

### Solvent solution

IN an epidemiological study published in *The Veterinary Record* (128, 199–203; 1991), J.W. Wilesmith *et al.* trace the onset of the bovine spongiform encephalopathy (BSE) epidemic in British cattle back to a change in the practice of meat and bone meal production. Around 1980, most British 'rendering' plants stopped using hydrocarbon solvents to separate fat from carcasses. This abrupt change fits in with the supposed transmission to cattle, through their feed, of the infective agent of scrapie (a similar disease afflicting sheep) in the winter of 1981–82. The authors point out that the additive effects of heat treatment and solvent action, or the additional steam heating needed to remove the solvents, may explain the agent's inactivation when solvents are used, and that regional variation in rendering practice could underlie the distribution of BSE cases.

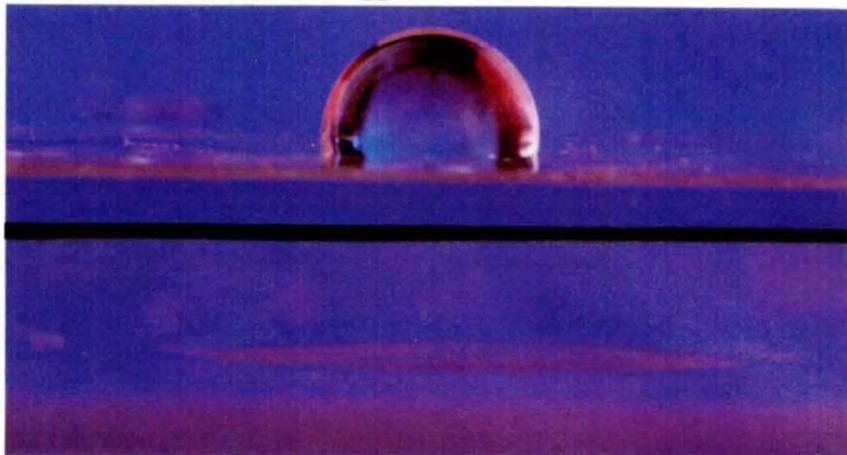
### Bated breath

Is the Earth warming up in response to increased atmospheric concentrations of greenhouse gases? The answer, according to T. Karl *et al.* (*Science* 251, 1058–1061; 1991), is that we won't be able to tell for some time yet. Although the past decade was the warmest on record, it is difficult to distinguish the effects of additional greenhouse gases from natural variations in the climate. Comparing the records of temperature and precipitation for the central United States with model projections for the past 95 years, the authors find no significant indications of warming trends. If the model projections are right, an unambiguous greenhouse signal will emerge only over the next 10–20 years. It will take even longer for any effects of greenhouse warming to show up in levels of precipitation. This leaves policy makers, the authors remark, "in an unenviable position".

### Sweet success

USING linkage studies, G.I. Bell and colleagues have mapped the gene responsible for one form of non-insulin-dependent diabetes mellitus (*Proc. natn. Acad. Sci. U.S.A.* 88, 1484–1488; 1991). First described in the 1970s, maturity-onset diabetes of the young (MODY) is an autosomal-dominant disorder that otherwise closely resembles late-onset non-insulin-dependent diabetes. Using 79 DNA markers, Bell *et al.* excluded about 40 per cent of the human genome, including several candidate genes such as the insulin-responsive glucose transporter on chromosome 17. Close linkage to the MODY locus was finally demonstrated in one large affected family with the adenosine deaminase gene, which maps to the long arm of chromosome 20. Although the location of the MODY gene does not betray its identity, its eventual isolation may provide insight into other forms of diabetes as well.

## Fashioning wet-look latex



WHEREAS water on the surface of most artificial plastics and elastomers beads up as shown in the top frame, Isao Noda has developed (page 143) an elastomeric latex that is fully wettable by water (bottom frame; the fully spread droplet is barely visible). The surface of this polystyrene–polybutadiene latex is rendered hydrophilic by adding a diblock copolymer during synthesis, one end of which is water-soluble (polyethylene oxide, PEO) and the other hydrophobic (ensuring solubility in the hydrocarbon latex). For reasons as yet unclear, the amphiphilic copolymer migrates to the surface during polymerization of the latex constituents, there becoming immobilized with the PEO ends exposed to the air.

Block copolymers, used already as emulsifying agents for polymer alloys, are thus able to influence surface as well as bulk properties. The immediate applications (nappies and bandages) may seem prosaic. But might this be a general way to make surfaces wettable only by specific adsorbates? P.B.

figure). Position 156 at which the two HLA-B44 subtypes differ provides a classic example; it is a peptide-interacting residue, the most variable residue of the  $\alpha 2$  domain helix, and substitutions at this position have repeatedly been shown to alter T-cell responses. In the course of human evolution class I subtypes have probably been selected for their capacity to bind and present different disease-related peptides to T cells. Substitutions that endow such properties are likely, given the common mechanism of T-cell recognition, to stimulate distinctive alloreactive cytotoxic T-cell responses. In fact that is precisely how subtypes were discovered in the first place<sup>2</sup>. Thus the experience of Fleischhauer *et al.* with HLA-B44 will probably have general application to cases where transplant donors and recipients are mismatched for class I HLA subtypes.

Another feature of the class I HLA subtypes is their distribution in populations of different geographical origins. For example the B\*2703 allele has been found only in African and American blacks, the B\*2704 and B\*2706 alleles only in orientals, and the B\*2701 and B\*2702 alleles only in caucasians<sup>3</sup>. These differences, which go undetected by current HLA typing and are likely to provoke allograft rejection, undoubtedly contribute to the difficulty of matching transplant donors and recipients of different racial origins. Such considerations substantiate the view that "The racial differences in ABO blood groups and MHC phenotypes make it more likely that a

candidate for transplantation will receive a well-matched kidney from a member of the same race"<sup>7</sup>.

Through the use of an ever-expanding arsenal of non-specific immunosuppressants, surgeons have been able increasingly to evade the immunogenetic rules of transplantation. As a consequence, the value of HLA matching for solid organs transplanted between unrelated donors is often called into question. For bone marrow transplantation there is in contrast little doubt as to the importance of obtaining the best HLA match, and over the past 20 years the procedure has evolved into the preferred therapy for a widening range of haematopoietic disease<sup>6,8,9</sup>. This impressive advance has primarily been achieved by the study of bone marrow transplantation between HLA-identical siblings. Now, however,

1. Fleischhauer, K., Kernan, N. A., O'Reilly, R. J., Dupont, B. & Yang, S. Y. *New Engl. J. Med.* 323, 1818–1822 (1990).
2. Biddison, W. E., Ward, F. E., Shearer, G. M. & Shaw, S. J. *Immun.* 124, 548–552 (1980).
3. López de Castro, J. A. *Immun. Today* 10, 239–246 (1989).
4. Hahn, A. W. A., Schendel, D. J., Hansen, P. W. & Ploegh, H. L. *Hum. Immun.* 11, 69–76 (1984).
5. Bodmer, J. G., Marsh S. G. E. & Albert, E. *Immun. Today* 11, 3–10 (1990).
6. Hows, J. M. & Bradley, B. A. *Br. J. Haemat.* 76, 1–6 (1990).
7. Kasiske, B. L. *et al. New Engl. J. Med.* 324, 302–307 (1991).
8. Martin, P. J., Hansen, J. A., Storb, R. & Thomas, E. D. *Adv. Immun.* 40, 379–438 (1987).
9. Santos, G. W. *Clinics Haemat.* 12, 611–639 (1983).
10. Saiki, R. K., Bugawan, T. L., Horn, G. T., Mullis, K. B. & Erlich, H. A. *Nature* 324, 163–166 (1986).
11. Bjorkman, P. J. & Parham, P. A. *Rev. Biochem.* 59, 253–288 (1990).
12. Bjorkman P. J. *et al. Nature* 329, 506–512 (1987).