

Editorial

The power of microarray technologies, when they were first introduced, lay in their ability to measure the relative copy numbers of thousands of genes within a single study; this made it possible to take snapshots of gene expression in cells under a variety of conditions, such as in tissues during development, in various tumour cells and in cells exposed to different environments and drugs. Microarray platforms have subsequently also been found to be powerful in other areas of genomics, including detecting subtle (sometimes involving only a one-fold difference) changes in the copy number of genomic fragments in tumour cells and conducting genotyping of thousands of single nucleotide polymorphisms (SNPs). In this issue of *Human Genomics*, three different applications of microarray technology are presented, which, coincidentally, all use Affymetrix platforms.

Gu and Gu investigate the difference in gene expression between humans and chimpanzees by comparing results based on several different statistics. The authors confirm an earlier discovery, that the pronounced changes in expression occurred in the human lineage after it differentiated from the chimpanzee and, more importantly, that the dramatic change in expression in the human brain is primarily driven by a set of genes with increased expression.

Shriver *et al.* study the genetic structure of human populations by typing 8,525 autosomal SNPs in four populations: African-American, European-American, Chinese and Japanese. Besides the observation on population-based clustering, they demonstrate strong correlations between inter-marker distance and both locus-specific population differentiation, measured by F_{ST} , and branch lengths. The non-uniform distribution of human genetic substructure is both instructive and a useful paradigm for education and research.

Huang *et al.* develop a novel algorithm that uses a high-density oligonucleotide array-based SNP genotyping method for detecting genome-wide chromosomal copy number changes at high resolution. They demonstrate that genomic regions with significant copy number changes can be detected using both single point analysis and contiguous point analysis. It is also shown that this approach is sensitive, specific and can tolerate mixed samples. By applying this method, several regions of amplification and deletion were detected and verified in a panel of human breast cancer cell lines.

Also in this issue of *Human Genomics*, Clark and Dean dissect the haplotype structure of two genomic regions: one containing four CC-chemokine receptor genes on chromosome 3p21 and the other containing three CC-chemokine genes on chromosome 17q11-12. They show that 14- and 6-tag SNPs respectively, are sufficient to encompass most of the haplotype variations in these two genes. The results of this study have strong implications for future association studies of HIV-1 disease and cancer.

The review by Gonzalez describes the approaches using humanised mouse models for the study of drug metabolism, pharmacokinetics and pharmacodynamics *in vivo* and for conducting human risk assessment towards xenobiotics.

Finally, in the Gene Annotation section, Nelson provides convincing examples to demonstrate the importance of improving pseudogene prediction.

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