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Review

Plant–Pathogen Warfare under Changing Climate Conditions

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Global environmental changes caused by natural and human activities have accelerated in the past 200 years. The increase in greenhouse gases is predicted to continue to raise global temperature and change water availability in the 21st century. In this Review, we explore the profound effect the environment has on plant diseases — a susceptible host will not be infected by a virulent pathogen if the environmental conditions are not conducive for disease. The change in CO_2 concentrations, temperature, and water availability can have positive, neutral, or negative effects on disease development, as each disease may respond differently to these variations. However, the concept of disease optima could potentially apply to all pathosystems. Plant resistance pathways, including pattern-triggered immunity to effector-triggered immunity, RNA interference, and defense hormone networks, are all affected by environmental factors. On the pathogen side, virulence mechanisms, such as the production of toxins and virulence proteins, as well as pathogen reproduction and survival are influenced by temperature and humidity. For practical reasons, most laboratory investigations into plant–pathogen interactions at the molecular level focus on well-established pathosystems and use a few static environmental conditions that capture only a fraction of the dynamic plant–pathogen–environment interactions that occur in nature. There is great need for future research to increasingly use dynamic environmental conditions in order to fully understand the multidimensional nature of plant–pathogen interactions and produce disease-resistant crop plants that are resilient to climate change.

Introduction

Plant diseases can have devastating economic, social and/or ecological consequences on a global scale. Some of the most infamous plant diseases, for example, potato late blight in Ireland in the 1840s [1] and chestnut blight in the United States in the early 1900s [2], resulted in massive human death and migration and/or drastic changes to the landscape. Not only do many plant diseases persist for centuries, but also new ones continue to emerge worldwide. Estimates of direct production losses for the major agricultural crops by biotic stress have been projected to be around 20% to 40% [3,4]. Together with associated indirect losses in crop quality and marketability, plant diseases are widely considered to be one of the most formidable obstacles to achieving global food security in the face of the rising human population in the 21st century. For plant scientists, a global challenge is how to speed up the understanding of the molecular, epidemiological and ecological bases of plant diseases and develop truly effective and long-lasting solutions for preventing, reducing, or managing some of the most devastating plant diseases facing modern agriculture today and in the future.

Not every disease is equal. Some plant pathogens have a more devastating socioeconomic effect than others, partly because of the host crop species they infect. We list some of the most important plant diseases in Table 1. Pathogens that commonly infect plants are diverse, ranging from intracellular viruses and bacteria, to those that live extracellularly, including other bacteria, fungi, oomycetes, and nematodes. Depending on how pathogens acquire their nutrients, they can be classified as biotrophs or necrotrophs. Biotrophs can only obtain nutrients from living host cells, while necrotrophs usually kill host cells in order to release nutrients. Necrotrophs are also often able to live as saprophytes. In nature, pathogen nutrient acquisition spans a continuum from biotrophy to necrotrophy, with many plant pathogens being hemibiotrophs, showing an initial biotrophic phase before eventually killing the host.

Pathogens and plants do not interact in isolation. The famous ‘disease triangle’ concept in plant pathology highlights the interaction of both pathogens and plants with the environment. For disease to occur, a susceptible plant host, a virulent pathogen, and the proper environmental conditions are required, as lack of favorable conditions for any of these three factors results in a failure for disease to develop [5]. The effect of environmental variables (e.g., high temperature) on pathogens and plants can have favorable, neutral or negative outcomes on plant disease development. Both pathogens and plants have an optimal environmental condition for their growth and reproduction, with an optimal environmental condition that is best for disease outbreak. The further the environmental conditions deviate from this ‘disease optimum’, the fewer disease symptoms will occur on the plant (Figure 1).

Due to genetic, environmental, and cultural reasons, not all cultivated crops have the same importance in human nutrition. Ten crops account for 58% of the total global cultivated area (Table 2). With respect to food security, only four crops

Table 1. Examples of the most destructive diseases and pathogens for crops with the highest area of production.

Crop	Most destructive diseases and pathogens
Banana and plantain	Banana bunchy top virus (BBTV), black sigatoka by *Mycosphaerella fijiensis*, and Panama disease by *Fusarium oxysporum* f. sp. *cubense* [139–141]
Barley	Fusarium head blight by *Fusarium graminearum*, powdery mildew by *Blumeria graminis* f. sp. *hordei*, and stem rust by *Puccinia graminis* f. sp. *hordei* [142]
Cassava	African cassava mosaic virus (ACMVD), bacterial blight by *Xanthomonas axonopodis* pv. *manihotis*, and cassava brown streak virus (CBSV) [143]
Cotton	Bacterial blight by *Xanthomonas citri* pv. *malvacearum*, Fusarium wilt by *Fusarium oxysporum* f. sp. *vasinfectum*, and Verticillium wilt by *Verticillium dahliae* [144–146]
Maize	Aspergillus ear rot by *Aspergillus flavus*, Giberella stalk and ear rot by *Fusarium graminearum*, and grey leaf spot by *Cercospora zeae-maydis* [147–149]
Palm fruit	Basal stem rot by *Ganoderma boninense*, and bud rot by *Phytophthora palmivora* [150,151]
Peanut	Groundnut rosette disease [a combination of groundnut rosette virus (GNV), its satellite RNA, and groundnut rosette assistor virus (GRAV)] [152]
Potato	Brown rot of potato by *Ralstonia solanacearum*, and late blight by *Phytophthora infestans* [1,153]
Rapeseed and mustard	Phoma stem canker by *Leptosphaeria maculans*, and Sclerotinia stem rot by *Sclerotinia sclerotiorum* [154,155]
Rice	Rice blast by *Magnaporthe oryzae*, rice bacterial blight by *Xanthomonas oryzae* pv. *oryzae*, and sheath blight by *Rhizoctonia solani* [156–158]
Sorghum and millet	Anthracnose by *Colletotrichum sublineolum*, and Turcicum leaf blight by *Exserohilum turcicum* [159]
Soybean	Soybean cyst nematode disease *by Heterodera glycines*, and Asian soybean rust by *Phakopsora pachyrhizi* [160]
Sugar beet	Cercospora leaf spot by *Cercospora beticola*, and rhizomania by beet necrotic yellow vein virus (BNYVV) [161,162]
Sugarcane	Ratoon stunting by *Leifsonia xyli* subsp. *xyli*, and red rot by *Colletotrichum falcatum* [163,164]
Sweet potato	Sweet potato virus disease [SPVD; a combination of sweet potato feathery mottle virus (SPFMV) and sweet potato chlorotic stunt virus (SPCSV)] [165]
Tomato	Late blight by *Phytophthora infestans*, and tomato yellow leaf curl virus (TYLCV) [1,166]
Wheat	Fusarium head blight by *Fusarium graminearum*, stem rust by *Puccinia graminis*, and wheat yellow rust by *P. striiformis* [167–169]
Yam	Anthracnose by *Colletotrichum gloeosporioides*, and yam mosaic virus (YMV) [170]

contribute up to 50% of the daily worldwide human calorie uptake (Table 2). In this Review, we will highlight the effect of the environment on plant diseases, whenever possible, focusing on the crops with the highest worldwide production, as these crops are fundamental for our global food security. However, we will also discuss studies that elucidated the molecular mechanisms controlling plant resistance and pathogen virulence, even though these mechanistic studies often use model plant–pathogen systems that may not involve major crops.

Environmental Factors Affecting Plant Disease

Many environmental conditions affect plant disease development, including temperature, light and water availability, soil fertility, wind speeds, and atmospheric ozone, methane and CO_2 concentrations. Among these, three are predicted to most likely change and affect the climate in this century — CO_2 concentration, temperature, and water availability. Therefore, this Review will focus on these three environmental factors. Readers are referred to excellent reviews that discuss the effects on disease of other environmental factors [6–9].

Atmospheric Carbon Dioxide Concentration

Data collected across the globe show a dramatic increase in atmospheric CO_2 concentrations since the dawn of the industrial revolution. Current atmospheric CO_2 concentration has surpassed the 400 ppm threshold (from less than 285 ppm at the start of the 19th century [10]) mainly because of anthropogenic influence [11]. How this will affect future climate conditions is still a matter of debate. It is projected that the increase in greenhouse gases will cause an increase in global temperature between 1.5°C and 4.8°C by the end of the century relative to the temperature before the industrial era (year 1800). A 0.85°C increase [90% uncertainty interval between 0.65 and 1.06°C] has been observed so far [11], with weather extremes (such as heat waves, droughts, floods, and heavy rains) predicted to increase in severity and/or duration [12,13]. For more information on climate change, please refer to the latest Intergovernmental Panel on Climate Change report [11], the U.S. Global Research Program report [14] and NASA (https://climate.nasa.gov/).

Globally, predicted future increases in temperature and regional changes in water availability [11] will change the areas in which crops will be produced, and the vector and pathogen populations causing disease [15]. Pathogen and pest distribution has even been observed to move towards the poles as global temperature increases [16]. By 2050, crop production is predicted to decrease throughout the world, especially in the poorest and more vulnerable countries (this study did not take into account the effect of increased CO_2 on photosynthesis) [17,18].

Higher CO_2 concentrations are expected to increase the photosynthetic rate and crop yield of C3 plants. Depending on the crop studied; there is between a 5 to 40% increase in yield in free-air CO_2 enrichment (FACE) experiments using the predicted CO_2 concentrations that will be reached by the end of

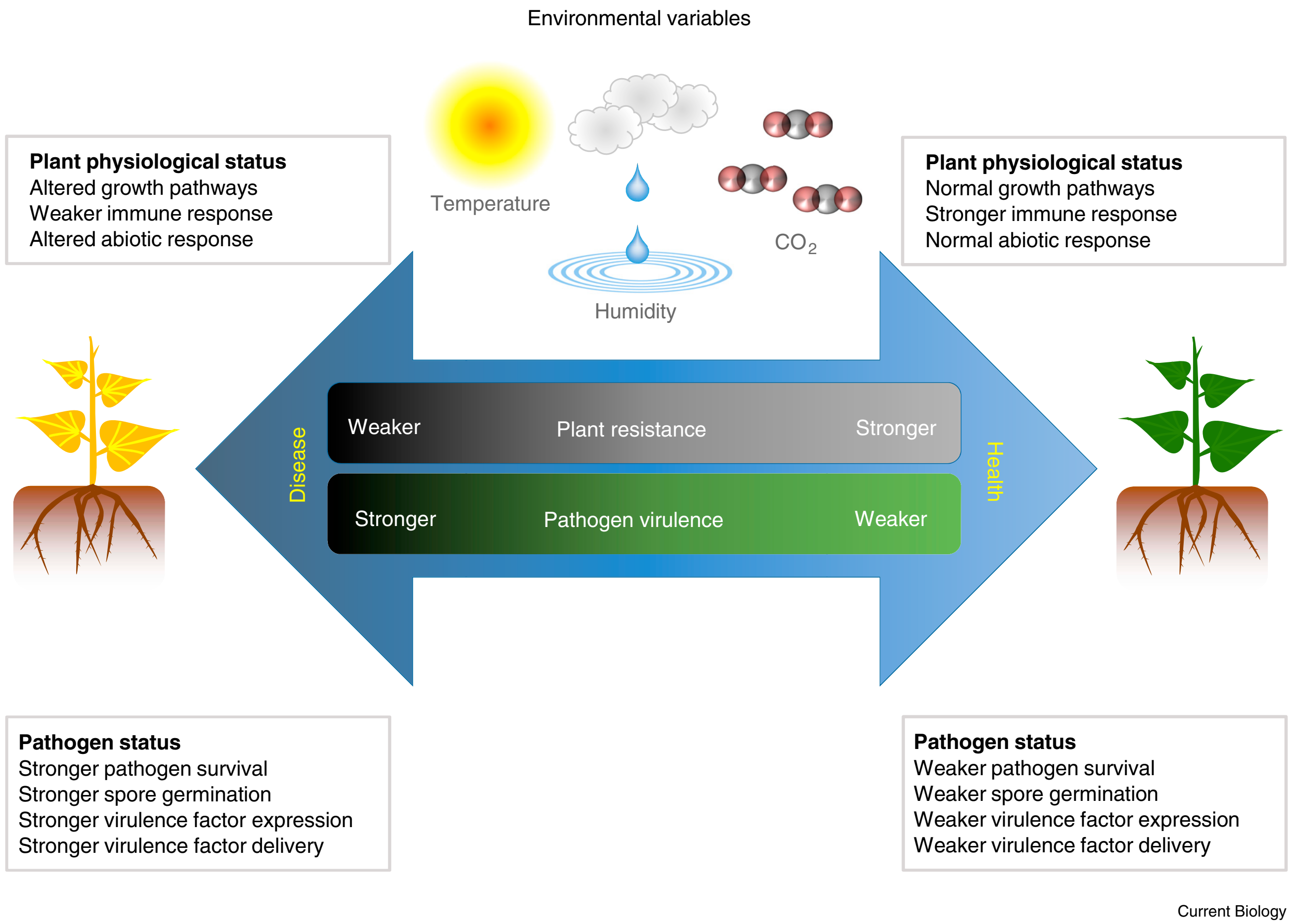


Figure 1. Impact of environmental conditions on plant–pathogen interactions.
The environment–host–pathogen tripartite interaction operates within a continuum, from interactions fully conducive for disease (disease optima) to those that maintain healthy plants. Environmental conditions can have profound effects on a host plant's physiological state, including its growth, immune signaling and abiotic stress response, as well as a pathogen's survival, germination, and expression and delivery of virulence factors. These variable environmental conditions can render the same plant being fully susceptible to being fully resistant, while the pathogen could range from being able to cause severe disease to being only weakly pathogenic. The three most important environmental variables predicted to change in this century are atmospheric CO_2 concentration, temperature, and water availability.

the century [19]. C4 plants (e.g., corn and sugarcane), however, will not benefit from this increase in atmospheric CO_2 due to their already inherent CO_2 concentration mechanisms [20]. While increased CO_2 concentrations can increase the yields of C3 crop plants, they also increase disease severity in rice and wheat (620 and 780 ppm, respectively), two of our staple crops with the highest worldwide production [21,22]. For the fungal pathogen *Fusarium graminearum*, elevated CO_2 levels not only increased the susceptibility of wheat varieties (irrespective of the resistant and susceptible genotypes evaluated), but also increased the virulence of the fungal isolate [22], resulting in more severe disease overall. In contrast, in some oomycete–plant interactions such as between soybean and *Peronospora manshurica*, CO_2 concentrations of 550 ppm decreased the severity of the disease by more than 50% [23]. The complicated relationship between CO_2 concentrations and disease is further reflected for the potato late blight disease caused by the oomycete *Phytophthora infestans*. A modeling study assessing the expected risk of late blight infection in potato-growing areas of Scotland for the next 60 years predicted an increase in *Phytophthora* infection during the first half of the potato growing season, but a decrease in infection for the second half [24]. The model did not separate the effect of increased CO_2 concentration from that of increased temperature, and as such, the predicted effect was most likely only indirectly influenced by CO_2. For tripartite biotic interactions involving a plant (wheat), a virus (barley yellow dwarf virus, BYDV), and a vector (the aphid *Rhopalosiphum padi*), non-infected plants had lower aphid populations at elevated CO_2, but higher CO_2 concentrations showed no effect on overall BDYV infection, highlighting complicated effects of CO_2 concentrations on the different biotic interacting components [25].

These disparate reports on the effects of CO_2 concentrations on plant–pathogen interactions are not sufficient to draw any unifying principles and, therefore, underscore the great need to expand research in this area. Most importantly, as different plant–pathogen interactions involve pathogens of different lifestyles (e.g., biotrophic vs. necrotrophic) and engagement of different plant defense pathways, increased efforts should be

Table 2. Crops with the highest area harvested worldwide.

Crop	Area harvested (million hectares)	Production (million metric tons)	Calories (C) consumed per day	% of calories of a 2,884-calorie[a] diet
Rice	162.72	741.48	544	18.9
Wheat	220.42	729.01	527	18.3
Sugarcane	27.12	1884.25	213	7.4
Sugar beet	4.47	269.71		
Maize	184.80	1037.79	155	5.4
Soybean	117.55	306.52	96	3.3
Potato	19.10	381.68	64	2.2
Palm fruit (for oil)	18.70	274.62	59	2.0
Peanut (including its shell)	26.54	43.92	38	1.3
Cassava	23.87	268.28	37	1.3
Sunflower seed	25.20	41.42	36	1.2
Rapeseed and mustard	36.93	74.48	36	1.2
Banana and plantain	5.39	114.13	29	1.0
Sorghum	44.96	68.94	28	1.0
Millet	31.43	28.38	27	0.9
Sweet potato	8.35	106.60	22	0.8
Yam	7.76	68.13	13	0.5
Cotton (seed)	34.75	79.07	13	0.5
Tomato	5.02	170.75	11	0.4
Barley	49.43	144.49	7	0.2
Watermelons	3.48	111.01	–[b]	–[b]

For 2014, the total worldwide area harvested was 1,384 million hectares, while production was 8,889 million metric tons. Almost 68% of the world calorie consumption is obtained from these 19 crops. Data for calories consumed per day from sugarcane and sugar beet combined. Data from FAOSTAT (Statistics Division, Food and Agriculture Organization of the United Nations), 2013 and 2014.

[a]Average calories consumed in the world. A calorie (C) is equivalent to 1 kcal.

[b]No data available.

devoted to determining whether the different effects could be attributed to different pathogen lifestyles and/or the CO_2 modulation of plant defense pathways.

Temperature

For every plant–pathogen interaction, there is an optimal temperature range at which disease develops. For example, 15°C is optimal for *Globodera pallida* nematodes to infect potato plants [26], daytime temperatures of 35°C and nighttime temperatures of 27°C are most favorable for *Xanthomonas oryzae* bacteria to colonize rice [27], whereas temperature between 26°C and 31°C are ideal for papaya ringspot virus (PRSV) to infect papaya [28]. However, temperature averages might not always be good predictors of the potential for an infection. The rust pathogen *Puccinia striiformis* was found to be unable to cause infections in the laboratory if inoculated in wheat seedlings at constant temperatures over 21°C, while in the field, infections occurred even when temperatures fluctuated between 18°C and 30°C [29]. This apparent discrepancy seemed to result from field temperatures being optimal for infection at night, as the fungus was able to survive and cause infection if the lethal temperature did not exceed a certain time threshold [30]. Also, even when the same average temperatures were used to study the interaction between *P. infestans* and potato, small 5°C fluctuations in temperature caused plants to be more susceptible than daily constant temperatures [31]. These findings have important implications, as extremes in temperature (e.g., heat waves) have become more frequent, despite the predicted small increase in global average temperature due to climate change. Consequently, in order to truly understand the effect of temperature on disease development, future research should use conditions that resemble the dynamic conditions observed in nature (e.g., changing temperature conditions for plant growth throughout the day).

It is anticipated that the projected increase in global temperature will most likely change the regional distribution in which a crop is susceptible to a particular pathogen. For areas outside of the tropics, a global trend is the higher prevalence of pathogen inocula overwintering for the next crop-growing season, with potential for more severe and frequent epidemics [32]. This will be particularly relevant for pathogens that already possess cold-, heat- or desiccation-tolerant surviving structures, some of which may last several years, even under adverse conditions [33,34]. Another outcome of warming temperatures is that new pathogen strains better adapted to these temperatures may become prevalent. For *P. infestans*, variability in the response to temperature has been observed in populations from different geographic regions [35]. Regarding the interaction between the rust fungus *Puccinia striiformis* and wheat, new pathogen races that are more aggressive in causing disease at higher temperatures have appeared since the year 2000 and have become more prevalent worldwide in only a few years [36,37]. It is important to note, however, that some diseases that can potentially cause an epidemic never develop into one simply because of transient temperature shifts. For example, temperatures fluctuating between a low of 12°C and a high of 25°C are conducive for soybean rust. However, exposing infected plants to as short as 1 hour at 37°C abolishes symptom development [38]. Transient temperature shifts, such as those that are predicted to occur more frequently this century [12], might stop or further enhance potential future epidemics. More studies on the distribution and variability in the virulence of pathogen strains are required to evaluate the future potential of disease outbreaks and infer the structure of future sweeping pathogen populations.

For tripartite biotic interactions involving a pathogen-transmitting vector, temperature could profoundly influence disease incidence and/or severity by affecting the vector. This is especially important for viruses, the majority of which are transmitted by insects [39]. Banana bunchy top virus (BBTV) is transmitted by an aphid (*Pentalonia nigronervosa*) whose fastest development, highest fecundity, and lowest mortality occur at 25°C [40], which is the same temperature at which transmission of BBTV is most efficient [41]. Temperatures favorable for the aphid could explain epidemics in banana, even if the conditions are suboptimal for BBTV replication. For many diseases, vector populations are an important factor that needs to be thoroughly considered when deciding on strategies for disease prediction and control under changing climate conditions.

Water Availability

Most plant diseases are favored by conditions of rain, high air humidity and high soil moisture. In particular, the virulence of pathogens that infect aerial tissues is greatly promoted by rain and high humidity. For example, the virulence of the fungus *Sclerotinia sclerotiorum* increases as air humidity increases, with highest disease development in lettuce plants when the air relative humidity surpasses 80% [42]. For fruit rot caused by *Phytophthora capsici*, pathogen growth and disease symptoms are highest at close to 100% relative humidity [43]. For many fungal pathogens, the duration in which the leaf has water on its surface (i.e., leaf wetness) is critical for disease development, with many devastating plant pathogens such as *Magnaporthe oryzae* (rice blast fungus) or *Puccinia striiformis* (stripe rust fungus) requiring a minimum of 5 hours of leaf wetness for disease to occur [44]. In conditions with clear night skies and light winds, the duration of leaf wetness is even longer due to dew accumulation in leaves [45]. As air can hold more water vapor at higher temperatures, the possibility for dew accumulation (and consequently, pathogen infection) increases at higher temperatures. On the other hand, soil moisture is more critical than air humidity for soil-inhabiting pathogens, many of which cause plant wilt diseases. Lower soil moisture decreases the incidence of infection of *Ralstonia solanacearum* in tomato plants [46]. In fact, balancing moisture levels to reduce disease incidence but still favor plant growth is an important cultural practice for disease management [47].

Contrary to the effect observed for *Ralstonia* infection of tomato plants, drought conditions caused more aggressive infections by *Magnaporthe oryzae* in rice, resulting in larger pathogen populations and more visible disease symptoms [48]. In the interaction between potato and the bacterial scab pathogen *Streptomyces* spp., lower soil moisture also favors disease development, and as such, increasing soil moisture can be used as a strategy to control this disease [49]. These two examples are exceptions, as there are few plant–pathogen interactions in which low humidity favors disease development.

Some of the effect of high humidity on pathogens does not directly translate into crop yield reductions but on reduction of product marketability, as exemplified by several pathogenic fungi (*Fusarium* spp. and *Aspergillus* spp.) that produce mycotoxins. Presence of mycotoxins even in minute quantities prevents the marketability of the crop. In wheat plants infected with *F. graminearum* (head blight fungus), higher humidity increased the concentration of the mycotoxin deoxynivalenol [50,51], which prevented the grain from being sold.

Combined Effect of Multiple Environmental Factors

Predicting the combined effect of changing environmental conditions on disease is not straightforward [52,53]. In *Arabidopsis*, combined heat, drought and turnip mosaic virus (TuMV) infection cause a more severe reduction in plant growth than each individual factor alone [54]. For many fungal pathogens, the combination of warm temperatures and high humidity provide the optimum conditions for disease development [42]. In the interaction between *Botrytis cinerea* and grapes, both air relative humidity close to 100% and temperatures between 20 and 25°C were required for optimal disease [55], and deviations from this optimum resulted in a drastic decrease in disease incidence. In the field, plants and pathogens experience multiple environmental factors. How the combined effects of multiple environmental conditions impact the disease outcome remains one of the most outstanding and challenging questions for the future study of plant–pathogen interactions.

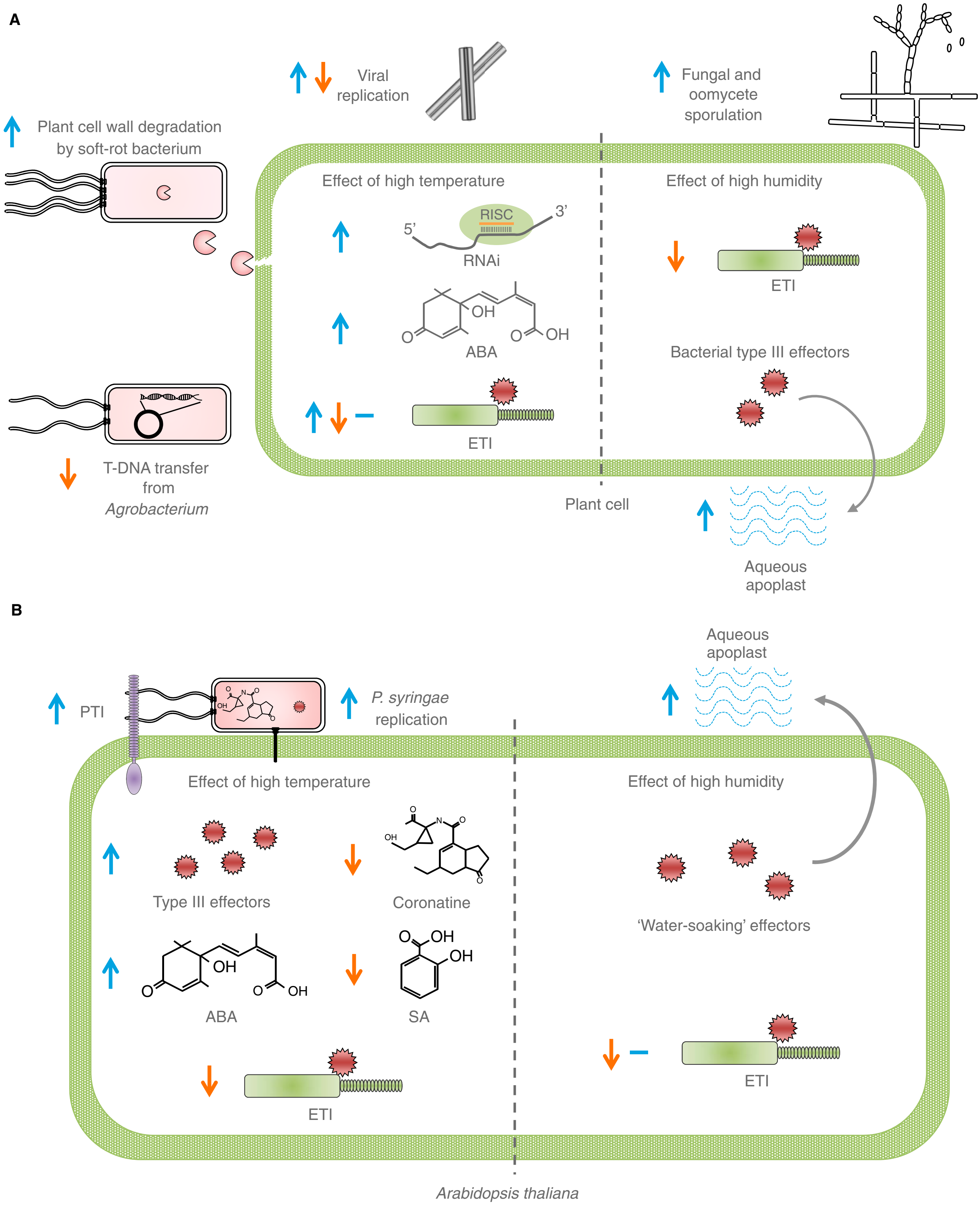
Molecular Mechanisms Underlying Environmental Modulation of Plant Immunity

Plants have evolved sophisticated defense mechanisms to fend off pathogen attacks. These mechanisms include PAMP-triggered immunity (PTI), effector-triggered immunity (ETI), RNA interference (RNAi), and intricate regulation of defense hormone pathways (Figure 2). Many excellent reviews have discussed the current understanding of these mechanisms [56–61]. Here, we focus on the emerging studies aimed to understand how environmental conditions modulate these essential defense mechanisms.

Environmental Impact on PAMP-triggered Immunity (PTI)

PTI is a mechanism in which conserved molecules of microbes (pathogen- or microbe-associated molecular patterns, PAMP or MAMP, respectively) are recognized by plant plasma-membrane-localized pattern recognition receptors (PRR). MAMP recognition causes a signaling cascade that includes protein phosphorylation, reactive oxygen species (ROS) production, Ca^{2+} concentration increases, and gene activation that lead, through a yet unknown mechanism, to halt microbial growth [56]. PTI is thought to be part of a mechanism that prevents multiplication of the vast number of nonpathogenic microbes that plants encounter in nature.

There is emerging evidence that humidity and water availability can influence the effectiveness of PTI. Virulent bacterial pathogens often cause the leaf apoplast (intercellular spaces) to be water-soaked as part of the infection cycle [62,63]. Keeping leaves water-soaked in the apoplast under high humidity allows PTI-inducing non-pathogenic *Pseudomonas* bacteria to



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grow to a significant level in *Arabidopsis*, bean, and tobacco plants [64].

Stomata are microscopic openings in leaves and stems that are involved in CO_2 and O_2 exchange with the atmosphere, and in water loss through transpiration. Under drought stress, the balance between the biosynthesis and catabolism of the hormone abscisic acid (ABA) favors increased concentration of this hormone in stomatal guard cells, which leads to stomatal closure [65]. In contrast, under high humidity, stomata open while ABA accumulation decreases [66]. Stomata are also commonly used as entry points for leaf pathogens. PTI activation triggers the closure of stomata after the recognition of MAMPs by stomatal guard cells [67,68], as part of the plant defense against bacterial entry into the leaf apoplast. It was recently shown that high humidity conditions blocked PTI-induced stomatal closure in both *Arabidopsis* and bean plants [69], suggesting that stomatal defense is another point of environmental regulation of PTI.

Likewise, PTI signaling can be altered by temperature changes. In *Arabidopsis*, short warm-temperature treatments (28°C for 15 minutes) induced higher PTI-associated MAPK (mitogen-activated protein kinase) phosphorylation and PTI marker gene expression after PAMP exposure, suggesting that PTI may be enhanced at warm temperatures [70].

Environmental Impact on Effector-triggered Immunity (ETI)

A widely used and arguably the most effective form of genetically controlled resistance against plant pathogens is ETI. During ETI, a virulence-promoting pathogen effector or its activity inside plant cells is sensed by a plant resistance (R) protein, most of which are NLR (nucleotide-binding domain and leucine-rich repeat) proteins [60]. This recognition triggers a signaling cascade that usually culminates in a hypersensitive response (HR), a type of programmed cell death thought to contain the pathogen at the site of infection [61]. While effective against biotrophic pathogens, ETI is ineffective against (and sometimes can be taken advantage of by) necrotrophs, which, as already mentioned, obtain their nutrients from dead host tissues [71].

In many pathosystems, high temperatures compromise ETI. For example, the tobacco N protein for the resistance against tobacco mosaic virus (TMV) [72], several tomato Cf proteins for resistance against the leaf mold pathogen *Cladosporium fulvum* [73], and several R proteins that confer resistance to the bacterial pathogen *Pseudomonas syringae* (RPM1, RPS2, and RPS4 from *Arabidopsis*) or potato virus X (PVX, recognized by Rx in potato) [74] do not mount an effective ETI at temperatures higher than 30°C. Contrary to most R proteins, however, Xa7, a rice disease resistance protein against *Xanthomonas oryzae*, is more effective at higher temperatures than at lower temperatures [75]. The suppressive effect of high temperature on ETI is also evident in certain 'autoimmune' mutant plants in which ETI and spontaneous cell death are constitutively activated. A prominent example is some *Arabidopsis snc1* mutant alleles. *SNC1* is an *Arabidopsis* NLR gene in which several mutations cause an autoimmune phenotype and increase resistance to *P. syringae* at 22°C but not at 28°C [76]. Transcription of the *SNC1* gene and the autoimmune phenotype of a *snc1* allele are suppressed by a gain-of-function allele of the pseudokinase ZED1 at 22°C [77]. ZED1 is an *Arabidopsis* protein required for the recognition of the bacterial effector HopZ1a by the R protein ZAR1 [78]. As such, SNC1 and ZED1 proteins may be part of the same high temperature-sensitive ETI pathway [77].

The effect of temperature on ETI appears to depend on the duration of the temperature regime. Recognition of *P. syringae* effector AvrRpt2 by *Arabidopsis* RPS2 and subsequent inhibition of bacterial growth is lost if plants are grown for 3 weeks at 28°C prior to infection [74]. However, if plants had been acclimated for only 1 day at 28°C, ETI containment of growth is still present, even though the ETI-associated HR cell death is absent [79]. Therefore, it seems that acclimation at high temperatures for short periods of time might only disarm part of the ETI signaling pathways. The variable results from using different durations of temperature treatment suggests a need to conduct future temperature experiments with diurnally fluctuating temperature cycles and heat waves that resemble those experienced by crops in the field.

How high temperature suppresses ETI is not fully understood. Several studies suggest that one effect may be at the level of nuclear localization of some R proteins. For example, autoimmunity-associated mutations in *SNC1* cause increased SNC1 nuclear localization at 22°C. A reduction of nuclear localization at 28°C is associated with suppression of autoimmune phenotypes. Similarly, nuclear localization of the R protein N was observed after recognition of the TMV coat protein at 22°C but not at 28°C [76]. A genetic suppressor screen identified an *Arabidopsis* mutant, *aba2-21* (an allele of a gene involved in ABA biosynthesis), that restores SNC1 autoimmunity at high temperatures [80]. The *aba2-21* mutation increases the nuclear localization of SNC1 and RPS4 (which recognizes the *Pseudomonas* effector AvrRps4) at 28°C [80]; therefore, the lack of R protein nuclear accumulation might cause ETI failure at high temperatures. It is important to note that not all R proteins show nuclear localization [81,82], so this phenomenon likely applies to only a subset of R proteins.

As with high temperatures, high humidity can also interfere with ETI-associated HR. For example, the response to *C. fulvum* Avr4 and Avr9 effectors by tomato Cf R proteins is

Figure 2. Environmental impact on the molecular mechanisms influencing disease.
(A) A diagram depicting examples of plant–pathogen–environment triangular interactions. High temperature (left half of plant cell) increases the production of bacterial plant cell wall-degrading enzymes by soft-rotting bacteria, while it decreases transfer DNA (T-DNA) delivery by *Agrobacterium* spp. Viral accumulation can be positively or negatively influenced by elevated temperature; however, the plant antiviral RNA interference (RNAi) mechanisms are generally increased by warm temperatures. Abscisic acid (ABA) accumulation is increased, whereas effector-triggered immunity (ETI) can be unaffected, heightened, or rendered ineffective at high temperatures. High humidity (left half of cell) increases sporulation by fungal and oomycete pathogens, and allows bacterial effectors to establish an aqueous apoplast in infected leaves. In tomato, ETI is negatively affected by high humidity. Abbreviation: RISC, RNA-induced silencing complex. (B) A diagram depicting the current knowledge of the *Arabidopsis*–*Pseudomonas*–environment triangular interaction. Elevated temperature increases effector translocation into plant cells and decreases production of the toxin coronatine from *P. syringae*. On the other hand, elevated temperature increases ABA and decreases salicylic acid (SA) hormone concentrations in *Arabidopsis*. A decrease in ETI and an increase in PAMP-triggered immunity (PTI) at elevated temperature have been reported. Higher air humidity conditions favor *P. syringae*, which produce effectors that establish an aqueous apoplast in the infected leaves to favor disease. Also, higher humidity affects some aspects of the ETI response.

dramatically reduced at air humidity levels above 95% [83]. Similarly, the ETI-associated HR is compromised under high humidity conditions for the recognition of the bacterial effector AvrRpt2 by RPS2 in *Arabidopsis* [63]. However, under conditions of high humidity, ETI-associated pathogen population restriction is still evident in certain *Arabidopsis* accessions [84].

The suppressive effect of high humidity on ETI extends to autoimmune mutants. The *Arabidopsis ssi4* mutant, which carries a mutation in an NLR-type R protein, shows spontaneous ETI-like cell death associated with increased levels of pathogen resistance (and the level of the defense hormone salicylic acid [SA]). These phenotypes are abolished under high humidity [85], suggesting the possibility that some *R* genes could be rendered less effective under high humidity conditions during agricultural production.

Environmental Impact on Quantitative Resistance Traits

Partial quantitative resistance to pathogens, which is less characterized at the molecular level than canonical PTI or ETI, reduces but does not completely abolish disease symptoms. It is usually controlled by quantitative trait loci (QTL), which collectively confer resistance [86]. In wheat, a kinase containing a START domain, Yr36, conferred wide-spectrum partial resistance to several races of *Puccinia striiformis* at temperatures as high as 35°C, but not at temperatures below 20°C [87]. *Sr13*, an NLR gene, is another wheat gene that confers partial resistance to *P. striiformis*. Plants carrying *Sr13* induced the transcription of resistance markers and resistance to *P. striiformis* only under high temperatures conditions [88]. As partial quantitative resistance is less prone to go through cycles of boom and bust compared to *R* genes, a moderate level of protection would be provided in plants carrying this type of resistance even under the higher temperature conditions that are predicted to occur in the future.

Environmental Impact on RNA Interference

RNA interference (RNAi) is a widespread defense mechanism against viruses [89]. During viral replication, double-stranded viral RNA intermediates are recognized and cleaved by the RNAi cellular machinery, generating small interfering RNAs (siRNA). These siRNAs are then used to 'silence' the production of new viral particles by targeting virus RNAs [89]. The importance of RNAi in viral defense can be inferred by the plethora of RNAi suppressors present in plant viruses [57]. Temperature affects the efficacy of RNAi-based plant defense. At low temperatures (15°C compared with temperatures between 21°C and 27°C), RNAi viral defenses in *Nicotiana benthamiana* are compromised against *Cymbidium* ringspot virus (CymRSV, an orchid-infecting virus) [90]. However, these results may not be generalized, as grape plants are able to mount efficient RNAi against transgenes even at temperatures as low as 4°C, whereas *Arabidopsis* silencing mechanisms are not functional below 10°C [91]. As with low temperatures, high temperatures also modulate RNAi defenses. In cassava, DNA geminiviruses, including the causal agents of cassava mosaic disease, cause more symptoms and have higher viral titres at 25°C compared with those observed at 30°C. The virus-specific siRNA concentration is lower at 25°C, indicating that the antiviral RNAi pathways may be enhanced at higher temperatures (or that RNAi suppressors are less active) [92].

Environmental Impact on Defense Hormone Pathways

Stress hormones, particularly ABA, SA, jasmonic acid (JA), and ethylene (ET), are well known regulators of abiotic and/or biotic responses in plants. In particular, ABA is involved in regulating multiple abiotic environmental stresses, including drought, heat, cold, and salinity responses [93], whereas SA [94], JA [95] and ET [96] are involved in regulating responses against pathogens.

Part of the environmental impact on plant disease development may be mediated through regulation of defense hormones and/or the hormone crosstalk between ABA signaling (abiotic responses) and SA/JA/ET signaling (biotic responses) [59]. Indeed, during infection with the hemibiotrophic pathogen *P. syringae*, both ABA and SA concentrations increase [97]. Furthermore, when ABA biosynthesis and signaling are impaired, plants become more resistant to *P. syringae* and the necrotroph *Botrytis cinerea* [97,98]. Hormonal crosstalk could cause this observed enhanced resistance for *P. syringae*, as SA accumulation is enhanced in ABA biosynthesis mutants [99]. However, other necrotrophs show the opposite effect. For example, infection of ABA biosynthesis- or signaling-deficient *Arabidopsis* mutant plants with the necrotrophs *Pythium irregulare* and *Alternaria brassicicola* cause disease to develop more aggressively [100]. The conflicting effect of ABA on resistance to fungal necrotrophs (*B. cinerea* vs. *A. brassicicola*) requires future in-depth studies.

A recent study examined the effect of warm temperatures on SA-mediated defense in the *Arabidopsis–P. syringae* interaction. It was found that at 30°C, *Arabidopsis* plants became more susceptible to *P. syringae* [101]. In contrast to a high level of SA accumulation normally induced by *P. syringae* at 23°C, *Arabidopsis* failed to accumulate SA in response to *P. syringae* infection at 30°C [101]. The biosynthetic pathway for pathogen-inducible SA starts from conversion of chorismate to isochorismate by ICS1 (ISOCHORISMATE SYNTHASE 1), from which SA is synthesized [102]. The lack of SA accumulation correlated with a lack of expression of a major group of SA-responsive genes, including the *ICS1* gene. Nuclear entry of NPR1, the master regulator of SA responses, was unaffected at 30°C [102], and as such, the temperature-sensitive component(s) of SA signaling, which awaits identification, likely lies downstream of NPR1 nuclear localization and/or upstream of *ICS1* gene expression.

In contrast to the negative effect of warm temperature on SA defense, exposing *Arabidopsis* plants to cold temperatures (4°C) for more than one week increased SA concentration, expression of SA-regulated defense genes, and resistance to *Pseudomonas* infection [103,104]. CAMTA (calmodulin-binding transcription activator) transcription factors are involved in cold-temperature-mediated expression of SA-responsive genes, including genes involved in SA biosynthesis (e.g., *ICS1*) [103,104]. Interestingly, CAMTA transcriptional factors in tomato were also important for negatively regulating the transcription of drought- and defense-associated genes, and resistance against pathogens and drought [105], further implicating a role of CAMTA transcription factors in connecting temperature, drought, and SA-regulated plant responses.

Compared with the increasing mechanistic understanding of the molecular basis of temperature effect on SA-mediated defense, the effect of environmental conditions on JA- and

ET-mediated defense pathways is much less understood. One study showed that higher relative air humidity increases the expression of genes required for the biosynthesis and signaling of JA [70]. Further research is needed to investigate JA- and ET-mediated defenses, as they affect plant resistance against many pathogenic necrotrophs.

Environmental Modulation of Pathogen Virulence Mechanisms

Pathogens have evolved many mechanisms to subvert plant defenses and other cellular processes in order to exploit the photosynthate-rich plants. These virulence mechanisms include specialized secretion systems for delivering a variety of virulence factors (collectively called effectors) into the plant cell, sporulation in fungal and oomycete pathogens, and intimate association of viruses (and some other pathogens) with their insect vectors (Figure 2). The expression and/or manifestation of many of these virulence mechanisms can be affected by environmental conditions. The environment could also have profound effects on the epiphytic (colonization of the surface of plants) and/or saprophytic (nutrient acquisition from dead organic matter) phase of certain pathogens.

Effect of Temperature on Virulence Mechanisms of Bacterial Pathogens

The interaction between environmental conditions and pathogen virulence mechanisms has been better studied in bacterial pathogens compared to other plant pathogens. *Agrobacterium tumefaciens* is famous for producing tumor-like growth in its hosts, a hallmark of crown gall disease. *Agrobacterium* pathogenesis is dependent on the formation of an extracellular pilus (T-pilus) involved in the translocation of virulence-promoting DNA and proteins, a function that requires the expression of a large number of virulence (*vir*) genes [106]. The expression of *vir* genes is controlled by a two-component system: VirA is a transmembrane sensory protein that detects plant phenolic compounds produced during wounding and phosphorylates VirG, a protein that binds the *vir* DNA box in *vir* genes to increase their expression [107]. Temperatures at or above 32°C reduced *vir* gene expression and rendered *Agrobacterium* strains non-pathogenic on *Kalanchoe* plants, probably due to loss of VirA phosphorylation activity [108]. In addition, temperatures higher than 28°C inhibited pilus formation and decreased Vir protein stability in some but not all *Agrobacterium* strains [109], which correlated with the inability of these strains to cause disease at 28°C.

Soft rotting bacteria are necrotrophs that macerate plant tissues using a multitude of bacterial plant cell-wall-degrading enzymes (PCWDE). For *Pectobacterium atrosepticum*, a soft rotting bacterium, there is a clear correlation between increased virulence at temperatures as high as 35°C and the production of PCWDE and quorum-sensing signals by certain strains [110]. Quorum-sensing signals are required for the production of macerating enzymes when a threshold bacterial population density has been reached [111], and their temperature-dependence limits *Pectobacterium* pathogenesis.

In *Pseudomonas syringae*, temperature has been shown to affect two well-studied virulence mechanisms — the production of phytotoxins and the type III secretion system, which is used by bacteria to deliver virulence effector proteins into the plant cells. In *P. syringae* pv. *glycinea*, the causal agent of bacterial blight in soybean, the coronatine toxin was preferentially produced *in planta* under low temperature conditions [112]. A modified two-component system in which the extracellular sensor CorS is involved in thermoregulation is required for coronatine production [113]. Cold-induced coronatine production is not observed in all coronatine-producing strains, even though epidemics for all those coronatine-producing strains are favored by cool and wet weather conditions [112]. *In vitro* expression of genes for the production of another *Pseudomonas* toxin, phaseolotoxin, [114] and, more importantly, for the expression of genes that encode the type III protein secretion system in culture are favored by low temperatures [115]. Cold temperature-induced expression of type III secretion genes *in vitro*, however, is in contrast with the increased translocation of effectors into the plant cell at high temperatures in *P. syringae*-infected *Arabidopsis* plants [101], highlighting a need for future investigation to untangle the dichotomy of *in vitro* vs. *in vivo* studies with respect to the temperature effects on the virulence of *P. syringae* and other pathogens.

Effect of Humidity on Virulence-promoting Mechanisms in Bacteria

Recent studies suggest that bacterial virulence mechanisms involve the establishment of a disease-promoting liquid environment in the apoplast of infected leaves [62,63]. In *P. syringae*, two highly conserved effectors of *P. syringae*, AvrE and HopM1, are redundantly involved in the establishment of the aqueous apoplast, but only under conditions of high humidity [63]. The environment-dependent virulence function of bacterial effectors can be observed not only for AvrE1 and HopM1, but also for *P. syringae* effector HopAM1, which contributes to virulence only in *Arabidopsis* plants grown under drought conditions [116]. As such, it is possible that the effect on pathogen virulence of some effectors may become apparent only under certain environmental conditions to which plants are exposed in nature, but not in laboratory conditions.

In another bacterial plant pathogen, *Xanthomonas gardneri* (which causes bacterial spot disease on tomato) a transcription activator-like (TAL) effector, AvrHah1, causes water soaking in tomato leaves and seems to be involved in promoting the intake of water from the environment into the apoplast [62]. AvrHah1 might affect cell wall permeability in order to make the apoplast more favorable for bacterial survival and/or dispersal [94].

Environmental Effect on Sporulation by Fungal and Oomycete Pathogens

It is well known that inoculum production and dispersal are critical for disease epidemics in crop fields. In some instances, wind can disperse fungal spores over thousands of kilometers, as has been observed for the wheat stem rust pathogen *Puccinia graminis* as a critical part of its disease cycle [117]. In maize, *Cercospora zeae-maydis* requires humidity higher than 95% for profuse sporulation, and only under this high humidity condition can the effect of temperature on disease be observed (25–30°C being the optimum temperature [118]). This requirement for high humidity for sporulation is observed for the majority of fungal and oomycete pathogens [1,42,119]. Interestingly, sexual and asexual spore production in *F. graminearum* required the protein-refolding chaperone HSP90 [120]. This requirement suggests a possible mechanistic connection between temperature response and

Table 3. Major causes for concern due to plant diseases in the context of climate change.

Increased temperatures:
• *Incidence of plant diseases.* In Asia, greater incidence of rice bacterial and sheath blight could be observed as temperatures increase. In Europe, increasing temperatures will affect sugar beet production due to increased incidences of leaf spot and rhizomania. In Brazil, higher temperatures may increase sugarcane infection by *Colletotrichum falcatum.*
• *Pathogen overwintering.* In the United States, warm temperatures would allow Asian soybean rust to overwinter in higher latitudes and also favor the development of grey leaf spot in maize. In China, warm winters have already contributed to severe epidemics of wheat head blight in recent years.
• *Emergence of new pathogenic strains.* Elevated temperature-adapted races leading to perturbed geographic distributions of late blight and stripe rust diseases.
• *Rise of aggressive plant disease vectors.* Higher populations of whiteflies transmitting viral diseases to cassava and yam in Western Africa.
Increased rainfall/humidity:
• *Increased incidence of moisture-requiring plant diseases.* In Europe, this could potentially threaten potato production by late blight.
• *Decreased crop marketability.* There may be more widespread manifestation of mycotoxin-producing fungi in valuable food staples.

sporulation in this fungus, as HSP90 is required for heat shock responses *in vitro* and colonization *in planta* [120].

Asexual zoospore production in the oomycete *Phytophthora infestans* requires low temperatures. Temperature regulation of zoospore production involves the expression of a protein phosphatase (PinifC3), which controls the phosphorylation status of RNA polymerase and other transcriptional regulators in this oomycete. *PinifC3* transcription is induced at 10°C, as the gene has a cold-induced promoter element [121]. The optimal temperatures where *Phytophthora* and *Fusarium* cause disease epidemics in the field correlate with those required for pathogen sporulation, which is essential for disease dispersal [1,122].

Thus, environmental conditions appear to have pervasive effects on many steps of pathogen infection, ranging from pathogen sporulation, pathogen growth and virulence gene expression in plants, and the overwintering of inocula, which is critical for initiating infections in subsequent plant-growing seasons. It should be pointed out that climate conditions near the optimum for the multiplication of pathogens could potentially increase the probability of pathogen evolution due to high population numbers. This could effectively change the pathogen population structures and create a condition for the emergence of new virulence strains, a cause of significant concern for future disease epidemics (Table 3).

Concluding Remarks and Future Outlook

Clearly, the ongoing changes in climate patterns have potential to threaten the already vulnerable global food security in multiple ways, including exacerbating major plant diseases and creating weather conditions for devastating new diseases to emerge in critical food-producing regions. Based on current climatic trends and plant and pathogen responses to major climatic factors in this century, we predict certain diseases to be of major concern for worldwide food security (Table 3). There is no more urgent time than now to call for intensifying global research efforts to understand how individual and combined environmental conditions influence plant immunity, pathogen virulence and disease development. From limited (and often disparate) literature in this important area of plant research, it is already obvious that the effects of CO_2, temperature and humidity and other environmental conditions on disease development could vary depending on plant and pathogen species. Therefore, predicting a general outcome for all diseases may be difficult. An overarching theme, however, is that there are environmental optima for plant immune and pathogen virulence systems to function properly. Environmental deviations from these optima inevitably reduce the strength of plant immunity or pathogen virulence. The final impact on disease reflects the combined environmental effects on plant immunity and pathogen virulence (Figure 1). However, with increased data, it may be possible to develop conceptual theories to unify the differential influences of environmental conditions on different plant diseases. For example, one could apply the mathematical relations used in the metabolic theory of ecology (MTE) to plant–pathogen interactions, as has been done in assessing the effect of climatic factors on human diseases [123]. MTE integrates the metabolic rate, mass and temperature of organisms [124]; the information may potentially be used to predict how host plants and plant pathogens would respond to dynamically changing environments.

Basic research into how temperature, humidity and other abiotic stresses reduce plant immune signaling will likely yield environment-vulnerable points in the plant immune system. Such knowledge will hopefully provide a foundation for developing a new generation of plant varieties in which the plant immune system is more resilient to environmental fluctuations. A well-known phenomenon associated with increasing stress tolerance is reduction in plant growth and yield. Recent efforts have been successful to reduce the detrimental effects from the expression of immunity-related genes [125]. It would be interesting to investigate whether such improved balance between plant growth/yield and disease resistance can be maintained under different climatic conditions.

Future breeding programs for new improved varieties should try to incorporate the abiotic tolerance, growth, and biotic resistance variabilities that favor plant immunity and disfavor pathogen virulence. These traits may be found in close wild relatives of cultivated crops, as they could have evolved combined abiotic and disease-resistance strategies over millennia that might not be present in the narrow germplasm from which a modern crop variety is derived. Traits could be introduced into cultivated plant varieties through gene pyramiding, marker-assisted selection, whole genome-wide association mapping (GWAS) analysis and other approaches. Some attempts to incorporate wild germplasm of both abiotic and biotic resistance into cultivated plant species have been carried out. Good examples are the introgression of chromosomal segments of the entire genome of a highly abiotic and biotic stress-tolerant wild grass species (*Festuca pratensis*) into forage grasses [126], and the extraordinary development of an advanced rice breeding line with six genes of resistance to bacterial and fungal diseases, two genes for resistance to an insect pest, and two major QTLs conferring salt and flood tolerance [127]. More effort of this type is needed to incorporate both pathogen resistance and environmental tolerance into commercial varieties. Another approach could be the use of planting mixtures of crop varieties that cover a range of climate and pathogen conditions (an idea that has been effectively employed for years when using multilines, near-isogenic lines that carry different resistance genes). This approach almost doubled the yields in rice fields affected by *M. oryzae* infection (compared with fields sown with monocultures) [128]. Similarly, planting mixed native landraces of potato in the Andes protects the farmer from the unpredictability of weather and pathogen infection [129].

A major concern of genetic manipulation of plant traits is the reluctance of the public to accept genetically modified crops. For example, many countries have significant restrictions on the use of transgenic organisms (only 28 countries have been planting transgenic organisms as of 2016) [130]. With the advent of the CRISPR (clustered regularly interspersed short palindromic repeats) technology, such concern may be reduced. New technologies will also enable us to more accurately forecast future pathogen epidemics by using nanosensors to detect pathogen populations in the field [131]. In addition, the identification of plants resistant to both disease and environmental stresses could benefit from the use of high-throughput phenotyping technology [132].

Another promising area of research aimed at making crop plants resilient to both abiotic and biotic stresses is that involving the emerging field of plant–microbiome interactions [133]. Disease-suppressive soils with enrichment in the bacterial clades Proteobacteria and Firmicutes protect against damping-off disease caused by *R. solani* [134], and numerous individual ‘biocontrol’ microbes have an ability to fight against pathogens [135,136]. Specific microbes are also known to enhance tolerance to drought and other abiotic stresses [137]. One can envision harnessing the potential of a community-defined microbiome in both suppressing disease and increasing environmental tolerance in the future to enhance crop resilience and productivity. Readers are referred to recent reviews on this topic [133,138].

Finally, we would like to point out that, although there is a widespread observation of the environmental effect on diverse diseases caused by a wide range of pathogens with different lifestyles, a major concern is that in-depth studies have been done mostly using only a few model plant species (e.g., *Arabidopsis*) and with biotrophic pathogens. For many crops with the highest world production, there are only descriptive studies on the environmental effects on major diseases. Knowledge on the molecular phenomena behind plant resistance and pathogen virulence is almost completely absent. This dearth of studies hampers the development of appropriate resistance strategies against the most devastating world plant diseases (Table 1). Also, future studies addressing the effect of environmental factors on plant disease will need to take into account the dynamic nature of conditions in the field. We need controlled growth chambers that more closely resemble the dynamic conditions to which plants and pathogens are subjected in nature, so that field trials can be reproduced, and any laboratory results can be validated. Current static laboratory experiments, although simple to perform, will not provide complete answers to solving the molecular mysteries of plant diseases. The classically accepted paradigms used to approach plant–pathogen interactions must now change. Future studies of plant–pathogen interactions should increasingly consider the multi-dimensional nature of ‘plant–pathogen–environment’ interactions, which are more reflective of what occur in crop fields and natural ecosystems.

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