greatest net conductivity maximizes this measure of diversity.

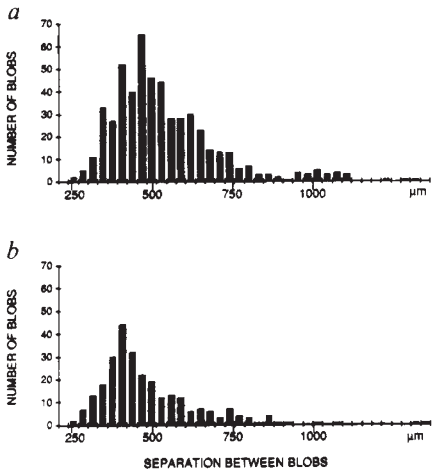
STEPHEN F. ALTSCHUL
DAVID J. LIPMAN
National Center for Biotechnology Information,
National Library of Medicine,
National Institutes of Health,
Bethesda, Maryland 20894, USA

1. May, R. M. *Nature* **347**, 129–130 (1990).
2. Atkinson, I. in *Conservation for the Twenty-First Century* (eds Western, D. & Pearl, M.) 54-69 (Oxford University Press, 1989).
3. Vane-Wright, R. I., Humphries, C. J. & Williams, P. H. *Biological Conservation* **55**, 235–254 (1991).
4. Orwell, G. *Animal Farm* (Secker & Warburg, London, 1945).
5. Felsenstein, J. *Am. J. hum. Genet.* **25**, 471–492 (1973).
6. Felsenstein, J. *Am. Nat.* **125**, 1–15 (1985).
7. Altschul, S. F., Carroll, R. J. & Lipman, D. J. *J. molec. Biol* **207**, 647–653 (1989).

Striate cortex periodicity

SIR—Cytochrome oxidase blobs form a regular repeating lattice in the primate striate cortex (area 17) directly related to its functional architecture[1]. Recently, Kuljis and Rakic[2] reported the size of these blobs following bilateral ablation of the retina on the 81st and 120th (E81 and E120) days of gestation. As early bilateral enucleation also reduces the size of striate cortex[2,3], this raises the question of whether the periodicity of the cytochrome oxidase lattice is also modified by this manipulation.

Kuljis and Rakic report[2] that the size of cytochrome oxidase blobs is unaffected by bilateral enucleation, confirming our previous findings[3]. We have measured the surface area of area 17 following enucleation at E59, E68, E77, E81 and E110. The extent of the reduction depends critically on the age at which the ablation is performed. At the ages examined by Kuljis and Rakic[2], enucleation leads to a reduction of striate cortex of only 14–40%, whereas enucleation at E59 and E68 leads to an areal reduction of more than 70%. We have therefore examined the distribution of blobs following bilateral enucleation at E68.



Frequency distribution histogram of adjacent blob separation on parasaggital sections for a normal neonate (*a*) and a neonate which underwent bilateral enucleation at E68 (*b*).

The mean blob separation in the normal neonate corresponds to published values in the adult[4] and is similar to that found in the neonate enucleated at E110 (normal, 560 μm; E110, 568 μm) and slightly larger than that in the neonate enucleated at E68 (514 μm; see figure). Although small, the difference between the E68 and the normal interblob spacing was statistically significant ($P<0.005$). The 8% linear reduction of interblob spacing following enucleation at E68 would correspond to an areal reduction of striate cortex of around 15%, considerably less than that which we observe in this neonate and possibly resulting from the cell atrophy following this operation[5].

The finding that the reduction in the dimensions of area 17 following early enucleation is not accompanied by an equivalent reduction in mean blob separation is relevant to current theories of cortical parcellation during corticogenesis and the potential role of the sensory periphery. It seems that two levels of specification need to be distinguished. The first occurs early in development, is critically dependent on the presence of the sensory periphery and determines the areal extent of area 17, at least partly by modulating the levels of cell death and proliferation. The second is apparently independent of the presence of the retinae, operates after the determination of areal borders and specifies the periodicity of cytochrome oxidase blobs.

HENRY KENNEDY
COLETTE DEHAY
Vision et Motricité,
INSERM U94,
16 Avenue Doyen Lépine,
69500 Bron,
France

GWYNN HORSBURGH
Department of Neurobiology,
Anatomy and Cell Science,
University of Pittsburgh Medical School,
3550 Terrace Street,
Pittsburgh,
Pennsylvania 15261, USA

1. Martin, K.A.C. *Trends Neurosci.* **11**, 380–387 (1988).
2. Kuljis, R.O. & Rakic, P. *Proc. natn. Acad. Sci. U.S.A.* **87**, 5303–5306 (1990).
3. Dehay, C. Horsburgh, G., Berland, M., Killackey, H. & Kennedy, H. *Nature* **337**, 265–267 (1989).
4. Tusk, T.C., Kaboored, W.S. & Wong-Riley, M.T.T. *Visual Neurosci.* **4**, 185–204 (1990).
5. Dehay, C., Horsburgh, G., Berland, M., Killackey, H. & Kennedy, H. *J. Eur. Neurosci.*

Forlorn hope for malaria vaccine?

SIR—Successful vaccination against viruses and bacteria has, it seems, led to the notion that vaccines could also be developed against malaria parasites. But malaria parasites reproduce sexually within the insect host. Sexual reproduction leads to the maintenance of and increase in heterozygosity, producing phenotypes ready to cope with almost any change that we may impose[1,2]. This important difference seems either to have been misunderstood or disregarded, and I wonder if the notion of finding a valid vaccine against malaria parasites is not flawed from the outset.

It is, of course, true that people living in regions where malaria is endemic develop an immunity, at least to the severe and dangerous symptoms of the disease. But, as anyone who has worked in such places well knows, if these immune people move away from the region, some of them contract malaria after two weeks or so. They have not lost the immunity to their 'home' parasites — although they will do so unless constantly challenged by them — they are being affected by different local phenotypes of *Plasmodium*.

It is this neglect or misunderstanding of the consequences of sexual reproduction, and the maintenance and constant enrichment of genetic diversity that is its corollary, that has in my opinion led the well-intentioned malaria vaccine lobby astray. The same may well apply to the search for a vaccine to combat other eukaryote parasites such as *Trypanosoma*, complicated even further with its sequential antigenic variation, and *Schistosoma*. One witnesses a similar flaw in recent approaches to find a vaccine to combat east coast fever (ECF) in cattle. The argument goes like this: an effective vaccinia virus recombinant vaccine has been developed against rinderpest and "this encourages us to pursue similar strategies in developing vaccinia for other livestock diseases [that is, ECF]", to cite ref. 3. Rinderpest and ECF are, to be sure, both livestock diseases but the former, like yellow fever, is a virus disease and the latter, like malaria, is a protozoal disease.

Perhaps the enormous and expensive effort now going into a search for a malaria vaccine would be better spent on a search for new methods to prevent contact between insect and man[4].

J. D. GILLETT
Department of Medical Parasitology,
London School of Hygiene and Tropical Medicine,
Keppel Street,
London WC1E 7HT, UK

1. Bell, G. *The Masterpiece of Nature* (Croom Helm, London, 1982).
2. Lively, C.M., Craddock, C. & Vrijenhoek, R.C. *Nature* **344**, 864–866 (1990).
3. Ole-Moi Yoi, O.K., Nayar, A., Iams, K., Musoke, A.J. & Yilma, T. *Ann. N.Y. Acad. Sci.* **569**, 174–182 (1989).
4. Gillett, J.D. *Trans. R. Soc. trop. Med. Hyg.* **79**, 12–20 (1985).