Viscosity of the asthenosphere

SIR — Foulger *et al.*[1] have used the observation of transient strain following dyke intrusion in northeast Iceland to estimate a newtonian viscosity for the asthenosphere; their value ($\sim 10^{19}$ Pa s) is of the same order as that obtained from postglacial rebound[2]. Observations such as those presented by Foulger *et al.* are very important, as they sample timescales ($\sim 1 - 1{,}000$ years) for which data are scarce and the rheological behaviour of the crust and mantle may not be steady-state. However, I argue here that the data do not provide a strong constraint on the viscosity of the asthenosphere, because (1) the viscous layers in stress relaxation and in postglacial rebound are not necessarily the same; (2) the estimated viscosity depends on the kinematics of flow; and (3) the inelastic response over $\sim$ 10 yr is likely to be transient and not steady-state.

The model of Foulger *et al.* assumes an elastic layer of thickness h = 8–30 km and modulus $M = 0.75 \times 10^{11}$ Pa overlying a viscous layer of thickness b = 5–10 km, identified with a layer of partial melt. A boundary displacement then propagates in the elastic layer according to the diffusion equation[3]. However, postglacial flow affects a sublithospheric layer at least one order of magnitude thicker[4], which, using the inferred diffusivity, would give a correspondingly higher viscosity. Furthermore, the diffusivity used in ref. 1 ($\kappa = Mhb/\eta$) is valid only for a linear velocity–depth distribution. If the distribution is quadratic (return flow within the viscous layer) the diffusivity is[3]

$$\kappa = \frac{Mhb^2}{2\eta\,(3h+2b)}$$

which, for the same parameter values as in the linear case, gives $\eta \approx 10^{17}$–10^{18} Pa s, and $\eta \approx 10^{19}$ Pa s if $b \approx 100$ km. A different kinematics of flow, therefore, makes a thick channel compatible with postglacial viscosity estimates.

Finally, as the Maxwell time of rocks is $\geqslant$ 1,000 yr, stress relaxation at plate boundaries, with a characteristic time of a few years, is unlikely to sample the steady-state rheology of the asthenosphere[4]. The transient strain rate of rocks at high temperature T usually depends on time t and stress σ as[4,5]

$$\dot{\varepsilon} = C\sigma^n t^{m-1}\exp(-E/RT)$$

where R is the gas constant, and the material parameters for silicate rocks are[5,6] $C \approx 10^{-4}$ Pa^{-n} s^{-m}, $n \approx 2.0 \pm 0.5$, $m \approx 0.4 \pm 0.1$ and $E \approx 400 \pm 150m$ kJ mol^{-1}. The transient viscosity $\eta(t) = \sigma/\varepsilon$ can be expressed in terms of strain rate, whose upper limit can be taken as $\dot{\varepsilon} = \varepsilon/t \approx 8.6 \times 10^{-15}$ s^{-1}, where $\varepsilon \approx 15$ cm per 50 km is the observed strain (one would obtain $\dot{\varepsilon} \approx 5.2 \times 10^{-15}$ s^{-1} assuming validity of the diffusion equation). For t = 11 yr (time of observation of the Iceland transient), T = 1,500 K, and central values of the material parameters, this yields $\eta \approx 4 \times 10^{18}$ Pa s, that is, not significantly different than in the linear model.

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1. Foulger, G. R. *et al. Nature* **358**, 488–490 (1992).
2. Sigmundson, F. *Geophys. Res. Lett.* **18**, 1131–1134 (1991).
3. Bott, M. H. P. & Dean, D. S. *Nature* **243**, 339–341 (1973).
4. Ranalli, G. *Rheology of the Earth* (Allen & Unwin, London, 1987).
5. Carter, N. L. & Kirby, S. H. *Pure appl. Geophys.* **116**, 807–839 (1978).
6. Ranalli, G. & Schloessin, H. H. *Am. Geophys. Un., Geophys. Monogr.* **49**, 55–63 (1989).

Mimicking ligands

SIR — S. Davis *et al.* (*Nature* **358**, 76–79; 1992) explore the interaction of the HIV-1 glycoprotein gp120 and anti-CD4 antibodies with the CD4 receptor. They have carefully documented the binding characteristics of 225 anti-CD4 antibodies, and conclude that none represents an "exact mimic" of the binding of gp120. They further suggest that the underlying concept behind the use of an antibody to mimic a naturally occurring ligand is inherently flawed and "unlikely to be rewarding."

One of their primary arguments against these antibodies appropriately mimicking gp120 is a significant difference in CD4 binding kinetics — k_{on} and k_{off} rates — between the antibodies and gp120. They therefore conclude that "interactions of antibodies and gp120 with CD4 may involve different types of binding sites or that regions outside the binding site affect the k_{on}." They further argue that potential variability in antibody binding sites is so large as to make the use of anti-idiotype antibodies as probes for a receptor essentially fruitless.

We believe that the binding of many agonists and antagonists to well-characterized receptors fails to satisfy the criterion of equivalent binding kinetics. A case in point is one of the best understood receptors, the nicotinic acetylcholine receptor. Nicotine, carbachol, tubocurare, α-bungarotoxin, maleimido-benzyl-trimethyl-ammonium and acetylcholine are agonists or competitive antagonists for the nicotinic receptor. They exhibit mutual competition, and furthermore have been of great utility in unravelling the structure and function of the nicotinic receptor. The structures of these agonists and competitive antagonists vary widely. If presented with these molecules *in vacuo*, it is unlikely that one would conclude that they bind in any biologically significant way to the same receptor. Anti-idiotype antibodies — which may bear little obvious resemblance in either chemical structure or binding kinetics with the naturally occurring ligand — might also prove informative in the characterization of novel receptors.

Although we concur with the authors' stringent criterion for the complete elucidation of a novel receptor, we would suggest that intermediate steps in the process might also be of general interest. We submit that the difficulty in the use of antibodies in unravelling the CD4/gp120 interaction is insufficient grounds for a complete indictment of either the technique or of results obtained with anti-idiotypic antibodies.

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SIR — On the basis of a study of one panel of antibodies specific for a ligand of one receptor, a limited review of the literature and a highly restricted criterion for a molecular mimic, Davis *et al.*[1] conclude that their experiments "undermine the concept of mimicry between antibodies and receptors". They go on to state that the "anti-idiotype approach [to the study of receptors] is unlikely to be rewarding". We disagree. Our experience has shown that the immune repertoire is large enough to yield suitable mimics if proper, rigorous screening procedures are used to find them.

Molecular mimicry by an antibody must be defined operationally. Let us take as one example a monoclonal anti-idiotypic antibody that mimics aldosterone, which was isolated in my laboratory using an auto-anti-idiotypic strategy[2,3]. It has the following characteristics: (1) It binds to Fab fragments of rabbit anti-aldosterone antibody with an affinity of 0.5 nM. (2) It inhibits the binding of [^{3}H] aldosterone to rabbit polyclonal antibodies and to seven different aldosterone-specific monoclonal antibodies. (3) It inhibits the binding of [^{3}H] aldosterone to rabbit kidney cytosolic aldosterone receptor preparations but does not bind to glucocorticoid