

A *soxS* Depended *pgi* Responsible for Activation to Paraquat Independently of *soxR* in *Escherichia coli*

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The promoter for gene *pgi* of *E. coli* induced to redox-cycling compounds such as paraquat (PQ), ethyl viologen (EV) and benzyl viologen (BV). The *soxS* dependent PQ-inducibility of promoter *pgi* was examined by increasing relative bioluminescence with increase in PQ or EV concentration in both wild type and *soxR* mutant *E. coli* carrying *pgi::luxCDABE* gene fusion, respectively. Mutant cells lacking *soxR* but carrying *pgi::luxCDABE* gene fusion showed similar response to PQ or EV as that of wild-type. However, *soxRS* mutant harboring *pgi::luxCDABE* failed to induce by either PQ or EV, suggesting that the *pgi* strongly responds to superoxides and is mediated by only *soxS* but seemed not by *soxR*. Our experiments with wild type recombinant PGI/RFM443 strain carrying *pgi::luxCDABE* fusion showed strong response to PQ under minimal conditions in presence of gluconate than with glucose. Further, *pgi::luxCDABE* also induced significantly 17-folds against H₂O₂ and four to six-folds against DNA-damage. Here we demonstrate that *pgi* gets activated against chemicals causing cellular stress and plays a contributing role to general cellular-stress mainly to maintain metabolic equilibrium and carbon flow.