Secondary Metabolites of Fusarium graminearum

Toxic to microbes and effectors in virulence

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Secondary Metabolites of *Fusarium graminearum*: Toxic to microbes and effectors in virulence

Fusarium graminearum forms complex infection cushions (IC) to infect its host plant wheat. Secondary metabolite gene clusters are upregulated in IC compared to epiphytically growing hyphae. We evaluated the impact of deoxynivalenol (DON), butenolides (BUT), and aurofusarin (AUR) on the early infection process of this fungus. Aur-deficient mutants show wild type like infection. However, a wild type extract with AUR is able to inhibit bacterial and fungal growth while the Aur-deficient mutant cannot. Therefore AUR may suppress competitors prior to colonization of the host plant. Don-deficient mutants are known to infect the spikelet, but cannot cross the rachis node and fail to colonize it. We show that even the early infection phase following the formation of IC is affected. During the first days of infection, Don-deficient mutants infect slower and with less mycelium compared to the DON producing wild type. DON is an effector molecule in the early infection phase. But-deficient mutants exhibit wild type like virulence. We detected an even increased virulence during the early infection phase compared to the wild type and are currently further evaluating this phenotype. Don/But/Aur triple knock out mutants show a similar reduced virulence as the Don-deficient single mutant. The increased virulence of the But-deficient phenotype cannot compensate for the loss of DON and the reduced virulence.