Climate Change: Impact on Plant Pathogens, Diseases, and Their Management

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Introduction

Anthropogenic emissions (combustion of fossil fuels resulting in the release of carbon dioxide [CO_2] as well as the release of other gases such as methane) and anthropological shifts in demographics, economics, technology and social behaviour have contributed significantly to climate change. With earth's temperatures that have already been increased by more than 1 °C since the 1880s, the Intergovernmental Panel on Climate Change (IPCC) has projected that global temperatures will have a further increase of 1.5 °C between 2030 and 2052 (IPCC 2014). The IPCC further predicts that increase in global mean temperature from 1 to 3 °C above 1990 levels will have either positive or negative impacts depending upon the region, with net annual cost set to surge as the temperature increases. The shared interactions between plant hosts, pathogens and their environment in causing plant diseases are described by the disease triangle (Garrett et al. 2006; Grulke 2011; Nazir et al. 2018), with environment playing a key role to determine the outcome of these interactions. Changes in climate such as increase in temperature and atmospheric CO_2 and the frequency and intensity of extreme weather fluctuations such as drought and flooding affect host plant resistance to pathogens (Dossa et al. 2015). The changes in these climate variables may reshape host-pathogen interactions and influence spatial and temporal development of disease epidemics (Chakraborty 2005; Burdon et al. 2006; Garrett et al. 2006; Crowl et al. 2008; Eastburn et al. 2011). Understanding the effects of climate change on disease dynamics is crucial in adopting appropriate control measures and identifying sources of resistance to diseases (Chakraborty and Pangga 2004; Ghini et al. 2008). Weather is a key driver of endemic bacterial dis-

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eases and the transient temperature shifts predicted to be increased during this century; these have potential to either suppress or cause disease outbreaks (Anderson et al. 2004; Fischer and Knutti 2015).

Although the outcome of host-pathogen interactions is driven by a number of different climate variables, temperature is the most important factor. Increase in temperature may result in greater crop vulnerability to disease (Coakley et al. 1999) due to the interactive effect of temperature on both the host plant and pathogen. In addition, elevated CO_2 has also shown substantial effect on disease development under controlled conditions. Most diseases studied by Chakraborty and Pangga (2004) increased in severity under the CO_2-enriched environments that resulted in alterations to the host-pathogen relationship. Within this context, we aim to consolidate information on the effects of different drivers of climate change on different groups of pathogens, on host-pathogen interactions and on disease management strategies with a primary focus on two key drivers: elevated CO_2 and temperature.

Impact of Climate Change on Plant Pathogens

A disease occurs only when a virulent pathogen attacks a susceptible host under suitable environmental conditions at an appropriate time (Agrios 2004). Climate change can change host resistance towards pathogens by altering host physiology and pathogen aggressiveness. Increases in global temperature will shift agroclimatic zones towards the poles, thereby changing the geographic distribution of current diseases by introducing pathogens into new areas. The quick acclimatization of pathogens in a region depends upon how the pathogen disperses, how it survives in the off-season and its ability to adapt to any change in its host's biology in a new environment. Concurrently, new pathogens and diseases may emerge in existing agroecosystems. More aggressive, climate-resilient strains of existing pathogens may emerge. The impact on different groups of pathogens may vary depending upon their biology, level of host-specialization, survival and dispersal modes and ability to cope with climate change. Here we discuss the impact of climate change on major groups of plant pathogens, viz. fungal, bacterial and viral.

Impact on Fungal Pathogens Fungi are the most dominant group of plant pathogens, displaying diverse modes of parasitic interaction with their host plants. Fungal pathogenesis is greatly influenced by prevailing atmospheric conditions, particularly temperature and moisture. Temperature is a critical factor during different life stages of a fungal pathogen, and any change in temperature will significantly influence fungal reproduction, rate of infection, number of infection cycles, long and short distance dispersal as well as off-season survival. Change in temperature might lead to activation of dormant races of pathogens, thus leading to sudden outbreak of an epidemic. Increase in temperature coupled with high soil moisture creates a warm and humid climate highly favourable to soilborne pathogens and foliar diseases. For example, increase in ambient temperature has increased potential for rice

blast (*Magnaporthe grisea*) incidence and epidemics for both cooler and subtropical rice-growing regions. Similarly, the severity of *Septoria* leaf spot (*Septoria lycopersici*) and stem rust (*Puccinia graminis*) in oat cultivars has also increased due to temperature sensitivity. Using a free-air CO_2 enrichment, Kobayashi et al. (2006) showed that elevating CO_2 by ~200–280 μmol mol^{-1} above ambient levels poses a potential risk for higher infection of leaf blast and sheath blight of rice in Japan.

Temperature regulates the reproduction rate of many plant pathogenic fungi (Legler et al. 2012). Elevated temperature favours accelerated spore germination in *Puccinia substriata* as well as the reproduction rate of *Monosporascus cannonballus.* Temperature increase will introduce the quick evolution of fungal pathogens due to longer seasons. It will also lead to the development of more aggressive fungal strains due to hastened gene recombination through overwintering sexual stages.

In recent years, changes in disease scenario have affected wheat production and have caused severe yield losses and reduced grain quality. Outbreak of yellow rust in the northern Indian state of Punjab in recent years is attributed to the new pathotype 78S84 which can cause infection at higher temperatures. In recent years, wheat yellow rust has started appearing in late December due to favourable weather conditions of increased temperatures (Prashar et al. 2007; Jindal et al. 2012). The incidence of powdery mildew and foliar blights has also increased in northern regions of India. The UG99 race of stem rust, a new threat to wheat production, has entered the Middle East and presents the potential for wide-scale yield losses. Powdery mildew has been predicted to cause serious losses in winter wheat in China under simulated elevated temperature conditions (Tang et al. 2017). Additionally, rice blast disease was not present in Punjab before the year 2000; however, neck blast has become a serious problem in Basmati rice in recent years.

Elevated CO_2 levels in the atmosphere cause physiological changes in plant morphology such as increases in leaf size, leaf thickness, and number of leaves (Pritchard et al. 1999). A thick canopy leads to increased duration of leaf surface moisture, presenting a highly favourable environment for spore germination and penetration in foliar pathogens leading to disease development (Garrett et al. 2006). Deutsch et al. (2008) suggested that warming may enhance fitness of pathogens in their environment on the host causing increased risk of disease epidemic outbreak. Elevated CO_2 levels (780 ppm) have been shown to increase the susceptibility of wheat variety *Remus* to *Fusarium* head blight and *Septoria tritici* blotch (Vary et al. 2015). In contrast, elevated concentrations of CO_2 and O_3 reduced downy mildew (*Peronospora manshurica*) severity but increased brown spot (*Septoria glycines*) severity in soybean (Eastburn et al. 2010). Elevated CO_2 and O_3 levels have also been shown to increase resistance in barley against powdery mildew (*Blumeria graminis*) through formation of papillae and silicon accumulation at sites of appressorial penetration. Greater fecundity in some pathogens as a result of increased levels of atmospheric CO_2 could lead to polycyclic epidemics in crops (Chakraborty and Datta 2003). It is difficult to generalize the effect of elevated CO_2 on a particular pathogen as it also depends on the effect of elevated CO_2 on the host plant, suggesting that the effect can be either positive or negative (Gautam et al. 2013).

Climate change leads to an increase in tropospheric ozone that hinders plant photosynthesis, resulting in poor growth due to water-soaked lesions. This also predisposes plants to biotic attack, particularly in the presence of necrotrophic and root-infecting fungi. It has been shown that *Botrytis cinerea* causing grey mould is more aggressive on onions exposed to ozone (Wukasch and Hofstra 1977).

Impact on Bacterial Pathogens For every disease to occur, there is optimal temperature range, e.g. *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), that fails to efficiently colonize rice xylem when the daytime temperature exceeds 35 °C (Horino et al. 1982). *Ralstonia solanacearum*, a major cause of bacterial wilt and brown rot in potato, fails to survive the high summer temperatures of Punjab, India. More than 40 genera of bacteria are reported to be associated with plants as pathogens. The emergence of a number of bacterial genera as a serious problem worldwide could possibly be due to global warming. Examples include *Acidovorax avenae* subsp. *avenae* infecting upland rice in southern Europe, *Burkholderia andropogonis* on jojoba in eastern Australia, *B. glumae* on rice in the southern United States and *Dickeya zeae* in rice in north India especially under high temperatures. Elevated temperature interferes with bacterial physiology, genetics and bacterial-plant interactions.

Exposure to high temperatures may increase the fitness of plant pathogenic bacteria on their respective host. Plant pathogenic bacteria, especially the xanthomonads and pseudomonads, produce copious amounts of extracellular polysaccharides (EPS) during their interaction with the host plant. This production is usually triggered by biotic and abiotic signals. Under natural conditions, bacteria use EPS to aggregate themselves and to make biofilms in order to protect themselves from unfavourable environmental conditions, antimicrobial compounds, etc. In addition, bacteria also use EPS for communication, adhesion, energy storage, etc. (Wingender et al. 1999; Vardharajula and Ali 2015). Exposure to high temperatures has been shown to trigger evolution of mutant strains with elevated levels of EPS (Nandal et al. 2005). High EPS producer strains of *Xoo* have been shown to be highly aggressive in rice (Kumar and Sakthivel 2001; Hunjan et al. 2014). High temperature (34.5 °C) attenuated the virulence of soft rot bacterium (*Erwinia carotovora* subsp. *carotovora*), but not in the particular strain EC153 that produced high levels of rRNA, N-acyl homoserine lactone and extracellular proteins causing extensive maceration of celery petioles and Chinese cabbage (Hasegawa et al. 2005).

Temperature also affects expression of effector genes in plant pathogenic bacteria and their recognition events in host plants during pathogenesis. Pathogen-associated molecular patterns (PAMPs) are conserved microbial signatures that evoke basal or PAMP-triggered immunity (PTI) in hosts upon recognition by PAMP recognition receptors in the host. PTI has been shown to be increased when *Arabidopsis* was exposed to 28 °C for short periods (Cheng et al. 2013), whereas *R* gene-based effector-triggered immunity (ETI) through *Arabidopsis RPM1*, *RPS2* and *RPS4* against *Pseudomonas syringae* is compromised at temperatures above 30 °C (Wang et al. 2009). An exception to this is rice *R* gene *Xa7*, which is more effective at high temperature against bacterial blight disease (Webb et al. 2010).

Virulence of *Agrobacterium* strains on *Kalanchoe* is attenuated at or above 32 °C possibly due to reduced *Vir* gene expression (Jin et al. 1993). The level of accumulation of virulence proteins in *Agrobacterium tumefaciens* was strongly reduced, and T-pilus assembly failed to occur at 28 °C as compared to 26 °C (Baron et al. 2001).

Impact on Viral Pathogens and Their Vectors Viral pathogens infect their plant hosts through their close association with vectors, thereby adding a new dimension to the disease triangle. The outbreak of a virus disease epidemic is therefore limited by the climatic requirements of virus vectors (Malmstrom et al. 2011). Climate change may affect both host plants and the type of vectors associated with them, determining the severity of its impact on viruses associated with that host (Jones 2009). It may also affect the range of vectors, their migration and biology (Canto et al. 2009). Jones and Barbetti (2012) have authored a comprehensive review where they tried to establish the possible effects of direct and indirect climate change parameters on the many vectors, viruses and host factors. Migrations of viruses such as potato leaf roll virus (PLRV) and potato yellow vein virus (PYVV) that are adapted to warmer regions are common at higher altitudes (Jones 2014). Elevated temperatures hastened yellow dwarf disease development and symptom expression in wheat infected with barley yellow dwarf virus-PAV (BYDV-PAV). BYDV-PAV inoculated wheat seedlings grown at higher temperatures (10.0–21.1 °C, night-day) showed higher titre than those grown at ambient (5.0–16.1 °C, night-day) temperatures (Nancarrow et al. 2014). In another study, elevated CO_2 (650 µmol mol^{-1}) was also shown to aggravate yellow dwarf disease and BYDV-PAV titre in wheat (Trebicki et al. 2015). High virus titres may not always result in enhanced symptom expression. Del Torro et al. (2015) reported that *Nicotiana benthamiana* plants inoculated with cucumber mosaic virus, PVY or potato virus X under elevated CO_2 did not affect symptom expression although virus titre was high. Elevated CO_2 reduced disease development and enhanced plant resistance in tobacco plants against potato virus Y (Matros et al. 2006) and in tomato against tobacco mosaic virus (Zhang et al. 2015) and tomato yellow leaf curl virus (Huang et al. 2012). Elevated temperature and CO_2 can significantly alter plant biochemistry, and hence plants may respond differentially to express defence responses towards insect vectors of viruses. It can affect insect fecundity, feeding rates, off-season survival and dispersal (Trebicki et al. 2017).

Most of the published reports on aphids have suggested that aphids will react strongly to environmental changes. Aphids have shorter generation times, and an increase in the number of virus carrying populations will eventually lead to higher viral load *in planta*. The aphid transmissible complex of BYDV in wheat and PVY in potato are amenable to show significant effects on the prevalence of infection because of elevated temperature and CO_2. In mild winters, high levels of aphid movement during spring have resulted in greater levels of PVY infection in potatoes. Greenhouse adapted viruses such as the pepino mosaic virus (PepMV) have moved out and became predominant in field conditions due to climate change. Natural climatic barriers in more temperate regions are deteriorating under changes in early winter temperatures, allowing for the natural spread of diseases, pests and

vectors at prolific rates. Increased likelihood for the outbreak of plant disease vectors such as aphids, whiteflies, thrips and beetles may cause severe epidemics in these regions.

Impact of Climate Change on *R* Gene-Mediated Disease Control The defence mechanism of *R* genes is considered the major source of resistance in various crop-breeding programs. Pathogen populations are subjected to selection pressure due to changing environmental conditions, thus affecting the durability of a deployed *R* gene. *R* genes can be sensitive to temperature and are only effective at either high or low temperatures (Dyck and Johnson 1983; Eizenberg et al. 2003; Webb et al. 2010; Dossa et al. 2015).

The wheat stripe rust resistance gene *Yr36* imparts resistance to a wide spectrum of *Puccinia striiformis* f. sp. *tritici (Pst)* races. The gene is reported to be effective at high temperatures (25–35 °C) although unable to resist fungal attack at temperatures below 15 °C (Uauy et al. 2005). Understanding the interconnectivity of environmental factors, pathogen evolution and *R* gene effectiveness requires knowledge of *R* gene function mechanisms and how a particular function is affected by various weather parameters. Loss of function in an effector protein through a single mutation may lead to loss of recognition by plant R proteins, resulting in evolution of new races or widening of the pathogen host range. In another context, a mutation in the effector protein can also result in reduced pathogenic fitness (Burdon et al. 2006). Effectiveness of bacterial blight resistance gene *Xa7* has been correlated with mutations in *Xoo* that resulted in reduced pathogenic fitness of the bacterium due to its reduced aggressiveness on susceptible host cultivars. These mutations in *Xoo* have occurred specifically in the pathogen effector gene *avra7*, and thus selection pressure imposed on the bacterium population by the *Xa7* gene resulted in reduced pathogenic fitness (Vera Cruz et al. 2000; Webb et al. 2010). The pathogen effectors block MAPK-mediated signalling pathways invoked by PRRs, however incite *R* gene-mediated effector-triggered immunity (ETI).

Elevated temperatures affect ETI in many pathosystems. At lower temperatures (10–23 °C), ETI signalling is preferentially activated, whereas PTI signalling is activated at higher temperatures (23–32 °C). The tobacco *N* gene against tobacco mosaic virus (Samuel 1931); tomato *Cf* against *Cladosporium fulvum* (de Jong et al. 2002); *Arabidopsis RPW8* against powdery mildew (Xiao et al. 2003); *Arabidopsis RPM1*, *RPS2*, and *RPS4* genes against *P. syringae*; or potato *Rx* fail to impart ETI at elevated temperatures (Wang et al. 2009). Although elevated temperature could inhibit ETI, *Xa7*, a rice disease resistance protein against *Xoo*, is more effective at higher temperatures (Webb et al. 2010).

Temperature also affects the pathogen fitness traits as well as genotype-environment interactions in case of *Pst* causing stripe or yellow rust of wheat. The pathogen shows increased aggressiveness on exposure to high temperatures, suggesting that rust fungi can adapt to warmer climatic conditions (Mboup et al. 2012). Some of the *R* genes are effective either at high or low temperatures. During a study conducted at Punjab Agricultural University, India), it was found that high temperature decreases the effectiveness of the bacterial blight resistance gene *Xa4*, while

Xa7, another bacterial blight resistance gene, was effective at high temperatures. The phenotypic results were corroborated by reduced production of different defence-related proteins in rice near isogenic line IRBB-4 at high temperatures. Hartleb and Heitefuss (1997) suggested that disease resistance can also be dependent on temperature, for example, phoma stem canker (*Leptosphaeria maculans*) of rapeseed mustard where resistance is expressed at 15 °C but not at 25 °C. Cohen et al. (2017) found certain differentially expressed genes as temperature responsive in rice cultivars containing *Xa7* using RNA-Seq technology. They suggested that plant hormone abscisic acid plays an important role in response to high temperature and pathogen attack. Tolerance to abiotic stresses may also induce enhanced resistance to biotic stresses.

Development of New Races and Diseases in an Ecosystem

Pathogens and their plant hosts are forever engaged in a co-evolutionary arms race. Pathogens evolve to evade recognition by plant hosts, while the latter evolve to recognize a wide array of pathogen effectors. To evolve new pathogenicity traits, pathogens undergo horizontal gene transfer where new mutations arise and the gene pool is widened through crossing of genetically diverse individuals leading to natural selection and subsequent adaptation of new variants. Plant pathogens are spreading globally with latitudinal shifts in their distribution for the last 60 years, largely in line with climate change projections. The damage imposed by these organisms may have serious consequences on economies of the developing world because of greater dependence on agriculture in these countries, as well as limited resources and technological interventions to manage crop diseases (Bebber et al. 2014).

Pst, the wheat pathogenic fungi with global prevalence and huge potential for long-distance migration, poses a serious invasion threat to new areas (Dean et al. 2012; Beddow et al. 2015; Ali et al. 2014; Chen et al. 2014). Often considered as a cold-loving pathogenic fungi (Brown and Hovmoller 2002), it has recently been able to invade wheat grown in warmer regions, demonstrating the thermal adaptation in it. New aggressive races of *Pst* (PstS1/S2) possessing broader virulence spectra suited to higher temperatures have been described (Milus et al. 2009; Walter et al. 2016). Incursion of a close lineage of *PstS2* was also observed in Mediterranean growing regions in 2004 (Bahri et al. 2009). Additionally, a warm temperature-loving race of *Pst* 78S84 broke down the resistance of *R* genes *Yr9* and *Yr27* in wheat mega variety PBW 343 in northwestern India.

Lyon and Broders (2017) stated that temperature and precipitation affect spore germination, infection and survival of *Pst.* Although *Pst* prefers temperatures between 7 and 12 °C, (Chen et al. 2014), temperature range adaptation has been observed among isolates (Sharma-Poudyal et al. 2014). Experiments conducted at increased temperature (18 °C) revealed that races PstS1/S2 had shorter latent periods and higher germination rates as compared to pre-2000 US *Pst* isolates (Milus et al. 2006, 2009; Walter et al. 2016). The post-2000 *Pst* populations were genetically

distinct (Ali et al. 2014) and coupled with phenotypic plasticity. This genetic differentiation might have resulted in their high temperature adaptation. Similarly, local thermal adaptation was also reported in French (Mboup et al. 2012) and Australian (Loladze et al. 2014) isolates. The warrior race of *Pst* that invaded northwestern Europe in 2011 showed wider adaptation to a range of temperatures due to its presence in both warm (Spain) and cold (Sweden) areas (Hovmoller et al. 2016). The strains of Warrior were categorized as thermal generalists as they showed the highest infection efficiency under optimal temperatures of 10 °C and 15 °C (de Vallavielle-Pope et al. 2018).

Mariette et al. (2016a) reported local thermal adaptation of *Phytophthora infestans* isolates in different climatic zones of western Europe. Their findings challenge the common notion that invasive behaviour is linked to increased aggressiveness for this pathogen (Mariette et al. 2016b). Their observations also predicted stable or reduced risk of late blight epidemics in future decades as already hypothesized by Sparks et al. (2014).

In the last century, Panama disease (*Fusarium oxysporum* f. sp. *cubense* Race 1) wiped out the banana industry in Central America. However, this was overcome by planting Cavendish bananas. However, Cavendish bananas succumbed to new race FocTR4 of this pathogen in Laos, Myanmar and Vietnam which has since spread from Southeast Asia across the Indian subcontinent, the Middle East and into Africa (Ordonez et al. 2015; Zheng et al. 2018). Climate change provided favourable temperature conditions and increased canopy wetness for spore germination and growth of *Pseudocercospora fijiensis*, the causal agent of black Sigatoka disease. The disease has been described as an emerging threat to banana cultivation (Churchill 2011) and is responsible for huge yield losses in banana plantations worldwide (Bebber 2019). Bebber (2019) parameterized an infection model on this disease that revealed an increased (44.2%) risk of infection in banana-growing areas of Latin America and the Caribbean since the 1960s.

Spatial and temporal dynamics of plant diseases are poorly understood, although models to forecast the impact of climate on crop production have been around for the last 40 years. Bregaglio et al. (2013) applied current climate as baseline and projected their simulations on potential infection events of fungal infections of wheat, rice and grapes in Europe. They forecasted brown rust (*Puccinia recondita*) to increase in wheat by 20–100%, whereas yellow rust was projected to increase by 5–20% in cold areas. Rice blast (*Pyricularia oryzae*) and brown spot (*Bipolaris oryzae*) were postulated to increase in all European rice-growing areas, specifically in northern Italy by almost 100%. Infection of *Plasmopara viticola*, the causal agent of grapes downy mildew, was estimated to increase by 5–20% throughout Europe. Their findings suggested presumed disease pressure on crops under changing climatic conditions and eventually the future challenges that farmers will face.

Impact of Climate Change on Disease Management Practices

Changing Planting Date Climate change may influence the sowing or planting date of many crops. Cultural practices and changing weather patterns influenced *Septoria tritici* blotch (STB) caused by *Zymoseptoria tritici*. The severity of the disease was decreased with low temperatures at −2 °C in the early stages of crop growth. Stewart's wilt, (*E. stewartii*), vectored by the corn flea beetle (*Chaetocnema pulicaria*) is highly dependent upon the survival of the beetle through winter. A forecast model based on winter temperatures predicted that the survival of vector is higher in warmer winters (Petzoldt and Seaman 2006).

Altering Fungicide Schedule *Septoria tritici* blotch (STB) is the major pathogen problem for wheat in the United Kingdom. Climate conditions have changed from the last decade and efforts have been made for forecasting of STB. The decision system for the timing of fungicide application has been made based upon different climate variables. There is a need to evaluate new broad-spectrum multi-target site antifungal treatments. In north-east of the United States, it was predicted that for each 1 °C rise in temperature, potato late blight would occur 4–7 days earlier, and the susceptibility period would be increased by 10–20 days. There may be a need for adding 1–4 fungicide foliar applications which would increase both farmer costs and environmental risks. In northern latitudes, a higher number of fungicide applications are required under frequent rainfall causing difficulty in retention of contact fungicides on the plant (Wolfe et al. 2008). The introduction of new fungicides with a larger effect under rainfall conditions might help to minimize this problem (Hannukkala et al. 2007). In response to higher CO_2 and elevated temperature, plant morphological characteristics like smaller stomatal openings or thicker epicuticular waxes on the leaves could reduce or delay the uptake and translocation of systemic fungicides (Juroszek and Tiedemann 2011). The efficacy of fungicides can be increased with proper timing of fungicide application (Bedos et al. 2002). Stem rot (*Sclerotium rolfsii*) of peanut managed by applying fungicide early in the morning improved spray deposition in the lower canopy of the plant (Augusto et al. 2010). The higher number of fungicide applications could be needed to control problematic diseases under high CO_2 and increased temperature. Additional fungicide sprays are required as rice plants become increasingly susceptible to leaf blast under higher CO_2 concentrations (Kobayashi et al. 2006). It was predicted that two extra applications of fungicide might become essential in order to manage downy mildew of grapevine under elevated temperatures in the northwestern part of Italy by the end of the twenty-first century (Juroszek and Tiedemann 2011).

Efficacy of Fungicides A high range of temperature and CO_2 concentrations support vegetative growth of plants (Gutierrez et al. 2008) that lowers the availability of pesticide per unit area in plants. It is accompanied by reduced pesticide uptake by the roots from the upper soil layer as they penetrate deeper soil layers. Temperature

increase, reduced rainfall and high concentrations of atmospheric CO_2 can develop fungicide resistance in pathogens resulting in an increased number of fungicide applications (Delcour et al. 2015).

Fungicide uptake and translocation in plants are influenced by precipitation and will be limited under dry conditions due to a reduced rate of transpiration (Keikotlhaile 2011).

Impacts of Climate Change on Food Safety Threats to food safety posed by the climate change can be predominantly categorized into three:

(i) **Food-borne contamination:** It has been reported that a warmer climate in combination with inappropriate food handling may contribute to increased incidences of food-borne diseases (IPCC 2014). In many countries, the main food-borne pathogens are *Salmonella*, *Campylobacter* and *Escherichia coli.* In temperate countries, strong seasonal patterns to the incidence of food-borne diseases have been observed (Seguin 2008). The links between ambient temperature and food-borne pathogen infections have been demonstrated (Seguin 2008).

(ii) **Mycotoxin contamination of food grains:** Mycotoxins are toxins produced by *Alternaria*, *Aspergillus*, *Fusarium* and *Penicillium.* Probably the most commonly known mycotoxin is the highly carcinogenic aflatoxin, which is produced by *Aspergillus flavus.* Mycotoxins are highly dependent on appropriate temperatures and water availability. Cool and temperate climates result in increased presence of aflatoxin due to increased *Aspergillus* occurrence, while tropical climates may experience a decline due to unsuitable high temperatures for *Aspergillus* (Paterson and Lima 2010). The major problem with the interactive effect of climate change on fungal growth and mycotoxin production is that mycotoxins may contaminate staple cereals such as wheat or corn (Medina 2017) which have an enormous importance for food security.

(iii) **Contaminants and residues:** Climate change will also affect the contamination of food sources with chemicals, such as plant protection product residues. As already discussed in this chapter, the increased use of pesticides on crops may lead to excessive residues and acute food safety risks.

Conclusions and Probable Strategies to Mitigate Effect of Climate Change on Plant Diseases

Over the next 100 years, global temperatures are expected to rise along with increasing levels of atmospheric O_3 and CO_2. Severe weather events like heat waves, erratic rainfall patterns and drought will probably become more common. Due to temperature rise, there may be an increase in crop production because of longer growing seasons in temperate regions. Simultaneously, climate change will affect diseases. There will be an increase in plant disease intensity, frequency of outbreaks and the

introduction of pathogens to new areas. According to Helfer (2014), increases in temperature had a beneficial effect on survival of the wheat leaf rust pathogen depending on the availability of leaf wetness and humidity. Contrary to this, the effect of increased CO_2 varied among susceptible cultivars, while increased O_3 had a negative effect. Stem rust resistance due to *R* gene *Sr31* is also in danger under threat of the Ug99 race of stem rust caused by *P. graminis* f. sp. *tritici* due to climate change. There is a threat perception of potato late blight and major diseases of rice, namely, sheath blight (*R. solani*) and blast (*P. oryzae*) due to elevated CO_2 concentrations and high temperatures. In arid, hot conditions, incidence of *M. phaseolina* is higher, and there are chances for the introduction of this pest to new areas under changing climatic conditions (Fones and Gurr 2017).

The geographical and temporal distribution of diseases and management methods will have to be modified according to climate change scenarios. The fungicide residue dynamics in foliage can be altered by temperature change and variable rainfall. The degradation of products can also be altered. Plant physiology or morphology changes because of high concentration of CO_2 in the atmosphere and variations in precipitation and temperature. Therefore, it is important to devise strategies for plant disease management in respect to climate change. The major consequence of climate change in the pathogen-host interaction is genetic resistance to diseases in plant. Plant physiological changes lead to modification in the resistance mechanisms of plant cultivars obtained as a result of genetic engineering and traditional methods. Current disease resistance breeding programs should include traits for tolerance to abiotic factors such as heat stress, elevated CO_2 and water stress. In addition, genome-wide association mapping can be used to understand and target quantitative trait loci for complex traits for abiotic and biotic stresses.

New climate-resilient strains of biocontrol agents with high competitive ability to survive in introduced environment may be needed. Region- and crop-specific strains may have to be incorporated and integrated into pest management approach. IPM is an ecosystem-based strategy, which emphasizes the long-term prevention of pests or their damage through a combination of methods such as cultural practices, habitat management, use of resistant varieties and biological control (Strand 2000). Detailed evaluation of different cropping systems can suggest changes in planting dates and cultural and other agronomic practices to avoid coinciding the susceptible stage of hosts with the virulent phase of the pathogen. While well-developed models for major crops exist, in the case of plant diseases, models are limited to a few major pathogens (Newbery et al. 2016). De Wolf and Isard (2007) suggested disease-forecasting models with improved quality are needed to guide farmers. This type of prediction tool may allow farmers to respond timely and efficiently with public-private partnership. Under changing climatic conditions, crops and varieties may spread to regions and locations where they have not been grown previously.

It is important to understand the potential effects of climate change on disease epidemics on a spatial and temporal scale. However, due to lack of multifactor plant disease simulation models, it is difficult to predict how climate variability will affect disease development and their management. In general, high temperatures and an increased concentration of atmospheric CO_2 will lead to changes in pathogen populations as well as an increased level of susceptibility to plant diseases.

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