Identification of the novel penicillin biosynthesis gene aatB of Aspergillus nidulans and its putative evolutionary relationship to this fungal secondary metabolism gene cluster.


Abstract

The final step of penicillin biosynthesis in the filamentous fungus Aspergillus nidulans is catalysed by isopenicillin N acyltransferase encoded by the aatA gene. Because there is no bacterial homologue, its evolutionary origin remained obscure. As shown here, disruption of aatA still enabled penicillin production. Genome mining led to the discovery of the aatB gene (AN6775.3) which has a similar structure and expression pattern as aatA. Disruption of aatB resulted in a reduced penicillin titre. Surface plasmon resonance analysis and Northern blot analysis indicated that the promoters of both aatA and aatB are bound and regulated by the same transcription factors AnCF and AnBH1f. In contrast to aatA, aatB does not encode a peroxisomal targeting signal (PTS1). Overexpression of a mutated aatB(PTS1) gene in an aatA-disruption strain (leading to peroxisomal localization of AatB) increased the penicillin titre more than overexpression of the wild-type aatB. Homologues of aatA are exclusively part of the penicillin biosynthesis gene cluster, whereas aatB homologues also exist in non-producing fungi. Our findings suggest that aatB is a paralogue of aatA. They extend the model of evolution of the penicillin biosynthesis gene cluster by recruitment of a biosynthesis gene and its cis-regulatory sites upon gene duplication.

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