PLANT PROTECTION | REVIEW ARTICLE

Impacts of nitrogen on plant disease severity and plant defense mechanism

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ARTICLE INFORMATION	ABSTRACT

Nitrogen (N) is considered the most important factor to help the growth and development of plants. This is the building block for plant protoplasm and the chlorophyll molecule component for the photosynthesis process. Although it is apparent that the availability of N affects disease, the underlying mechanisms remain unknown. Many data indicate that the greater use of N fertilizers affected crop disease incidence. In comparison, cases are also recorded in which a decrease in N fertilization increases the severity of the disease, suggesting a complex relationship between them. N plays a significant role in regulating signaling networks that are active in reacting to a broad variety of biotic and abiotic stresses. In terms of physical, biochemical, and molecular mechanisms, the relationship between N and plant defense is considered. N has negative effects on physical defenses and the development of anti-microbial phytoalexins, but positive effects on defense-related enzymes and proteins that influence both local defense and systemic resistance. These all factors are implicated in plant defense signaling pathways but their role in plant defense is less well studied. This review aims to explain current knowledge of pathways connecting plant N status with the plant disease severity and plant defense. While this analysis highlights the crucial role of N nutrition in plant defense, further research is desperately required to provide a thorough overview of how interacting networks affect competing virulence and defense mechanisms.

Keywords: Nitrogen, disease severity, plant defense, Physical defense, biochemical defense, molecular defense

1 Introduction

The crops need at least 14 mineral elements in addition to carbon dioxide, oxygen, and water for their nutrition. These elements are taken from the soil and are usually grouped as primary nutrients (nitrogen, phosphorous, potassium), secondary nutrients (calcium, magnesium, sulphur) and micronutrients (boron, chlorine, manganese, iron, zinc, copper, molybdenum and nickle) (White and Brown, 2010). Nutrients are key factors in disease resistance and tolerance as well as for the growth and development of plants (Huber and Haneklaus, 2007). The resistance of the host plant is their ability to restrict the development and reproduction of invasive pathogens while the tolerance of the host is measured as being able to sustain its growth or yield, even in the infection (Graham and Webb, 1991). There are two forms of key defense mechanisms that may influence mineral nutrition either through creating mechanical barriers, mainly by the formation of thicker cell walls, or through synthesizing natural defense compounds, such as phytoalexins, antioxidants, and flavonoids, which protect pathogenic agents (Bhaduri et al., 2014). Although genetically regulated resistance and tolerance to plant disease are impaired by the environment, and in particular by nutrient and toxicity deficiencies (Krauss, 1999; Agrios, 2005). However, some nutrients are more likely to cause plant diseases. It

should be noted that a single nutrient may have opposite effects on different diseases and in different ecosystems, i.e. same element that raises the occurrence of one disease while reducing the occurrence of others (Agrios, 2005).

Nitrogen plays a vital role in agriculture not only by increasing the yield but also by enhancing the quality of the product (Leghari et al., 2016). The optimum N rate improves photosynthetic processes, leaf area production, leaf area duration, and the net assimilation rate (Horvath et al., 2014). Production and yield attributing parameters such as plant height, number of tillers, number of spikelets, weight of 1000 grams, etc. are significantly affected by fertilization of nitrogen (Ali et al., 2011). N fertilizers became a vital component in the Green revolution which changed farming practices and progress all over the world. In the second half of the 20th century, the drastic rise in the use and usage of N fertilizers became essential for feeding the growing world population (San Martín, 2017). The form of N can change the pH of the soil which in turn regulates the availability of other nutrients with residual N, time of fertilizer usage, earlier crop, and the ratio of NH_4^+ to NO_3^- control disease susceptibility as a whole (Bhaduri et al., 2014). Effects can depend on the nutritional characteristics of the host, the type of pathogen (obligate vs. facultative parasites) or even the timing of N application, but there is a dearth of rigorous research to study the impact of N supply on disease tolerance under controlled conditions (Hoffland et al., 2000). Even though the excess of N in plants is one of the most significant factors affecting disease growth, many studies remain obscure and contradict each other about the influence of N on disease production, and the reasons for this elusiveness were still little understood (Gupta et al., 2017). This review summarizes the relationship of N nutrition with the disease severity and its role in the defense mechanism.

2 Nitrogen and disease severity

A significant factor in increasing crop production has been the increase in the use of N fertilizers over the last few decades. Agricultural practices that rely on extensive application of synthetic N fertilizer will dramatically affect the interplay for N between host and pathogen (Kant et al., 2011). Nitrogen is consumed by most plant species in different forms such as Nitrate (NO_3^-) and Ammonium (NH_4^+) and can influence plant physiological processes such as enzyme activity, respiration rate, water balance, photosynthetic rate, and signaling pathway, ultimately causing plant diseases. Various forms of N may also regulate disease tolerance by influencing metabolic pathogen adaptation and signals regulating stimulation of the virulence factor (Horchani et al., 2010;

Lopez-Berges et al., 2010; Yang et al., 2012; Zhou et al., 2017). While N input comprises plant defense, it has been shown that over-application of nitrogen fertilizers improves the production of diseases (Solomon and Oliver, 2001). It was suggested that increased supply of N induces greater susceptibility to disease by changes in the structure of the canopy, decreases in phenol, increases in crop density which could provide a favorable microclimate for pathogen development. Increase in the rate of N increased the severity of sheath blight in rice (caused by *Rhizoctonia solani*) as influenced by canopy structure (Wu et al., 2014). High dose of N increased the disease severity of sheath blight in rice with reduced phenolic contents (Prasad et al., 2010). Biswas et al. (2016) suggested that increased in amount of total phenolic content is associated with decrease in severity of spot blotch in wheat. In yellow rust (caused by *Puccinia striiformis* f.sp. *tritici*), however, leaf N content was essential to support epidemics on winter wheat, rather than canopy structure (Neumann et al., 2004). Increased dosage of nitrogen fertilizer has a possible detrimental effect on soil carbon cycling, and encourages fungal genera with established pathogenic characteristics, exposing a negative effect of intense fertilization. The relative abundance of Ascomycota was usually higher in high N dose conditions than low N, whereas it was lower in Basidiomycota (Paungfoo-Lonhienne et al., 2015). Higher dose of N fertilizer promotes late blight in potato (Dey and Chakraborty, 2016) whereas Jha et al. (2019) revealed that both less and high doses of N application invites more disease.

Nitrogen-induced susceptibility (NIS) affects both wheat and rice blast diseases and may be related to NUE (Nitrogen use efficiency). In addition, it showed that complete resistance triggered by major resistance genes may also be affected (Ballini et al., 2013). Increased supply of N to the plant led to higher development of spore by the powdery mildew fungus *Oidium lycopersicum*, and increased colonization of the leaf by the bacterium *Pseudomonas syringae* pv. *tomato* indicated that increased leaf N caused greater susceptibility to these diseases. Increased supply of N to the plant contributed to higher colony density and spore production of *Erysiphe graminis* f.sp. *hordei* on seedlings of six spring barley cultivars showing that these pathogens were more vulnerable (Jensen and Munk, 1997). Besides, different forms of nutrition influence pathogenic microorganism growth. In contrast to NO_3^- feeding, NH_4^+ can inhibit the proliferation of *Verticillium dahlia* (Tenuta and Lazarovits, 2002) and *Elsinoe fawcettii* (Wang et al., 2009) while the contrary events were also reported in *Aspergillus* which produce aflatoxin (Calvo et al., 2002). Some impacts of N on disease severity are discussed in Table 1.

Table 1. Effects of nitrogen on disease severity of different crops

Host, disease & pathogen	Severity	Remarks	References
Rice Blast (*Magnaporthe oryzae*)	High N regime enhanced susceptibility.	↑ expression of the fungal pathogenicity program overpassed plant defense.	Huang et al. (2017)
Stripe rust of wheat (*Puccinia striiformis* f.sp. *tritici*)	Increased severity during the grain filling stage due to an increased rate of Nitrogen.	↓ yields of susceptible rust varieties and their impact is most likely associated with reduced N intake.	Devadas et al. (2014)
Powdery mildew of wheat (*Blumeria graminis*)	Disease incidence (DI) and Disease Severity Index (DSI) increased due to an increase in N fertilizer.	Microclimate and biological control mechanisms can affect severity.	Chen et al. (2007)
Gummy stem blight of watermelon (*Didymella bryoniae*) Downy mildew of watermelon (*Pseudoperonospora cubensis*)	Highest severity when N doses (80 & 120 kg ha^{-1} as compared to control).		Santos et al. (2009)
Foliar disease of barley (*Rhynchosporium secalis*, *Drechslera teres Cochliobolus sativus*)	The increasing rate of N from 50% to 100% did not affect.		Turkington et al. (2012)
Obligate parasite (*Puccinia graminis, Erysiphe graminis, Oidium lycopercicum*)	High N supply increases the severity of the infection.	Obligate parasites require assimilates of apoplast or directly from living cells by a haustorium.	Gupta et al. (2017); Hoffland et al. (2000); Snoeijers et al. (2000); Agrios (2005)
Facultative parasite (*Fusarium oxysporum, Alternaria solani, Xanthomonas* spp.)	High N supply decreases the severity of the infection.	These are semi-saprophytes that favor senescent tissues or release toxins to damage or destroy host plant cells.	
Take-all disease of wheat (*Gaeumannomyces graminis* var. *tritici*)	High N reduced the severity of the disease.	Fertilizer with a low rate of nitrification reduces the severity. N fertilizer ↑ plant vigor resulting production of more roots.	Brennan (1993)
Leaf spot disease of wheat [Tan spot (*Pyrenophora tritici-repentis*), blotch (*Stagonospora nodorum, Phaeosphaeria nodorum*)]	Adequate fertilizer with a Non-till condition appears to reduce the risk of leaf spot.	Production of lesions may be facilitated by N deficiency or by an imbalance of nutrients.	Krupinsky et al. (2007)
Botrytis cinerea of tomato	↑ fertilizer lowers the susceptibility.	Low availability of substrate hinders the growth of moderately aggressive isolates which are more efficient against plant defense.	Lecompte et al. (2010)

3 Pathogen recognition by plants

Defense mechanism in the plant can be simply classified into Constitutive defense (production by and present in the plant irrespective of attack by pathogens) and Induced defense (production by and present in the plant in respective of attack by pathogens) (Dietrich et al., 2004). Constitutive (passive / continuous) defense is the first line of defense that consists of preformed barriers such as cell wall, barks, waxy epidermal cuticles as well as pre-formed chemical compounds that not only aid in the defense against pathogens but also provide strength and rigidity to plants (Boots and Best, 2018; Serrano et al., 2014). Different antimicrobial chemicals, antimicrobial proteins, antimicrobial enzymes as well as secondary metabolites (Terpenoids, Phenolics, N compounds like alkaloids) are activated after the infection (Freeman and Beattie, 2008; Ribera and Zuñiga, 2012). Defense-related hormones like Salicylic acid (SA), Jasmonic acid (JA), Ethylene, Abscisic Acid (ABA), Auxins may augment their importance on plant immunity. Accumulation of Reactive oxygen species (ROS), defense-related genes like pathogenesis related genes (PR genes), activation of defense-related signaling pathways, etc. are also inducible defenses for pathogens (Ponce de Leon and Montesano, 2013; Garcion et al., 2014).

Pathogen's cell surface contains highly conserved molecules known as Pathogens (or Microbes or Damage) Associated Molecular Patterns (PAMPs/MAMPs/DAMPs) like bacterial flagellin and fungal chitin which has an important microbial fitness or survival Feature. Pattern recognition receptors (PRRs) are present in the cell surface of the host which recognizes MAMPs and activates an innate immune response called PAMP-triggered immunity (PTI) (Bahia et al., 2018). Efficient pathogens can overcome PTI by secreting effectors that suppress PTI responses leading to Effector-triggered susceptibility (ETS). During the course of evolution, plants responded to these effectors by developing cytoplasmic resistance (R) proteins which recognize (presence or action of) single effectors and activate the effector-triggered immunity (ETI). So simply, a second class of perception, ETI involves the identification of pathogen virulence molecules (effectors) by intracellular receptors (Sun et al., 2020). ETI is a prolonged and more robust solution than PTI but can only be used against particular infections of the pathogen. Both PTI and ETI function across interconnected signaling networks to protect against pathogens more precisely with differing responses depending on the pathogen that interacts. When the result of a plant-pathogen interaction is a disease, the relationship is considered compatible, while it is considered incompatible when the result is resistance (Fagard et al., 2014).

BAK1 is a PAMP-triggered central immunity regulator. Most identified PRRs require the leucine-rich repeat (LRR) receptor kinase, Brassinosteroid insensitive 1-associated Kinase 1 (BAK1) for function. An exception is the Chitin Elicitor Receptor Kinase 1 (CERK1), a fungal chitin receptor that also responds to mysterious bacterial PAMP. However, many are recognized by intracellular nucleotide-binding (NB)-LRR receptors, which induces effector-triggered immunity (ETI) (Dodds and Rathjen, 2010). The PTI and ETI activate downstream defense including the local defense response and Hypersensitive responses (HR) (rapid plant cell death), mediated by a series of signal or regulatory factors such as reactive oxygen species (ROS), Reactive nitrogen species (RNS), Mitogen-activated protein kinase (MAPK) cascades and hormones. The antioxidant systems are also stimulated to maintain intracellular redox balance. Furthermore, signals including nitric oxide (NO), salicylic acid (SA), and NADPH oxidase (RBOH)-generated ROS act to induce systemic acquired resistance (SAR) in uninfected tissues (Tsuda and Katagiri, 2010). The relationship between plants and pathogenic microorganisms is complex and also several environmental factors can affect it. Among them, N is the one which can limit pathogen growth and affect the elicitation and deployment of plant defenses (Bolton and Thomma, 2008). The N status of the plant may affect plant resistance to various abiotic and biotic stresses. Numerous studies have shown that N supply can potentially alter plant resistance to abiotic stress through effect-associated plant growth patterns and N-mediated signal transduction (Xuan et al., 2017). Different forms of N (NH_4^+/NO_3^-) have different effects on signaling and metabolism pathways so still the basic N-mediated defense mechanisms are not yet fully understood. Within this study, we find the relationship between Nutrition and plant disease occurrence as indicated from physical, biochemical, and molecular perspective by its impact on host and pathogen.

4 Impact of N on plant defense

N supply can influence plant-pathogen interactions. Some literature insights that N has an impact on the trade-off between plant growth and defense (Hakulinen et al., 1995) while some literature showed that poor nutrition weakens plants and is thus not conducive to defense (Snoeijers et al., 2000). Therefore, there is still a lot of contradiction in the effect of nutritional status on the development of plant defense.

4.1 Physical defense mechanism

Plant waxes give defense against bacterial, fungal infections, and reduce encounters between plants and insects (Ahmad et al., 2015). Kohlrabi (*Brassica oleracea*

var. *gongylodes*) administered N as ammonium ions produced glaucous leaves relative to nitrate supplies that formed glossy leaves. The open wax structures and reduced deposits found on nitrate-fed kohlrabi leaves would increase surface wetting and pesticide absorption, thus promoting pathogenic infection compared to plants fed with ammonium (Blanke et al., 1996). It was found that there were negative effects of N fertilization on the thickness of the epidermal cuticles and found susceptible to brown rot diseases on a peach (Daane et al., 1995). ATL31 (Arabidopsis Toxicos en Levadura31), a ubiquitin ligase plays an important role in connecting the C/N response with basal immunity to prevent the penetration of powdery mildew fungus by promoting the establishment of cell wall appositions (papillae) at fungal entry sites through its association with SYP121 (Syntaxin of Plants121) (the plasma membrane-localized soluble N-ethylmaleimide-sensitive fusion protein) (Maekawa et al., 2014). However, Lignin biosynthesis genes are downregulated by higher nitrogen fertilizer, which causes lignin deficiency in the secondary cell walls and mechanical tissue structure to collapse. Subsequently, this results in internodes with decreased mechanical strength and low resistance to lodging in japonica rice (Zhang et al., 2017). Depending on the modifications made and the pathogens tested, plants with altered secondary cell walls can either have an increased or reduced resistance to the pathogen, or no effect at all. A more thorough study of the role of the plant cell wall in pathogen resistance and of the biochemical networks that underlie this resistance is required (Miedes et al., 2014).

4.2 Biochemical and enzymatic defense

As pathogens invade plants, their stress can trigger biochemical and physiological as well as enzymatic behavior that can reduce the rate of spread of disease (Prasannath, 2017). Most alkaloids enable plants to combat pathogen attacks. Important examples of this group include berberine, tubocurarine, colchicine, morphine, sanguinarine, etc. are synthesized from tyrosine (Ahmed et al., 2017). Root exudes such as amino acids and other chemical compounds that are the source of N will change both physical and chemical characteristics of the soil, which in turn help to make the soil a suitable environment for microbial communities to flourish (Doughari, 2015). Different amino acids like glutamine, glutamate, alanine, and γ-aminobutyric acid (GABA) were found to be increased in the apoplast of tomato leaves supporting the pathogen growth (*Cladosporium fulvum*) through increased protease activity possibly due to induction of an extracellular serine protease P69B (Solomon and Oliver, 2001). The use of NO_3^- or NH_4^+ fertilizers influences the outcome of plant – pathogen interactions, in addition to considering only N material. NO_3^- feed-

ing improves hypersensitive reaction- (HR) mediated resistance, whereas nutrition with ammonium can compromise the protection. Metabolically, NO_3^- increases the production of polyamines such as spermine and spermidine, which are identified defensive signals, with NH_4^+ feeding contributing to increased amounts of γ-aminobutyric acid (GABA) which may be a nutritional source for the pathogen (Mur et al., 2005).

Commonly studied chemical elicitors include salicylic acid, methyl salicylate, benzothiadiazole, benzoic acid, chitosan etc. which affect the production of phenolic compounds and the activation of various enzymes related to defense (Thakur and Sohal, 2013). DCA (3,5-dichloroanthranilic acid) works as WRKY 70- dependent step of SA defense signaling networking with no requirement of NPR1. NC1 (N-cyanomethyl-2-chloroisonicotinamide) induces SAR by triggering defense signaling steps operating between SA and NPR1. Isotianil triggers the accumulation of defense-related enzymes such as lipoxygenase in rice. Sulfanilamides induces PR1 gene expression. Imprimatin activates signaling steps downstream of SA accumulation and induces expression of known SA responsive genes (Tripathi et al., 2019; Bektas and Eulgem, 2015). Major nitrogen-based enzymes or chemical compounds for plant defense are presented in Table 2.

4.3 Molecular defense

Plant hormones play a major role in regulating the reaction of plant defense against a broad range of biotic and abiotic stress. Salicylic acid (SA), Jasmonates (JA), and Ethylene (ET) are important plant defense reaction regulators. As Abscisic acid (ABA), Auxin, Gibberellic acid (GA), Cytokinin (CK), Brassinosteroids (BR), and Peptide hormones are also involved in signaling pathways for plant defense, but their function in plant defense is less well known (Bari and Jones, 2009). The infection of plants with different pathogens leads to changes in specified phytohormone levels (Adie et al., 2007). SA plays a significant role in activating defenses against biotrophic and hemibiotrophic pathogens, as well as the development of systemic acquired resistance (SAR). The rise in SA levels in pathogen-challenged plant tissues and exogenous applications contributes to the activation in pathogenesis-related (PR) genes and increased resistance to a wide variety of pathogens (Bari and Jones, 2009). However, JA and ET are most generally concerned with protecting against necrotrophic pathogen attacks. Also, SA and JA / ET are mutually antagonistic defense pathways (van Loon et al., 2006). While mechanistic theories of this antagonistic and cooperative crosstalk are scarce, the antagonism between JA and SA pathways involves the activation of proteins like NPR1 (Ankyrin repeat protein)

Table 2. Nitrogen-based enzymes or chemical compounds for plant defense

Disease/ Host/ Pathogen	Enzymes/Chemical compound	References
Late Blight of potato (*Phytophthora infestans*)	Peroxidase (POD), Polyphenol oxidase (PPO), Phenylalanine ammonia lyase (PAL)	Kumar et al. (2017)
Anthracnose in cucumber (*Colletotrichum lagenarium*)	POD, PPO, Chitinases, β-1,3 glucanases, Phenylalanine	Franzener et al. (2018)
Brown spot of wheat (*Bipolaris sorokiniana*)	Superoxide dismutase, Catalase, Glutathione, Flavonoid	Pittner et al. (2019)
Panama disease of banana (*Fusarium oxysporum* f.sp. *cubense*)	POD, PPO, Chitinase, Phenolics	Thakker et al. (2012)
Bacterial speck of tomato (*Pseudomonas syringae*)	Superoxide dismutase	Gonzalez-Hernandez et al. (2019)
Fusarium wilt of tomato (*Fusarium oxysporum*)	Phenols, Peroxidase	Sarhan et al. (1982)
Rice blast (*Magnaporthe oryzae*)	PAL, Glucanases, Chitosanase, Superoxide dismutase	Thapa et al. (2018); Ballini et al. (2013)
Potato (*Xanthomonas axonopodis*, *Ralstonia solanacearum*)	PPO, POD	Poiatti et al. (2009)
Plum (*Taphrina pruni*)	O-diphenols	Fuchs and Spiteller (1998)
Shoot blight in *Populus tremuloides* (*Venturia moreletii*)	Tannins	Holeski et al. (2009)
Poplar leaf rust (*Melampsora medusae*)	Proanthocyanidin	Miranda et al. (2007)

and WRKY70, which trigger the expression of SA-responsive genes while suppressing JA-responsive genes (Li et al., 2004). Conversely, coordination between JA and ET in stimulating necrotrophic defenses can be explained by their coordinated activation of the Ethylene Response Factor1 (ERF1), which causes PDF 1.2 (Plant defensin) gene expression for plant resistance (Lorenzo et al., 2003; Pieterse et al., 2009). Contrary to the pathogen infection scenario, however, ET and JA antagonize each other in the triggering of wound responses. The fine-tuning of this antagonism depends on the activation equilibrium of both ERF1 and MYC2 hormones, a further transcription factor that differentially controls two branches of the JA signaling pathway (Lorenzo et al., 2004). Vega et al. (2015) showed that the *Solanum lycopersicum*-*Botrytis cinerea* relationship repressed ERF1 under N-deficiency. OPR3 gene also reported a similar repressive effect with N deficiency, which improves tomato resistance to *B. Cinerea* impacting the JA biosynthesis route (Scalschi et al., 2015). However, Fagard et al. (2014) found that high rates of JA-related defenses inhibited by the N-environment resulted in higher numbers of bacterial pathogen cells. ABA may induce stomatal closure to avoid pathogenic bacterial infection but may also harm plant immunity as a signal molecule (Berens et al., 2017). N deficiency can increase the synthesis of ABA whereas NO_3^- decreases levels of ABA compared with NH_4^+ (Garnica et al., 2010). Huang et al. (2017) reported that high N fertilization not only encourages the production of protection genes such as PR and those involved in chemical defense biosynthesis but also induces multiple negative regulators of defense during rice-*Magnaporthe oryzae* relationships. The research also revealed the essential function of the OsNAP gene in plant immunity, the expression of which was decreased with high N. A similar finding has been noted in the relationship between *Arabidopsis* and *Erwinia amylovora*, with the up-regulation of multiple resistance genes by N-starvation and pathogen challenge, in particular PR1, WRYK33 and WRYK60, well-known positive plant defense regulators (Farjad et al., 2018). It may reflect the survival of plant protection programs under N-starvation stress, while high N cannot draw the opposite conclusion. Accumulating evidence suggests that gaseous radical NO is a crucial signaling factor for resistance determination and can regulate the expression of different genes involved in the responses to plant pathogen resistance (Vitor et al., 2013).

Key defense-related products like phytoalexins and lignin in phenylpropanoid pathway are regulated by NO (Ahuja et al., 2012). Moreover, Salicylic acid (SA) is a crucial aspect of the acquired local and systemic resistance associated with the accumulation of pathogenesis-related proteins (PRs) and is another component of the phenylpropanoid pathway (Durrant and Dong, 2004). The role of NO as a trigger of hypersensitive cell death and as a regulator of defense gene expression was well-clear in host–pathogen interaction between *Arabidopsis thaliana* and *Pseudomonas syringae* (Chen et al., 2014). Mur et al. (2005) suggested that NO influenced the resistance with two bacterial pathogens, *Pseudomonas syringae* pv. *phaseolicola* and *Pseudomonas syringae* pv. *tabaci* in tobacco. Most of the study indicate that NR (Nitrate Reductase) pathway is the main source of NO during plant-pathogen interactions (Gupta et al., 2013). NO_3^- nutrition will increase NO generation through NR activity, while NO signals are inhibited under NH_4^+ (Modolo et al., 2005). Growth on NO_3^- nutrition leads to increased levels of NO, SA, PR gene expression, induction of the polyamine pathway, a decrease in apoplastic sugars and amino acids, and an overall increase in plant resistance while NH4+ acts as vice versa reducing plant defense responses (Mur et al., 2017).

5   Conclusions

Nitrogen fertilizer doses or forms have a complicated relationship between plant disease severity. There is not any obvious basic model or mechanism to explore the linkage between N uptake, metabolism, and disease infection process. Numerous evidences are found in various literature about the impact of N fertilizer on crop disease incidence during the green revolution, but also different disease severity is reported when low N fertilizers are applied. The understanding of the underlying mechanism can be a great importance for agricultural practices. The first line of plant defense like cell wall, barks, waxy epidermal cuticle, lignin contents as well as preformed chemical substances are greatly influenced by the N contents. Many antimicrobial chemicals, enzymes, proteins, and secondary metabolites also acts for the plant defense mechanisms. Finally, many genes, proteins, and plant hormones like SA, JA, and ET are also involved in regulating many signaling pathways for plant defense. In all these cases, N has a great influence on the underlying mechanisms. Nonetheless, the fundamental molecular pathways are not fully known so there are still many questions to be addressed. A better understanding of the relationship between N and the severity of plant disease and the responses to plant defense is critical in designing successful strategies for disease and pest resistance.

Additional studies should discuss both system biology and natural variation mechanisms to highlight the role of plant metabolites in plant-pathogen interaction as well as the plant nutrition effect on plant defense and pathogen virulence. Further study should also address the effect of forms of N fertilization on physical, biochemical as well as molecular defense when suffering from disease. To find the regulatory pathway between N and HR at local site and SAR at distal site, transcriptomics approaches should be studied broadly. Continued advancement in the production of disease-resistant crop plants involves more knowledge of how N, NO, and polyamines relate to PTI and ETI, along with explicating the pathways involved in transferring N capital away from the infection site.

Abbreviations

N: Nitrogen; NH_4^+: Ammonium ion; NO_3^-: Nitrate ion; NIS: Nitrogen-induced susceptibility; NUE: Nitrogen use efficiency; DI: Disease incidence; DSI: Disease severity index; SA: Salicylic acid; JA; Jasmonic acid; ET: Ethylene; ABA: Abscisic acid; ROS: Reactive oxygen species; PR: Pathogenesis related; PAMP: Pathogens associated molecular patterns; PRRs: Pattern recognition receptors; PTI: PAMP-triggered immunity; ETS: Effector-triggered susceptibility; BAK1: Brassinosteroid insensitive 1-associated kinase 1; LRR: leucine-rich repeat; CERK1: Chitin elicitor receptor kinase 1; NB-LRR: Nucleotide binding- leucine-rich repeat; HR: Hypersensitive response; RNS: Reactive nitrogen species; MAPK: Mitogen-activated protein kinase; NO: Nitrous oxide; NADPH: Nicotinamide adenine dinucleotide phosphate hydrogen; RBOH: Respiratory burst oxidase homologs; SAR: Systemic acquired resistance; ATL31: Arabidopsis Toxicos en Levadura31; C/N: Carbon: Nitrogen; SYP121: Syntaxin of Plants121; POD: Peroxidase; PPO: Polyphenol oxidase; PAL: Phenylalanine ammonia lyase; GA: Gibberellic acid; CK: Cytokinin; BR: Brassinosteroids; NPR1: Ankyrin repeat protein; ERF1: Ethylene Response Factor1; PDF 1.2; Plant defensin 1.2; OPR3: Oxophytodienoate reductase 3.

Conflict of Interest

The author declares that there is no conflict of interests regarding the publication of this paper.

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