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CRYSTAL STRUCTURE OF THE CYTOPLASMIC DOMAIN OF THE SERINE TRANSMEMBRANE RECEPTOR FROM *E. COLI*

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All bacterial chemotaxis receptors are dimeric and have highly conserved cytoplasmic domains that transmit signals from different periplasmic domains to a common signalling pathway. The crystal structure of the cytoplasmic domin of Tsr, the serine chemotaxis receptor of *E. coli* at 2.6 angstrom resolution, reveals that the domain is a 20 nm long coiled coil of two antiparallel helices connected by a U-turn, and two such domains form a supercoiled four helical bundle. Futhermore, three dimers join together at the signaling regions to form a trimer of the dimers. Combined with the information from the crystal structure of the ligand-binding domain of another chemotaxis receptor, a model of an intact dimeric chemotaxis receptor emerges as a 40 nm long molecule of mostly supercoiled four-helical bundles supplemented by four additional short helices at the ligand-binding domains.

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STRUCTURE, FUNCTION, AND REGULATION OF THE E. COLI GLUCOSE TRANSPORTER

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Mutations arose from an *Escherichia coli* strain defective in the high (Rbs/ribose)- and low (Als/allose and Xyl/xylose)-affinity D-ribose transporters, which allow cells to grow on D-ribose. Genetic tagging and mapping of the mutations revealed that two loci in the *E.coli* linkage map are involved in creating a novel ribose transport mechanism. One mutation was found in *ptsG*, the glucose-specific transporter of PTS system, and the other in *mlc* recently reported to be involved in the regulation of *ptsG*. Five different mutations in *ptsG* were characterized, whose growth on D-ribose medium were about 80% of that of the high-affinity system (Rbs^+). Two of them were found in the predicted periplasmic loops while three others are in the transmembrane region. Ribose uptakes in the mutants, competitively inhibited by D-glucose, D-xylose, or D-allose, were much lower than that of the high-affinity transporter, but higher than those of the Als and Xyl systems. Further analyses of the mutants revealed that the *rbsK* (ribokinase) and *rbsD* (function unknown) genes are involved in the ribose transport through PtsG, indicating that the phosphorylation of ribose is not mediated by PtsG and that some unknown metabolic function mediated by RbsD is required. It was also found that D-xylose, another sugar not involving phosphorylation, was efficiently transported through the wild-type or mutant PtsG in *mlc*-negative background. The efficiencies of xylose and glucose transports are variable in the PtsG mutants, depending on their locations, either in the periplasm or in the membrane. In an extreme case of the transmembrane change (I283T), xylose transport is virtually abolished, indicating that the residue is directly involved in determining sugar specificity. Taken together, it was proposed that there are at least two domains for substrate specificity in PtsG with slightly altered recognition properties.