Des Cooper from the Macquarie group have published a paper in *Genetics*, detailing a comprehensive linkage map of the tammar wallaby. This is the first for any marsupial. Most of 64 markers used were microsatellites or anonymous DNA markers, but importantly, 14 are known coding genes. The coding markers allow some of the linkage groups to be anchored to chromosomes, and potentially will allow the map to be aligned with those of other species.

A collection of 353 informative meioses, largely from male hybrid animals, linked all but four markers to at least one other, suggesting that most of the genome is included in the map. The 60 linked markers fell into nine linkage groups. Based on the smattering of physical assignments available, three of these could be assigned to chromosomes 1 and 3, and one to the X chromosome (Graves, 1995).

True, the map is still sketchy. However, it contains many features of immediate interest. Chief among these is the analysis of the sex differences between recombination rates.

Previous work on limited linkage data in the other two model marsupials, the dunnart and the Brazilian opossum, established that recombination in females was much lower than in males (Bennett *et al*, 1986; van Oorschot *et al*, 1992). This is just the opposite of eutherian mammals, in which recombination is much reduced in males. This exception strikes at the heart of Haldane's venerable hypothesis that recombination is lower in the heterogametic sex, perhaps due to reduced crossing over between differentiating sex chromosomes (Haldane, 1922). The tendency has been to ignore marsupials on the grounds that the data were too fragmentary.

The new and much more complete data from the tammar supports these earlier findings, and implies that the lower recombination in females is an ancient marsupial trait. The authors rightly insist that this reverse sex difference in recombination in marsupials can no longer be ignored when assessing theories of sex and recombination.

Once again, therefore, marsupials are caught breaking the genetic rules. This is what makes them particularly valuable subjects for genetic study. Perhaps this map will herald the start of a much-needed onslaught on the Kangaroo genome (Wakefield and Graves, 2002). ■

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Comparative Genomics

Islands of genetic novelty

JJ Ferretti and AN Suvorov

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Two new reports of bacterial genome sequences provide revealing insights into the evolution of virulence mechanisms in an important group of human pathogens. Interestingly, the new work also demonstrates there is substantial heterogeneity among the genomes of this group, even among those of the same serotype that provoke a similar immune response in humans.

Streptococcus agalactiae, also known as group B streptococcus (GBS), is responsible for invasive, and sometimes life-threatening, infections in humans, particularly newborn babies. Neonatal infections of these bacteria have decreased since 1996 after the Centers for Disease Control issued guidelines recommending antibiotic treatment before birth for babies at high risk (Centers for Disease Control, 2002). However, GBS still remains a leading cause of newborn sepsis and meningitis, as well as of severe invasive diseases in adults.

GBS is traditionally classified into serotypes according to the degree to which its polysaccharide capsule stimulates an immune response (ie its antigenicity). Nine serotypes have been identified to date.

Now complete genome sequences for representatives of two of these serotypes, III and V, have been reported in September issues of, respectively, *Molecular Microbiology* and *Proceedings of the National Academy of Science*. Philippe Glaser and his co-authors report the sequence of the circular chromosome of serotype III GBS, which is highly associated with early onset disease in the newborn (Glaser *et al*, 2002). Hervé Tettelin and colleagues have sequenced serotype V GBS, associated with severe invasive diseases of nonpregnant adults (Tettelin *et al*, 2002).

In the *PNAS* study, Tettelin and colleagues used DNA microarrays to do comparative genomic hybridization between the serotype V strain that was sequenced and 19 other strains of various serotypes. Interestingly, at least 18% of the GBS-specific genes were either absent or divergent from these 19 other strains, including those of the same serotype. It seems there is an enormous amount of genetic heterogeneity within these bacterial serotypes.

One of the most intriguing findings of the two reports was the discovery that most genes apparently unique to specific strains of the same serotype were clustered together in regions (islands). These islands consisted of 7 to 81 kb and encoded at least five contiguous genes. Of the 15 islands identified in the serotype V genome, 10 contained atypical nucleotide compositions differing from the 35.7% G + C content of the entire genome. One possibility is that these divergent islands correspond to horizontal gene transfer events.

The serotype III strain genome had 14 islands that contained the majority of known or putative GBS virulence factors. Importantly, all of these islands also contained sequences known to be associated with mobile genetic elements, eg insertion sequences, proteins of phages, plasmids and transposons. However, the role that these mobile genetic elements had played in the acquisition and spread of these islands remains uncertain because of substantial gene rearrangements among the islands. Such evidence of genetic deterioration may indicate that at least some of these elements may have been present in GBS for a long period of the organism's evolutionary history. Regardless, the strong association of genetic transfer elements and virulence factors in these chromosomal islands suggests that they may be pathogenicity islands and thus have an important role in virulence acquisition and genetic diversity.

Although a large number of phage and plasmid-related genes were found in the

chromosome of the serotype III strain, no complete temperate phage genomes were identified. Interestingly, three copies of a 47 kb sequence were present that had the characteristics of an integrative plasmid. These elements may also be important for horizontal gene transfer in GBS.

Tettelin and colleagues estimated that 650 proteins encoded by the serotype V genome were surface exposed and therefore were potential virulence factors. They then used a proteomic approach to identify which of these proteins was expressed.

Of 291 recombinant proteins made from this group and used to immunize mice, 139 sera were found to recognize a major GBS protein, 55 of which were shown by fluorescence-activated cell sorter analysis to be expressed on the cell surface. These data are important for understanding the physiology of GBS, as well as identifying new candidate antigens for vaccine development.

A remarkably large number of two-component regulatory systems were found in both new GBS genomes, compared with other bacteria in the Gram-positive group sequenced to date. The type III genome contained 20 histidine kinases and 21 response regulators and the type V genome had 17 of each. By comparison 14 are found in *S. pneumoniae*, 13 in *S. pyogenes*, and only eight in *Lactococcus lactis*.

This finding together with discovery of 107 transcriptional regulators suggests that the GBS have greater flexibility than the other Gram-positive cocci to react and survive in response to fluctuations in the external environment. Such versatility may explain why GBS are able to infect animals such as cattle in addition to humans.

Comparative genome analyses revealed that *S. agalactiae* is more closely related to *S. pyogenes* (group A streptococci, GAS) than *S. pneumoniae*. *S. agalactiae* also shares more orthologous genes with *S. pyogenes* than *S. pneumoniae*. *S. galactiae* or *S. pyogenes* also differ from *S. pneumoniae* in that they are not generally known for their ability to uptake exogenous DNA (transformation). In contrast, *S. pneumoniae* has a highly developed transformation system.

These new genomes allow important insights into the sequence heterogeneity found among and within GBS serotype strains. Future drug development and identification of candidate antigens for a universal vaccine will critically depend on these data. Ultimately these studies, and similar future ones, will lead to improved prevention and treatment of GBS diseases. ■

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Plant population genetics

What maintains male-sterility factors in plant populations?

D Charlesworth

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Most flowering plants are hermaphrodites whose flowers have both female parts (ovaries and a style and stigma) and male parts (anthers and stamens). However, 5–10% of species exhibit gynodioecy, ie females as well as hermaphrodites are present (Darwin, 1877). Sexually polymorphic species, such as these gynodioecious species, are particularly useful for studying the advantages and disadvantages of different reproductive modes. There have been many interesting studies of plant breeding systems, and those of gynodioecy have yielded a series of fascinating results. There has been an interplay between theoretical and empirical analyses of gynodioecy that has led to steady progress in understanding, though many puzzles remain. A major question is whether the genetic factors controlling the sexual polymorphism are maintained in gynodioecious species for long time periods or are lost, only to re-evolve again later. Mitochondrial DNA sequence variability data are starting to shed light on this question (Städler and Delph, 2002).

In most gynodioecious populations, females are in the minority, but female frequencies vary greatly and, surprisingly, sometimes exceed 50% (Olson and McCauley, 2002). The genetics of male sterility can account both for this variability and the high female frequencies. Male sterility is sometimes maternally inherited, and even a slight advantage to a maternally transmitted cytoplasmic male sterility (CMS) factor causing loss of male functions – for instance, some energy saving that could allow higher seed output – allows females to increase in the population until their seed output becomes limited by the pollen supply. CMS is a classic 'selfish' genetic element. However, such gynodioecious populations can be invaded by nuclear factors that restore male fertility of individuals with the sterility cytoplasm. Depending on the effects of the genetic factors, the restorer will either spread throughout the population, causing reversion to hermaphroditism, or the population may remain gynodioecious, with both sterility and fertility cytoplasms present, and both restorers and non-restorers. In theoretical models, such systems behave in complex ways. They can exhibit permanent cycles in the frequencies of the genetic factors (Gouyon *et al*, 1991), or may approach an equilibrium slowly, with large frequency fluctuations (Charlesworth, 1981).

In maize, where male sterility is used in hybrid breeding, there are three sterility cytoplasms, and several restorers. Many other crop plants have similar systems. In these, male sterility is due to mitochondrial genome rearrangements causing expression of chimaeric proteins, and the genes causing fertility restoration are now starting to be identified.

In natural populations, the genetics is probably similar to that in crops. Since gynodioecious plants often have several different sterility cytoplasms, each with several restorers, analysing these complex polymorphisms is very difficult. However, even without identified CMS genes, sequence variants anywhere in the mitochondrial genome could provide helpful genetic markers, allowing identification of plants' cytoplasmic genotypes, assuming strictly maternal inheritance, and no (or rare) recombination. Mitochondrial genome variants indeed exist in several gynodioecious plants, and distinct haplotypes coexisting within popu-