



WASHINGTON STATE
UNIVERSITY

WSU PLANT PATHOLOGY SEMINAR
September 8, 2025, 4:10 PM



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Plant Pathogens Manipulate Host mRNA Alternative Splicing

ABOUT THE PRESENTER

Nosheen Fatima is a Ph.D. student in the Department of Plant Pathology at Washington State University, advised by Dr. Xianming Chen. Her research focuses on the genetics of disease resistance in wheat, particularly stripe rust, with an emphasis on QTL mapping and marker development in wheat. Beyond her research, she has been actively involved in student leadership, previously serving as a Graduate and Professional Student Association Senator and currently contributing to the Plant Pathology Graduate Student Organization, where she actively contributes to student leadership and community engagement.

Attend in Person

9/8/25 @ 4:10 pm (Pacific)

Clark 149, Pullman, WA

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ABSTRACT

Eukaryotes use alternative splicing (AS) as a conserved post-transcriptional regulatory mechanism to generate multiple messenger RNA (mRNA) isoforms from one gene, which increases transcriptome complexity and proteome variability (1,2). In plants, AS also plays a significant role in plant immunity, but various plant pathogens actively manipulate the process to evade host defenses (3). Gaining an in-depth understanding of these plant pathogen interactions at the molecular level will facilitate the development of sustainable resistance. In this seminar, insights from three different host-pathogen systems demonstrating the complex nature of this interaction will be discussed. One of the prominent examples of pathogen-mediated AS is the maize-sugarcane mosaic virus (SCMV) pathosystem. The mRNA surveillance pathway, comprising nonsense-mediated mRNA decay, regulates transcriptomic homeostasis in maize and SCMV disrupts this pathway to compromise plant immunity. This disruption is caused by SCMV nuclear inclusion protease (NIa-Pro), directly targeting the conserved splicing component ZmU2AF65B and restricting its normal binding to ZmUPF3 pre-mRNA (4). Ultimately, this affects the correct splicing of ZmUPF3, impairing RNA surveillance and allowing the proliferation of viral particles. Similarly, fungal pathogens manipulate AS to alter the signaling networks in plants, as seen in the case of rice blast. In the rice-*Magnaporthe oryzae* pathosystem, *Pi21* gene in rice negatively regulates resistance. Comparative AS profiling between the *Pi21*-mutated line and the susceptible cultivar Nipponbare revealed significant isoform changes. The fungal infection causes bidirectional shifts of the rice plant splice site usage in defense transcription factors, ubiquitin-dependent endoplasmic reticulum stress regulators, and protein-protein interaction networks (5). Such AS modifications affect hormone signaling pathways (jasmonic acid and ethylene) and stress accommodation responses, consequently tuning the growth immunity balance to promote pathogen survival. In bacterial pathosystems, AS acts as a defense regulator that operates at specific stages. The roots of Arabidopsis plants infected by *Ralstonia solanacearum* showed significant changes in splicing patterns prior to the transcriptional changes. The differentially spliced genes (DSGs) showed minimal overlap with differentially expressed genes, which demonstrated that AS operates as an independent regulatory mechanism. The DSGs accumulated in post-transcriptional and translational processes, but NLR receptors and receptor-like kinases, which function as main immune regulators, showed splicing changes that indicate AS shapes immune receptor diversity. Collectively, these examples show the splicing apparatus of hosts serves as a crucial regulatory network in plant-pathogen interactions, which pathogens use to disable immune responses. Significant progress has been made to identify pathogen effectors that target splicing factors and infection-induced isoform changes, yet many aspects remain unclear. The combination of large-scale effector-host interactome studies with advanced epigenome profiling and single-cell RNA sequencing methods will reveal the intricate mechanisms of pathogen-controlled AS regulation (1). These approaches will not only uncover pathogen mechanisms for host spliceosome control but also establish novel molecular targets for the development of broad-spectrum resistance. In conclusion, combining molecular knowledge of host-pathogen interactions with crop improvement strategies can support resilient agroecosystems to withstand the ongoing challenges posed by evolving plant pathogens.

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ADDITIONAL SOURCES

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