Editorial

The pattern of linkage disequilibrium (LD) in human populations was one of the most discussed subjects in the 54th Annual Meeting of the American Society of Human Genetics, which was held in October this year. Many of the conference participants had high hopes for the results of the International Hapmap Project, although the utility of the LD structure in delineating the aetiology of common diseases is yet to be demonstrated. Two problems were of particular interest: (1) What is the relationship between the shared history and the correlation of LD pattern between human populations? (2) What is the role of recombination in shaping the LD pattern in the human genome?

In this issue of *Human Genomics*, González-Neira *et al.* compare different patterns of LD in a chromosome 22 region between world populations. They show that haplotype structure has a distinct continental structure, with marked heterogeneity within Africans and Americans. In Eurasian populations, East Asians form a stronger cluster, while the pattern of LD in the populations in the west, including Europeans, turns out to be more complicated, with two major LD patterns along with some very specific outliers. In the fourth paper of this issue, de Silva *et al.* study the recombination rate behaviour in humans. In their paper they show that levels of recombination correlate with levels of nucleotide diversity. Approximately 12 per cent of genes carry distinct signatures of recombination hotpots, which are not always confined to introns but can stretch across exons.

As a proof of principle of genotyping pooled samples using a high-density oligonucleotide array, Hinds *et al.* conducted an association study of high-density lipoprotein cholesterol level by typing 7,283 single nucleotide polymorphisms, encompassing 71 genes.

In a further paper in this issue, Kennerly *et al.* compare the regional gene expression differences between the human brain and that of the domestic dog, and suggest the potential use of transcriptional profiling of the canine brain as a suitable animal model for creating a hypothesis to explain how the human brain behaves.

Also in this issue, Milos and Seymour examine emerging strategies and applications of pharmacogenomics.

In the Software Review section, the 'Statistical Analysis for Genetic Epidemiology' (SAGE) software package is reviewed and provides updated information to those who are studying the genetics of complex diseases.

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