Miles of isles

Pathogenicity islands are now known to be the culprits that supply virulence traits to a wide range of pathogens. Based on features of the genes within, or the sequences flanking, these chromosomal regions and their absence from non-pathogenic strains, all known pathogenicity islands are thought to have been gained through horizontal transfer. By applying these criteria, Covacci and co-workers have recently shown that the *cag* region of *Helicobacter pylori* – a locus expressed in clinical strains associated with gastritis, ulcers and adenocarcinomas – is part of a 40-kb pathogenicity island that has integrated into the glutamate racemase gene[1].

H. pylori is a Gram-negative microorganism that is harboured by the majority of readers of this journal; but, thankfully, clinical symptoms will develop in <10% of infected individuals. *H. pylori* strains recovered from humans have been grouped into two types (I and II) based on whether they express the CagA antigen and the VacA cytotoxin. The *vacA* gene is present in both type I and type II strains, whereas *cagA* is absent from type II strains, which are relatively benign. This suggests that the acquisition of the *cag* pathogenicity island may be responsible for the devastating effects of *Helicobacter* infections.

The disease symptoms promoted by type I strains of *H. pylori* are further underscored by a remarkable feature of the *cag* pathogenicity island; the degree of virulence appears to be modulated by the structure of the *cag* island itself, which, in certain strains, has been disrupted by the insertion of IS elements and the rearrangement of internal sequences. However, the structural heterogeneity in the *cag* island is not the only genetic difference among strains. For example, type I strains of *H. pylori* display a degree of genetic divergence in *vacA* sequences that is similar to that observed in comparisons of homologous genes from *Escherichia coli* and *Salmonella* spp. In the time required to accrue this level of sequence divergence, there could also be large numbers of additions or deletions to the genome. Therefore, additional experimentation is needed to identify, or discount, the contribution of genetic background to the phenotypic differences displayed by strains of *Helicobacter*. But given the intriguing association between the spectrum of alterations within the *cag* pathogenicity island and the observed variation in virulence properties, it would not be surprising if a simple causal relationship exists.

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Reference

1 Covacci, A. *et al.* (1997) *Trends Microbiol.* 5, 205–208

Horizons

Intercontinental promiscuity

The influence of inter- and intra-specific gene flux among bacteria on their population genetics is a current hot debate. It is important to have some measure of this phenomenon because, although the spread of antibiotic resistance genes is well documented for clinically important bacteria, relatively little is known about the effect of the potential dissemination of genetically engineered elements and the impact of environmental pollutants on the genetics of other microbial populations. These authors have evaluated this phenomenon in 'wild' bacteria by tracing the concurrence of the *mer* operon, which occurs in a wild bacterial plasmid (*Acinetobacter* pKLH2), a wild transposon (Tn*5041*) and a transposon found in clinical contexts (Tn*21*), which confers resistance to mercury in nonmedical bacteria. They have found *mer* operons in environmental Gram-negative bacteria worldwide, some of which have sequences virtually identical to the corresponding operons in clinical bacteria. They also noted dissemination of *mer* operon sequences by homologous recombination, resulting in mosaic *mer* operons.

Yurieva, O. *et al.* (1997) Intercontinental spread of promiscuous mercury-resistant transposons in environmental bacteria, *Mol. Microbiol.* 24, 321–329

Alternative segregation

There are insufficient microtubules in cells of the flagellated parasitic protozoan *Trypanosoma brucei* for the mitotic segregation of its complement of both large and minichromosomes. Minichromosomes act as a reserve for variant surface glycoprotein genes, which have to be transferred to the large chromosomes before they can become transcriptionally active. Early in mitosis, minichromosomes congregate in the centre of the nucleus. After the spindle is established, they divide into two clusters and move to the poles of the spindle. Late in mitosis, the spindle extends to the nuclear envelope to which the minichromosomes are attached. After disassembly of the spindle, the minichromosomes distribute close to the nuclear envelope. In contrast, the large chromosomes segregrate to the central segment of the spindle, consistent with the position of putative kinetochore structures. It is not clear whether segregation of the minichromosomes is faithful, but the inherited clusters of minichromosomes are the same size.

Ersfield, K. and Gull, K. (1997) Partitioning of large and minichromosomes in *Trypanosoma brucei*, *Science* 276, 611–614

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