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Glucose Repression of *sdhCDAB* Expression Is Mediated by cAMP–CRP Complex in *Escherichia coli*

Tae-Wook Nam*, Young-Ha Park and Yeong-Jae Seok

Institute of Microbiology, College of Natural Sciences, Seoul National University

The *Escherichia coli sdhCDAB* operon encodes succinate dehydrogenase complex involved in tricarboxilic acid (TCA) cycle. The transcription of this operon is regulated in response to various growth conditions, such as anaerobiosis and carbon sources. ArcA, the response regulator of ArcA/B two component system, represses the transcription of *sdh* operon in anaerobic condition. The expression of *sdh* operon is also repressed in the presence of glucose, but it has been reported that the general catabolite regulators, CRP, FruR and Mlc, may not be directly involved in this glucose repression. Recently, new experimental evidences have been presented that glucose repression of *sdhCDAB* transcription involves a mechanism dependent upon the *ptsG* expression, but the molecular mechanism underlying the glucose repression is still unknown yet. In this work, we show that CRP directly regulates the expression of *sdh* operon and the glucose repression of this operon also occurs in cAMP-dependent manner. Disruption of *crp* or *cya* completely abolished the glucose repression of *sdhC-lacZ* expression. The levels of phosphorylated $EIIA^{Glc}$ on various sugar substrates and intracellular cAMP are proportional to the β-galactosidase activities of *sdh-lacZ* expression. Growth on glucose did not lead to a remakable decrease in *sdhC-lacZ* expression in cells lacking *crr* expression compared to wild type cells, and glucose repression of *sdhC-lacZ* expression was completely disappeared in *ptsG* deletion mutant cells. Comparison of the levels of intracellular cAMP and CRP between mutants and wild type suggests that the decrease in cAMP-CRP level on glucose is the major determinant of glucose repression of *sdh* operon.