Plant Physiol. Rep. (October–December 2019) 24(4):541–549
https://doi.org/10.1007/s40502-019-00483-7

REVIEW ARTICLE

Plant-pathogen interaction in the presence of abiotic stress: What do we know about plant responses?

Prachi Pandey[1] · **Muthappa Senthil-Kumar**[1]

Received: 8 September 2019 / Accepted: 22 November 2019 / Published online: 28 November 2019

Abstract Climate change has led to an increased number of abiotic and biotic stresses, and the plants experience these stresses simultaneously. Abiotic stresses like drought, heat, and salinity greatly influence plant-pathogen interaction when co-occurring with the biotic stressors. Recent studies have shown that combined stresses induce unique physiological and molecular responses, which involve rewiring of the hormonal pathways, accumulation of various metabolites and induction or suppression of immunity genes in plants. The net impact of the interaction, which depends on a multitude of factors, thus, modulates the effect of biotic stressors on plants by either increasing or decreasing plants susceptibility towards them. The present review aims to provide an overview of the current knowledge on the biotic and abiotic stress interactions in plants. We have discussed the role of drought, salt, and heat stress in influencing pathogen infection in plants in brief. Plants responses to the three types of combined stresses are compared to decipher the common and unique plant responses to these stresses.

Keywords Stress interaction · Biotic · Abiotic · Drought · Heat · Salinity

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s40502-019-00483-7) contains supplementary material, which is available to authorized users.

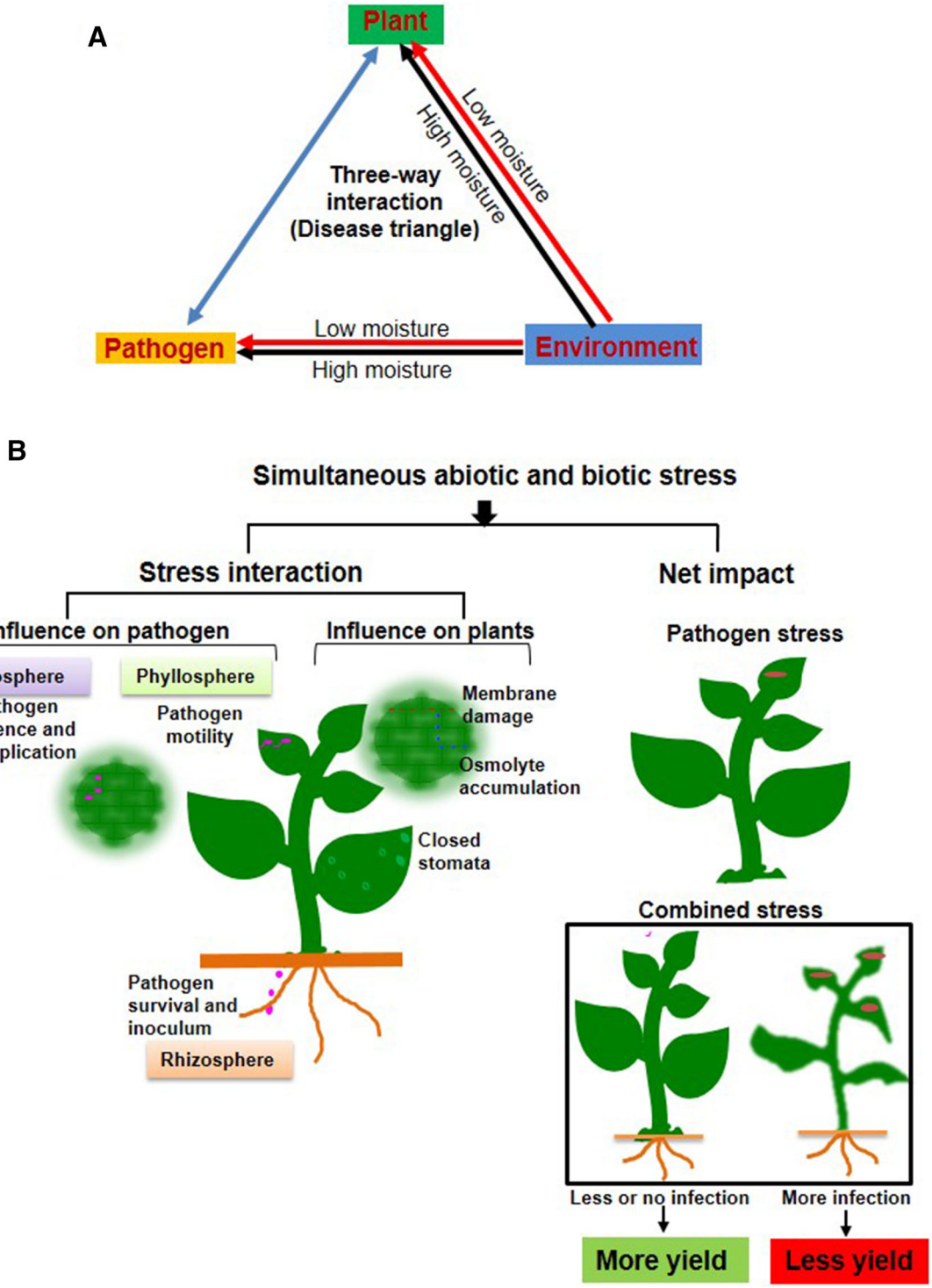
✉ Muthappa Senthil-Kumar
skmuthappa@nipgr.ac.in

[1] National Institute of Plant Genome Research, Aruna Asaf Ali Marg, New Delhi 110067, India

Introduction

Global warming pose increasing threats to plants as it leads to the evolution of complex environmental conditions involving several abiotic and biotic stressors. Many abiotic stress conditions like high and low temperatures, drought, and salinity affect the survival, dispersal, and virulence of many biotic stressors. For instance, high temperature plays a vital role in the increased occurrence of diseases caused by *Ralstonia solanacearum*, *Acidovorax avenae,* and *Burkholderia glumea* (Kudela 2009). Moreover, increase in temperature during winters (warmer winters) ease the survival of aphids thus spreading *Barley yellow dwarf virus* (BYDV) and also increase viruses of potato and sugar beet (Thomas 1989; Mackerron et al. 1993).

The role of environmental factors in influencing the occurrence and progression of plant diseases has been earlier identified and represented as 'disease triangle'. Abiotic stresses like high temperature, salinity, and drought along with affecting plant growth also modulate the response of plants towards pathogen infection by either affecting the pathogen per se or enhancing or suppressing the defense response of plants to the pathogens (Fig. 1A). Pathogens are equipped with constantly evolving virulence mechanism that helps them to successfully invade their hosts. The tug of war between the plants and pathogens is widely affected by these abiotic stresses which modulate plant defense as well as pathogen multiplication, virulence, motility and survival in the endosphere, phyllosphere and the rhizosphere (Fig. 1B). It is the interaction between plants and pathogens at these levels (endo-, phyllo- and rhizosphere) that influences the net impact of the combined stresses on plants. Recent studies have thrown light on the transcriptional, physiological, and metabolic changes occurring during combined stresses and unique responses


A
Plant
Low moisture
High moisture
Three-way
interaction
(Disease triangle)
Pathogen
Low moisture
High moisture
Environment
B
Simultaneous abiotic and biotic stress
Stress interaction
Net impact
Influence on pathogen
Influence on plants
Endosphere
Phyllosphere
Pathogen
virulence and
multiplication
Pathogen
motility
Membrane
damage
Osmolyte
accumulation
Closed
stomata
Pathogen
survival and
inoculum
Rhizosphere
Pathogen stress
Combined stress
Less or no infection
More infection
More yield
Less yield

◀**Fig. 1** Schematic representation of the interaction between abiotic and biotic stresses. **A** Disease triangle showing three-way interaction between host, environment and pathogen is depicted here. Pathogen infection or a disease is a net effect of interaction between the three elements of the triangle. Environmental conditions (here exemplified as moisture) affect both plant, pathogen and plant-pathogen interaction. Low soil moisture levels cause wilting of plants and may negatively affect mobility of pathogens and can decide the fate of plant pathogen interactions. **B** Effect of simultaneous abiotic and biotic stresses on plants is shown here. The effect of simultaneously occurring abiotic and biotic stresses can be assessed either by studying the interaction between two stresses or the net impact of the two stresses on plant, depending on the stress combination and the plant species. The interaction between biotic and abiotic stresses can occur by influence on pathogen or plant responses. Pathogen responses can be perturbed by abiotic stresses at various levels in and outside plant interface e.g., at phyllosphere where abiotic stress conditions can affect pathogen adherence and motility. For example, temperature stress enhances the motility of *Tobcco mosaic virus* in *Nicotiana tabaccum*. Similarly, at endosphere (inside the plant), abiotic stress induced plant responses can modulate pathogen multiplication. For example, temperature stress (indicated as T) increases multiplication of *Turnip crinkle virus* (TCV; indicated by orange oval) in *A. thaliana* and drought stress (indicated as D) decreases multiplication of *Pseudomonas syringae pv tomato (indicated as purple rectangle)* in *A. thaliana*. At rhizospheric level, survival and inoculum levels of pathogen can be affected by abiotic stresses. For example, drought increases inoculum levels of smut causing soil borne pathogen *Urocystis agropyri (indicated as green hexagon) and decreases the levels of root rot causing oomycetes Pythium* sp. (indicated by green hexagon). Abiotic stresses also affects plant responses like membrane architecture, closing or opening of stomata, or accumulation of osmolytes which in turn affect pathogen survival, virulence and multiplication inside plant. The net impact of the combined stresses is thus the resultant of impact of individual stresses as well as the interaction between the two stresses. The net impact of the combined stress is negative if (**i**) either or both the stresses are too severe (e.g. very high temperatures, salt concentrations or prolonged drought & very high inoculum levels) or (**ii**) the interaction between the two stresses is positive i.e. pathogen can modulate the abiotic stress induced physiological and molecular changes in their favour and are able to thrive and multiply inside the plant resulting into increased infection. The net impact of the combined stress is positive if (**i**) abiotic stress induces non conducive environment for pathogen growth and infection outside and inside the plants (high salt concentration, less water etc.); (**ii**) abiotic stress induce strong defense responses which suppresses pathogen growth and multiplication e.g. induction of PR proteins by drought stress. Uag—*Urocystis agropyri, Pyt—Pythium* sp., *TMV—Tobacco mosaic virus*, *TCV—Turnip crinkle virus*, *Pst—Pseudomonas syringae pv tomato* (color figure online)

characteristic to combined stresses have been observed in plants. Modulation of highly complex and intertwined hormone signaling has been observed in all combined stresses (Prasch and Sonnewald 2013).

This review highlights the ways by which heat, salinity, and drought stress affect pathogen infections in plants. Both negative and positive effects of these three abiotic stresses on pathogen infection are briefly discussed, and new avenues for further understanding the interaction between the two stresses are highlighted (Fig. 2).

Effect of heat stress on pathogen infection

Heat stress has varied effects on pathogens and plant-pathogen interaction depending upon the type of pathogen, host plant, geographic location and season (Elad and Pertot 2014; Velásquez et al. 2018) Warmer winters facilitate the survival and virulence of pathogens evolved under cooler climates thereby increasing the risk of diseases caused by them (Deutsch et al. 2008). For example, increase in temperature during winters resulted in early outbreak of phoma stem canker of oilseed rape (caused by *Leptosphaeria maculans*) in southern United Kingdom (Sun et al. 2000). An increase in annual mean temperature by 0.8–1.0 °C led to early onset of Cercospora leaf spot of sugar beet (Richerzhagen et al. 2011). Increase in temperature modulates life cycle of pathogens by affecting their survival, multiplication, and dissemination by vectors. Among all the abiotic factors temperature is the most crucial parameter that affects the life cycle and density of viral vectors thereby affecting the occurrence of viral diseases (Bale et al. 2002).

Plants transcriptome and metabolome undergoes extensive changes to cope with the effect of high temperature. This not only affects plants physiology and growth but also their ability to fight with pathogens and pests. Heat stress may either increase or decrease the susceptibility of plants to a pathogen depending on the tolerance capacity of host and pathogen to the elevated temperature. In general, pathogen infection is aggravated when the host plant is more adversely affected by the increased temperature than the pathogen. Heat stress basically affects plant pathogen interaction by either modulating the expression of host resistance genes or modifying the pathogen virulence. Heat stress impairs both the basal and R gene-mediated resistance (Table S1). For example, high temperature not only modulates the inoculum density of *L. maculans* (Huang et al. 2005), it also impairs the Rlm6-mediated resistance to pathogen in *Brassica napus* (Huang et al. 2006). Contrarily, higher temperatures (25–35 °C) enhance Yr36 mediated resistance to *Puccinia striiformis* in *Triticum aestivum* (Uauy et al. 2005) and Xa7 mediated resistance to *X. oryzae* in *O. sativa* (Webb et al. 2010). Moreover, high temperature (30 °C) triggers more efficient RNA silencing-mediated plant defense thus enhancing the resistance of plants to some geminiviruses like *Cassava geminivirus* and *Cotton leaf crinkle virus* (Chellappan et al. 2005; Tuttle et al. 2008). High temperature also modulates plant pathogen interaction by modulating virulence strategies of pathogens. In *Pseudomonas syringae-Arabidopsis thaliana*

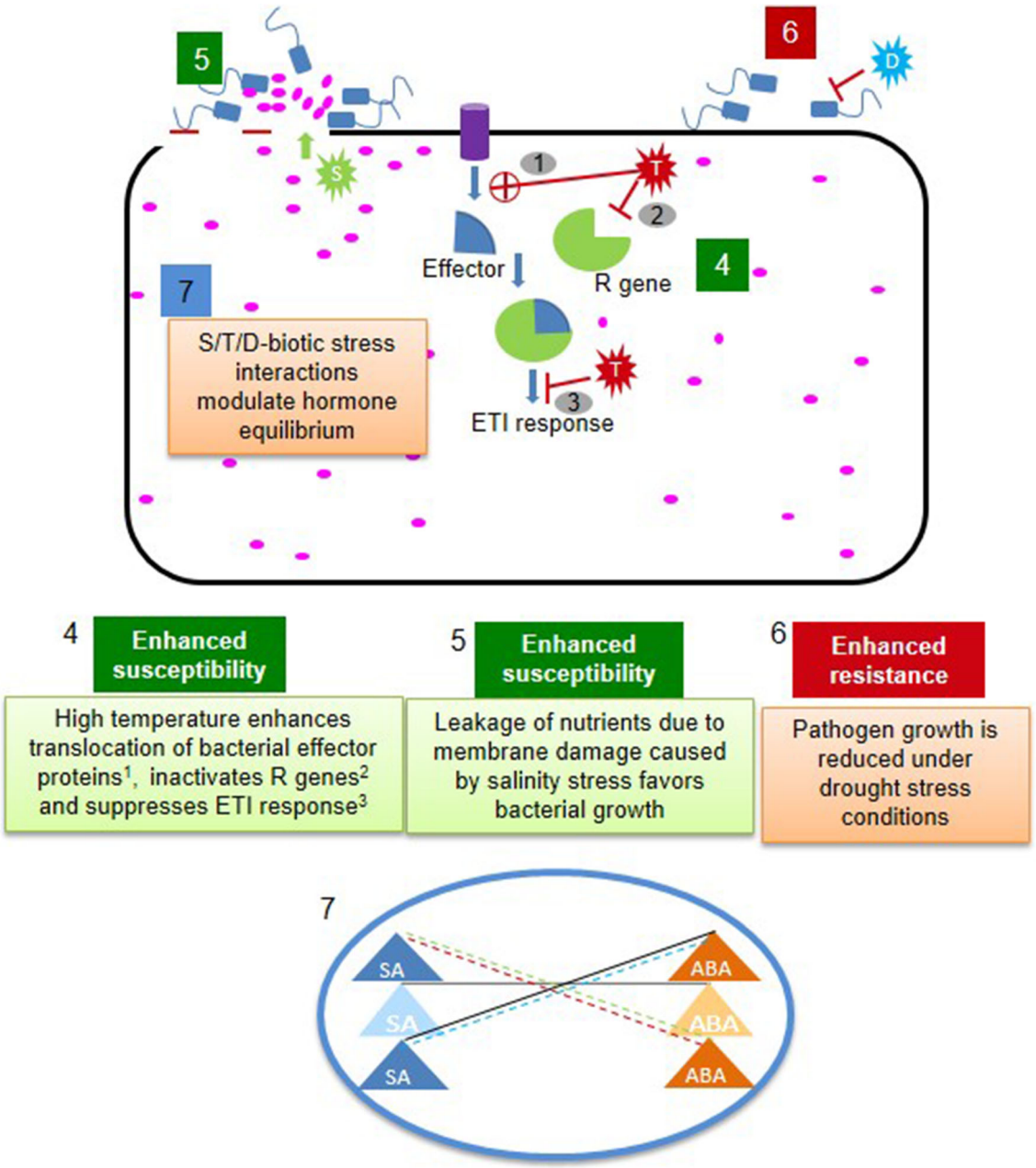


Fig. 2 Schematic representation of the effect of temperature, salinity, and drought on *Pseudomonas syringae* infection in *Arabidopsis thaliana*. High temperature (marked as T red star) enhances translocation of bacterial effector proteins (indicated by grey circle numbered 1) inactivates *R* genes (indicated by grey circle numbered 2) and suppresses ETI response (indicated by grey circle numbered 3) leading to enhanced susceptibility (indicated in red square numbered 4). Salinity, on the other hand, causes enhanced membrane damage, which leads to nutrient leakage into the apoplast, which helps the bacterial population to feed and grow rapidly and resulting in enhanced susceptibility (indicated in red square numbered 5). Drought stress, on the contrary, inhibits bacterial multiplication resulting in enhanced resistance against the bacteria (indicated in red square numbered 6). The abiotic stress conditions also modulate the hormone equilibrium (enclosed in big blue oval) in the plant ((indicated in blue square numbered 7). Pathogen infection activates SA related genes resulting in enhanced production of SA (indicated by the black line and faded blue (SA levels) and red (ABA levels) triangles. Abiotic stress interferes with this condition. High temperature (indicated by the broken red arrow) leads to downregulation of SA biosynthetic genes leading to less production of SA and raising the SA-ABA see-saw on ABA side. Salinity also has the same effect (indicated by green broken arrow) and enhances the production of ABA, which inhibits SA production. On the contrary under drought conditions (indicated by light blue broken arrow), SA production is enhanced, which results in enhanced resistance to the pathogen. The figure is based on studies by Huot et al. (2017), Chojak-Koźniewska et al. (2017) and Gupta et al. (2016). ABA—abscisic acid, SA—salicylic acid, ETI—effector triggered immune response, T—heat stress, S—salinity, D—drought (color figure online)

pathosystem, elevated temperature (30 °C) caused increased translocation of bacterial T3E effector proteins into the plant cell thereby facilitating bacterial growth in plants (Fig. 2) (Huot et al. 2017). High temperature (26 °C) enhances the replication of *Turnip crinkle virus* (TCV) in *A. thaliana* (Zhang et al. 2012), *Peanut stunt virus* in *Nicotiana benthamiana* (Obrepalska-Steplowska et al. 2015) and spread of *Tobacco mosaic virus* in *Nicotiana tabacum* (Király et al. 2008). High temperature (35 °C) also enhances expression of cell wall degrading enzymes of *Pectobacterium atrosepticum* leading to increased virulence (Hasegawa et al. 2005). Contrastingly, high temperature is known to suppress production of bacterial toxins in *P. syringae* pv. *glycinea* (causal agent of bacterial blight in soybean) (Hockett et al. 2013).

To better understand the impact of heat stress on pathogen infection, studies have been performed wherein the effects of both individual and combined heat and pathogen infection on plants have been studied and compared. Prasch and Sonnewald (2013) showed heat stress co-occurring with *Turnip mosaic virus* infection modifies viral specific signaling leading to impairment of defence responses and increased susceptibility to the virus. Transcriptome studies in *Oryza sativa* plants exposed to combined heat and *Xanthomonas oryzae* infection have indicated the role of genes involved in ABA biosynthesis, metabolism and signaling in Xa7 mediated resistance to *Xanthomonas oryzae* (Cohen et al. 2017). ABA biosynthetic genes were upregulated under both individual and combined heat and *X. oryzae* infection at all time points indicating their role in modifying plant pathogen interaction under high temperature conditions. Heat stress also represses SA signalling which is otherwise induced under *Pseudomonas syringae* pv. *tomato* DC3000 infection in *A. thaliana* leading to increased susceptibility to the pathogen (Huot et al. 2017).

Effect of salinity stress on pathogen infection

The effect of salinity stress on pathogen infection in plants also depends on several factors, namely, type of pathogen, the intensity of salt stress imposed, etc. Whereas viral infection under salinity stress is majorly seen to aggravate infection (Elsheikh and Osman 2002; Cui et al. 2015; Li et al. 2018), fungal infection under salinity stress is either enhanced or suppressed depending on the intensity of salt stress (Wiese et al. 2004; Triky-Dotan et al. 2005; DiLeo et al. 2010). For instance, at low concentration, salt stress suppressed *S. lycopersicum* defense against *Oidium neolycopersi* (causal agent of powdery mildew), high salt concentration provided resistance against the pathogen (Kissoudis et al. 2016). A high concentration of salt inhibits fungal growth, and this can be attributed to increased resistance conferred at a higher concentration of salt. Low concentration of salts, seemingly tolerated by the fungi, may induce ABA signaling which interferes with SA mediated defense, thereby enhancing the susceptibility. Salt stress also increases the intensity of infection caused by *P. syringae* pv. tomato in *S. lycopersicum*. The enhanced susceptibility was attributed to salinity induced ABA accumulation in roots, which suppressed SA mediated defense (Pye et al. 2013). In another study, it was found that combined salinity stress and *P. syringae* pv. *lachrymans* infection had a more detrimental impact on water status and stomatal functioning of *Cucumis sativus* than the individual stresses (Chojak-Koźniewska et al. 2017). The increased disease severity was correlated with increased bacterial population due to increased nutrient leakage caused by salinity induced membrane damage, thus, rendering the apoplastic environment more favorable for pathogen growth. Enhanced infection in combined stress plants also corresponded with increased ABA/SA ratio and decreased Pathogenesis-related 1 gene (*PR1*). Moreover, combined stress-specific reactive oxygen species production and scavenging were also seen. Combined stressed plants exhibited an increased production of ROS followed by upregulation of Iron superoxide dismutase (FeSOD) enzyme activity (Chojak-Koźniewska et al. 2017).

Effect of drought stress on pathogen infection

Drought stress causes various physiological changes in plants including stomatal closure, reduction in photosynthesis, inhibition of leaf growth and changes in root/shoot ratio. In some cases, drought also impairs production of plant defense substances, favouring the progress of the pathogen. Conversely, several diseases are less severe when the availability of moisture is limited (Achuo et al. 2006). The effect of drought on plants less susceptibility to a pathogen largely depends on the nature of pathogen and occurrence and intensity of drought (Gupta et al. 2016; Ramegowda and Senthil-Kumar 2015; El Aou-ouad et al. 2016). Although most studies till date have focussed on the effect of drought on plants responses towards pathogen infection, a few have also highlighted the direct impact of drought on pathogen growth inside the plant (Gupta et al. 2016; Sinha et al. 2016). The interaction between drought-induced responses and pathogen infection inside the plants can be positive, negative, or neutral. In cases of positive interaction, both stresses may have additive damaging effects on plants as indicated by enhanced damage incurred by *Beet yellows virus* and *Maize dwarf mosaic* virus under drought stress (Olsen et al. 1990). Prasch and Sonnewald

(2013) showed that combined virus and drought stress caused more reduction in plant biomass as compared with plants treated with either virus or drought alone. Drought stress may also exacerbate the damage caused by xylem invading pathogens as indicated by *Xyllela fastidiosa* infection in drought stressed *Vitis vinifera* wherein the combined stress caused more severe disease symptoms and greater reductions in physiological parameters like total photosynthetic activity and stomatal conductance (Choi et al. 2013). Drought-induced changes in plant architecture or physiology can also suppress pathogen infection as well as growth in plants. For example, drought stress induced reduction in root lengths decrease the chances of soil borne pathogens to reach the roots thereby causing suppression of infection (Huisman 1982). Inside the plant interface, drought stress can also supress pathogen growth as indicated by reduced *P. syringae* growth in *A. thaliana* (Gupta et al. 2017) and decreased multiplication of *R. solanacearum* in *Cicer arietinum* under drought stress (Sinha et al. 2016). In cases of neutral interaction, both the stresses though cast negative effects on plants, there is no interaction between the two stressors inside the plant. For instance, combined drought and GLRaV-3 infection in *V. vinifera* did not cause any additive damage to the plants. However, both the individual drought, virus infection and combined stress reduced the net photosynthetic rate, stomatal conductance, leaf transpiration and hydraulic conductance in plants (El Aou-Ouad et al. 2016, 2017).

The interaction between drought stress and viral diseases is much more intricate, as shown by Davis et al. (2015) in the study that involved the application of simulated drought stress on wheat plants infected with *Barley yellow dwarf virus* (BYDV). Under well-watered conditions, BYDV infection led to reduced plant growth. Drought stress (chronic) did not have any significant effect on plant growth; however, acute water deprivation had a positive effect on BYDV infected plants making the plants more tolerant to BYDV infection. However, there is a possibility that drought conditions enhance the build-up of the vector population and the spread of the virus (Davis et al. 2015).

The effect of drought on plant-pathogen interaction can be attributed to the cross-talk between the drought stress-induced the ABA pathway and pathogen-induced SA and JA pathway (Lievens et al. 2017). The LC–MS based profiling of the three hormones in *A. thaliana* exposed to combined drought and *P. syringae* infection, drought being the first stressor, revealed repression of ABA, concomitant induction of SA and JA and reduced bacterial growth in the plants. However, when the pathogen was the first stressor, accumulation of ABA, SA, JA levels, and unaltered bacterial growth as compared to individually stressed plants (pathogen-infected) was observed. Thus, repression of ABA synthesis seems to be responsible for the observed resistance in combined stressed plants. Furthermore, it was found that prolonged duration of drought leads to suppression of plant defense, mainly SA and ET signaling leading to the susceptibility of plants to the bacteria (Gupta et al. 2017). Interestingly, secondary cell wall modifications are also differentially affected by the severity of drought in *R. solanacearum* infected plants. Early drought led to up-regulation of lignin biosynthesis and prolonged drought-induced cellulose biosynthesis which was correlated with enhanced susceptibility (Sinha et al. 2017).

Avenues for improving plant resistance under combined abiotic and biotic stresses

Although the role of abiotic stress on pathogen infection was observed and documented long ago in terms of disease triangle, extensive molecular studies to understand the molecular changes under combined stress have just begun. The effect of heat, salt and drought on pathogen infection and plant pathogen interaction is complicated and cannot be generalized. Greenhouses and plant growth chambers must be designed in such a way that they mimic field conditions and wherever possible filed studies should be performed to assess the role of abiotic factors on plant diseases. Predictive climatic models and simulation studies have been developed for a few plant-pathosystems, like phoma stem canker (*L. maculans*) of oilseed rape in the United Kingdom (Evans et al. 2008). To predict the future severity of epidemics, weather-based models were combined with climate change models. The result of the study showed the increased severity of the disease and also that the area affected by the canker disease will extend to areas which are currently not affected by the disease, Similar predictive climatic models have been developed for downy mildew (caused by *Plasmopara viticola*) (Salinari et al. 2006), and powdery mildew (caused by *Erysiphe necator*) (Caffarra et al. 2012) of grapevine and Cercospora leaf spot (caused by *Cercospora beticola*) of sugar beet (Richerzhagen et al. 2011). There is a need to develop many more such predictive climatic models to understand the effect of abiotic factors on plant diseases. Carefully designed experiments under controlled conditions can help in further uncovering the molecular intricacies of plant patho-systems under heat, salt or drought stress. The knowledge gained from advances in transcriptomics, metabolomics and phenomics on the effect of abiotic factors on plant-pathogen interaction can be coupled with agronomic practices, like crop rotation, tillage, fertilization, irrigation, use of resistant/tolerant varieties, to reduce the risk of increased infection under combined abiotic and biotic stresses. (Juroszek and von Tiedemann 2011).

Conclusion and future perspectives

In an environment where plants are exposed to multiple stresses, the consequences of the interaction between the different stresses inside and outside the plant interface can be diverse and complicated. Abiotic stresses have been known to impact the disease cycle of pathogens and predispose plants to various pathogens (Bostock et al. 2014). Abiotic stresses interfere with the strategy used by pathogen for infection. Different factors drive the response of plants to combined abiotic and biotic stresses. The suppression or accumulation of phytohormones mainly ABA and SA are some of the common responses observed under all abiotic and biotic stress combination. The other important target affected by combined stresses is *R* gene-mediated immunity genes (Fig. 1). Apart from affecting plant defense responses, abiotic stresses induced changes like altered water potential and or ionic concentration may interfere with pathogen virulence at plant interface. Thus, the pathogen effectors might also be affected by the abiotic stressors. However, studies in this direction are not available. Analysis of the plant-pathogen interactome at the site of infection during combined stress may reveal the mechanism behind the altered defense under combined stress. Another major stage of plant-pathogen interaction vulnerable to abiotic stress is the root and leaf interface. Plants secrete anti-microbial metabolites as a part of defense mechanism against a wide variety of pathogens. Analysis of metabolites at the root and leaf interface under combined stress conditions can also shed light on the effect of abiotic stress on the establishment of infection.

The transcriptomic data generated has opened up new areas of research in combined stress. RNA silencing is an important defense arsenal of plants and being targeted by combined heat and pathogen stress, stands as an important area to be probed under other abiotic, biotic stress combinations. It would be quite interesting to see the regulation of small RNA under combined stresses. Moreover, analysis of stress modulated metabolome and secretome of both plant and pathogen can shed light on newly evolved defense strategies of plants. The effect of abiotic stresses on pathogens post-infection remains unknown, and it will be interesting to study if abiotic stresses affect pathogens capability to hijack plants defense for successful infection. Since each plant-pathogen interaction is unique, and the effect of abiotic stresses on plant-pathogen interaction is furthermore specific, studying the effect of combined stress on crop plants is important. Effective designing of experiments mimicking the environmental conditions prevailing at the time of infection in fields should be undertaken. This further warrants the role of wide-scale field studies to analyze the weather conditions at the time of infections and carefully understand the correlation, if any, between environmental conditions and plant's susceptibility to diseases. Although the combined stress research is still in its infancy, it is rapidly unfolding the intricate mechanisms of plant defense and providing avenues for the further improvement of crops under current climatic conditions.

Acknowledgements Combined stress tolerance related projects at MS-K lab were supported by DBT-innovative young biotechnologist award (BT/09/IYBA/2015/07). PP acknowledges financial support from SERB young scientist (SB/YS/LS-71/2014) scheme. We acknowledge DBT-eLibrary Consortium (DeLCON) and NIPGR library for providing access to e-resources. We also thank Dr. Mahesh Patil for critical reading of the manuscript.

Authors' contribution MS-K conceived the concept and provided an outline. PP drafted and revised the manuscript. MS-K edited and finalized the manuscript.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

Achuo, E. A., Prinsen, E., & Höfte, M. (2006). Influence of drought, salt stress and abscisic acid on the resistance of tomato to *Botrytis cinerea* and *Oidium neolycopersici*. *Plant Pathology, 55,* 178–186.

Bostock, R. M., Pye, M. F., & Roubtsova, T. (2014). Predisposition in plant disease: exploiting the nexus in abiotic and biotic stress perception and response. *Annual Review in Phytopathology., 52*, 517–549.

Bale, J. S., Masters, G. J., Hodkinson, I. D., Awmack, C., Bezemer, T. M., Brown, V. K., et al. (2002). Herbivory in global climate change research: Direct effects of rising temperature on insect herbivores. *Global Change Biology, 8,* 1–16.

Caffarra, A., Rinaldi, M., Eccel, E., Rossi, V., & Pertot, I. (2012). Modeling the impact of climate change on the interaction between grapevine and its pests and pathogens: European grapevine moth and powdery mildew. *Agriculture, Ecosystem and Environment, 148,* 89–101.

Chellappan, P., Vanitharani, R., Ogbe, F., & Fauquet, C. M. (2005). Effect of temperature on geminivirus-induced RNA silencing in plants. *Plant Physiology, 138,* 1828–1841.

Choi, H. K., Alberto, I., Francisco, G. S., & Douglas, C. (2013). Water deficit modulates the response of *Vitis vinifera* to the Pierce's disease pathogen *Xylella fastidiosa*. *Molecular Plant Microbe Interactions, 26,* 1–46.

Chojak-Koźniewska, J., Linkiewicz, A., Sowa, S., Radzioch, M., & Kuzniak, E. (2017). Interactive effects of salt stress and *Pseudomonas syringae* pv. lachrymans infection in cucumber: Involvement of antioxidant enzymes, abscisic acid and salicylic acid. *Environmental and Experimental Botany, 136,* 9–20.

Cohen, S. P., Liu, H., Argueso, C. T., Pereira, A., Vera Cruz, C., Verdier, V., et al. (2017). RNA-Seq analysis reveals insight into enhanced rice Xa7-mediated bacterial blight resistance at high temperature. *PLoS ONE, 12*(11), e0187625.

Cui, Z.-H., Bi, W.-L., Pan, C., Xu, Y., & Wang, Q.-C. (2015). Abiotic stress improves in vitro biological indexing of Grapevine

leafroll-associated virus-3 in red grapevine cultivars. *Australian Journal of Grape and Wine Research, 21,* 490–495.

Davis, T. S., Bosque-Pérez, N. A., Foote, N. E., Magney, T., & Eigenbrode, S. D. (2015). Environmentally dependent host–pathogen and vector–pathogen interactions in the Barley yellow dwarf virus pathosystem. *Journal of Applied Ecology, 52*(5), 1392–1401.

Deutsch, C. A., Tewksbury, J. J., Huey, R. B., Sheldon, K. S., Ghalambor, C. K., Haak, D. C., et al. (2008). Impacts of climate warming on terrestrial ectotherms across latitude. *Proceedings in Natural Academy of Sciences USA, 105,* 6668–6672.

DiLeo, M. V., Pye, M. F., Roubtsova, T. V., Duniway, J. M., MacDonald, J. D., Rizzo, D. M., et al. (2010). Abscisic acid in salt stress predisposition to Phytophthora root and crown rot in tomato and chrysanthemum. *Phytopathology, 100,* 871–879.

El Aou-ouad, H., Montero, R., Medrano, H., & Bota, J. (2016). Interactive effects of grapevine leafroll-associated virus 3 (GLRaV-3) and water stress on the physiology of *Vitis vinifera* L. cv. Malvasia de Banyalbufar and Giro-Ros. *Journal of Plant Physiology, 196–197,* 106–115.

El Aou-ouad, H., Pou, A., Tomás, M., Montero, R., Ribas-Carbo, M., Medrano, H., et al. (2017). Combined effect of virus infection and water stress on water flow and water economy in grapevines. *Physiologia Plantarum, 160,* 171–184.

Elad, Y., & Pertot, I. (2014). Climate change impacts on plant pathogens and plant diseases. *Journal of Crop Improvement, 28*(1), 99–139.

Elsheikh, E. A. E., & Osman, A. G. (2002). Alleviation of salinity and viral disease effect on faba bean by inoculation and nitrogen fixation. *Sudan Journal for Scientific Research, 8*(1), 73–89.

Evans, N., Baierl, A., Semenov, M. A., Gladders, P., & Fitt, B. D. L. (2008). Range and severity of a plant disease increased by global warming. *Journal of the Royal Society, Interface, 5,* 525–531.

Gupta, A., Hisano, H., Hojo, Y., Matsuura, T., Ikeda, Y., Mori, I. C., et al. (2017). Global profiling of phytohormone dynamics during combined drought and pathogen stress in *Arabidopsis thaliana* reveals ABA and JA as major regulator. *Scientific Reports*. https://doi.org/10.1038/s41598-017-03907-2.

Gupta, A., Sarkar, A. K., & Senthil-Kumar, M. (2016). Global transcriptional analysis reveals unique and shared responses in *Arabidopsis thaliana* exposed to combined drought and pathogen stress. *Frontiers in Plant Science, 7,* 686.

Hasegawa, H., Chatterjee, A., Cui, Y., & Chatterjee, A. K. (2005). Elevated temperature enhances virulence of *Erwinia carotovora* subsp. carotovora strain EC153 to plants and stimulates production of the quorum sensing signal, N-acyl homoserine lactone, and extracellular proteins. *Applied Environmental Microbiology, 71*(8), 4655–4663.

Hockett, K. L., Burch, A. Y., & Lindow, S. E. (2013). Thermoregulation of genes mediating motility and plant interactions in *Pseudomonas syringae*. *PLoS ONE, 8*(3), e59850.

Huang, Y.-J., Evans, N., Li, Z.-Q., Eckert, M., Chevre, A.-M., Renard, M., et al. (2006). Temperature and leaf wetness duration affect phenotypic expression of Rlm6-mediated resistance to *Leptosphaeria maculans* in *Brassica napus*. *New Phytologist, 170,* 129–141.

Huang, Y.-J., Fitt, B. D. L., Jedryczka, M., Dakowska, S., West, J. S., Gladders, P., et al. (2005). Patterns of ascospore release in relation to phoma stem canker epidemiology in England (*Leptosphaeria maculans*) and Poland (*Leptosphaeria biglobosa*). *European Journal of Plant Pathology, 111,* 263–277.

Huisman, O. C. (1982). Interrelations of root growth dynamics to epidemiology of root-invading fungi. *Annual Review Phytopathology, 20,* 303–327.

Huot, B., Castroverde, C. D. M., Velásquez, A. C., Hubbard, E., Pulman, J. A., Yao, J., et al. (2017). Dual impact of elevated temperature on plant defence and bacterial virulence in Arabidopsis. *Nature Communication, 8*(1), 1808.

Juroszek, P., & von Tiedemann, A. (2011). Potential strategies and future requirements for plant disease management under a changing climate. *Plant Pathology, 60,* 100–112.

Király, L., Hafez, Y. M., Fodor, J., & Király, Z. (2008). Suppression of tobacco mosaic virus-induced hypersensitive-type necrotization in tobacco at high temperature is associated with downregulation of NADPH oxidase and superoxide and stimulation of dehydroascorbate reductase. *Journal of General Virology, 89,* 799–808.

Kissoudis, C., Sunarti, S., van de Wiel, C., Visser, R. G. F., van der Linden, C. G., & Bai, Y. (2016). Responses to combined abiotic and biotic stress in tomato are governed by stress intensity and resistance mechanism. *Journal of Experimental Botany, 67,* 5119–5132.

Kudela, V. (2009). Potential impact of climate change on geographic distribution of plant pathogenic bacteria in Central Europe. *Plant Protection Science, 45,* S27–S32.

Li, J.-W., Chen, H.-Y., Li, J., Zhang, Z., Blystad, D.-R., & Wan, Q.-C. (2018). Growth, microtuber production and physiological metabolism in virus-free and virus-infected potato in vitro plantlets grown under NaCl-induced salt stress. *European Journal of Plant Pathology, 152,* 417.

Lievens, L., Pollier, J., Goossens, A., Beyaert, R., & Staal, J. (2017). Abscisic acid as pathogen effector and immune regulator. *Frontiers in Plant Science, 8,* 587.

Mackerron, D., Boag, B., Duncan, J. M., Harrison, J. G., & Woodford, J. A. T. (1993). The prospect of climate change and its implications for crop pests and diseases. In D. Ebbels (Ed.), *Plant health and the European single market* (pp. 181–193). Farnham: British Crop Production Council.

Obrepalska-Steplowska, A., Renaut, J., Planchon, S., Przybylska, A., Wieczorek, P., Barylski, J., et al. (2015). Effect of temperature on the pathogenesis, accumulation of viral and satellite RNAs and on plant proteome in peanut stunt virus and satellite RNA-infected plants. *Frontiers in Plant Science, 6,* 903.

Olsen, A. J., Pataky, J. K., D'arcy, C. J., & Ford, R. E. (1990). Effects of drought stress and infection by Maize dwarf mosaic virus (MDMV) in sweet corn. *Plant Disease.*, *74*, 147–151.

Prasch, C. M., & Sonnewald, U. (2013). Simultaneous application of heat, drought, and virus to Arabidopsis plants reveals significant shifts in signaling networks. *Plant Physiology, 162,* 1849–1866.

Pye, M. F., Hakuno, F., MacDonald, J. D., & Bostock, R. M. (2013). Induced resistance in tomato by SAR activators during predisposing salinity stress. *Frontiers in Plant Science, 4,* 116.

Ramegowda, V., & Senthil-Kumar, M. (2015). The interactive effects of simultaneous biotic and abiotic stresses on plants: Mechanistic understanding from drought and pathogen combination. *Journal of Plant Physiology, 176,* 47–54.

Richerzhagen, D., Racca, P., Zeuner, T., Kuhn, C., Falke, K., Kleinhenz, B., et al. (2011). Impact of climate change on the temporal and regional occurrence of Cercospora leaf spot in Lower Saxony. *Journal of Plant Disease Protection, 118,* 168–177.

Salinari, F., Giosue, S., Tubiello, F. N., Retorri, A., Rossi, V., Spanna, F., et al. (2006). Downy mildew (*Plasmopara viticola*) epidemics on grapevine under climate change. *Global Change Biology, 12,* 1299–1307.

Sinha, R., Gupta, A., & Senthil-Kumar, M. (2016). Understanding the impact of drought on foliar and xylem invading bacterial pathogen stress in chickpea. *Frontiers in Plant Science, 7,* 902.

Sinha, R., Gupta, A., & Senthil-Kumar, M. (2017). Concurrent drought stress and vascular pathogen infection induce common and distinct transcriptomic responses in chickpea. *Frontiers in Plant Science, 8,* 333.

Sun, P., Fitt, B. D. L., Gladders, P., & Welham, S. J. (2000). Relationships between phoma leaf spot and development of stem canker (*Leptosphaeria maculans*) on winter oilseed rape (*Brassica napus*) in southern England. *Annals of Applied Biology*, *137*, 113–125.

Thomas, T. (1989). Sugar beet in the greenhouse: A global warning. *Brown Sugar, 59,* 24–26.

Triky-Dotan, S., Yermiyahu, U., Katan, J., & Gamliel, A. (2005). Development of crown and root rot disease of tomato under irrigation with saline water. *Phytopathology, 95,* 1438–1444.

Tuttle, J. R., Idris, A. M., Brown, J. K., Haigler, C. H., & Robertson, D. (2008). Geminivirus-mediated gene silencing from Cotton leaf crumple virus is enhanced by low temperature in cotton. *Plant Physiology, 148,* 41–50.

Uauy, C., Brevis, J. C., Chen, X., Khan, I., Jackson, L., Chicaiza, O., et al. (2005). High-temperature adult-plant (HTAP) stripe rust resistance gene *Yr36* from *Triticum turgidum* ssp. *dicoccoides* is closely linked to the grain protein content locus Gpc-B1. *Theoretical Applied Genetics, 112,* 97–105.

Velásquez, A. C., Castroverde, C. D. M., & He, S. Y. (2018). Plant–pathogen warfare under changing climate conditions. *Current Biology, 28*(10), R619–R634.

Webb, K. M., Ona, I., Bai, J., Garrett, K., Mew, T., Cruz, V., et al. (2010). A benefit of high temperature: Increased effectiveness of a rice bacterial blight disease resistance gene. *New Phytologist, 185*(2), 568–576.

Wiese, J., Kranz, T., & Schubert, S. (2004). Induction of pathogen resistance in barley by abiotic stress. *Plant Biology, 6,* 529–536.

Zhang, X., Zhang, X., Singh, J., Li, D., & Qu, F. (2012). Temperature-dependent survival of Turnip crinkle virus-infected Arabidopsis plants relies on an RNA silencing-based defense that requires dcl2, AGO2, and HEN1. *Journal of Virology, 86,* 6847–6854.