predators, even within a species, can show enormous variation in sensitivity to toxins (*3*). But we point out that homobatrachotoxin affects voltage-dependent sodium channels of nerve and muscle cell membranes; these channels are highly conserved in vertebrates and invertebrates (*4*), the only known exception being poison-dart frogs, in which the recognition site on the sodium channel for batrachotoxins is no longer functional (*5*). Thus numbing and burning probably occur in buccal tissue of predators such as snakes, raptors, and arboreal marsupials, neurons of which have the requisite sodium channels. We emphasize that we chose to keep the possible identification of homobatrachotoxin as a chemical defense in birds a tentative one, by framing it as a question in the title of our report, until further field work can be conducted.

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Corrections and Clarifications

The Research News article "Getting it together at the synapse" by Jean Marx (20 Nov., p. 1304) gave the incorrect impression that the chick agrin gene was the first to be cloned. However, Fabio Rupp, James Campanelli, and Werner Hoch (working in the laboratory of Richard Scheller at Stanford University) cloned the rat agrin gene about a year earlier and showed that the protein is made in motor neurons and is active in inducing acetylcholine receptor aggregation on muscle cells.

In the News & Comment article "Will Fermilab find its future by looking to the stars?" by Faye Flam (1 Jan., p. 24), two participants in the Sloan Digital Sky Survey, Johns Hopkins University and the Institute for Advanced Study, were inadvertently omitted.

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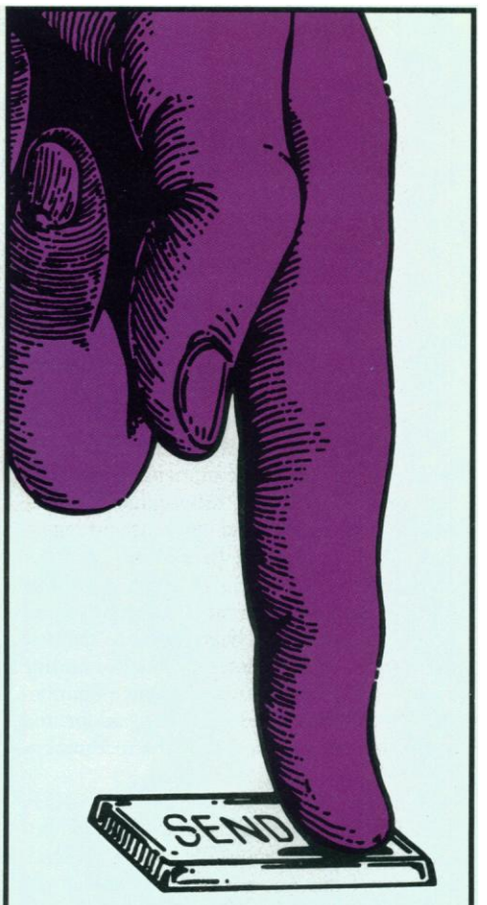

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