Ferroelectricity in oxides

SIR — Cohen[1], using total energy calculations, ascribes the origin of ferroelectricity in $BaTiO_3$ and $PbTiO_3$ to transition metal–oxygen *p–d* hybridization effects. Here, we emphasize that this approach is, of course, not unique because dynamical properties, which are very important in ferroelectrics, are neglected by Cohen.

A unified interpretation of ferroelectricity that is not restricted to perovskite oxides has been offered by Migoni *et al.*[2], who argued that the O^{2-} ion may be crucial for the polar ferroelectric state to occur. These authors took into account the outstanding properties of O^{2-} by introducing a shell model description for the lattice dynamics of perovskite oxides. The temperature dependence of the oxygen-ion polarizability, which is assumed to drive the lattice instability, has been taken into account by introducing a fourth-order repulsive term in addition to a local attractive electron–ion coupling, thus inducing a local double-well potential in the core–shell interaction at the oxygen-ion lattice site. Within self-consistent phonon theory, quantitative agreement with experimental data has been achieved for phonon dispersion relations, second-order Raman spectra, temperature dependence of the soft-mode frequency and coupled branches as well as defect properties.

Extensions to systems other than perovskite oxides have been carried out within a simplified version[3] of the model of Migoni *et al.*, which quantitatively describes the temperature dependence of soft modes in various structurally different compounds[4]. The extremely transparent, physically understandable and analytically tractable model possesses, besides a wide range of applicability, highly interesting nonlinear solutions which exist on the lattice as well as in a continuum approach to the lattice[5]. From these studies on ferroelectric systems, we conclude that the origin of ferroelectricity in oxides is a consequence of the critical dynamics which are driven by crucial interplay of ionic and electronic interactions.

A. Bussmann-Holder
Max Planck Institut für Festkörperforschung, Heisenbergstr. 1, D-7000 Stuttgart 80, Germany
H. Büttner
Institut für Physik, Universität Bayreuth, D-8580 Bayreuth, Germany

1. Cohen, R. E. *Nature* **358**, 136 (1992).
2. Migoni, R., Bilz, H. & Bäuerle, D. *Phys. Rev. Lett.* **37**, 1155 (1976).
3. Bilz, H., Benedek, G. & Bussmann-Holder, A. *Phys. Rev.* B**35**, 4840 (1982).
4. Bussmann-Holder, A., Bilz, H. & Benedek, G. *Phys. Rev.* B**39**, 9214 (1989).
5. Bussmann-Holder, A., Büttner, H. & Bilz, H. *Solid State Physics* **48** (ed. Turnbull, D.) (Academic, Ehrenreich, in the press).

Deviant TATA-box binding protein

SIR — In their paper reporting the crystal structure of one variant of the *Arabidopsis* TATA-box binding protein (TBP), Nikolov *et al.*[1] draw attention to the remarkable similarity of the C-terminal domains of all known TBP proteins, as does Greenblatt in the accompanying News and Views article[2]. Thus, in this 180-residue region, TBP molecules of fungal, plant, insect and mammalian origin are a minimum of 70% identical at the amino-acid level. On this basis, and the fact that variant residues map to positions unlikely to perturb the folding pattern, it is suggested that the reported structure of the TBP DNA-binding domain may be universal.

We have characterized the TBP homologue from the human malaria parasite *Plasmodium falciparum*[3], which we find to be strikingly divergent from these examples. In this case, the corresponding amino-acid identities range from 38 to 44%. In this organism, whose genome shows a highly biased base composition, putative promoter regions have an A+T content averaging close to 90% (ref. 4). Under these circumstances, the unambiguous recognition of conventional TATA-boxes will be difficult, and the observed divergence in protein composition may reflect an evolutionary response to this problem.

In any case, this dramatically altered sequence composition means that caution is needed in regarding the *Arabidopsis* TBP structure as being universal. In particular, the *P. falciparum* molecule may be subtly different from the structure presented. Any such differences between it and its equivalent from the human host might well be reflected in functional differences that in future could be exploited chemotherapeutically.

We would also point out that the residual sequence identities conserved between the *P. falciparum* TBP protein and those of other organisms (57, compared to the 126 seen in the data set of Nikolov *et al.*[1]) may help to define a much reduced number of residues likely to be critical to the interaction of TBP with other members of the transcription initiation complex. This information could aid the informed design of site-directed mutagenesis experiments to investigate further the nature of intermolecular interactions in other eukaryotic transcription systems.

John E. Hyde
Paul F. G. Sims
Martin Read
Michael B. McAndrew
Department of Biochemistry and Applied Molecular Biology, UMIST, PO Box 88, Manchester M60 1QD, UK

1. Nikolov, D. B. *et al. Nature* **360**, 40–46 (1992).
2. Greenblatt, J. *Nature* **360**, 16–17 (1992).
3. McAndrew, M. B. *et al. Gene* (in the press).
4. Hyde, J. E. and Sims, P. F. G. *Gene* **61**, 177–187 (1987).

More on motor neurons

SIR — Vrbová *et al.*[1] raise questions with regard to our paper on the effect of ciliary neurotrophic factor (CNTF) on the mouse mutant progressive motor neuronopathy (*pmn*)[2]. They wonder whether the "relatively modest loss of facial motor neurons" (40%) can account for the death of untreated homozygotes. It certainly cannot.

However, the denervation of the diaphragm at the fourth postnatal week caused by the almost complete loss of motor axons in the phrenic nerve[3] would be sufficient to lead to the death of the animals 2 weeks later. The putative "sharp contrast" they note between the original observations[3] and our manuscript[2], that motor neurons of the spinal cord and cranial nuclei appear qualitatively normal at 5 weeks of age[3] whereas 40% of the facial motor neurons have degenerated at 6–7 weeks[2], simply reflects the rapid progression of the degeneration process in the *pmn* mutant. Moreover, cell numbers of spinal motor neurons were not determined in the original paper[3] so that loss of these neurons cannot be excluded. Similar observations (no loss of anterior horn cell somata) have also been reported for other animal models such as the Brittany spaniels with hereditary canine spinal muscular atrophy (HCSMA)[4]. If one should follow the criterion of Vrbová *et al.*, that any useful model for motor neuron disease should show an initial degeneration of the motor neuron cell body, then the HCSMA model should also be dismissed.

The *pmn* mutant is certainly not identical with amyotrophic lateral sclerosis (ALS) (we never claimed that), but the pathological manifestations of ALS show striking similarities to those observed in