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Biosynthetic regulation of echinocandin B: From pathway specific to environmental cues responsive regulation

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Echinocandin B is a well-known potent antifungal which is considered to be the front-line antifungal against the treatment of candida infections due to the rare emergence of resistance. It is a cyclic hexapeptide synthesized by the two ecd and hty gene clusters of *Emericella rugulosa* NRRL 11440. It acts on the fungal cell wall by blocking the 1,3 β- glucan synthase activity. The present work is targeted to elucidate the regulation of echinocandin B biosynthesis. For this, we have deleted the ecdB transcription factor encoded gene, located in the ecd gene cluster by homologous recombination. This deletion of ecdB in *Emericella rugulosa* NRRL 11440 was successfully made and completely arrogate the ecdB expression. The ecdB deletion did not significantly affect the echinocandin B production and found to be similar to the wild type. Furthermore, the expressions of other genes of the ecd and hty cluster were also not significantly altered in the knockout background. We also focused to explore the role of pH and nitrogenous sources on echinocandin B production. Unlike Nitrate which has repressive function, arginine remarkably increased the echinocandin B production by 10 folds as compared to the nitrate. Remarkably production of echinocandin B was induced suitably at acidic pH (range 4.5- 6.6), highest production was observed at 6.6 pH which is two folds higher than 4.5 pH. Taken together our results indicate that in-clustered transcription factor ecdB may have no direct role in the regulation of echinocandin B biosynthesis while environmental cues, nitrogen and pH-responsive global regulatory factors are involved in the regulation of Echinocandin B biosynthesis in *Emericella rugulosa* NRRL 11440.

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