

The nucleotide sequence of *greA*, a suppressor gene that restores growth of an *Escherichia coli* RNA polymerase mutant at high temperature

Jason Sparkowski and Asis Das*

Department of Microbiology, University of Connecticut Health Center, Farmington, CT 06030, USA

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We have recently reported the isolation of a suppressor RNA polymerase mutant of *Escherichia coli* that is susceptible to modification to a termination-resistant form by the phage lambda *N* gene product utilizing a mutant host factor, *nusA1* (1). The mutation, *rpoB_{ts8}*, resulting from a serine to phenylalanine substitution at residue 522 of the beta subunit, also renders the cell temperature sensitive for growth (1). The proficiency of the mutant RNA polymerase in general transcription activities and gene expression at high temperature indicates that the F522 substitution has caused a loss of some specialized function of RNA polymerase, perhaps the interaction with a transcription factor vital for cell growth (1). This hypothesis predicts that it may be possible to suppress the defect of the polymerase mutant by independent genetic events restoring the wild type interaction or bypassing the requirement for that interaction. Accordingly, we have tested whether the cell growth at high temperature can be restored by an *E. coli* gene cloned in a multi-copy plasmid. Here, we report the entire DNA sequence of a NcoI-BamHI fragment encoding one such suppressor gene, we name *greA* for growth regulator.

The sequence reveals a putative promoter approximately 150 bp upstream of the ATG initiator and a Shine-Dalgarno sequence 8 bp upstream. A putative attenuator that might be involved in regulation of *greA* transcription is found within the leader region. The predicted protein, consisting of 158 amino acids (MW 17,630), contains two putative helix-turn-helix motifs (2) corresponding to residues 48–67 and 124–133. The GreA protein contains regions of similarity to portions of the following proteins which may be of interest with regard to its function: i) 75% identity in residues 61–68 to the C-terminal helix-turn-helix motif of the sigma 70 subunit of *E. coli* RNA polymerase, believed to interact with the –35 region of promoter sequences (3). ii) 46% identity in residues 58–70 to the *E. coli* protein AraC, responsible for regulation of the arabinose operon, though it is not in either of the two putative helix-turn-helix motifs described by Francklyn and Lee (4). iii) 32% identity in residues 129–156 with the *E. coli* (2), 50S ribosomal protein L3 that binds to the 3' half of the 23S RNA (5). iv) 46% identity in residues 53–65 with the human transforming protein jun (6). v) 24% identity in residues 10–42 with the cdc10 DNA synthesis start control protein of *Schizosaccharomyces pombe* (7).

We have detected the GreA protein expressed from the multi-copy plasmid. The potential role of the homology regions in the suppressor function of GreA is being investigated.

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* To whom correspondence should be addressed