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INTERACTIONS BETWEEN TOMATO PLANTS AND PATHOGENIC FUNGI: PTI AND ETI MECHANISMS (REVIEW)

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ABSTRACT----------
Tomato (Solanum lycopersicum L.) is among the most economically important vegetable crops worldwide, yet its productivity and fruit quality are severely constrained by fungal pathogens employing biotrophic, necrotrophic, or hemibiotrophic lifestyles. Major pathogens-including Oidium neolycopersici, Alternaria solani, Botrytis cinerea, Fusarium oxysporum f. sp. lycopersici (FOL), Verticillium dahliae, and Phytophthora infestans-reshape host physiology through distinct infection strategies. This review synthesizes current knowledge on the early immune responses of tomato to fungal invasion, focusing on pattern-triggered immunity (PTI) initiated by pattern-recognition receptors (PRRs) and subsequent signaling via reactive oxygen species (ROS), Ca^{2+} transients, and MAPK cascades, as well as on effector-triggered immunity (ETI) mediated by intracellular R proteins. We compare host physiological outcomes across pathogen lifestyles, highlighting SA-dominant signaling against biotrophs versus JA/ET-dominant responses against necrotrophs, and the two-phased immune dynamics typical for hemibiotrophs. We further discuss effectors that suppress PTI and the circumstances under which hypersensitive-like cell death may paradoxically benefit necrotrophs (effector-triggered susceptibility). Finally, we outline integrative disease management directions-breeding for durable resistance, immune priming, biological control, and informed fungicide rotation-framed by a mechanistic understanding of PTI-ETI crosstalk in tomato. This mechanistic perspective provides a conceptual basis for developing resistant cultivars and robust integrated disease management strategies.

KEYWORDS: *Solanum Lycopersicum, Fungal Pathogens, Biotroph, Necrotroph, Hemibiotroph, PTI, ETI, PRR, ROS, Ca^{2+} Signaling, MAPK.----------*

INTRODUCTION

Tomato suffers from a broad spectrum of fungal diseases that cause substantial yield and quality losses globally. In the field, biotrophic powdery mildew (*Oidium neolycopersici*), necrotrophic early blight and gray mold (primarily *Alternaria solani* and *Botrytis cinerea*), and hemibiotrophic wilts and late blight (FOL, *Verticillium dahliae*, and *Phytophthora infestans*) are particularly damaging (Panno et al., 2021; Srinivas et al., 2019; Mazumdar et al., 2021). Plant–pathogen interactions proceed through rapid physiological and biochemical reprogramming in the host: pathogen perception (PAMP/MAMP recognition by PRRs), oxidative bursts, calcium influx, and activation of mitogen-activated protein kinase (MAPK) cascades culminate in defense gene expression and the accumulation of pathogenesis-related (PR) proteins, chitinases, β-1,3-glucanases, and antioxidant enzymes (Jones & Dangl, 2006; Bigeard et al., 2015; Meng & Zhang, 2013). Understanding how tomato tailors these responses to pathogen lifestyle is essential for durable resistance breeding and for integrated control strategies.

LIFESTYLES OF KEY TOMATO FUNGAL PATHOGENS AND THEIR PHYSIOLOGICAL IMPACT.

Biotrophs. Biotrophic fungi colonize living tissues and often deploy apoplastic and cytoplasmic effectors to attenuate PTI and avoid recognition, enabling prolonged symptom-poor phases (Bai et al., 2005; Mapuranga et al., 2022). In tomato powdery mildew, *O. neolycopersici* forms superficial mycelia and haustoria, gradually depressing photosynthesis, altering stomatal dynamics, and perturbing gas exchange while eliciting constrained ROS bursts and SA-biased signaling (Bai et al., 2005; Panno et al., 2021).

Necrotrophs. Necrotrophs thrive on dead tissue and actively induce host cell death via toxins, cell wall-degrading enzymes (CWDEs), and ROS, generating rapidly expanding necrotic lesions that sharply reduce photosynthesis

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and exacerbate water loss (Singh et al., 2023; Panno et al., 2021). Defense is typically coordinated through jasmonic acid (JA) and ethylene (ET) pathways; strong hypersensitive responses (HR) can be counterproductive, facilitating effector-triggered susceptibility (ETS) (Mapuranga et al., 2022; Pandey et al., 2023).

Hemibiotrophs. Hemibiotrophs initiate infections with a stealth biotrophic phase before transitioning to necrotrophy. FOL spreads through xylem vessels, causing vascular browning, reduced water potential, and wilt despite irrigation; occlusion of conduits is common (Srinivas et al., 2019). V. dahliae induces similar but often slower symptoms, frequently at cooler temperatures (Panno et al., 2021). P. infestans penetrates via appressoria, grows biotrophically, then triggers rapid tissue collapse and severe photosynthetic decline during necrotrophy (Mazumdar et al., 2021; Srinivas et al., 2021).

Physiological contrasts. Against biotrophs, SA-centered signaling predominates; ROS bursts are typically tempered to avoid self-damage. Necrotroph challenges bias toward JA/ET signaling, where excessive ROS promotes lesion expansion. Hemibiotroph infections exhibit biphasic immunity: a moderate early response followed by intensified ROS production, callose deposition, and wall fortification during the necrotrophic shift (Dodds & Rathjen, 2010; Pandey et al., 2023).

EARLY PATTERN-TRIGGERED IMMUNITY (PTI) IN TOMATO. PAMP recognition by PRRs. Tomato membranes harbor PRRs that detect chitin oligomers, β-glucans, lipopolysaccharides, and other conserved patterns. CERK1/LYK-family chitin receptors initiate defense signaling upon binding fungal chitin fragments (Couto & Zipfel, 2016; Saijo & Loo, 2020). PRR activation launches ROS production, MAPK cascades, Ca^{2+} influx, and broad transcriptional reprogramming (Jones & Dangl, 2006; Bigeard et al., 2015).

ROS burst and redox control. Plasma-membrane NADPH oxidases (RBOHs) drive the oxidative burst, generating O_2•– and H_2O_2 that restrict pathogens, strengthen cell walls, and function as secondary messengers (Torres, 2010). Antioxidant systems-superoxide dismutase, peroxidases, and catalase-maintain redox homeostasis and mitigate photo-oxidative damage (Sharma et al., 2012).

Ca^{2+} signaling. Rapid Ca^{2+} influx from the apoplast and intracellular stores activates Ca^{2+}-dependent protein kinases (CDPKs) and calmodulin-dependent kinases that feed forward to ROS generation and defense gene expression in tomato (Ranty et al., 2016; Romeis & Herde, 2014).

MAPK cascades and transcriptional control. Tomato MAPKs (e.g., SlMPK1/3/6 and upstream MAPKKKs) are phosphorylated upon PAMP perception, activating WRKY and ERF transcription factors and promoting PR proteins, phytoalexins, and antioxidant enzymes (Melech-Bonfil & Sessa, 2010; Meng & Zhang, 2013).

Cell wall fortification and antimicrobial metabolites. Callose and lignin deposition, together with phenylpropanoid-derived phenolics, reinforce the cell wall, constraining pathogen ingress (Underwood, 2012). Tomato phytoalexins, including tomatine and related saponins, contribute to antifungal activity (Iriti & Faoro, 2009).

Effector-Triggered Immunity (ETI), Crosstalk, and Susceptibility. Fungal (and oomycete) effectors suppress PTI at multiple nodes; when recognized by intracellular NLR-type R proteins, robust ETI ensues, often accompanied by localized cell death and heightened defense (Jones & Dangl, 2006; Bigeard et al., 2015). In P. infestans, RXLR and CRN effectors target nuclear and apoplastic hubs to repress defense gene expression, especially during early biotrophy (Fan et al., 2021). Notably, for necrotrophs, HR-like death can enhance colonization (ETS), demanding careful breeding strategies that avoid “death-promoting” specificities (Mapuranga et al., 2022; Afzal et al., 2025).

SA–JA/ET crosstalk. Lifestyle-contingent hormone crosstalk shapes outcomes: SA pathways typically antagonize JA/ET, favoring anti-biotroph defense, whereas JA/ET promote anti-necrotroph resistance (Couto & Zipfel, 2016; Pandey et al., 2023). Hemibiotrophs exploit this crosstalk to time phase transitions.

Implications for Disease Management. Recurring epidemics of early blight, powdery mildew, gray mold, and vascular wilts threaten tomato in temperate continental climates; late blight can devastate crops within days (Panno et al., 2021; Afzal et al., 2021). Mechanistic insights suggest several complementary avenues:

Breeding for durable resistance: Stack broad-spectrum PRR-based traits with well-chosen R genes that avoid ETS risks against necrotrophs (Bai et al., 2005; Jones & Dangl, 2006).

Immune priming and biological control: PGPR and beneficial endophytes can prime PTI/ETI and modulate metabolism, lowering FOL and Phytophthora severity (Ansari et al., 2025; Pereira et al., 2025).

Fungicide stewardship: Rotate modes of action in line with FRAC guidance; match chemistry to lifestyle (e.g., anti-necrotroph actives during lesion expansion) while integrating canopy microclimate management.

Physiology-aware agronomy: Practices that maintain photosynthetic efficiency and water balance can reduce susceptibility windows during hemibiotrophic phase shifts.

CONCLUSIONS

Tomato-fungus interactions are governed by fast, multilayered immunity. PTI begins with PRR detection of PAMPs and proceeds through ROS bursts, Ca^{2+} transients, and MAPK signaling to deploy structural and chemical defenses. ETI strengthens this response when effectors are recognized, but necrotrophs may exploit host cell death, leading to effector-triggered susceptibility. A lifestyle-aware understanding of SA/JA/ET crosstalk and PTI-ETI integration provides a rational framework for breeding and integrated disease management in tomato.

REFERENCES

1. *Afzal K. et al. Effectors of plants pathogenic fungi and fungal like microbes: a comprehensive review on mechanisms, roles, and host interactions //Frontiers in Plant Science. – 2025. – T. 16. – C. 1626960.*
2. *Ansari W. A. et al. Relevance of plant growth-promoting bacteria in reducing the severity of tomato wilt caused by Fusarium oxysporum f. sp. lycopersici by altering metabolites and related genes //Frontiers in Microbiology. – 2025. – T. 15. – C. 1534761.*
3. *Bai Y. et al. Tomato defense to Oldium neolycopersici: dominant OI genes confer isolate-dependent resistance via a different mechanism than recessive oI-2 //Molecular Plant-Microbe Interactions. – 2005. – T. 18. – №. 4. – C. 354-362.*
4. *Bigeard J., Colcombet J., Hirt H. Signaling mechanisms in pattern-triggered immunity (PTI) //Molecular plant. – 2015. – T. 8. – №. 4. – C. 521-539.*
5. *Couto D., Zipfel C. Regulation of pattern recognition receptor signalling in plants //Nature Reviews Immunology. – 2016. – T. 16. – №. 9. – C. 537-552.*
6. *Dodds P. N., Rathjen J. P. Plant immunity: towards an integrated view of plant–pathogen interactions //Nature Reviews Genetics. – 2010. – T. 11. – №. 8. – C. 539-548.*
7. *Fan K. T. et al. Quantitative proteomics reveals the dynamic regulation of the tomato proteome in response to Phytophthora infestans //International Journal of Molecular Sciences. – 2021. – T. 22. – №. 8. – C. 4174.*
8. *Iriti M., Faoro F. Chemical diversity and defence metabolism: how plants cope with pathogens and ozone pollution //International journal of molecular sciences. – 2009. – T. 10. – №. 8. – C. 3371-3399.*
9. *Jones J. D. G., Dangl J. L. The plant immune system //nature. – 2006. – T. 444. – №. 7117. – C. 323-329.*
10. *Mapuranga J. et al. Infection strategies and pathogenicity of biotrophic plant fungal pathogens //Frontiers in Microbiology. – 2022. – T. 13. – C. 799396.*
11. *Mazumdar P. et al. Late blight in tomato: insights into the pathogenesis of the aggressive pathogen Phytophthora infestans and future research priorities //Planta. – 2021. – T. 253. – №. 6. – C. 119.*
12. *Melech-Bonfil S., Sessa G. Tomato MAPKKKε is a positive regulator of cell-death signaling networks associated with plant immunity //The Plant Journal. – 2010. – T. 64. – №. 3. – C. 379-391.*
13. *Meng X., Zhang S. MAPK cascades in plant disease resistance signaling //Annual review of phytopathology. – 2013. – T. 51. – №. 1. – C. 245-266.*
14. *Morales Bello J. New insights into the regulation of NADPH oxidase dependent reactive oxygen species signaling during the plant immune response: ∂uc. – Agronomos, 2015.*
15. *Pandey A. K. et al. Insight into tomato plant immunity to necrotrophic fungi //Current Research in Biotechnology. – 2023. – T. 6. – C. 100144.*
16. *Panno S. et al. A review of the most common and economically important diseases that undermine the cultivation of tomato crop in the mediterranean basin //Agronomy. – 2021. – T. 11. – №. 11. – C. 2188.*
17. *Pereira E. C. et al. Diaporthe atlantica improves tomato resistance against the vascular pathogen Fusarium oxysporum f. sp. lycopersici //Biological Control. – 2025. – C. 105852.*
18. *Ranty, B., et al. (2016). Calmodulin and calcium signaling in plant immunity. Plant Cell.*
19. *Romeis T., Herde M. From local to global: CDPKs in systemic defense signaling upon microbial and herbivore attack //Current opinion in plant biology. – 2014. – T. 20. – C. 1-10.*
20. *Saijo Y., Loo E. P. Plant immunity in signal integration between biotic and abiotic stress responses //New Phytologist. – 2020. – T. 225. – №. 1. – C. 87-104.*
21. *Sharma P. et al. Reactive oxygen species, oxidative damage, and antioxidative defense mechanism in plants under stressful conditions //Journal of botany. – 2012. – T. 2012. – №. 1. – C. 217037.*
22. *Singh D. P. et al. Metabolomics of early blight (Alternaria solani) susceptible tomato (Solanum lycopersicum) unfolds key biomarker metabolites and involved metabolic pathways //Scientific reports. – 2023. – T. 13. – №. 1. – C. 21023.*

Volume: 13 | Issue: 9 | September 2025 | Journal DOI: 10.36713/epra0813| Impact Factor SJIF(2025): 8.733| ISSN: 2321 – 7847

23. *Srinivas C. et al. Fusarium oxysporum f. sp. lycopersici causal agent of vascular wilt disease of tomato: Biology to diversity–A review //Saudi journal of biological sciences. – 2019. – T. 26. – №. 7. – C. 1315-1324.*
24. *Torres M. A. ROS in biotic interactions //Physiologia plantarum. – 2010. – T. 138. – №. 4. – C. 414-429.*
25. *Underwood W. The plant cell wall: a dynamic barrier against pathogen invasion //Frontiers in plant science. – 2012. – T. 3. – C. 85.*