Fight hard or die trying: when plants face pathogens under heat stress

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Summary

In their natural environment, plants are exposed to biotic or abiotic stresses that occur sequentially or simultaneously. Plant responses to these stresses have been studied widely and have been well characterised in simplified systems involving single plant species facing individual stress. Temperature elevation is a major abiotic driver of climate change and scenarios have predicted an increase in the number and severity of epidemics. In this context, here we review the available data on the effect of heat stress on plant–pathogen interactions. Considering 45 studies performed on model or crop species, we discuss the possible implications of the optimum growth temperature of plant hosts and pathogens, mode of stress application and temperature variation on resistance modulations. Alarmingly, most identified resistances are altered under temperature elevation, regardless of the plant and pathogen species. Therefore, we have listed current knowledge on heat-dependent plant immune mechanisms and pathogen thermosensory processes, mainly studied in animals and human pathogens, that could help to understand the outcome of plant–pathogen interactions under elevated temperatures. Based on a general overview of the mechanisms involved in plant responses to pathogens, and integrating multiple interactions with the biotic environment, we provide recommendations to optimise plant disease resistance under heat stress and to identify thermotolerant resistance mechanisms.

I. Introduction

During their life cycle, wild and cultivated plants have to deal with multiple environmental constraints that often occur simultaneously. The complex sequence of genetic, molecular and physiological responses of the plant subjected to these constraints is defined as a stress. Depending on the nature of the triggering factor, stress can be classified into two categories, biotic and abiotic. Biotic stress is induced by other living organisms, such as weeds, insects, bacteria, fungi and viruses, that may be either beneficial (symbiotic interactions) or harmful (i.e. competitive and pathogenic interactions). In the second category, abiotic stress is caused by nonliving factors such as drought, pollution, soil salinity, nonoptimal temperature and light conditions or variations in water or in nutrient availability. Due to their sessile nature, the ability of plants

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to adapt to stress is crucial. Until recently, the identification and the study of physiological, genetic and molecular mechanisms involved in plant disease responses were mainly focused on stress applied separately, while studies that integrated combined stresses remain under-represented (Suzuki *et al.*, 2014; Pandey *et al.*, 2017; Zhang & Sonnewald, 2017). Studies related to plant–pathogen–abiotic factor interactions are even scarcer. Overall, it appears that the mechanisms that occur on the plant side are complex and quite different from those involved when individual biotic or abiotic constraints are considered (Pandey *et al.*, 2015; Zhang & Sonnewald, 2017).

To face pathogen attack, plants have developed different defence strategies. Preformed components on the surface of plant organs, such as wax layer, rigid cell walls, cuticular lipids (Reina-Pinto & Yephremov, 2009), antimicrobial enzymes (Habib & Fazili, 2007) or secondary metabolites (Ahuja *et al.*, 2012; Piasecka *et al.*, 2015), constitute a first barrier that restricts pathogen entry. Pathogens that overcome these first obstacles are then confronted with induced plant defence responses that have been studied extensively and well characterised (Jones & Dangl, 2006; Dodds & Rathjen, 2010; Miller *et al.*, 2017). The plant immune system relies on two layers of defence. The first layer involves pattern recognition receptors (PRRs) on the cell surface that perceive conserved pathogen-associated molecular patterns (PAMPs) leading to PAMP-triggered immunity (PTI). This immune response is nonspecific and confers a basal resistance level to a broad spectrum of pathogens. Adapted pathogens have evolved sophisticated virulence strategies that rely on virulence factors that can interfere with various host processes, including PTI. For plant bacterial pathogens, effector proteins are secreted and injected into the host cytoplasm through a type III secretion system (T3SS), promoting effector-triggered susceptibility (ETS) (Deslandes & Rivas, 2012). The second layer of defence involves intracellular nucleotide-binding oligomerisation domain (NOD)-like receptors (NLRs) that can specifically detect effector virulence activities and activate a strong innate immune response called effector-triggered immunity (ETI) (Wang *et al.*, 2019a). ETI is often associated with a localised programmed cell death (called HR, for hypersensitive response) that restricts further spread of the pathogen (Mur *et al.*, 2008). Members of the NLR protein family share common structural features, including a nucleotide-binding domain (NB) and a leucine-rich-repeat domain (LRR) (Jones & Dangl, 2006). Depending on the amino acid domain localised at their N-termini, these immune receptors are classified into CC-NB-LRR (coiled coil) or TIR-NB-LRR (Toll, interleukin-1 receptor) proteins (Eitas & Dangl, 2010). ETI is generally species- and strain-specific and often leads to full resistance, resulting in a strong selective pressure on pathogens to overcome immune responses (Roux *et al.*, 2014). However, not all pathogenic microbial determinants fit the classic definition of PAMPs or effectors, such that PTI and ETI immune responses involve similar mechanisms such as reactive oxygen species (ROS) production, kinase signalling and transcriptome reprogramming. The distinction between these defence responses therefore remains ambiguous. Indeed, some studies have suggested that a microbial effector might have driven the emergence of plant pattern recognition systems mediating PTI. For example, necrosis and ethylene-inducing peptide 1 (Nep1)-like proteins (NLPs) from bacteria, fungi and oomycetes are effectors that activate PTI through phytotoxin-induced host cell damage. In addition, a conserved pattern of 20 amino acid residues (nlp20) found in these NLPs has been shown to confer broad-spectrum resistance through pattern recognition, a mechanism reminiscent of the immune receptor mediators of the ETI (Böhm *et al.*, 2014). Furthermore, the direct association between an R-like protein, SlNRC4a, and PRRs leads to enhanced PTI signalling in the absence of effectors (Leibman-Markus *et al.*, 2018). Therefore, plant resistance has been suggested to be in a continuum between PTI and ETI, based on the recognition of pathogen molecules by that appropriate plant receptors to activate an efficient immune response (Thomma *et al.*, 2011). As many partial resistance responses are more frequently observed than ETI in natural populations and crop fields (Young, 1996; Bartoli & Roux, 2017), several studies have proposed that this phenomenon is explained by quantitative disease resistance (QDR). Unlike ETI, which is defined as a qualitative resistance, QDR is characterised by a continuous distribution of resistance phenotypes within a population rather than a total absence of disease, and by polygenic architecture (Roux *et al.*, 2014; French *et al.*, 2016; Bartoli & Roux, 2017).

Fluctuating climate parameters are among the types of abiotic stress to which plants must also adapt. In the context of climate change, scenarios predict variations in all the components of the climate system, resulting in more intense, frequent and long-lasting extreme weather events worldwide (IPCC, 2019). The speed, brutality and severity of projected changes represent a major threat of unknown magnitude that would increase the likelihood of altering species distribution areas and ecosystem equilibrium (Bebber, 2015), therefore affecting natural biodiversity (Pimm *et al.*, 2014) and global food security (IPCC, 2014). Amongst climate risks, an increase in mean temperature is one of the main abiotic fluctuations to which plants will have to adapt (Bita & Gerats, 2013; Suzuki *et al.*, 2014; Velasquez *et al.*, 2018). Based on average global surface temperature is predicted to rise from 1.5°C to 4.8°C by the end of the century (IPCC, 2014). While a shift in the geographic expansion of pathogens polewards has already been observed (Bebber *et al.*, 2013), temperature elevation (TpE) is also expected to favour the emergence of new pathogens and to increase the occurrence and severity of epidemics (Elad & Pertot, 2014; Bebber, 2015; McDonald & Stukenbrock, 2016).

Interestingly, a growing number of studies have shown that TpE can balance the plant immune response in different ways, although the underlying mechanisms remain poorly understood. The purpose of this review was to gather and discuss studies that describe the effect of TpE either (1) on plants or pathogens, (2) on both partners in interaction, or finally (3) on the outcome of the interaction. By considering 45 studies that describe the effect of temperature on plant response to pathogens, we found a predominantly negative effect of TpE on the main known resistance mechanisms. We discuss the implications of the diversity of the experimental conditions used, the way combined stresses were applied, the level of explored genetic diversity, and the cellular, genetic and molecular mechanisms modulated by TpE on either plants or pathogens. Finally, we provide some recommendations

on research directions that may improve our understanding of the combinatorial effects of TpE on plant–pathogen interactions. This should pave the way for maintaining efficient disease resistance under heat stress and for identifying molecular mechanisms that would provide sustainable crop resistance under changing temperature conditions.

II. Round one: plants or pathogens facing heat stress

1. Effect of heat stress on plants

Most higher plants, classified as mesophilic organisms, have an optimum growth in a thermal niche that ranges from 10 to 30°C (Nievola *et al.*, 2017). However, the incidence of a TpE depends on the applied temperature range and on the plant species studied. For instance, for *Arabidopsis thaliana*, according to temperatures applied, heat is defined as warm ambient temperature between 22°C and 27°C, high temperature between 27°C and 37°C and extremely high temperature between 37°C and 42°C (Liu *et al.*, 2015). The effect of TpE on plants has been studied extensively and reviewed recently in several articles (Bita & Gerats, 2013; Liu *et al.*, 2015; Gray & Brady, 2016; Nievola *et al.*, 2017), however we would like to point out that, to date, most studies have investigated the effects of an elevation from 5°C to 10°C or even more than 10°C above the optimum growth temperature, which corresponds to high or extremely high TpEs. Heat stress severely affects plant homeostasis and vital functions. Fig. 1 puts together the main findings obtained for plants and we invite readers to consult listed reviews for more information. At the developmental level, vegetative and reproductive organs are affected (Zinn *et al.*, 2010; Gray & Brady, 2016; Yang *et al.*, 2018; Wang *et al.*, 2018a). Plants exposed to TpE are affected at the cell physiology level during all developmental stages. TpE increases membrane fluidity and permeability leading to: (1) lipid-based signalling cascades that modulate membrane-localised heat-sensing factors; and (2) a re-organisation of cellular structures such as microtubules, organelles and cytoskeleton, affecting cell differentiation, elongation and expansion (Saidi *et al.*, 2011; Bita & Gerats, 2013). TpE interferes

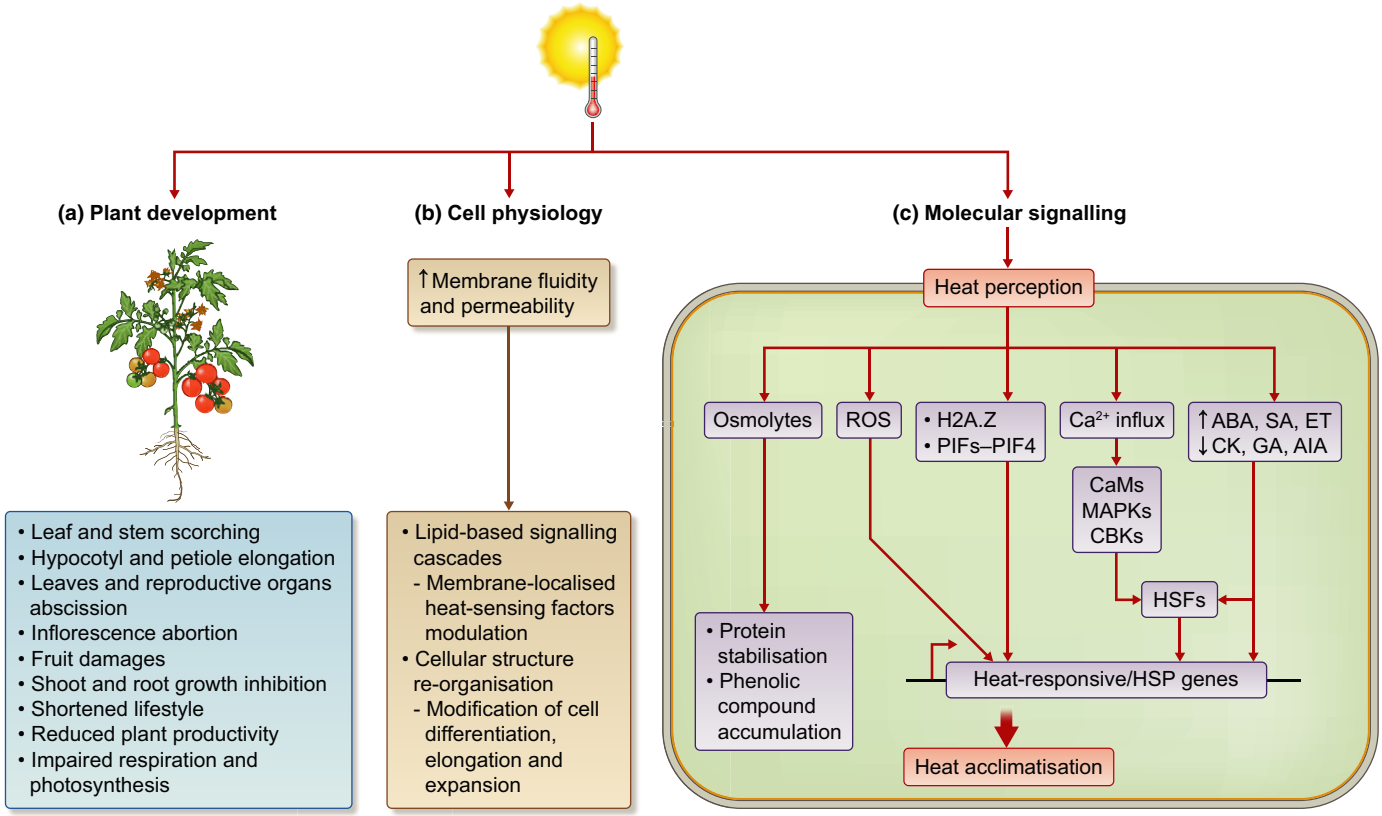

Fig. 1 Effect of elevated temperature on plant development, cell physiology and molecular signalling. (a) Plant development is altered at different levels, affecting different organs such as leaves and reproductive organs (Zinn *et al.*, 2010; Gray & Brady, 2016; Yang *et al.*, 2018; Wang *et al.*, 2018a). (b) TpE affects cell physiology, increasing membrane fluidity and permeability leading to the modulation of membrane-localised heat-sensing factors and to the modification of cell differentiation, elongation and expansion (Saidi *et al.*, 2011; Bita & Gerats, 2013). (c) Heat tolerance is promoted by osmolytes production that help protein stabilisation (Mirzaei *et al.*, 2012) and stimulate phenolic compound accumulation (Wahid *et al.*, 2007). At the cellular level, TpE interferes with many signalling processes involving reactive oxygen species (ROS) (Königshofer *et al.*, 2008), Ca^{2+} influx across the plasma membrane and its signalling through calmodulins (CaMs), mitogen-activated protein kinases (MAPKs) or CaM-binding protein kinases (CBKs) (Saidi *et al.*, 2011), differential accumulation of key hormones, phytochrome-interacting factors (PIFs) that belong to a class of basic helix–loop–helix (bHLH) transcription factors (Huai *et al.*, 2018) and perception of temperature through DNA–nucleosome fluctuations involving histone H2A.Z (Kumar & Wigge, 2010). Together, these actions contribute to the transduction of heat signals and the coordination of temperature-dependent gene transcription leading to heat acclimatisation. ABA, abscisic acid; AIA, auxin; CK, cytokinin; ET, ethylene; GA, gibberellic acid; SA, salicylic acid.

with many signalling processes within the cell. Production of osmolytes is triggered, participating in heat tolerance by helping protein stabilisation (Mirzaei *et al.*, 2012) and stimulating phenolic compound accumulation (flavonoids, anthocyanins, steroids) (Wahid *et al.*, 2007). Signalling pathways are also regulated, and phosphorylation of some proteins is promoted (Larkindale & Vierling, 2008; Saidi *et al.*, 2011). In addition, the biosynthesis and compartmentalisation of metabolites are disturbed (Maestri *et al.*, 2002). Production of ROS contributes to transduction of the heat signal, leading to the regulation of expression of heat shock protein (HSP) genes involved in thermotolerance (Königshofer *et al.*, 2008). Thermosensory signalling also relies on phytochrome-interacting factors (PIFs) that belong to a class of basic helix–loop–helix (bHLH) transcription factors (TFs) (Huai *et al.*, 2018). Among those, PIF4 coordinates temperature-dependent growth of *A. thaliana* plants and negatively regulates plant immunity (Gangappa *et al.*, 2017). In *A. thaliana*, direct perception of temperature occurs through DNA–nucleosome fluctuations that involve alternative histone H2A.Z required for proper coordination of temperature-dependent gene transcription (Kumar & Wigge, 2010). TpE also induces extracellular Ca^{2+} influx across the plasma membrane. Perception of Ca^{2+} fluctuations by calcium sensors, such as calmodulins (CaMs), and the activation of specific mitogen-activated protein kinases (MAPKs) or CaM-binding protein kinases (CBKs), are part of the Ca^{2+}-dependent heat stress downstream signalling pathway that regulates the activity of heat shock transcription factors (HSFs) and expression of *HSP* genes (Saidi *et al.*, 2011). Furthermore, TpE also induces modulation of gene expression in various processes including primary and secondary metabolism, transcriptional regulation, translation and response to environmental stress.

Depending on the plant species and genotypes, TpE can also differentially affect host genome methylation, as exemplified in *A. thaliana* and cotton, whose genomes are hyper- methylated and hypo-methylated under heat stress, respectively (Liu *et al.*, 2015). Furthermore, TpE can trigger the transient activation of repetitive elements or silenced gene clusters and, by contrast, transient inhibition of gene silencing (Lang-Mladek *et al.*, 2010; Pecinka *et al.*, 2010). However, it seems difficult to define a general trend of heat effects on DNA methylation changes in different species (Liu *et al.*, 2015). Finally, TpE can interfere with protein homeostasis in plant cells, leading to the denaturation of some proteins already present or misfolding of newly synthesised proteins (Volkening *et al.*, 2019).

Following a nonlethal exposure to TpE, plants can enhance their ability to cope with and respond more efficiently to a repeated heat stress. This phenomenon is commonly referred to as 'priming' or acquisition of thermotolerance. This rapid, highly conserved, and actively maintained response leads to heat stress memory, recently reviewed in Bäurle (2016) and Friedrich *et al.* (2019). This priming induces HSF and heat shock protein (HSP) expression and stabilisation, thus enhancing protein homeostasis (Finka *et al.*, 2015; Haslbeck & Vierling, 2015). Although the underlying molecular mechanisms remain elusive, the heat shock transcription factor A2 (HsfA2) was shown to regulate heat stress memory genes encoding small HSPs such as HEAT STRESS-ASSOCIATED 32kD PROTEIN (HSA32) or ASCORBATE PEROXIDASE 2 (APX2) (Charng *et al.*, 2007; Liu & Charng, 2012; Lämke *et al.*, 2016). Interestingly, *APX2A* and another *HPS22* loci can be dimethylated or trimethylated with histone H3 lysine 4 (H3K4me2 and H3K4me3) upon heat stress priming, and regulating their ability to be transcribed (Lämke *et al.*, 2016). This phenomenon is linked to HsfA2 and shows how epigenetic markers are affected by heat stress.

2. How do pathogens cope with heat stress?

The overall effect of climate changes on pathogens is difficult to determine as the optimal infection conditions, host specificity and plant responses greatly differ from one pathogen to another (Elad & Pertot, 2014). Temperature is one of the most significant climatic variables for phytopathogen infection, along with relative humidity (Huber & Gillespie, 1992). The main effects of TPE on pathogens at the macroscopic level are summarised in Fig. 2(a). For pathogens that have evolved at higher latitudes, TpE is predicted to improve their fitness and to increase the risk of epidemics due to their adaptation to temperatures below their physiological optimum (Deutsch *et al.*, 2008). The effect of temperature also depends on pathogen trophic behaviour. Indeed, elevated temperatures increase tissue necrosis and favour colonisation by necrotrophic pathogens (Elad & Pertot, 2014). Furthermore, modification of plant physiology under TpE can result in profoundly altered colonisation of host tissues by biotrophic pathogens (Agrios, 2005). Both temperature and relative humidity often govern pathogen reproduction rate (Caffarra *et al.*, 2012). Longer growing seasons due to climate warming will increase the length of pathogen reproduction and dissemination periods. For instance, studying the effect of temperature on life-history traits of the fungal pathogen *Podosphaera plantaginis*, which causes powdery mildew, on *Plantago lanceolata* showed an acceleration of spore germination and a stimulation of spore production at higher temperatures. This suggests that under such conditions, all asexual traits perform better, unlike sexual traits (Vaumourin & Laine, 2018). At the epidemiological level, prolonged periods of optimum temperatures during pathogen development, along with optimum precipitation and/or humidity conditions, increase crop losses (Agrios, 2005). The strongest consequences of global warming on the spread of pathogens are expected to be in regions where the average temperature reaches their optimum growth temperature. Additionally, TpE will probably affect the fitness of pathogen species with a narrower temperature growth range, as such species are expected to be more sensitive to extreme temperature fluctuations (Elad & Pertot, 2014).

Several mechanisms involved in the sensing of thermal fluctuations have been investigated extensively in fungal and bacterial pathogenic species, mainly in humans and mammals. We will present them briefly, as they have already been described in reviews (Shapiro & Cowen, 2012; Lam *et al.*, 2014). Relevant examples that could help to understand the mechanisms involved in plant–pathogen interactions under TpE are presented in Fig. 2(b). Temperature affects developmental transitions, promotes virulence of bacteria and fungi, and influences replication and growth

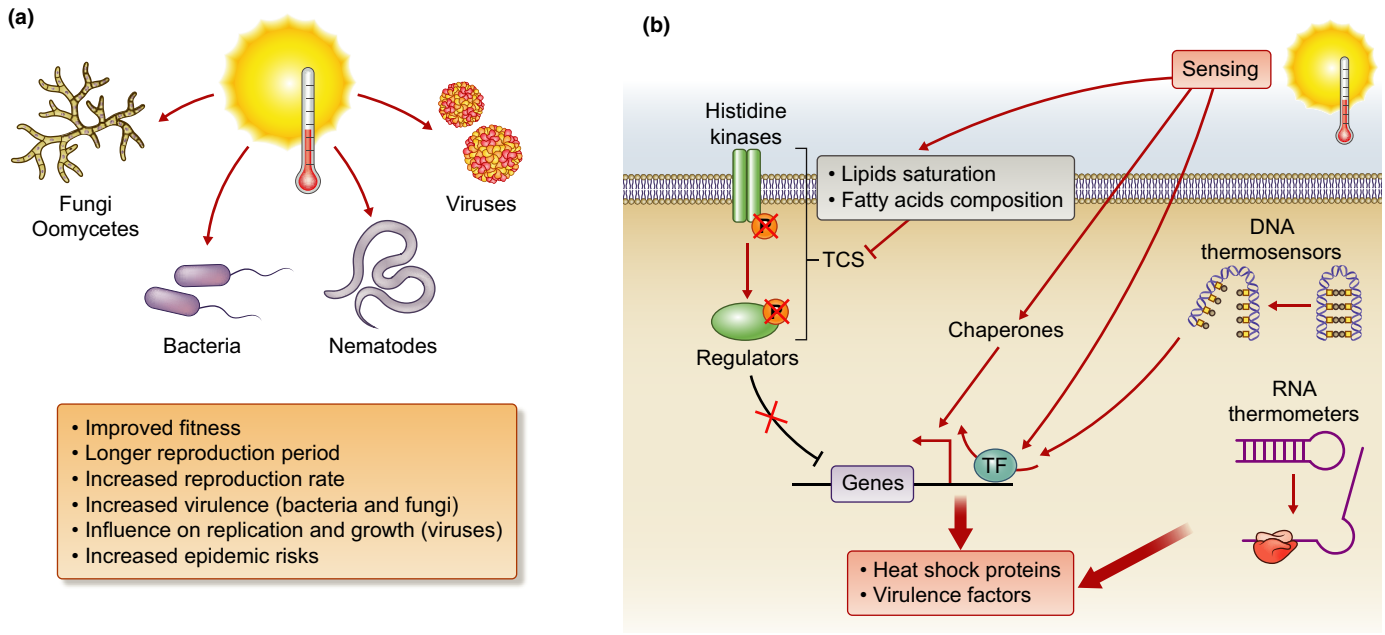

Fig. 2 Effect of elevated temperature on pathogens. (a) Heat stress has an effect at a macroscopic scale, influencing the plant–pathogen life cycle. (b) In many animal pathogens, heat stress can be perceived at the membrane level and through the regulation of various molecular mechanisms involving kinase receptors and DNA-dependent and/or RNA-dependent sensor mechanisms. Red lines indicate TpE-dependent regulation. Blunted and pointed arrows indicate inhibition and activation, respectively. TCS, two-component regulatory system; TF, transcription factor.

properties of viruses that infect human and mice. For mammalian bacterial pathogens, temperature is an indicator of successful host infection as it modulates significant virulence determinants such as the T3SS functions, the delivery of type III effectors (T3Es), flagella motility and the production of toxins and adhesins (Lam *et al.*, 2014). The cellular membrane contributes to sensing temperature fluctuations through different processes. For bacteria, extreme temperatures alter membrane properties, fatty acid composition and the level of unsaturated lipids, thus allowing membranes themselves to act as thermosensors. For instance, change in the membrane lipid composition, through LpxDs acyltransferase activities or the expression level of acyl-lipid desaturases, have been demonstrated to be involved in temperature-dependent remodelling of membrane lipids in *Francisella* bacteria and *Synechocystis* cyanobacteria, respectively (Suzuki *et al.*, 2000; Li *et al.*, 2012). A second mechanism involves transcriptional regulators, kinases and chaperones as temperature sensors (Shapiro & Cowen, 2012). It also includes a two-component regulatory system (TCS), ubiquitous in prokaryotes, and composed of a membrane-anchored sensor like histidine kinase and a cytoplasmic regulator. TCSs have key roles in temperature fluctuation perception and can activate type III and type IV secretion systems, which are considered as major determinants of bacterial virulence. Temperature sensing and regulation mechanisms for plant pathogens remain poorly understood. To date, *Agrobacterium tumefaciens* and *Pseudomonas syringae* TCS have been the most studied (Jin *et al.*, 1993; Braun *et al.*, 2007). In *A. tumefaciens*, autophosphorylation of VirA (sensor) and phosphorylation of VirG (regulator) are both suppressed above 32°C, leading to impaired bacterial virulence (Jin *et al.*, 1993). In *P. syringae*, elevated temperatures trigger conformational changes in CorS (sensor kinase), leading to CorR inactivation (regulator) (Braun *et al.*, 2007). Interestingly, Shapiro & Cowen (2012) suggested that the temperature-dependent modulation of the TCSs from different bacterial species could be part of a mechanism shared by animal and plant pathogens to promote host infection at temperatures described as optimum for the hosts themselves. Other studies have shown that the structure and topology of DNA can also act as thermosensor, either through its supercoiling or the temperature-dependent accessibility of promoter regions occupied by histone-type proteins (Shapiro & Cowen, 2012). Finally, an 'RNA thermometers' mechanism involves temperature-sensitive noncoding RNA regions that are often located in the 5′ untranslated region (5′UTR) of bacterial RNAs. Temperature can either modulate their expression or stability, making ribosome-binding sites accessible and facilitating translation initiation (Shapiro & Cowen, 2012).

III. Round two: effect of heat stress on plant–pathogen interactions

1. Key features of combined heat and pathogenic stresses studies

In recent decades, many review articles have addressed the potential effect of climate change on plant pathogens and diseases (Juroszek *et al.*, 2020). Surprisingly, Juroszek and colleagues noticed a decrease since 2014 in the number of reviews in biological research dealing with the effects of climate changes on plant pathogens and

crop diseases, suggesting a decline of interest in this topic. However, in agreement with the observed effects of global warming and the prediction of its effect on living organisms and ecosystems, the number of studies reporting an alteration of plant disease resistances under TpE has increased tremendously in recent years. Therefore, we decided to review current available knowledge to assess the effect of TpE on plant–pathogen interactions, using 'high temperature, temperature elevation, pathogens, plants, resistance, immune response and combined stresses' as keywords to perform bibliographic searches on the Web of Science, Google Scholar and PubMed–NCBI websites. We selected 45 studies or reviews combining 142 cases of pathogen resistance responses tested under TpE (Tables 1, 2). Among those, 36 pathosystems could be distinguished, corresponding to a combination of 21 plant species (including 20 crop species) with 27 pathogen species (including eight fungi, three oomycetes, three nematodes, three bacteria and nine viruses). Studies describing negatively impacted resistances under TpE are listed in Table 1, whereas heat-stable resistances and those enhanced under TpE are presented in Table 2. We draw attention to the fact that observations must be nuanced because, in some cases, the effect of TpE on other characterised resistance genes is unknown.

Overall, studies are mainly descriptive. Most of the resistance responses were examined under controlled conditions (42 studies). Only two responses, conferred by the *Mi-1* gene in tomato to three nematode species and by the *Xa-7* gene, in rice to *Xanthomonas oryzae* pv *oryzae* (Xoo), respectively, were assessed in both controlled (Jablonska *et al.*, 2007; Cohen *et al.*, 2017) and field conditions over several years (Dropkin, 1969; Webb *et al.*, 2010). Additionally, one study investigated the thermosensitivity of wheat resistances linked to *Lr22b* and *Lr34* genes against *Puccinia recondita*, during 3 years only in field conditions (Plotnikova & Stubei, 2013). The fact that most experiments were carried out in glasshouses or growth chambers allowed easy application and fairly precise control of specific abiotic and biotic factors. However, although much more complex to perform, field experiments, which can take several years to complete, allow the robustness and transferability of the resistance to be assessed under more agro-ecologically realistic conditions. Apart from field studies, depending on how temperature and pathogen stresses were applied, we classified studies into three groups, presented in Fig. 3 and listed in Table 1 and Table 2. Eleven, seven and 23 studies reported simultaneous stress application (Fig. 3a), sequential stress application (Fig. 3b), and acclimatisation (Fig. 3c), respectively. The few remaining studies assessed the effect of TpE using several modes of application of stresses within each study, with no change in the final effect on plant immune response (Gijzen *et al.*, 1996; Djian-Caporalino *et al.*, 1999; Webb *et al.*, 2010).

Strikingly, in 55% of the studied resistances, TpE resulted in an increased plant susceptibility or an inhibition of plant defences (Table 1). The negative effect on plant resistances is not restricted to specific plant species or pathogen species and their related lifestyles. In Table 1, there is a balanced distribution of plant and pathogen species studied. Interestingly, most pathogen species have an optimal growth temperature close to the temperature stress applied. However, there are few exceptions (i.e. Martens *et al.*, 1967; Mayama *et al.*, 1975; Gousseau *et al.*, 1985; Xiao *et al.*, 2003). The mode of application of the stresses was predominantly simultaneous (Fig. 3b). In Table 2, which references positive or neutral effect of TpE on resistance, a high number of studies involved wheat, rice and fungi. This higher proportion, corroborated by Juroszek and colleagues, could be related to the fact that some diseases, such as the wheat stripe rust caused by *Puccinia striiformis* f. sp. *tritici*, were among the first to be studied because of their major economic importance (Chen, 2013; Juroszek *et al.*, 2020). Contrasting with Table 1, the acclimatisation mode (Fig. 3c) is predominant in Table 2. This difference could be explained by the fact that most studies in Table 2 were performed on pathosystems involving pathogenic fungi requiring a plant tissue infection phase prior TpE. Overall, TpE negatively affects all types of resistance responses (PTI, ETI and QDR), although cases of immune response inhibition mainly concern ETI (Table 1). In addition, these data highlight stable defence responses mostly involving QDR (Table 2) (i.e. Uauy *et al.*, 2005; Fu *et al.*, 2009; Ren *et al.*, 2012; Chen, 2013; Zhou *et al.*, 2014; Lu *et al.*, 2014; Aoun *et al.*, 2017; Toa *et al.*, 2018; Feng *et al.*, 2018; Wang *et al.*, 2019b). Interestingly, QDRs were proposed to confer sustainable resistance often efficient against a wide range of pathogen species and also specific to a developmental stage, spatially and temporally regulated, and dependent on the environment (Chen, 2013; Debieu *et al.*, 2016; French *et al.*, 2016).

The high frequency of negatively impacted resistances observed under TpE (Table 1) may be explained by the ability of many pathogens to cope with and to adapt quickly to conditions above the optimal growth temperature of their host plants. Alternatively, in all the maintained resistances to *P. striiformis* f. sp. *tritici* (Table 2), the ability of wheat to grow in a wider range of temperatures and to develop specific resistance responses at higher temperatures gives this crop a great advantage over the pathogen, which has a lower optimum growth temperature. Noteworthy, the effect of TpE on plant resistance could depend on the mode of TpE application. Acclimatisation to TpE for 7 d or more before inoculation with the pathogen led to enhanced resistance (Ge *et al.*, 1998; Cohen *et al.*, 2017; Onaga *et al.*, 2017a). The effect of TpE acclimatisation on ETI in *A. thaliana* is highlighted by different studies. For instance, while Cheng and colleagues applied TpE for 3–6 h on *AvrRpt2* expressing transgenic plants, Menna and colleagues primed plants for 24 h prior to inoculation with *P. syringae* pv *tomato* (*Pst*) strains and delivering either HopZ1a or AvrRpt2 effectors (Cheng *et al.*, 2013; Menna *et al.*, 2015). In these two studies, ETI was affected, but in different ways. Indeed, Cheng and colleagues showed that TpE fully inhibits AvrRpt2-triggered immunity (Cheng *et al.*, 2013). By contrast, Menna and colleagues showed that, after acclimatisation, although HR triggered by *Pst* effectors was suppressed at 28°C, the resistance response remained efficient enough to restrict bacteria multiplication, but to a lower extent compared with 22°C (Menna *et al.*, 2015). Whether these differences are directly related to the mode of TpE application remains to be demonstrated. However, acclimatisation period and priming effects following a chronic and intermittent exposure to abiotic stress are known to allow plants to better resist biotic stress (Hilker *et al.*, 2015). For example, wheat plants exposed to 15 or

Table 1 List of studies carried on plant–pathogen interactions under heat stress with a negative effect on resistance.

Plant species	Optimal vegetative growth temperature	Pathogen species	Pathogen optimal growth temperature	Phylum	Lifestyle[a]	Type of study[d]	Culture conditions[c]	Δ in temperature	Time of TpE exposure	Mode of stress application[b]	Resistance genes	Effect of TpE	References	Mechanism of resistance
Arabidopsis (*Arabidopsis thaliana*)	22–23°C (Suzuki *et al.*, 2014)	*Golovinomyces cichoracearum*	20°C (Ward & Manners, 1974)	Ascomycete	B	M	C	8°C (22°C or 30°C)	Up to 30 d	Seq (B-2)	*RPW8*	HR suppressed	Xiao *et al.* (2003)	ETI
		Peronospora parasitica	20°C (Achar, 1998)	Oomycete	B	M	C	6°C (22°C or 28°C)	Weeks before inoculation	Acc (C-4)	*SNC1*	*SNC1*, *EDS1 and PAD4 expression downregulated*	Yang & Hua (2004)	PTI, ETI
		Pseudomonas syringae pv *tomato* DC3000	28°C (Young *et al.*, 1977)	Proteobacterium	N	M	C	6°C (22°C or 28°C)	1 wk	Acc (C-6)	*RPS2*, *RPM1*, *RPS4*	HR suppressed	Wang *et al.* (2009)	ETI
								6–28°C (4°C to 32°C)	6 h	Sim (A-1)	*RPS2*, *RPM1*	Resistance inhibition HR suppressed	Cheng *et al.* (2013)	ETI
								6–9°C (21°C/24°C or 28°C/30°C)	24 h/4 d	Acc (C-5)	*RPS2*, *ZAR1*	HR suppressed, higher bacterial multiplication	Menna *et al.* (2015)	ETI
		Ralstonia solanacearum	28°C (Huerta *et al.*, 2015)	Proteobacterium	H	M	C	3°C (27°C or 30°C)	10 d	Sim (A-1)	*RPS4/RRS1-R*	Resistance inhibition	Aoun *et al.* (2017)	ETI
Pepper (*Capsicum annuum*)	20–25°C (Saha *et al.*, 2010)	*Phytophthora capsici*	25–28°C (Hausbeck & Lamour, 2004)	Oomycete	H	M	C	12°C/7°C (25°C/18°C or 37°C/25°C)	0 , 1, 3, 5–7 d	Sim (A-1)	na	More symptoms	Lu *et al.* (2017)	QDR
		Paprika mild mottle virus (*PaMMV*)	na	Virus	B	D	C	4°C (24°C→28°C)	5–12 d	Acc (C-1)	L^{1a}	HR less effective, systemic infection	Sawada *et al.* (2004)	ETI
		Tobacco mild green mosaic virus (*TMGMV*)	na	Virus	B	D	C	6°C (24°C→30°C)	5–12 d	Acc (C-1)	L^{1}- L^{2}	HR less effective, systemic infection	Sawada *et al*, 2004	ETI
Pepper (*Capsicum chinense*)		*Tomato spotted wilt virus* (*TSWV*)	na	Virus	B	D	C	10°C (22°C or 32°C→22°C)	9/12 d at 32°C/22°C until 1 month	Seq* (B-2)	*Tsw*	HR suppressed	Moury *et al.*, 1998	QDR
Potato (*Solanum sucrense*, *S. sparsipilum*)		*Potato virus Y* (*PVY*)	na	Virus	B	D	C	37°C (16°C/18°C→19°C/24°C)	Week before inoculation	Acc (C-4)	*Ny*	HR less effective, systemic infection	Valkonen (1997)	ETI
Soybean (*Glycine max*)	12–20°C (Hopper *et al.*, 1997)	*Phytophthora sojae*	25–30°C (Scott *et al.*, 2020)	Oomycete	H	D	C	8°C (25°C or 33°C) 11°C – 19°C (44°C→25°C or 33°C)	48 h	Sim (A-1) Seq (B-3)	*Rps1-a*,*Rps1-b*, *Rps1-c*,*Rps1-d*, *Rps1-k*,*Rps2*, *Rps3-a*,*Rps4*, *Rps5*, *Rps6*	Resistance inhibition, isolate dependent	Gijzen *et al.* (1996)	QDR
Tobacco (*Nicotiana tabacum*)	23.5°C (Yang *et al.*, 2018)	*Cucumber Mosaic Virus* (*CMV*)	na	Virus	B	M	C	4–10°C (18°C or 24°C or 28°C)	28 d	Sim (A-1)	*na*	More symptoms	Zhao *et al.* (2016)	QDR
Tobacco (*Nicotiana benthamiana*)		*Potato virus X* (*PVX*)	28°C (Choi *et al.*, 2017)	Virus	B	D	C	6–8°C (22°C→22°C or 28°C or 30°C)	Up to 4 d	seq (B-2)	*Rx*	HR suppressed	Wang *et al.* (2009)	ETI
Tobacco (*Nicotiana rustica*, *N. acuminata*, *N. glutinosa*)		*Tobacco mosaic virus* (*TMV*)	24°C (Lebeurier & Hirth, 1966)	Virus	B	D	C	6–8°C (22°C→22°C or 28°C or 30°C)	3 d	seq (B-2)	*N*	HR suppressed	Wang *et al.* (2009)	ETI
						M		9°C (21°C or 30°C)	5 d	Seq (B-2)		HR suppressed	Whitham *et al.* (1994)	

Table 1 (Continued)

Plant species	Optimal vegetative growth temperature	Pathogen species	Pathogen optimal growth temperature	Phylum	Lifestyle[a]	Type of study[d]	Culture conditions[c]	Δ in temperature	Time of TpE exposure	Mode of stress application[b]	Resistance genes	Effect of TpE	References	Mechanism of resistance
Tomato (*Solanum lycopersicum esculentum*)	18–25°C (Hussey, 1965; Schwarz *et al*., 2014)	*Cladosporium fulvum*	22°C (Gravesen, 1979)	Ascomycete	B	D	C	13°C (20°C or 33°C)	3 wk	Sim (A-1)	*Cf4*, *Cf9*	HR suppressed	de Jong *et al*. (2002)	ETI
Tomato (*Solanum lycopersicum*)		*Meloidogyne javanica*, *M. incognita*, *M. arenaria*	22–28°C (Tyler, 1933)	Nematode	B	M	C	8°C (24°C or 32°C)	6 d	Sim (A-1/A-2)	*Mi-1*	HR suppressed	Hwang *et al*. (2000)	ETI
Tomato (*Solanum arcanum*, *Solanum lycopersicum*)		*Meloidogyne incognita*	22–28°C (Tyler, 1933)	Nematode	B	D	C/F	6–8°C (32°C→ 22°C or 26°C)	2–3 d/until 4 wk/3 wk	Acc (C-6) Field	*Mi-1*	Resistance inhibition	Dropkin (1969), Jablonska *et al*. (2007)	ETI
Tomato (*Solanum lycopersicum peruvianum*)							C	7°C (25°C or 32°C)	7 d/30 d	Acc (C-4)	*Mi-7*, *Mi-8*	Resistance inhibition	Veremis & Roberts (1996)	ETI
Tomato (*Lycopersicum esculentum*)		*Ralstonia solanacearum*	28°C (Huerta *et al*., 2015)	Proteobacterium	H	D	C	8°C (24°C or 32°C)	10–12 d	Sim (A-1/A-2)	*Polygenic Resistances*	Resistance inhibition	Krausz & Thurston (1975)	QDR
Maize (*Zea mays*)	20–25°C (Hardacre & Turnbull, 1986)	*Puccinia sorghi*	25°C (Dey *et al*., 2015)	Basidiomycete	B	D	C	4°C (26°C→ 30°C)	Up to 3 wk	Sim (A-1/A-2)	*Rp1-D21*	HR suppressed	Negeri *et al*. (2013)	ETI
Oat (*Avena sativa*)	20–25°C (Hopper *et al*., 1997)	*Puccinia graminis* f.sp. *avenae*	20–25°C (Martens *et al*., 1967)	Basidiomycete	B	D	C	5–10°C (20–24°C→15°C or 20°C or 25°C or 30°C)	28 h–30 h/Na	Acc (C-1)	*H*, *F*, *B*, *E*	Resistance inhibition	Martens *et al*. (1967)	ETI
Rice (*Oryza sativa* L. ssp. *japonica* cv Nipponbare)	20–27°C (Wang *et al*., 2018a)	*Magnaporthe oryzae*	28°C (Uddin *et al*., 2002)	Ascomycete	B	M	C	8°C (28°C→ 35°C)	7 d/7 d	Acc (C-5)	*Pi1*, *Pi11(t)*, *Pi12 (t)*, *Pi19*, *Pi20*, *Pi7(t)*, *Pia*, *Pib*, *Pi-h*, *Pik*, *Pik-m*, *Pik-p*, *Pik-s*, *Pish*, *Pit*, *Pita*, *Pita2 (Pi No.4)*, *Piz*, *Piz5*, *Pl3*, *Pi5(t)*	Resistance inhibition	Onaga *et al*. (2017b)	QDR
Rice (*Oryza sativa*)		*Xanthomonas oryzae* pv *oryzae*	25–30°C (Sullivan *et al*., 2011)	Proteobacterium	B/H	D	C	Controlled: 6°C (29°C/21°C or 35°C/31–27°C)	21–28 d	Acc (C-3)	*Xa3*, *Xa4*, *Xa5*, *Xa10*	Resistance less effective	Webb *et al*. (2010)	QDR

Table 1 (Continued)

Plant species	Optimal vegetative growth temperature	Pathogen species	Pathogen optimal growth temperature	Phylum	Lifestyle[a]	Type of study[d]	Culture conditions[c]	Δ in temperature	Time of TpE exposure	Mode of stress application[b]	Resistance genes	Effect of TpE	References	Mechanism of resistance
Wheat (*Triticum aestivum*)	20–25°C (Acevedo *et al.*, 2002)	*Puccinia graminis* f.sp. *tritici*	15–23°C (Burrage, 1970)	Basidiomycete	B	D	C	7°C (26°C→ 19°C)	48 h	Acc (C-4)	*Sr6*	Resistance inhibition, isolate dependent	Mayama *et al.* (1975), Harder *et al.* (1979)	ETI
								3–15°C (15°C or 18°C or 22°C or 26°C or 30°C)	na	Seq (B-2)	*Sr15*	More symptoms	Gousseau *et al.* (1985)	ETI
		Puccinia recondita also known as *P. triticina*	10–22°C (Kramer & Eversmeyer, 1992)	Basidiomycete	B	D	C	15°C (10°C or 25°C)	na	Seq (B-2)	*Lr10*, *LrB*, *LrEch*, *Lr22a*, *Lr30*, *RL6057*, *RL6058*, *RL6059*, *Lr11*, *Lr12*, *Lr13*, *Lr14a*, *Lr14b*, *Lr18*	Resistance inhibition, isolate dependent	Dyck & Johnson (1983)	QDR
							F	Field HT (20°C) vs MT (16°C) (3 yr)	Until 9 d	Field	*Lr22b*, *Lr34/Yr18*	Resistance inhibition	Plotnikova & Stubei (2013)	QDR
		Triticum mosaic virus (*TriMV*)	na	Virus	B	D	C	6°C (18°C or 24°C)	28 d	Sim (A-1/A-2)	*Wsm3*	Symptoms on plants	Liu *et al.* (2011)	na
		Wheat streak mosaic virus (*WSMV*)	na	Virus	B	D	C	6°C (18°C or 24°C)	28 d	Sim (A-1/A-2)	*Wsm1*,*Wsm2*	Symptoms on plants Isolate dependent	Liu *et al.* (2011), Seifers *et al.* (2013)	na
								2°C (18°C or 20°C)	Up to 21 d					

ETI, effector-triggered immunity; HR, hypersensitive response; na, not available; P, pathogen; PTI, PAMP-triggered immunity; QDR, quantitative disease resistance; TP, temperature; TpE, temperature elevation.

In this table, the optimum temperature for plant and pathogen species is given along with the mode of stress applied presented in Fig. 3, resistance gene and mechanism of resistance if known.

[a]B, biotrophic; H, hemibiotrophic; N, necrotropic.

[b]Seq, sequential (Tp, temperature stress; p, pathogen stress); Sim, simultaneous stress; Acc, acclimatisation; Nat, natural.

[c]C, controlled; F, field.

[d]M, mechanistic; D, descriptive.

*Experiment considered repeated twice.

Table 2 List of studies carried on plant-pathogen interactions under heat stress with a neutral or positive effect on resistance.

Plant species	Optimal vegetative growth temperature	Pathogen species	Pathogen optimal growth temperature	Phylum	Lifestyle[a]	Type of study[d]	Culture conditions[c]	D in temperature	Time of TPE exposure	Mode of stress application[b]	Resistance genes	Effect of TPE	References	Mechanism of resistance
Arabidopsis (*Arabidopsis thaliana*)	22–23°C (Suzuki *et al.*, 2014)	*Ralstonia solanacearum*	28°C (Huerta *et al.*, 2015)	Proteobacteria	H	M	C	**3°C** (27°C→30°C)	10 d	Sim (A-1)	*SSL4-SSL5*	Heat stable resistance	Aoun *et al.* (2017)	QDR
Tomato (*Solanum arcanum*, *Solanum lycopersicum*)	18–25°C (Hussey, 1965; Schwarz *et al.*, 2014)	*Meloidogyne incognita*	22–28°C (Tyler, 1933)	Nematode	B	D	C	**6–8°C** (32°C→22°C, 26°C)	2–3 d / till 4 weeks / 3 weeks	Acc (C-6)	*Mi-9*	Heat stable resistance	Jablonska *et al.* (2007)	ETI
Tomato (*Solanum lycopersicum peruvianum*)						D	C	**7°C** (25°C or 32°C)	7 d/30 d	Acc (C-4)	*Mi-2*, *Mi-3*, *Mi-4*, *Mi-5*, *Mi-6*	Heat stable resistance	Veremis & Roberts (1996)	ETI
Pepper (Capsicum annuum)	20–25°C (Saha *et al.*, 2010)	*Meloidogyne javanica*, *M. incognita*, *M. arenaria*, *M. Hapla*	22–28°C (Tyler, 1933)	Nematode	B	D	C	**10–20°C** (32°C or 42°C)	7 to 28 d	Sim (A-2) Acc (C-1 & C-4)	*Me1-Me3*	Heat stable resistance	Djian-Caporalino *et al.* (1999)	ETI
		Tobacco mild green mosaic virus (TMGMV)	na	Virus	B	D	C	**6°C** (24°C → 30°C)	7 to 14 d	Seq (B-2)	L^{1a}	Heat stable resistance	Sawada *et al.* (2004)	ETI
Soybean (*Glycine max*)	12–20°C (Hopper *et al.*, 1997)	*Phytophthora sojae*	25–30°C (Scott *et al.*, 2020)	Oomycete	H	D	C	**8°C** (25°C or 33°C)	48 h	Sim (A-1/A-2)	*Rps1-c*, *Rps2*, *Rps5*	Heat stable resistances, isolate dependent	Gijzen *et al.* (1996)	QDR
Rice (*Oryza sativa-indica*, *O. sativa-japonica*)	20–27°C (Wang *et al.*, 2018a)	*Magnaporthe oryzae*	28°C (Uddin *et al.*, 2002)	Ascomycete	B	M	C	**8°C** (28°C or 35°C)	7 d / 10 d	Acc (C-5)	*Pi54*	Enhanced resistance, lower expression of *Pi54* at HT	Onaga *et al.* (2017a)	ETI
Rice (*Oryza sativa* L. ssp. Japonica cv. Nipponbare)								**8°C** (28°C or 35°C)	7 d /7 d	Acc (C-5)	*Pi9*, *Pii*, *Pik-h*, *Pita2*, *Piz-t*	Enhanced resistance	Onaga *et al.* (2017b)	QDR
Rice (*Oryza sariva*)		*Xanthomonas oryzae* pv. *oryzae*	25–30°C (Sullivan *et al.*, 2011)	Proteobacteria	B / H	D/M	C/F	**Controlled: 6°C** (29°C/ 21°C→35°C/31–27°C) **Field: Cool season** 29°C **Hot season** 33°C	14–21 d/Two seasons	Acc (C-3) Nat	*Xa7*	Enhanced resistance	Webb *et al.* (2010) Cohen *et al.* (2017)	ETI
Oat (*Avena sativa*)	20–25°C (Hopper *et al.*, 1997)	*Puccinia graminis* f. sp. *avenae*	20–25°C (Martens *et al.*, 1967)	Basidiomycete	B	D	C	**5–10°C** (20–24°C→ 15°C or 20°C or 25°C or 30°C)	28 –30 h / na	Acc (C-1)	*A*, *D*	Heat stable resistance	Martens *et al.* (1967)	ETI
Wheat (*Triticum aestivum*)	20–25°C (Acevedo *et al.*, 2002)	*Blumeria graminis* f. sp. *tritici*	20°C (Ward & Manners, 1974)	Ascomycete	B	D	C	**10°C** 15°C→15°C or 25°C / 25°C→15°C or 25°C	na	Acc (C-3)	*Pm1*, *Pm8*, *Pm4a*, *Pm4b*	*Pm1* and *Pm8* heat stable, *Pm4a-b* enhanced resistance	Ge *et al*. (1998)	QDR
		Puccinia graminis f. sp. *tritici*	15–23°C (Burrage, 1970)	Basidiomycete	B	D	C	**3– 15°C** 15°C →15°C or 18°C or 22°C or 26°C or 30°C	12 h / na	Seq (B-2)	*Sr14*, *Sr9b*	Less symptoms	Gousseau *et al.* (1985)	ETI
		Puccinia recondita also known as *P. triticina*	10–22°C (Kramer & Eversmeyer, 1992)	Basidiomycete	B	D	C	**15°C** (10°C or 25°C)	na	Acc (C-1)	*Lra*, *Lr2a*, *Lr2b*, *Lr2c*, *Lr3*,*Lr3bg*, *Lr1*, *Lr3ka*, *Lr9*, *Lr15*, *Lr16*, *Lr17*, *Lr19*, *Lr21*, *Lr23*, *Lr24*	Heat stable resistance	Dyck & Johnson (1983)	QDR
							F	**Field HT (20°C) vs MT (16°C)** (3 yr)	Until 9 d	Field	*Lr37*	Heat stable resistance	Plotnikova & Stubei (2013)	QDR

Table 2 (Continued)

Plant species	Optimal vegetative growth temperature	Pathogen species	Pathogen optimal growth temperature	Phylum	Lifestyle[a]	Type of study[d]	Culture conditions[c]	D in temperature	Time of TPE exposure	Mode of stress application[b]	Resistance genes	Effect of TPE	References	Mechanism of resistance
							C	**4–7°C** (18°C → 22°C or 25°C)	24 h /14 d	Acc (C-2)	*LrZH22*	Heat stable resistance	Wang *et al.* (2016)	QDR
		Puccinia striiformis f. sp. *tritici*	13–16°C (Newton & Johnson, 1936)	Basidiomycete	B	M	C	**6–10°C** 10°C → diurnal cycle 4°C/20°C (seedling test) or 10°C / 30°C (adult test)	24 h / several d	Acc (C-1)	*Yr18, Yr29, Yr34, Yr36, Yr39, Yrxy1, Yrxy2, YrR61, YrQ1, YrQ2, YrQ3, YrQ4, Yr52*	HTAP temperature sensitive resistance	Chen (2013)	QDR
						M	C	**5–10°C** 10°C→15°C or 20°C	24 h / 24 h to 8 d	Acc (C-1)	*TaXa21*	HTSP temperature sensitive resistance	Toa *et al.* (2018) Wang *et al.* (2019b)	PTI/QDR
						D	C	**6–10°C** 10°C → diurnal cycle 4°C/20°C (seedling test) or 10°C / 30°C (adult test)	24 h / 18–22 d	Acc (C-1)	*Yr79*	HTAP temperature sensitive resistance	Feng *et al.* (2018)	QDR
						D	C	**6–10°C** 10°C → diurnal cycle 4°C/20°C (seedling test) or 10°C / 30°C (adult test)	24 h / 20–25 d	Acc (C-1)	*Yr62*	HTAP temperature sensitive resistance	Lu *et al.* (2014)	QDR
						D	C	**6–10°C** 10°C → diurnal cycle 4°C/20°C (seedling test) or 10°C / 30°C (adult test)	24 h / 18–21 d	Acc (C-1)	*Yr59*	HTAP temperature sensitive resistance	Zhou *et al.* (2014)	QDR
						D	C	**6–10°C** 10°C → diurnal cycle 4°C/20°C (seedling test) or 10°C / 30°C (adult test)	24 h / 18–22 d	Acc (C-1)	*Yr52*	HTAP temperature sensitive resistance	Ren *et al.* (2012)	QDR
Wheat (*Triticum turgidum* L. ssp. *Durum*)		*Puccinia striiformis* f. sp. *tritici*	13–16°C (Newton & Johnson, 1936)	Basidiomycete		D	C/F	**10°C** 10°C→diurnal cycle 10°C / 25°C or 10°C / 35°C **Field:** High diurnal temperatures followed by cool nights	24 h / na	Acc (C-1) Field	*Yr36*	HTAP resistance	Uauy *et al.* (2005) Fu *et al.* (2009)	QDR
Wheat (*Triticum aestivum*)		*Wheat streak mosaic virus (WSMV)*	na	Virus	B	D	C	**6°C** (18°C or 24°C)	28 d	Sim (A-1/A-2)	*wsm3*	Heat stable resistance	Liu *et al.* (2011)	na
						D	C	**2°C** (18°C or 20°C)	up to 21 d	Sim (A-1/A-2)	Temperature-sensitive resistance (TSR)	Heat stability isolate dependent	Seifers *et al.* (2013)	na

In this table, the optimal temperature for plant and pathogen species is given along with the mode of stress applied presented in Figure 3, resistance gene and mechanism of resistance if known HR, Hypersensitive Response; na, not available; P, Pathogen; TP, Temperature; TPE, temperature elevation; PTI, PAMP triggered immunity; ETI = effector triggered immunity, QDR = quantitative disease resistance.

[a]B, Biotrophic; H, Hemibiotrophic; N, Necrotropic

[b]Seq, Sequential (Tp, Temperature stress ; P, Pathogen stress) ; Sim, Simultaneous stress ; Acc, Acclimatization ; Nat, Natural ; Comb, Combined

[c]C, Controlled; F, Field

[d]M, Mechanistic; D, Descriptive

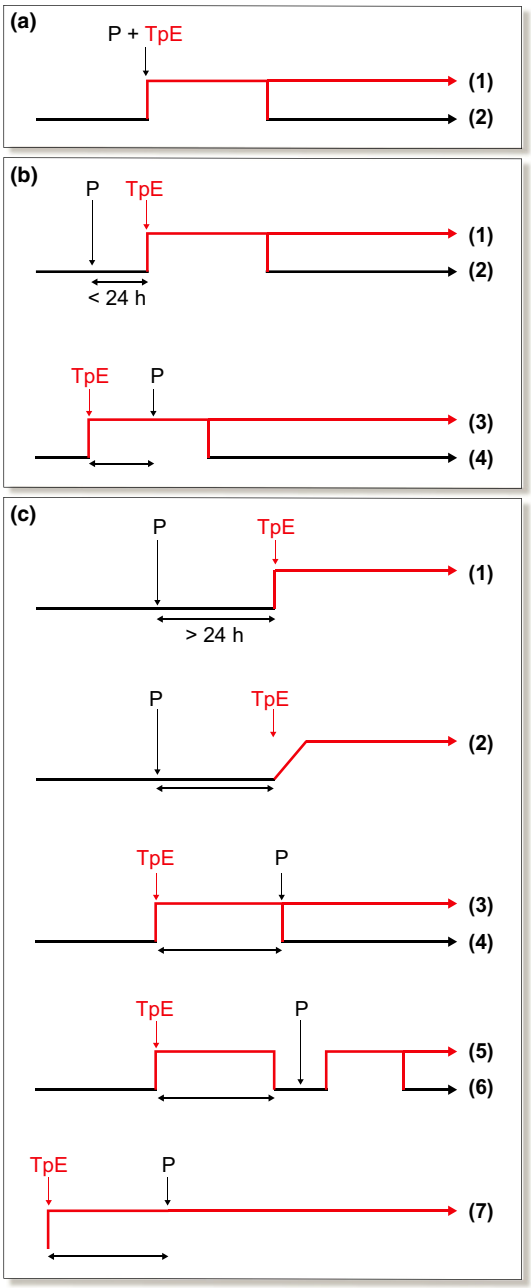

Fig. 3 Classification of the different modes of stress application, for temperature elevation and pathogen inoculation, described in the studies listed in Tables 1 and 2. (a) In a simultaneous mode of stress application, both stresses are applied at the same time or at least within < 1 h. (b) Sequential stress application: both stresses are applied one after the other over a period of time <24 h. (c) Acclimatisation studies: both stresses are applied one after the other with a >24 h delay between each application. (c2) denotes a gradual transition from growth temperature to stressful temperature with one or more transition temperature thresholds. In each class, the experimental design is described, differing according to the duration or cycle of heat stress and in the order in which both stresses are applied. The numbers to the right of the figure illustrate different experimental designs found in the studies for each class. P, pathogen inoculation; TpE, temperature elevation.

25°C until the booting stage prior to inoculation with *Blumeria graminis* f. sp. *tritici*, were more resistant. Expression of *Pm4a* and *Pm4b* resistance genes was correlated with the applied temperature before inoculation, with resistance being more efficient at 25°C (Ge *et al.*, 1998). Wang and colleagues also reported that the expression of some race-specific *R* genes, such as *Pib* rice-blast resistance genes, could be primed by abiotic stresses including fluctuating temperature or light and water availability (Wang *et al.*, 2001).

Although TpE interferes with various plant resistance responses, the underlying molecular mechanisms remain poorly understood. Throughout the different studies, most of the investigations were performed at the transcriptomic level (Chen *et al.*, 2013; Prasch & Sonnewald, 2013; Rasmussen *et al.*, 2013; Cohen *et al.*, 2017; Huot *et al.*, 2017; Onaga *et al.*, 2017a; Toa *et al.*, 2018). Only a few studies have assessed in detail how TpE affects plant immune responses, mainly NLR-dependent autoimmune responses and specifically on *A. thaliana* interacting with *Pst* (Zhu *et al.*, 2010; Mang *et al.*, 2012; Cheng *et al.*, 2013; Menna *et al.*, 2015; MacQueen & Bergelson, 2016; Huot *et al.*, 2017).

2. Transcriptome specificities in response to combined heat and pathogenic stress

Several studies have pointed out commonalities and differences between plant transcriptome analyses in response to individual and combined stresses (Atkinson & Urwin, 2012; Suzuki *et al.*, 2014; Pandey *et al.*, 2015; Zhang & Sonnewald, 2017). Based on previously published transcriptomic studies, whatever the pathogen species considered, the range of TpE used or the way both biotic and abiotic stresses were applied, two groups of studies could be distinguished.

The first group concerns studies in which disease susceptibility is increased under TpE (Prasch & Sonnewald, 2013; Rasmussen *et al.*, 2013; Huot *et al.*, 2017). Prasch and Sonnewald investigated the effect of a moderate TpE (increase of 4°C) applied after challenging plants with *Turnip mosaic virus* (*TuMV*) on *A. thaliana* immunity (Prasch & Sonnewald, 2013). Huot and collaborators studied the effect of TpE on plants infected with *Pst* DC3000 at 23°C or 30°C, focusing on the salicylic acid (SA)-mediated plant response. In particular, they showed a drastic negative effect of TpE on plants immunity, with an increased susceptibility dependent on the highest temperature applied after inoculation (Huot *et al.*, 2017). By contrast, Rasmussen and colleagues compared the responses of various *A. thaliana* genotypes exposed for 3 h either to TpE (24°C to 38°C) or to the bacterial flagellin peptide 22 (flg22), or both (Rasmussen *et al.*, 2013).

The second group corresponds to studies investigating the molecular bases of thermostable resistances. In rice, *Xa7*-mediated and *Pi54*-mediated resistance to *X. oryzae* pv *oryzae* (*Xoo*) and *Magnaporthe oryzae* (*Mo*), respectively, were defined as thermostable as they are unaffected by TpE (Cohen *et al.*, 2017; Onaga *et al.*, 2017a). In wheat, the *TaXa21* resistance gene mediates high temperature seedling plant (HTSP) resistance, whereas *Yr18, Yr29, Yr36* and *Yr39* genes confer high temperature adult plant (HTAP) resistance to *P. striiformis* f. sp. *tritici*, (Chen *et al.*, 2013; Toa *et al.*, 2018; Wang *et al.*, 2019b). We provide a general overview in Fig. 4 of the main regulated genes or pathways for combined TpE and pathogen inoculation stress in both types of studies. In total, five main observations could be made:

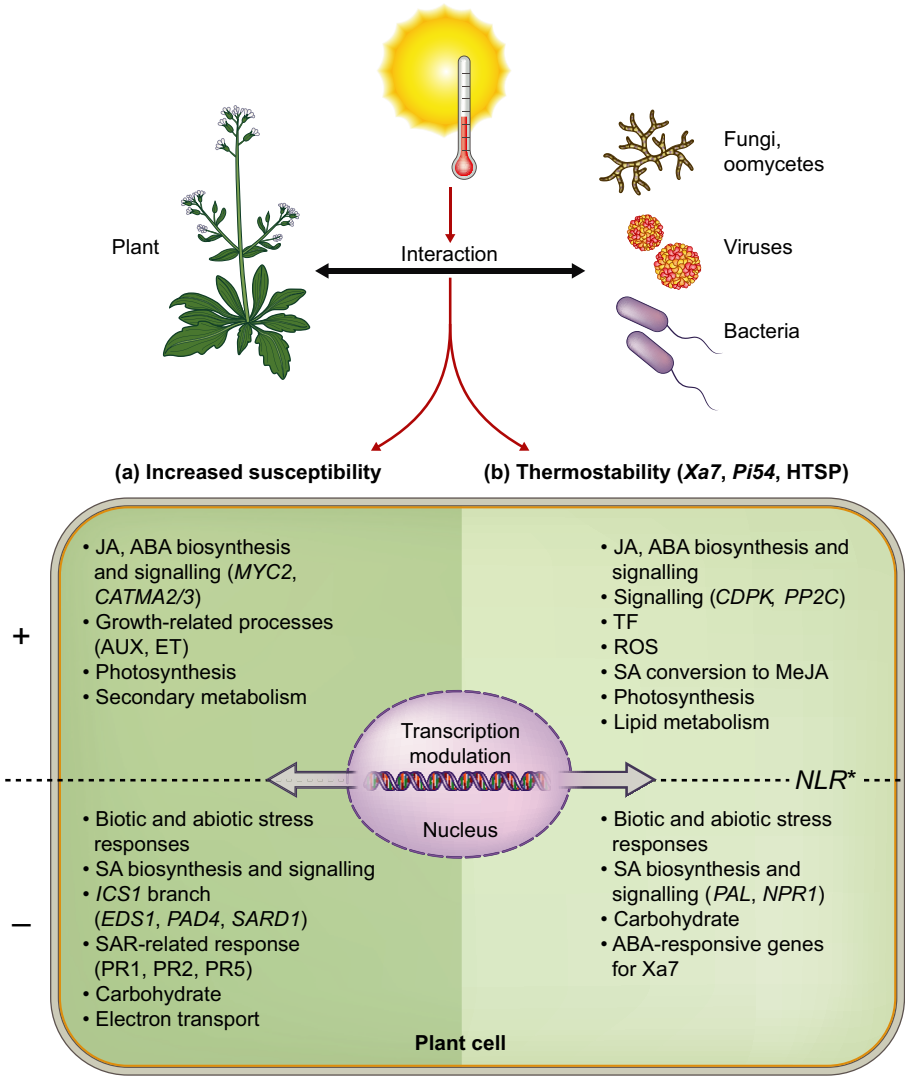

Fig. 4 Main pathways and genes positively and negatively regulated at the plant level and related to increased susceptibility or resistance stability upon combined stress. The grey arrows on either side of the nucleus indicate genes or pathways that are specifically induced or repressed during combined TpE and pathogenic stress in the different transcriptome studies on: (a) increased susceptibility to disease in *Arabidopsis thaliana* (Prasch & Sonnewald, 2013; Rasmussen *et al*., 2013; Huot *et al*., 2017); and (b) thermostable resistance (Chen *et al*., 2013; Cohen *et al*., 2017; Onaga *et al*., 2017a; Toa *et al*., 2018; Wang *et al*., 2019b). *, Different sets of *NLR* genes are differentially regulated in combined stress compared with single-stress treatment. ABA, abscisic acid; AUX, auxin; ET, ethylene; JA, jasmonic acid; MeJA, methyl jasmonate; NLR, nucleotide-binding oligomerisation domain (NOD)-like receptors; ROS, reactive oxygen species; SA, salicylic acid; SAR, systemic acquired resistance; TFs, transcription factors.

(**1**) The plant transcriptional response induced by combined stresses is specific and unpredictable from an individual stress application.

(**2**) Few common responses can be identified between individual and combined stresses, involving the activation of TFs and stress-responsive genes, while photosynthetic and primary carbon metabolism-related genes are downregulated (Rasmussen *et al*., 2013; Pandey *et al*., 2015; Suzuki & Katano, 2018).

(**3**) The number of differentially regulated genes is significantly higher under combined stresses. This observation associated with the weak overlap between genes deregulated in individual and combined stresses suggests that the application of both biotic and abiotic constraints exerts an extreme change to the plant that effects different sets of genes.

(**4**) Plant response to combined stresses is close to the response to the most severe individual stress or to the latest stress applied. For instance, for thermostable *Pi54*-mediated resistance to *Mo* in rice, transcriptomic responses of a rice genotype pretreated for 7 d at either at 28 or 35°C prior *Mo* inoculation were very similar, suggesting that the plant response to *Mo* was mostly driven by the latest stress applied (Onaga *et al*., 2017a).

(**5**) Signalling networks involving NRL or LRR receptor-like kinase (RLK) proteins, serine/threonine protein kinases or specific TFs seem to be critical for stable resistance responses under combined TpE and pathogen attack (Chen *et al*., 2013; Cohen *et al*., 2017; Onaga *et al*., 2017a; Toa *et al*., 2018). Indeed, Wang and collaborators recently demonstrated the involvement of wheat TaXa21 LRR-RLK in HTSP resistance to *P. striiformis* f. sp. *tritici*.

TaXa21 interacts with TaWRKY76 and TaWRKY62, two WRKY TFs that positively regulate HTSP resistance (Wang *et al.*, 2019b). In *Caspicum annuum*, infection with *Ralstonia solanacearum* under TpE (42°C for 24 h), activates the expression of both CaWRKY40 and CaWRKY46 TFs, which promote the activation of defence-related genes and genes encoding heat shock proteins such as NtHSF2. These observations suggest that these two WRKY TFs are involved in defence responses to *R. solanacearum* and tolerance to TpE (Dang *et al.*, 2013; Cai *et al.*, 2015).

3. Mechanisms involved in the thermosensitivity of immune responses

To date, few studies have been conducted to elucidate how TpE modulates plant immunity. They focused on a few of the well studied models and the knowledge acquired so far mainly concerned the plant side, although it is obvious that TpE can also modulate pathogen virulence. Well known molecular agents playing a role in the modulation of immune responses under TpE (Fig. 5) are detailed in the following sections.

Plant determinants related to thermosensitive immunity Several hypotheses give some clues on putative causal factors that lead to the thermosensitivity of plant immunity. Combined TpE and pathogen attack inhibits the plant defence through modulation of immune-related gene expression. Few studies have addressed this aspect for PTI-mediated resistance and the TpE-dependent effect on defence gene expression appears to be controversial (Fig. 5a). Two studies that were performed in *A. thaliana* reported the repression of systemic acquired resistance (SAR)-related gene

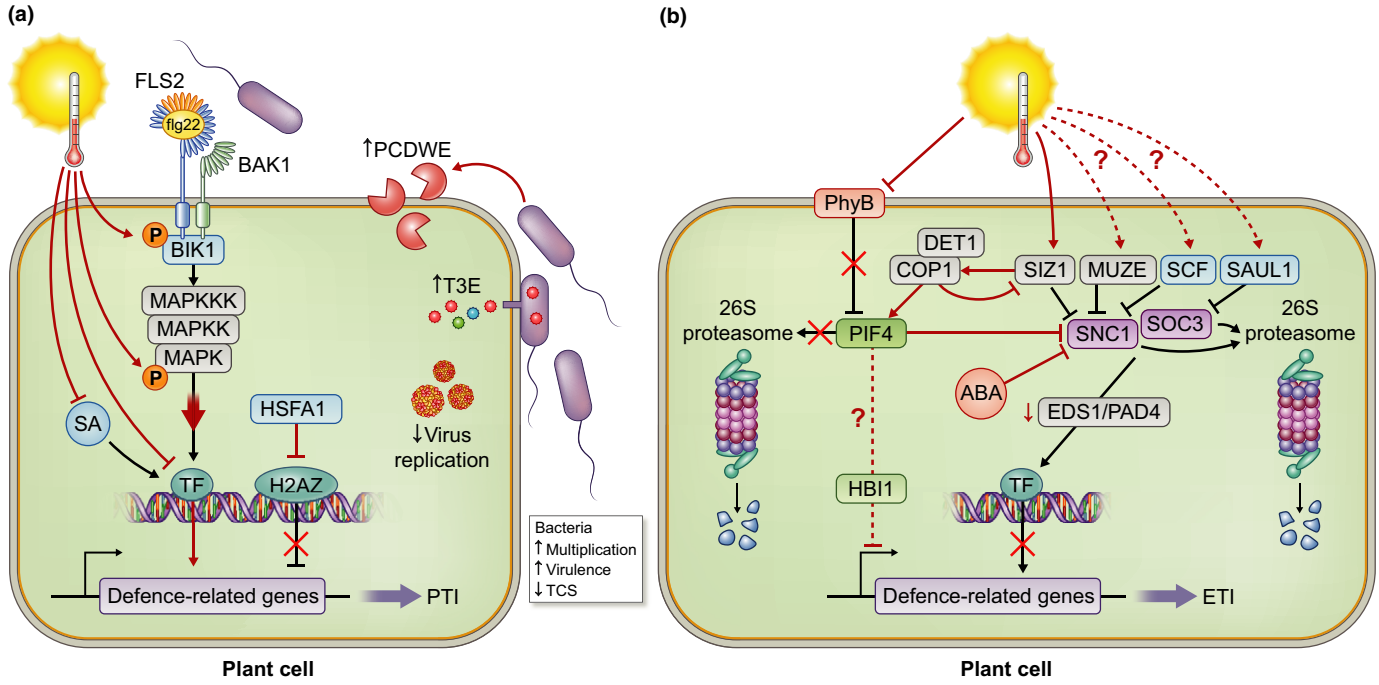

Fig. 5 Schematic model of temperature elevation–interference with plant immune responses and pathogen virulence. (a) TpE interference with PAMP-triggered immunity (PTI) signalling and phytopathogens. At elevated temperature, on the plant side, perception of flg22 by the FLS2 membrane receptor leads to increased BIK1 and MAPK phosphorylation, enhancing expression of downstream PTI-related genes (Cheng *et al.*, 2013). Alternatively, TpE represses *FLS2* expression, salicyclic acid (SA) biosynthesis and signalling and SAR-related gene expression. Also, TpE impairs PTI-related ROS production and decreases SA levels (Rasmussen *et al.*, 2013; Huot *et al.*, 2017; Janda *et al.*, 2019). At the chromatin level, TpE promotes the rapid replacement of H2A.Z, mediated by HSFA1, allowing the expression of heat-responsive and defence-related genes (Cheng *et al.*, 2013; Cortijo *et al.*, 2017). On the pathogen side, TpE can increase bacterial virulence, for example by stimulating the production of bacterial plant cell wall-degrading enzymes (PCDWE) (Hasegawa *et al.*, 2005). TpE enhances the secretion of type III effectors (Huot *et al.*, 2017). The thermosensory two-component system (TCS) is affected, inhibiting DNA transfer delivery by *Agrobacterium* spp. and decreasing the production of virulence factors such as coronatine for *P. syringae* (Shapiro & Cowen, 2012). Accumulation of viruses in plant cells is repressed (Chung *et al.*, 2015). (b) Effect of elevated temperature on ETI-related signalling. At ambient temperature, immune response involving SNC1 NLR protein relies on EDS1 and PAD4. In the absence of pathogen, EDS1 is sequestered by PAD4 in the cytoplasm. Upon pathogen perception, EDS1 is released from PAD4 and translocated to the nucleus and triggers transcriptome reprogramming, leading to the activation of the plant defence response. SIZ1 interferes with SNC1 activation at the transcriptional and/or the protein level (Hammoudi *et al.*, 2018). Heat stress promotes sumoylation of COP1 by SIZ1, which in turn results in ubiquitination and degradation of SIZ1. Under TpE, DET1 and COP1 increase the activity of PIF4 by controlling both its transcription and its protein levels. SNC1 autoactivation, observed in the autoimmune *snc1-1* mutant, is suppressed by PIF4 at elevated temperatures (Sreeramaiah *et al.*, 2018). PhyB negatively regulates PIF4 and promotes its degradation at ambient temperature. Heat stress inactivation of PhyB enables PIF4 accumulation and indirect repression of defence-related genes by HBI1 TF (Gangappa *et al.*, 2017). The SCF E3 ligase complex and MUSE proteins are responsible for temperature-dependent SNC1 degradation (Cheng *et al.*, 2011; Copeland *et al.*, 2016). The same mechanisms are described for SAUL1 and the NLR SOC3 (Disch *et al.*, 20120166; Tong *et al.*, 2017). Enhanced accumulation of abscisic acid (ABA) at elevated temperatures contribute to NLR translocation from the nucleus to the cytosol, leading to resistance inhibition (Mang *et al.*, 2012). Red lines indicate TpE-dependent regulations. Blunted and pointed arrows indicate inhibition and activation, respectively. Dashed lines indicates controversial results.

expression of the ICS1-mediated SA biosynthesis and signalling pathway, as well as a lower SA accumulation level upon TpE and flg22 treatment (Rasmussen *et al.*, 2013; Huot *et al.*, 2017). Conversely, a recent study showed that a short, but extremely high, TpE combined with flg22 treatment represses the expression of *flagelin sensing 2* (*FLS2*) receptor gene and transiently inhibits ROS production, whereas the expression of *FRK1* and *ICS1*, two PTI-responsive genes, was not induced (Janda *et al.*, 2019). By contrast, TpE combined with flg22 treatment of *A. thaliana* protoplasts leads to the induction of *WRK29* and *FRK1* PTI-responsive genes and to increased phosphorylation of the serine/threonine kinase BIK1 and of MAPKs (Cheng *et al.*, 2013). Taken together, these results clearly demonstrate that TpE modulates the expression of PTI-related genes; the discrepancies observed between studies are probably explained by the differences in plant material used, TpE magnitude and duration of TpE. More evidence is available for ETI-mediated resistance. Yang and Hua reported a lower expression of *ENHANCED DISEASE SUSCEPTIBILITY1* (*EDS1*) and *Phytoalexin Deficient4* (*PAD4*) immune regulators at 28°C compared with 22°C (Yang & Hua, 2004) (Fig. 5b). In maize, induction of spontaneous HR-like lesions by the Rp1-D21 NLR variant in the absence of pathogen is suppressed at 30°C and correlates with a lower expression of defence-related genes, such as *PR1, PR5, PRms* and *WIP1*, at 30°C, when compared with 18°C (Negeri *et al.*, 2013). Similarly, in tomato, Cf-4- and Cf-9-mediated immunity triggered by *Cladosporium fulvum* Avr4 and Avr9 effectors is inhibited upon exposure to high temperatures. Alteration of these immune responses correlates with misregulation of various HR-related and defence-related genes at elevated temperature (de Jong *et al.*, 2002). Other studies have reported a specific regulation of several *NLR* genes under combined TpE and pathogen stress (Chen *et al.*, 2013; Lu *et al.*, 2017; Onaga *et al.*, 2017a; Toa *et al.*, 2018). More recently, MacQueen and Bergelson investigated the expression profile of 13 *NLR* genes in a subset of *A. thaliana* natural accessions that were treated under different environmental conditions, including TpE, before and after inoculation with various strains of *Pst*. Prior pathogen challenging and under all tested environmental conditions, the expression of *NLR* genes was increased. Interestingly, interference of TpE with *NLR* gene expression correlates with the historical climate of the geographical regions where the accessions originated. Indeed, accessions from dry climate zones showed a more drastic reduction of *NLR* expression compared with accessions from wet climate areas (MacQueen & Bergelson, 2016). By contrast, other examples indicate that transcriptional modulation of *NLR* and defence-related genes at elevated temperature is not sufficient to explain the thermosensitivity of immune responses. For instance, the resistance conferred by *Pi54 NLR* gene in rice to *Mo* remains efficient despite its downregulation at elevated temperatures, suggesting that other genotype-dependent factors related or not to *Pi54* are involved (Onaga *et al.*, 2017a). Moreover, when applying a short-term TpE simultaneously with flg22 treatment, mimicking a *Pst* DC3000 infection, the expression levels of *NLR* genes, such as *RPM1* and *RPS2*, or genes encoding key immune components, including RIN4, RAR1, SGT1b or NDR1, are not altered.

In addition, an interplay between TpE-dependent immune transcriptional reprogramming and chromatin remodelling is supported by the identification of the Arabidopsis ACTIN-RELATED PROTEIN6 (*arp6*) mutant (Cheng *et al.*, 2013). ARP6 is a component of the SWR1 complex (SWR1c), which is involved in replacement of histone H2A (HTA) with H2A.Z variant in the nucleosome. A recent transcriptomic analysis showed that H2A.Z-containing nucleosomes are evicted specifically from TpE-sensitive target genes by heat stress factor A1 class transcription factors (HSFA1), therefore facilitating induction of downstream stress-responsive transcriptional regulators (Cortijo *et al.*, 2017) (Fig. 5a). Interestingly, the *arp6* mutant displays constitutive expression of TpE-responsive genes and an enhanced resistance to *Pst* (Kumar & Wigge, 2010; Cheng *et al.*, 2013).

TpE-dependent regulation of immune responses may also rely on NLR protein stabilisation. Among systems that contribute to the homeostasis of proteins, the 26S proteasome, requiring ubiquitination of substrate proteins through a cascade of reactions involving different E ubiquitin ligases (Smalle *et al.*, 2004) is the most common (Fig. 5b). The temperature-dependent modulation of the defence response by E3 ligase complexes is well described. For example, mutations in the F-box CONSTITUTIVE EXPRESSOR OF PR GENES 1 (CPR1) involved in the SKP1-CULLIN1-F-BOX E3 ligase complex (SCF) promote SUPPRESSOR of npr1-1, CONSTITUTIVE 1 protein (SNC1)-mediated autoimmunity at lower temperatures (Cheng *et al.*, 2011). Similar results were obtained with a mutation in the SENESCENCE-ASSOCIATED E3 UBIQUITIN LIGASE1 (*SAUL1*) gene for the SUPPRESSOR OF CHS-2,3 (SOC3) NLR (Disch *et al.*, 2016; Tong *et al.*, 2017) and for the double mutant *muse13-2 muse14-1* of MUTANT SNC1-ENHANCING (MUSE) proteins 13 and 14 that interact with the SCF complex and regulate the degradation of SNC1 and RPS2 NLRs (Huang *et al.*, 2014; Copeland *et al.*, 2016) (Fig. 5b). However, the role of these proteins at elevated temperatures remains to be demonstrated. The formation of a signalling module with ubiquitin E3 ligase activity, involving DE-ETIOLATED 1 (DET1) and CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1), participates in PIF4 stabilisation at elevated temperatures. PIF4-mediated thermosensory signalling plays a significant role in the suppression of defence under elevated temperatures, probably through transcriptional repression of *SNC1* (Sreeramaiah *et al.*, 2018) (Fig. 5b). TpE-dependent inhibition of PIF4 interaction with Phytochrome B (PhyB), a canonical light receptor involved in thermosensing, would lead to PIF4 accumulation and the negative regulation of defensive genes through activation of bHLH TF HOMOLOG OF BEE2 INTERACTING WITH IBH 1 (HBI1) (Gangappa *et al.*, 2017) (Fig. 5b). However, as there is no evidence of TpE-dependent PIF4 involvement in the plant defence response, its role is still a matter of debate (Huot *et al.*, 2017). Again, these contrasting findings could be due to differences in experimental designs and to the mode of stress application.

TpE-dependent suppression of immune responses also correlates with the mislocalisation of key immune components. This is well exemplified with nuclear targeted SNC1 and N immune receptors, whose reduced nuclear accumulation at elevated

temperatures probably contributes to the inhibition of defence responses (Zhu *et al.*, 2010). The underlying mechanisms of this mislocalisation remain poorly understood. Compelling data support a central role for ABA in the interplay between TpE and immune responses. Indeed, ABA deficiency was shown to promote the nuclear accumulation of SNC1 and antagonise the immune response inhibition by TpE (Mang *et al.*, 2012) (Fig. 5b). In addition, ABA responsive *cis*-regulatory elements were found in promoter regions of genes that were specifically downregulated in thermostable *Xa7*-mediated resistance (Cohen *et al.*, 2017). Overall, the hormonal cross-talk between SA, MeJA/JA and ABA clearly participates in the regulation of the plant response under combined stresses, with sometimes opposite effects depending on the pathosystem considered (de Jong *et al.*, 2002; Mang *et al.*, 2012; Chen *et al.*, 2013; Cohen *et al.*, 2017).

Finally, TpE-dependent modulation of immunity probably also relies on the plasticity and adaptability of different plant genetic backgrounds to elevated temperatures. In wheat, HTAP and HTSP-mediated resistance conferred by genes such as *Yr36 or Yr39* are nonrace specific, durable and influenced by specific environmental conditions (Chen *et al.*, 2013; Bryant *et al.*, 2014). Interestingly, Bryant and colleagues demonstrated that instability of temperature-dependent resistance mediated by *Yr36* was host specific (Bryant *et al.*, 2014). Similar results were obtained for different soybean isolines carrying different *Rps* genes involved in resistance to *Phytophthora sojae* (Gijzen *et al.*, 1996). Interestingly, the 'spontaneous lesion' phenotype induced by the *Rp1-D21* mutation in maise depends on the genotype, although it is not known whether the heat sensitivity of this phenotype also relies on the genotype (Negeri *et al.*, 2013). Furthermore, for rice *Pi54*-mediated resistance against *Mo* under TpE, the genetic background of *Oryza sativa* ssp. *japonica* seems to significantly contribute to the thermostability of resistance compared with the *Oryza sativa* ssp. *indica* genetic background (Onaga *et al.*, 2017a).

Effect of heat stress on pathogens in interaction with their host plants Much fewer studies have investigated the effect of TpE on phytopathogens during interactions with their hosts. Findings available are illustrated in Fig. 5(a). In several cases, bacterial and virus multiplications were enhanced *in planta* under TpE (Menna *et al.*, 2015; Zhao *et al.*, 2016; Huot *et al.*, 2017). TpE can also negatively affect pathogen multiplication, as reported with TuMV whose coat protein accumulation *in planta* is repressed by elevated temperatures (Chung *et al.*, 2015). For bacterial effectors, expression of the *avrB/avrRpm1* and *avrRpt2* effector genes from *Pst*, triggering RPM1-dependent and RPS2-dependent immunity respectively, were not found to be influenced by TpE (Cheng *et al.*, 2013). By contrast, Onaga and co-workers reported the upregulation of several putative effector genes in *Mo* that infected the rice cultivar Nipponbare under TpE, partly explaining how elevated temperatures could promote pathogen virulence and infection (Onaga *et al.*, 2017b). Furthermore, the increased susceptibility of *A. thaliana* under heat stress was associated with an enhanced multiplication of *Pst* in plant tissues requiring T3E secretion (Huot *et al.*, 2017). A correlation has also been demonstrated between the increased virulence of soft rotting necrotrophic bacteria, such as *Pectobacterium atrosepticum*, and elevated temperatures (Velásquez *et al.*, 2018). This phytobacterium is responsible for the maceration of plant tissues using several bacterial plant cell-wall-degrading enzymes (PCDWE). At elevated temperatures (up to 35°C), the population density of some strains can reach a threshold that activates quorum-sensing signals and promotes the production of PCDWE, therefore increasing the virulence of the bacterium (Hasewagua *et al.*, 2005).

IV. Round three: future avenues for robust thermostable resistances in the context of global warming

Climate change is already affecting ecosystems worldwide. Among the components of climate change, TpE is one of the main factors that affects both plant development and plant–pathogen interactions. Adapting agricultural systems to minimise crop yield losses, while limiting the use of pesticides and fertilisers, is even more challenging under global warming. To achieve these goals, the combination of complementary approaches, while considering plants and pathogens from the individual to the population level (Fig. 6), should give us a more global vision of the mechanisms involved in the plant defence response under TpE.

1. Towards deciphering temperature-sensitive and temperature-resilient immune mechanisms

Genetic sources of resistance are often the most effective and environmentally friendly way of controlling plant diseases. Maintaining or stimulating the effectiveness of already known resistance mechanisms in a changing environment is a priority. Given the increasing number of studies that have reported an alteration in plant immunity under TpE, it becomes essential to evaluate more systematically the thermostability of known resistances, if possible over several years or generations, not only in controlled but also in agro-ecologically relevant conditions. Even if TpE negatively affects many resistance responses, whatever the pathosystem and the mode of stress application, it is highly likely that a wide variety of mechanisms are involved. This could be due to the diversity of adopted experimental designs and of the studied plant and pathogen species. Therefore, a better understanding of the physiological, metabolic, molecular, genetic and epigenetic mechanisms involved in TpE-dependent plant immunity modulation is required to identify upstream and downstream signalling components. This knowledge could help to increase the resilience of immune responses to combined biotic and TpE stresses (Fig. 6). To this end, well studied models in which the main molecular agents have been identified and their modes of action well characterised should be reassessed in combined stress conditions, considering the different hypotheses proposed that could explain the inhibition. Attention should be paid to the genetic background of the plant material studied, as several studies highlight a genotype effect on the TpE-dependent modulation of defence responses independently of the *R* gene involved (Gijzen *et al.*, 1996; Negeri *et al.*, 2013; Bryant *et al.*, 2014) (Fig. 6). Comparative transcriptomic analysis on plants whose ETI-mediated immune response is inhibited or NLR

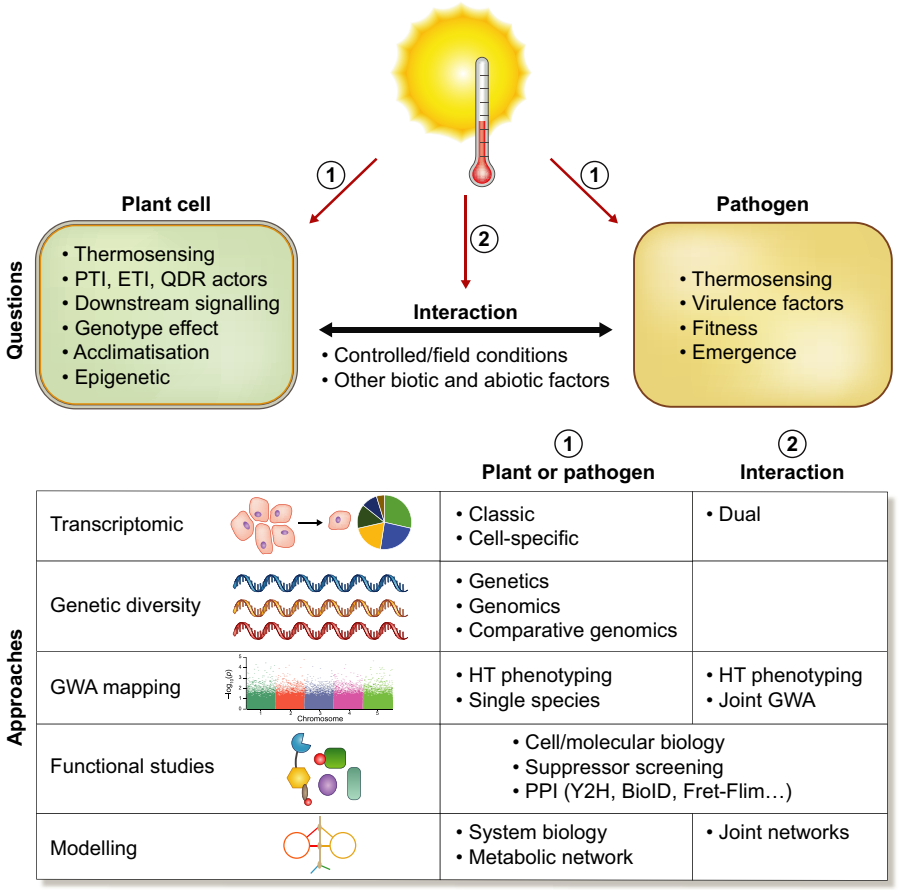

Fig. 6 Future perspectives to decipher the temperature elevation-dependent mechanisms involved in plant immune responses and pathogen virulence. Schematic representation of the questions that need to be addressed in future studies and examples of complementary approaches to be developed at the plant (1), the pathogen (1) and the plant–pathogen interaction (2) levels. GWA, genome-wide association; HT, high throughput; PPI, protein–protein interaction.

autoactivation suppressed under TpE, as for example in the study of the temperature-conditioned RPS4 autoimmunity (Heidrich *et al.*, 2013), would undoubtedly help to identify the host components and signalling pathways involved. Single-cell analyses involving approaches such as IntAct or GFP-strand systems (Deal & Henikoff, 2010; Henry *et al.*, 2017) would allow a specific focus on cells interacting directly with the pathogen under TpE, and give access to profiling their transcriptomic and epigenetic perturbations. Moreover, the availability of thermostable allelic forms of genes conferring thermosensitive resistance, as described for the thermostable SNC1 variant in *int102* mutant (Zhu *et al.*, 2010), may also be useful to unravel the underlying molecular mechanisms. Indeed, this genetic material could be used to identify the key host components involved either:

(1) by suppressive mutagenesis genetic screening, looking for reversion of thermosensitive resistance as seen with *SNC1* (Zhu *et al.*, 2010; Mang *et al.*, 2012);

(2) by classic approach (e.g. yeast two-hybrid);

(3) by more elegant proteomic approaches such as proximity-based labelling, enabling the detection of physiologically more relevant protein interactions (Roux *et al.*, 2018).

2. Elucidating the temperature elevation-dependent mechanisms regulated at the pathogen level

Under combined stress, plant immune responses depend not only on their ability to cope with TpE but also on the effect of TpE on pathogens and plant–pathogen interactions. Therefore, other ways to find innovative solutions to identify thermoresilient resistance would also require a better characterisation of the pathogen thermosensory mechanisms during the interaction. The approaches listed above are all plant centred and do not give access to the pathogen transcriptome. Solutions could come from dual-transcriptome analyses, as recently used to study plant–pathogen interactions (Zhang *et al.*, 2019). In addition to providing valuable information on the gene networks that control cross-kingdoms interactions, they would allow the identification of factors that regulate pathogen fitness and virulence under heat stress during the interaction. Moreover, biological resource centres give access to collections that represent the genetic diversity of well studied pathogen species such as the complex of *P. syringae* species or *Sclerotinia sclerotiorum*. Genomic resources are already available or are easy to produce using next generation sequencing technologies.

Combined with comparative genomic analyses, the phenotyping of representative collections of a given pathogen species under TpE, on different nutrient sources or in interaction with plants, could reveal the molecular agents necessary for their TpE-mediated virulence. Finally, for intensively studied pathogen species, relevant systems biology approaches are developed. For instance, a genome-scale reconstruction metabolic network, together with a macromolecule network module accounting for the production and secretion of *Ralstonia solanacearum* virulence determinants, has been generated (Peyraud *et al*, 2016). Integration of phenotypic, transcriptomic and metabolic data that were generated under elevated temperature conditions in such models could help to predict the nature of the trade-off between the increased virulence and proliferation of the pathogen under combined stress and facilitate the identification of heat-sensitive pathogenicity determinants.

3. Identification and study of uncovered robust resistance mechanisms

Unravelling novel resistance mechanisms that remain efficient under TpE is also essential. So far, immune mechanisms altered by TpE have been mostly investigated from a limited number of genotypes, from both the host and the pathogen perspectives. For

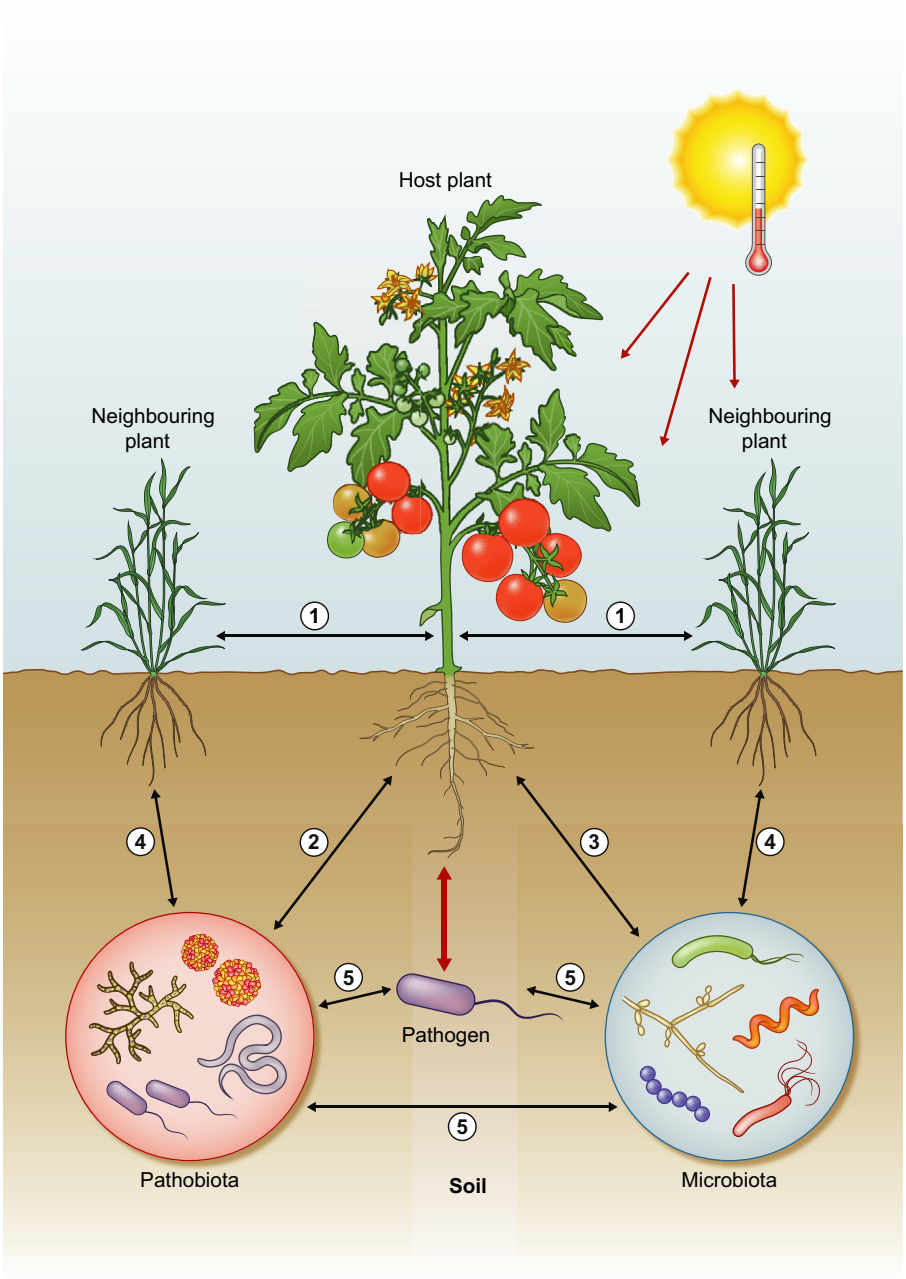

Fig. 7 Different ecological scenarios involving relationships with other biotic factors potentially affecting plant–pathogen interactions under abiotic stresses that could be investigated in future studies. Under natural conditions, the outcome of a plant–pathogen interaction relies on an immune system modulated by abiotic stress such as TpE. The issue may also depend on direct or indirect effects of neighbouring plants (1) and on the plant ability to respond to interactions (beneficial or harmful) with other pathogens (co-infections, pathobiota) (2) and its microbiota (root microbiota here) (3). Neighbouring plants may directly or indirectly modulate the effects of pathogens or microbiota (4), also shaping the outcome of the interaction. The species constituting the microbiota can compete, cooperate or coexist with each other and with phytopathogens (5). In turn, all plant–living organism interactions can be affected by abiotic factors in the environment (here TpE).

instance, a bibliometric analysis carried out between 1979 and 2016 by Gimenez and co-workers on factors related to plant–pathogen interactions revealed that most studies had been carried out on a single (or few) genotype(s) of *A. thaliana* and on a limited number of pathogens, mainly *Pst* (Gimenez *et al.*, 2018). High-throughput phenotyping tools, combined with the production of new adapted genomic resources using new sequencing technologies and genome-wide association (GWA) mapping approaches, has provided the opportunity to consider and explore more broadly the genetic diversity of the plant response to specific traits. The development of such strategies on model plants and crop species has already demonstrated their great potential with regards to the identification of genes underlying QDR to bacteria, fungi and oomycetes (French *et al.*, 2016; Bartoli & Roux, 2017; Bruessow *et al.*, 2019). Because of the durable nature and the broad spectrum resistance conferred by *QDR* genes (Chen *et al.*, 2013; Roux *et al.*, 2014; French *et al.*, 2016), it has become relevant to use such strategies to uncover thermoresilient resistance mechanisms or to consider other climate parameters in the changing environment. Moreover, new statistical methods that allow joint GWA mapping on two interacting species makes it possible to map a phenotypic trait on a pair of genomes (Wang *et al.*, 2018b). Its application, taking into account the genetic diversity of both the plant and the pathogen for a given pathosystem, should facilitate the identification of molecular agents that govern the interaction under TpE. Implementing such strategies directly onto model crop species under field conditions over several years, while integrating climate parameters is another major challenge that should be addressed. Finally, obtaining a high level of protection with thermostable resistance will require their reasoned use in combination with effective genetic resistance sources already exploited.

4. Next step: taking into account the complexity of natural interactions

Although they have demonstrated their value in characterising the mechanisms involved in plant immunity, most studies on plant–pathogen interactions were still carried out on simplified pathosystems composed of a single host plant interacting with a single pathogen. However, in their natural environment, plants often interact with a wide variety of pathogens (also called the plant pathobiota) (Bartoli *et al.*, 2018). Therefore, to predict and optimise plant responses to pathogens under abiotic stress, it is crucial to study how the plant can manage such interactions by considering, as much as possible, all microorganisms involved and also the potential effect of neighbouring plants. Recent studies on plant–multipathogenic systems have shown that interactions between pathogens depend on various parameters, including coexistence, cooperation or competition, and result in very different outcomes for the hosts (Abdullah *et al.*, 2017). The importance of the microbiota in helping plants cope with biotic or abiotic stress was also reported (Berendsen *et al.*, 2012; Muller *et al.*, 2016; Cheng *et al.*, 2019). Indeed, plants' 'beneficial' microbiota can improve and even contribute to broaden the defence response to various diseases by: (1) direct modulation of plant immunity, or (2) competition between members of the microbiota that can indirectly influence the host (Vannier *et al.*, 2019). Deciphering microbiota effects on plant–pathogen–environment interactions is the next challenge. Microbiota description and functional characterisation, together with the elucidation of plant immune mechanisms that are modulated by natural or synthetic microbial consortia, becomes accessible. Furthermore, the effect of plant–plant interactions on immune responses has long been neglected and yet could be relevant (Subrahmaniam *et al.*, 2018). Some examples of the facilitation processes under changing environments have been described (Brooker, 2006). For instance, ground vegetation cover facilitates the establishment of young trees at the altitude limit of alpine tree lines and promotes the upward movement of forest species in response to climate change (Germino *et al.*, 2002). A recent study has also demonstrated that the tree neighbours of a host plant, belonging to different species, have a significant negative influence on root-associated host-specific pathogenic fungi as well as on other phytopathogens (Cheng & Yu, 2020). Fig. 7 presents different ecological scenarios that could lead to a modulation of host immune responses under heat stress and that should be explored in future studies. All the interesting aspects of plant–pathogen interactions clearly need further investigation. Answering the emerging questions discussed in this review is crucial to better understand how to maintain or stimulate plant immunity in a global warming context.

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