

HspR Mutations Are Naturally Selected in *Bifidobacterium longum* When Successive Heat Shock Treatments Are Applied[▽]

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The development of molecular tools allowed light to be shed on several widespread genetic mechanisms aiming at limiting the effect of molecular damage on bacterial survival. For some bacterial taxa, there are limited tools in the genetic toolbox, which restricts the possibilities to investigate the molecular basis of their stress response. In that case, an alternative strategy is to study genetic variants of a strain under stress conditions. The comparative study of the genetic determinants responsible for their phenotypes, e.g., an improved tolerance to stress, offers precious clues on the molecular mechanisms effective in this bacterial taxon. We applied this approach and isolated two heat shock-tolerant strains derived from *Bifidobacterium longum* NCC2705. A global analysis of their transcriptomes revealed that the *dnaK* operon and the *clpB* gene were overexpressed in both heat shock-tolerant strains. We sequenced the *hspR* gene coding for the negative regulator of *dnaK* and *clpB* and found point mutations affecting protein domains likely responsible for the binding of the regulators to the promoter DNA. Complementation of the mutant strains by the wild-type regulator *hspR* restored its heat sensitivity and thus demonstrated that these mutations were responsible for the observed heat tolerance phenotype.

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