Editorial

Population isolates used to be favourites for mapping genes underlying genetic susceptibility to diseases. Besides the reduced genetic heterogeneity and increased phenotypical homogeneity, their appeal to human geneticists rests upon the extended linkage disequilibrium (LD) in such populations, which facilitates localisation of disease genes using fewer markers even for a genome scan with a population-based design. The question is, what qualifies a population isolate? Furthermore, does a population isolate warrant an elevated LD across its genome? In this issue, Tsai *et al.* explore the magnitude of LD in the Samoan population, a known 'population isolate', and contrast it with a general Caucasian population. Even though the density of the markers (~10 centimorgans) is insufficient to address the question fully, the observed elevation of LD in the Samoan versus the Caucasian population does strongly mitigate in favour of the use of population isolates in mapping.

High-order genomic structure, such as segmental duplications (SDs), may mediate genomic variations. Mehan *et al.* conduct a database search for SDs in the human genome and find that a large proportion of SDs, either palindromic or tandemly orientated, can mediate genomic variation.

Macpherson *et al.* apply two existing Bayesian approaches (one based on all of the data and one based on summaries) to seven Y chromosome microsatellite loci typed in a worldwide sample of 677 males. Both methods are explored in detail, and the comparison of the two methods demonstrates how much information is lost by using the more computationally feasible approach.

Besides the three afore-mentioned articles, this issue also includes a thorough review on the application of microarray technology to cardiovascular diseases including coronary artery disease, myocardial infarction, congestive heart failure and congenital heart disease. The software review provides a comprehensive description and comparison of the methods for Bayesian-based gene mapping. In addition, a piece by Kalow provides a stimulating and personal account of the rich history of pharmacogenomics. The recounting of Kalow's initial discovery of genetic variation in human pseudocholinesterase illustrates the advances in both pharmacology and genetics, highlighting how current studies in pharmacogenetics will have an increasing importance in optimising drug therapy. Finally, the update in this issue describes the problems with cyclophilin nomenclature and offers a logical solution.

Li Jin
Managing Editor
Human Genomics