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

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Details



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 FB_1 producer *Fusarium verticillioides*. FB_1 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 FB_1. 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 FB_1 and increases resistance by providing *FUM* cluster-encoded ceramide synthase activity.

Beteiligte Forschungseinheiten

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