The transcriptional regulators SteA and StuA contribute to keratin degradation and sexual reproduction of the dermatophyte *Arthroderma benhamiae*.

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Abstract

Most superficial fungal infections are caused by dermatophytes, a specialized group of filamentous fungi which exclusively infect keratinized host structures such as hair, skin and nails. Since little is known about the molecular basis of pathogenicity and sexual reproduction in dermatophytes, here we functionally addressed two central transcriptional regulators, SteA and StuA. In the zoophilic species *Arthroderma benhamiae* a strategy for targeted genetic manipulation was recently established, and moreover, the species is teleomorphic and thus allows performing assays based on mating. By comparative genome analysis homologs of the developmental regulators SteA and StuA were identified in *A. benhamiae*. Knock-out mutants of the corresponding genes as well as complemented strains were generated and phenotypically characterized. In contrast to *A. benhamiae* wild type and complemented strains, both mutants failed to produce sexual reproductive structures in mating experiments. Analysis of growth on keratin substrates indicated that loss of steA resulted in the inability of ΔsteA mutants to produce hair perforation organs, but

did not affect mycelia formation during growth on hair and nails. By contrast, Δ stuA mutants displayed a severe growth defect on these substrates, but were still able to produce hair perforations. Hence, formation of hair perforation organs and fungal growth on hair per se are differentially regulated processes. Our findings on the major role of SteA and StuA during sexual development and keratin degradation in *A. benhamiae* provide insights into their role in dermatophytes and further enhance our knowledge of basic biology and pathogenicity of these fungi.

Involved units

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