Sequence of the *Salmonella typhimurium* LT2 *lexA* gene and its regulatory region

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Submitted January 13, 1992 EMBL accession no. X63562, GenBank accession no. M83220

The SOS regulon of *Escherichia coli* is composed of over 20 genes that are expressed in response to DNA damage (1). LexA protein acts as a repressor of these genes under normal growth conditions. The SOS system is induced when DNA damage activates RecA protein to a form which mediates self cleavage of LexA (2). Cleaved LexA protein no longer functions as a repressor.

A functional assay was used to identify plasmids which carry the *S.typhimurium lexA* gene. *E.coli* strain ATT575, which has two genetic constructs sensitive to LexA (3), was transformed with a plasmid library prepared by complete digestion of *S.typhimurium* DNA with *Eco*RI. The success of the cloning strategy proved that *S.typhimurium* LexA protein represses *E.coli sulA* and *recA* promoter-operators *in vivo*.

E.coli has two tandem consensus LexA binding sites 5′ to the lexA gene which are used for autoregulation of LexA protein levels (4). *S.typhimurium* also has two such binding sites (in boxes below), but the downstream site is not a consensus site. The consensus binding site for LexA protein in *E.coli* is TACTGTATATATATACAGTA, and the CTG and CAG are highly conserved in other known SOS genes and have been shown to be the most important for LexA binding by genetic analysis (5). The downstream *S.typhimurium* binding site differs by containing a TTG instead of the conserved CTG.

Previous studies in *E.coli* have implicated the amino terminus as being involved in DNA binding. Although LexA protein structure is different from the typical helix-turn-helix motif, two-dimensional NMR analysis of the amino terminus revealed three α-helices comprised of amino acids Gln8 to Ser2O, Arg28 to Leu35, and Asn41 to Gly54 (6). There is also a hydrophobic pocket formed by Ala29, Ala42, and Ala43 into which Phe37 may fit (6). None of the amino acid differences in *S.typhimurium* are in the helices, or the hydrophobic pocket. Mutations effecting DNA binding in *E.coli lexA* have been characterized (3, 7), and none of the changes in the *S.typhimurium lexA* match these known mutations.

The carboxy-terminus of *E.coli* LexA contains the amino acid residues necessary for the cleavage reaction, interaction with RecA protein, (2) and dimerization (8). Activated RecA mediates the cleavage of *E.coli* LexA between Ala84 and Gly85, and as expected, this region is conserved in *S.typhimurium* LexA. Many mutants of *E.coli lexA* that affect cleavage and dimerization have been isolated (8, 9, 10, 11) however, none of the amino acid changes in *S.tymphimurium* correspond to these mutants.

The fact that *S.typhimurium* LexA functions in *E.coli* to repress the *recA* and sulA operators, that *E.coli* LexA represses DNA damage inducible loci in *S.typhimurium* (12), and that the amino acids important for DNA binding are conserved, suggests that *S.typhimurium* LexA recognizes the same DNA binding sites as *E.coli* LexA. The *S.tymphimurium lexA* sequence is accessible through the EMBL and GenBank database and is also available on request.

ACKNOWLEDGMENTS

We are indebted to Paul Sharp for his analysis of codon usage and sequence divergence of the *S.typhimurium* and *E.coli lexA* genes. We also wish to thank L. Kevin Lewis for many helpful discussions and John Levy for technical assistance. This work was supported in part by NIH grant GM24496.

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