On the day the completion of the Human Genome Project was announced, the President of the Royal Society, Lord May of Oxford, pointed out that humans share about 50% of their genes with bananas. The parallels between our genome and those of other species are remarkable. Approximately 40% of our genes are shared with yeasts, 60% are shared with worms and about 80-90% are shared with mammals, such as mice or rabbits. Around 99% are shared with chimpanzees and other great apes. The difference in the genome between ourselves and chimpanzees are therefore tiny compared with the overwhelming overall differences. However, they are crucial because they make humans 'human.'

It has become evident that the two 'genomes' that each of us carry, inherited from our parents, differ from each other and from the genomes of other humans, in terms of single changes in nucleic acids. The 21st century has witnessed exponential growth in the identification of these so-called single nucleotide polymorphisms (SNP). The International SNP Map Working Group estimates that they have identified 1.42 million SNPs within genes.[1] However, the main use of the human SNP map will be in delineating the contributions of individual genes to diseases that have a complex, multi-gene basis. Knowledge of genetic variation already affects patient care to some degree. For example gene variants lead to tissue and organ incompatibility, affecting the success of transplants. The mainstay of medical genetics has also been the study of the rare gene variants that are the basis of inherited diseases such as cystic fibrosis. The most valuable aspect of variation in genome sequences underlie differences in our susceptibility to, or protection from all kinds of diseases including the age of onset, severity of illness, and how our bodies respond to treatment. Therefore, by comparing patterns and frequencies of SNPs in patients and controls, researchers can identify which SNPs are associated with which diseases.

The clinical application of SNPs is currently being investigated in two projects here at Trinity College, the LIPGENE Project and the IMAGE Project. LIPGENE is an ambitious five-year interdisciplinary research project aiming to reveal the link between genes and obesity. It involves a consortium of 25 research laboratories across Europe, led by Professor Michael Gibney and Dr. Helen Roche of the Institute of Molecular Medicine and the Department of Clinical Medicine. A primary focus of LIPGENE will be to identify interactions between dietary lipids and SNPs involved in the development of the 'metabolic syndrome'. The IMAGE (International Multicenter ADHD Genetics) Project is another ambitious collaboration between the Neuropsychiatric Genetics Group, led by Professor Michael Gill of the Department of Psychiatry, the Institute of Psychiatry in London, Harvard Medical School and clinical centres throughout Europe. One of the aims of this project is to identify SNPs in the genome that predispose and lead to the development of attention-deficit hyperactivity disorder (ADHD). The genetic basis of obesity and ADHD is explored further in this issue of the TSMJ.

Finally, we should pay heed to Aravinda Chakravarti's warning in the issue of *Nature* which announced the completion of the Human Genome Project, *"To some, there is a danger of genomania, with all differences (or similarities, for that matter) being laid at the altar of genetics. But I hope this does not happen. Genes and genomes do not act in a vacuum, and the environment is equally important in human biology."*[2]

References

1. Sachidanandam R, Weissman D, Schmidt SC, et al. A map of human genome sequence variation containing 1.42 million single nucleotide polymorphisms. *Nature* 2001;409:928-33.
2. Chakravarti A. Single nucleotide polymorphisms: . . .to a future of genetic medicine. *Nature* 2001;409:822-3.