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The role of AmtB, GlnK and glutamine synthetase in regulation of of transcription factor TnrA in Bacillus subtilis

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Abstract

Nitrogen is a macroelement for all living cells, from bacteria to animals. Although ammonia ions (NH3/NH4 +) are toxic to animals, they are the most preferable nitrogen source for the majority of bacteria and are assimilated by glutamine synthetase (GS) in the so-called GOGAT cycle. A lack of nitrogen for a cell triggers cascade regulatory processes and activation of a large group of genes for utilization of nitrogen from other compounds. Thus, in Bacillus subtilis, genes of nitrogen metabolism are regulated by the transcription factor TnrA. In the cell, it is bound to AmtB-GlnK proteins, with interaction with glutamine synthetase repressing its DNA-binding activity. Deletion of the protein AmtB responsible for ATP-dependent transport of ammonium ions into the cell from the medium has been shown to lead to a lack of nitrogen in the cell and, as a result, to an increased level of expression of TnrA-regulon genes. With a deficit of protein GlnK, the factor TnrA is constitutively associated with GS, with its activity also decreasing under conditions of deficit of nitrogen source. The factor TnrA activity in cells seems to be constantly repressed by GS: in the absence of GS, the TnrA activity is significantly increased as compared with control, even under conditions of nitrogen starvation, in which GS is highly active. These facts allow it to be suggested that the factor TnrA activity is regulated by competitive binding to GS and protein GlnK. © 2013 Pleiades Publishing, Ltd.

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Keywords

Bacillus subtilis, glutamine synthetase, nitrogen metabolism, transcription factor TnrA