OPEN ACCESS Freely available online

Journal of
Plant Pathology & Microbiology

Review Article

Role of Individual Components of Disease Triangle in Disease Development: A Review

Alina Pokhrel*

Tribhuvan University, Institute of Agriculture and Animal Sciences, Nepal

ABSTRACT

Food security has become an important issue at present world. It is estimated that, on a global scale, pathogens and pests are reducing crop yields for five major food crops namely, rice, wheat, maize, soybean and potato by 10 to 40 percent. Mitigating the losses due to plant diseases will obviously help in meeting the growing demand for quality food production. For this, all the factors affecting disease development must be understood clearly and appropriate management strategies must be followed. This review discusses these three major factors; host, pathogen and environment. Temperature, light, relative humidity/moisture are important aerial and edaphic environmental factors that are considered to have major impact on disease development. Development of resistant cultivars and suppressing virulence factors in pathogens are critical to disease control.

Keywords: Disease development; Disease triangle; Pathogen; Host; Environment; Host resistance; Virulence factor

INTRODUCTION

Plant disease, an impairment of the normal state of a plant that interrupts or modifies its vital functions [1]. It is a sustained or progressive impairment of an organism's cells or tissues that causes structural or functional abnormalities. Symptoms of diseases vary from marring only the physical appearance of the plant they infect to affecting tissues critical to the organism's growth, weakening or even killing the infected plant. The disease triangle is a concept that illustrates the importance of the necessary three elements; just as there are three sides of a triangle, there are three critical factors necessary for a disease to develop. A disease occurs when a virulent disease causing agent, or pathogen, meets the susceptible host under environmental conditions favourable to disease development [2]. However, some literatures have added either 'time' or 'human interference' as fourth factor (making a disease square), but, as the other three aspects have a degree of human influence on a particular time, the disease triangle is sufficient as a framework for discussing the various factors that affect disease [3]. It is the balance of the interactions among three elements that determines whether or not disease develops to destructive levels in a particular situation. The susceptibility of the host and the activity of pathogen can be affected by the environment. For example, by providing the conditions of leaf wetness required for spore germination and infection, plant becomes more susceptible to pathogen. Similarly, by secreting chemical factors, the pathogen and the host can affect each other. As well as the hosts can influence the environment by influencing the microclimate within their canopy. Therefore, it is necessary to study the environmental factors influencing disease development, the population genetics of the host resistance and the susceptibility and evolutionary potential of the pathogen populations to produce new races that may be more virulent to hosts or more resistant to pesticides, in order to develop a forecasting system. This review aims at understanding the factors and their interactions as a whole as understanding this will allow prediction of disease outbreaks at a particular place at a particular time.

LITERATURE REVIEW

Pathogen Components: For a pathogen to be able to develop disease in host, it must be virulent, be able to adapt to the host environment, along with having high dispersal and survival efficiency. In certain part of the time in their life cycles, most pathogens go through dormancy. When a pathogen is dormant, no disease can occur. Plants have evolved a complex and multi-layered immune system over time that is effective in warding off most microbial infections [4]. At the same time, numerous microbes - such as bacteria, fungi, oomycetes and viruses have evolved the ability to cause disease in plants [5]. Virulence is the ability of a pathogen to invade and multiply within the host. The unique virulence factors produced by individual pathogens, which determine the extent and severity of disease they may cause, makes some pathogens more virulent than others. Microbial virulence factors include all those molecules produced by pathogens that invade and harm a susceptible host. Any molecule such as plant cell-degrading enzyme, toxin, hormone, capsules, lipoproteins,

Correspondence to: Alina Pokhrel, Tribhuvan University, Institute of Agriculture and Animal Sciences, Nepal, Tel: +9860417504; E-mail: pokhrelaleena@gmail.com

Received: June 19, 2021; **Accepted:** October 01, 2021; **Published:** October 08, 2021

Citation: Pokhrel A (2021) Role of Individual Components of Disease Triangle in Disease Development: A Review 12:573.

siderophore, or extracellular polysaccharide that occurs on the microbial cell surface or is translocated to an extracellular environment where it harms the host cells, is a virulence factor [6]. Not only this, intracellular changes in metabolic regulatory networks, governed by protein sensors or regulators and non-coding regulatory RNAs are also known to contribute to virulence. Furthermore, some secreted microbial products have the ability to manipulate host machinery by entering inside the host cell, contributing to the success of the infection.

Exploiting the platforms such as Next-Generation Sequencing (NGS) and its companion applications like proteomics have helped to gain insights into how these plant pathogens develop the sophisticated strategies to attack. Various pathogenic organisms use various virulence factors to develop disease in plants. Gram-negative bacterial pathogens such as *Pseudomonas syringae, Ralstonia solanacearum, Xanthomonas* and *Erwinia spp*. use the conserved Type III Secretion System (TTSS) to translocate virulence-mediating effector proteins into the host cell [7]. *Agrobacterium tumefaciens*, the causal agent of crown gall disease, injects into the plant cell a fragment of its plasmid DNA (T-DNA) and several virulence proteins via a Type IV Secretion System (TFSS) [8]. The soil-borne pathogen *Ralstonia solanacearum* uses a Type III Secretion System (T3SS) to inject effector proteins into plant cells (15). Different bacteria, interacting with different hosts, have independently evolved Type 3 Secretion System (T3SS) effectors capable of interfering with similar functions in their host cells. These bacteria manage to do so by mimicking components of their eukaryotic hosts and some have been shown to target key cell mechanisms [9]. Bacterial T3SS effectors that directly interfere with their host ubiquitin/proteasome pathway have recently been identified and studied in *Pseudomonas syringae* [10]. Effectors are generally associated with the biotrophic growth phase of fungi, where they play a crucial role in keeping host cells alive and the immune system suppressed. This has been observed in transcriptomics studies with *Colletotrichum higginsianum, Colletotrichum orbiculare, Colletotrichum graminicola, Fusarium oxysporum, Magnaporthe oryzae, Zymoseptoria tritici, Leptosphaeria maculans, and Ustilago maydis* [11]. The roles of the peroxisomes in the rice blast fungus *Magnaporthe oryzae* and *C. higginsianum* gene *ChSTE7* is involved in regulation of vegetative growth, appressorial formation, and invasive growth in host tissues. This is an important and conserved virulence factor affecting infection of *C. higginsianum* on cruciferous plants [12]. As a necrotrophic fungus, the wheat pathogen Fusarium graminearum Schwabe (teleomorph Gibberellae zeae) produces, among other things, cell wall-degrading enzymes and toxins [13]. The dimorphic maize (Zea mays) smut fungus *Ustilago maydis* utilizes pheromone role for pathogenicity [14]. Some pathogens use pectic enzymes as virulence factors such as the pathogenic bacterium *Erwinia chrysanthemi* utilizes apectolytic enzyme, pectate lyase C for pathogenesis [15]. Similarly, small proteins *CgDN3* from *Colletotrichum gloeosporioides* is required for pathogenicity on Stylosenthes [16] Plant viruses need to confront plant defence mechanisms and to hijack the functions of different host factors in order to accomplish their life cycle [17]. A virus can cause productive infection only in those plants that have not developed specific defensive responses to its virulence factors. Although viruses may adopt various strategies to go about achieving this, it is believed the most usual strategy they adopt is that of producing silencing suppressors [18]. They utilize the plant machinery for their nucleic acid and protein synthesis and they take advantage of the plant's transport system to spread, locally through plasmodesmata and systemically through the phloem [19].

Not only virulence, study of the adaptive potential of plant pathogen to host environment and external environment is essential to manage them. Pathogens evolve due to various forces such as spatial dispersion, recombination, genetic drift, selection by the host plant resistance, etc. [20]. Many pathogens have developed resistance to chemical control measures to overcome host resistance. For example, many researches have reported that *Colletotrichum gloeosporioides* has developed ability to overcome host resistance [21,22] and fungicide resistance [23]. Similarly, *Plasmopara viticola* have showed high evolutionary potential and reported for fungicide resistance [24]. Similarly, if we light on spatial adaptation, though yellow rust of wheat caused by *Puccinia striiformis* f. sp. tritici, has previously shown preferential development in cold areas, certain strains of it were recently able to invade warmer regions due to their developed adaptation [25]. Population density also has significant effect on disease development. It is reported that the appearance of bacterial canker symptoms is closely related to the population of the pathogen colonizing the host. The pathogen can survive as an effective endophyte in tomato but apparently has to establish an endophytic population of >108 CFU g−1 plant tissue to induce disease symptoms [26]. Knowledge of the pathogen adaptation ability and virulence factors is crucial for designing effective crop protection strategies.

Host factors: A pathogen at critical stage of its life cycle to infect, must come in contact with a susceptible host at appropriate growth stage of host, for a disease to develop. Pathogen-host interactions are often very specific as plant pathogens cause diseases to specific hosts only where they can get their food source for growth and development. Some pathogens attack only a particular genus or even species of organism, while other pathogens have a wide range of hosts belonging to many families. For example, *Cercospora sojina* causes frogeye leafspot disease on soybean but does not cause disease on corn, alfa-alfa, etc. At the same time, *Fusarium oxysporum* causes fusarium wilt in several hundred species of crops including potatoes, tomatoes, legumes, melons, bananas, etc. This may be related to evolution of the pathogen and the host. Additionally, not all individuals within that species will have the same level of susceptibility, even if a species is susceptible. The infection process is either successful or unsuccessful depending on the type of host, whether susceptible or resistant, respectively [27]. 'Makassane', a new IRRI-bred rice variety has improved resistance to blast and the variety 'Vijay' of wheat is resistant to stem rust Ug99 [28]. An individual plant may have resistant genes that defend it against infection. The capability of plant pathogen for reaching the full infection occurs after it overcomes the plant defences. If the host is resistant to a particular pathogen, even when the pathogen is present under favourable environmental conditions, a disease will not occur. The prior health of the host is often important in determining the occurrence of disease as it can be seen in most cases that vigorous, strong-growing, non-stressed plants are less susceptible to disease than plants growing under stress conditions.

Certain growth stages of plants are more vulnerable while other stages may be resistant to pathogen for a particular pathogen to attack. The response of host plants to pathogens often depends on the developmental stage of the host when challenged by the pathogen [29]. For example, in young tomato plants, disease

symptoms caused by *Clavibacter michiganensis* sub sp. *michiganensis* appeared earlier than in older plants [30]. Infestation by E. cruciferarum on oilseed rape increases as the host plant ages or matures [31]. Cramer reported that older onion plants were less susceptible than seedlings/dormant bulbs to Fusarium basal rot, incited by *Fusarium oxysporum* f. sp. Cepae [32]. Similarly, strawberry becomes more susceptible to the pathogen *Botrytis cinerea* once it flowers [33]. Additionally, it has been found that younger leaf tissues of wheat had greater disease severity to wheat stem rust disease [34]. Not only this, onset of developmental resistance to *Xanthomonas oryzae pv. Oryzae* can be seen in rice as the crop enters its vegetative phase [35,36]. These all examples represent age-related resistance. In this age-related resistance, as plant tissues age, they become less susceptible to pathogens infection. The molecular mechanism for the resistance in older plants has not been described clearly in literatures. However, changes in contents and biochemical activities of proteins, enzymes, and phenolics during developmental stages of host plants could assist resistance.

Plant densities have significant influence on the development of foliar diseases in many crops. A denser canopy provides ideal moisture and temperature conditions for the development of *Sclerotia sclerotiorum* [37,38] Higher plant densities support foliar diseases like botrytis gray mold of chickpea [39]. The severities of Late leaf spot and rust diseases were significantly more in higher plant densities than in low plant densities in groundnut [40]. On the contrary to commonly held belief, powdery mildew severity showed negative correlation on barley with increasing plant density [41].

Environmental factors: Environmental factors have been considered to have the major impact on disease development. Even if a susceptible host and a virulent pathogen are present in a certain locality, serious disease will not occur unless the environment favours its development. Some pathogens are capable of causing disease throughout the world, in areas where plants susceptible to them are grown, for they can tolerate a wide range of environmental conditions. Other pathogens may be less tolerant of environmental variations and are likely to be restricted in their geographical distribution. Not only may environment directly influence either host or pathogen, it may also affect disease incidence and development, because of the interaction of effects on the host and pathogen. Host plant's physiological state, including its growth, immune system and abiotic stress response are affected by the environmental condition. Similarly, environment has profound effect on a pathogen's survival, germination, expression and delivery of virulence factors. The same plant can be fully resistant or susceptible, and the pathogen could range from being able to cause severe disease to being only weakly pathogenic due to these variable environmental conditions. Many environmental conditions affect plant disease development, including temperature, light, water availability, soil fertility, wind speeds, and atmospheric ozone, methane and CO_2 concentration. Plant diseases develop under an optimal range of climatic variables; however, the occurrence and severity of a disease in an individual plant is defined by the deviation of each climatic variable within the optimal range for disease development, thus climate affects all life stages of the pathogen and host [42]. The environment may affect plant pathogen, therefore, survival, vigour, rate of multiplication, sporulation, direction, distance of dispersal of inoculums, rate of spore germination and penetration can be affected [43].

Temperature and CO_2 concentration in air: Temperature is the determining factor in the seasonal and regional incidence of disease and determines the geographic distribution. There is an optimal temperature range for every plant-pathogen interaction, at which disease develops. There is also an optimum temperature for a pathogen for its growth. Different stages in a life cycle of a pathogen may have different optimum temperatures. If we take an example of fungi, different growth stages of fungi, such as the production of spores, their germination, and the growth of the mycelium, may have slightly different optimum temperatures. Daytime temperatures of 35 °C and night temperatures of 27°C are most favourable for *Xanthomonas oryzae* bacteria to colonize rice [44]. Yellow rust caused by *Puccinia striformis* f. sp. tritici (Pst) is a major disease of wheat in temperate areas with cool and wet summers and mild winters [45]. High temperatures during the growing season have been considered a constraining factor for Pst development and long-distant dissemination [46]. High atmospheric CO_2 concentrations, temperatures, and changes in precipitation patterns as well as frequency of extreme weather phenomena will significantly affect crop production and therefore the presence of diseases will be altered under these conditions [47]. Not only the incidence, but the climatic condition influences the temporal and spatial distribution of plant diseases. It is anticipated that the projected increase in global temperature will most likely change the regional distribution in which a crop is susceptible to a particular pathogen [48]. New races may evolve on a faster rate under elevated temperature and CO_2 as evolutionary forces act on massive pathogen populations boosted by a combination of increased fecundity and infection cycles under favourable microclimate [49]. When the CO_2 level and the temperature were elevated, an increase on powdery mildew disease was observed on zucchini, Alternaria leaf spot on rocket salad, black spot and downy mildew on basil, Allophoma tropica on lettuce and Phoma leaf spot on garden beet [50]. Increased CO_2 concentration has been found to increase disease severity in rice [51]. Similarly, the severity of wheat diseases also increases when plants and pathogens are acclimatized to elevated CO_2. For the fungal pathogen *Fusarium graminearum*, elevated CO_2 levels not only increased the susceptibility of wheat varieties (irrespective of the resistant and susceptible genotypes evaluated), but also increased the virulence of the fungal isolate [52], resulting in more severe disease overall. In contrast, in oomycete-plant interactions between soybean and *Peronospora manshurica*, CO_2 concentrations of 550 ppm decreased the severity of the disease by more than 50% [53]. The development of powdery mildew disease of Brassica napus was more at 22/17°C than at 14/10°C [54]. Higher temperatures resulted in more rigorous replication of Turnip Crinkle Virus (TCV) in Arabidopsis plants [55], and facilitated the spread of tobacco mosaic virus or turnip mosaic virus by weakening the plant defence responses [56]. Higher temperature caused faster disease symptom development on plants affected by peanut stunt virus, which after reaching a plateau, dropped precipitously [57]. Schwartz et al. reported that leaf blight, caused by *Pontoea. ananatis*, is favored by prolonged periods of rain, high Relative Humidity (RH), and temperatures of 28 to 35°C during the bulbing stage [58]. Disease severity caused by *Pontea* spp. and *Erwinia cowanii* on onion increased with both increasing temperature and RH, whereas disease progression was restricted under cool temperatures (15°C) and low RH (40 to 60%) [59].

Light: Light plays an important role in regulating the chemical and physiological processes in most organisms. There are many

reports that have indicated that light influences both the defence responses in the host and the virulence of the pathogens [60]. Light has indirect effects on moisture on leaf surfaces, which in turn has impact on disease development. In a study conducted. [61], both the establishment of local defence responses and Systemic Acquired Resistance (SAR) are affected by light density. SAR development in response to infection by virulent bacteria is completely lost when primary infection occurs in the absence of light. Another experiment Chen LJ, et al. [62], demonstrated that higher light intensity increased susceptibility of *Nicotiana tabacum* to Cucumber mosaic virus and accelerated systemic symptoms compared with plants grown in lower light intensities. Expression of Salicylic Acid mediated defence genes was strongly depressed in virus inoculated plants grown in high light compared to low light intensities. It is also known to have effect on sporulation of several fungal and oomycete species, and the circadian clock of one fungal phytopathogen has been linked to the pathogen's virulence programme [63]. The interaction of light intensity and photoperiod have been found to affect the germination of pathogen. Studies with *Brassica graminis f*.sp. hordei showed that increased light intensity lengthened the latent period, with the highest light intensities causing a reduction in sporulation [64]. Ayres found that white light and shorter wavelengths stimulated germ tube development in *Erysiphe pisi* [65]. Plants acclimated to high light displayed increased resistance against virulent *Pseudomonas Syringae* DC3000 both in tissues exposed directly to excess light and in systemic leaves, analogous to the establishment of Systemic acquired resistance [66]. A study showed that constant dark inhibited *Hyaloperonospora arabidopsidis* (Hpa) growth while constant light supported higher level of Hpa biomass production than that in normal light/dark or constant regimes [67]. Similarly, disease incidence and internal bacterial growth in the tomato seedlings were significantly reduced when grown under green and red light [68]. Thus, light intensity, photoperiod and wavelength have direct or indirect effect on disease development.

Relative humidity and precipitation: Relative Humidity (RH) has been widely studied or applied as a predictor for disease development in various pathosystems at different stages, including fungal growth and sporulation of numerous plant pathogens [69]. Atmosphere humidity and precipitation in the form of rain, fog and dew affect seasonal and local incidence of diseases. Moisture makes the plant cells turgid which facilitates the pathogen to grow in the host. Raindrops facilitate the liberation and dispersal of pathogens like fungi and bacteria, and when the propagules are dispersed, conditions are likely to be suitable for germination and infection. Many diseases get worse as leaf wetness period increases. For example, gray mold disease caused by *Botrytis cinera* on roses increases with longer wet periods. Such is the case with anthracnose of tomato (*Colletotrichum coccodes*), leaf rust in wheat (*Puccinia recondita)* and stem rot in soybean(*Diasporthe phaseolorum*) [70]. Majority of leaf and fruit diseases caused by fungi, water molds and bacteria are favoured by high RH. The infection, colonization and conidiation of *M. oryzae* on perennial ryegrass were highly dependent on the atmospheric moisture levels. Near-saturated humidity conditions were most favourable for *M. oryzae* infection and conidiation [71]. Similarly, higher relative humidity around the plants resulted in significantly greater level of disease caused by *Erysiphe graminis* f. sp. Hordei [72]. The optimum relative humidity for the initiation of *Rhizopus* soft rot is in the range of 75-84% and few infections occur at 93-99% [73]. Thus, if we become able to maintain RH of 85-90%, the disease doesn't develop. There was a large increase in disease severity by *Didymella rabiei* on chickpea with increase in RH from 95% to 100% [74]. In contrast, the severity of tomato powdery mildew caused by *Oidium lycopersici* decreased with increase in RH from 80% to 95% [75], which indicates tomato powdery mildew is favoured by dry condition. While in case of grapevine powdery mildew, disease incidence and severity increased increasing humidity to an optimum near 85% RH, and then appeared to plateau or decrease marginally at higher values [76].

Edaphic factors: The soil moisture, soil type, pH, temperature, soil microbiomes and nutrients play an important role in disease incidence and development. The prevalence of infection and severity of diseases due to soil borne pathogens is determined by these edaphic factors. Soil moisture may exist as rain or irrigation water on plant surface or around the roots. Moisture is indispensable for the germination of fungal spores, penetration of the host by germ tube and initial development and severity. The disease development by stem gall of coriander was maximum (77.5%) at 25°C soil temperature giving 52.5% disease incidence and 30% disease severity, as well as 45% of soil moisture level was favourable for disease development and resulted in 70.25% disease incidence and 35.25% disease severity. Very low and very high soil moisture and temperature were not favourable for disease development [77]. High soil moisture favours development of water mold fungi such as *Pythium* and *Phytophthora* whereas low moisture or drought favours diseases like charcoal rot diseases of field crops, aspergillus ear rot and fusarium diseases [78]. Elevated moisture in soil raises CO_2 level that makes roots more susceptible to root rotting organisms. The clubroot disease caused by *Heteroconium chaetospira* in Chinese cabbage was non-symptomatic in alkaline soils at a pH range from 7 to 7.2. Also the pathogen-treated plants were completely protected from clubroot at low to moderate soil moisture contents (40 to 60%), but they were severely damaged at high soil moisture contents (80%) [79]. Likewise, in general, root rot caused by Phytophthora are inhibited by lower soil pH as low soil pH restricts sporangium formation, zoospore release and motility of the pathogen [80]. In certain cases, high pH reduces the availability of soil nutrients to the pathogen and hence restricts their effects as in the case of Fusarium wilt [81]. When nutrient levels are below optimum, the effects of soil-borne pathogens are more serious, especially for immobile nutrients. Insufficiency of nutrients can cause root starvation and plant death, even though the pathogen itself may not be toxic. It can be said that healthy, well-nourished plants are less susceptible to pathogens and disease than nutrient-deficient plants. Potassium deficiency impairs the synthesis of proteins, starch, cellulose and cell wall development, which may directly correlate to a pathogen's ability to enter plant tissue; Calcium plays an important role in plant disease resistance as it maintains structural integrity of plant membranes. Under Ca deficiency, compounds such as sugars and amino acids may leak into areas between cells where these compounds become available to pathogens [82]. Similarly, boron is known to be toxic to pathogenic fungi [83]. Phosphorus is also found to increase resistance to diseases in plants. The role of Nitrogen in disease resistance is quite easily demonstrated but the effect of N is also seen variable. This is due to the different response depending on the type of the pathogen. Regarding the obligate parasites, e.g., Puccinia graminis and

Erysiphe graminis, when there is high N supply there is an increase in severity of the infection; however, when the disease is caused by facultative parasites, e.g., *Alternaria, Fusarium* and *Xanthomonas spp*., high N supply decreases the severity of the infection [84]. It is found that soil type also affects the activity of pathogens in soil. High clay content in soil with high cation exchange capacity can suppress the infection rate of Pythium ultimum[85] and high organic matter content can suppress disease like Fusarium wilt of banana [86]. Addition of composts or organic matter enhances the plant defence system through induced system resistance [87]. Soil microbial community also influence the incidence of disease. For example, actinobacteria [88] and arbuscular mycorrhizal fungi [89] have the ability to inhibit the growth of soil-borne pathogens in many ways. Thus working on the concept of disease suppressive soil is a most to manage soil-borne pathogens.

CONCLUSION

The world population is increasing at a faster rate. But the pace of agricultural production is not parallel with the increasing population. One of the major limiting factors in agricultural production is the incidence of diseases. The use of chemical control measures has severely affected the sustainability of the environment. Therefore, in order to increase food production keeping in mind the safety of environment, it is very much important to understand each individual factors affecting disease development in detail. For successful management of disease, some components of the disease triangle must be disturbed. The first and best defence against pathogens is a healthy and resistant plant. Though breeding for resistant cultivars is one of the most important methods and long-practised idea for dealing with disease out-breaks, this alone is not sufficient to eliminate risk. Although the manipulation of the environmental parameters is difficult at field levels, negative effects of environment can be avoided through some management strategies such as choice of planting date and place. Understanding the combined effects of environmental parameters on pathogens is equally important to control disease. This will help predict and prepare for the challenges ahead. Similarly, knowing and understanding the disease cycle for a particular disease and disturbing any step in the disease cycle will be very helpful in managing the disease. All in all, the concept of integrated disease management should be followed by carefully understanding all the factors affecting disease incidence and development.

REFERENCES

1. Kelman A, Pelczar MJ, Pelczar RM, Shurtleff MC. Encyclopedia Britannica. Plant Dis. 2020.
2. Agrios GN. Plant Pathology. (5th edn), Elsevier-Academic Press. 2005.
3. Keane P and Kerr A. Factors affecting disease development. In J.F. Brown and H.J. ogle(Ed.). Plant pathogens and plant diseases (pp 287-299). Rockvale Publication. 1997.
4. Chisholm ST, Coaker G, Day B, Staskawicz BJ. Host-microbe interactions: Shaping the evolution of the plant immune response. Cell. 2006;124:803–814.
5. Speth EB, Lee YN, He SY. Pathogen virulence factors as molecular probes of basic plant cellular functions. Curr Opin Plant Biol. 2007;10(6):580–586.
6. Surico G. The concepts of plant pathogenicity, virulence/avirulence and effector proteins by a teacher of plant pathology. Phytopathol Mediterr. 2013;52:399-417.
7. He SY, Nomura K, Whittam TS. Type III protein secretion mechanism in mammalian and plant pathogens. Biochim Biophys Acta. 2004;1694:181–206.
8. Citovsky V, Kozlovsky SV, Lacroix B, Zaltsman A, Dafny-Yelin M. Biological systems of the host cell involved in Agrobacterium infection. Cell Microbiol. 2007;9:9–20.
9. Angot A, Peeters N, Lechner E, Vailleau F, Baud C. *Ralstonia solanacearum* requires F-box-like domain-containing type III effectors to promote disease on several host plants. Proc Natl Acad Sci USA. 2006;103(39):14620-14625.
10. Janjusevic R, Abramovitch RB, Martin GB, Stebbins CE. A Bacterial Inhibitor of host programmed cell death defenses is an E3 ubiquitin ligase. Science. 2006;311(5758):222-226.
11. Charlotte DH, Rep M. Adaptation to the host environment by plant-pathogenic Fungi. Annu Rev Phytopathol. 2017;55:427-450.
12. Yang J, Hsiang T, Bhadauria V, Chen X, Li G. Plant fungal pathogenesis. Biomed Res Int. 2017.
13. Rauwane ME, Ogugua UV, Kalu CM, Ledwaba LK, Woldesemayat AA. Pathogenicity and virulence factors of fusarium graminearum including factors discovered using next generation sequencing technologies and proteomics. Microorganisms. 2020;8(2):305.
14. Vadlapudi V, Naidu KC. Fungal pathogenicity of plants: Molecular approach. Eur J Exp Biol. 2011;1(1):38-42.
15. Herron SR, Benen JAE, Scavetta RD, Visser J, Jurnak F. Structure and function of pectic enzymes: Virulence factors of plant pathogens. National Academy Of Sciences. (NAS Colloquium) Virulence and Defense in Host-pathogen interactions: Common features between plants and animals. 2001; 10.17226/10099.
16. Stephenson S, Hatfield J, Rusu AG, Maclean DJ, Manners JM. CgDN3: An essential pathogenicity gene of *Colletotrichum gloeosporioides* necessary to avert a hypersensitive-like response in the host *Stylosanthes guianensis*. Molecular Plant- Microbe Interaction. 2000;13:929-941.
17. Pallas V, Garcia JA. How do plant viruses induce disease? Interactions and interference with host components. J Gen Virol. 2011;92:2691–2705.
18. Li F, Ding SW. Virus counter defense: Diverse strategies for evading the RNA-silencing immunity. Annu Rev Microbiol. 2006;60:503–531.
19. Nelson RS, Citovsky V. Plant viruses. Invaders of cells and pirates of cellular pathways. Plant Physiol. 2005;138:1809–1814.
20. McDonald BA, Linde C. Pathogen population genetics, evolutionary potential and durable resistance. Annu Rev Phytopathol. 2002;40:349–379.
21. Abang MM, Green KR, Wanyera NW, Iloba C. "Characterization of *Colletotrichum gloeosporioides* penz from yam (*Dioscorea* spp.) in nigeria," in Proceedings of the 7th Triennial Symposium of the International Society for Tropical Root Crops, Cotonou. 2001;613–615.
22. Abang MM. Genetic Diversity of *Colletotrichum gloeosporioides Penz*. Causing anthracnose disease of yam (*Dioscorea* spp). 2003.
23. Bayart JD, Pallas B. Tolerance of yam anthracnose to benzimidazoles: Results of the first study conducted in Guadeloupe. Phytoma. 1994;461:37–40.
24. Blum M, Waldner M, Gisi U. A single point mutation in the novel *PvCesA3* gene confers resistance to the carboxylic acid amide fungicide mandipropamid in *Plasmopara viticola*. Fungal Genet Biol. 2010;47:499–510.
25. Vallavieille CP, Bahri B, Leconte M, Zurfluh O, Belaid Y. Thermal generalist behaviour of invasive *Puccinia striiformis* f. sp. tritici strains under current and future climate conditions. Plant Pathol. 67:1307–1320.

26. Gartemann KH, Kirchner O, Engemann J, Grafen I, Eichenlaub R. *Clavibacter michiganensis* subsp. michiganensis: first steps in the understanding of virulence of a Gram-positive phytopathogenic bacterium. J. Biotechnol. 2003;106:179-191.

27. Abdulkhair WM, Alghuthaymi MA. Plant Pathogens, Plant Growth. 2016;10.5772/65325.

28. Timsina K, Ghimire Y, Gauchan D, Subedi S, Adhikari S. Lessons for promotion of new agricultural technology: A case of Vijay wheat variety in Nepal. Agric Food Secur. 2018;7:63.

29. Whalen MC. Host defence in a developmental context. Mol Plant Pathol. 2005;6:347-360.

30. Sharabani G, Shtienberg D, Borenstein M, Shulhani R, Lofthouse M. Effects of plant age on disease development and virulence of *Clavibacter michiganensis* subsp. michiganensis on tomato. Plant Pathol. 2013;62:1114-1122.

31. Kennedy R. Brassicas: forecasting light leaf spot and powdery mildew in vegetable Brassica crops based on 'in field' detection of airborne spores. Agriculture and Horticulture Development Board, 2010.

32. Cramer CS. Breeding and genetics of Fusarium basal rot resistance in onion. Euphytica. 2000;115:159–166.

33. Mertely JC, MacKenzie SJ, Legard DE. Timing of fungicide applications for *Botrytis cinerea* based on development stage of strawberry flowers and fruit. Plant Dis. 2002;86:1019-1024.

34. Farber DH, Mundt CC. Effect of plant age and leaf position on susceptibility to wheat stripe rust. Phytopathology. 2016;107:412-417.

35. Wang YS, Pi LY, Chen X, Chakrabarty PK, Jiang J. Rice XA21 binding protein 3 is a ubiquitin ligase required for full Xa21-mediated disease resistance. Plant Cell. 2006;18:3635– 3646.

36. Panter SN, Jones DA. Age-related resistance to plant pathogens. Adv Bot Res. 2002;38:251-280.

37. Boland GJ, Hall R. Epidemiology of sclerotinia stem rot of soybean in Ontario. Phytopathology. 1988;78:1241-1245.

38. Jaccoud-Filho DS, Sartori FF, Manosso-Neto M, Vrisman CM, Pierre MLC. Influence of row spacing and plant population density on management of "white mould" in soybean in southern Brazil. 2016;10:161-168.

39. Pande S, Johansen C, Rao NJ (1998) Management of botrytis gray mold of chickpea. A review. In: Recent advances in research and management of botrytis gray mold of chickpea. Summary proceedings of the Fourth Working Group Meeting to discuss collaborative research on Botrytis gray mold of chickpea. 1998;23-40.

40. Pande S, Rao JN. Effect of plant population densities on the severity of late leaf spot and rust of groundnut. Plant Pathol J. 2002;18(5):271-278.

41. Finckh MR, Gacek ES, Czembor HJ, Wolfe MS. Host frequency and density effects on powdery mildew and yield in mixtures of barley cultivars. Plant Pathol. 1999;48:807-816.

42. Agrios GN. Plant Pathology. 5th Ed. Elsevier, 2005.

43. Kang WS, Yun SC, Park EW. Nonlinear regression analysis to determine infection models of *Colletotrichum acutatum* causing anthracnose of chili pepper using logistic equation. Plant Pathol J. 2010;26(1):17-24.

44. Horino O, Mew TW, Yamada T. The effect of temperature on the development of bacterial leaf blight on rice. Ann Phytopathol Soc Japan. 1982;48:72–75.

45. Chen W, Wellings C, Chen X, Kang Z, Liu T. Wheat stripe yellow rust caused by *Puccinia striiformis* f. sp. tritici. Mol Plant Pathol. 2014;15:433-446.

46. Brown JKM, Hovmoller MS. Aerial dispersal of pathogens on the global and continental scales and its impact on plant disease. Science. 2002;297:537-541.

47. Rosenzweig C, Tubiello FN. Adaptation and mitigation strategies in agriculture: an analysis of potential synergies. Mitig Adapt Strateg Glob Chang. 2007;12:855-873.

48. Velásquez AC, Castroverde C, He SY. Plant-Pathogen Warfare under Changing Climate Conditions. Curr biol. 2018;28(10): R619-R634.

49. Garrett KA, Nita M, De Wolf ED, Esker PD, Gomez-Montano L. plant pathogens as indicators of climate change. in climate change: Observed impacts on planet earth: (Second Edn). Clim Change.2016;325-338.

50. Gullino ML, Pugliese M, Gilardi G, Garibaldi A. Effect of increased CO_2 and temperature on plant diseases: a critical appraisal of results obtained in studies carried out under controlled environment facilities. J Plant Pathol. 2018;100:371–389.

51. Kobayashi T, Ishiguro K, Nakajima T, Kim HY, Okada M. Effects of elevated atmospheric CO_2 concentration on the infection of rice blast and sheath blight. Phytopathology. 2006;96:425–431.

52. Váry Z, Mullins E, McElwain JC, Doohan FM. The severity of wheat diseases increases when plants and pathogens are acclimatized to elevated carbon dioxide. Global Change Biology. 2015;21:2661–2669.

53. Eastburn DM, Degennaro MM, Delucia EH, Dermody O, McElrone AJ. Elevated atmospheric carbon dioxide and ozone alter soybean diseases at SoyFACE. Glob Change Biol. 2010;16:320–330.

54. Uloth MB, You MP, Barbetti MJ. Plant age and ambient temperature: significant drivers for powdery mildew (*Erysiphe cruciferarum*) epidemics on oilseed rape (*Brassica napus*). Plant Pathol. 2018;67:445-456.

55. Zhang X, Zhang X, Singh J, Li D, Qu F. Temperature-dependent survival of Turnip crinkle virus-infected arabidopsis plants relies on an RNA silencing-based defense that requires DCL2, AGO2, and HEN1. J Virol. 2012;86:6847–6854.

56. Prasch CM, Sonnewald U. Simultaneous application of heat, drought, and virus to Arabidopsis plants reveals significant shifts in signaling networks. Plant Physiol. 2013;162:1849–1866.

57. Obrępalska-Stęplowska A, Renaut J, Planchon S, Przybylska A, Wieczorek P, Barylsky J et al. 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. Front plant sci. 2015;6:903.

58. Schwartz HF, Otto KL, Gent DH. Relation of Temperature and Rainfall to Development of Xanthomonas and Pantoea Leaf Blights of Onion in Colorado. Plant Dis. 2003;87:11-14.

59. Tho KE, Brisco-McCann E, Wiriyajitsomboon P, Hausbeck MK. Effects of temperature, relative humidity, and plant age on bacterial disease of onion plants. Plant Health Prog. 2019;20:200-206.

60. Oberpichler I, Rosen R, Rasouly A, Vugman M, Ron EZ. Light affects motility and infectivity of *Agrobacterium tumefaciens*. Environ Microbiol. 2008;10:2020–2029.

61. Zeier J, Pink B, Muller MJ, Berger S. Light conditions influence specific defense responses in incompatible plant-pathogen interactions: uncoupling systemic resistance from salicylic acid and PR-1 accumulation. Planta. 2004;219:673-683.

62. Chen LJ, Ren H, Deng XG, Li YN, Cha WQ. Effects of light intensity on the susceptibility of *Nicotiana tabacum* to cucumber mosaic virus. J Gen Plant Pathol. 2015;81:399-408.

63. Hevia MA, Canessa P, Müller-Esparza H, Larrondo LF. A circadian oscillator in the fungus *Botrytis cinerea* regulates virulence when infecting *Arabidopsis thaliana*. P N A S. 2015;112:8744– 8749.

64. Aust H. Light intensities affecting germination, incubation and latent period, and sporulation of Erysiphe graminis DC.

f.sp. hordei Marchal. Proceedings of the 3rd Congress of the Mediterranean Phytopathology Union.1972;542-546.

65. Ayres PG. Conidial germination and germ-tube growth of *Erysiphe pisi* in relation to visible light and its transmission through pea leaves. Trans Brit Mycol Soc. 1983;81:269-274.

66. Roden LC, Ingle RA. Lights, rhythms, infection: the role of light and the circadian clock in determining the outcome of plant-pathogen interactions. Plant cell. 2009;21(9):2546–2552.

67. Telli O, Jimenez-Quiros C, McDowell JM, Tor M. Effect of light and dark on the growth and development of downy mildew pathogen *Hyaloperonospora Arabidopsidis*. Plant Pathol. 2020;69:1291– 1300.

68. Nagendran R, Lee YH. Green and red light reduces the disease severity by *Pseudomonas cichorii* JBC1 in tomato plants via upregulation of defense-related gene expression. Phytopathology. 2015;105:412-418.

69. Carisse O, Kushalappa AC. Influence of interrupted wet periods, relative humidity, and temperature on infection of carrots by *Cercospora carotae*. Phytopathology. 1992;82:602– 606.

70. Huber L, Gillespie TJ. Modeling leaf wetness in relation to plant disease epidemiology. Annu rev Phytopathol. 1992;30:553-557.

71. Li Y, Uddin W, Kaminski JE. Effects of relative humidity on infection, colonization and conidiation of *Magnaporthe orzyae* on perennial ryegrass. Plant Pathol. 2014;63:590-597.

72. Harris JG, Manners JG. Influence of relative humidity on germination and disease development in *Erysiphe graminis*. Trans Brit Mycol Soc. 1983;81:605-611.

73. Nelson S. Rhizopus soft rot of sweetpotato. Plant Dis. 2009;68.

74. Jhorar OP, Butler DR, Mathauda SS. Effects of leaf wetness duration, relative humidity, light and dark on infection and sporulation by *Didymella rabiei* on chickpea. Plant Pathol. 1998;47:586-594.

75. Whipps JM, Budge SP. Effect of Humidity on Development of Tomato Powdery Mildew (Oidium lycopersici) in the Glasshouse. Eur J Plant Pathol. 2000;106:395–397.

76. Carroll JE, Wilcox WF. Effects of humidity on the development of grapevine powdery mildew. Phytopathology. 2003;93(9):1137-1144.

77. Gupta M, Leharwan M, Shukla A. Effect of temperature and moisture levels on disease development of stem gall of coriander. Agricultural Science Digest - A Research Journal. 2019;38(4):307-309.

78. Wegulo S, Giesler L, Harveson R, Jackson-Ziems TA, Liu B. Impacts of drought on disease development and management. Plant Pathol. 2013;537.

79. Narisawa K, Shimura M, Usuki F, Fukuhara S, Hashiba T. Effects of pathogen density, soil moisture, and soil pH on biological control of clubroot in Chinese cabbage by *Heteroconium chaetospira*. Plant Dis. 2005; 89:285-290.

80. Jambhulkar PP, Sharma M, Lakshman D, Sharma P. Natural mechanisms of soil suppressiveness against diseases caused by Fusarium, *Rhizoctonia*, *Pythium*, and *Phytophthora*. In: Meghvansi MK, Varma A (eds) Organic amendments and soil suppressiveness in plant disease management, Soil Biol. 2015;46:95-123.

81. Jones JB, Jones JP, Stall RE, Zitter TA. Compendium of tomato diseases. The American Phytopathological Society. 1991.

82. Spann TM, Schumann AW. The role of plant nutrients in disease development with emphasis on citrus and Huanglongbing. Proc Fla State Hort Soc. 2009;122:169–171.

83. Pratt JE. Effect of inoculum density and borate concentration in a stump treatment trial against *Heterobasidion annosum*. For Pathol. 2000;230:277–283.

84. Christos D. Role of nutrients in controlling plant diseases in sustainable agriculture: A review. Agronomy for Sustainable Development. 2008;28(1):33-46.

85. Martin FN, Hancock JG. Association of chemical and biological factors in soils suppressive to *Pythium ultimum*. Phytopathology 1986;76:1221–1231.

86. Stotzky G, Martin RT. Soil mineralogy in relation to the spread of Fusarium wilt of banana in Central America. Plant Soil. 1963;18:317–337.

87. Yogev A, Raviv M, Hadar Y, Cohen R, Wolf S, Katan J, et al. Induced resistance as a putative component of compost suppressiveness. Biol Control. 2010;54(1):46–51.

88. Claessen D, Rozen DE, Kuipers OP, Søgaard-Andersen L, van Wezel GP. Bacterial solutions to multicellularity: A tale of biofilms, filaments and fruiting bodies. Nat Rev Microbiol. 2014;12:115–124.

89. Li B, Ravnskov S, Xie G, Larsen J. Biocontrol of pythium damping-off in cucumber by arbuscular mycorrhiza-associated bacteria from the genus *Paenibacillus*. Bio Control. 2007;52:863–875.