well as for the pathogenic microorganism. Some phytoalexins [e.g., glyceollin from *Glycine max* (soybean)] are specific inhibitors of complex I of the mitochondrial electron-transport chain (Sect. 2.2.1 of Chapter 2B on plant respiration). Glyceollin can be found in soybean roots as soon as 2 hours after inoculation with the fungus *Phytophthora megasperma* (Hahn et al. 1985).

In several pathosystems, the hypersensitive response to an avirulent pathogen (incompatible interaction) as well as the response to a virulent pathogen (compatible interaction) is accompanied by an **induced systemic resistance** to infection by other pathogens (Ryals et al. 1994). It involves the accumulation of **salicylic acid** (Tenhaken & Rübel 1998) and activation of ***PR* genes** (Linthorst 1991). **Salicylic acid** is required for the expression of **systemic acquired resistance** (SAR) (Durrant and Dong 2004). Although salicylic acid can be transported in the plant, reciprocal graftings of transgenic plants, in which salicylic acid is degraded, and nontransformed plants as rootstocks or scions, show that salicylic acid is not the translocated signal in SAR (Vernooij et al. 1994). **Methyl salicylate**, which is a volatile liquid known as oil of wintergreen, is produced from salicylic acid by a number of plant species. It is a major volatile compound released by *Nicotiana tabacum* (tobacco) inoculated with tobacco mosaic virus. Methyl salicylate may act as an airborne signal that activates disease resistance and the expression of defense-related genes in neighboring plants and in the healthy tissues of the infected plants (Shulaev et al. 1997). Plants treated with salicylic acid or acetylsalicylic acid (aspirin) for 12–24 hours are primed to respond much faster to pathogen-derived signals with the production of phytoalexins and their biosynthetic enzymes, PRs, and the production of reactive oxygen species (Kauss & Jeblick 1996, Conrath et al. 2002). People are therefore not the only organisms that benefit from use of aspirin.

Resistance genes are also activated by exposure to ethylene or the vapor of methyl jasmonic acid [e.g., in *Solanum lycopersicum* (tomato), *Medicago sativa* (alfalfa), or *Nicotiana tabacum* (tobacco)]. Methyl jasmonic acid is a cyclopentanone that is synthesized from linolenic acid; it is well known as a fragrant constituent of the essential oil of *Jasminum grandiflorum* (Spanish jasmine). Jasmonic acid or methyl jasmonic acid from either a synthetic solution or from undamaged twigs of *Artemisia tridentata* (sagebrush) are equally effective. They are common secondary metabolites that often occur in higher levels in damaged plants (Bruin et al. 1995). Plants like *Artemisia tridentata* are promising for use as an "**intercrop**" (i.e., plants used in combination with a crop plant to protect the crop against pests in an environmentally friendly way). "Intercropping" has been proposed as a method to contribute to **pest control**.

Plants can also become resistant by exposure to nonpathogenic root-colonizing bacteria [e.g., to fluorescent *Pseudomonas* sp. in *Dianthus caryophyllus* (carnation) (Van Peer et al. 1991) and *Raphanus sativus* (radish) (Hoffland et al. 1996b), and to *Serratia plymuthica* in *Cucumis sativus* (cucumber) (Wei et al. 1991)]. In this **induced systemic resistance**, however, salicylic acid does not play a role (Van Loon et al. 1998, Pieterse et al. 2001, 2002).

4. Cross-Talk Between Induced Systemic Resistance and Defense Against Herbivores

The responses to microbial pathogens (Sect. 3) have much in common with responses to herbivory (Sect. 3 of Chapter 9B on ecological biochemistry). For example, the expression of resistance to **pathogens** as well as to **insect herbivores** involves two signaling pathways, one involving **salicylic acid** and another involving **jasmonic acid**. Stimulation of induced systemic resistance in field-grown *Solanum lycopersicum* (tomato) plants with benzothiadiazole (a salicylate mimic) (1) attenuates the jasmonate-induced expression of the antiherbivore defense-related enzyme **polyphenol oxidase**, and (2) compromises host-plant resistance to larvae of the beet armyworm, *Spodoptera exigua.* On the other hand, treatment of plants with jasmonic acid at concentrations that induce resistance to insects reduces **pathogenesis-related protein** gene expression induced by benzothiadiazole; this partially reverses the protective effect of benzothiadiazole against bacterial speck disease, which is caused by *Pseudomonas syringae*. This suggests that the sharing of elements of the two defense pathways may involve trade-offs. Therefore, effective utilization of induced systemic resistance to multiple pests typically encountered in agriculture requires understanding potential signaling conflicts in plant defense responses (Thaler et al. 1999).

Considering the example of *Solanum lycopersicum* (tomato), discussed above, variable outcomes can be expected, because there can be **cross-talk** between signaling leading to induced defense as well as signaling resulting in induced resistance against herbivores. On one hand, resistance elicited by one group

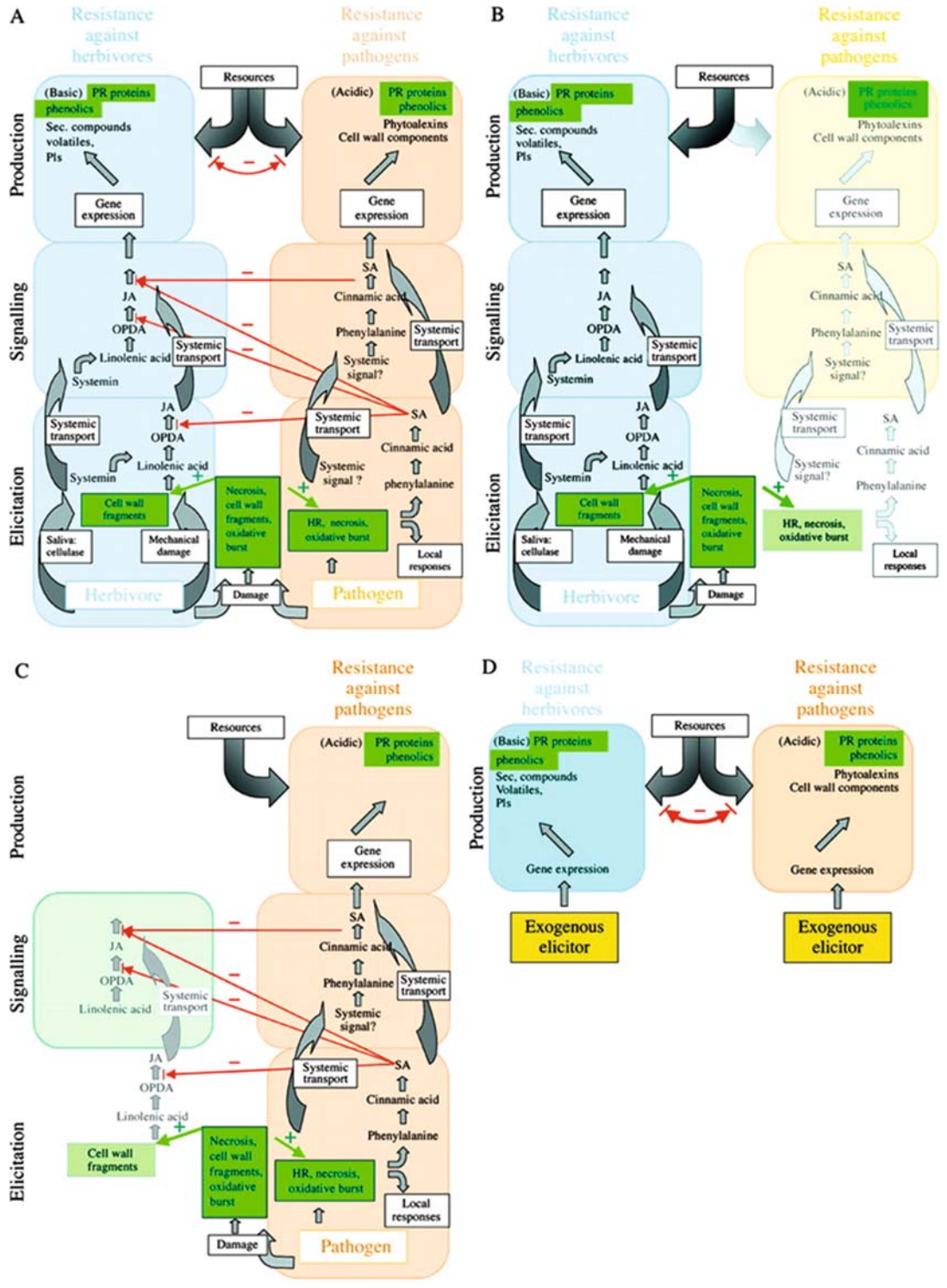

FIGURE 6. Variable outcomes of cross-talk between signaling leading to induced systemic resistance and signaling resulting in induced resistance against herbivores. (A) Overview of interactions. At the level of elicitation and production, several "common factors" (in *green boxes*) appear in both signaling pathways (necrosis,

of enemies and active (also) against another is called **cross-resistance** (e.g., resistance against pathogens induced by herbivores, and vice versa). Cross-resistance has been found in different systems. For example, feeding by thrips and aphids reduces infection of *Cucurbita citrullus* (watermelon) by the fungus *Colletotrichum orbiculare*. Defoliation of *Glycine max* (soybean) by *Pseudoplusia includens* (soybean looper moth) reduces the severity of two different fungal infections. Beetle grazing can induce resistance against fungal infections in *Rumex obtusifolius* (bitter dock). *Helicoverpa zea* (corn earworm) feeding can increase resistance of *Solanum lycopersicum* (tomato) plants to an aphid species (*Macrosiphum euphorbiae*), a mite species (*Tetranychus urticae*), corn earworm (*Spodoptera exigua*), and to a bacterial phytopathogen, *Pseudomonas* (Bostock 2005). In *Arabidopsis thaliana* (thale cress), feeding by caterpillars of the cabbage white butterfly (*Pieris rapae*) results in enhanced resistance against the bacterial pathogens *Pseudomonas syringae* and *Xanthomonas campestris*, and turnip crinkle virus (De Vos et al. 2006).

Instead of cross-talk, there may be trade-offs, i.e., compromised resistance against one group of enemies when the plant is in the induced stage against the other group. For example, chemical induction of induced systemic resistance decreases the plants' ability to express wound-inducible proteinase inhibitors. Similarly, salicylic acid treatment inhibits wound- and jasmonic-acid-induced responses in the same plant, and application of jasmonic acid partially reduces the efficacy of chemically induced systemic resistance elicitors. Results available so far show that salicylic acid can inhibit the signaling cascade at different steps that are located both upstream and downstream of jasmonic acid (Heil & Bostock 2002, Spoel et al. 2003).

In summary, there are three steps in the induction pathway leading to defense: (1) **elicitation**, (2) **signaling**, and (3) **production**, i.e., gene expression and synthesis of enzymes and other proteins involved in the establishment of the resistant phenotype. Interactions probably occur independently on all three levels (Fig. 6).

(1) **Elicitation: S**alicylic acid is synthesized in response to mechanical damage, necrosis, and oxidative stress. Compounds resulting from the degradation of cells or cell walls might be involved in eliciting the systemic signal, and induced systemic resistance can thereby be induced by different types of enemies. Correspondingly, jasmonic acid can be induced in response to cell-wall degradation. Further elicitors in the context of both wound response and induced systemic response include the development of reactive oxygen species (Fig. 6A and B). Therefore, events at the elicitation level will mainly lead to the expression of a rather nonspecific cross-resistance.

(2) **Signaling**: Further interactions can occur at the signaling level. Different activities of the various intermediates leading to jasmonic acid may lead to a diversity of potential outcomes. Similar regulatory properties might characterize the salicylic acid-dependent signaling. An inhibition of the jasmonic acid pathway by salicylic acid has been described in different plant species. While herbivores can induce both an induced systemic response and induced resistance to herbivores (Fig. 6B), an induction by pathogens leads to synthesis of high concentrations of salicylic acid, and thus blocks later steps in signaling involving the jasmonic acid pathway. Phenotypically, pathogen attack thus induces mainly (or only) induced systemic response compounds (Fig. 6C).

(3) **Production**: The trade-offs might, in contrast, occur mainly at the production level (i.e., signal–response coupling; Fig. 6D). Production of defensive compounds can be limited by the

FIGURE 6. (continued) cell wall fragments, and oxidative burst during elicitation; phenolics and PR proteins at the production level) and these might represent factors leading to cross-resistance phenomena. (B) Elicitation by a herbivore. While inducing mainly the pathway leading to jasmonic acid, the "common" elicitors might lead to partial induction of induced systemic resistance signaling. Resources are mainly allocated to herbivore resistance, but some resistance against pathogens is expressed, too. (C) Elicitation by a pathogen. The partial induction of the pathway leading to jasmonic acid by the "common" elicitors might lead to the occurrence of some early metabolites in the pathway leading to jasmonic acid, but later the pathway is blocked by the inhibitory effects of salicylic acid. On the phenotypic level, only resistance against pathogens is expressed. (D) Exogenous elicitation bypasses regulatory mechanisms at the elicitation and the signaling levels. The competition between both pathways for limiting resources therefore dominates the outcome and leads to phenotypically visible trade-offs when both pathways are induced at the same time (Heil & Bostock 2002, by permission of Oxford University Press).

supply of available precursors such as amino acids, ATP, and other biosynthetic cofactors, and so does not depend only on the outcome of events at the signaling level. Induction of salicylic-acid-responsive and jasmonic-acid-responsive genes appears to occur each at the cost of the other group, presumably since plants are compromised in the total amount of defensive compounds that can be produced during a limited time span.

5. Messages from One Organism to Another

Plants continually receive messages from their environment, including chemical messages (elicitors) released by pathogenic and nonpathogenic microorganisms. Resistant plants respond to these messages by defending themselves. This involves sacrificing a small number of cells in a programmed manner, and trapping the pathogen inside dead cells; it also involves both a physical and chemical defense of the surviving cells. Upon attack by pathogens, both resistant and surviving sensitive plants acquire greater resistance to subsequent attack, be it by the same or by a different pathogen. In recent years remarkable surveillance mechanisms have been discovered that have evolved in plants to recognize microbial factors and combat pathogenic microbes (Chisholm et al. 2006). The discovery of resistance that is induced by nonpathogenic rhizobacteria which is a process of plant **immunization** to diseases is receiving increased attention. It may help us protect our crops against pathogens in an environmentally friendly manner.

Many plants may be damaged by herbivores at the same time as being attacked by microbial pathogens. The subsequent defense responses may either induce cross-resistance or involve a trade-off, depending on the plant species and the attacking organism. If nonpathogenic rhizobacteria would induce cross-resistance, this would offer potential for applications in intensive agricultural systems.

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