The expression of small heat shock protein AlIbpA from mycoplasma Acholeplasma laidlawii in E. coli cells promotes the formation of amyloid structures

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The main function of small heat shock proteins (sHSPs) is the prevention of proteins aggregation in cell. Besides, sHSPs have been shown to be involved in many other processes including the biofilm formation. Thus, in E. coli, two sHSPs IbpA and IbpB indirectly influence biofilm formation. When absent, cells are subjected to endogenous oxidative stress and consequent overproduction of indole, that in turn inhibits formation of the biofilm.

Here we show that the overproduction of sHSP AlIbpA from phytopathogenic mycoplasma Acholeplasma laidlawii restores at high temperatures the biofilm formation and increases the amyloid structures content in E. coli cells lacking their own IbpB.

The full-length AlIbpA and proteins with deletions of putative functional terminal motifs (AlIbpAΔN12, AlIbpAΔC14, AlIbpAΔN12C14) were overexpressed in Escherichia coli wild-type and strains with deletions of own sHSPs (ΔEcIbpA or ΔEcIbpB). The crystal violet staining of the biofilms revealed that the biofilm formation was restored in ΔEcIbpB cells producing the full-length AlIbpA. Furthermore, the Thioflavin S and Congo red staining of the biofilms revealed that the removal of one of sHSP in E. coli, with the simultaneous expression of AlIbpA, leads to increased formation of amyloid structures. Increased amyloids were also observed during overexpression of AlIbpAN12 in the E. coli ΔEcIbpA and AlIbpAC14 by cells of the E. coli ΔEcIbpB, respectively. Amyloids in the matrix were also detected during overexpression of AlIbpA with double deletion (AlIbpAΔN12C14) in both knockout E. coli strains.

Thus, the sHSPs can be involved in biofilm formation also via amyloids synthesis, however, the molecular mechanism of regulation requires further study.

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