YbcM, a transcriptional regulator from prophage origin involved in Escherichia coli physiology

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Résumé

When integrated into the bacterial genome, temperate phages are called prophages. When expressed, genes carried by these prophages can confer advantages to their host such as increased virulence, fitness or stress resistance. However, only few studies have been undertaken to elucidate the molecular mechanisms leading to these changes in bacterial physiology.
In this work, we investigate the role of YbcM, a transcriptional regulator encoded by the DLP12 prophage in *E. coli*. By combining RNA-Seq and ChIp-Seq approaches, we identified 48 genes whose expression varies more than 4-fold when YbcM is overproduced. Our data show that YbcM directly represses 17 of them, which is odd for a regulator from the AraC/XylS family. A large number of these genes are involved in motility, adherence and biofilm formation. As a consequence, YbcM overproduction inhibits motility and promotes a particular type of biofilm called macrocolony biofilm. Interestingly, YbcM overproduction also decreases the sensitivity of different *E. coli* strains to several phages, suggesting that this surface-dependent biofilm can have a negative impact on the interaction between phages and their host.

We have also demonstrated that YbcM directly represses the expression of *crp* that encodes the central regulator of the catabolic repression in *E. coli*. This regulatory pathway is still being characterized and may have important consequences on the host physiology, particularly under glucose starvation since CRP favors the use of carbon sources other than glucose.
Overall, our results show that YbcM is integrated into the bacterial regulatory network and changes in depth the bacterial physiology. An important consequence of these changes is the immunity conferred by YbcM against potential future phage infections. This work will provide a better understanding of how prophages can genetically interact with their bacterial hosts and deeply affect their physiology.

Mots-Clés: prophage, phage resistance, biofilm, motility, metabolism

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