Predisposition in Plant Disease: Exploiting the Nexus in Abiotic and Biotic Stress Perception and Response

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Abstract

Predisposition results from abiotic stresses occurring prior to infection that affect susceptibility of plants to disease. The environment is seldom optimal for plant growth, and even mild, episodic stresses can predispose plants to inoculum levels they would otherwise resist. Plant responses that are adaptive in the short term may conflict with those for resisting pathogens. Abiotic and biotic stress responses are coordinated by complex signaling networks involving phytohormones and reactive oxygen species (ROS). Abscisic acid (ABA) is a global regulator in stress response networks and an important phytohormone in plant-microbe interactions with systemic effects on resistance and susceptibility. However, extensive cross talk occurs among all the phytohormones during stress events, and the challenge is discerning those interactions that most influence disease. Identifying convergent points in the stress response circuitry is critically important in terms of understanding the fundamental biology that underscores the disease phenotype as well as translating research to improve stress tolerance and disease management in production systems.

INTRODUCTION

Abiotic stresses can dramatically alter the outcome of plant-pathogen interactions, and depending on the pathosystem and stress intensity, the stress may enhance or reduce disease. Recognition of the importance of predisposing environmental stress in plant pathology goes at least as far back as 1874, when Sorauer et al. formally introduced the concept and later discussed predisposition in their *Manual of Plant Diseases* (172). Hartig also recognized the importance that abiotic stress plays in altering the proneness of the host plant to disease, using the terms "predisposition" and "tendency to disease" interchangeably (73). Yarwood defined predisposition as "the tendency of nongenetic factors, acting prior to infection, to affect the susceptibility of plants to disease" (205). Implicit, and important, in the latter definition is that predisposing stresses can shift the outcome toward resistance or susceptibility.

The quest to improve plant tolerances to abiotic and biotic stresses is an ongoing effort. What is probably not fully appreciated, however, is that episodes of relatively mild abiotic stress can override disease resistance. Apparent from field experience and from the literature is that abiotic stress levels that are thresholds for predisposition occur routinely in agricultural, forest, and nursery production systems. Furthermore, in the absence of a pathogen, plants often recover rapidly and fully when the stress is relieved (27). This is significant for plant breeding and in food and fiber production in which abiotic stress can (*a*) reduce inoculum thresholds necessary for disease, (*b*) degrade the consistency and reliability of pathogenicity tests and disease resistance screens, and (*c*) diminish or nullify the efficacy of other disease management measures. Although the problem of predisposing stress in plant diseases is not new, the specter of climate change adds urgency for a better understanding of abiotic and biotic stress interactions (65).

SCOPE OF REVIEW

This review examines recent progress in our understanding of the interaction between abiotic stress and disease, and builds upon themes presented previously on cross talk and trade-offs in phytohormone signaling in relation to induced resistance (24). Here, we emphasize stresses that predispose plants to levels of pathogen inoculum that would not be damaging in the absence of the stress and discuss how phytohormone networks may engage unproductively to compromise the host plant. Although abiotic stresses affect pathogens, we do not delve into this aspect, except to mention it as an experimental consideration. For additional background, the reader is referred to excellent reviews and the references therein that address plant abiotic stress perception, signaling, and response (143, 208), and others that address biotic and abiotic stress interactions in plants (13, 148, 157). Desprez-Loustau et al. (45) thoroughly cover the predisposing effect of drought in diseases of forest trees. For population- and landscape-level perspectives and the impact of climate change on plant diseases, the reader is referred to an excellent special issue of the journal *Plant Pathology* (33).

ABIOTIC AND BIOTIC STRESSES AND THE DILEMMA OF SHARED SIGNAL-RESPONSE NETWORKS

Stress can be defined as "a sudden change in the environment that exceeds the organism's optimum and causes homeostatic imbalance, which must be compensated for" (92). Stress imposes strains on a biological system that can be distinguished as plastic or elastic (15, 181). Strains that are plastic result in irreversible physical or chemical changes that often lead to death of the plant. Elastic strains impose physical or chemical changes that are reversible when the stress is removed. Elastic strains, such as water deficit or the hypoxia incurred by waterlogging, may have consequences that become irreversible with sufficient duration (15).

As sessile organisms, plants have effective physiological mechanisms to maintain homeostasis during stress events. These are adaptive, at least initially, in order to promote the plant's health and survival (96, 97). The dilemma that arises in predisposition, however, is that an adaptation in one context, such as adjustment to water deficit, may be maladaptive when there is a need to also resist pathogens and pests. The notion of a response hierarchy attempts to rationalize the apparent dominance of one response over another in the face of concurrent stresses (71). An evolutionary interpretation is that the dominant response has undergone stronger selection pressure, for example, a trait arising from the continual need for plants to minimize water stress or damage from UV irradiation versus a threat of a sporadic nature, as attack by pathogens might be viewed. The notion of accepting alternative evolutionary scenarios was raised previously in the context of why some current traits may not result in the highest fitness, the example being trade-offs we observe in defense signaling when plants are challenged by different attackers (24). Such traits may have been pulled along by pleiotropy, linkage with a correlated trait, genetic drift, and/or lack of variation. The degree to which the stress response circuitry is shared to counter an abiotic stress but not the biotic stress, and vice versa, could be a result of these genetic processes.

A general adaptation syndrome (GAS) posits a common stress response in plants to evoke similar coping mechanisms (104). Responses common to anoxia, drought, heat, chilling, flooding, salinity, desiccation, and freezing, which include both physiological and morphological adaptations, support this view (149). Numerous investigations, including contemporary transcriptome, proteome, and metabolome studies, reveal that abiotic and biotic stressors engage common signals and share responsive genes and products (34, 97, 113). Plant stress response pathways are conserved (161) and are viewed as highly integrated, overlapping, and nonlinear to balance and optimize plant performance in the face of diverse challenges (59). Different stressors may cause similar cellular damage to initiate the shared signal-response cascades through perturbation of ion channels and mechano- and osmosensors, membrane disruption, and generation of reactive oxygen species (ROS) (38, 141, 195) (**Figure 1**). Implicit and perhaps desired is that subfunctionalization downstream from the common signaling events will afford opportunities for tapping selective responses to improve tolerance to different stresses.

Certainly, positive interactions and synergies occur, examples being cross tolerance to chilling following adaptation to water stress and a mild stress that may enhance disease resistance (176). It is important to acknowledge the positive effects of environmental stresses, particularly when the stress is imposed gradually rather than abruptly, thereby enabling the physiological adjustments necessary for tolerance (96). The GAS model is instructive in that the stress response is viewed in three stages: an initial alarm reaction, an acclimation stage during which a degree of tolerance is acquired, and, if the stress persists, an exhaustion phase resulting in collapse or severe compromise (**Figure 2**).

EXAMPLES OF PREDISPOSING ABIOTIC STRESSES

Plant pathology textbooks include a section on abiotic diseases with discussion of water stress, heat, chilling, freezing, light quality, air pollution, nutrient deficiency or excess, salinity, and herbicide injury as well as other stresses. Any of these at sufficient intensity can damage or kill plants, and this damage can be further exacerbated by pathogen attack. All of these at intensities below an irreversible damage threshold, and often in the absence of visible injury, can predispose plants to pathogens. This is to say that predisposition occurs with both plastic and elastic strains. However, of special interest are mild elastic strains that occur with brief or episodic stress from which the plant normally recovers (**Figure 2**). Such reversible strains present experimentally tractable

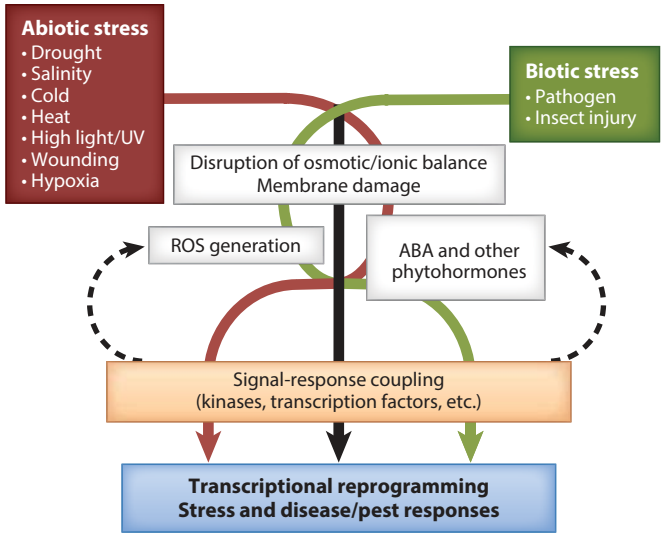


Figure 1

General signal-response sequence in plants following abiotic and biotic stress events that illustrates the concept of shared pathways and phytohormone cross talk to shape transcriptional reprogramming and response. Factors involved in signal-response coupling include various kinases, phosphatases, transcriptional activators and repressors, Ca^{2+} signaling elements, and so forth (*light orange box*), and downstream outputs include gene expression and biochemical and physiological responses (*light blue box*). Dashed arrows indicate feedback, which could be positive or negative, to enhance or attenuate reactive oxygen species (ROS) and phytohormone action. Adapted from Fujita et al. (62). Abbreviation: ABA, abscisic acid.

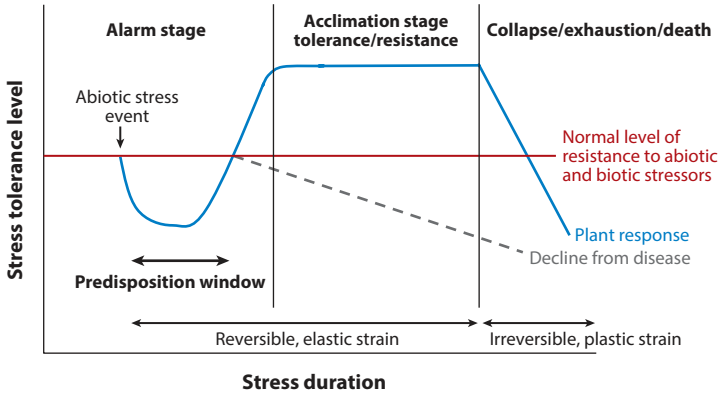


Figure 2

Model of the plant response to abiotic and biotic stresses that integrates features of Selye's general adaptation syndrome, concepts of strain severity and duration, and disease predisposition. The phase of greatest vulnerability to disease occurs immediately following or soon after a stress event and prior to onset of acclimation. The dashed line illustrates predisposition and abrogation of stress adaptation as disease develops. In the absence of the abiotic stress, the plant would normally resist the pathogen or at least the level of inoculum present. Adapted from Leshem et al. (104).

approaches for mechanistic inquiry and, relative to studies of severe stress, may afford greater opportunities to identify genetic strategies to mitigate predisposition.

A classic review in this journal (165) provided a generalized model for predisposing stress that accommodated different outcomes, either enhanced resistance or susceptibility, depending on the parasitic strategy of the pathogen. The underlying paradigm interprets these different outcomes as a gain or loss in resistance resulting from the stress's impact on host defenses. However, it is fair to note that the stress may also cause a gain or loss in susceptibility, attributable to what might be viewed as susceptibility factors, such as cell death programs (49). The difference may seem nuanced and semantic, but the paradigm selected can influence the framing of hypotheses and the selection of analytical targets.

Abiotic stress generally results in reduced severity or incidence of diseases caused by obligate, or biotrophic, pathogens (3, 166), although there are exceptions, such as diseases caused by some viruses, fungi, and nematodes (177, 180). Abiotic stresses can predispose plants to potentially aggressive hemibiotrophic pathogens, resulting in severe disease from very low levels of inoculum. Notable here are *Phytophthora* root and crown rots, where episodes of plant water stress in its various forms can be a critical determinant for disease development and severity (56, 99, 115) (**Figure 3**). Perhaps the most pronounced impact of abiotic stress is to facilitate diseases caused by weakly aggressive facultative pathogens and those usually present in association with their hosts as saprophytes or endophytes (45). These include the root- and crown-infecting pathogens *Pythium ultimum* and *Fusarium* spp.; pathogens of aerial parts, such as *Alternaria* spp. and *Botrytis cinerea*; and many canker-causing pathogens of trees and woody perennials (45, 118, 177) (**Figure 4**).

An analysis of recent literature by Cramer et al. (40) found that of the more than 35,000 papers published between 2001 and 2011 on abiotic stress, 14% dealt with water stress, 28% with temperature stress, 22% with light stress, and 35% with chemical/soil stresses (e.g., nutrients and minerals, salinity, air pollutants). In terms of their importance as factors in disease predisposition, water, nutrient/salinity, temperature, and air pollution, stresses are perhaps the most common environmental triggers. Variation in light intensity or duration is generally not of significance to impact diseases in the field but can be a consideration in highly managed production or experimental systems, in natural ecosystems in studies of understory plants, or in combination with other stresses (58, 165). Subtoxic levels of synthetic herbicides can also increase or decrease disease in crops in the field, particularly those caused by facultative pathogens, and are commonly reported (55). Direct effects of the herbicide on the pathogen have also been reported.

Physiological and Experimental Considerations

As with any experimental pathosystem developed for mechanistic inquiry, the environmental conditions and disease assays in a predisposition study must be carefully considered and optimized. Typically, one environmental parameter is isolated and varied, and disturbing influences that may confound interpretation are eliminated or minimized. To discern stress treatment effects on the plant-pathogen interaction, it is desirable to use plants at the same developmental stage with uniform growth conditions and maintain sampling consistency while using a sensitive and quantitative disease assay. Diurnal changes and circadian phases are also important to consider, as these influence gene expression and response during stress events (63, 198). Whether plants are actively transpiring can also influence the impact of a stress on host physiology, an example being salinity (an accumulation of salts in leaves is higher as a result of transpiration) (134). Most contemporary studies incorporate biochemical and molecular methods that can be exquisitely sensitive to subtle external changes, so experiments are typically conducted within tightly

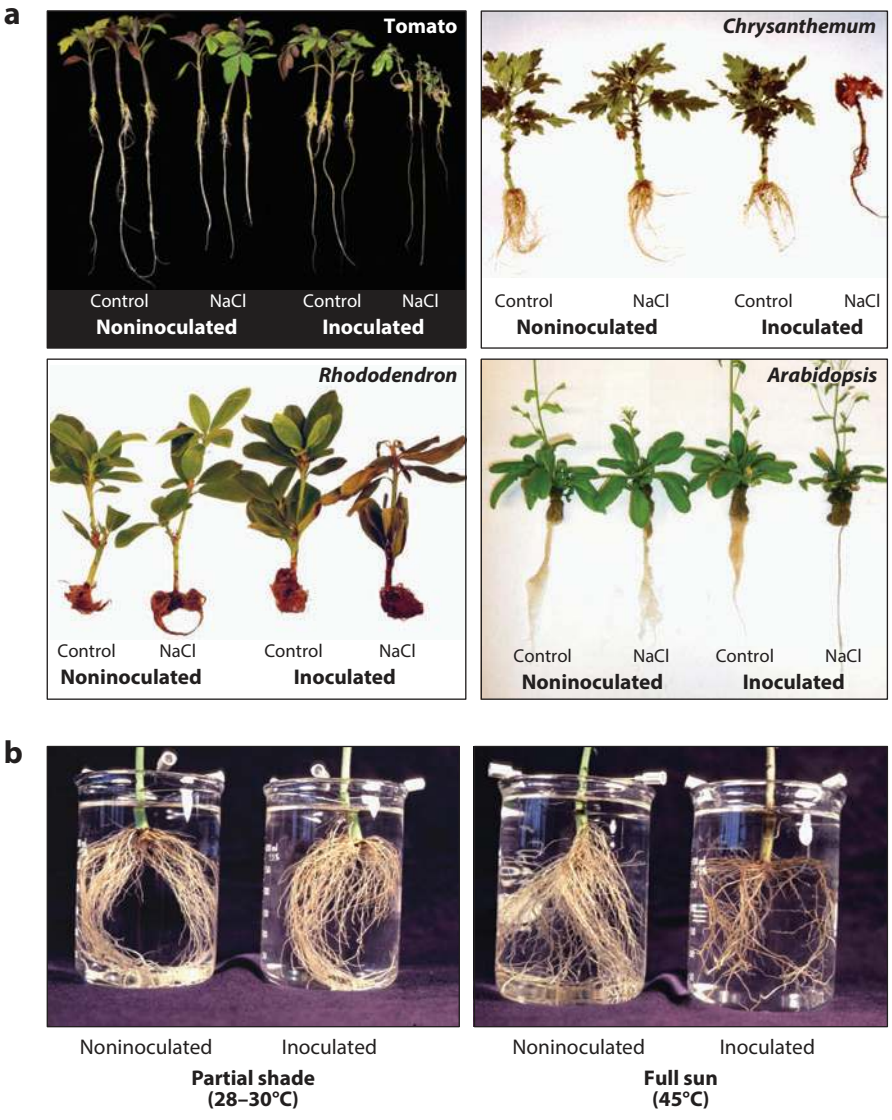


Figure 3

Predisposition in *Phytophthora* root and crown rot. (*a*) Impact of salinity stress prior to inoculation in four *Phytophthora*-plant interactions: *Phytophthora capsici*–tomato (*Solanum lycopersicum*); *Phytophthora cryptogea–Chrysanthemum*; *Phytophthora ramorum–Rhododendron*; *P. capsici–Arabidopsis*. Roots were exposed to 0.2 M NaCl/0.02 M $CaCl_2$ in half-strength Hoagland for 16–24 h, rinsed and returned to half-strength Hoagland to recover, and then inoculated with zoospores (10^4–10^5 per ml). Controls included no salt and noninoculated treatments as indicated. Images taken after an appropriate incubation period. Portions of panel *a* adapted from References 50 and 159. (*b*) Impact of an episode of heat stress on development of *Phytophthora* root rot caused *P. cryptogea* in potted *Chrysanthemum* plants. Potted plants were removed from the greenhouse and placed in an outdoor nursery for one day either in partial shade or in full sun, achieving maximum soil temperatures as indicated. Plants were then inoculated with a zoospore suspension and returned to the greenhouse; several days later the roots were washed for isolations and examined for symptoms. Note the discoloration and necrosis of the inoculated roots from the pots exposed to full sun. Images in panel *b* courtesy of J.D. MacDonald (117).

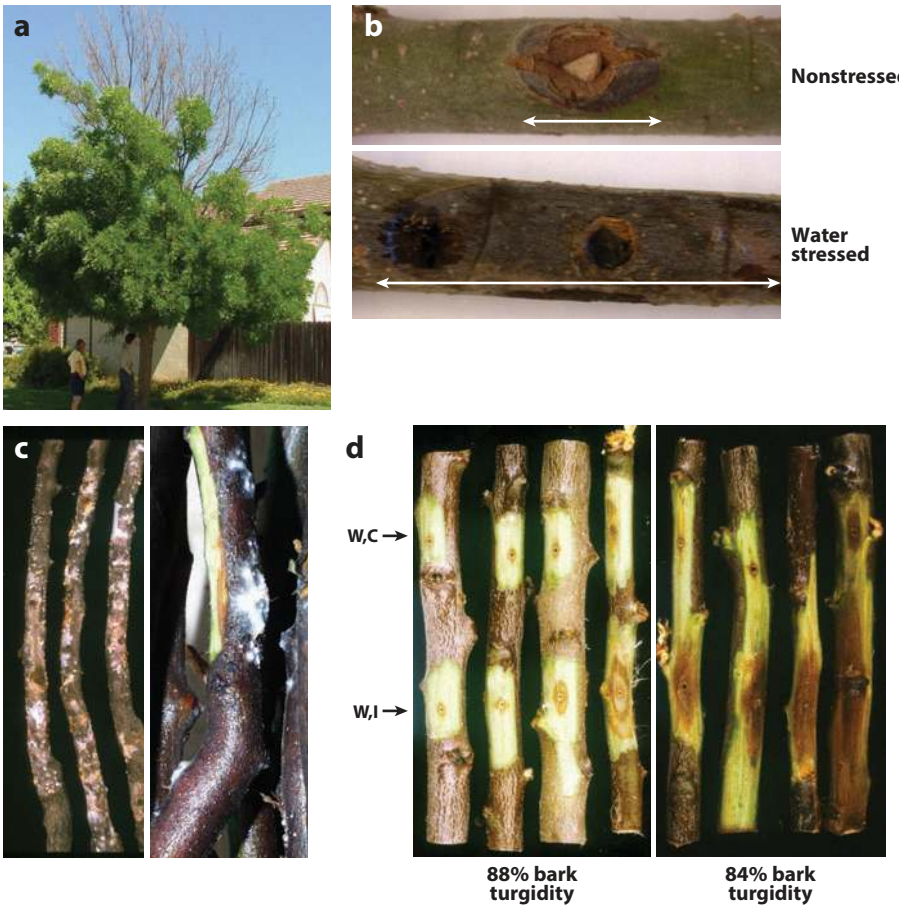


Figure 4

Examples of predisposition to fungal canker diseases in trees. (*a*) Dieback of raywood ash (*Fraxinus oxycarpa* Raywood) following drought stress caused by *Botryosphaeria stevensii*. (*b*) *Botryosphaeria* canker development in inoculated nonstressed (stem water potential $= -1.0 \pm 0.5$ MPa) and water-stressed (-3.1 ± 0.4 MPa) raywood ash branch segments. Arrows indicate canker lengths. Photos in panels *a* and *b* courtesy of T.R. Gordon. (*c*) Almond (*Prunus dulcis*) stems from cold-stored seedling trees showing symptoms and signs of cankers caused by *Fusarium* spp. and *Cylindrocarpon* spp. (*d*) Influence of bark moisture measured as relative bark turgidity on the susceptibility of dormant almond tree segments to *Fusarium acuminatum*. Branch segments were inoculated following a 14-day desiccation period (84% relative bark turgidity) or without desiccation (88% relative bark turgidity) and then examined 14 days after inoculation. W,C: wound-only control; W,I: wound inoculation. Panels *c* and *d* adapted with permission from Reference 118.

controlled growth environments. We refer the reader to excellent reviews of experimental design and analytical considerations for studies of water stress (189) and stress transcriptomes (92).

Adjusting the inoculum to an appropriate titer is also an important consideration. Inoculum density that is too high can mask the predisposing effect of a stress, so levels are adjusted to where there is little or no disease in the absence of the stress. Plants may resist relatively high inoculum levels, as with weak, facultative pathogens, and it is only with predisposing stress that disease manifests. This can be an important consideration for establishing etiology, whereby Koch's postulates should be performed, if necessary, on both nonstressed and moderately stressed

plants to evaluate the potential of a species to cause disease in a given host (165). Predisposing stresses can also affect pathogen physiology or behavior (57), and, if so, this needs to be considered in the experimental design. The pathogen may be physiologically compromised by the imposed stress, inactivating inoculum or rendering it temporarily incompetent until favorable conditions are restored. The pathogen may be stimulated by the stress, as with salinity (115) and flooding (99) to increase colonization of roots by *Phytophthora* spp. Isolating the stress effect to the plant can be done experimentally to some degree by separating in time or place the stress treatment from the inoculation (50, 152). The time course over which stress is imposed can influence how the plant responds. Where plants are grown in containers, roots proliferate at the sides and bottom of the pot. When water is withheld it disappears quickly from these locations so stress comes on much more quickly than it would under field conditions. Consequently, plants cannot adjust physiologically to the same degree, and the effects of water stress may overwhelm subtler effects on disease susceptibility than might otherwise be apparent.

Water Stress in Predisposition

Abiotic stresses that impact plant water potential (ψ_w) have provided an important focus in predisposition research (27) and include dehydration from water deficit, hypoxia from waterlogging, and osmotic stress from soil salinity. Chilling and freezing, in addition to their direct injury to plant membranes and other structures, also impact plant ψ_w (181). Classic studies of fungal canker diseases of trees (165) demonstrated predisposition when bark moisture dipped below a critical threshold of approximately 80% relative turgidity, although higher bark turgidities (e.g., 84% in almond stems) can be predisposing to fungal pathogens (118) (**Figure 4**). In general, stem xylem potentials of −1.2 to −1.5 MPa provide a threshold for predisposition to nonaggressive canker fungi during episodes of water stress in woody plants (167). Desiccation of woody tissues due to drought stress leads to loss in the mechanical strength of the bark-wood bond (131) and can result in bark cracks that can be invaded by opportunistic endophytes (21). Transplanted tree seedlings can also experience a severe physiological shock due to injury to root systems and consequent loss in capacity for water absorption (96), resulting in the activation of cryptic infections (118).

Root and crown diseases caused by *Phytophthora* spp. and other soilborne pathogens have provided models whereby cycles of soil saturation, drought, and salinity were shown to be dominant predisposing factors to markedly increase disease severity (39, 42, 56, 115). This research defined many of the physiological parameters of stress-induced predisposition in the laboratory, greenhouse, and field, and demonstrated that predisposition can occur as a result of relatively minor stress. Brief exposure to water potential deficits from −0.5 to −1 MPa predisposes various plant species to *Phytophthora* root and crown rots (**Figure 3**). Waterlogging compromises the plant, although the impact varies across species (30, 168). Tomato and tobacco plants will show signs of wilting and leaf yellowing within a few hours of flooding, indicative of the rapid dehydrating effect and impact on hydraulic conductance (81). These studies consider stress effects on both the host and the pathogen and have shown that the *Phytophthora* spp. of concern function effectively in soil during (or, in the case of drought, immediately after) the stresses that cause predisposition. This research has raised grower awareness of the need to optimize watering regimes where possible and brought attention to the importance of predisposing stress in assessing plant performance against certain pathogens, resulting in the incorporation of stress-screens in disease resistance breeding programs (76).

For typical mesophytic crop plants, as the ψ_w declines over the range −0.2 to −2.0 MPa, which encompasses plants that are well-watered to plants experiencing mild water stress, cell expansion

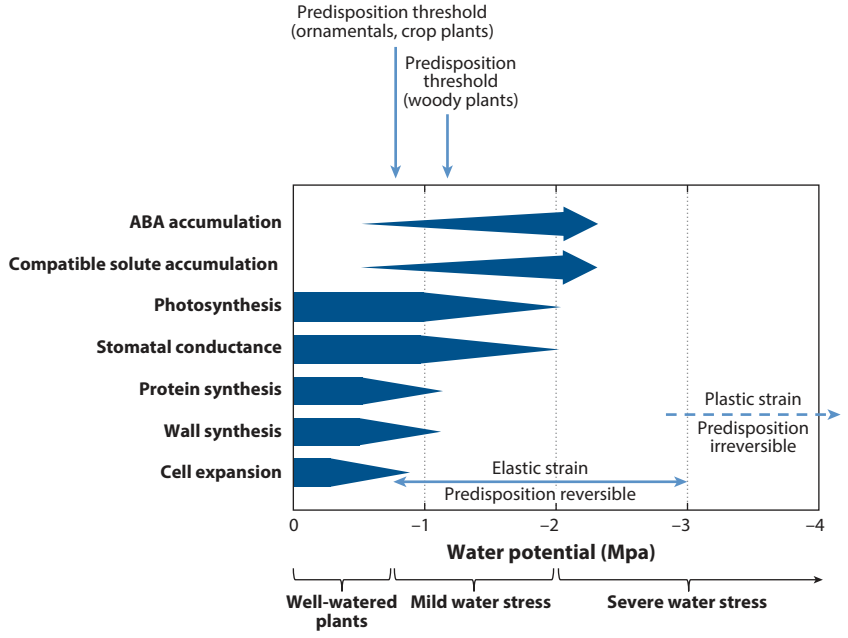


Figure 5

Predisposition thresholds in relation to water potential and major physiological and biochemical changes in plants under water stress. Cell expansion and wall and protein synthesis are most sensitive to a decline in water potential, followed by stomatal conductance and photosynthesis. Abscisic acid (ABA) and osmocompatible solutes begin to accumulate at the threshold for predisposition. Adapted with permission from References 167 and 181.

and wall and protein synthesis are most sensitive, declining rapidly within this range (**Figure 5**). This rapid decline in protein synthesis (48) would seem to compromise the defense budget of the plant and is probably underappreciated in contemporary studies that focus on the dramatic redirection of gene expression and compensatory metabolic adjustments during the stress response. Photosynthesis and stomatal conductance subsequently decline with concomitant accumulation of solutes and induction of the phytohormone abscisic acid (ABA) (181). In many plants, root:shoot ratios increase as part of the strategy to adapt to reduced water availability.

Temperature and Other Factors in Predisposition

Temperature extremes, similar to severe water stress, can create a plastic strain that is directly injurious, particularly when they occur abruptly and the plant is insufficiently acclimated. Such injuries provide infection courts for facultative pathogens. Generally, freezing temperatures between −20°C and −30°C can predispose woody plants in temperate climes to fungal canker diseases and diebacks (165). Mild to moderate freezing contributes to development of bacterial canker and short-life disorders in stone fruit trees (29).

Temperature stresses of a milder form provoke elastic strains that increase disease proneness. Chilling injury can occur in plants at temperatures >0°C and up to 15°C. Chilling is a key predisposing factor in cotton leaf senescence and susceptibility to *Alternaria alternata*, leading to premature defoliation (210), and tropical and subtropical plants are typically quite sensitive to chilling. In the rice blast pathosystem, cold temperature and ABA suppress resistance to

Magnaporthe oryzae (94). Cold temperatures induce water stress, wilting and leaf necrosis, and ABA (184). Nonetheless, transcriptome analysis shows both positive and negative regulation of gene expression by ABA in chilled pepper plants, and ABA application can alleviate some symptoms of chilling injury (69). The physiological changes occurring during cold acclimation can also enhance disease resistance in some plants, such as occurs in various winter cereals to snow mold diseases (101). Salicylic acid (SA) biosynthesis is induced by cold temperatures with a corresponding induction of pathogenesis-related (PR) proteins, possibly as a preemptive strategy to minimize the impact of potential infections of injured host tissue (93).

The integration of downstream signaling generated by the seemingly disparate stresses of cold and pathogen infection is evident from studies with a chilling sensitive mutant (*chs2*) in *Arabidopsis* in which a shift from 22°C to temperatures below 16°C causes a hypersensitive response (HR)-like cell death accompanied by electrolyte leakage, PR gene expression, and ROS and SA accumulation (79). *CHS2* encodes the R protein RPP4, which is effective against the downy mildew pathogen *Hyaloperonospora arabidopsidis*. A single amino acid substitution in the nucleotide binding domain confers the temperature-dependent deregulation of RPP4 in the *chs2* mutant that results in chilling sensitivity and the associated reactions that mimic a response to infection. The integration of cold and canonical defense responses is further evidenced by shared regulators and transcriptional repressors and activators (187).

Transient episodes of heat stress can increase susceptibility or resistance to pathogens. In nurseries, pots exposed to direct sunlight can achieve soil temperatures of 45°C or more, which can cause heat injury to ornamental roots and significantly increase severity of *Phytophthora* root rot (117) (**Figure 4*b***). Even mild temperature elevation can nullify host resistance, the classic examples being interactions conditioned by temperature-sensitive *R* genes (212). Elevated temperatures can also induce resistance, as in cucumber seedlings exposed to a heat shock of 50°C for 40 s (174). Heat-shock factors, which recognize consensus sequences in the promoters of heat-induced genes, and PR and heat-shock proteins are associated with heat shock–induced resistance in various species (100).

Although all mineral nutrients in inappropriate amounts can impact severity of disease (53), the impact of nitrogen (N) fertility is the most commonly reported and studied (80). Although excess N often increases disease, insufficient N can have a similar effect (171). Also important is the form of N as ammonium or nitrate because each can affect diseases differently. In addition to N form, a number of factors contribute to N's impact, including genetics of the host, applied N rate and availability, and the influence of soil pH and redox on the availability of other ions (80). Ammonium-N increases *Fusarium* wilt of tomato, particularly in acidic soils, and this can be managed with lime and nitrate-N fertilizer, a treatment that also decreases the availability of Mn and Fe. However, wheat take-all disease is reduced by ammonium-N, acidic soil, and Mn. N stress, whether resulting from insufficient or excessive N, results in a decline in photosynthesis and reduced growth (35). N deficiency consistently results in increased ABA levels in plants (35), as does excessive N in the form of ammonium-N (105). There is also a range within which increased N enhances growth and disease susceptibility, as in stem rot of rice (90) and pitch canker of Monterey pine (109). This is interpreted as resulting from a trade-off between growth and defense, which includes less lignification and greater succulence of the tissue.

Atmospheric pollutants, notably SO_2, NO_x, and ozone (O_3), are directly injurious to plants but also indirectly influence plant-pathogen interactions (19). O_3, for example, can increase severity of diseases caused by necrotrophic pathogens and reduce severity of those caused by biotrophs, although this effect can be quite variable (162). The physiological and cellular impacts of O_3 in plants are similar to the effects of treatment with elicitors, with the associated induction of antioxidant systems, expression of PR proteins, and other responses.

PHYTOHORMONE NETWORKING IN PREDISPOSITION

Phytohormones, together with ROS, provide important signals to help orchestrate the similar signaling cascades, transcriptional changes, and metabolic and cellular responses to abiotic and biotic stresses (24, 95, 157). Stress-induced changes in both concentration and perception of phytohormones are important to consider in conducting and interpreting these studies. ABA, jasmonic acid (JA), ethylene (ET), SA, auxin [indole-3-acetic acid (IAA)], and cytokinins (CKs) figure prominently in the literature on disease resistance and susceptibility (157). More recently, gibberellic acid (GA) and its interaction with DELLA proteins as well as the brassinosteroids (BRs) have gained interest in studies of abiotic-biotic stress interactions (18, 137, 146). Whereas SA, JA, and ET have shaped much of the current understanding of induced resistance, it has become evident that there is considerable cross talk among all the major phytohormones in abiotic and biotic stress signaling and response. Phytohormone and ROS modulation of immunity in plant-microbe interactions have been reviewed recently (126, 148, 157, 191). These excellent reviews cover much of the territory, so here we discuss the aspects most germane to predisposition toward susceptibility, with an emphasis on ABA and its interactions (**Figure 6**).

Abscisic Acid

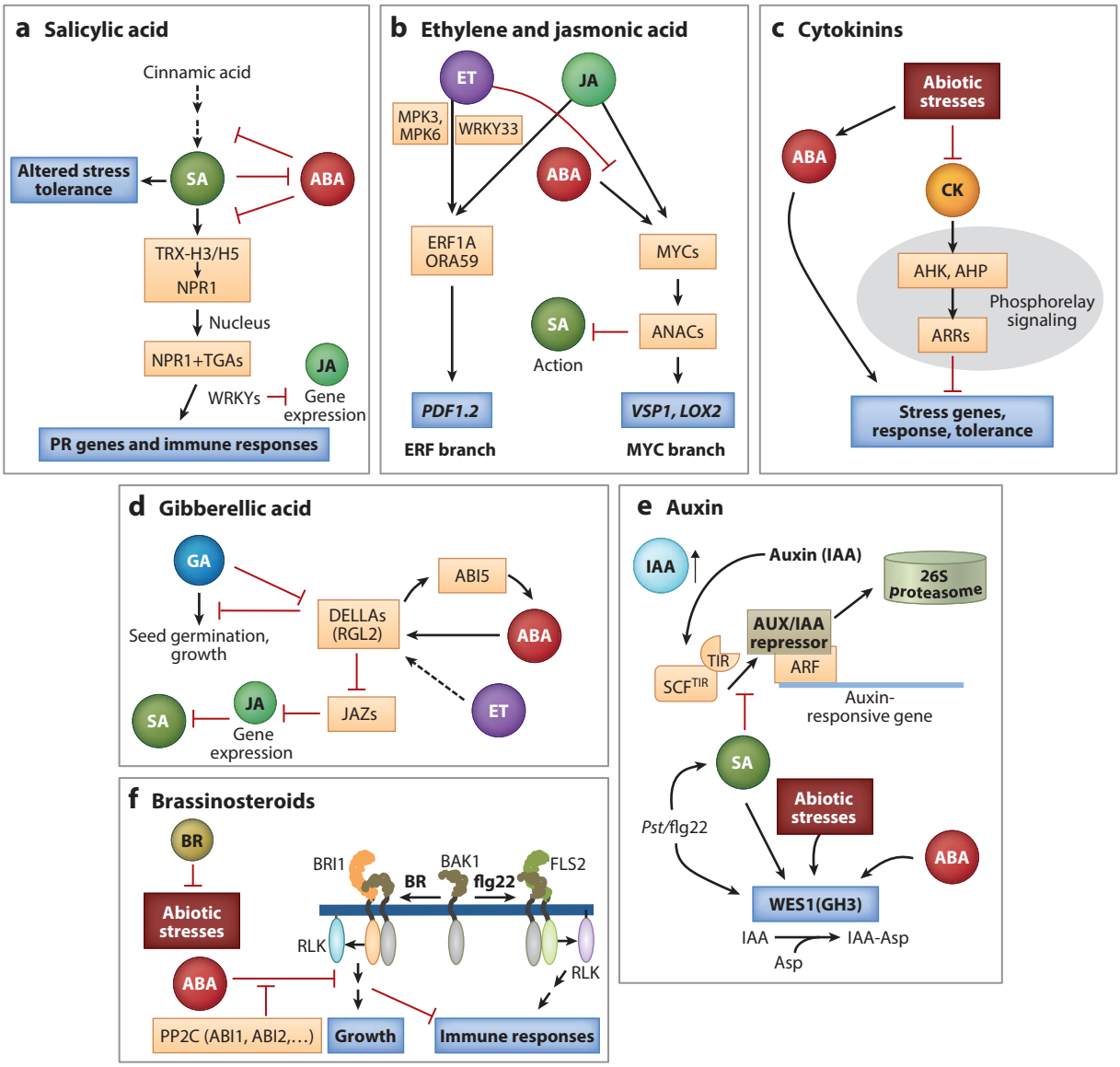
ABA is a highly conserved stress-related signal that occurs throughout all kingdoms except Archaea and appears to be universally triggered under conditions of limited cellular water availability (74). In higher plants, ABA, a C_{15} isoprenoid, is synthesized by the plastidial 2-*C*-methyl-D-erythritol-4-phosphate (MEP) pathway via cleavage of C_{40} carotenoids (136) (**Figure 7**). Discovery and characterization of the PYR/PYL/RCAR family of ABA receptors and the core signaling pathway are leading to a deeper understanding of ABA action within different developmental and environmental contexts (41) and revealing opportunities that may lead to improved stress tolerances in plants (83, 202). Many stress responses engage ABA, and, remarkably, it is estimated that as many as 10% of all protein coding genes in *Arabidopsis* show differential expression following ABA treatment. Many of these genes respond to abiotic and biotic stresses (92). Several studies, including a meta-analysis of a series of stress transcriptomes (4), provide evidence for ABA as a signal for defense-related gene expression (119). ABA-responsive elements are highly represented in the promoters of many of these genes, although ABA alone may be insufficient to fully engage their expression and, in some cases, may even suppress expression.

ABA's primary role in stress responses is to evoke adaptive physiological changes toward water balance and cellular dehydration tolerance (38, 74, 188), which includes guard cell regulation (86). ABA, acting in concert with other phytohormones, helps maintain root growth while reducing shoot growth and photosynthetic rate during stress episodes, alters the capacity for nutrient uptake, and invokes gene expression leading to protective proteins (e.g., LEA proteins and dehydrins) and osmocompatible solutes (35, 78).

Abscisic Acid in Disease

A causal role for ABA in disease is supported by studies with ABA-modified plants (12, 119, 162). Early investigations of ABA action in plant-microbe interactions in potato (75) and soybean (31, 120, 127) demonstrated that ABA pretreatment abrogated race-cultivar resistance to *Phytophthora* spp. and suppressed phytoalexin accumulation and defense responses, with ABA-synthesis inhibitors enhancing resistance. ABA predisposes wheat seedlings to *Fusarium culmorum*, causal agent of foot rot, a classic drought-incited disease (123), and pine seedlings to *Cylindrocarpon destructans*

(98). Most studies now incorporate ABA-synthesis and -perception mutants, and some have examined the connection between abiotic stress, ABA, and disease (50, 94, 183, 206). In *Arabidopsis*, ABA treatment and drought stress induced susceptibility to *Pseudomonas syringae* pv. *tomato* (*Pst* DC3000) (128). The ABA-deficient *aba1-1* mutant displayed reduced susceptibility to compatible isolates of the downy mildew pathogen *H. arabidopsidis*. An ABA-insensitive mutant, *abi1-1*, which has wild-type levels of endogenous ABA, was not altered in its host reaction to *Pst* or to *H. arabidopsidis*, although it is possible that other *ABI* genes may have compensated (41). There is also strong evidence that *Pst* DC3000 co-opts ABA biosynthesis and signaling in *Arabidopsis*, apparently through the action of its AvrPtoB effector, to promote disease in compatible interactions



(47). Whether targeting of host ABA signaling is a strategy used more widely among diverse pathogens remains to be determined.

Roots most directly encounter the predisposing impacts of waterlogging, salinity, and drought. Studies of root-pathogen interactions suggest shared elements and common themes in stress and defense network dynamics with leaves, even if details differ qualitatively and quantitatively. An elegant study of cell type–specific transcriptional responses in *Arabidopsis* roots found that ABA marker genes, unlike those for other phytohormones, are induced by salt stress in a semiubiquitous manner in all cell layers of the root (51). Because of the importance of predisposing stress in soilborne diseases, we have examined ABA-regulated susceptibility in roots with experimental treatments that are informed by predisposition in the field (23, 56, 116).

The tomato (*Solanum lycopersicum*)–*Phytophthora capsici* interaction provides an important experimentally tractable crop model that can give a clear disease phenotype within 48–72 hours post infection in hydroponic formats. Seedlings are predisposed by brief root immersion in saline solutions, followed by rinse and then return to a standard hydroponic solution and inoculation with zoospores (50). Disease and host colonization are evaluated by seedling collapse and qPCR of pathogen DNA. The acute stress regime results in a rapid phase 1 response as defined in salinity research (133), and noninoculated but stressed plants recover completely. In tomato roots, ABA increases rapidly, peaking 6 to 12 hours after salt treatment, and then declines (50), although the timing may vary depending on the size and age of the plant. ABA induction precedes or temporally parallels the onset of the predisposed state, which is evident within 4–6 hours of salt exposure and persists for up to 24 h after salt removal, well after the decline in ABA levels to near prestress levels. Salinity stress in roots and its systemic impact are evident in various interactions, with similar effect in leaves challenged with the bacterial speck pathogen *Pst* (152, 183) and in soilborne infections by *Phytophthora* spp. in several ornamental species (50, 159).

Roots and shoots of wilty ABA-deficient tomato mutants accumulate only a fraction of the ABA present in wild-type plants and do not show the adaptive responses to water stress. Following a

Figure 6

Phytohormone interactions in abiotic and biotic stress signaling and response. In each panel, black arrows indicate a positive or feed-forward effect and red blocked lines indicate inhibition. Dashed lines imply indirect or multiple steps in the sequence. Factors involved in signal-response coupling (e.g., kinases, phosphatases, transcription factors, etc.) are in light orange boxes, and downstream outputs (gene expression and responses) are in light blue boxes. The models are not intended to be comprehensive and are derived from those presented in detail in References 148, 153, 157, 196, and 209. (*a*) Salicylic acid (SA) path for induced resistance and antagonism between SA and abscisic acid (ABA). SA has various effects on abiotic stress tolerance as well. (*b*) Ethylene (ET) and jasmonic acid (JA) and the ERF (ethylene responsive factor) and MYC response branches. (*c*) Cytokinins (CKs) counteract ABA responses to water stress, but CKs can also enhance stress tolerance. In *Arabidopsis*, CK signal perception and transduction are mediated by a two-component histidine-kinase phosphorelay system composed of *Arabidopsis* histidine kinases (AHKs), histidine phosphotransfer proteins (AHPs), and response regulators (ARRs), with members of the latter providing positive and negative feedback regulation of CK signaling. (*d*) DELLAs act as repressors of gibberellic acid (GA) signaling and of JAZ (JASMONATE-ZIM DOMAIN) repressors, the latter resulting in the release of JA-mediated gene expression and cross talk with SA. (*e*) Auxins, such as indole-acetic acid (IAA), as well as ABA, SA, and biotic and abiotic stressors, can induce the GH3 auxin–amino acid–conjugating enzyme WES1 to lower free auxin pools in the cell. SA can also interfere with auxin-mediated gene expression. (*f*) Brassinosteroids (BRs) can mitigate abiotic stresses. They share BAK1 (BRI-associated receptor kinase) with the PAMP (pathogen-associated molecular pattern) flg22, and thus may compete for BAK1's attention. BAK1 complexes with other receptor-like kinases (RLKs), leading to downstream effects. Abbreviations: ABI5, abscisic acid insensitive 5; ANAC, abscisic acid–responsive NAC; ARF, auxin response factor; Asp, aspartic acid; BRI1, brassinosteroid insensitive 1; DELLA, DELLA protein; GH3, Gretchen Hagen 3; *LOX2*, lipoxygenase 2; MPK, MAP kinase; MYC, MYC transcription factor; NPR1, nonexpressor of pathogenesis-related genes; ORA, octadecanoid-responsive *Arabidopsis AP2/ERF*; *PDF1.2*, plant defensin 1.2; PR, pathogenesis related; RGL2, RGA-like 2 transcriptional regulator; SCF, Skp1-Cullin1-F-box; TGA, TGA transcription factor; TIR, transport inhibitor response; TRX, thioredoxin; *VSP1*, vegetative storage protein 1; WES1, Weso 1; WRKY, WRKY transcription factor.

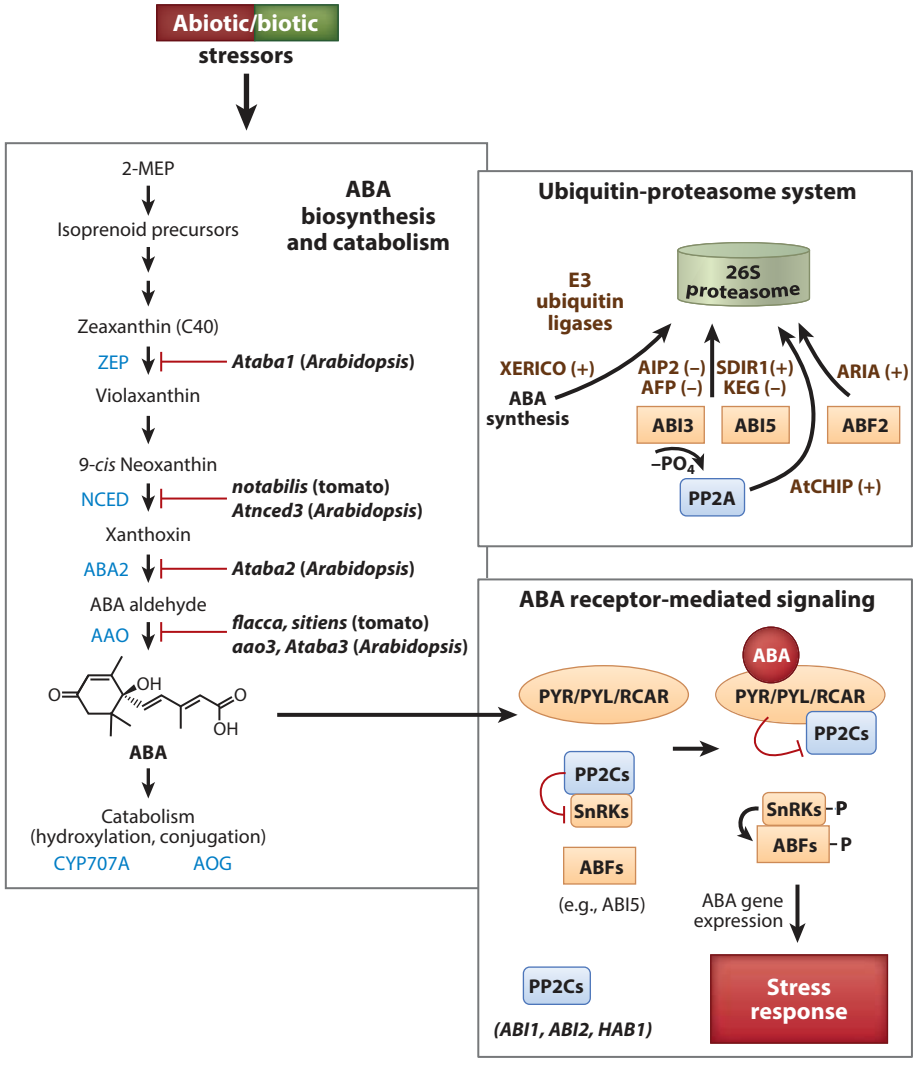


Figure 7

Abscisic acid (ABA) biosynthesis and features of ABA signaling. Black arrows indicate a positive or feed-forward effect, and red blocked lines indicate inhibition. ABA biosynthesis and catabolism abbreviations: 2-MEP, 2-C-methyl-D-4-erythritol phosphate; ZEP, zeaxanthin epoxidase; NCED, 9-*cis*-epoxycarotenoid dehydrogenase; ABA2, short chain alcohol dehydrogenase; AAO, abscisic aldehyde oxidase; CYP707A, a family of ABA-8′-hydroxylases; AOG, ABA O-glucosyl transferases. Various biosynthesis mutants in *Arabidopsis* and tomato are indicated. ABA receptor-mediated signaling: binding of ABA to the PYR/PYL/RCAR family of receptors inhibits PP2Cs, a family of serine-threonine protein phosphatases (e.g., ABI1, ABI2, HAB1). PP2Cs normally keep SnRKs (SNF1-related protein kinases) at bay. In the presence of ABA, SnRKs phosphorylate transcription factors [ABFs (ABA response-element binding factors); e.g., ABI3, ABI5] to activate gene expression. Ubiquitin-proteasome system: selected E3 ubiquitin ligases that target various elements to promote (+) or inhibit (–) ABA signaling. PP2A is another family of serine-threonine protein phosphatases. See References 41, 54, 61, 111, and 136 for further details. Abbreviations: ABI, abscisic acid insensitive; AIP2, ABI3-interacting protein 2; AFP, ABI5 binding protein; ARIA, arm protein repeat interacting with ABF2; CHIP, C-terminal HSP interacting protein; KEG, keep on going E3 ligase; SDIR1, salt- and drought-induced RING finger 1; XERICO, a RING-H2 zinc finger protein, from the Greek, meaning "drought tolerant."

stress episode, these mutants show a significantly diminished predisposition to *P. capsici* relative to wild-type plants (50). The difference is most dramatic in the *sitiens* mutant, having the most depressed phenotype, with less than 10% of the ABA levels of wild-type seedlings. However, exogenous ABA only partially complements the *sitiens* mutant to full susceptibility (50), suggesting additional ABA-independent factors contribute to the predisposing effect of salt or, perhaps, synergy between salt and ABA in conditioning the plant response, as observed, for example, in rice cells (25). In fungal (11) and bacterial (10) diseases of tomato in which ABA pushes the interaction toward susceptibility, *sitiens* displays a rapid induction of defense responses that are otherwise diminished in the wild type.

Although there are many examples of ABA shifting the host-pathogen dynamic toward susceptibility (12), there are also examples in which abiotic stress and ABA enhance disease resistance (32, 185). Notable among these are powdery mildew (3) and other fungal diseases and several bacterial diseases. In the latter case, stomatal closure triggered by ABA in concert with SA may prohibit ingress of the pathogen (121), although recent evidence indicates an important role for ABA-independent oxylipin signaling in stomatal function during drought stress and pathogen infection (130, 163). Recognition of this complexity prompted a model that considers parasitic strategy (66), the timing and intensity of ABA signaling during the course of infection, and ABA's interaction with SA and JA to explain different outcomes (185).

Defense Network Signaling During Predisposing Stress: Does Abscisic Acid Trump Salicylic Acid, Jasmonic Acid, and Ethylene?

Changes in SA, JA, and ET that strongly modulate defense against biotic attackers coincide with changes in ABA during abiotic stress events (24, 181). Central to the downstream signaling that couples SA to induced resistance are the thioredoxins TRX-H3 and TRX-H5 and the transcription cofactor NPR1 (nonexpressor of PR genes 1). Monomerization and nuclear localization enable NPR1 to combine with members of the TGA subclass of bZIP family transcription factors (TFs) to activate defense gene expression (148, 191). However, SA has additional, and sometimes conflicting, physiological effects other than its importance in defense signaling (191). SA can increase or decrease plant tolerance to various abiotic stresses, depending on how much and where it is applied, the developmental stage of the plant, and the plant's overall redox status and stress history (78). SA can interfere with or synergize ABA- and osmotic stress–regulated gene expression and response (190, 206), disrupt the plant's ability to maintain water balance (17), induce partial tolerance to predisposing levels of salt stress (175), and contribute to redox homeostasis and protection against oxidative stress in various contexts (190) (**Figure 6*a***).

ABA inhibits SA-mediated acquired resistance to *Pst* in *Arabidopsis* and tomato, whether the ABA is applied exogenously or induced by drought or NaCl stress (46, 60, 129, 183). Details of the cross talk between ABA and SA have been further delineated in *Arabidopsis* in a compelling study by Yasuda et al. (206), who used various signaling mutants and two resistance-inducing SA mimics, one that acts upstream of SA to stimulate its synthesis, and one that acts downstream of SA to engage its targets. ABA and NaCl stress inhibited the action of both SA mimics, with evidence from various mutants for reciprocal inhibition by SA of stress-induced ABA synthesis and ABA-regulated gene expression. The results implicate multiple points of interaction between ABA and SA as well as both dependence and independence of NPR1 in the outcome of the cross talk. ABA's inhibition of SA-mediated defenses also occurs in monocots, as recently reported in the interaction of rice with the bacterial leaf blight pathogen *Xanthomonas oryzae* pv. *oryzae* (201).

We examined the influence of SA on NaCl predisposition to *Pst* and *P. capsici* in wild-type and *nahG* transformed tomato seedlings that are compromised in SA-mediated resistance. In

both diseases, *nahG* plants were more susceptible than the wild type, with salt stress causing the expected additional increase in disease severity in both host backgrounds (152). However, the impact of predisposing stress in the *nahG* lines was proportionally the same as in the wild type. One interpretation is that the impairment of SA action following osmotic stress contributes little relative to the dominating impact of ABA and brings into question any direct role of SA-mediated processes to counteract predisposition within our brief stress regime.

JA and ET also interact with ABA and SA, although whether these interactions are positive or negative in target gene expression and response depends on the context (4, 7, 28, 44, 60). Various abiotic and biotic stresses induce JA and/or alter JA sensitivity to induce defense gene expression and adaptive responses. JA is an oxylipin derived from α-linolenic acid in plastids, with further metabolism in peroxisomes (197). JA is converted to its isoleucine conjugate, JA-Ile, which binds to the F-box factor COI1 in association with an SCF E3 ubiquitin ligase. This activated SCF^{COI1} complex then binds to JAZ (JASMONATE-ZIM DOMAIN) transcriptional repressors to release and target them for degradation by the proteasome, alleviating the block on transcription (197).

There appear to be two pathways through which JA acts in stress responses, the ERF (ethylene responsive factor) branch and the MYC branch, each with different outputs and regulation, that are mutually antagonistic (148) and are conserved in *Arabidopsis* and tomato (26) (**Figure 6*b***). In the ERF branch, ET conspires with JA to induce expression of TFs, ERF1A/ORA59, and their target PR genes (e.g., *PDF1.2*), responses typically associated with resistance to necrotrophs. In the MYC branch, MYC TFs are induced by the coordinated action of ABA and JA to induce other TFs (ANAC019, ANAC055, and ANAC072) that engage gene expression to suppress SA accumulation, coordinate responses to water stress, and counter insect herbivory (89). MYC2 is induced by JA-JAZ derepression and serves as a core master regulator that forms homo- and heterodimers with MYC3 and MYC4 in complex with other proteins to orchestrate transcription in diverse JA responses (88). MYC2 also shows regulation by ABA and is required for ABA-dependent gene expression. The synergy of JA and ABA as translated through MYC2 may explain the enhanced disease resistance in some interactions.

The model that emerges is that JA-mediated stress responses are highly coordinated and finely tuned to address various challenges. There are many examples in which JA synthesis and signaling mutants display reduced resistance to bacterial, fungal, and oomycete pathogens and insect herbivores, affirming JA's position as a defense mediator (197). NPR1 is important in the negative cross talk between JA and SA (148), and a number of WRKY TFs, notably WRKY70, further contribute to modulating the SA-JA interaction to balance these different defense pathways (106). Although sophisticated circuitry engages JA to address different stresses, as with SA, it is unclear if JA and SA coordinate to counteract predisposition and how they negotiate ABA's strong induction during stress episodes. Also unresolved is the degree to which the antagonism between JA and SA is expressed to increase vulnerability to pathogens during stress events (24).

Molecular mechanisms governing induction and perception of ET as well as ET action and cross talk with other hormones in stress responses have been extensively studied (95). In *Arabidopsis*, stress-responsive mitogen-activated protein kinases (MAPK; i.e., MPK3 and MPK6) are important for inducing ET and phosphorylating specific ERF and WRKY TFs to trigger gene expression during infection (122). One of these targets, WRKY33, has been shown to be important for integrating collaborative signaling between JA and ET (22). ET can also be an important player in defense response activation by altering the JA-SA conflict. Depending on the timing of the ET signal during attack, NPR1's intermediary role can be circumvented (103) or SA's suppression of JA signaling can be abolished (102). Although ET interacts with multiple hormones and can modify disease resistance in plant-microbe interactions, including potentiating programmed cell death (PCD) (132), its contribution to predisposition is unclear. In tomato, we found that ET

was not induced by the brief episode of salt stress in our predisposition regime. Furthermore, the disease phenotype to *P. capsici* in the ET-insensitive *Never-ripe* mutant was not different from that in the wild type (50). Although ET can be induced in tomato during salt stress (6), this occurs well after ABA induction and far later than the time window of our stress regimes and disease assays. ET could be a factor during stress events of longer duration, and as with ABA and other hormones, ET is known to contribute to salt-stress adaptation (114), but additional research is needed to address any contributing role in predisposition.

Cytokinins Temper Abiotic Stress

CKs generally oppose ABA action in plants under water stress, with extensive cross talk between CKs and ABA and impacts on both ABA-dependent and ABA-independent stress responses (70). Short-term stresses, such as the acute salinity regime in our predisposition format, briefly elevate CK levels, but longer or more severe stress episodes reduce growth as well as CK levels and shift resources toward defense (**Figure 6*c***). CKs are adenine derivatives containing either isoprenoid or aromatic side chains (112). The principal isoprenoid CKs are isopentenyl, *trans*-zeatin, *cis*-zeatin, and dihydrozeatin derivatives, and the rate-limiting enyzmatic step in their synthesis is isopentenyl transferase (IPT). CK-modified tobacco plants in which IPT is transgenically expressed under control of the maturation- and stress-inducible promoter for the senescence-associated receptor-like kinase (SARK) show an increased tolerance to drought stress, attributable in part to CK-induced delayed leaf senescence (155). However, CK-deficient mutants in *Arabidopsis* also show strong stress tolerance to drought and salinity as well as ABA hypersensitivity and reduced levels of ABA (140). These results suggest finely tuned mechanisms for maintaining CK homeostasis and action, in part through adjustments in ABA synthesis and signaling.

CKs appear to contribute to the successful establishment of biotrophic infections (192). Recently, it was reported that the *Pst* effector HopQ1 suppresses immune responses in *Arabidopsis* and induces CK signaling in the plant (72). However, CKs may also interact with SA through their activation of ARR2 TFs that participate in SA-mediated defense gene expression (37) (**Figure 6*c***). How the plant negotiates these apparent countervailing CK actions is unclear, although the levels of CKs may be the critical factor here, with low concentrations promoting susceptibility and high concentrations promoting resistance (72). Although the adaptive role of CKs in plants under drought and salinity stress is complicated, there is intense interest in how plants balance the stress-adaptive functions of ABA and CK in efforts to improve environmental stress tolerance, which may have implications for tolerance to pathogens as well (112, 199).

DELLA Proteins and Gibberellic Acid

DELLA proteins are a nuclear family of TFs that are now recognized as integrators of phytohormone signaling. DELLAs repress growth and GA signaling, and GA reciprocates by derepressing its pathway to promote degradation of DELLAs via the ubiquitin-proteasome system (190) (**Figure 6*d***). In *Arabidopsis*, there are five DELLA genes that encode proteins directed at different and overlapping targets in processes associated with germination, cellular redox, growth, and abiotic and biotic stress responses, and mutations in these genes lead to a number of altered GA and stress phenotypes (67, 190). One of these DELLA proteins, RGL2, is important in repressing seed germination and is induced by ABA, and in turn increases ABA levels and the expression of the TF ABI5. In addition to their role as repressors of GA signaling and of growth, DELLAs are stabilized by ABA and ET to potentiate cross talk between SA and JA through promoting JA action and inhibiting SA synthesis and signaling (67, 137). JA's inhibition of plant growth in favor

of defense appears to be attributable in large part to interference with GA signaling that leads to DELLA protein degradation, indicating antagonism between JA and GA (204). DELLAs bind to JAZ repressor proteins to liberate MYC2 and promote JA-responsive gene expression. DELLAs mediate salt stress–induced inhibition of growth, and an *Arabidopsis* mutant with knockouts of four of the DELLA genes is more resistant to salt than are wild-type plants (1). In *Arabidopsis*, DELLAs also increase susceptibility to *Pst* DC3000 (137). Likewise, the quadruple DELLA knockout shows increased resistance to *Pst* DC3000, generally regarded as a hemibiotroph, but increased susceptibility to the necrotrophic fungi *Alternaria brassicicola* (137) and *Botrytis cinerea* (2). The evidence positions DELLAs as potentially important contributors to the predisposing effects of salt and other stresses on disease, and further research is needed to address this issue.

Auxin

Auxin integrates the effects of multiple phytohormones to regulate plant growth and development and root system architecture in response to changing environments (64, 82). Auxin stimulates lateral root formation as a response to nutrient limitation; however, drought and salinity stress repress auxin signaling and lateral root development to favor primary root growth to tap water deeper in the soil (70). The interplay of auxin and the counteracting effects of ABA and CK contribute to these different outcomes, due in part to differential regulation of the TF ABI4 and adjustments to polar auxin transport (170).

Auxin's role in plant-microbe interactions is well established in the traditional sense, with pathogen- and host-derived IAA contributing to disease phenotypes such as galls and fruit russeting. However, auxin action in predisposition has not been thoroughly investigated, although there is compelling evidence for auxin modulating SA and JA defense networks at several levels (**Figure 6*e***). The major categories of auxin-responsive genes are grouped in the *Aux/IAA*, *GH3*, and small auxin-up RNAs (SAUR) families. AUX/IAA proteins are transcriptional repressors that are removed from their targets by auxin-activated SCF^{TIR1} receptor complexes and tagged for proteasome degradation by the SCF E3 ubiquitin ligase pathway (20). In *Arabidopsis*, the eubacterial PAMP flg22 binds to the receptor kinase FLS2 to trigger an immune response that includes posttranscriptional silencing of mRNAs for the F-box auxin receptors TIR1, AFB2, and AFB3 (138). This stabilizes Aux/IAA repressors, and the repression in auxin signaling further enhances resistance to *Pst* DC3000. SA treatment also downregulates auxin signaling in a similar fashion (194), providing evidence that auxin and SA counteract each other, with auxin diminishing resistance and SA having the opposite effect, at least on *Pst*. In contrast to *Pst*, necrotrophic fungal pathogens exploit auxin signaling during pathogenesis by stabilizing another group of auxin transcriptional repressors (i.e., AXRs), resulting in enhanced susceptibility (108). The different outcomes might be explained by *Arabidopsis*'s contrasting defense strategies in dealing with biotrophs and necrotrophs (66).

Another level of control of auxin action is provided by the auxin-inducible *GH3* genes, which encode acyl acid amido synthetases that regulate the endogenous auxin pool by catalyzing formation of auxin–amino acid conjugates (144). One of these, *WES1*, displays complex regulation and induction by SA, ABA, and *Pst* infection as well as drought, cold, and heat stresses. An overexpressing mutant (*wes1-D*) displays severe dwarfing characteristic of auxin deficiency as well as altered phenotypes to the various abiotic and biotic stresses. Other *GH3* family members encode enzymes that conjugate amino acids to JA (i.e., JA-Ile) and SA, providing another mechanism for adjusting levels of these phytohormones and cross talk (173).

An intriguing model positions auxin as a transitional signal between phases of JA and SA signaling to establish systemic acquired resistance (SAR) (186), predicated in part on the above-cited

studies but also on the key observation of an early transient spike in JA that precedes SA signaling in *Arabidopsis* following inoculation with incompatible *Pst*. In this model, the initial JA phase induces indole biosynthesis that includes auxin as well as JAZ repressors to begin to dampen JA signaling. As JA action subsides, the auxin phase follows and engages the auxin importer AUX1 to import and position IAA for interaction with the TIR receptor to derepress auxin-regulated transcription. Auxin induction of *GH3* genes provides a mechanism to diminish auxin signaling, facilitating the next transition to SA-mediated defense activation and priming for SAR. It will be interesting to see if this model can be generalized to other host-pathogen interactions and whether these signaling transitions are disrupted by ABA and predisposing abiotic stresses.

Brassinosteroids

BRs, e.g., brassinolide, are a group of more than 70 polyhydroxysteroids with impacts on various aspects of plant growth and development as well as a potential protective role in abiotic and biotic stress responses (142). Application of BRs can induce protection against osmotic, drought, and temperature stresses, suggesting that they could counter predisposition to diseases. BRs induce oxo-phytodienoic reductase in JA biosynthesis and when applied with other phytohormones also show additive or synergistic effects (e.g., GA and auxin) or mutual antagonism (e.g., ABA) (209). The impact of BR treatment on host-pathogen interactions is mixed, with examples of increased resistance (135) as well as increased susceptibility (16). BR signaling has emerged as a potential target for engineering tolerance to both abiotic and biotic stresses (52).

BR perception and signaling are tightly regulated through phosphorylation and dephosphorylation reactions (**Figure 6*f***). The BR INSENSITIVE 1 (BRI1) receptor is a leucine-rich repeat receptor kinase (LRR-RK) that requires BAK1 (BRI1-associated kinase) as a coreceptor to positively regulate downstream BR signaling. An interesting connection between BR and PAMP-triggered immunity occurs at the level of their perception by RKs. The PAMP receptor FLS2 almost immediately after binding flg22 forms a heteromer with BAK1 to trigger transphosphorylation and a phosphorylation cascade of downstream targets (178). Although FLS2 and BRI1 would appear to negatively regulate the other's action by competing for BAK1, experimental evidence indicates that the outcome of their interaction can be positive or negative, depending on levels of BR, BRI1, and BAK1 (196). The evidence points to coordination of BR signaling and PAMP-triggered immunity during growth and abiotic/biotic stress events. Additional details of receptor-like kinases and BR perception and response are reviewed in Osakabe et al. (142).

Oxidative Stress and Reactive Oxygen Species Signaling in Predisposition

ROS generation is an initiating event in biotic and abiotic stress responses, and ROS signaling is integrated with multiple phytohormone networks (126). ROS function as both localized and distance signals through their propagation as waves in the plant from an initiating stimulus. Phosphorelay systems are important both in generating ROS and in modulating ROS signaling. ROS generation by various isoforms of the *Arabidopsis* NADPH-dependent respiratory burst oxidase homolog oxidase complex (i.e., AtRBOH) involves calcium signaling and phosphorylation (126). In turn, MAP kinase cascades are engaged by elevated ROS levels to regulate TFs and gene expression, leading to stress adaptation, such as cold- and salt-stress tolerance (182). A MAPK cascade that couples ABA, catalase induction, and H_2O_2 generation is mediated by MKK1 and MPK6 (200).

Tight regulation of the steady-state levels of ROS is required in multiple cellular processes in plants (8), with more than 150 genes in the network managing ROS in *Arabidopsis* (125). Analysis of

the NaCl-induced transcriptome in roots suggests a high degree of subfunctionalization within this network at the regulatory and catalytic levels (85). The majority of genes in the ROS-scavenging network are unresponsive or downregulated within the salt-stress regimes we have found to be capable of predisposing. Water stress induces changes in ROS levels, and many ABA-regulated genes are also induced by oxidative stress (36). *AtrbohD* and *AtrbohF* appear to be required for ABA-induced ROS generation and are linked to ABA-induced changes in guard cell turgor and stomatal function as well as hypersensitive cell death in response to avirulent pathogens (160). ABA and water stress upregulate transcripts of abscisic aldehyde oxidase (AO; e.g., *AAO3*) (**Figure 7**) and transcripts for *LOS5/ABA3*, responsible for AO and xanthine dehydrogenase (XDH) activation (207). Thus, water stress can enhance ROS production via AO and XDH in an ABA-dependent manner, and place Rboh, AO, and XDH as candidates for ABA-dependent ROS transducers involved in predisposition.

Transcription Factors and Network Interactions

There are a number of important TFs not mentioned above that provide points of convergence in abiotic and biotic stress response networks and have the potential to alter disease outcomes during predisposing stress (63, 203). Atkinson & Urwin (13) discussed TF functions more from the point of view of coordinating gene expression to enhance stress tolerance and defense against pathogens. Notable among these are various members belonging to the MYB, NAC, AREB/ABF (ABA response-element binding factor), GBFs (G-box binding factors), and AP2/ERF families that are regulated by ABA. MYB family members are important in gene expression for the biosynthesis of phenylpropanoids, flavonols, and cuticular wax as well as other defensive products, including ABA-dependent SA accumulation, and seem to enable the plant to discriminate between different stress signals (164). NAC family members, such as ATAF1, ATAF2, and RD26 in *Arabidopsis*, are inducible by ABA and various abiotic and biotic stresses (84). The AREB/ABFs and GBFs are subgroups within the bZIP class of TFs and are important integrators of ABA signal-response coupling (77). AP2/ERF family members were discussed in relation to ET signaling, but others, such as DEAR1 (*Arabidopsis*) and TS1 (tobacco), contribute to abiotic and biotic stress regulation of gene expression. Overexpression of *TS1* enhances tolerance to osmotic stress and resistance to *Pseudomonas syringae* pv. *tabaci* (145). DEAR1 represses *DREB* genes to diminish tolerance to cold stress but enhances pathogen resistance (187). The DREB/CBF TFs are ERF family members that bind to *cis*-acting promoter elements of genes conferring dehydration and cold tolerance (5). In *Arabidopsis*, DREB1A is induced by cold but not by drought or ABA. ABA-independent targets should also be considered in predisposition studies.

PROGRAMMED CELL DEATH AND PREDISPOSITION

The connection of PCD with abiotic stress and disease is indicated by hallmark PCD features in cells responding to various stresses and the broad-spectrum protection afforded by transgenic expression of antiapoptotic genes (49). Expression of proapoptotic (e.g., *Bax1*) and antiapoptotic genes (e.g., *Bcl-2*, *CED9*, *P35*, *AtBI1*) from diverse organisms respectively induce and protect plants against PCD, providing evidence for cross-kingdom commonalities in cell death mechanisms. For example, SfIAP, a negative regulator of PCD from the insect *Spodoptera frugiperda*, is a member of the IAP (inhibitor of apoptosis) family of proteins (49). Transgenic expression of SfIAP in tobacco and tomato plants confers tolerance to heat, salt, and the mycotoxin FB1 as well as resistance to the necrotrophic fungal pathogens *A. alternata* and *Sclerotinia sclerotiorum* (107). Also, SfIAP-transgenic tomatoes are impaired in *EIN3* and ET-mediated gene expression, display delayed fruit

ripening, and are less sensitive to the inhibitory effect of ABA on plant growth. SfIAP appears to have a conserved function for inhibiting stress-induced cell death, and when expressed in plants may operate through ABA- and ET-regulated mechanisms. SfIAP's protection is attributable to its E3 ubiquitin ligase activity, and regulated protein degradation via the proteasome is essential for SfIAP-induced stress tolerance and delayed fruit ripening (87). Targeting PCD is an attractive strategy with the potential for improving abiotic and biotic stress tolerance in plants. Ensuring tight regulation to avoid unwanted effects on plant growth and development and understanding the limitations of PCD within different biological and environmental contexts will be crucial for deployment.

SMALL RNAs IN ABIOTIC STRESS AND DISEASE

Small RNAs (sRNA) play various roles in plant processes, such as developmental patterning and genome integrity, but are best known for RNA silencing in plant-virus interactions (14). These short, 18–25 nucleotide sequences are diverse in genomic distribution, sequence, biogenesis, and regulatory function. MicroRNAs (miRNAs) are a class of sRNAs that target complementary mRNAs for translational repression or cleave their targets through association with RNA-induced silencing complexes (RISCs). Emerging evidence indicates important roles for miRNAs in abiotic stress responses (179). The first implication of sRNAs in ABA signaling was from experiments with the *Arabidopsis hyl1* mutant, which is hypersensitive to ABA. HYL1 partners with DICER LIKE 1 (DCL1) for the biogenesis and accumulation of miRNAs but is not required for posttranscriptional gene regulation (110). The first ABA-induced miRNA to be discovered is *miR393*, which is also strongly upregulated by dehydration, increased salinity, and cold (179). Subsequently, *miR393* was found to be induced by flg22 and to repress auxin signaling as described above (138). *miR159* is also induced by ABA and targets two MYB TFs that are positive regulators of ABA responses in *Arabidopsis* (147).

Plants may rely on miRNA-mediated degradation of ABA signaling factors following stress episodes in order to reduce ABA levels and restore the prestress physiological state (154). In *Arabidopsis*, miRNAs upregulated by ABA include *miR160*, *miR417*, and *miR319*; ABA-downregulated miRNAs include *miR167*, *miR169*, and *miR398* (91). The sets of miRNAs associated with salt and drought stress both overlap with and differ from those induced by ABA, with some showing inverse responses to ABA and stress treatments (91). Similarities in the miRNA expression profiles between drought and salt stress and ABA strengthen a role for miRNAs in ABA-mediated stress responses. These miRNAs mainly target diverse TF families, such as SBPs (squamosa promoter binding protein), MYBs/TCPs (myeloblastosis/teosinte branched1, cycloidea, and PCF), ARFs (auxin response factor), HD-ZIPs (homeodomain leucine zipper), and NFY (nuclear transcription factor Y) subunits.

miRNA regulation in biotic stress responses is highly variable and is likely influenced by the host, the pathogen, and the tissue infected (91). Differential miRNA regulation has been reported in symbiotic interactions and in pathogenic viral, bacterial, fungal, and nematodal interactions. In the soybean–*Phytophthora sojae* interaction, infection altered the expression of a small group of host miRNAs with known roles in abiotic stresses (68). Many of the aforementioned stress-related miRNAs are also upregulated by bacterial pathogens and downregulated by fungal pathogens (91). Recent work has shown that many solanaceous species possess miRNAs of the *miR482/miR2118* superfamily. Members of this miRNA superfamily specifically target the P-loop motif of mRNAs that encode NBS-LRR resistance proteins (169). Degradation of the target mRNA results in the creation of phased, secondary small RNAs (phasiRNAs), which allows basal expression of *miR482* to simultaneously silence multiple *R* genes (151). *miR482* is also suppressed by viral, bacterial,

and fungal pathogens, allowing R proteins to accumulate (169, 211), and is upregulated by abiotic stress (9). Deciphering complex small RNA regulatory networks will require the identification of sets of miRNAs and their targets. Such information will provide additional insights into how plants cope with concurrent biotic and abiotic stresses.

SUMMARY

During evolution, plants developed finely tuned mechanisms to cope with diverse abiotic and biotic stresses. This is our operational paradigm and there are many studies that affirm cooperative signaling and response to mitigate environmental challenges and enhance stress tolerance. Yet we must reconcile the fact that plants often succumb to pathogens that they would normally resist when confronted with transient and relatively mild abiotic stresses. Modern agricultural crops must balance disease and pest resistance, stress tolerance, and growth with associated fitness costs (43), but how well they do this can depend on selection criteria during breeding as well as on the impact of production practices designed to maximize yields or to conserve increasingly limited resources. The details of stress network interactions within current cultivars, which are often adapted to rich resource environments, may differ from land races and old world varieties and depart from the experimental models. The circuitry, although complex and interconnected, may not be as elegantly wired as we would wish to offset the unproductive interactions.

ABA has emerged as a global regulator of abiotic stress responses and an important phytohormone in plant-microbe interactions with systemic effects on resistance and susceptibility (**Figures 6**, **7**, and **8**). It has become apparent that cross talk occurs among all the major phytohormones during stress events, and the challenge is discerning which interactions most influence disease development in particular contexts. Transcriptomics has enabled identification of large sets of coordinately regulated stress genes, but interpretation of the results in the context of predisposition can be confounded in that the output may lack sufficient resolution to ascribe functional correspondence to disease outcomes. For example, many ROS- or ABA-regulated defense genes are induced in both susceptible and resistant interactions as well as by other stresses, but issues of timing and intensity as well as whether mRNAs are translated may not be apparent from the data without further analysis. Critical for interpretation of such studies are corroborative quantitative assays that rigorously distinguish disease phenotypes.

Delineation of stress network interactions has implications for disease management, such as in deploying chemically or biologically induced resistance (156, 193). The efficacy of plant activators depends on the environmental context, and abiotic stresses are expected to influence JA and SA network dynamics during pathogen attack (24). Encouraging are results acquired when using the SA mimic tiadinil, albeit conducted under highly controlled experimental conditions, which induces disease protection in tomato under salinity stress (152). This and other studies suggest that plant activators may offset or at least function adequately under conditions of predisposing stress, but further assessment under the environmental rigors of the field is needed.

Various strategies under consideration to enhance plant stress tolerance include using agonists and antagonists of phytohormone action, engineering phytohormone response pathways, and breeding (143, 146, 199). An interesting example of the latter is in soybean, where a quantitative trait loci analysis of recombinant inbred lines found that genes for both flooding tolerance and resistance to *P. sojae* were necessary to reduce disease under flooded conditions (139), illustrating the potential synergy derived from combining traits for stress tolerance and disease resistance. Regardless of the strategy, it is important to critically assess how plants modified for abiotic stress

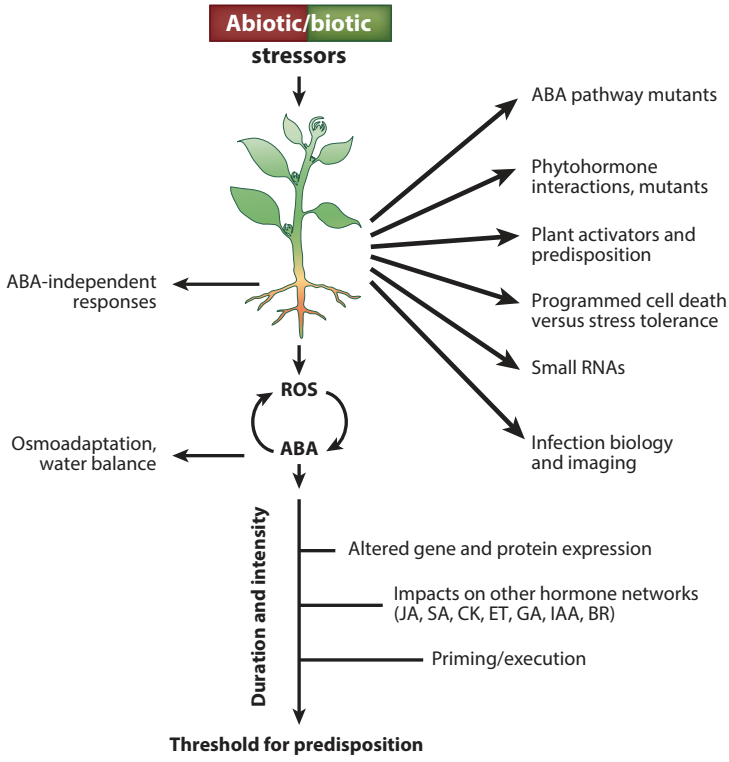


Figure 8

General model and recent areas for mechanistic inquiry into the role of stress-induced abscisic acid (ABA) in predisposition. Abbreviations: BR, brassinosteroid; CK, cytokinin; ET, ethylene; GA, gibberellic acid; IAA, indole-acetic acid; JA, jasmonic acid; ROS, reactive oxygen species; SA, salicylic acid.

tolerance perform when challenged by pathogens and pests. An understanding of the adverse interactions among phytohormone networks in the face of concurrent abiotic and biotic stresses is emerging, and the challenge is to determine how and to what extent the underlying processes can be manipulated to effect positive outcomes.

SUMMARY POINTS

1. Mild, episodic stresses can severely predispose plants to pathogens they would otherwise resist.
2. Molecular and biochemical studies indicate extensive overlap in abiotic and biotic stress responses, with evidence for a universal stress response transcriptome (113). Understanding the degree to which there is fine-tuning to tailor responses to different stresses is an ongoing quest.
3. Strong evidence supports ABA's determinative role in predisposition, but there is also extensive cross talk among all the phytohormones in stress events.

4. A model for the recruitment of ROS and phytohormones to sequentially engage defense responses is emerging (186), but how the sequence is disrupted by predisposing stress events is unclear.
5. Strategies for improving stress tolerance in plants should also consider impacts on disease resistance.

FUTURE ISSUES

1. Comparative studies are needed to assess how stress network dynamics discovered in *Arabidopsis* translate to agricultural pathosystems.
2. Studies that compound multiple stresses are needed to fully characterize the abiotic-biotic stress interactome as it may occur in the field (124, 150) and likely will require a robust systems biology approach involving transcriptomics, proteomics, and metabolomics to identify key regulatory hubs and outputs (40, 158).
3. There is a need for high-throughput methods for phenotyping root-pathogen interactions.
4. There are more than 600 receptor-like kinases in *Arabidopsis* (142). Many appear to be involved in abiotic and biotic stress perception but require functional characterization.
5. Small RNAs, chromatin remodeling, and epigenetics are emerging areas in plant stress biology. How these processes impact disease outcomes is an active area of inquiry.

DISCLOSURE STATEMENT

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Errata

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