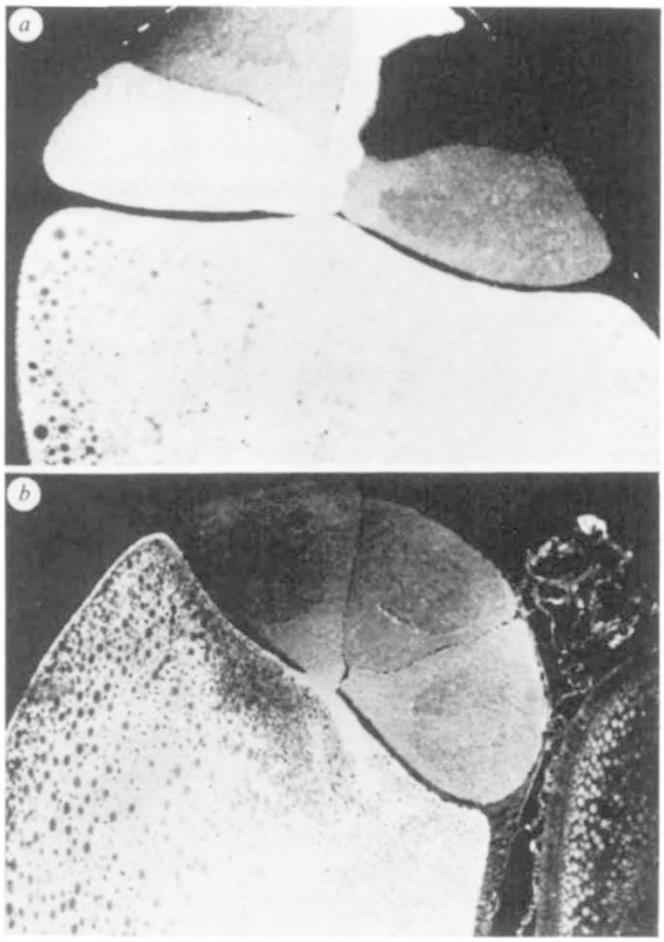

Fig. 3 *a*, Central nurse cell micro-injected with fluorescein-labelled haemoglobin. The protein has passed from the injected nurse cell into a co-bridged cell (lower left nurse cell), into the oocyte and from the oocyte to varying degrees into other nurse cells. *b*, An oocyte micro-injected with fluorescein-labelled myoglobin. A portion of an uninjected control follicle may be seen on the right. This neutral protein was found to migrate throughout the follicle whether oocyte or nurse cell was injected.

even when these were bridged directly to the injected cell (Fig. 1, centre quartet and right hand pair of nurse cells). Polarity apparently extends throughout the bridge complex, and whenever the negatively charged FSG entered this zone, it was unable to move in a direction away from the oocyte. By contrast, when nurse cells were injected with FLy, label entering the zone containing the bridge complex, although unable to proceed into the oocyte, was able to enter the nurse cells directly bridged to the injected cell (Fig. 2*b*). FLy thus seems to diffuse against the electrical potential gradient more readily than FSG.

To confirm that the differences in behaviour resulted entirely from the positive charge of lysozyme and not in part from its small size (molecular weight 14,600), the electrical properties of this protein were altered by methylcarboxylation of its ε-amino groups[10]. The polarity of lysozyme movement in the bridges was now completely reversed—it behaved exactly as FSG had behaved in the earlier work (Fig. 2*e–h*), passing from nurse cell to oocyte (48 injections), but not from nurse cell to nurse cell or from oocyte to nurse cell (40 injections). To extend the correlation between charge and mobility through the bridges, we also injected fluorescein-conjugated haemoglobin (21 injections) and myoglobin (28 injections), both of which have isoelectric points close to neutrality, and found that they were able to move in both directions across the bridges (Fig. 3*a*, *b*). When the fluorescein-labelled proteins were compared by electrophoresis in agarose plates at *p*H 7.0, they were found to exhibit the following sequence of mobilities towards the cathode: lysozyme > haemoglobin = myoglobin > methylcarboxylysozyme ≥ serum globulin. The charge dependence of the mobility of these proteins decisively supports the idea that the potential gradient across the bridges is steep enough to polarize the movement of soluble proteins.

Is the intracytoplasmic electrophoresis observed here a unique adaptation to the transport requirements of the oocyte–nurse cell syncytium or an exceptionally clear example of a more ubiquitous mechanism of polarity? The widespread occurrence of transcellular ion fluxes in developing cells[1,11] suggests that intracytoplasmic potential differences are not unusual. The intercellular bridges, which are a special feature of our system, focus the potential gradient at the equator of the complex, and allow a qualitatively clear result in which fluorescent proteins either do or do not pass through to the uninjected cell. Although the diffusion of our fluorescent probes may prove not to be so dramatically restricted in polarized cells that lack an equatorial constriction, intercellular bridges occur more commonly than might be supposed. Many dividing cells generate transitory bridges of cytoplasm late in cytokinesis[12,13], and ionic communication through these channels attenuates gradually until division is complete[14]. Any dividing cell that is electrically polarized, therefore, should generate a transiently focused gradient in potential steep enough to restrict diffusion to the degree seen here for the oocyte–nurse cell syncytium.

This work was supported by US NSF grant PCM77-16455.
R.I.W. is Professor of Biology at West Chester State College.

Received 18 March; accepted 8 May 1980.

1. Jaffe, L. F. *Membrane Transduction Mechanisms* (eds Cone, R. A. & Dowling, J. E.) (Raven, New York, 1979).
2. Jaffe, L. F. *Proc. natn. Acad. Sci. U.S.A.* **56**, 1102–1109 (1966).
3. Nuccitelli, R. *Devl Biol.* **62**, 13–33 (1978).
4. Robinson, K. R. & Jaffe, L. F. *Science* **187**, 70–72 (1975).
5. Quatrano, R. S., Brawley, S. H. & Hogsett, W. E. in *Determinants of Spatial Organization* (eds Subtelny, S. & Konigsberg, I. R.) (Academic, New York, 1979).
6. Mandelbaum, I. *J. Morph.* (In the press).
7. Woodruff, R. I. & Telfer, W. H. *J. Cell Biol.* **58**, 172–188 (1973).
8. Pollack, S. & Telfer, W. H. *J. exp. Zool.* **170**, 1–24 (1969).
9. Hughes, M. & Berry, S. *Devl Biol.* **23**, 651–664 (1970).
10. Grundlach, H. G., Stein, W. H. & Moore, S. *J. biol. Chem.* **234**, 1754–1761 (1959).
11. Jaffe, L. F. & Nuccitelli, R. *A. Rev. Biophys. Bioengng* **6**, 445–476 (1977).
12. Flemming, W. *Arch. microsk. Anat. Entw. Mech.* **37**, 685–751 (1891).
13. Byers, B. & Abramson, D. H. *Protoplasma* **66**, 413–435 (1968).
14. Ashmau, R. F., Kanno, Y. & Loewenstein, W. R. *Science* **145**, 604–605 (1964).
15. Nairn, R. C. *Fluorescent Protein Tracing* (Churchill Livingston, New York, 1976).
16. Woodruff, R. I. *Devl Biol.* **69**, 281–295 (1979).

Corrigendum

In the *News and Views* item on 'Dendochronology' by Sara Champion, *Nature* **284**, 663 (1980), reference should have been included in paragraph four to the work of Hubert Polge and in particular to his paper, *Br. Arch. Rep.* S51, 77 (1978).

Erratum

In the letter 'Inhibin is absent from azoospermic semen of infertile men' by R. S. Scott and H. G. Burger, *Nature* **285**, 246–247 (1980), the following was omitted from line 13 on page 247 'relationship of inhibin concentrations to those of serum FSH in spontaneous disorders of the seminiferous epithelium has not been studied. It has been previously shown that a reciprocal'.