Histidine degradation via an aminotransferase increases the nutritional flexibility of *Candida glabrat*a.

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Abstract

The ability to acquire nutrients during infections is an important attribute in microbial pathogenesis. Amino acids are a valuable source of nitrogen if they can be degraded by the infecting organism. In this work, we analyzed histidine utilization in the fungal pathogen of humans *Candida glabrata.* Hemiascomycete fungi, like *C. glabrata* or Saccharomyces cerevisiae, possess no gene coding for a histidine ammonia-lyase, which catalyzes the first step of a major histidine degradation pathway in most other organisms. We show that *C. glabrata* instead initializes histidine degradation via the aromatic amino acid aminotransferase Aro8. Although ARO8 is also present in *S. cerevisiae* and is induced by extracellular histidine, the yeast cannot use histidine as its sole nitrogen source, possibly due to growth inhibition by a downstream degradation product. Furthermore, *C. glabrata* relies only on Aro8 for phenylalanine and tryptophan utilization, since ARO8, but not its homologue ARO9, was transcriptionally activated in the presence of these amino acids. Accordingly, an ARO9 deletion had no effect on growth with aromatic amino acids. In contrast, in S. cerevisiae, ARO9 is strongly induced by tryptophan and is known to support growth on aromatic amino acids. Differences in the genomic structure of the ARO9 gene between *C. glabrata* and *S. cerevisiae* indicate a possible disruption in the regulatory upstream region. Thus, we show that, in contrast to *S. cerevisiae*, *C. glabrata* has adapted to use histidine as a sole source of nitrogen and that the aromatic amino acid aminotransferase Aro8, but not Aro9, is the enzyme required for this process.

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