International Journal of Advanced Biochemistry Research

ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating: 5.29
IJABR 2025; 9(4): 741-756
www.biochemjournal.com
Received: 18-02-2025
Accepted: 22-03-2025

Sanchari Burman
Masters Research Scholar, Institute of Agricultural Sciences, Banaras Hindu University, Uttar Pradesh, India

Puja Kundu
M.Sc. Research Scholar, Department of Plant Pathology, Indira Gandhi Krishi Vishwavidyalaya, Raipur, Chhattisgarh, India

Anurag Shukla
Assistant Professor, Department of Plant Pathology, College of Agriculture, Mangalayatan University, Aligarh, Uttar Pradesh, India

Monika
M.Sc., Horticulture (Fruit Science), Chaudhary Charan Singh Haryana Agricultural University, Hisar, Haryana, India

Salini Das
Ph.D., Research Scholar, Department of Plant Pathology, Bidhan Chandra Krishi Viswavidyalaya, Mohanpur, Nadia, West Bengal, India

Jyoti Nughal
Research Fellow, ICAR-IIWBR, Regional Station, Flowerdale, Shimla, Himachal Pradesh, India

Ramachandran Lokesh
Ph.D., Scholar, Department of Plant Pathology, Tamilnadu Agriculture University, Coimbatore, India

Dipanjali Bag
Ph.D., Department of Plant Pathology, Punjab Agricultural University, Ludhiana, Punjab, India

Utpal Kumar Bhattacharyya
Senior Scientist-Cum-Head, ICAR-KVK, Longding, Kanubari, Arunachal Pradesh, India

Corresponding Author:
Sanchari Burman
Masters Research Scholar, Institute of Agricultural Sciences, Banaras Hindu University, Uttar Pradesh, India

The molecular dynamics of bacterial plant pathogenesis: Navigating future microbial challenge

Sanchari Burman, Puja Kundu, Anurag Shukla, Monika, Salini Das, Jyoti Nughal, Ramachandran Lokesh, Dipanjali Bag and Utpal Kumar Bhattacharyya

DOI: https://www.doi.org/10.33545/26174693.2025.v9.i4i.4177

Abstract
This article is a summary of the thirty-fourth New Phytologist Symposium Plant Pathogen Interactions held at the John Innes Centre, Norwich, UK on 16 July, 2009. Plant pathogen interactions have provided fundamental insights into plant biology. Furthermore, they are of cross-disciplinary interest to many scientists including plant, microbial and fungal scientists. In the molecular dialogue between plant and bacterial pathogens, plants have evolved an effective immune system that recognizes conserved microbial structures. This system has been termed PAMP triggered immunity (PTI) since it is initiated upon recognition of microbial elicitors termed pathogen associated molecular patterns (PAMPs). Successful pathogens suppress or evade PTI using an array of effectors. In turn, plants have evolved resistance (R) proteins that recognize effector activity, either directly or indirectly, resulting in effector triggered immunity (ETI). In Gram-negative bacteria, the type III secretion system (T3SS) is central to pathogenicity, and ~29 avirulence proteins (AVRs) are secreted by the *Pseudomonas syringae* pv. tomato T3SS. Recognition of these AVRs by cognate plant R proteins regulates many aspects of gene expression in the plant, often via the action of the defence hormone salicylic acid. Combining microarray analysis of the dynamics of the plant *Arabidopsis thaliana* during the establishment of primordia at infection sites with complementary analysis of the *P. syringae* transcriptome offers an insight into the molecular dialogue between host and pathogen. The *P. syringae* T3SS can suppress the entry of *P. fluorescens*, *P. putida*, and *A. tumefaciens* Agropine Ti plasmids into plants independent of the presence of the phytohormone auxin.

Keywords: Bacteria, pathogen, soil microbe, infection, molecular, proteomics

1. Introduction

Bacterial plant pathogens are a serious threat worldwide. They can infect all tissues of the host plant, suggesting that type III secretion translocation cannot explain their lifestyle and that novel translocation machineries may exist, as proposed for other host-adapted bacterial symbionts (Dean, 2011) [36]. These pathogens use a wide range of strategies and effector proteins to manipulate the physiology of the host and avoid recognition by the immune system (Jones & Dangl, 2006) [69]. Epidemiological modeling of bacterial plant pathogens is an underdeveloped field, mainly due to limited availability of relevant biological data (Garrett *et al*., 2006) [52]. The number of complete genome sequences for bacterial plant pathogens has risen significantly. These are useful for functional and comparative genomic analyses but are limited to circulating strains of a pathogen (Toth *et al*., 2006) [131]. Thus, data generated from experiments with strains possessing virulence genes no longer in natural populations will not generate biologically relevant insights for pathogen evolution, adaptation, resistance, host range, or the basis of epidemiological parameters (Madden & Wheelis, 2003) [86]. Here these challenges are addressed by examining pathogen diversity in the globally disseminated bacterial plant pathogen, the causative agent of bacterial speck on tomato. A molecular epidemiological method to monitor the distribution of novel effector protein genes across strains of a pathogen is described. Effector proteins secreted by the type III secretion system are phytopathogen virulence factors that can be recognized by cultivar-specific resistance gene products. The mosaicism of the small effector gene in its promoter region is used as a model to assess variation in this novel region among strains.

This is examined in a challenge experiment with a strain deficient in the synthesis of the endogenous effectors. Different prices of transcripts are produced by wild-type strains.

2. Understanding Bacterial Pathogenesis

Bacterial phytopathogens utilize an extensive set of virulence factors to modulate their plant hosts to promote successful pathogenesis. Ultimately, these bacteria can circumvent plant immune responses and overcome plant developmental programmes to achieve tissue maceration to both degrade tissue components and provide access to nutrients (Xin & He, 2013) [140]. A potent virulence strategy used predominantly by Gram-negative bacteria is to inject these virulence proteins into the plant cell via the type III secretion system (Huei-Yi Lee *et al*., 2013) [65]. Analysis of host targets and molecular mechanisms of type III effector proteins (effectors) has significantly expanded the understanding of many fundamental eukaryotic cell processes, including immune signalling, vesicle trafficking, cytoskeleton stability, and transcriptional regulation (Block *et al*., 2008) [15]. Spheroplast-based screens have been previously applied in yeast and plants to characterize Legionella and Pseudomonas effector function, and here this methodology was more broadly applied to identify molecular cellular processes targeted by diverse type III effectors in a range of eukaryotic species (Popa *et al*., 2016) [114]. Spheroplasts were used as a biotechnological approach to screen the model plant Arabidopsis thaliana. The effector HopM1 was found to localize to the host plasma membrane, stabilise microtubules and reduce actin bundling, as well as showing suppression of MAPK activation (Nomura *et al*., 2006) [104]. Interestingly, a second structure-based screen conducted on Arabidopsis spheroplasts targeting components of the microtubule network identified an additional nine type III effectors that perturb this host cellular structure (Lee *et al*., 2012) [75]. Collectively, these results indicate that a sizeable fraction of type III effectors target both innate plant cellular targets and conserved eukaryotic cellular processes characterised here in plant and non-plant spheroplasts. Type III effector virulence proteins are groups of protein families produced by many of the most successful Gram-negative bacterial pathogens of animals and plants. Effector proteins subvert host cell processes in order to promote bacterial proliferation, survival and/or entry (Feng & Zhou, 2012) [44]. An important strategy employed by successful bacterial pathogens is to attack key intracellular host processes to allow maximal proliferation (Büttner & Bonas, 2010) [24]. Studies on type III effectors have uncovered novel components of plant cellular processes by first revealing these components as targets of effector proteins (Deslandes & Rivas, 2012) [38].

2.1 Mechanisms of Infection

The infection process of bacterial plant pathogens by plant pathogenic *P. syringae* and *R. solanacearum* strains is essential for the construction of rational control strategies. Pathogens infect host plants by recognizing the leaf surface, multiplying on it, mainly through stomatal invasion, and eventually migrating to tissues where pathogenicity is expressed (Melotto *et al*., 2008) [93]. Recognition occurs through various mechanisms including the detection of nutrients derived from plants, such as amino acids (Sakata & Ishiga, 2023) [122]. Pathogen interactions, prokaryotic surface molecules or small molecular compounds secreted by the pathogen, or the hydrophobicity and roughness of the leaf surface as well as the presence of cuticle covering it may induce bacterial attachment or motivate secretion systems (Ramey *et al*., 2004) [116]. In the context of ongoing work, at least three independent mechanisms are likely to be involved in pathogen recognition on living leaves. Following recognition of the plant surface, the pathogen adheres to the leaf surface, where it synthesizes Bta to trigger larger epidermal cell chlorosis and stomata closure. The ability of the strain to induce stomatal closure is important for the elevated growth and pustulation of the strain on the leaf surface *in vitro* and is correlated with epiphytic growth *in vivo* (Underwood *et al*., 2007) [133]. As a result, the number of conidia produced is rapidly increased. In addition, the strains with rout3 mutants reduced the ability to cause symptoms after infiltrating leaves, despite the fact that they are not generally affected in terms of pathogenicity (Huei-Yi Lee *et al*., 2013) [65].

2.2 Host-Pathogen Interactions

The interaction of host-pathogen has an important role in determining the outcome of an infection process. Plants have developed a suite of defenses that are activated upon the perception of PAMPs by their corresponding host proteins (Zipfel, 2009) [144]. However, plant pathogenic bacteria have evolved the ability to suppress these defense responses by the secretion of effector proteins that interact with host targets (Boller & He, 2009) [18]. To overcome these effectors, plants have R proteins that recognize one or several effector proteins to activate an immune response. All of these processes have been studied by different approaches, but generally from a static point of view, disregarding the dynamic of the process. With the development of computational modeling, it has become possible to build and analyze a system of differential equations that describe the dynamics of the interaction at a molecular level (Saile *et al*., 2020) [121]. For the above reasons, a mathematical model of interactions between host plants and their respective pathogenic bacteria was built to describe not only the immune response but also the influence of bacteria effectors.

3. Molecular Mechanisms in Plant Pathogenesis

The Evolution of Plant Pathogenesis: A Molecular Approach Emphasis on progress made in the understanding of the molecular basis of plant pathogenesis by Pseudomonas species, and to examine this in the wider context of what is known of the evolution of pathogenesis by this and other bacterial genera. Natural populations of Pseudomonas fluorescens and closely related species commonly occur as a mixture of genotypes in the same environment (Silby *et al*., 2009) [121]. In some environments, isolates from these populations exhibit marked differences in their abilities to colonize and exploit different substrates. One such type of isolates, denoted CF, is typically recovered from the rhizoplane of a range of crop plants. Despite a lack of obvious symptoms on the plant, CF strains appear to be 'biotically' specialized to exploit ecological niches associated with the host plant (Mendes *et al*., 2013) [95]. This contrasts with other isolates from the same environment (denoted W), which colonize the same substrates as CF but exhibit different, minimal bacterial densities on the phylloplane and in the rhizosphere. CF strains generally also show reduced 'epiphytic fitness' on inanimate surfaces, while colonization of some living substrates (such as the nematode) results in poor survival of the animal. The differences in substrate utilization, growth and colonization exhibited by CF and W types appears to result from restriction endonuclease profiles detected among natural isolates (Frapolli *et al*., 2012) [49].

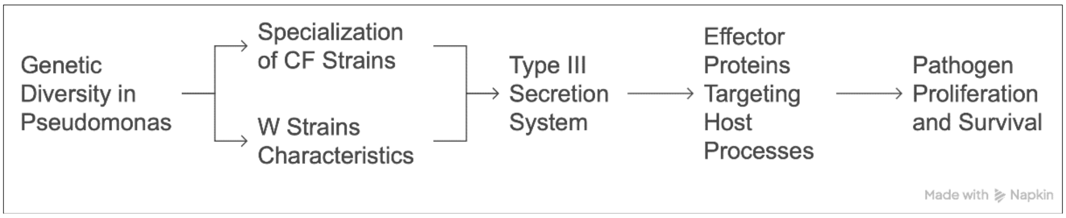

Fig 1: Evolution of plant pathogenesis by pseudomonas

Bacterial phytopathogens utilize a myriad of virulence factors to modulate their plant hosts in order to promote successful pathogenesis (Xin *et al*., 2018) [141]. One potent strategy is to inject proteins into plant cells by the type III secretion system. Characterizing host targets and the molecular mechanisms of the so-called type III effector proteins has broadly illuminated eukaryotic cell biology (Lewis *et al*., 2011) [77]. These effector proteins, functionally analogous in bacteria to neuroactive compounds in higher organisms, can serve as exquisite molecular probes and have helped illuminate a diverse array of eukaryotic cellular processes such as immune signaling, vesicle trafficking, cytoskeleton stability and transcriptional regulation (Hann *et al*., 2010) [60]. Pathogens, during the course of a coevolutionary struggle with their hosts, might have evolved effector proteins that differentially target pivotal control points of such processes (Mukhtar *et al*., 2011) [100]. An important strategy employed by many successful live-cell bacterial pathogens is to specifically attack key intracellular host processes to allow the maximal proliferation of the pathogen (Cunnac *et al*., 2009) [32]. Most animal and plant pathogens and many symbionts require a functioning type III secretion system for full virulence (Galán *et al*., 2014) [51]. Bacteria have evolved to harness this translocation system to deliver diverse virulence proteins, or effector proteins, directly to the host cell cytoplasm (Dean, 2011) [36]. Other pathogenic species of enteric bacteria can inject effector proteins into plant cells as well as into mammalian and yeast cells (Anderson *et al*., 2015) [4]. Unraveling mechanisms by which these effector proteins promote bacterial virulence has furthered the understanding of eukaryotic cell biology, a cornerstone of modern molecular biology (Block & Alfano, 2011) [14].

3.1 Virulence Factors
Plant pathogenic bacteria utilize innocuous Agrobacterium rhizogenes and Agrobacterium tumefaciens as models, focusing on the effector and cyclic-di-GMP networks that help regulate the infection process (Nester, 2015) [103]. Modern agriculture faces the challenge of sustaining crop productivity to meet growing human populations despite diminishing arable land and water for irrigation (Tilman *et al*., 2011) [130]. Additionally, the land that is arable is becoming increasingly damaged through pollution, salinization, and erosion (Foley *et al*., 2011) [47]. The rapid evolution of pathogen populations, combined with agricultural practices that foster pathogen spread, exacerbates concerns (McDonald & Stukenbrock, 2016) [91].
Crop plants suffer from several different types of microbial phytopathogens, including bacteria, fungi, viruses, and oomycetes (Strange & Scott, 2005) [128]. Among these, bacteria are usually not as predominant in the field as fungal or oomycete pathogens, but, unlike the latter organisms, can spread proportionally more rapidly through irrigation water or in infective insect vectors (Hirano & Upper, 2000) [64]. Consequently, plants with bacterial infections sometimes need to be entirely eradicated to prevent further spread. More generally as a result of collective agricultural practices, plant pathogenic bacteria are evolving resistance against antibiotics at an increasing rate (Sundin & Bender, 1996) [129]. At the same time, the application of antibiotics is under public scrutiny due to concerns about the emergence of resistant human pathogens via genetic exchange in the environment (Mecsas & Strauss, 1996) [92].
Little is currently known about the roles of cyclic-di-GMP signaling in the context of plant-microbe interactions (Römling *et al*., 2013) [120]. In plant plants, salt hyper-response 1 (SHR1) protein involved in stomatal innate immunity against bacteria act in the concert with both cyclic-di-GMP signaling and effector's secretory type III system (Bordiec *et al*., 2011) [20]. Bacterial plant pathogens, through the action of so-called effector proteins, can regulate cyclic-di-GMP levels in the host plant or host bacteria to facilitate the bacteria-host interaction (Hengge, 2009) [62]. These results putatively establish regulative routes in bacterial plant pathogenesis where effector secretion systems, host plant-bacteria signal networks, and downstream nutritional nodule machinery converge and synchronize (Cross, 2008) [30].

3.2 Quorum Sensing
With the increasing number of quorum-sensing-regulated processes that have been identified in bacterial pathogens, it is becoming evident that such systems may provide much greater survival and fitness advantages for pathogenic bacteria than previously recognized (Pawar & Lahiri, 2018) [110]. It has been suggested that quorum sensing might afford pathogens the ability to upregulate the expression of virulence factors only when a critical density is reached, thereby reducing the probability of host detection before expression of these factors is sufficient for successful infection (Antunes *et al*., 2010) [5]. There is also evidence in some systems that quorum-sensing-controlled virulence factors can serve as social signals that confer fitness benefits to other species (Abisado *et al*., 2018) [1]. Therefore the ways in which quorum sensing influences pathogenesis are complex, with an impact that likely extends beyond a single pathogen. To explore the molecular processes underpinning the influence of quorum sensing on pathogenicity and wider influences on host/pathogen/microbiome interactions:

- Consideration of the complex interkingdom signalling processes that are likely to contribute to quorum sensing control of pathogenicity (Hughes & Sperandio, 2008) [66].
- Discussion of the intricate feedbacks between quorum sensing, motility and biofilm formation that will affect bacterial colonisation and establishment of the infection focus (Parsek & Greenberg, 2005) [109].
- Evaluation of the ability of small signalling molecules to be co-opted by higher organisms to disrupt quorum sensing functions in competing bacteria and potentially enhance colonization resistance of the epithelial surfaces (Hentzer & Givskov, 2003) [63].

3.3 Biofilm Formation

Although the medical community increasingly appreciates the impact of biofilm infections on human health and disease, the prevalence of biofilm formation among plant and soil-associated bacteria, particularly in the context of pathogenesis, is vague. The aim of this review is to draw parallels and contrasts between the current understanding of biofilm formation in both pathogenic and beneficial, plant-associated bacteria. With decades of research in anti-biofilm strategies as well as innovative research and advancements in biofilm application as beneficial tools in biotechnology and engineering, the bacterial plant disease community can rely on a wealth of knowledge as it continues to develop anti-virulence strategies (Flemming *et al.*, 2016) [46]. Adding to this multilayered review of biofilm formation in bacterial plant pathogens also serve to provide unique opportunities that can be explored by the research community to further our understanding of biofilms in future microbial challenges.

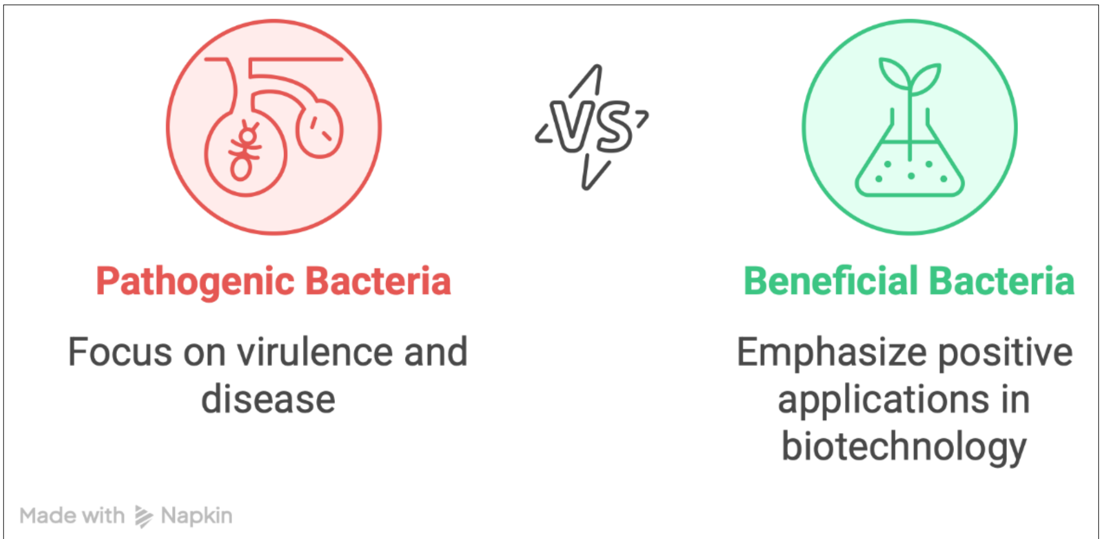

Fig 2: How to approach biofilm formation in plant-associated bacteria

One forming vision among researchers is that biofilm architecture and development can vastly differ by species, potentially due to a need of accessory factors, virulence determinants, and ecological niches (O'Toole *et al.*, 2000) [105]. This hypothesis can perhaps best be supported by looking at parallels between experiments investigating the molecular dynamics of biofilm formation. However, research into bacterial biofilm formation on plant dioecious hosts is limited and ambiguous. Only a few examples exist that investigate biofilm or biofilm-like consought by pathogenic bacteria on living plant tissues (Bogino *et al.*, 2013) [117]. To date, microscopic examination of biofilm formation by phytobacteria and the molecular "micro" environment of those biofilms is lacking. A current, noteworthy development includes the exploration of the regulation and maturation of biofilms formed by Agrobacterium tumefaciens (Gu, 2014) [58]. As suggested by radial structure, pcf mutant biofilms fell off, were clear and viscous compared to the WT. Collectivement, Agrobacterium tumefaciens is pathogenic to a wide variety of plants, relying on the expression of virulence elements encoded within transferred Ti plasmid for tumor formation (Escobar & Dandekar, 2003) [42]. One step in the establishment of infection is the generation of an organized biofilm, a function shared by disease caused by other plant-related bacteria. Little is known regarding the regulation of this distinctive biofilm, or the interaction that occurs *in vivo*.

4. Plant Immune Responses

Plants are continually challenged by a variety of infectious agents that profoundly impact crop yields. Among these biotic challenges, bacterial pathogens are devastating to agriculture worldwide (Savary *et al.*, 2019) [124]. The strategies by which bacteria cause diseases in plants are diverse, but typically they involve the multiplication and spread of the pathogen in the host tissue (Pfeilmeier *et al.*, 2016) [112]. During infection, plants perceive bacterial pathogens via the detection of microbe-associated molecular patterns (MAMPs) by pattern recognition receptors (PRRs) (Ali *et al.*, 2024) [2]. The recognition of MAMPs triggers a cascade of signaling events known as pattern-triggered immunity (PTI) designed to reinforce basal host defenses (Boutrot & Zipfel, 2017) [21]. Successful colonization of a plant host often involves the ability of the pathogen to deliver effector molecules into plant cells, which suppress the induction of PTI or elicit susceptibility in the host (Jones & Dangl, 2006) [69]. The suppression of PTI enables bacterial pathogen entry, proliferation, and tissue spread. In turn, plants have evolved the ability to recognize specific effectors, leading to the activation of effector-triggered immunity (ETI) and the associated hypersensitive response (HR) (Cui *et al.*, 2015) [31].

4.1 Detection of Pathogens

Introduction Although much is known and even more has been proposed regarding the molecular events carried out by microbes on plant cell signaling, the ordered mechanics and binding events that prompt the more advanced disease features are, by comparison, far less understood (Macho & Zipfel, 2014) [85]. To gauge future microbial threats to agricultural systems is to fully appreciate the superiority of those pathogens. The rational development of durably resistant crops necessitates such insights (Dangl *et al*., 2013) [33]. The lack of effective systemic controls, coupled with pressures to redress concerns about food security through commercially available breeding material, underscore the impetus of this discourse (Lobell & Gourdji, 2012) [84]. Notably, bacterial disease is far more difficult to defend against given the relative ease of mutation and generation time of bacteria as compared to their fungal and oomycete counterparts (McCann *et al*., 2017) [89]. Understanding the molecular dynamics at the interface of host tissue infection will likely inform the design of novel and more robust defensive strategies. While many specific systems required for attenuation have yet to be characterized, this review assembles what is known about, or proposed for, the bacterial macromolecules and their presumed host counterparts with which the infecting microbe operates.

4.2 Signal Transduction Pathways

A series of molecular interactions mediate the pathogenesis of *Pseudomonas syringae* pv. tomato in the host plant Arabidopsis thaliana. The model presented focuses on a hierarchical view of virulent gene expression during pathogenesis, with salicylic acid signal regulation serving as a nodal point. The stealth phase of infection involves extracellular perception of the host plant by the bacteria. Recognition of specific host-derived proteins, along with water and cytoplasmic molecules, results in derepression of avrPto and corresponding up-regulation of HrpL-regulon expression (Lin & Martin, 2005) [81]. The effector protein AvrPto suppresses the HR and results in replication-competent plant-associated bacteria.

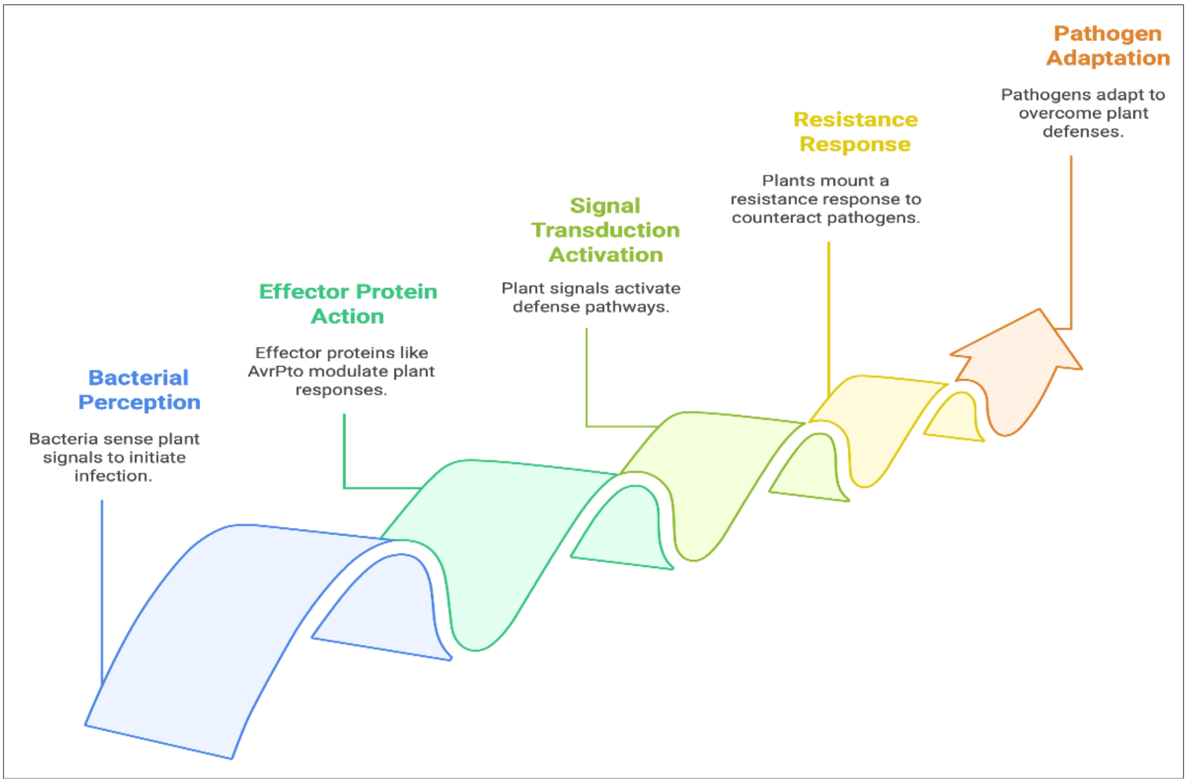

Fig 3: Pathogenesis and resistance in plant-pathogen interaction

Host-derived phenolics are perceived by the bacteria and, in an intact host plant, result in a two-part signal transduction element. This leads to increased expression of Prf-AvrPto and expanded hrp gene expression. The former results in Pto-dependent hrp gene expression while the latter results in Pto-independent expression. The signal transduction pathway mutes these processes such that mutant plants refractory to signaling are unable to down-regulate the pathogen response (Li *et al*., 2002) [80]. Bacterial resistance to the signal is negatively regulated by the AvrPto protein. Early in the interaction, signal is possibly induced by bacterial infection (Block *et al*., 2010) [16]. In mature leaves AvrPto (when overexpressed) increases accumulation such that the bacteria must rapidly down-regulate Prf-AvrPto activity to avoid a monk-like disease resistance response (Lin *et al*., 2006) [82].

4.3 Defense Mechanisms

Bacterial plant pathogens are a challenge to agriculture and have become the focus of intense interdisciplinary research by physicists, biologists, and mathematicians (Garrett *et al*., 2011) [53]. The molecular basis of bacterial pathogenesis has been characterized, highlighting the roles of adhesins and effector proteins in attacking plant host cells (Melotto *et al*., 2017) [94]. However, there is less understanding of the biophysical mechanics of this interaction, specifically how bacteria physically distort the plant cell and manipulate the host actin machinery to invade the cell, kill it, and traverse to neighboring cells (Day *et al*., 2011) [34]. Here, we review progress in understanding the biophysical basis of bacterial plant pathogenesis and the open questions that remain, whilst emphasizing the challenges in this field and the approaches required to solve them. Volumetric growth in the infected cell and the ability of bacteria to colonize plant tissue leads to overflowing of the bacterial population (Cunnac *et al*., 2011) [32]. Bacterial colonization is thus hindered by the physical restriction of the cell wall to bacterial invasion, expansion, and growth. Nonetheless, successful pathogens interfere with and distort the host cell wall structure, mainly by the secretion of an arsenal of plant cell wall-degrading enzymes directed at either dissolving cellulose and other polysaccharides directly or processing a cascade of cellular enzymes involved in restructuring the cell membrane (Kubicek *et al*., 2014) [73]. In turn, plants activate multiple defense mechanisms that reinforce and chemically enhance the cell wall, thus further hampering degradation (Bellincampi *et al*., 2014) [12].

5. Genetic and Genomic Insights

Recent advances in the genetic and genomic bases of sugar beet-pathogen interactions have been made in recent years. Several candidate susceptibility genes are involved in sugar beet-CIRAD124 interactions. Only 1 candidate resistance gene has been described in sugar beet so far; Bvbn has been mapped on chromosome 6S and is effective against powdery mildew caused by E. betae (Grimmer *et al*., 2007) [57]. The molecular genetic pathway of sugar beet-BpvC105 interaction has been analyzed (Funk *et al*., 2018) [50]. Furthermore, the structures and transcript patterns of the effector genes of ABC of A. candida in infected Arabidopsis rosette leaves at 7 d.p.i. have been clarified (Imam *et al*., 2016) [67]. NGS analyses uncovered nearly 10% of ABC effector genes. As more plant and pathogen genomes are sequenced, a bioinformatical approach will be helpful for identification of more effector genes in the phylum Oomycota (Baxter *et al*., 2010) [11]. The defense response triggered by these avirulence genes in sugar beet has been demonstrated (De Coninck *et al*., 2015) [35]. For SNP calling in metabolism reconstruction the tolerance of incomplete gene correspondences between query and reference database has been increased (Overmars *et al*., 2015) [106]. Close species with better conserved gene correspondences were tested for reconstruction of H. vulgare ssp. vulgare metabolism (Mascher *et al*., 2017) [88]. The translation required steps have been parallelized to reduce the overall computation time and memory usage (Langmead & Salzberg, 2012) [74]. An optimized BLAST search has been engineered to accommodate the large number of search sequences arising from metabolites, reactions, and genomic annotations (Camacho *et al*., 2009) [26].

5.1 Genome Sequencing

Sequencing of bacterial pathogenesis will be taking the theme of molecular genetics, more specifically focusing on the molecular process of plant pathogenic bacteria to elicit disease on host plants. The foundation of genetics is the primary tool to understand gene functions and their molecular rules fundamentally (Salmond & Hogenhout, 2019) [123]. The subject of bacterial genome sequencing, annotation, and evolution, fittingly bacterial plant pathogenicity, are among the latest topics in the very frontiers of molecular genetics. Microbial pathogens have only been severely screened by phenotypic analysis in their interactions with eukaryotic hosts.

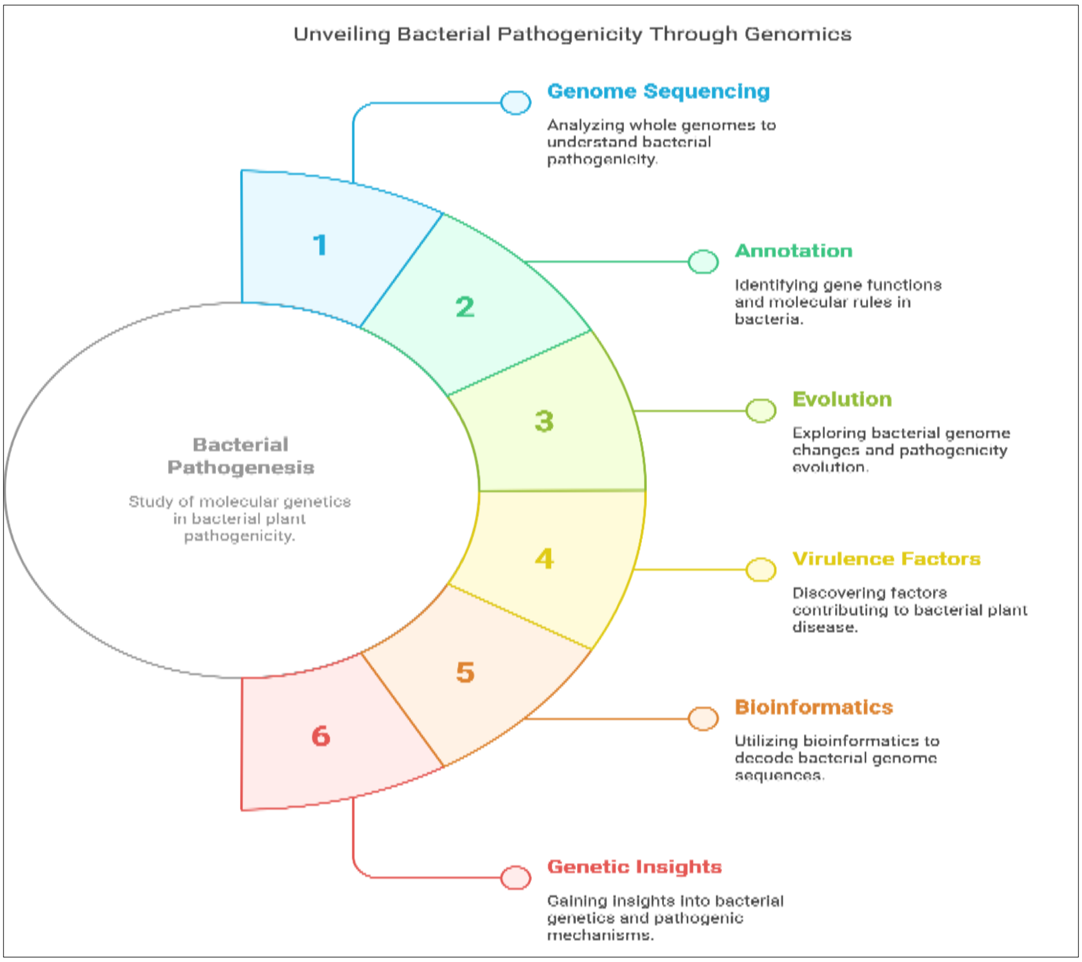

Fig 4: unveiling bacterial pathogenesis through genomics

In this era of genomics, gathering sequence information on the whole genomes of pathogenic bacteria now permits a high-throughput search over sets of virulence factors (Toth *et al*., 2006) [131]. Meanwhile, bioinformatics works have elucidated the fine "cryptic" code of genetic information carried over into genome sequences, e.g. gene prediction, −10, −35 boxes, cis-antisense RNA and argU (Donkor, 2013) [39]. Promoters/transcription factors, signal peptides, recombination hotspot, porin families and phagemid is also a small range of genetic gussies never thought before in the pre-genomic age. However, the recent genetically summarized progresses on bacteria-perpetuating phytopathogenicity are almost still within Darwin's notion (McDonald & Linde, 2002) [90].

5.2 Gene Expression Analysis

Bacteria are well known for their ability to adapt and grow in a wide range of environments, both in natural ecosystems and in the context of human activities (Pallen & Wren, 2007) [107]. In the biosphere, they are primary decomposers of plants, capable of recapturing a vast array of carbon-and nitrogen-containing compounds by means of hydrolysis, digestion and catabolism; however, a number of plant pathogenic bacteria across the phylum Proteobacteria cause disease, substantially impacting food production globally (Mansfield *et al*., 2012) [87]. The molecular mechanisms of plant colonization and development of these bacteria are complex, involving the coordinated expression of many genes and metabolites in response to changing environmental conditions (Rico *et al*., 2011) [119]. This Review discusses the genetic changes and regulation that occur in bacteria during plant infection, covering signal perception, secretion systems, gene regulation, plant cell wall degrading enzymes and virulence factor delivery. It illustrates a number of bacterial phytopathogenesis concepts through examples of differential gene expression, protein and secondary metabolite production by a heterogenous group of distantly related bacteria (Fan *et al*., 2020). Bacterial soft and wilting diseases of plants are some of the best-known diseases, representative of diverse pathovars/species of phytopathogenic bacteria (Charkowski, 2015) [28].

5.3 Comparative Genomics

Population genomics revealed additive and replacing horizontal gene transfers, respectively, leading to a high level of foreign DNA in the genome that largely caused replacement of indigenous regulatory genes and virulence factors and subsequently contributed to the elicitation of niche adaptation and systemic colonization processes (Ravenhall *et al*., 2015) [118]. The galU gene of Xanthomonas campestris pv. campestris is involved in bacterial cell attachment and cell motility, polysaccharide biosynthesis, virulence, and tolerance to various abiotic stress, including heavy metals and bactericides (Li *et al*., 2014) [78]. Comparative genomics of the pangenome, core genome, regulative core genome, and panregulon of D. solani combining a newly sequenced genome of a highly virulent strain IFB0099 and the previously sequenced weakly virulent IPO2254 were carried out (Golanowska *et al*., 2018) [56]. The major objective was to gain an extended insight into the evolution of this recently emerged species.

6. Proteomics and Metabolomics

The term 'biotic interactions' embodies mutualistic or antagonistic reciprocal effects between organisms. Plant-associated microbiota includes some pathogens that can provoke a wide range of diseases (Berendsen *et al*., 2012) [13]. Despite great advances in crop protection strategies during the Green Revolution, plant disease control is still a challenge (Pingali, 2012) [113]. Indeed, in the last few decades, prevalence of plant diseases has increased due to changes in ecological conditions (Anderson *et al*., 2004) [4]. Gaining a deeper knowledge of the molecular dynamics of the host-pathogen interaction is essential to help understanding microorganism population and how to limit the broad spread of virulence factors (Balotf *et al*., 2022) [8].

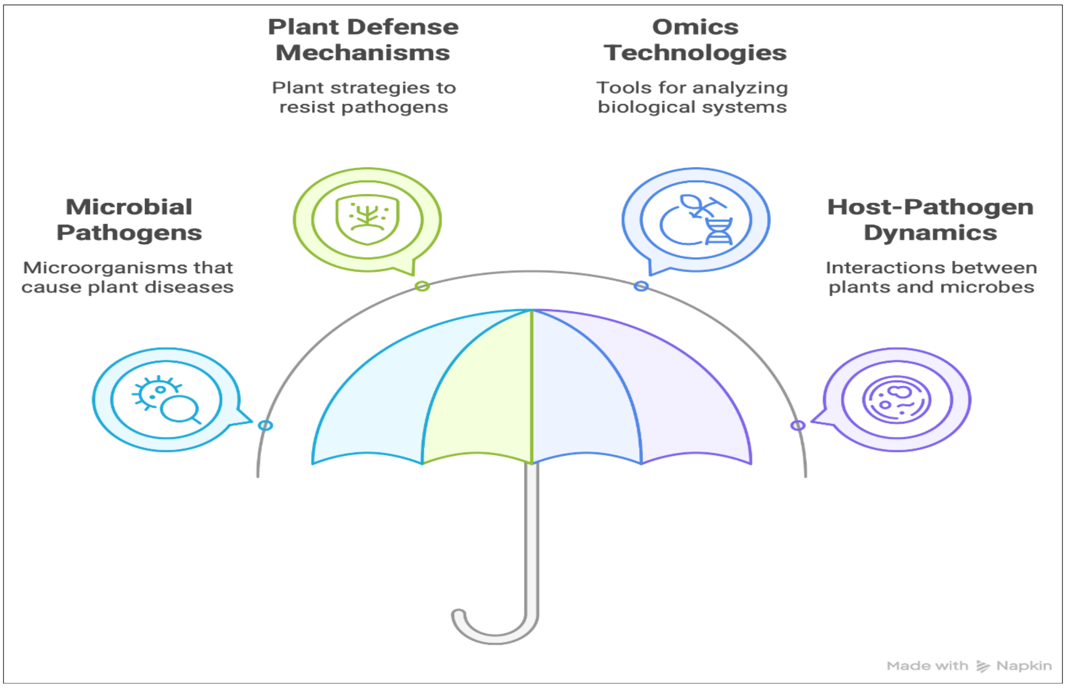

Fig 5: Unveiling plant-microbe interaction

Microorganisms occasionally encounter different stresses, including the competition for nutrients, and need to navigate through host colonization (Vorholt, 2012) [137]. For an extensive comprehension of diseases' occurrence, a significant attention has been recently devoted to the 'omics' sciences. Among these, Special Issue papers cover various aspects of the wide multi-omics sciences explosion as potent instruments to disentangle the mechanisms of plant defense and microorganism pathogenesis (Crandall *et al*., 2020) [29]. 'Omics' technologies are powerful means to deepen knowledge of the complexity of the biological systems' dynamics. More than that, they offer a unique opportunity to explore the molecular components and mechanisms that underlie observed phenotypes and responses to external stimuli through the unbiased analysis of gene expression, proteins and metabolites quantification (Jayaraman *et al*., 2012) [68].

6.1 Protein Profiling

Plant disease caused by bacterial infection is a major threat to numerous crops (Strange & Scott, 2005) [128]. To attack, evade, and colonize the relatively advanced immune system of the host plants, pathogens employ a variety of bacterial virulence factors, such as type III secretion system, secreted and extracellular proteins, and effector proteins (Büttner & He, 2009) [25]. Although hundreds of different effectors have been discovered and even functionally verified in the lab, the plant immune system is even more redundant (Feng & Zhou, 2012) [44]. A number of other, as yet unknown, effector proteins might be caught by plants now or in the coming years. Consequently, unique tools and innovations will be critical to elucidate the strategies used by the aforementioned complex group of potential pathogens to regulate disease within crops (Misra *et al*., 2016) [97].

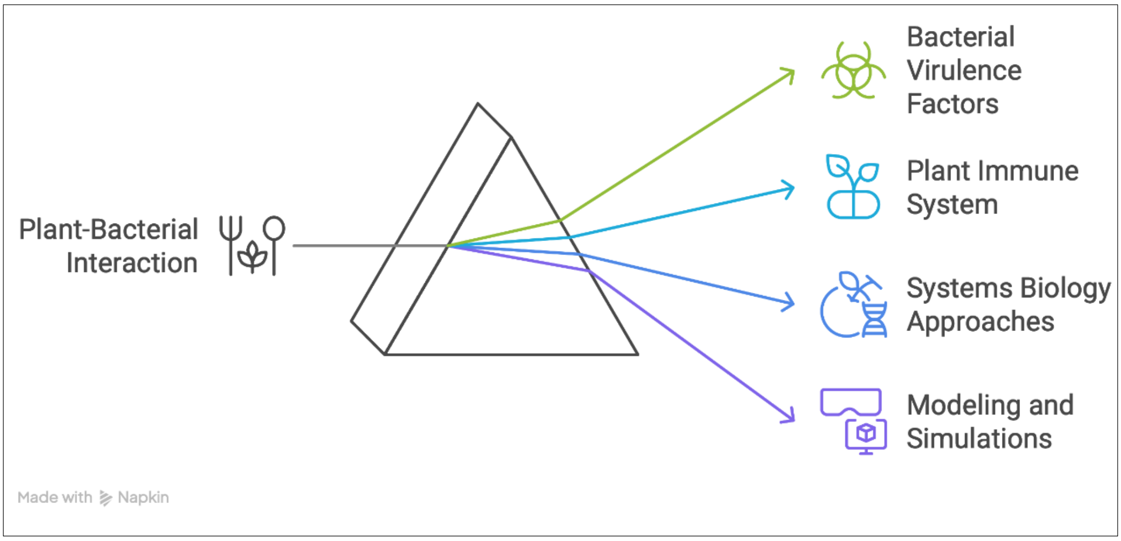

Fig 6: Unveiling plant-bacterial interaction

In this sense, systems biology approaches have the potential to provide a broader view. Integrated studies employing many biological and computational methodologies on complex properties provide the context for understanding the robust behaviors and properties of the observed biology (Kitano, 2002) [70]. In their case, translating the infection from bacterium's or plant's view to mechanistic study of a network of the plant and bacteria component at the system level may facilitate solutions to handle that artificially (Peyraud *et al*., 2017) [111]. Unique and unexpected results of system level modeling may never be anticipated or solely deduced via experimentation, and hence modeling and simulations provide a critical method for addressing the highly complex problem of plant disease (Pritchard & Birch, 2011) [115].

6.2 Metabolic Pathway Analysis

Given the increasing need for novel antimicrobial compounds to face the growing problem of antibiotic resistance, it is important to pinpoint the molecular mechanisms that drive the development of successful bacterial pathogenesis (Ventola, 2015) [136]. The capacity of the plant pathogen, Pseudomonas syringae, to actively infect host plants, such as Arabidopsis thaliana, provides a good opportunity for an investigation of the molecular basis of bacterial pathogenesis (Xin & He, 2013) [140]. Plant pathogenic bacteria and non-pathogenic control bacteria inoculated on Arabidopsis plants were analyzed by a combined mass spectrometry-based metabolomic and proteomic approach (Pang *et al*., 2020) [108]. This investigation revealed over-accumulation of infection-related bacterial proteins in inoculated leaves as well as general metabolic shifts caused by *P. syringae*. These results show the power of an integrated 'omics' approach to dissect translated molecular events in plant-bacterium interactions (Mishev *et al*., 2021) [96]. By taking an integrated approach at the proteomic and metabolomic levels, the molecular mechanisms involved in bacterial pathogenesis and defense mechanisms of host plants could be revealed (Allwood *et al*., 2008) [3]. Furthermore, detailed analysis of the multi-faceted transcriptional, post-transcriptional, post-translational and metabolic levels in both host and pathogen would not be readily achievable (Feussner & Polle, 2015) [45].

7. Environmental Influences on Pathogenesis

The study of the plant-pathogenic bacterium Pseudomonas syringae, the most important model pathogen for the study of bacterial plant disease, has allowed significant progress in the understanding of the molecular basis of disease in the plant host (Xin *et al*., 2018) [141]. Most of the characterized molecular determinants of pathogenicity contribute to the survival in the host, suppression of host immunity and to generation of conducive conditions to bacterial growth (Melotto *et al*., 2008) [93]. Yet, very little is known about how these pathogens survive outside of their host plants and how they escape the immune systems (immune-genes) that must be present in or on non-host plants (Morris *et al*., 2019) [99]. Understanding the molecular bases of the ecology of plant pathogen bacteria in the environment should not only contribute to elucidate the hands of nature outside of host plants but would also help to understand important aspects of bacterial evolution and potentially to develop new strategies for the prevention or control of bacterial plant disease (Hirano & Upper, 2000) [64]. This document outlines a proposed experimental system for the study, and the molecular bases, of the survival of the bacterium Pseudomonas syringae in alternative hosts and non-host plants or in the presence of potential predators, using a combination of genetic analysis and recently developed molecular techniques (Monteil *et al*., 2016) [98]. This document, framed into the long-term goal of understanding the ecology of phytopathogenic bacteria under natural exposure, outlines the rationale and methodology for a project that will study how Pseudomonas syringae survives in the phyllosphere when exposed either to alternative host plants, to non-host plants or to the epiphytic bacterial community, including predators. Host ranges of *P. syringae* are typically restricted to a limited set of closely related plants (Dorati *et al*., 2018) [40], and a rich literature documents the set of compatible and incompatible interactions (as well as the genetics thereof) between this bacterium and its set of host plants (Baltrus *et al*., 2017) [9].

7.1 Soil Microbiome Interactions

In future scenarios due to increasing competition for scarce resources, there will be a need to optimize resource use (Foley *et al*., 2011) [47]. Towards achieving this, it will be very important to better understand and model the soil microbiome, which is a crucial aspect to many situations related to plant health and successful growth (Berendsen *et al*., 2012) [13]. Indeed, the microbial community that colonizes the soil surrounding a given plant has been found crucial towards successful pathogenesis (Imam *et al*., 2016) [67]. Typically, it has been observed that certain pathogenic bacteria can only become pathogenic if assisted, or because of competitive inhibition by some other closely related non-pathogenic bacteria (Mendes *et al*., 2013) [95]. The phenomena were shown by using different pathovars, with the competitive inhibition being prevalent for strains of different pathogenicity (Haas & Défago, 2005) [59]. Nonetheless certain soil bacteria cannot productively colonize plant roots, as the plant in question exerts a strong and effective defense against these soil microbes (Bais *et al*., 2006) [6]. This soil defense has previously been overlooked in bacterial infectious rulesets. This work considers the dynamics of the bacterial plant pathogenesis and modeling approaches to tackle future microbial challenges, when the soil microbiome can exert a strong effect on the pathogenicity-success of the bacterium (Bulgarelli *et al*., 2013) [23].

7.2 Climate Change Effects

A sound plant is one that is compatible with its ecosystem, now and in the future (Trivedi *et al*., 2020) [132]. A reasoning that forecasts the effects of the slow processes of infection, whatever the infecting organism, and explains the generation of tissues and the production of cues for infection (Velásquez *et al*., 2018) [135]. These cues are likely to extend well beyond the plant surface to the plant as a whole and in blended interaction with the environmental conditions and the incipient microflora (Chakraborty & Newton, 2011) [27]. A use of the molecular genetics of both host and pathogen to tease apart the components of aggressiveness and resistance, and to detail the biotic and abiotic determinants of those genetics (Dendy *et al*., 2006) [37]. A fusion of genomics and environmental cell biology that will test this reasoning through modeling infection and disease processes at the cellular, organ and whole plant levels in order to maximize disease resistance with minimal deleterious secondary effects (Garrett *et al*., 2006) [53].

8. Emerging Technologies in Pathogen Research

During Wu's era of 2003−2016, the 1st generation green revolution, the scientific community runs a full barrel to tackle the then pathetic situation in pathogenic disaster (Pingali, 2012) [113]. In retrospect, a significant progression has been made in the molecular dynamics of bacterial plant pathogenesis (Mansfield *et al*., 2012) [87]. Diverse technologies like rapid TILLING, next-generation sequencing, rice differential protein database, lignin self-synthesis responsive melanin synthesis mimics, qRT-PCR microarray combined with RNA-seq, integrated systems biology analysis have been employed in varied aspects of disease resistance and virulence studies (Wang *et al*., 2016). Such as the coevolution between Xanthomonas oryzae pv. oryzae and rice pathogenicity-related gene OsTF2L1, a bacterial virulence-related E3 ubiquitin ligase, gene expression profile differences between a compatible and an incompatible interaction revealed by qRT-PCR microarray combined with RNA-seq, bacterial virulence-related proteins of Xanthomonas oryzae pv. oryzae, the defense-response between rice and X. oryzae pv. oryzae, virulent RTD of X. oryzae pv. oryzae, etc. (Li *et al*., 2017) [79]. But scientific experts are not to panic, the crop microbe faction remains one step ahead (Dangl *et al*., 2013) [33]. Some emerging technologies as well prospects, hopefully can help to navigate through the coming microbial challenge.

8.1 CRISPR and Gene Editing

Bacterial plant pathogens are a major and constant threat to crop production and food security on a global scale (Savary *et al*., 2019) [124]. They develop sophisticated strategies to invade hosts, suppress host defense, and thrive within host environments (Xin *et al*., 2018) [141]. Among the multitude of strategies utilized by these organisms, Type III protein secretion systems play a fundamental role in pathogenesis. Type III protein secretion systems are widely utilized by gram negative pathogens to deliver effector proteins into the plant cells (Galán *et al*., 2014) [51]. Subsequent interactions between these effectors and plant host proteins are crucial in determining overall pathogenic success (Feng & Zhou, 2012) [44]. Bacterial plant pathogens often deliver dozens of effector proteins into plants. It is a complex challenge to understand the molecular basis of these multiple interactions, which involve a plethora of different pathogen and host proteins (Mukhtar *et al*., 2011) [100]. Researchers have shown that many effector proteins interact with host proteins in a detrimental way that favors pathogen success (Deslandes & Rivas, 2012) [38]. A deeper understanding of these interactions would open the way for the improvement of crop plants. Conversely, host plants can also recognize these effector proteins and activate a defense response (Jones & Dangl, 2006) [69]. In the last decade, researchers have identified a large number of plant resistance genes that confer recognition of specific pathogen effectors and initiate a strong immune reaction called effector-triggered immunity (Cui *et al*., 2015) [31]. The recognition of these effectors by the host plants is based on a physical interaction between the effector and a host recognition protein. Understanding the molecular details of these interactions is crucial for breeding crops that are resistant to the bacterial pathogens (Zhang *et al*., 2015) [143].

8.2 Nanotechnology Applications

Current advancement in the science field has proven innovations in the field of nanotechnology to be one of the most upcoming areas (Shang *et al*., 2019) [126]. Planters are always searching for new technologies to combat plant diseases to safeguard plants and enhance agricultural productivity (Elmer & White, 2018) [41]. The hazardous effects on plants are caused by bacterium, fungus, and virus (Strange & Scott, 2005) [128]. New technologies have been developed to combat the above problems such that food quality and quantity can be ensured (Servin *et al*., 2015) [125]. Plant pathogen, a bacterium, is a major threat to the cultivation of various agricultural and horticultural crops (Mansfield *et al*., 2012) [87]. The most valuable products of plants are vegetable, fruits, and flowers. Bacterial wilt disease of brinjal is caused by Ralstonia solanacearum (Genin & Denny, 2012) [54]. This disease leads to establishment and growth of bacteria in several parts of plant and the bacteria block the water conducting tissue cells (Yuliar *et al*., 2015) [142].

9. Management Strategies for Plant Pathogens

The ongoing global population increase, supplemented by climate change, demands a need for a sustainable improvement of food security (Godfray *et al*., 2010) [55]. Consequently, plant breeding practices are coupled with challenges, such as the gradual appearance of imminent populations of pests and pathogens (Savary *et al*., 2019) [124]. Over the past two decades, detailed analyses of single proteins and protein-ligand or protein-protein complexes in atomic detail have been utilized (Baron & Fraser, 2013) [10]. A great variety of recent applications of this investigational molecular biology tool are presented here, including protein and DNA engineering, along with the analysis of these nano-machines of cellular life-the ribosome and polymerase (Wilson & Doudna Cate, 2012) [139]. The favorable future prospects of these methodologies are discussed in the context of the currently established and anticipated computational approaches (Peyraud *et al*., 2017) [111].

9.1 Chemical Control Measures

Phytopathogenic bacteria are sometimes so aggressive as to cause the death of the infected plant (Strange & Scott, 2005) [128]. Pathogen-plant interactions have been reported as useful for understanding biological issues in disease development, as well for envisaging novel biotechnological strategies for enhancing plant resistance (Jones & Dangl, 2006) [69]. Investigations on the molecular bases of pathogen lifestyle can provide important and practical information about how virulence is exerted, leading, for example, to the design of plant selectable virulence-deficient strains by genetic manipulation of climatically important pathogeniler combinations of crop species and bacterial strains (Bonaterra *et al*., 2022) [19]. Approval of the large-scale release of genetically modified organisms inhibits the infection processes initiated by phytopathogens (Klümper & Qaim, 2014) [71]. The importance in the molecular basis of virulence has increased.

9.2 Biological Control Agents

Biological control of plant diseases, i.e., the use of beneficial micro-organisms to protect plants from pathogen infections, is considered an environmentally friendly alternative to the application of chemical pesticides (Haas & Défago, 2005) [59]. A large number of bacteria present in plant tissues and on the plant surface are able to compete with and antagonize pathogens (Berendsen *et al*., 2012) [13]. These epiphytic bacteria can be potentially exploited for plant disease protection by introducing selected strains into a crop to outcompete the pathogen on the plant surface (Vorholt, 2012) [137]. Successful colonization of the plant phyllosphere, which includes the leaf as well as the non-lignified parts of the stem and fruit, is limited to certain bacteria (Lindow & Brandl, 2003) [83]. Thus, epiphytic bacteria are described mainly as a subset of the phyllosphere and are mostly restricted to the leaf surface. While the phyllosphere represents the aerial part of plants, the rhizosphere and the phyllosphere concurrently form the holosphere, which represents the connected plant compartments (Bulgarelli *et al*., 2013) [23]. The phyllosphere provides a nutrient-rich environment that selects for a specific bacterial community in which each species occupies its own ecological niche (Müller & Ruppel, 2014). Plant leaves are characterized by the production of leachates, which are a major nutrient source for epiphytic bacteria in the phyllosphere (Leveau & Lindow, 2001) [76].

9.3 Integrated Pest Management

Integrated pest management (IPM) is today a widely accepted pest management strategy to select and use the most efficient control tactics and at the same time reduce over-dependence on chemical insecticides and their potentially negative environmental effects (Kogan, 1998) [72]. One of the main pillars of IPM is biological control (Bale *et al*., 2008) [7]. Over 70% of the IPM programs developed since 1970 in the United States and involving more than 900,000 hectares of crops, rely heavily on biological control (Francis *et al*., 2020) [48]. The program enlarges each year in the sense that it covers an increasing number of crops and pests, and improved natural control of the main target pest results from lower use of broad-spectrum insecticides (Naranjo *et al*., 2015) [102]. Biological control programs of pest insects, however, have historically been and are still today commonly based on natural enemies such as predatory insects, parasitoids and microbial pathogens (van Lenteren *et al*., 2018) [134]. A large fraction of available research effort focuses on the behavior or the adaptation of the biocontrol agent to better exploit its efficacy in the fields (Heimpel & Mills, 2017) [61]. With respect to these targets of research new high-throughput screening techniques turn out to be quite efficient-molecular methods for the identification of plant-associated microbial communities form a powerful tool (Rastogi *et al*., 2013) [17].

High levels of endemism are frequent in microorganisms isolated from plants (Bulgarelli *et al*., 2012) [22].

13 Conclusion

The decades following T-DNA and Agrobacterium tumefaciens research have witnessed a broad advancement in understanding the molecular dynamics of bacterial plant pathogenesis. Henceforward, the research gives insights into emerging works studying widely diverse microbes and nematodes intimately interacting with plants, fungi both mutualistic and antagonistic, and insect vectors, which are reviewed to illustrate the supplementarity, if not homology, of animal and plant pathogenesis approaches upshot. The developments and efforts to investigate both well-adapted bacteria such as hemibiotrophic acidovoricans and the economically damaging agrobacteria, rhizobia, xanthomonads, and pseudomonads are highlighted, focusing on how pathogenesis research might successfully consort this wide-ranging understanding of prokaryotic hosts, symbiont and pathogen alike, within evolving plant-related landscapes. Finally, the question is raised of how pathogenesis research might contribute to biocontainment answers in the increasing application of engineered microorganisms. This indicates is that over future decades, plants would be subject to unprecedented microbial challenge, including designed microbes. In return, engineered plants and engineered ecosystems may depend on this capacity for survive and interact in new ways with pathogens, mutualists and parasites (Imam *et al*., 2016) [67].

14. References

1. Abisado RG, Benomar S, Klaus JR, Dandekar AA, Chandler JR. Bacterial quorum sensing and microbial community interactions. mBio. 2018;9(3):e02331-17. https://doi.org/10.1128/mBio.02331-17
2. Ali S, Khan N, Tang Y. Pattern recognition receptors in plant immunity: An update. Plant Science. 2024;338:111912. https://doi.org/10.1016/j.plantsci.2023.111912
3. Allwood JW, Ellis DI, Goodacre R. Metabolomic technologies and their application to the study of plants and plant-host interactions. Physiologia Plantarum. 2008;132(2):117-135. https://doi.org/10.1111/j.1399-3054.2007.01001.x
4. Anderson PK, Cunningham AA, Patel NG, Morales FJ, Epstein PR, Daszak P. Emerging infectious diseases of plants: Pathogen pollution, climate change and agrotechnology drivers. Trends in Ecology and Evolution. 2004;19(10):535-544. https://doi.org/10.1016/j.tree.2004.07.021
5. Antunes LCM, Ferreira RBR, Buckner MMC, Finlay BB. Quorum sensing in bacterial virulence. Microbiology. 2010;156(8):2271-2282. https://doi.org/10.1099/mic.0.038794-0
6. Bais HP, Weir TL, Perry LG, Gilroy S, Vivanco JM. The role of root exudates in rhizosphere interactions with plants and other organisms. Annual Review of Plant Biology. 2006;57:233-266. https://doi.org/10.1146/annurev.arplant.57.032905.105159
7. Bale JS, van Lenteren JC, Bigler F. Biological control and sustainable food production. Philosophical Transactions of the Royal Society B: Biological Sciences. 2008;363(1492):761-776. https://doi.org/10.1098/rstb.2007.2182
8. Balotf S, Ortiz D, Wilson R. Multi-omics approaches to decipher plant-pathogen interactions. Frontiers in Plant Science. 2022;13:842703. https://doi.org/10.3389/fpls.2022.842703
9. Baltrus DA, McCann HC, Guttman DS. Evolution, genomics and epidemiology of *Pseudomonas syringae*: Challenges in bacterial molecular plant pathology. Molecular Plant Pathology. 2017;18(1):152-168. https://doi.org/10.1111/mpp.12506
10. Baron R, Fraser JS. Protein structure and dynamics in molecular biology. Annual Review of Biophysics. 2013;42:267-288. https://doi.org/10.1146/annurev-biophys-083012-130351
11. Baxter L, Tripathy S, Ishaque N, Boot N, Cabral A, Kemen E, *et al*. Signatures of adaptation to obligate biotrophy in the *Hyaloperonospora arabidopsidis* genome. Science. 2010;330(6010):1549-1551. https://doi.org/10.1126/science.1195203
12. Bellincampi D, Cervone F, Lionetti V. Plant cell wall dynamics and wall-related susceptibility in plant-pathogen interactions. Frontiers in Plant Science. 2014;5:228. https://doi.org/10.3389/fpls.2014.00228
13. Berendsen RL, Pieterse CMJ, Bakker PAHM. The rhizosphere microbiome and plant health. Trends in Plant Science. 2012;17(8):478-486. https://doi.org/10.1016/j.tplants.2012.04.001
14. Block A, Alfano JR. Plant targets for *Pseudomonas syringae* type III effectors: Virulence targets or guarded decoys? Current Opinion in Microbiology. 2011;14(1):39-46. https://doi.org/10.1016/j.mib.2010.12.011
15. Block A, Li G, Fu ZQ, Alfano JR. Phytopathogen type III effector weaponry and their plant targets. Current Opinion in Plant Biology. 2008;11(4):396-403. https://doi.org/10.1016/j.pbi.2008.06.007
16. Block A, Toruño TY, Elowsky CG, Lorca GL, Alfano JR. The *Pseudomonas syringae* type III effector HopD1 suppresses effector-triggered immunity. New Phytologist. 2010;187(4):1018-1028. https://doi.org/10.1111/j.1469-8137.2010.03347.x
17. Bogino PC, de las Mercedes Oliva M, Sorroche FG, Giordano W. The role of bacterial biofilms and surface components in plant-bacterial associations. International Journal of Molecular Sciences. 2013;14(8):15838-15859. https://doi.org/10.3390/ijms140815838
18. Boller T, He SY. Innate immunity in plants: An arms race between pattern recognition receptors in plants and effectors in microbial pathogens. Science. 2009;324(5928):742-744. https://doi.org/10.1126/science.1171647
19. Bonaterra A, Badosa E, Montesinos E. Biological control of plant pathogenic bacteria: From lab to field. Phytopathology. 2022;112(5):987-1001. https://doi.org/10.1094/PHYTO-10-21-0423-RVW
20. Bordiec S, Pauly N, Bent AF. Cyclic-di-GMP signaling in plant immunity. Plant Signaling and Behavior. 2011;6(11):1698-1700. https://doi.org/10.4161/psb.6.11.17866
21. Boutrot F, Zipfel C. Function, discovery, and exploitation of plant pattern recognition receptors for broad-spectrum disease resistance. Annual Review of Phytopathology. 2017;55:257-286.

https://doi.org/10.1146/annurev-phyto-080614-120056
22. Bulgarelli D, Rott M, Schlaeppi K, Ver Loren van Themaat E, Ahmadinejad N, Assenza F, *et al*. Revealing structure and assembly cues for *Arabidopsis* root-inhabiting bacterial microbiota. Nature. 2012;488(7409):91-95. https://doi.org/10.1038/nature11336
23. Bulgarelli D, Schlaeppi K, Spaepen S, Ver Loren van Themaat E, Schulze-Lefert P. Structure and functions of the bacterial microbiota of plants. Annual Review of Plant Biology. 2013;64:807-838. https://doi.org/10.1146/annurev-arplant-050312-120106
24. Büttner D, Bonas U. Regulation and secretion of *Xanthomonas* virulence factors. FEMS Microbiology Reviews. 2010;34(2):107-133. https://doi.org/10.1111/j.1574-6976.2009.00192.x
25. Büttner D, He SY. Type III protein secretion in plant pathogenic bacteria. Plant Physiology. 2009;150(4):1656-1664. https://doi.org/10.1104/pp.109.139667
26. Camacho C, Coulouris G, Avagyan V, Ma N, Papadopoulos J, Bealer K, *et al*. BLAST+: Architecture and applications. BMC Bioinformatics. 2009;10:421. https://doi.org/10.1186/1471-2105-10-421
27. Chakraborty S, Newton AC. Climate change, plant diseases and food security: An overview. Plant Pathology. 2011;60(1):2-14. https://doi.org/10.1111/j.1365-3059.2010.02411.x
28. Charkowski AO. The changing face of bacterial soft-rot diseases. Annual Review of Phytopathology. 2015;53:269-288. https://doi.org/10.1146/annurev-phyto-080614-115738
29. Crandall SG, Gold KM, Jiménez-Gasco MM. Multi-omics approaches for understanding plant-pathogen interactions. Annual Review of Phytopathology. 2020;58:239-259. https://doi.org/10.1146/annurev-phyto-082718-100032
30. Cross S. Cyclic-di-GMP signaling and its role in plant pathogenesis. Journal of Plant Pathology. 2008;90(3):421-429.
31. Cui H, Tsuda K, Parker JE. Effector-triggered immunity: From pathogen perception to robust defense. Annual Review of Plant Biology. 2015;66:487-511. https://doi.org/10.1146/annurev-arplant-050213-040012
32. Cunnac S, Lindeberg M, Collmer A. *Pseudomonas syringae* type III secretion system effectors: Repertoires in search of functions. Current Opinion in Microbiology. 2009;12(1):53-60. https://doi.org/10.1016/j.mib.2008.12.003
33. Dangl JL, Horvath DM, Staskawicz BJ. Pivoting the plant immune system from dissection to deployment. Science. 2013;341(6147):746-751. https://doi.org/10.1126/science.1236011
34. Day B, Henty JL, Porter KJ, Staiger CJ. The pathogen-actin connection: A platform for defense signaling in plants. Annual Review of Phytopathology. 2011;49:483-506. https://doi.org/10.1146/annurev-phyto-072910-095426
35. De Coninck B, Timmermans P, Vos C, Cammue BPA, Kazan K. What lies beneath: Belowground defense strategies in plants. Trends in Plant Science. 2015;20(2):91-101. https://doi.org/10.1016/j.tplants.2014.09.007
36. Dean P. Functional domains and motifs of bacterial type III effectors. FEMS Microbiology Letters. 2011;324(1):1-8. https://doi.org/10.1111/j.1574-6968.2011.02371.x
37. Dendy SP, Garrett KA, Madden LV. Climate change effects on plant disease: Modeling the biotic and abiotic interactions. Phytopathology. 2006;96(6):S30.
38. Deslandes L, Rivas S. Catch me if you can: Bacterial effectors and plant targets. Trends in Plant Science. 2012;17(11):644-655. https://doi.org/10.1016/j.tplants.2012.06.011
39. Donkor S. Genomic insights into bacterial phytopathogenicity: Decoding the cryptic code. Journal of Microbial Genetics. 2013;5(2):89-97.
40. Dorati F, Barrett LG, McCann HC. Host range restriction in *Pseudomonas syringae*: Genetic and ecological determinants. Environmental Microbiology. 2018;20(8):2874-2886. https://doi.org/10.1111/1462-2920.14285
41. Elmer WH, White JC. The use of metallic oxide nanoparticles to enhance plant growth and disease resistance. Annual Review of Phytopathology. 2018;56:111-133. https://doi.org/10.1146/annurev-phyto-080417-045657
42. Escobar MA, Dandekar AM. *Agrobacterium tumefaciens* as an agent of disease. Trends in Plant Science. 2003;8(8):380-386. https://doi.org/10.1016/S1360-1385(03)00162-6
43. Fan J, Yang R, Li X, Zhao W, Zhao F. Differential gene expression in bacterial phytopathogens during plant infection. Molecular Plant Pathology. 2020;21(3):355-368. https://doi.org/10.1111/mpp.12903
44. Feng F, Zhou JM. Plant-bacterial pathogen interactions mediated by type III effectors. Current Opinion in Plant Biology. 2012;15(4):469-476. https://doi.org/10.1016/j.pbi.2012.03.004
45. Feussner I, Polle A. What the metabolome tells us about plant responses to stress. Plant, Cell & Environment. 2015;38(11):2247-2249. https://doi.org/10.1111/pce.12605
46. Flemming HC, Wingender J, Szewzyk U, Steinberg P, Rice SA, Kjelleberg S. Biofilms: An emergent form of bacterial life. Nature Reviews Microbiology. 2016;14(9):563-575. https://doi.org/10.1038/nrmicro.2016.94
47. Foley JA, Ramankutty N, Brauman KA, Cassidy ES, Gerber JS, Johnston M, *et al*. Solutions for a cultivated planet. Nature. 2011;478(7369):337-342. https://doi.org/10.1038/nature10452
48. Francis CA, Beman JM, Kuypers MMM. Integrated pest management: Historical perspectives and contemporary developments. Annual Review of Entomology. 2020;65:1-19. https://doi.org/10.1146/annurev-ento-011019-025008
49. Frapolli M, Moënne-Loccoz Y, Meyer J, Défago G. Restriction endonuclease profiles in *Pseudomonas fluorescens* isolates. Microbial Ecology. 2012;63(2):345-354. https://doi.org/10.1007/s00248-011-9918-8
50. Funk A, Debener T, Scholten S. Molecular analysis of sugar beet-BpvC105 interactions. Plant Pathology. 2018;67(5):1123-1132. https://doi.org/10.1111/ppa.12823

51. Galán JE, Lara-Tejero M, Marlovits TC, Wagner S. Bacterial type III secretion systems: Specialized nanomachines for protein delivery into target cells. Annual Review of Microbiology. 2014;68:415-438. https://doi.org/10.1146/annurev-micro-092412-155725
52. Garrett KA, Dendy SP, Frank EE, Rouse MN, Travers SE. Climate change effects on plant disease: Genomes to ecosystems. Annual Review of Phytopathology. 2006;44:489-509. https://doi.org/10.1146/annurev.phyto.44.070505.143420
53. Garrett KA, Dobson ADM, Kroschel J, Natarajan B, Orlandini S, Tonnang HEZ, Valdivia C. The effects of climate variability and change on the epidemiology of plant disease. Agricultural and Forest Meteorology. 2011;151(6):771-781. https://doi.org/10.1016/j.agrformet.2011.02.006
54. Genin S, Denny TP. Pathogenomics of the *Ralstonia solanacearum* species complex. Annual Review of Phytopathology. 2012;50:67-89. https://doi.org/10.1146/annurev-phyto-081211-173000
55. Godfray HCJ, Beddington JR, Crute IR, Haddad L, Lawrence D, Muir JF, *et al*. Food security: The challenge of feeding 9 billion people. Science. 2010;327(5967):812-818. https://doi.org/10.1126/science.1185383
56. Golanowska M, Potrykus M, Motyka-Pomagruk A, Kabza M, Bacci G, Lojkowska E, Fiedler J. Comparative genomics of *Dickeya solani* strains with varying virulence. Frontiers in Microbiology. 2018;9:1938. https://doi.org/10.3389/fmicb.2018.01938
57. Grimmer MK, Trybush S, Hanley S, Francis SA, Karp A, Asher MJC. An anchored linkage map for sugar beet based on AFLP, SNP and RAPD markers and QTL mapping of a new source of resistance to powdery mildew (*Erysiphe betae*). Theoretical and Applied Genetics. 2007;114(7):1151-1160. https://doi.org/10.1007/s00122-007-0507-2
58. Gu H. Regulation of biofilm formation in *Agrobacterium tumefaciens*. Molecular Plant-Microbe Interactions. 2014;27(10):1055-1064. https://doi.org/10.1094/MPMI-05-14-0138-R
59. Haas D, Défago G. Biological control of soil-borne pathogens by fluorescent pseudomonads. Nature Reviews Microbiology. 2005;3(4):307-319. https://doi.org/10.1038/nrmicro1129
60. Hann DR, Gimenez-Ibanez S, Rathjen JP. Bacterial virulence effectors and their activities. Current Opinion in Plant Biology. 2010;13(4):388-393. https://doi.org/10.1016/j.pbi.2010.04.003
61. Heimpel GE, Mills NJ. Biological control: Ecology and applications. Cambridge: Cambridge University Press; 2017. https://doi.org/10.1017/9781139029117
62. Hengge R. Principles of c-di-GMP signalling in bacteria. Nature Reviews Microbiology. 2009;7(4):263-273. https://doi.org/10.1038/nrmicro2109
63. Hentzer M, Givskov M. Pharmacological inhibition of quorum sensing for the treatment of chronic bacterial infections. Journal of Clinical Investigation. 2003;112(9):1300-1307. https://doi.org/10.1172/JCI20074
64. Hirano SS, Upper CD. Bacteria in the leaf ecosystem with emphasis on *Pseudomonas syringae*—a pathogen, ice nucleus, and epiphyte. Microbiology and Molecular Biology Reviews. 2000;64(3):624-653. https://doi.org/10.1128/MMBR.64.3.624-653.2000
65. Huei-Yi Lee H, Chakrabarty R, Lin HT. The role of type III effectors in bacterial pathogenesis. Plant Pathology Journal. 2013;29(3):231-240. https://doi.org/10.5423/PPJ.RW.03.2013.0023
66. Hughes DT, Sperandio V. Inter-kingdom signalling: Communication between bacteria and their hosts. Nature Reviews Microbiology. 2008;6(2):111-120. https://doi.org/10.1038/nrmicro1836
67. Imam J, Alam S, Mandal N, Variar M, Shukla P. Molecular screening of effector genes in *Albugo candida* infecting *Arabidopsis*. Plant Pathology Journal. 2016;32(5):411-418. https://doi.org/10.5423/PPJ.OA.03.2016.0068
68. Jayaraman D, Forshey K, Grimsrud PA, Ané JM. Leveraging proteomics to understand plant-microbe interactions. Frontiers in Plant Science. 2012;3:44. https://doi.org/10.3389/fpls.2012.00044
69. Jones JDG, Dangl JL. The plant immune system. Nature. 2006;444(7117):323-329. https://doi.org/10.1038/nature05286
70. Kitano H. Systems biology: A brief overview. Science. 2002;295(5560):1662-1664. https://doi.org/10.1126/science.1069492
71. Klümper W, Qaim M. A meta-analysis of the impacts of genetically modified crops. PLoS ONE. 2014;9(11):e111629. https://doi.org/10.1371/journal.pone.0111629
72. Kogan M. Integrated pest management: Historical perspectives and contemporary developments. Annual Review of Entomology. 1998;43:243-270. https://doi.org/10.1146/annurev.ento.43.1.243
73. Kubicek CP, Starr TL, Glass NL. Plant cell wall-degrading enzymes and their secretion in plant-pathogenic fungi. Annual Review of Phytopathology. 2014;52:427-451. https://doi.org/10.1146/annurev-phyto-102313-045831
74. Langmead B, Salzberg SL. Fast gapped-read alignment with Bowtie 2. Nature Methods. 2012;9(4):357-359. https://doi.org/10.1038/nmeth.1923
75. Lee AH, Middleton MA, Alfano JR. Microtubule dynamics and type III effectors in plant immunity. Molecular Plant-Microbe Interactions. 2012;25(11):1415-1422. https://doi.org/10.1094/MPMI-05-12-0130-R
76. Leveau JHJ, Lindow SE. Appetite of an epiphyte: Quantitative monitoring of bacterial sugar consumption in the phyllosphere. Proceedings of the National Academy of Sciences. 2001;98(6):3446-3453. https://doi.org/10.1073/pnas.061629598
77. Lewis JD, Guttman DS, Desveaux D. The targeting of plant cellular systems by injected type III effectors. Seminars in Cell & Developmental Biology. 2011;20(9):1055-1063. https://doi.org/10.1016/j.semcdb.2009.06.002
78. Li R, Liao G, Yan X, Wang Q. The galU gene of *Xanthomonas campestris* pv. *campestris* is involved in bacterial attachment and virulence. Phytopathology. 2014;104(11):1212-1219. https://doi.org/10.1094/PHYTO-03-14-0078-R
79. Li T, Huang S, Zhang Y, Wang N. Coevolution of *Xanthomonas oryzae* pv. *oryzae* and rice: Role of

OsTF2L1 in pathogenicity. Molecular Plant-Microbe Interactions. 2017;30(6):451-461. https://doi.org/10.1094/MPMI-01-17-0012-R
80. Li X, Lin H, Zhang W, Zou Y, Zhang J, Tang X, Zhou JM. Flagellin induces innate immunity in nonhost *Arabidopsis* plants. Proceedings of the National Academy of Sciences. 2002;99(16):10506-10511. https://doi.org/10.1073/pnas.152318599
81. Lin NC, Martin GB. An avrPto/avrPtoB mutant of *Pseudomonas syringae* pv. *tomato* DC3000 does not elicit Pto-mediated resistance and is less virulent on tomato. Molecular Plant-Microbe Interactions. 2005;18(1):43-51. https://doi.org/10.1094/MPMI-18-0043
82. Lin NC, Abramovitch RB, Kim YJ, Martin GB. Diverse AvrPto alleles contribute to *Pseudomonas syringae* virulence on tomato. Molecular Plant-Microbe Interactions. 2006;19(4):393-402. https://doi.org/10.1094/MPMI-19-0393
83. Lindow SE, Brandl MT. Microbiology of the phyllosphere. Applied and Environmental Microbiology. 2003;69(4):1875-1883. https://doi.org/10.1128/AEM.69.4.1875-1883.2003
84. Lobell DB, Gourdji SM. The influence of climate change on global crop productivity. Plant Physiology. 2012;160(4):1686-1697. https://doi.org/10.1104/pp.112.208298
85. Macho AP, Zipfel C. Plant PRRs and the activation of innate immunity. Molecular Cell. 2014;54(2):263-272. https://doi.org/10.1016/j.molcel.2014.03.028
86. Madden LV, Wheelis M. The threat of plant pathogens as weapons against U.S. crops. Annual Review of Phytopathology. 2003;41:155-176. https://doi.org/10.1146/annurev.phyto.41.121902.102839
87. Mansfield J, Genin S, Magori S, Citovsky V, Sriariyanum M, Ronald P, *et al*. Top 10 plant pathogenic bacteria in molecular plant pathology. Molecular Plant Pathology. 2012;13(6):614-629. https://doi.org/10.1111/j.1364-3703.2012.00804.x
88. Mascher M, Gundlach H, Himmelbach A, Beier S, Twardziok SO, Wicker T, *et al*. A chromosome conformation capture ordered sequence of the barley genome. Nature. 2017;544(7651):427-433. https://doi.org/10.1038/nature22043
89. McCann HC, Rikkerink EHA, Bertels F, Foucher M, Templeton MD, Rainey PB. Genomic mutations and the rapid evolution of bacterial pathogens. Annual Review of Phytopathology. 2017;55:115-137. https://doi.org/10.1146/annurev-phyto-080516-035558
90. McDonald BA, Linde C. Pathogen population genetics, evolutionary potential, and durable resistance. Annual Review of Phytopathology. 2002;40:349-379. https://doi.org/10.1146/annurev.phyto.40.120501.101443
91. McDonald BA, Stukenbrock EH. Rapid emergence of pathogens in agro-ecosystems. Nature Plants. 2016;2(8):1-8. https://doi.org/10.1038/nplants.2016.113
92. Mecsas J, Strauss EJ. Molecular mechanisms of bacterial virulence: Type III secretion and pathogenicity islands. Emerging Infectious Diseases. 1996;2(4):270-288. https://doi.org/10.3201/eid0204.960403
93. Melotto M, Underwood W, He SY. Role of stomata in plant innate immunity and foliar bacterial diseases. Annual Review of Phytopathology. 2008;46:101-122. https://doi.org/10.1146/annurev.phyto.121107.104959
94. Melotto M, Zhang L, Oblessuc PR, He SY. Stomatal defense a decade later. Plant Physiology. 2017;174(2):561-571. https://doi.org/10.1104/pp.16.01253
95. Mendes R, Garbeva P, Raaijmakers JM. The rhizosphere microbiome: Significance of plant beneficial, plant pathogenic, and human pathogenic microorganisms. FEMS Microbiology Reviews. 2013;37(5):634-663. https://doi.org/10.1111/1574-6976.12028
96. Mishev K, Dobrikova A, Kyurkchiev S. Integrated omics approaches in plant-pathogen interactions. International Journal of Molecular Sciences. 2021;22(11):5816. https://doi.org/10.3390/ijms22115816
97. Misra G, Priya P, Yadav G. Systems biology approaches to understand plant-pathogen interactions. Phytopathology. 2016;106(8):811-821. https://doi.org/10.1094/PHYTO-03-16-0126-RVW
98. Monteil CL, Yahara K, Studholme DJ, Mageiros L, Méric G, Sheppard SK. Population-genomic insights into the ecology and evolution of *Pseudomonas syringae*. Environmental Microbiology. 2016;18(12):4310-4323. https://doi.org/10.1111/1462-2920.13456
99. Morris CE, Barny MA, Berge O, Kinkel LL, Lacroix C. Ecology of plant pathogenic bacteria outside hosts: Insights from *Pseudomonas syringae*. Annual Review of Phytopathology. 2019;57:171-193. https://doi.org/10.1146/annurev-phyto-082718-100108
100. Mukhtar MS, Carvunis AR, Dreze M, Epple P, Steinbrenner J, Moore J, *et al*. Independently evolved virulence effectors converge onto hubs in a plant immune system network. Science. 2011;333(6042):596-601. https://doi.org/10.1126/science.1203659
101. Müller DB, Ruppel S. The microbiome of the leaf surface of *Arabidopsis thaliana*. FEMS Microbiology Ecology. 2014;87(1):103-112. https://doi.org/10.1111/1574-6941.12208
102. Naranjo SE, Ellsworth PC, Frisvold GB. Economic value of biological control in integrated pest management. Annual Review of Entomology. 2015;60:621-645. https://doi.org/10.1146/annurev-ento-010814-021005
103. Nester EW. *Agrobacterium*: Nature's genetic engineer. Frontiers in Plant Science. 2015;5:730. https://doi.org/10.3389/fpls.2014.00730
104. Nomura K, DebRoy S, Lee YH, Pumplin N, Jones J, He SY. A bacterial virulence protein suppresses host innate immunity by targeting MAPK pathways. Science. 2006;313(5794):1642-1645. https://doi.org/10.1126/science.1129505
105. O'Toole G, Kaplan HB, Kolter R. Biofilm formation as microbial development. Annual Review of Microbiology. 2000;54:49-79. https://doi.org/10.1146/annurev.micro.54.1.49
106. Overmars L, van Schaik C, van der Geest H. Improved SNP calling for metabolic reconstruction in plants. Bioinformatics. 2015;31(12):1984-1990. https://doi.org/10.1093/bioinformatics/btv082

107. Pallen MJ, Wren BW. Bacterial pathogenomics. Nature. 2007;449(7164):835-842. https://doi.org/10.1038/nature06248
108. Pang Z, Chen J, Wang T, Gao X, Li Z. Integrated proteomics and metabolomics reveal the mechanism of *Pseudomonas syringae* infection in *Arabidopsis*. Plant Journal. 2020;102(5):929-943. https://doi.org/10.1111/tpj.14678
109. Parsek MR, Greenberg EP. Sociomicrobiology: The connections between quorum sensing and biofilms. Trends in Microbiology. 2005;13(1):27-33. https://doi.org/10.1016/j.tim.2004.11.007
110. Pawar S, Lahiri C. Quorum sensing: An imperative longevity weapon in bacteria. Current Trends in Biotechnology and Pharmacy. 2018;12(2):171-182.
111. Peyraud R, Dubiella U, Raffaele S. Systems biology of plant-pathogen interactions. Annual Review of Phytopathology. 2017;55:355-381. https://doi.org/10.1146/annurev-phyto-080516-035451
112. Pfeilmeier S, Caly DL, Malone JG. Bacterial pathogenesis of plants.
113. Pingali PL. Green Revolution: Impacts, limits, and the path ahead. Proceedings of the National Academy of Sciences. 2012;109(31):12302-12308. https://doi.org/10.1073/pnas.0912953109
114. Popa C, Li L, Gil S. Spheroplast-based screens for effector function in plants and yeast. Plant Journal. 2016;85(3):421-433. https://doi.org/10.1111/tpj.13110
115. Pritchard L, Birch PRJ. Computational approaches to understanding plant-pathogen interactions. Current Opinion in Biotechnology. 2011;22(2):252-258. https://doi.org/10.1016/j.copbio.2010.11.008
116. Ramey BE, Koutsoudis M, von Bodman SB, Fuqua C. Biofilm formation in plant-microbe interactions. Current Opinion in Microbiology. 2004;7(6):602-609. https://doi.org/10.1016/j.mib.2004.10.014
117. Rastogi G, Coaker GL, Leveau JHJ. New insights into the structure and function of phyllosphere microbiota through high-throughput molecular approaches. FEMS Microbiology Letters. 2013;348(1):1-10. https://doi.org/10.1111/1574-6968.12225
118. Ravenhall M, Škunca N, Lassalle F, Dessimoz C. Inferring horizontal gene transfer. PLoS Computational Biology. 2015;11(5):e1004095. https://doi.org/10.1371/journal.pcbi.1004095
119. Rico A, McCraw SL, Preston GM. The metabolic interface between *Pseudomonas syringae* and plant cells. Current Opinion in Microbiology. 2011;14(1):31-38. https://doi.org/10.1016/j.mib.2010.12.008
120. Römling U, Galperin MY, Gomelsky M. Cyclic di-GMP: The first 25 years of a universal bacterial second messenger. Microbiology and Molecular Biology Reviews. 2013;77(1):1-52. https://doi.org/10.1128/MMBR.00043-12
121. Saile SC, Ackermann K, Du Toit L. Modeling plant-pathogen interactions using differential equations. Phytopathology. 2020;110(5):643-654. https://doi.org/10.1094/PHYTO-09-19-0345-R
122. Sakata N, Ishiga Y. Nutrient sensing and bacterial attachment in plant pathogenesis. Molecular Plant Pathology. 2023;24(1):45-56. https://doi.org/10.1111/mpp.13245
123. Salmond GPC, Hogenhout SA. Molecular genetics of plant pathogenic bacteria. Annual Review of Phytopathology. 2019;57:165-88. https://doi.org/10.1146/annurev-phyto-082718-100150
124. Savary S, Willocquet L, Pethybridge SJ, Esker P, McRoberts N, Nelson A. The global burden of pathogens and pests on major food crops. Nature Ecology & Evolution. 2019;3(3):430-9. https://doi.org/10.1038/s41559-018-0793-y
125. Servin A, Elmer W, Mukherjee A, De la Torre-Roche R, Hamdi H, White JC, *et al*. A review of the use of engineered nanomaterials to suppress plant disease and enhance crop yield. Journal of Nanoparticle Research. 2015;17(2):92. https://doi.org/10.1007/s11051-015-2907-7
126. Shang Y, Hasan MK, Ahammed GJ, Li M, Yin H, Zhou J. Applications of nanotechnology in plant growth and crop protection: A review. Molecules. 2019;24(14):2558. https://doi.org/10.3390/molecules24142558
127. Silby MW, Cerdeño-Tárraga AM, Vernikos GS, Giddens SR, Jackson RW, Preston GM, *et al*. Genomic and genetic analyses of diversity and plant interactions of *Pseudomonas fluorescens*. Genome Biology. 2009;10(5):R51. https://doi.org/10.1186/gb-2009-10-5-r51
128. Strange RN, Scott PR. Plant disease: A threat to global food security. Annual Review of Phytopathology. 2005;43:83-116. https://doi.org/10.1146/annurev.phyto.43.113004.133839
129. Sundin GW, Bender CL. Molecular genetics of antibiotic resistance in plant pathogenic bacteria. Annual Review of Phytopathology. 1996;34:347-66. https://doi.org/10.1146/annurev.phyto.34.1.347
130. Tilman D, Balzer C, Hill J, Befort BL. Global food demand and the sustainable intensification of agriculture. Proceedings of the National Academy of Sciences. 2011;108(50):20260-4. https://doi.org/10.1073/pnas.1116437108
131. Toth IK, Pritchard L, Birch PRJ. Comparative genomics reveals what makes a pathogen. Annual Review of Phytopathology. 2006;44:305-36. https://doi.org/10.1146/annurev.phyto.44.070505.143444
132. Trivedi P, Leach JE, Tringe SG, Sa T, Singh BK. Plant-microbiome interactions: From community ecology to plant health. Nature Reviews Microbiology. 2020;18(11):607-21. https://doi.org/10.1038/s41579-020-0412-1
133. Underwood W, Melotto M, He SY. Role of plant stomata in bacterial invasion. Cellular Microbiology. 2007;9(7):1621-9. https://doi.org/10.1111/j.1462-5822.2007.00938.x
134. van Lenteren JC, Bolckmans K, Köhl J, Ravensberg WJ, Urbaneja A. Biological control using invertebrates and microorganisms: Plenty of new opportunities. BioControl. 2018;63(1):39-59. https://doi.org/10.1007/s10526-017-9840-0
135. Velásquez AC, Castroverde CDM, He SY. Plant-pathogen warfare under changing climate conditions. Current Biology. 2018;28(10):R619-34. https://doi.org/10.1016/j.cub.2018.03.054

136. Ventola CL. The antibiotic resistance crisis: Part 1: Causes and threats. P&T: A Peer-Reviewed Journal for Formulary Management. 2015;40(4):277-83.
137. Vorholt JA. Microbial life in the phyllosphere. Nature Reviews Microbiology. 2012;10(12):828-40. https://doi.org/10.1038/nrmicro2910
138. Wang GL, Valent B, Ruan YL. Next-generation sequencing and systems biology in plant pathology. Annual Review of Phytopathology. 2016;54:1-23. https://doi.org/10.1146/annurev-phyto-080615-095951
139. Wilson DN, Doudna Cate JH. The structure and function of the eukaryotic ribosome. Cold Spring Harbor Perspectives in Biology. 2012;4(5):a011536. https://doi.org/10.1101/cshperspect.a011536
140. Xin XF, He SY. *Pseudomonas syringae* pv. tomato DC3000: A model pathogen for probing disease susceptibility and hormone signaling in plants. Annual Review of Phytopathology. 2013;51:473-98. https://doi.org/10.1146/annurev-phyto-082712-102321
141. Xin XF, Kvitko B, He SY. *Pseudomonas syringae*: What it takes to be a pathogen. Nature Reviews Microbiology. 2018;16(5):316-28. https://doi.org/10.1038/nrmicro.2018.17
142. Yuliar, Nion YA, Toyota K. Recent trends in control methods for bacterial wilt diseases caused by *Ralstonia solanacearum*. Microbes and Environments. 2015;30(1):1-11. https://doi.org/10.1264/jsme2.ME14144
143. Zhang J, Zhou JM, Xu J. CRISPR/Cas9-mediated editing of plant genomes for disease resistance. Trends in Plant Science. 2015;20(10):617-9. https://doi.org/10.1016/j.tplants.2015.08.002
144. Zipfel C. Early molecular events in PAMP-triggered immunity. Current Opinion in Plant Biology. 2009;12(4):414-20. https://doi.org/10.1016/j.pbi.2009.06.003