LuxF: a potential rescue factor in bacterial bioluminescence?

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Abstract

In bioluminescent bacteria the heterodimeric enzyme luciferase catalyzes the monooxygenation of aliphatic aldehydes to the corresponding acids utilizing FMN as redox cofactor. The proteins involved in bacterial bioluminescence are encoded by the *lux* operon. A number of bioluminescent *Photobacteria* were identified with an additional *lux* gene, termed *luxF*. LuxF is a homodimeric protein that binds the unusual flavin derivative 6-(3'-(R)-myristyl)-FMN (myrFMN).

It was postulated that myrFMN is a potential inhibitor of the luciferase. We hypothesized that LuxF is the putative scavenger of myrFMN, due to a higher affinity to LuxF than to the luciferase, thereby preventing inhibition. Replication of the bioluminescent reaction *in vitro* was achieved by applying an enzyme cascade with a cofactor recycling system. In multiple turnover reactions, exhibiting light for more than 48 hours, the formation of myrFMN was confirmed via HPLC analysis. In vivo analysis of a range of bioluminescent bacteria (luxF+ and luxF) revealed that myrFMN formation is independent of luxF occurrence. However, there seems to be a correlation between light intensity, myrFMN formation and luxF occurrence. Therefore, we are further investigating the role of LuxF via heterologous expression of luxF and luxF+ operons within an *E.coli* model system that results in the production of light. These "enlightened *E.coli* cells" will be compared to other bioluminescent bacterial strains to elucidate the influence of luxF on light intensity.

In conclusion, these findings suggest that LuxF not only plays a role in preventing inhibition but also influences the catalytic activity of the bacterial luciferase and thereby light production.

Keywords: bacterial bioluminescence, luciferase, myrFMN, luxF+ strains

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