for their ability to recognize random patterns in different orientations, networks evolve an increasing sensitivity to particular symmetries. Thus, by analogy, natural selection acting on organisms to recognize signals seen in different orientations offers a plausible source for some of the symmetries observed in biological signals (which evolve to become increasingly efficient at eliciting responses from the receiver). It is largely irrelevant whether we label our starting patterns as "bird", "tail" or otherwise[1]; the point applies to any signal.

It is true that our results can be explained simply by correlations between inputs and outputs that develop when a pattern undergoes geometrical transformation on the pseudo-retina. But to regard our results as an "artefact" of such correlations distracts from the important question of whether such correlations would not also arise when a biologically significant pattern is projected in different orientations onto a real living retina. It seems difficult to avoid the conclusion that this will be the case.

Finally, we take issue with Cook's implication that results obtained from artificial neural network studies are meaningful only if a net conceptualizes input patterns in the same way that humans (or other animals) do. To us at least, it seems questionable whether any model can do this. Although this may be a worthy aim for neurobiologists, who seek a description of the actual computational processes undertaken by the brain, as evolutionary biologists we use such models to explore how neural mechanisms may, in very general ways, influence the evolution of biological signals. In keeping with the best traditions of natural science, it would seem prudent to start with a simple model, rather than attempt to mimic the enormous complexity of real brains.

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Yet more danger for coelacanths

SIR — The living coelacanth *Latimeria chalumnae* is the sole survivor of the old lineage of crossopterygian fishes and is of great importance for evolutionary biology[1]. Recently coelacanths were assigned to 'category 1' of the Convention of International Trade of Endangered Species (CITES). During the past three years we have witnessed an alarming population decline which we believe is due to fishing habits by local fishermen. We wish to alert the scientific community to the plight of the coelacanth and to suggest ways in which the apparent decline may be arrested.

We have been monitoring a coelacanth population, occupying 8 km of coastline off Grande Comore, for the past 6 years[2]. Between 1991 and 1994 the average number of coelacanths in all underwater caves fell from 20.5 to 6.5 individuals. In 1991 we surveyed 59 individuals, but in 1994 only 40.

The fall in the number of individuals could be due to natural population fluctuation, emigration or even disturbances due to the presence of the submersible, but these possibilities seem unlikely. The decline is more probably attributable to human predation. Comorian fishermen, originally using paddle canoes, have regularly fished using long lines for food fish and for the oilfish (*Ruvettus pretiosus*) which live close to shore and at about the same depth as the coelacanth. Occasionally, they hook a coelacanth. Between 1989 and 1991 an almost stable number of coelacanths was sighted; this coincided with a fishing development programme. Fish-attracting devices were installed offshore, far outside the coelacanths' habitat, which dramatically reduced fishing pressure on these animals. However, in 1994, our canoe survey revealed that fishermen, unable to afford repairs to their motorized canoes, were again forced to fish close to shore in the 'coelacanth zone'.

Because the CITES convention is signed by the Comorian government, it is strictly forbidden to land coelacanths, so not all accidentally caught coelacanths were reported. Further, our interviews revealed that several fishermen had killed coelacanths to retrieve their hooks.

We are concerned that the survival of the coelacanth, with an estimated population of about 200 individuals on Grande Comore[2], is severely threatened. We suggest that fish-attracting devices close to shore and within paddling distance, but set at well above the coelacanth zone, would be a useful fishing alternative. An information centre in one of the local villages and a permanently installed on-line underwater television system in front of one of the coelacanth caves could attract tourists and provide the community with income. The World Bank is interested in funding this project through the Global Environment Facility. International efforts should help to continue the population monitoring and scientific investigation of the coelacanth, whose survival depends not only on financial aid but on the education and cooperation of local communities.

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Knockout mouse fault lines

SIR — Despite the prevailing view that valuable information can be obtained with null-mutation experiments, concern has been expressed about the interpretation of the results in some biological systems (see refs 1–3). I believe that recent studies on memory now emerging from several different laboratories[4–7] show that the null-mutant strategy, although a technological *tour de force*, is wholly inappropriate for resolving the issues for which it was intended. It is incapable of allowing the conclusion to be drawn that a particular molecule is necessary for an adult physiological process such as memory or long-term potentiation.

Put quite simply, null mutants are not animals without that protein, but are "reactionisms", organisms that respond to the mutation. This is so because the absence of a single gene is well known to alter expression of other genes and developmental programmes. These reactions to the 'knockouts'[4–8] remain largely undocumented.

The mutation can be either with or without effect. In the case where the long-term potentiation or memory of the null mutant is altered, there is no compelling reason to ascribe the impairment to the loss of that protein when it is equally likely that the altered response results from the fact that the organism itself has been altered by the single gene mutation. In the case where the null mutation yields no loss of function, can one conclude that the molecule is not necessary for that function? I do not think so.

The phrase "considering the evidence for the apparently necessary and sufficient role of [protein x] in [function y] it is surprising that in the 'X' null mutant there is so little functional impairment" has become commonplace. Yet examples from the muscle differentiation field[3] illustrate that a single knockout of an apparently critical transcription factor can be without effect. Because of documented increased expression in related genes in these null mutants leading to compensation, it would not be surprising if a similar event were also present in other single-gene knockouts. Therefore, one cannot conclude, when the protein but not the function is knocked out, that that protein is not necessary. Double knockouts now show that muscle differentiation no longer occurs[3]. Even here the conclusion that

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