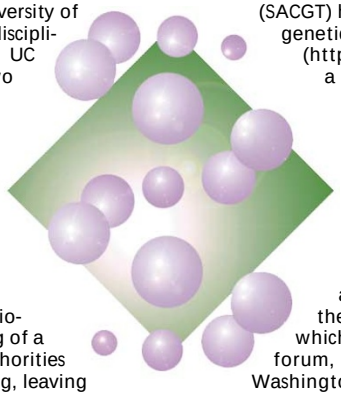
TOUCHING**base**

QB3 Valley

California Governor Gray Davis has allocated $100 million of state funds over the next four years to three University of California (UC) campuses, to create a new multidisciplinary institute of research named 'QB3'. Other UC campuses will receive comparable funding for two other 'California Institutes for Science and Innovation', devoted to telecommunications, computing and nanotechnologies. QB3, the 'Institute for Bioengineering, Biotechnology and Quantitative Research', will offer joint programs to students and researchers of the UC in Northern California. UC Santa Cruz aims to develop its bioinformatics resources; UC Berkeley, its structural and chemical biology facilities; and UC San Francisco is looking to upgrade its facilities in bioengineering and biotechnology. This is something of a departure from the norm, as the US state authorities have traditionally invested in educational training, leaving it to the federal government to fund research. Although California is not the first state to finance basic research, its current initiative dwarfs those of other states. Remarkably, corporations have already topped the governor's goal of obtaining two-to-one matching funds from private sources in a bid to replicate the Silicon Valley entrepreneurial model. In order to catalyze the kind of economic chain reaction initiated in Silicon Valley, however, the partnership between academia and industry must evolve into a culture of collaboration, with technology transfer. In any event, new faculty positions in bioinformatics, genomics and biological chemistry should soon be available.



All together now

The US Secretary's Advisory Committee on Genetic Testing (SACGT) has published its final report on the oversight of genetic tests. The report, available on the web (http://www4.od.nih.gov/oba/), is the culmination of a process that began in June 1999, when the SACGT was asked to assess existing programs for assuring the accuracy, safety and effectiveness of a range of genetic tests, while keeping in mind the health and well-being of patients and communities. The ambitious goal was to develop a general method to categorize all genetic tests so as to define for each of them an appropriate degree of 'scrutiny'. This was not a straightforward task. A remarkable aspect of the project was the extensive public consultation, which included targeted mailing, a web-based forum, a Federal Register and a public meeting in Washington, DC, involving advocacy groups, health professionals and patients with genetic conditions. Some pointed out that predictive tests, or those for low-penetrance conditions, present a greater ethical challenge than do diagnostic ones and therefore require a higher level of scrutiny. Others suggested that tests be categorized based on their stage of development or on the availability of treatments or preventive measures. The result is a multidimensional space where a test can now be placed graphically and its benefits, risks and recommended degree of scrutiny can be visualized. Another topic of public discussion is the supervision of testing and whether private consortia might fruitfully instruct the Food and Drug Administration, which is responsible for maintaining high standards.

"What ever will we think about now that the genome project is almost complete?"

"It's the straight line that connects functionality.... the sweat beads on your forehead. The impact of a hard blow. It's fashion manifested into action. Undisputed. Since day one."

—the Mecca DNM "Jean Therapy" campaign

A new receptor for HIV?

Variants of the human immune deficiency virus (HIV) has been isolated that can infect cells through the CD8 receptor—from one person with AIDS (*Nature Med.* **7**, 65–72; 2001). This observation, if confirmed, might explain the decline of CD8-expressing cytotoxic T cells in late stages of the disease. It is generally believed that the virus gains entry to the CD4-positive helper T cells or macrophages through the binding of its envelope protein, gp120, to the CD4 receptor. And it is thought that CCR5 or CXCR4 co-receptors are required for virion entry. The findings of Kunal Saha and colleagues (Ohio State Univ.) thus breathe new life into a 'play' whose stage seemed set some time ago. They found that two variant forms of HIV1 can infect cells that express CD8 and lack CD4. Although others have found evidence of HIV in CD8+ cells, it was assumed that the virus had infected them at an early stage of development; precursor T cells transiently express both CD4 and CD8. Using highly purified CD8+, CD4– T cells, or HeLa and COS cells engineered to express CD8, Saha *et al*. show that CD8 mediates the entry of the new HIV1 variant. It seems that its altered tropism has been effected through mutation of residues irrelevant to interaction with CD4, as the virus is still able to infect CD4+ cells. How could such a variant come to be? The authors provide plausible explanations of how CD8-tropic HIV1 strain might have been overlooked. As CCR5 or CXCR4 are not required for CD8-mediated infection, one would predict the discovery of additional players before the mechanism of HIV infection is fully understood.