

Newsdesk

Gene therapy in trouble?

Concerns have been raised (see editorial, *Nature* 1999;402:107) about possible failure to disclose side effects related to gene therapy, which may include a number of fatalities, since the financing of gene therapy research has been progressively handed over to the commercial sector following its initial support from US government agencies such as the National Institutes of Health. At least six deaths have occurred recently in patients who were participating in trials of gene therapy for various conditions, including attempts to grow new cardiac vessels in heart attack patients. Not all of the deaths can be attributed directly to the gene therapy itself and the researchers involved justify their lack of disclosure of these events on this basis, indicating that the wrong message concerning the value of the therapy would get into the public domain. However, more ominously there appears to be a reluctance to involve the NIH's Recombinant DNA Advisory Committee (RAC), whose mandate is to debate the ethics of gene therapy in public, and who would finally offer opinion as to the true cause of the fatalities/side effects of gene therapy objectively. As the editorial states, "companies loathe disclosing anything that could harm share prices". These must be the wrong reasons for lack of disclosure and will only harm the prospects for gene therapy in the long run.

PubMed Central

The writing, reading, and processing of scientific papers by authors, referees, and editors alike will take a very different form in the future, and in the near future at that. The NIH has established a new mechanism of scientific information dissemination in which a free global repository for the primary literature in the life sciences will be available for use by the research community. It is called PubMed Central and will be available on the world wide web just as PubMed Medline services are available at present. Journals such as the *BMJ* are likely to participate and in time it is probable that all journals that wish to remain competitive (or even to survive) will be published by this means. A similar mechanism is being set up by the European Molecular Biology Organisation and developments on both sides of the Atlantic are being viewed with interest.

World hunger

A new website has been created that allows individuals who are browsing to do some good for the world's hungry. The following review appeared in the 22 October issue of the *Washington Post* (USA): "Think of all the time you spend clicking aimlessly or fruitlessly around the Web. At the Hunger Site,

one click actually accomplishes something! It sends a serving of food to a starving person, at no cost to you. Corporate sponsors provide the food in exchange for free advertisements and links. Since its June 1 start-up, the site has sent enough money to the United Nations' World Food Program to purchase more than 4 million servings of dietary staples; a WFP official calls it 'an extraordinary testimony to the power of the Internet'. The privacy-protected site is run without profit by John Breen, an Indiana software programmer who initially wanted to support Third World education but decided hunger was the priority. As his world map arrestingly illustrates, starvation kills 24,000 people daily, most of them children." The website to click is <http://www.thehungersite.com/>

Genes for type 2 diabetes

Susceptibility to type 2 diabetes has been known for some time to be linked to genetic defects but their nature is not clear. A recent paper in *Nature Genetics* (Malecki *et al*, 1999;23:323-8) has identified a new mutation which may be responsible for type 2 diabetes in humans. Mutations in the gene *Neurod1* which is a helix-loop-helix (HLH) protein that regulates endocrine pancreatic development have been found to be associated with severity of type 2 diabetes. In mice homozygous for a targeted disruption of *Neurod*, the pancreas fails to develop properly and overt diabetes develops as a result, in part, of failure of insulin secretion. The authors describe two mutations in *Neurod1*, which are associated with the development of type 2 diabetes in the heterozygous state. The first is a missense mutation at Arg 111 in the DNA binding domain which interferes with function of *Neurod1* while the second truncates the polypeptide. Patients with the truncated *Neurod1* gene have more severe diabetes than patients with the Arg 111 mutation. The authors suggest the possibility that promoters in pancreatic islets which fail to bind *Neurod1* or bind a transcriptionally inactive *Neurod1* are the main reason for the development of type 2 diabetes in humans.

Guide Dogs for the Blind Association announces new scheme for ophthalmic research

The Guide Dogs for the Blind Association (GDBA) has announced its return to supporting ophthalmic research by launching a £5m scheme. Described as the "New way forward" strategy, GDBA has pledged to support research over the next seven years. This builds on its previous support going back to 1990 since when it has funded over 80 projects in diverse areas such as diabetic retinopathy, age related macular degeneration, glaucoma, retinal detachment, and ocular tumours. GDBA

was founded in 1934 to provide welfare for blind and partially sighted people, including the training and provision of guide dogs. Its mission today is to provide guide dogs, mobility, and other rehabilitation services that meet the needs of the visually impaired community. GDBA states that the key objectives of the new scheme are to concentrate on the causes, treatment, and prevention of sight threatening diseases and conditions, with priority given to those conditions with a high prevalence, particularly if they lack effective treatments. A second strand to its activities will be to develop stronger communication between the ophthalmic profession and the general visual impairment sector.

1999 Golden Brain Award

Nikos Logothetis has been awarded the Golden Brain Award for 1999 by the Berkeley based Minerva Foundation, established in 1984 to promote basic research in vision and the brain, for pioneering work that explores visual perception. Logothetis, professor of neuroscience and director of the Max-Planck Institute for Biological Cybernetics in Tübingen, has shown how the eye makes sense of conflicting images when the eyes see an optical illusion—for example, a three dimensional cube in which the walls appear to face alternating directions. By combining single neuron tracking experiments with functional magnetic resonance imaging, he has produced data which suggest that neurons whose function it is to interpret such images are distributed over the entire visual pathway and are not located to a single higher vision association area. He has also shown that other areas such as the frontal lobe may control the processes that select a particular image when there are conflicting images as in the case of optical illusions. This selection process is not limited to visual stimuli but may apply generally to sensory systems such as auditory networks.

NSAIDs may be toxic to the cornea

Recent reports suggest that some non-steroidal anti-inflammatory drugs may be toxic to the cornea especially if used frequently or intensively. As reported in *Eyenet*, the magazine of the American Academy of Ophthalmology, a survey conducted by the American Society of Cataract and Refractive Surgery has found at least 200 cases of corneal toxicity related to the use of NSAIDs. No relation with associated corticosteroid use was noted but patients with dry eye were more at risk. Both diclofenac and ketorolac (Acular) were implicated. According to *Eyenet*, Alcon the suppliers of generic diclofenac have suspended distribution of the drug while investigating these reports.