RÉSUMÉ

Speedy genes

THE technique of transferring foreign genes into living cells by coating inert gold particles with DNA and literally bombarding them through the cell membrane has been fairly well documented for plants. But the successful application to mammalian somatic cells now reported by N.-S. Yang *et al.* (*Proc. natn. Acad. Sci. U.S.A.* **87**, 9568–9572; 1990) suggests that the method may prove of value for gene therapy. Five different reporter genes were coated onto gold beads and, by accelerating them at high voltage, were successfully introduced and expressed in rodent liver, skin and muscle tissue. The method works equally well *in vitro* with a variety of cultured cell lines, and offers a potentially useful alternative to viral gene transfer.

Getting tough

RESEARCHERS at Los Alamos National Laboratory have discovered a new way of toughening the surfaces of ceramics — a vital step if these hard but brittle materials are to be put to widespread use in mechanical engineering. Silicon carbide, one of the hardest materials known, is liable to fracture owing to defects on its surface. But T. R. Hervis, J.-P. Hirvonen and M. Nastasi (*J. Mater. Sci.* **6**, 146–151; 1991) find that incorporating titanium into the SiC surface, using laser heating, has dramatic effects on the material's properties: surface cracking and flaking can be prevented almost entirely. Similar results have been achieved using ion implantation of titanium, but the new process, involving the evaporation of a thin layer of titanium onto the ceramic's surface followed by rapid heating in air by an excimer laser, is more convenient, faster and produces a surface that is in thermal equilibrium. A puzzling variation of friction with humidity apparently has no effect on the durability.

How cats purr

THE gentle purring of contented cats results from laryngeal modulation of respiratory flow, as shown by D. E. Frazer Sissom *et al.* (*J. Zool., Lond.* **223**, 67–78; 1991). The authors' comprehensive monitoring of obliging domestic cats (*Felis silvestris* f. *catus*) with oscilloscopes and sound recording equipment revealed that the sound emanates from the oscillation of the vocal folds as the cat inhales and exhales. This is independent of phonation, the pitch of which is controlled by stretching of the vocal chords, which explains why cats can purr and mew at the same time. Purr frequency is independent of age, size, weight and sex, and ultimate control is exercised by an oscillator in the brain. Parallel studies of purring in cheetahs (*Acionyx jubatus*) and pumas (*Puma concolor*) were understandably less thorough, but yielded similar results.

Vβ-selective elements and their linkage to retroviruses

Vbse	Vβ	MHC	Chromosome	Retrovirus	Reference
Mls-1a	6,8.1,9*	II, E>A	1	Mtv-7	6
Mls-2a	3	E>>A	4	Mtv-13	6
Mls-3a	3	E>>A	16	Mtv-6	6
Mls-2-like	3	?	7	Mtv-1	6, R. Abe (pers. comm.)
Etc-1 (Dvb 11-2)	5.1, 5.2, 11	E	12	Mtv-9	4,7,8
Dvb11-1	11	E	6	Mtv-8	8
Dvb11-3	11	E	14	Mtv-11	8
Unnamed	14	E	Extrinsic	MMTV	5
MAIDS B cell	5	II	Extrinsic	MuLV	9, H. C. Morse (pers. comm.)
Unnamed	17a	E	Not mapped†	?	29, 30
Unnamed	12	E	Not mapped	?	31, R. J. Hodes (pers. comm.)
Unnamed	16	E	Not mapped	?	31, R. J. Hodes (pers. comm.)
Unnamed	19a	E	Not mapped	?	R. J. Hodes (pers. comm.)

* Mls-1a is often stated to delete Vβ7 as well, but this is not always confirmed (R. Abe, personal communication).

† Non-polymorphic

tioned. One issue raised by the new findings is why retroviruses encode Vbse, and why mice retain such large numbers of these sequences in their genome. Stimulation *in vivo* with Vbse[19,20] is known to induce widespread immune suppression, so one possibility is that retroviral Vbse facilitate viral invasion of the host by suppressing the immune response; alternatively, activation of T cells by retroviral Vbse may make them susceptible to retroviral infection, facilitating spread of the virus. The mouse may counter these strategies by retaining viral sequences which eliminate those T cells able to respond to viral Vbse.

The previously undefined nature of *Mls* genes has led several groups to study a similar T-cell response driven by the bacterial toxic mitogens[14,21]. These substances cause food poisoning, toxic shock syndrome and other human disorders. They are potent mitogens for T cells whose receptors are encoded by one or a few Vβ gene segments, require class II MHC molecules to stimulate, act selectively on CD4 T cells, and cause clonal deletion of developing T cells. Thus, the bacterial toxic mitogens were thought to be possible homologues of endogenous Vbse[22]. If so, they seem to have achieved this result by a distinct evolutionary pathway, because they show no structural similarity to endogenous retroviral genes. An alternative hypothesis is that the bacterial toxic mitogens act on the endogenous Vbse, mimicking their effects by perturbing their structure[23]. If so, then all species whose T cells respond to bacterial toxic mitogens must have Vbse, most notably humans. Although less is known about endogenous retroviruses in man, possible retroviral insertions have been reported[24]. The new findings should allow a rapid definition of Vbse genes in several species.

The fact that Vbse may be encoded by a retrovirus and act primarily on CD4 T cells raises the question of whether the most notorious retrovirus, the human immunodeficiency virus (HIV), which causes AIDS, may also encode a Vbse that is involved in pathogenesis. CD4 T-cell depletion in AIDS could occur as follows. HIV infects CD4 T cells, remaining latent until the T cell is activated. As activated human T cells express MHC class II proteins, HIV-infected CD4 T cells would express both HIV retroviral proteins and MHC class II, allowing them to interact with T cells expressing a complementary Vβ. It has recently been shown that activated human T cells presenting bacterial toxic mitogens induce *inactivation* of cells expressing the corresponding Vβ[25]. Furthermore, *in vivo* administration of bacterial toxic mitogens[26] or Mls-disparate cells[27] leads first to activation and then to profound depletion of CD4 T cells

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