Virulence strategies of fungal biotrophs: lessons from the Ustilago maydis - maize model system

The outcome of plant-microbe interactions is determined by the interplay of the microbial virulence repertoire with the plant immune system. The plant pathogenic fungus *Ustilago maydis* establishes a biotrophic interaction and causes smut disease in its host plant maize. While formation of prominent plant tumors is a major hallmark of this disease, for many years it was not understood how the pathogen orchestrates the transformation of different cell types into tumor tissue. Combination of cytology and transcriptomics identified the cellular basis of tumor formation and showed that secreted effector proteins act in cell-type specific manner to trigger tumor development. Important hubs for the coordination of plant defence are papain-like cysteine proteases (PLCPs). In maize, these enzymes are crucial for the activation of salicylic acid (SA)-dependent defenses. Consequently, activity of PLCPs is modulated by both host factors, as well as fungal effector proteins. Recently, we discovered a peptide, *Zea mays* immune signalling peptide 1 (Zip1), which is released by SA-activated PLCPs and, in turn, itself activates SA-mediated defence resulting in maize resistance to biotrophic infection. The fungal effector Pit2 blocks those maize PLCPs, which are responsible for Zip1 release. This modulation of maize immunity depends on a conserved sequence motif within Pit2. Biochemical analyses revealed that Pit2 acts as substrate for the maize PLCPs, to release the inhibitory domain, which in turn blocks the plant PLCPs. We therefore propose that the Pit2 effector evolved as a molecular decoy to overcome PLCP-mediated plant defenses. Remarkably, the PID14 core motif is conserved in unrelated plant associated fungi and bacteria. The existence of a conserved microbial protease inhibitory motif to facilitate host colonization substantiates the importance of PLCPs as central hubs in the regulation of maize biotic interactions.