



Secondary Metabolites of *Fusarium graminearum*

Toxic to microbes and effectors in virulence

Glasenapp, Anika; Malz, Sascha; Frandsen, Rasmus John Normand; Schäfer, Wilhelm

Published in:

Book of abstracts from the 13th European Conference on Fungal Genetics

Publication date:

2016

Document Version

Publisher's PDF, also known as Version of record

[Link back to DTU Orbit](#)

Citation (APA):

Glasenapp, A., Malz, S., Frandsen, R. J. N., & Schäfer, W. (2016). Secondary Metabolites of *Fusarium graminearum*: Toxic to microbes and effectors in virulence. In *Book of abstracts from the 13th European Conference on Fungal Genetics* (pp. 528-528). [CS7W1]

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

POSTER SESSION ABSTRACTS
Session CS7 Metabolism and physiology
CS7W1

Wednesday 6th April
14:00 - 16:00

GLASENAPP Anika (1), **MALZ Sascha** (1), **FRANDBSEN Rasmus J. N.** (2), **SCHÄFER Wilhelm** (1)

(1) University of Hamburg, Hamburg, GERMANY

(2) Technical University of Denmark, Lyngby, Denmark

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.
