Abstract

Carbon source is one of the basic requirements for bacterial growth. Dgalactonate, an aldonic sugar acid, can be used as a carbon source by Escherichia coli, a common gram-negative bacterium. Galactosemic patients who cannot utilize galactose in their diet excrete large amounts of D-galactonate in urine. It has been shown by microarray studies that E. coli grown in urine up regulates the expression of enzymes involved in D-galactonate metabolism thereby suggesting that galactosemic patients might be more prone to infections by bacteria that have the ability to utilize Dgalactonate. Classical mutagenesis and mapping studies performed in 1970's identified the genes involved in D- galactonate metabolism; however, the phenotypes of strains carrying clean deletion in these dgo genes were not studied. The D-galactonate metabolic pathway includes a transporter, a dehydratase, a kinase and an aldolase, and the genes encoding these components are organized in a putative D-galactonate operon (dgo). The first gene of the operon, dgoR, encodes a putative transcriptional regulator. Till date, the regulation of D-galactonate metabolism has not been studied. In the present work, we have used various dgo gene deletion strains to show that dgo genes are involved in Dgalactonate metabolism. We also provide evidence that DgoR is a negative regulator of dgo operon. We observed that deleting dgoR shortens the lag phase of bacteria when Dgalactonate is used as the carbon source; hence it acts as a repressor of dgo genes. Western blotting experiments to detect the expression of 3X-FLAG tagged DgoR from chromosome revealed the induction of DgoR by D-galactonate. During the course of exploring the regulation of dgo genes, we identified a second transcriptional regulator of D-galactonate metabolism, ArcA. In future, we will carry out detailed characterization of the regulatory role of DgoR and ArcA in D-galactonate metabolism.