

Self-protection against the sphingolipid biosynthesis inhibitor fumonisin B1 is conferred by a *FUM* cluster-encoded ceramide synthase.

Janevska S, Ferling I, Jojić K, Rautschek J, Hoefgen S, Proctor RH, Hillmann F, Valiante V (2020) Self-protection against the sphingolipid biosynthesis inhibitor fumonisin B1 is conferred by a *FUM* cluster-encoded ceramide synthase. *mBio* 11(3), e00455-20.

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Abstract

Fumonisin (FB) mycotoxins produced by species of the genus *Fusarium* detrimentally affect human and animal health upon consumption, due to the inhibition of ceramide synthase. In the present work, we set out to identify mechanisms of self-protection employed by the FB₁ producer *Fusarium verticillioides*. FB₁ biosynthesis was shown to be compartmentalized, and two cluster-encoded self-protection mechanisms were identified. First, the ATP-binding cassette transporter Fum19 acts as a repressor of the *FUM* gene cluster. Appropriately, *FUM19* deletion and overexpression increased and decreased, respectively, the levels of intracellular and secreted FB₁. Second, the cluster genes *FUM17* and *FUM18* were shown to be two of five ceramide synthase homologs in *Fusarium verticillioides*, grouping into the two clades CS-I and CS-II in a

phylogenetic analysis. The ability of *FUM18* to fully complement the yeast ceramide synthase null mutant *LAG1/LAC1* demonstrated its functionality, while coexpression of *FUM17* and *CER3* partially complemented, likely via heterodimer formation. Cell viability assays revealed that Fum18 contributes to the fungal self-protection against FB₁ and increases resistance by providing *FUM* cluster-encoded ceramide synthase activity.

Beteiligte Forschungseinheiten

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Identifier

doi: 10.1128/mBio.00455-20.

PMID: 32546615