Trends in Plant Science

CellPress

Review

The plant disease triangle facing climate change: a molecular perspective

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Variations in climate conditions can dramatically affect plant health and the generation of climate-resilient crops is imperative to food security. In addition to directly affecting plants, it is predicted that more severe climate conditions will also result in greater biotic stresses. Recent studies have identified climate-sensitive molecular pathways that can result in plants being more susceptible to infection under unfavorable conditions. Here, we review how expected changes in climate will impact plant–pathogen interactions, with a focus on mechanisms regulating plant immunity and microbial virulence strategies. We highlight the complex interactions between abiotic and biotic stresses with the goal of identifying components and/or pathways that are promising targets for genetic engineering to enhance adaptation and strengthen resilience in dynamically changing environments.

Highlights

To cause disease in plants, pathogens not only require a susceptible host, but also often require specific environmental conditions suitable for pathogenesis.

Multiple aspects of climate are predicted to change dramatically over the coming decades, which will result in increases in CO_2 and, depending on location, temperature, humidity, salinity, flooding, or drought.

Climate change will result in increases in the prevalence, dispersal, and range of different plant pathogens, while simultaneously affecting the virulence mechanisms of microbial pathogens.

The plant immune system can be compromised by extremes in climatic conditions and crosstalk with abiotic stress signaling pathways.

Several recent reports have unraveled how climatic factors affect plants at the molecular level, raising the possibility of modifying these pathways for climate-smart, pathogen-resistant crops.

The plant disease triangle: mechanistic perspectives under global climate change

In 1960, Russell B. Stevens introduced the concept of the 'plant disease triangle', which posits that three conditions are required for plant disease development: a susceptible host plant, a virulent pathogen, and favorable environmental conditions. This concept is particularly important today since climate change projections indicate that global environmental conditions will undergo significant shifts in the coming decades, including increased frequency and severity of extreme weather events. These changes, such as elevated temperatures, altered precipitation patterns, and increased atmospheric CO_2 and soil salinity levels, will have a significant impact on plant growth and productivity. Field observations have established a correlation between abiotic stressors, similar to those anticipated under projected climate change scenarios, and plant susceptibility to pathogenic infections [1].

Observational research shows that temperature and humidity are the main drivers of the distribution and impacts of plant pathogens, and that climate-associated disease risk will track crop yields [2]. Certain climatic conditions can create microenvironments beneficial to pathogen proliferation. For example, many foliar pathogens benefit from high air humidity levels following precipitation, because high humidity favors aqueous microenvironments in the extracellular spaces of plant tissues, which promotes pathogen virulence [3,4]. Enhanced soil water content is also an important factor for disease outbreaks caused by soil-borne pathogens. Although pathogens can benefit from extreme environmental conditions to infect their hosts because they provide ideal conditions for growth and spread, the molecular mechanisms by which these abiotic factors contribute to the infection process are not well understood.

The influence of environmental factors on plant–pathogen interactions extends beyond the establishment of conditions favorable to the pathogen. Infection outcomes also depend on the plant immune system, and it is well established that microbial pathogens use immune evasion

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mechanisms as a strategy for successful host infection [5]. Recent studies revealed the importance of environmental factors, such as elevated temperatures and humidity levels, in modulating the immune response of the host plant [6,7]. Specifically, these abiotic stresses have been shown to alter phytohormone production and other defense signals mediating responses to pathogens. However, the effects of multiple abiotic stressors on host responses to microbial infection are complex and much remains to be explored.

In this review, we explore how abiotic stresses projected to increase in future climates reshape our understanding of the plant disease triangle concept, with particular emphasis on their impact

Box 1. The plant immune system

Plants detect and defend against pathogens using a two-tiered and mutually potentiated immune system comprising: (i) pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI); and (ii) effector-triggered immunity (ETI) [179,180] (Figure I). PTI confers basal disease resistance to a broad spectrum of pathogens and is induced when plant pattern recognition receptors (PRRs) recognize conserved PAMPs [181]. This, in turn, leads to the production of ROS and calcium influx at the plasma membrane, which activate immune signaling through calcium-dependent protein kinases (CDPKs) and/or mitogen-activated protein kinases (MAPKs). Activation of plasma membrane-associated immune signaling increases the expression of immune-related transcripts and defense-related phytohormone biosynthesis. However, most pathogens have developed mechanisms to suppress PTI and promote virulence, including the secretion of effector proteins, many of which are delivered to the host cytoplasm [4,5,180]). In response, plants have evolved intracellular immune receptors called nucleotide-binding leucine-rich repeat (NLR) proteins, which can perceive pathogen effector proteins and subsequently induce ETI [180]. Mutual potentiation of PTI and ETI leads to various responses, including MAPK activation, Ca^{2+} influx, defence gene upregulation, and synthesis of defence hormones, such as salicylic acid (SA), jasmonic acid (JA) and ethylene (ET) [179,180].

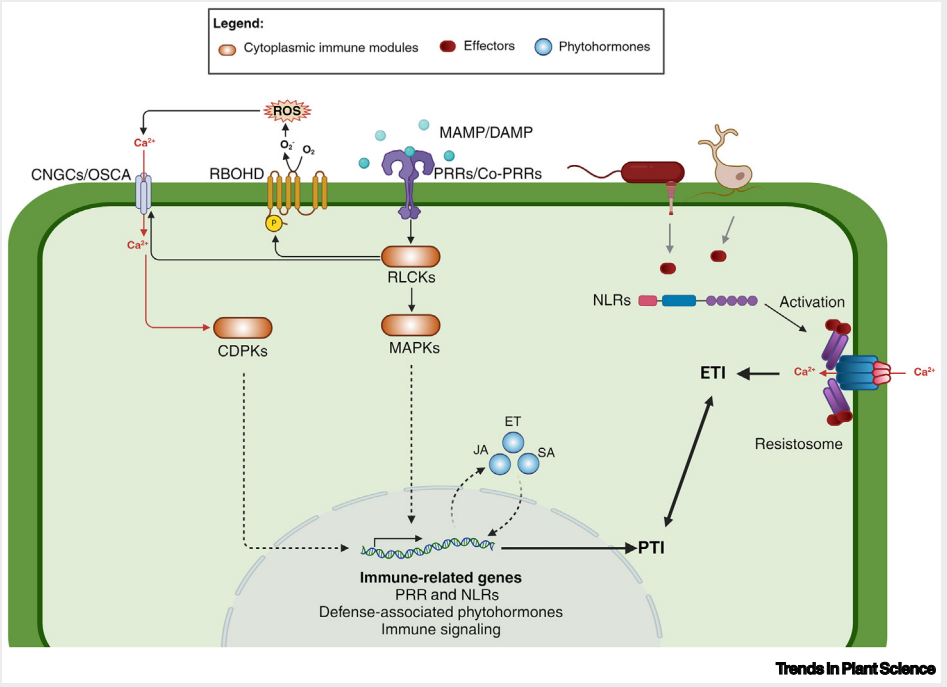

Figure I. Simplified overview of the plant immune system. Figure created with BioRender (biorender.com). Abbreviations: CDPK, calcium-dependent protein kinases; CNGC, cyclic nucleotide-gated channel; DAMP, damage-associated molecular pattern; ET, ethylene; ETI, effector-triggered immunity; JA, jasmonic acid; MAMP, microbe-associated molecular pattern; MAPK, mitogen-activated protein kinases; NLR, nucleotide-binding leucine-rich repeat; OSCA, hyperosmolality-gated calcium-permeable channel; PRR, pattern recognition receptor; PTI, pathogen-associated molecular pattern (PAMP)-triggered immunity; RBOHD, respiratory burst oxidase homolog D; ROS, reactive oxygen species; SA, salicylic acid.

on plant immune systems and microbial virulence strategies. A comprehensive understanding of how changing climatic conditions mechanistically affect the interactions between plant immunity, microbial pathogenesis, and the environment can help design and implement much-needed mitigation and adaptation strategies to reduce plant vulnerabilities to climate change.

Climate change affects plant immune responses

Temperature

Rising temperatures can lead to new or higher incidences of plant disease outbreaks or epidemics due to regional changes in pathogen presence and/or increased plant susceptibility [8,9]. Additionally, shorter/warmer winters can lead to increased survival between growing seasons [10]. Moreover, some pathogens normally constrained by high temperatures have been shown to adapt to survive better in warm conditions [11–13]. Finally, global warming could indirectly impact the development and fitness of vectors (e.g., insects) that transmit plant viruses [14,15]. While understanding how climate change will impact insect and other pathogen vectors is of great interest for food security, this subject has been covered elsewhere [1]. In terms of the plant host, climate change-linked warm temperatures modulate the production and/or effects of various plant defense hormones, as well as other defense-related molecules, such as Ca^{2+} and reactive oxygen species (ROS) [16,17]. A brief overview of the plant immune system is described in Box 1 to facilitate reading of this review. Figure 1 summarizes the climatic factors described in this section and their major reported impacts on plant immune processes.

Mechanisms that can be affected by elevated temperatures range from initial pathogen perception to downstream defense responses. In terms of pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) [18], it has been reported that exposing plants to 42°C for only 45 min decreases immune signaling. However, differences in the length and intensity of an elevated temperature treatment may lead to different immune outcomes, since another study

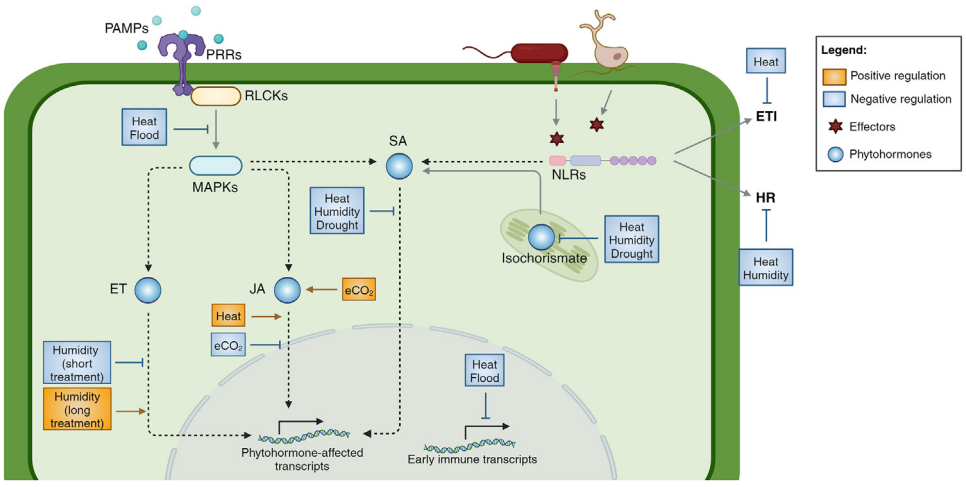

Figure 1. Global impact of climatic factors on the plant immune system. While climatic factors may increase certain aspects of jasmonic acid (JA)-related immune responses, salicylic acid (SA), and ethylene (ET)-mediated immunity are generally negatively affected by climate extremes. In addition to suppressing pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI), high temperatures and high humidity can negatively impact effector-triggered immunity (ETI). Overall, the climate factors expected to change as a result of climate change will negatively impact host immune responses and benefit pathogen proliferation. Figure created with BioRender (biorender.com). Abbreviations: eCO2, elevated CO_2; HR, hypersensitive response; MAPKs, mitogen-activated protein kinases; NLR, nucleotide-binding leucine-rich repeat; PRR, pattern recognition receptor; RLCKs, receptor-like cytoplasmic kinases.

showed that arabidopsis (*Arabidopsis thaliana*) grown at 28°C displayed enhanced responsiveness in terms of PTI-activated mitogen-activated protein kinase (MAPK) and BOTRYTIS-INDUCED KINASE1 (BIK1) pathways compared with plants kept at 23°C [19]. Downstream of pathogen recognition, warm temperatures (28–30°C) can suppress pathogen-induced salicylic acid (SA) biosynthesis and signaling [20]. SA reduction under elevated temperature increased disease susceptibility in arabidopsis and tobacco plants to *Pseudomonas syringae* pv. *tomato* (*Pst*) DC3000 at 28–30°C [6,21,22] and tobacco mosaic virus at 32°C, respectively [23]. Interestingly, an elevated temperature of 30°C did not affect the nuclear localization of the SA receptor NPR1 [22], indicating that not all components of the SA pathway are vulnerable to warm conditions. Indeed, analyses of the SA-regulated transcriptome showed that there are distinct sets of genes that are either affected or unaffected by elevated temperature [22]. The reason for this differential regulation remains elusive, but a recent study demonstrated that temperature suppression of SA biosynthesis at 28–30°C can be attributed to the downregulation of two master transcription factors involved in SA biosynthesis, CALMODULIN-BINDING PROTEIN 60-LIKE G (CBP60g) and SYSTEMIC ACQUIRED RESISTANCE DEFICIENT 1 (SARD1) [6]. Transcription of *CBP60g* and *SARD1* appears to be rate-limiting in disease resistance in arabidopsis, since constitutive *CBP60g* or *SARD1* expression restores not only basal immunity at high temperature, but also other immune phenotypes [6].

Reduced *CBP60g* and *SARD1* expression at high temperatures (28–30°C) is related to the action of thermosensitive GUANYLATE-BINDING PROTEIN-LIKE (GBPL) defense-activated condensates (GDACs). The GBPL3 protein forms condensates through phase separation, which control transcriptional responses to pathogen infection, including *CBP60g* and *SARD1* [6,24]. However, these condensates are greatly reduced at warm temperatures, resulting in loss of recruitment of the general transcriptional machinery to target promoters [6]. Interestingly, not all GDACs are affected by warm temperatures, which could explain why SA-regulated signaling is not entirely abrogated under such conditions. It remains an open question why plants would evolve this warm temperature vulnerability of SA, or whether this phenomenon is a trade-off with some other trait. This could be linked to the thermogenic (heat-producing) quality of SA in certain species [25]. Additionally, because of the intimate physical and functional connections between SA biosynthesis, immunity, and chloroplasts (where SA biosynthesis is initiated) [26,27], it is also possible that differences in photosynthetic strategies (C3 versus C4 versus CAM) could account for the varying sensitivity of the SA pathway in diverse plants. Consistent with this, CAM plants have higher SA levels compared with C3 plants upon infection [28], although any differential effects of temperature between photosynthesis strategies have yet to be explored. Further investigations are needed to understand the role of SA in plant species that naturally exhibit high levels of SA, and whether trends observed in model dicots (e.g., arabidopsis and tomato) are broadly applicable. It is likely that many aspects are conserved because, for example, SA pathways have been reported to be downregulated under warmer temperatures (~35°C) in rice [6,29].

SA signaling is also intricately linked to the function of certain nucleotide-binding leucine-rich repeats (NLRs) in activating effector-triggered immunity (ETI). Intriguingly, temperatures slightly above the optimal growth range have been shown to suppress ETI-associated cell death, known as the hypersensitive response (HR) [19,30]. In some cases, this results in a break of resistance to a bacterial pathogen at 35°C [31], but in others, despite an apparent lack of HR, ETI suppression of bacterial growth is unaffected by elevated temperatures in the 24–35°C range [31–34]. At the same time, nematode resistance conferred by the tomato *Mi-1* gene is compromised at high temperatures (3 h at 35°C) [35], as is virus resistance conferred by the tobacco *N* gene and pepper *TSW* gene at 30°C or higher [36,37]. By contrast, some NLRs, such as Rx1, which confers resistance to potato virus X, display clear HR and disease resistance under elevated temperatures of

30°C or higher [21,34]. Other NLRs, such as Sr15 and Sr21 of durum wheat, exhibit better ETI function at elevated temperature (25°/22°C versus 18°/15°C day/night) against *Puccinia graminis* f. sp. *tritici* Ug99 [32,33]. Interestingly, while Xa7-mediated ETI in rice is stronger at high temperatures (35°/29°C versus 29°/23°C day/night), the SA-responsive pathway in rice is downregulated by heat, suggesting SA-independent ETI resilience [29]. Understanding the variable effects of temperature on HR and ETI requires further analyses. However, NLR proteins, such as GDACs, may be intrinsically sensitive to high temperatures. Indeed, it has been proposed that the accumulation of certain NLR proteins, such as the N protein and SUPPRESSOR OF NPR1-1, CONSTITUTIVE1 (SNC1) is compromised at elevated temperature of 28°C or higher [38,39]. At the same time, this phenomenon could be linked to the variable requirements of NLRs for SA, as well as the fact that SA induces the expression of many NLRs [40], which, as noted earlier, is compromised at high temperatures. An intriguing aspect of ETI under elevated temperature relates to the resilience of SA accumulation in some cases. Indeed, while compatible interactions between arabidopsis and *Pst* result in increased disease severity caused, in part, by reduced SA accumulation under elevated temperature (28°C or higher), SA accumulation is sustained in ETI induced by the *Pst* effector AvrRpt2 [41], but not the *Pst* effector AvrRps4 [6]. How ETI protects the SA pathway under elevated temperatures and leads to different outcomes based on the nature of the NLR-effector pair remains unresolved.

Although SA-dependent immunity is compromised under elevated temperature, another key phytohormone, jasmonic acid (JA), appears to be positively regulated [17,22,42]. JA is involved in defense against necrotrophic pathogens and has been shown to be canonically antagonistic to SA [43,44]. The extent by which increased JA might contribute to the reduction of SA potentiation at elevated temperatures requires further investigation. Interestingly, because JA is required in certain ETI-mediated HR reactions, and functions through the negative SA receptors NPR3/4, it is interesting to speculate that HR could still occur under elevated temperatures in certain immune reactions as a result of increased JA biosynthesis [45]. Remarkably, increased JA biosynthesis under warmer conditions (28°C versus 22°C) can enhance immunity against the hemibiotrophic fungi *Magnaporthe oryzae* in rice [42] and, possibly, against other diseases caused by necrotrophic pathogens [46]. However, SA–JA antagonism is not necessarily conserved in other species, such as rice [47] and poplar [48]; thus, detailed spatiotemporal measurements in such species are still needed. Even in arabidopsis, the interaction between the SA and JA pathways can become synergistic in certain cases. For example, in the absence of the central ETI regulator PAD4, the production of JA augments SA biosynthesis [49]. Given that PAD4 is negatively regulated by high temperature [6,22], one can hypothesize that SA–JA antagonism might be reversed under warmer conditions, potentially to provide robustness to the plant immune network. Considering the relative implication of phytohormone networks in diverse pathosystems, it is critical to take into account both the plant host and the microbial pathogen when examining the effect of elevated temperatures on basal immunity.

Finally, viral infection is often countered by plant RNA silencing (in addition to NLRs), wherein viral double-stranded (ds)RNA is recognized and degraded through the action of DICER-like (DCL) proteins. The resulting small interfering RNA (siRNA) fragments are then bound by Argonaute endoribonucleases, which use them to target homologous single-stranded viral RNA [50]. In turn, viruses have evolved viral suppressors of RNA silencing (VSRs) [51]. Virus infection and symptoms have long been known to be attenuated at high temperature (above 25°C) and exacerbated at low temperatures [52]. Indeed, virus-infected plants can turn from asymptomatic to highly symptomatic when temperatures drop in the autumn [53]. The reasons for this are not always clear, but this phenomenon may be affected in the future by changes in growing season length. In some cases, however, the outcomes of virus infections in plants appear to be due, at least in part, to more efficient RNA silencing. For example, the targeting of viral dsRNA by DCLs appears to be much less efficient

at lower temperatures [54]. At the same time, higher temperatures appear to increase expression of RNA-dependent RNA polymerases, which act to amplify RNA silencing by increasing production of virus-targeting siRNAs, although the effects can differentially affect different viruses [55]. The exact mechanisms responsible for increased RNA silencing at higher temperatures remain to be fully explored (e.g., higher enzymatic activity versus increased expression of RNA-silencing components). At the same time, it remains to be seen whether increased pressure from RNA silencing in a warmer world will result in a concomitant evolution of viral strains with more effective VSRs. Likewise, RNA silencing is known to have important roles in interactions between plants and filamentous pathogens and it will be of interest to determine whether changes in temperature affect this phenomenon.

Humidity

Increased temperatures will drive future global water evaporation, leading to an increase in precipitation and atmospheric humidity levels in certain regions [56]. High relative humidity (HRH; generally considered to be >90% relative humidity) and soil moisture have long been correlated with disease development in plants [57]. Recent studies have started to provide insights into the impacts of humidity levels on plant immune responses. In the aerial parts of the plant, pathogens often gain access to internal tissues through natural openings, such as stomata and hydathodes [58,59]. PTI activation in guard cells is known to lead to stomatal closure, which prevents further pathogen entry [60]. This stomatal closing effect is dependent on SA, and it has been reported that HRH results in reduced stomatal immune responses due to stomatal guard cells being less affected by SA [61]. In agreement with these findings, a recent study reported that HRH results in a generalized reduction in SA signaling, which the authors propose to be caused by the accumulation of 'inactive' forms of NPR1 in the nucleus, thus providing a potential mechanism for the effects of HRH on SA signaling [7]. Prolonged exposure to HRH before microbial perception appears to exacerbate effects on plant immunity [7]. However, plants kept under normal relative humidity before pathogen exposure and HRH incubation are still protected by the immunogenic effects of flg22 pretreatment, suggesting that, once immune responses are mounted, HRH does not affect basal immunity [7]. As such, the length of exposure to HRH, as well as fluctuations in humidity levels, may both be important in affecting plant immunity.

HRH affects the levels of two other immune-related phytohormones: JA and ethylene [7,62]. Humidity levels can affect the response to a methyl-JA (MeJA) treatment. The expression patterns of JA-responsive genes are contradictory under different humidity conditions following MeJA treatment, because some marker genes, such as *MYC2* and *VSP2*, are downregulated under HRH, while *JAZ8* and *PDF1.2* are upregulated [7]. In the rice-*M. oryzae* pathosystem, accumulation of the ethylene precursor, 1-aminocyclopropane-1-carboxylate (ACC), as well as the expression of ethylene-responsive genes, are reduced under HRH compared with 70% RH. In turn, these contribute to increased *M. oryzae* virulence [63]. Given that HRH stimulates ethylene accumulation and signaling in arabidopsis in the absence of pathogens, it is possible that plant responses to humidity levels vary due to the adaptation of the plant to its environment [63].

High humidity not only affects plant basal immunity, but also represses the induction of ETI-induced HR [3,64–67]). However, in many cases, this lack of macroscopically visible HR under HRH does not result in a loss of ETI-mediated suppression of microbial growth [3]. High humidity in combination with high temperature triggers particular immune responses in solanaceous plants, such as activating subsets of NLR genes [68]. In roots of pepper plants, a cytokinin-mediated immune response is mounted to cope with this stress combination, rather than the classical SA/JA pathways found in leaves [69].

Overall, HRH reduces the effectiveness of plant immunity by disrupting the effects of multiple defense-related phytohormones. Although our understanding of the effects of HRH on leaf immunity is increasing, more research is needed to understand its impact on root immunity, because soil moisture benefits many soil-borne pathogens. In particular, it will be of interest to understand how plants perceive changes in humidity and how this information translates to intracellular molecular changes.

Flooding

Future climate changes are predicted to result in elevated rainfall and/or glacier melting, leading to more frequent floods in vulnerable regions. Plant submergence negatively affects gas exchange, leading to reduced oxygen acquisition and eventual hypoxic stress. The extent to which hypoxia affects the plant immune system is poorly understood. A submergence stress was found to trigger an immune response capable of reducing bacterial load in infected arabidopsis via a program dependent on the transcription factor WRKY22 [70]. However, a recent study revealed that a combined treatment of the immunomodulatory peptide flg22 with hypoxia in arabidopsis reduced the amplitude of the PTI transcriptional response compared with flg22 in plants not undergoing hypoxia [71]. These observations raise the question of whether a submergence stress and a hypoxia stress (O_2 <2%) can be considered similar stresses in the context of a combination with a biotic stress. Nevertheless, it has been reported that, under hypoxic conditions, transcript levels of defense-related extensin and SA-related phenylalanine ammonia-lyase (PAL) gene families, as well as PAL activity, are reduced in potato tubers and *Eucalyptus*, respectively, increasing potato susceptibility to *Erwinia* infections [72,73]. Apart from differential gene regulation and enzymatic functions, in soybean and tobacco cells undergoing ETI elicitation by avirulent *Phytophthora*, hypoxia can suppress both the oxidative burst and HR cell death [74]. Potential clues to the impact of flooding on immune pathways could be derived by studying aquatic plants, some of which, such as monocot duckweeds, have lost the central ETI regulator EDS1 during evolution but have gained other molecular innovations [75]. Taken together, it appears that hypoxia suppresses plant immune functions, although some variation may exist between plant species.

Drought

Conflicting hormonal responses were found to affect plant immunity during drought stress. Plants appear to prioritize drought responses over immune activation, which may stem from an antagonistic interaction between the plant immune phytohormone SA and the water-stress phytohormone abscisic acid (ABA) [76]. Drought-stressed plants produce less SA when challenged with a pathogen and display overall downregulation of defense-related transcripts, thereby increasing disease susceptibility [77,78]. The ineffective SA response in drought-stressed infected plants could be explained by the antagonistic effects of ABA [79,80]. Moreover, ABA suppression of SA-mediated immunity appears to operate in an age-dependent manner, with young leaves exhibiting less SA–ABA antagonism compared with older leaves [76]. This suggests that plants prioritize the survival of young leaves when experiencing a combination of drought and biotic stress [76]. Therefore, it is not surprising that drought responses have been suggested to have coevolved with certain immune pathways [81].

The interplay between viruses and drought is complex and not fully understood. For example, the major drought-responsive hormone, ABA, induces the expression of RNA silencing components and virus resistance in arabidopsis [82]. However, it is unclear how widespread this phenomenon might be. At the same time, however, multiple studies have reported that virus infection can increase plant drought tolerance [83] and can even result in the evolution of mutualism between viruses and drought-stressed plants [84].

Until recently, our understanding of plant immunity under drought did not consider the post-stress recovery phase. However, a recent study using fine-scale RNA-sequencing time course analysis of arabidopsis identified an important set of genes involved in both plant immunity and drought recovery, referred to as drought recovery-induced immunity (DRII) [85,86]. Activation of DRII was reported to contribute to disease resistance against *P. syringae* [85,86]. This is further supported by observations that mild drought can increase resistance of avocado plants to *Rosellinia necatrix*, possibly through a process similar to DRII [87]. It has been suggested that plants have adapted to rapidly shift from drought responses to defense to prevent pathogen infection when they are in an immune-compromised state [86]. Alternatively, recovery from drought may result in a downregulation of ABA signaling, the latter being required for susceptibility to *Pst*. Whether these mechanisms have evolved to prevent pathogen induction of water soaking during rehydration in plants experiencing drought and most likely containing high levels of ABA remains to be investigated.

Salinity

Soil salinity is expected to increase as a result of drought and irrigation, which favor the concentration of salts, as well as sea-water floods [88]. Notably, activation of plant immunity by immunogenic molecules is sufficient to trigger salt stress tolerance in several plant systems [89]. Crosstalk between plant immunity and salt stress pathways appears to converge on signaling associated with the loss of cell wall integrity. Indeed, salt-induced cell wall damage activates the damage-associated molecular pattern (DAMP) pathway through Pep signaling [89]. Increased salt tolerance in PTI-activated plants may result from commonly shared signaling components activated by both DAMP-triggered immunity (DTI) and PTI. It is also possible that PTI activation enhances salt tolerance by increasing cell wall reinforcement. However, there is conflicting evidence regarding whether this involves callose. Mutants lacking the defense-related callose synthase PMR4 behave similar to wild-type plants under salt stress [89], whereas mutants of the callose synthase gene *CALS1* appeared to be less tolerant of salt stress [90].

A peculiar observation is the apparent cooperation between SA and ABA under salt stress, wherein both phytohormones are required for salinity stress resilience, but are normally antagonistic during pathogen infection [79,80,91]. This could have negative consequences in salt-stressed infected plants. Given that many effectors target either or both SA and ABA networks, this could result in plants being more sensitive to salt stress, further diminishing plant health [5]. This would be consistent with the observation that arabidopsis is more resistant to salt stress when infected with nonpathogenic, but not virulent, *P. syringae* strains [89], because the former induces SA production, while the latter disrupts SA signaling. Understanding how pathogenic infections impact plant tolerance to high salinity by affecting immune-related processes will be of interest to develop disease-resistant and salt-tolerant crops.

Carbon dioxide

Plant responses to elevated CO_2 (eCO_2) are highly variable, depending on the plant species, ecotype, and cultivar, which are discussed in greater detail in a recent review [92]. Notable examples include *Pst* infections, which display contrasting responses between their natural host, tomato (enhanced resistance under eCO_2) [93,94], and the experimental model plant, arabidopsis (increased susceptibility under eCO_2) [95,96]. Intriguingly, arabidopsis constitutively synthesizes SA under eCO_2 conditions [97]. Furthermore, arabidopsis (which uses C3 photosynthesis) displays improved growth under eCO_2 despite elevated SA levels, which also contradicts the expectation of a growth-to-defense trade-off in plants with constitutive SA accumulation [98]. Since SA can induce the expression of aquaporins [99], some of which are involved in increasing CO_2 entry through stomata, and have also been associated with increased defense [100,101], it is

interesting to speculate that a SA–CO_2-defense network may regulate defense by as-yet undefined mechanisms. These observations provide a paradox to our understandings of the growth-to-defense trade-off mediated by SA in plant immunity and present a potential research avenue to untangle this trade-off.

While stress-induced JA biosynthesis is negatively affected at eCO_2 [102,103], contrasting observations have been reported concerning disease resistance toward necrotrophic pathogens. In most cases, reduced JA responses at eCO_2 have been associated with greater susceptibility to *Botrytis cinerea* and *Fusarium* sp. [104–106]. However, increased resistance was observed in arabidopsis upon infection with *B. cinerea* at extremely high CO_2 concentrations (3000 ppm) [97]. Whether extreme CO_2 levels act as a distinct stress signature triggering JA-related defenses remains to be investigated. The effects on JA-related defenses may be indirectly relevant to virus infections. JA is important for defense against insects and several viruses are known to manipulate this, and other, pathways to favor insect feeding and subsequent virus transmission [107]. Studies of the direct effects of eCO_2 suggest that it often results in decreased negative impacts of virus infection [92]. However, the ultimate effects of increased eCO_2 on viruses will require integrated studies on the effects of the latter on plant growth, metabolism, and vector behavior and biology [108].

Effects of climate change on microbial virulence

Beyond the effects of global climate change on host plant immunity, changing abiotic factors can influence pathogen fitness outside the host by allowing increased growth, survival, and transmission [1]. Likewise, environmental factors can influence pathogen virulence by affecting the establishment of an ideal niche for growth or by promoting the production of virulence-related molecules. In this section, we highlight our current conceptual understanding of these processes (Figure 2) and provide forward-looking outlooks for potential future investigations.

Temperature

Temperature can dramatically impact the effectiveness of initial pathogen invasion and colonization. For example, certain bacterial pathogens, such as *Pectobacterium*, produce higher levels of cell wall-degrading enzymes and quorum-sensing signals at higher temperatures (>30°C), which correlate with increased virulence [109]. Another example is that of the epiphytic bacterium *P. syringae*, which becomes pathogenic upon entry, via openings such as stomata, to the plant apoplast (intercellular spaces). In this case, *P. syringae* produces the virulence factor coronatine, which promotes stomatal opening, over-riding the plant immune response that induces stomatal closure upon pathogen detection [58]. It has been shown that coronatine production by *P. syringae* pv. *glycinea* in soybean plants is enhanced at lower temperature (18°C versus 28°C), potentially via the CorS sensor [110,111]. By contrast, *Pst*-induced stomatal closure in arabidopsis is delayed at higher temperatures (28°C versus 22°C), suggesting that these effects are plant species and/or bacterial strain specific [112]. Overall, the temperature dependence of plant pathogen enzymatic activities and toxin production has implications not only for the efficiency of pathogen invasion, but potentially even the extent of plant host damage induced during infection.

To facilitate successful host infection, pathogens have evolved host immune evasion mechanisms, which are mediated by their effectors [5]. Given that protein translation and activity are regulated by changing temperatures, it is unsurprising that effector production is also temperature dependent. For example, several plant pathogenic bacteria use secretion systems that are affected by temperature to deliver virulence effectors into plant cells. Likewise, the *Agrobacterium* Type IV Secretion System does not function efficiently at elevated temperatures

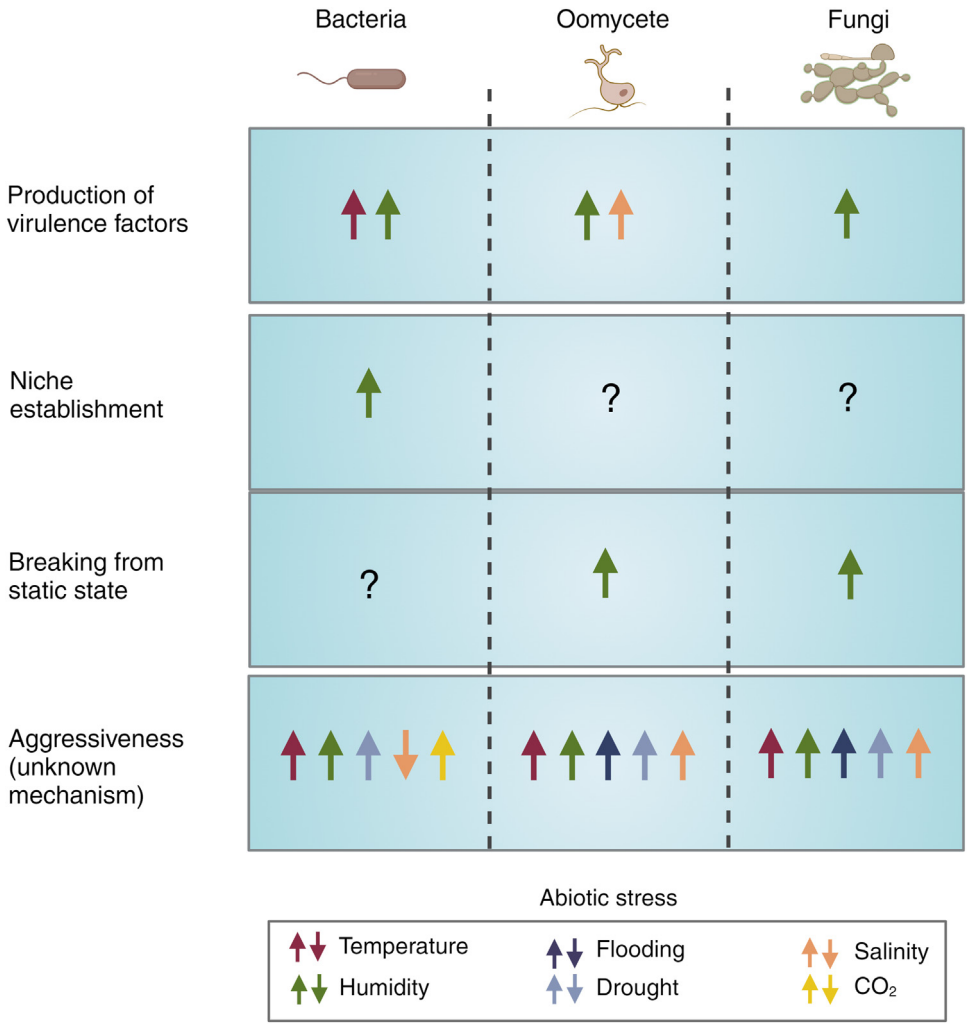

Figure 2. Known impacts of climate change on microbial virulence. The climatic factors discussed here can have a variety of impacts on pathogen proliferation. Here, they are described as affecting different aspects of microbial virulence, such as: (i) production of virulence factors; (ii) niche establishment; (iii) breaking from a static state; or (iv) unknown factors associated with aggressiveness. We consider the production of virulence factors as the transcriptional increase in effector gene expression, effector translocation/secretion, and/or the biosynthesis of toxins involved in virulence. Niche establishment is defined as the ability of pathogens to induce an ideal microenvironment in the apoplast, commonly referred to as an aqueous and/or nutrient-rich apoplastic space. 'Breaking from a static state' refers to a change in metabolism from stasis to active growth, spore germination, or appressorium formation, depending on the nature of the pathogen. 'Aggressiveness' is based on known cases of increased pathogen aggressivity, but for which mechanisms are unknown. Abiotic factors affecting these factors are represented as different colored arrows for each abiotic factor, up and down arrows representing an increase or decrease in the microbial virulence aspect, respectively. Figure created with BioRender (biorender.com).

of 32°C or higher [113,114]. Interestingly, the *P. syringae* Type III Secretion System (T3SS) exhibits diverging properties in relation to temperature. T3SS-related genes are upregulated *in vitro* by colder temperatures at 18°C [115], but translocation of effector proteins into arabidopsis cells is increased below (16°C) and above (30°C) ambient temperature [22,116].

In addition to bacterial pathogens, filamentous pathogens (fungi and oomycetes) also cause major plant diseases and threaten various ecosystems. Notably, pathogenic fungi and oomycetes

appear to have wider climatic tolerances globally compared with their beneficial counterparts [117,118]. Temperature effects on filamentous pathogens can occur at various stages of the pathogen life cycle [119,120]. Given that protein folding is affected by temperature, a central mechanism relies on well-conserved temperature-regulated molecular chaperones. In particular, HSP90 is crucial for asexual and sexual spore production of *Fusarium graminearum* [121], suggesting an intricate interplay between fungal heat shock responses and development. It is possible that these mechanisms will be key for projected increases in fungal pathogen abundance [1,122] and/or virulence [123]. However, spore production in filamentous pathogens is predicted to decrease under climate warming [124,125]. Taken together, these findings underscore the potential effects on the sexual and asexual life cycles of fungi and oomycetes, which could have implications on filamentous pathogen distribution, persistence, and virulence.

Successful host invasion of filamentous pathogens requires effective penetration, mediated by appressoria formation. Appressoria formation is more effective at 22–28°C compared with lower temperatures, although it is abrogated above 30°C [126]. This is interesting since the appressoria of several fungal species require melanin to generate pressure for host invasion [127]. This is congruent with the theory of thermal melanism, which posits that pigmentation is an ancient adaptation for absorbing thermal energy, consistent with variation in yeast colorations [128]. As with pathogenic bacteria, filamentous pathogens also secrete pathogenesis-promoting enzymes, which can be influenced by temperature in the 25–37°C range [129]. Interestingly, peptidases of thermotolerant and thermosensitive fungi diverge in their proportions of hydrophobic, charged, and polar amino acids [130], which could serve as an initial screen to identify strains that might become greater threats due to global warming. Apart from analyzing molecular changes, it may be worthwhile examining the phase transition dynamics of thermally dimorphic fungi, since conversion to the unicellular yeast form has implications on virulence (Box 2).

Humidity

The relationship between leaf wetness and plant disease has been studied for over a century and monitoring leaf wetness duration is crucial for plant disease management [57]). Microbes living on the leaf surface (called epiphytic microbes) benefit from leaf wetness to penetrate the leaf interior [131]. Epiphytic bacteria exhibit increased motility (swimming and twitching) when water is

Box 2. Microbial thermosensors

It is unclear whether plant bacterial pathogens have thermosensors *per se*, and whether such players are involved in increasing their virulence. However, it has been reported that, in *Pseudomonas aeruginosa*, the thermosensory diguanylate cyclase TdcA modulates temperature-dependent motility, biofilm development, and virulence through the perception of the second messenger c-di-GMP [182]. Given how conserved TdcA homologs and c-di-GMP are from gamma- to beta-proteobacteria, it is likely that such systems are involved in temperature-dependent modulation of virulence in plant bacterial pathogens.

Beyond prokaryotes, what governs temperature-mediated changes in filamentous pathogen virulence? It has been reported that the fungal wheat pathogen *Zymoseptoria tritici* switches from its yeast-like blastospore phase to hyphae or chlamydospores in response to heat stress, and that this is controlled by the transcription factor ZtMsr1 and the protein phosphatase ZtYvh1 [183]. In certain animal pathogenic fungi, Hsp90 governs thermoregulated growth by modulating cell cycle regulators (such as the cyclin-dependent kinase Pho85 and the cyclin Pcl1) [184]. Finally, although *Saccharomyces cerevisiae* is nonpathogenic, studies have shown that key transcription factors (e.g., HSF1) and kinases may have a role in cell temperature perception or heat-induced chromatin remodeling [185,186]. Collectively, these molecular players are promising candidates for further investigation in plant pathogenic fungi [187,188].

Upstream of signal transduction and transcriptional reprogramming are thermosensing mechanisms. The opportunistic human pathogen *Aspergillus nidulans* uses thermosensing via the heterohistamine kinase TcsB and photochrome FphA [189], similar to plant phytochrome B-controlled thermosensing [190,191]. Fungal thermosensors crosstalk with thermosensitive MAPKs, which can be linked with the surface sensor protein Sho1 for fungal recognition of nearby host surfaces [192]. These pathways could be of interest for mechanistic analyses and/or for customized immune targeting.

abundant on the leaf surface [57]. Increased flagellar motility under these conditions enhances the opportunity for bacteria and fungi to invade interior tissues via stomata [132].

Endophytic foliar pathogens benefit from HRH following precipitation to create an aqueous environment in the apoplast, known as water-soaked lesions when macroscopically visible [3]. Both fungal and bacterial pathogens have evolved the ability to induce these lesions, suggesting convergent evolution of pathogenicity strategies. Current evidence suggests a conserved mechanism between *Pseudomonas* and *Xanthomonas* species in inducing water-soaked lesions by triggering host ABA responses, which result in stomatal closure, leading to a decrease in evapotranspiration and accumulation of water in the intercellular space [133–136]. Further supporting the role of water in increasing bacterial virulence, artificial flooding of the apoplastic space or genetic perturbations leading to spontaneous water-soaked lesions in plants was shown to benefit pathogen proliferation [3,7]. As for root pathogens, soil moisture has disparate effects on virulence. For example, high soil moisture enhances the virulence of the soil-borne bacterial pathogen *Ralstonia solanacearum* in tomato [137], while it reduces the virulence of *Streptomyces* sp. and the appearance of common scab of potato [138].

Although the induction of water-soaking is a critical component in bacterial pathogenesis, its importance in fungal disease progression has received less attention. Multiple fungal and oomycete pathogens, such as *Blumeria graminis*, *Uncinula necator*, *Colletotrichum* sp., *Alternaria alternata*, *Cercospora* sp., and *B. cinerea* also cause reductions in host stomatal aperture and induce water-soaking symptoms early during the infection process [139]. Some of these fungal and oomycete species have been shown to either produce ABA or to induce ABA signaling in their host [140]. Production of ABA by *B. cinerea* has been suggested to be key for its virulence [141], potentially by inducing an aqueous apoplast under HRH. Beyond its role in microenvironment establishment, HRH and leaf wetness have been reported to act as cues for fungal pathogens to break from dormancy and initiate disease development in their host plants [142]. Indeed, HRH has been shown to be essential for conidial germination, appressoria formation, and expression of virulence traits in several important fungal pathogens, such as *M. oryzae*, *Gibberella zeae*, *Sclerotinia*, *Phytophthora*, and certain *Fusarium* species [9,63,143–146]. At the same time, HRH can cause abnormalities in the appressorial formation process of *B. graminis*, thus potentially reducing its virulence [147].

In the case of protist pathogens from the *Plasmodiophora* genus, which cause clubroot disease, high soil moisture has been found to increase spore germination [148]. These examples highlight the need for further research to understand how multiple aerial and soil-borne pathogens from multiple kingdoms of life respond to HRH, particularly in regions where HRH is predicted to increase in the future.

Flooding

As previously mentioned, submergence induces hypoxia in plants. Interestingly, various microbial pathogens also induce hypoxia-like responses during infection [149,150]. It is tempting to speculate that pathogens that induce stomatal closure and/or water-soaking may directly trigger local hypoxia responses. As such, we refer to hypoxia herein as being external (caused by flooding or waterlogging of the roots) or internal (i.e., caused by the activities of microbial effectors and/or toxins).

External hypoxia can affect the activities of virulence factors in pathogenic microbes. For example, *Pectobacterium* spp., *Dickeya* spp., and *Erwinia carotovora* pectate lyases, which are key enzymes involved in host cell wall degradation, exhibit higher expression and activity under hypoxia

compared with aerobic conditions [73,151]. Hypoxia perception in *Dickeya solani* also increases expression of virulence genes required for successful disease development [73,152]. Bacterial toxins and effectors may also trigger internal hypoxia through the reduction of gas exchange via manipulation of stomatal aperture and/or reduction of photosynthetic rates [27,134,135,153], with the two not being mutually exclusive. Since internal hypoxia may be a part of a virulence strategy, it will be interesting to evaluate how external hypoxia might benefit pathogens in a similar fashion. While reducing photosynthetic rates can benefit some bacterial pathogen proliferation, whether this is solely related to hypoxia stress is unknown.

Fungal and oomycete root pathogens also benefit from hypoxia responses to infect their hosts. Reduced oxygen exchange at the root level causes growth arrest and damage, which may render this structure more susceptible to pathogen colonization. Indeed, root hypoxia has been associated with increased disease severity caused by multiple fungal pathogens [74,154]. Oxygen consumption by fungal pathogens during colonization has been reported to be a potential factor involved in the reduction of host oxygen levels, leading to internal hypoxia [155]. Studies with *M. oryzae* in rice provided evidence that both the host and pathogen experience hypoxia stress during colonization, and hypoxia-induced genes in *M. oryzae* have been shown to contribute to its pathogenicity, further suggesting pathogen adaptation to internal hypoxia [156,157]. By contrast, localized hypoxia affects mycelial growth of *Collybia fusipes* and *Heterobasidion annosum*, as well as mycotoxin production in *F. graminearum* and *Fusarium porotrichioides*, resulting in reduced virulence [156,158].

Root hypoxia also benefits *Plasmodiophora brassicae*, because it contributes to clubroot disease development in arabidopsis [159]. More investigations are required to better understand whether microbially induced hypoxia is a part of a broader conserved infection mechanism or simply evidence of coevolution in environments with reduced oxygen availability.

Drought

The mechanisms by which drought stress affects microbial virulence strategies are not well understood. Nevertheless, there are reports suggesting that drought can increase disease severity in several pathosystems, including *Xanthomonas oryzae*–rice, *Xyella fastidiosa*–grapevine, *Streptomyces* spp.–potato, *Pseudomonas syringae*–arabidopsis, and *M. oryzae*–rice [78,138,160–162]. Drought stress leads to the rapid biosynthesis of ABA in plants [163], and two primary modes of action have been proposed for how ABA increases microbial virulence. First, ABA has been shown to suppress plant immunity [164] and, second, it has been implicated in the induction of water-soaking lesions by some pathogens [133–135]. However, because water soaking requires high atmospheric humidity and soil hydration, it is unlikely to be the sole factor responsible for higher pathogen populations in these pathosystems. It is also unlikely that increased microbial growth results solely from a reduction in plant immune performance, because other factors significantly contribute to virulence in the absence of an effective plant immune system [3].

Salinity

Similar to drought, high soil salinity impacts various aspects of plant–pathogen interactions. Indeed, *Pst*, as well as *Alternaria brassicicola* and *B. cinerea*, displayed increased aggressivity in arabidopsis plants that were salt stressed before an infection [165]. However, classical immune responses were not affected by salt stress in arabidopsis, suggesting that the increase in pathogen proliferation is not caused by reduced immunocompetency [165]. The increased pathogenicity of *B. cinerea* was associated with elevated ABA levels induced by salt stress. However, whether salt stress favors pathogenic microenvironments remains unknown. In the case of the oomycete pathogen *Phytophthora* sp. involved in tomato root rot, salt stress increased virulence

compared with unstressed plants [166]. Similar observations were made in the *Phytophthora ramorum–Rhododendron* pathosystem, where salt stress increased virulence and disease development [167]. It would be of interest to examine whether salinity-induced osmotic and/or ion toxicity stresses promote host vulnerability independent of canonical immune signaling pathways, versus whether the effects largely occur on the induction of increased virulence in the pathogen. Such research is required to better understand the basis of salt stress-induced susceptibility to infection to design crops that are both tolerant to salt and resistant to biotic stresses.

Carbon dioxide

Finally, CO_2 levels are known to affect stomatal development and physiology [92,168,169]. Studies have shown that eCO_2 can decrease leaf stomatal density, which in turn reduces potential pathogen entry points. Interestingly, recent reports suggest that eCO_2 reduces the ability of *Pst* to reopen closed stomata compared with ambient or low CO_2 levels [95,170]. However, bacterial population in plants inoculated with *Pst* at eCO_2 displayed enhanced susceptibility toward infection [95]. One possible explanation is that, once in the apoplast, water soaking is triggered more swiftly by *Pst*, because water condensation should occur more rapidly due to the predisposition to stomatal closure. However, the mechanisms by which CO_2 influences microbial virulence require further research for a comprehensive understanding of this phenomenon.

Concluding remarks

Changing climate conditions impact plant disease resistance and microbial pathogenesis in various ways. Dynamically changing abiotic factors can modulate host invasion and/or colonization ability, as well as the production of effectors and other virulence factors. These have consequences for the degree of damage that these pathogens can inflict on plants and the extent to which they can evade plant immune responses. Direct changes mediated by temperature and other environmental parameters are mostly due to their effects on pathogen enzymatic activity at the molecular level and overall metabolism and growth. At the same time, these environmental factors can affect the plant by impinging on numerous molecular nodes, from pathogen perception to cellular signaling, and from gene regulation to downstream defense outputs. Understanding these climate-mediated effects on plant immunity and pathogen virulence underpin successful predictions of the impact of climate change on plant disease severity and incidence.

With a greater understanding of the molecular pathways and proteins affected by climatic changes comes the potential to engineer climate-resilient plant immune systems. For example, mutation of certain amino acids in heat-sensitive NLR proteins, such as SNC1 and N, can result in proteins that are functional at higher temperatures [38]. Likewise, several components of the RNA silencing machinery are known to show altered expression levels at lower temperatures. As such, it may be possible to engineer these components for greater functionality. In addition, plants have adapted to a wide range of climatic conditions and part of this process has involved the evolution of immune components adapted to different environments. Indeed, it will be of interest to determine whether certain components known to be sensitive to, for example, high temperature in one plant might have more heat-resistant homologs in plants adapted to such conditions. Indeed, GDAC proteins responsible for inducing SA production in arabidopsis are heat sensitive, whereas other members of this protein family are not [6]. This observation suggests that GDAC proteins differ in thermosensitivity between species, representing a potential adaptive strategy for plants normally growing in hot environments. Understanding whether changes in thermosensitivity impact the thermotolerance–biotic stress trade-off will be of great interest to the research community [171].

So far, our understanding of the mechanistic impact of climate warming on plant immunity and pathogen virulence has largely relied on disparate experimental designs, some using constant

Outstanding questions

Do plant pathogens have sensors for abiotic stresses other than temperature and, if so, are they involved in virulence?

How do climate extremes affect microbial virulence at the transcriptional and metabolic levels?

Can we engineer plants to be resistant to both biotic and abiotic stresses by creatively bypassing known trade-offs?

How have plants, thriving in harsh environments, evolved to face both abiotic and biotic stresses and can this information be used to engineer climate-resilient crops?

How will climate change impact the host holobiont and the advantages provided by beneficial microbes interacting with them?

How do plants, adapted to extreme climate conditions, deal with biotic stresses, and can we translate this knowledge to nonadapted crops?

environmental factor averages while others use fluctuating conditions [9,172]. This is an important consideration since fluctuating temperatures can have significantly different impacts compared with constant temperatures, even if the resulting daily average temperature is the same, as has been shown with *P. infestans* and *Puccinia striiformis* [173,174]. Like all organisms, there is a certain temperature breaking point at which plant pathogens no longer thrive. For example, soybean rust can be effectively abolished with a brief heat stress treatment at 37°C [175]. In the future, it will be important that both laboratory and field studies model these interactions to accurately reflect the anticipated real-world conditions of climate change depending on the geographic area. The timing of the environmental changes is also crucial, since varying effects have been observed when warming conditions have been applied before, during, and/or after plant pathogen infection [172,176]. Investigating the effect of the environmental stressors described earlier beyond pathogenesis in the context of the host and its microbiome (i.e., the holobiont) will be essential for modeling plant health in the coming decades. Furthermore, changes in temperature and rainfall patterns may negatively affect soil health due to increased nutrient runoff, leaching, and erosion. This makes it paramount to fundamentally understand how soil health will affect plant immunity and microbial virulence.

While this review has mainly focused on the negative impacts of climate change on plant immunity and positive impacts on microbial virulence, certain combined stresses can lead to neutral or even beneficial gains for the host plant [177]. Indeed, plants in the field are likely to be subjected to combinations of stresses, and recent efforts have sought to integrate and synthesize the literature on the effects of combinatorial stresses on plant phenomic and molecular responses [178] in

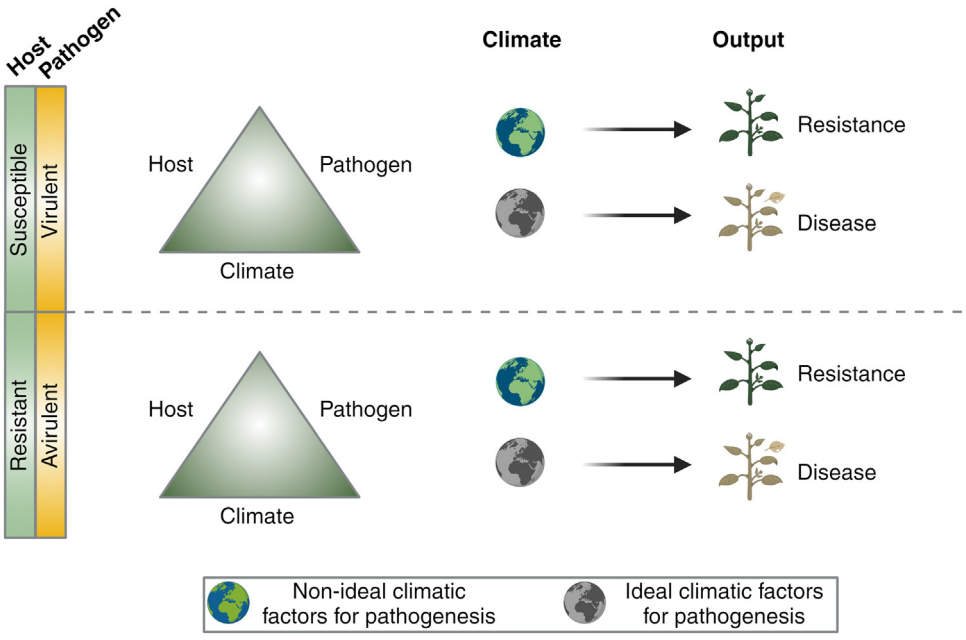

Figure 3. Continuum of the plant disease triangle as a function of climatic factors. The plant disease triangle has long been considered as a model in which a susceptible host, virulent pathogen, and ideal climatic conditions for pathogenesis are required for disease development. Indeed, when climatic factors are not favorable for pathogenesis, effective resistance is often observed against a large spectrum of microbial pathogens. It is also often assumed that a resistant plant facing an avirulent pathogen will lead to resistance to disease. However, such a 'defense-forward' triangle can be broken by climatic factors, such as high temperature and high humidity, allowing disease in plants that are normally genetically resistant to a particular pathogen/pathogen strain. As such, the paradigm of genetically encoded resistance and avirulence is not rigid, but rather plastic, and will be heavily challenged under future climates.

a comprehensive database (SCIPDb; http://www.nipgr.ac.in/scipdb.php). Many examples covered here are from the field of arabidopsis research because of our understanding of the molecular biology of this model plant. However, it is notable that certain plants naturally accumulate high levels of SA without inducing autoimmune phenotypes, which often occurs in arabidopsis. Understanding how plants can manage constitutive immune system activation without a growth-to-defense trade-off may be crucial to developing climate-resilient and pathogen-resistant plants. In addition, while SA and JA exhibit antagonistic effects in arabidopsis, both phytohormones increase during certain pathogen infection in rice. With respect to humidity and flooding, it will be of interest to understand whether plants adapted to such environments have evolved strategies to prevent pathogen-induced water-soaked lesions, because these environmental conditions are favorable to the development of this conserved disease-associated phenomenon. The examples discussed earlier underline the need to study the molecular vulnerabilities and/or resilient pathways in multiple crops, as well as in plants adapted to extreme environments to create a better portrait of the plant disease triangle facing climate change (Figure 3) (see also Outstanding questions).

Acknowledgments

Research in the Moffett laboratory is supported by funding from the Natural Sciences and Engineering Research Council (NSERC) of Canada, from the Fonds de Recherche Québécois - Nature et Technologies, from the Centre SÈVE; C.R-L. is supported by an NSERC doctoral fellowship. C.D.M.C. is supported by research funding from the NSERC and Wilfrid Laurier University. C.A.M.R. is supported by the Ontario Graduate Scholarship and NSERC Canada Graduate Scholarship. The Castroverde Lab at Wilfrid Laurier University is located on the shared traditional territory of the Neutral, Anishinaabe, and Haudenosaunee peoples.

Declaration of interests

None declared by authors.

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