Microbiology Comment provides a platform for readers of Microbiology to communicate their personal observations and opinions in a more informal way than through the submission of papers.

Most of us feel, from time to time, that other authors have not acknowledged the work of our own or other groups or have omitted to interpret important aspects of their own data. Perhaps we have observations that, although not sufficient to merit a full paper, add a further dimension to one published by others. In other instances we may have a useful piece of methodology that we would like to share.

The Editors hope that readers will take full advantage of this section and use it to raise matters that hitherto have been confined to a limited audience.

Jon Saunders, Editor-in-Chief

Presence of the Cpx system in bacteria

The CpxA/R two-component signal transduction system is involved in the sensing of (via CpxA) and adaptation to (via CpxR in phosphorylated form) envelope protein distress in Escherichia coli (1, 9). The Cpx system has also been implicated in host-cell invasion and virulence. In uropathogenic E. coli, the Cpx pathway seems critical for the expression and assembly of P-pili (4, 5). In Shigella sonnei, CpxR-P directly activates the synthesis of VirF, the master regulator of virulence expression (7, 8). In Salmonella typhi, a cpxA::TnphoA mutant proved unable to invade human small intestinal epithelium cells (6). Studies on the transcriptional regulation of cpxRA in non-pathogenic E. coli have yielded insights that may be relevant to related pathogens. CpxR-P autoactivates cpxRA transcription in concert with RpoS and an unknown activator at the onset of stationary-phase growth. In addition, Cpx signalling is feedback-inhibited by CpxP, a periplasmic protein which is under positive control of CpxR-P (2, 10).

Because of the importance of the Cpx pathway in stress adaptation and pathogenicity, we explored further the presence and distribution of cpxR, cpxA and cpxP in other prokaryotic genomes (http://wit.mcs.anl.gov/CGI; http://www.sanger.ac.uk/Projects/Microbes; http://www.tigr.org/tdb/CMR/) using the E. coli gene and protein sequences as references (default search settings). Among the genomes analysed (Archaeoglobus fulgidus; Aquifex aeolicus; Bacillus subtilis; Borrelia burgdorferi; Caulobacter crescentus; Campylobacter jejuni; Chlamydia trachomatis; Chlorobium tepidum; Clostridium acetobutylicum; C. difficile; Coxiella burnetii; Deinococcus radiodurans; Enterococcus faecalis; Haemophilus influenzae; Rd; Helicobacter pylori; Klebsiella pneumoniae; Methanobacteriumthermoautotrophicum; Methanococcus jannaschii; Mycobacterium avium, M. bovis, M. leprae, M. tuberculosis; Mycoplasma genitalium; Neisseria meningitidis; Porphyromonas gingivalis; Pseudomonas aeruginosa, P. putida; ‘Pyrococcus horikoshii’; Rhodobacter capsulatus, R. sphaeroides; Salmonella typhi; Shewanella putrefaciens; Staphylococcus aureus; Streptococcus pneumoniae; Streptomyces coelicolor; Vibrio cholerae; Yersinia pestis), cpxR, cpxA and cpxP were found only in Salmonella typhi and Yersinia pestis.

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The divergently transcribed \textit{cpxRA/cpxP} intergenic region of \textit{Y. pestis} shares only 55\% nucleotide sequence identity with that of \textit{E. coli}. Except for two extra sequence stretches (21 and 3 bp) in \textit{Y. pestis}, this intergenic region shows 63\% identity. In the \textit{Y. pestis} promoter region, one CpxR-P recognition box is found at the same distance from the CpxR start codon as in \textit{E. coli}, whereas the other box lies 21 bp further upstream of the start codon (Table 1). \textit{Y. pestis} CpxA, CpxR and CpxP are 82, 88 and 47\% identical, respectively, to those of \textit{E. coli}. However, no box is present in the promoter region of \textit{degP} homologue. Importantly, the \textit{cpxR} promoter region of \textit{H. influenzae} itself has no detectable similarity to that of \textit{E. coli}, \textit{S. typhi} or \textit{Y. pestis} and does not contain a CpxR-P box, indicating that the expression of the \textit{cpxR} homologue (HI0837) is regulated differently. It thus seems possible that \textit{H. influenzae} Rd strain KW20 has evolved to use the response regulator in a particular way. The absence of loci present in virulent \textit{H. influenzae} isolates, such as pathogenicity islands and genes involved in capsule and fimbriae formation (3), implies that CpxA is yet another missing virulence factor in strain KW20. The only known natural niche for \textit{H. influenzae} is within the human host, primarily in the upper respiratory tract. Therefore, this species may not require the ability to sense a transition from the free environment to the host milieu. If sensing that transition defines the role of CpxA/R, then \textit{Haemophilus} would not require the typical CpxA/R pathway.

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