
Characterization of a gene that regulates toxin A synthesis in *Pseudomonas aeruginosa*

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The plasmid pFHK10 has been shown to contain a 3 kb XhoI fragment from PA103 chromosomal DNA which increases exotoxin A production (1). Subcloning and complementation analysis indicated that the positive regulatory gene (regA) resided on a 1.9 kb PstI-XhoI fragment (2) which was further characterized by DNA sequence analysis (3) (figure 1). Deletion derivatives identified a single open reading frame responsible for RegA synthesis (2). Composite proteins produced with the pT7-7 expression system (4) revealed a close correlation between the observed and predicted molecular weight which is 27,755. A comparison of the sequence presented here and previously published sequence data for the same gene (denoted toxR) (5) revealed several notable differences. Our sequence includes a C at position 6, an A at 784, a G at 785, and a C at 788 and excludes a T at 202 and a G at 778. The result of these differences is that the toxR sequence predicts a start codon 32 nucleotides upstream and a stop site 107 nucleotides short of the predicted regA start and stop, giving a protein with a predicted molecular weight of 24,626.

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GTACCCTCGG CGGCCGATTG CCGAGCCGAT CTCCTACCTG CCCCCTGGGT TTTCCGACGA
AAGACCTTGA TTCGTGGGAG GTAGGGTCGT CTCCGCTAGA TACCCCTCAA CCCTGCGTGC
GGGCTCCATG CCGGAGCGCC TTGGCGAGAT TTGCCCATAG AGCCATCACT TATGACTGCG
ACAGACAGAA CGCCCCGCC ATGAAATGGC TCTGCCTCGG CAACCGTGAT GCGAACGACG
GATTCGAGCT CTTCGCCCAT GGCATCTATG CGAGGAACGG CGCGTTGGTC GGCAGCAAGC
TCTCCCTGCG CGAACGGCGC CAGCGCGTGC ACCTGTCGGC CTTCCTTTC GCGCACCGC
CGCTGCTTGC TGAGGCGGCG GTC AAGCACC TGCTGGCGCG CCTCCTGTGC GTGCACCGC
ACAACACCGA CCTCGAACTG CTCGGCAAGA ACTTCATTCC CCTGCATGCC AGCAGCCTGG
GCAACGCCGG GGTCTGCGAG CGGATCCTGG CCTCGGCCAG GCAATTGCAG CAGCACCAGG
TCGAACTCTG CCTGCTGCTG GCCATCGACG AGCAGGAACC CGCCTCGGG GAGTACCTGG
CGTCCCTTGC CCGGCTACGC GACAGCGGCG TGCGCATCGC GCTGCACCCG CAACGCATCG
ATACCGACGC TCGCCAGTGC TTCGCCGAGG TCGACGCCGG CCTCTGCGAT TACCTGGGCC
TGGACGCGCG CCTGCTTGCC CCCGCCCGC TGACCGGTAA CCTGCGCCAG CGCAAGAGCA
TCGAGTACCT GAACCGGCTG CTGGTGGCAC AGGACATCCA GATGCTTTC CTCAACGTCG
ACAATGAGGA ACTGCACCAA CAAGCCAACG CACTCCCTT CGCCTCCGT CACGGCAGGC
ACTATTCGGA GCCTTTCAG GCCTGGCCGT TCAGCAGTCC GGCCTGCTGA ACGCCGATGC
  
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References 1. Hedstrom, R., et al. (1986) *Infect. Immun.* **51**, 37-42. 2. Hindahl, M., et al. (1987) *Antibiot. Chemother.* **39**, 279-289. 3. Sanger, F., et al. (1977) *Proc. Natl. Acad. Sci.* **74**, 5463-5467. 4. Tabor, S., et al., (1985) *Proc. Natl. Acad. Sci.* **82**, 1074-1078. 5. Wozniak, F., et al. (1987) *Nucl. Acids Res.* **15**, 3123-2135.