Molecular basis of the rice blast fungus *Magnaporthe oryzae* utilizing autophagy and cell wall integrity signaling to govern pathogenicity

With the financial support provided by the National Natural Science Foundation of China, the research team led by Prof. Zhang ZhengGuang (张正光) at the Key Laboratory of Integrated Pest Management on Crops of Education, Department of Plant Pathology, College of Plant Protection, Nanjing Agricultural University, went a step further into examining how the rice blast fungus *Magnaporthe oryzae* integrates autophagy and cell wall integrity signaling pathways to govern pathogenicity. The result was recently published in *Autophagy* (2019, 24: 1—17).

Blast caused by the filamentous ascomycete *M. oryzae* has been the most destructive disease of rice during the widespread crop cultivation, resulting in significant yield losses of varying between 10% and 30%, which poses serious threats to the global food supply security. With the availability of genome sequences and advanced research tools for classical and molecular genetics, *M. oryzae* is rapidly becoming a model organism in the research field of phytopathology, especially for the study of the plant-pathogen interaction.

As a hemibiotrophic fungus, *M. oryzae* undergoes an initial biotrophic stage before switching to a necrotrophic stage that promotes plant cell death. During the biotrophic stage of infection, the fungus synthesizes and secretes a repertoire of effector proteins to suppress plant immunity and to manipulate host cell physiology for the next stages of infection. Most of the secreted and transmembrane proteins are synthesized, folded, and matured in the endoplasmic reticulum (ER). However, as much as a third of newly synthesized proteins fail to achieve native structures correctly owing to imperfections in transcription, translation, and post-translational modifications or protein folding. Thus, an inevitable consequence of the high rate of protein synthesis is the accumulation of misfolded proteins in the ER. To counteract this, cells activate the unfolded protein response (UPR) pathway to degrade these misfolded proteins. At the same time, autophagy, a highly conserved catabolic process in the eukaryotic cells, is responsible for vacuolar (lysosomal) degradation of proteins, membranes, and organelles according to functional and energetic needs upon the environmental stress or pathogen invasion. Prof. Zhang's group has found that ER stress could activate cell wall integrity (CWI) signaling independent of the upstream kinase MoMck1. Considering that ER stress is also associated with autophagy, they investigated and identified a likely link between autophagy and CWI signaling.

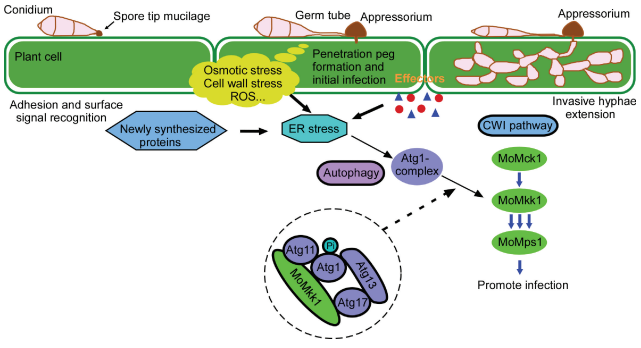

Figure Model of *M. oryzae* utilizing MpAtg1-dependent MoMkk1 phosphorylation to stimulate the CWI signaling and enhance its infection on rice.

Interestingly, they found that, during *M. oryzae* infection, the accumulation of ER stress could stimulate MoAtg1, a core autophagy-related protein, to directly phosphorylate MoMkk1, an essential kinase downstream of MoMck1. This MoAtg1-dependent MoMkk1 phosphorylation could further enhance the CWI signaling pathway to govern the pathogenicity of *M. oryzae* on rice.