" ! ... Comment ... ? "

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 new section and use it to raise matters that hitherto have been confined to a limited audience.

Jon Saunders
Editor-in-Chief

***Haemophilus* virulence**

I noted with interest that the published nucleotide sequence of the *Haemophilus influenzae* Rd genome (1) lacks equivalents of the *Escherichia coli* *sbcC* and *sbcD* genes (no similarities were found by searching the translated genome with either gene). All other known recombination genes from *E. coli* are represented. Presumably efficient streamlining of the genome omitted *sbcCD* since they are unnecessary for normal cell growth and recombination. However, their absence may in fact confer an evolutionary advantage. Several of the genes associated with virulence in *H. influenzae* carry tandem tetramer repeats that are subject to deletion or addition by replication slippage (1,2). These events allow variation of cell surface antigens important for infection. The products of *sbcC* and *sbcD* genes are critical for the replication of palindromic sequences in *E. coli*. They are thought to control replication of secondary structures by eliminating slipped bases (3). Although the tetrameric repeat sequences (5'-CAAT-3') seen in these *Haemophilus* species genes are not obviously palindromic, their repetitive composition is likely to stimulate replication slippage. The AT-richness of the repeat may also be a factor (2). That recombination is unnecessary

for this phenomenon is in keeping with the nature of the process. Lack of SbcCD proteins in *H. influenzae* may therefore serve to promote antigenic phase variation, enhancing its ability to survive in a particular host.

The absence of *sbvCD* in *Haemophilus* may be compensated by the natural transformability of the organism. Random deletion/insertion episodes as a result of errors in replication could potentially disrupt essential genes. DNA released from adjacent cells can be acquired and transferred to the chromosome by homologous recombination to mediate repair.

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2. Roche, R. J. & Moxon, E. R. (1995). Phenotypic variation of carbohydrate surface antigens and the pathogenesis of *Haemophilus influenzae* infections. *Trends Microbiol* **3**, 304–309.
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Identifying bacterial genes – a cautionary tale

An ORF capable of encoding a 296 residue protein (predicted molecular mass 33 kDa) has been detected at around 1275 kb in the complete genome sequence of *Haemophilus influenzae* (3). This ORF has been named *sulA*, and inferred to encode a cell division inhibitor, on the basis of similarity between its predicted product and that of an ORF named *sulA* in a sequence from *Vibrio cholerae*. However, neither of these putative proteins exhibit any significant similarity to the *sulA* (cell division inhibitor) gene products previously characterized from other members of the γ subdivision of the Proteobacteria. In fact, the *H. influenzae* sequence exhibits most similarity to an alternative ORF within the *V. cholerae* sequence, and is also similar to a previously unrecognized probable gene downstream of the histidine transport operon in *Salmonella typhimurium*. These genes are referred to as *orfX* below.

The *V. cholerae* sequence has a curious

history. Three overlapping sequences from the same laboratory (GenBank/EMBL accession numbers X56017, X56018 and X61383) are present in the DNA sequence databases. Two of these sequences are almost identical. Database entry X61383 is 841 bp long and lies completely within entry X56018 (899 bp), covering nucleotides 3–842. X61383 differs by the deletion of one of three A nucleotides at sites 175–177 (in X56018) and the addition of two C nucleotides to two already present at sites 801–802 (in X56018). Identical ORFs (at sites 177–794 and 174–791 in X56018 and X61383, respectively) are alternatively annotated as encoding the *recP* (X56018) and *sulA* (X61383) genes. Database entry X56017 (submitted simultaneously with X56018) is 542 bp long, from the complementary strand downstream of and substantially overlapping X56018: nucleotides 542–219 of X56017 correspond to nucleotides 576–899 of X56018, except for a transition at site 422 (696 in X56018). X56017 contains an ORF (named *pcdA*) at sites 23–400 reported to encode a 14 kDa protein conferring resistance to choleraeophage ϕ 149 (1). There is a 26 codon overlap between the 3' ends of *pcdA* and the putative *recP/sulA* gene.

The putative *rcp/slaA* gene seems unlikely to be functional because codons 24–65 correspond to a copy of the Enterobacterial Repetitive Intergenic Consensus (ERIC) sequence (4) or Intergenic Repeat Unit, IRU (6). As the names imply, this repetitive sequence has been found

► GUIDELINES

Communications should be in the form of letters and should be brief and to the point. A single small Table or Figure may be included, as may a limited number of references (cited in the text by numbers, and listed in alphabetical order at the end of the letter). A short title (fewer than 50 characters) should be provided.

Approval for publication rests with the Editor-in-Chief, who reserves the right to edit letters and/or to make a brief reply. Other interested persons may also be invited to reply. The Editors of **Microbiology** do not necessarily agree with the views expressed in **Microbiology Comment**.

Contributions should be addressed to the Editor-in-Chief via the Editorial Office.