

touching base

What's in a Name

Looking for approval? The Human Genome Nomenclature committee will be more than happy to give it to you — for your gene name, that is. The committee is working hard to provide approved names and symbols for newly identified human genes. Sue Povey, nomenclature committee editor, suggests researchers with a novel gene first investigate the information available on the Nomenclature Home page <<http://www.gene.ucl.ac.uk/nomenclature/>>, which provides information about what gene symbols are likely to be acceptable as well as makes available a list of current names and symbols in use. The nomenclature committee is working towards a semi-automated system for acquiring gene names, but human checking at some stage will likely always be necessary. To obtain an approved gene symbol, researchers should contact the nomenclature committee with information about the gene and a name and symbol suggestion; negotiations will commence! Currently the best contact point is via E-mail at: nome@galton.ucl.ac.uk, which is examined every day by two experienced post-doctoral scientists who work virtually full-time on nomenclature information. (Queries can also be sent by FAX to : [+44] 171 387 3496.) Attempts to turn things around quickly are made for submitted manuscripts, and information is of course treated confidentially. The committee maintains a 'private' database that includes prepublication information, thus reducing name duplication while manuscripts are in press. The necessity and importance of obtaining and using approved gene names will only increase as the human genome project proceeds.

Follow the Money

The editorial in the May issue of *Nature Genetics* provided details of the new wave of genomics companies and their pharmaceutical ties. These links are laid bare in a fold-out map appearing in the October issue of *Nature Biotechnology*. Were the financial flow chart to illustrate intra-company cash flow, an extra arrow would soon be added to the scheme. In a move designed to consolidate its genetic component, Glaxo-Wellcome is to create a separate genetics directorate — to operate autonomously from (but maintain and develop links with) its three current directorates of development, research, and commercial application. The new directorate may only last a few years, as its very formation is considered a means to an end, resulting in a company in which genetics is more fully integrated with other aspects of pharmaceutical development.

Late-Breaking, Anyone?

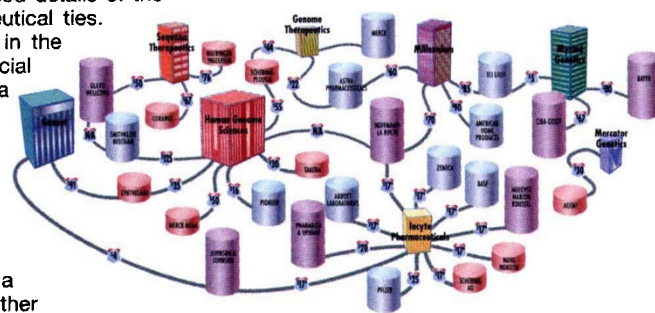
As advertised elsewhere in this issue, *Nature Genetics* hosts its fifth international conference next year (17–18 April, 1997) in Washington, D.C., on the subject of *Functional Genomics: From Genes to Drugs*. As at this year's meeting, there will be time devoted to at least two late-breaking research presentations. Anyone interested in being considered to give a talk at the conference is invited to contact one of the editors informally after the New Year. A decision on the talks will be made as close to the meeting as possible.

Less Than Zero

New findings from the group of James Lupski and colleagues show that a third peripheral neuropathy is associated with mutations in the myelin protein zero gene (*MPZ*), and suggest a correlation between the severity of the respective disorders and the nature of the mutation involved. Three years ago, reports demonstrated that a form of Charcot-Marie-Tooth disease (CMT1B) and the more severe Dejerine-Sottas syndrome (DSS) were caused by mutations in the *MPZ* gene. Warner *et al.* (*Neuron* 17, 451–460; 1996) find that a patient with congenital hypomyelination (characterized by early onset of hypotonia and very slow nerve conduction velocities) has a *de novo* nonsense mutation in the intracellular domain of the protein. Mutations in DSS probably act in a dominant-negative manner, producing an inactive dimeric complex, whereas the milder phenotype in CMT1B results from a variety of heterozygous mutations presumably causing a loss of function and reducing the amount of normal protein. These suggestions are supported by the analysis of one family, in which the parents, heterozygous for a frameshift mutation, are mildly affected with CMT1B, but their two homozygous children have DSS.

It's So Easy

A new 'proprietary functional genomics initiative' — or in plain English, a protein development platform called *DiscoverEase*TM — has been launched by Genetics Institute (GI), one of the most successful biotechnology companies in the United States, to identify therapeutically useful secreted proteins, the leading class of biotechnology drugs. Last July, GI was awarded a patent for a novel method of cloning cDNAs 'which encode cytokines ... and other secreted proteins'. Known as the signal sequence trap, the strategy relies on the fact that *Saccharomyces cerevisiae* must secrete the enzyme invertase (which cleaves sucrose and raffinose) to thrive on certain carbon sources. Mammalian cDNAs are spliced to the cDNA for a non-secreted invertase, and transformed into a yeast strain lacking invertase. If the yeast invertase is ligated to a mammalian secretory leader sequence, the yeast will be viable. From that point, it is a relatively straightforward process to isolate the full-length secretory cDNA and purify the corresponding proteins. GI says it has 70 employees working on the project, and hopes to identify and express 5,000 secreted proteins within the next year. GI has already forged a collaboration with Genentech and Chiron to screen the proteins, but will make the resource available to academic institutions as well.



USA over DNA

This month's sign that the trend in genetic reductionism is going a little too far: in celebration of the United States' dramatic recent victory in the Canada Cup — the World Cup of ice hockey — the weekly magazine *Sports Illustrated* served up an amusing testament to the virtues of nurture over nature. 'Canadian players have a hockey DNA', wrote *SI*'s Michael Farber, 'that's supposed to remind them who and what they are in the dying moments of important one-goal games.' Alas for the Canadians, their genetic legacy failed them. Team USA climbed back from a 2–1 deficit in the final game in Montreal, remarkably scoring 4 goals in the final three minutes, sending a proud nation into shock.