Glaxo Wellcome continue the battle against TB
The re-emergence of TB as a global threat comes not simply as part of the legacy of AIDS but, in fact, is more directly the result of the appearance of multidrug resistant strains of the mycobacterium. Current statistics suggest that eight million new cases of TB occur each year with about three million dying from the disease. As such, TB is once more a significant global threat and commercial pharmaceutical companies such as Glaxo Wellcome have joined the battle against TB as announced on World TB day in March. Glaxo Wellcome have underwritten a research initiative, named Action TB to the tune of £10 million with the object of discovering new treatments for TB. According to Glaxo Wellcome chairman Sir Richard Sykes, one of the main reasons for failure of current treatments is compliance with the minimum course of 6 months’ therapy. After an initial successful phase one of Action TB in which several new drug targets have been identified, phase 2 will narrow down on the more promising drugs targets and develop them in models of infection. Vaccines are also a major thrust of the campaign. Action TB believes that success of the initiative will only come if targets are kept clear and well defined.

Wellcome Trust promotes research on biomedical ethics
The Wellcome Trust has established a programme for the development of evidence based policy making which aims to inform and facilitate public debate of scientific issues. The programme is named the Medicine in Society Programme and has extended its brief to include biomedical ethics. Part of the remit of the programme is to promote research in many of those issues which, according to Dr Tom Wilkie who heads up one section of the programme, requires a strong intellectual community of bioethicists engaged in research of direct practical relevance. In addition, there will be a greater involvement in social research to include novel approaches to public consultation. The trust will draw on information from existing research programmes it supports in genetics and neuroscience research but will not be confined by them, drawing also on a wider resource from many fields of biological research. The programme will support both PhD studentships in the form of fellowships and project grants.

A simple idea brings braille writing to many more users
Braille, invented in 1824 by the French educator Louis Braille, has brought literacy to many visually impaired people and remains the major source of such material for many. The system is based on a series of raised dots organised into cells of two dots wide and three dots high, the number and position of the dots corresponding to letters, numbers, words, and phrases. However, only an estimated 20–30% of those who can read braille have the facility to write the code since writing is normally performed in reverse—that is, writing braille is performed by placing a sheet of paper on a perforated slate and using a solid stylo to punch the paper down into the holes in the slate, thereby producing the embossed dots. Now, an American engineer and inventor, Lawrence Hawk, has developed a technique which using raised bumps or pins and a hollow stylus to press the sheet of paper so allowing the paper to be pushed up and around the bumps rather than in the opposite direction This “direct manual braille slate” now permits braille users to write braille from left to right in the “normal” manner rather than having to perform writing in reverse. Hawk has had his idea for some time—in 1993 he was awarded a Rolex Honourable Mention—but it has taken some time to catch on, mainly because of manufacturing and production difficulties, since Hawk wishes to make his invention available to as many as possible at low or even nil cost for the neediest. Currently, supported by the Matrix Tool and Mold Company in California, Hawk still needs financial support to achieve his dream of making this simple invention available to all who need it.

Continuing progress in gene therapy
The possibility of providing normal genes to replace or even to modify existing genes which may be causing disease through gene therapy methods has continued to excite the scientific community during the past few years. Ophthalmological and, in particular, retinal diseases appear to offer a unique opportunity to test the potential of gene therapy since any of these diseases are the result of single gene abnormalities. Two major difficulties present themselves in the application of gene therapy: how best to deliver the healthy gene in a sustainable form and how to prevent host immunological responses to the new gene and/or its vector. Viral vectors are a popular choice for this type of therapy and in particular adenoviral or adeno associated viral vectors have been frequently used in animal models of retinal degeneration. However, persistence of the exogenous gene has usually been short lived either due to spontaneous viral decay or more likely due to immunological clearance of the virus from the host tissues. Now, a recent paper in Nature Genetics (Feb 1998) has described a form of adenovirus which appears to evade the immune system because almost all of its viral coding sequences have been deleted. So far this vector has been used only for carrying the gene for a1 anti-trypsin in a proposed therapy for emphysema but if it proves stable its value for ocular disease will be apparent.

Is aminoguanidine coming of AGE?
Advanced glycation end products (AGEs) are well known to ophthalmologists and ophthalmic scientists as the culprits in diabetic associated changes to the extracellular matrix, to circulating proteins such as haemoglobin and albumin, and even to intracellular proteins particularly enzymes where they are considered to be potentially harmful (e.g. alteration of the normal metabolism of intracellular metabolism and other normal homeostatic functions. However, AGEs also appear in greater quantities in tissues affected by age ie increased longevity and are known to be deposited for example in conditions such as age related cataract and age related macular degeneration (AMD). They also appear to be components of other age related non-ocular disease such as cardiac hypertrophy associated with hypertension and with arterial stiffening which occurs with age. Inhibitors of AGE deposition have been available for some time, particularly the drug aminoguanidine but it has been considered somewhat toxic for use in vivo. Now a study in the Proceedings of the National Academy of Sciences has reported that aminoguanidine fed in the drinking water of rats selectively prevented cardiac hypertrophy and collagen deposition while having no effect on other arterial wall components such as elastin content and smooth muscle cells numbers. The authors attribute these improvements to a decrease in AGE induced cross linking of the extracellular matrix. This may have implications for similar changes in Bruch’s membrane in AMD.