Regulation of fructooligosaccharide metabolism in an extra-intestinal pathogenic Escherichia coli strain

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cluster expression is under the control of an epigenetic switch depending on methylation: Fur binding prevents methylation of a GATC motif, whereas methylation at this specific site decreases the affinity of Fur for its binding box. A model is proposed in which the sci1 promoter is regulated by iron availability, adenine methylation, and DNA replication.

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A gene cluster involved in the metabolism of prebiotic short-chain fructooligosaccharides (scFOS) has recently been identified in the extra-intestinal avian pathogenic *Escherichia coli* strain BEN2908. This gene cluster, called the *fos* locus, plays a major role in the initiation stage of chicken intestinal colonization. This locus is composed of six genes organized as an operon encoding a sugar transporter and enzymes involved in scFOS metabolism, and of a divergently transcribed gene encoding a transcriptional regulator, FosR, belonging to the LacI/GalR family. To decipher the regulation of scFOS metabolism, we monitored the *fos* operon promoter activity using a luciferase reporter gene assay. We demonstrated that the expression of *fos* genes is repressed by FosR, controlled by catabolite repression and induced in the presence of scFOS. Using electrophoretic mobility shift assays and surface plasmon resonance experiments, we showed that FosR binds to two operator sequences of the *fos* operon promoter region. This binding to DNA was inhibited in the presence of scFOS, especially by GF2. We then propose a model of scFOS metabolism regulation in a pathogenic bacterium, which will help to identify the environmental conditions required for *fos* gene expression and to understand the role of this locus in intestinal colonization.

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YO02/34 The regulation of the Cad system in *Escherichia coli*

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On their way from the stomach to the gut enterobacteria are exposed to substantial changes in the pH. As the stomach can reach a pH value as low as 1.0, neutrophil bacteria had to evolve strategies to survive this extreme stress condition. One acid response system is the lysine dependent Cad system. It consists of the enzyme CadA which catalyses the decarboxylation of lysine to cadaverine while consuming a cytoplasmic proton and releasing CO_2. This results in the increase of the internal pH. The lysine/cadaverine antiporter CadB, the membrane-integrated protein CadC and the lysine transporter LysP are other components of the system. Under non-inducing conditions, LysP represses CadC, whereas at low pH, anaerobiosis and in presence of lysine CadC functions as transcriptional activator of the *cadBA* operon. In a large scale transposon mutagenesis we identified the lysine 2,3-aminomutase YjeK to play a role in the regulation of the Cad system. A *yjeK* deletion mutant is unable to express the *cadBA* genes. It was previously shown that YjeK is involved in the posttranslational modification of proteins. Therefore, this study investigates whether the transcriptional regulator CadC is directly modified or additional proteins are involved in the regulatory network of the Cad system.