gave up for reburial a skeleton dated by radiocarbon at 10,600 years old, possibly the oldest directly dated human remains in the New World.

Our government officials, having been told to lay off the regulation of commercial interests for a while, have discovered a fertile new field of political activity in regulating all aspects of scientific study. The burden of this falls mostly on legitimate scholars and institutions because they are easily identified. Even if the conviction and penalty are dismissed, those caught in this trap will have to pay for attorneys to defend themselves. There is the additional cost to the taxpayers of putting these ridiculous cases into the court system. Scholars should be very worried—it is a short step from what is happening now to the point where a government office will decree what can be studied, who can study it, and what will happen to the scientific evidence.

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Super Collider, 2000 B.C.

In his letter of 30 October, "The pre-druid Super Collider?" (p. 725), Leon Lederman forgets to mention another potential similarity between Stonehenge and the Superconducting Super Collider. After 4000 years it is still not certain that Stonehenge ever had any scientific value.

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Lederman may be trying to argue that we should build the Superconducting Super Collider just as the old Britons built Stonehenge, but his analogy suggests the very opposite. He says that Stonehenge was completed on schedule in 2000 B.C. Glyn Daniel discussed (*1*) the chronological details of Stonehenge: The first phase was from 2800 to 2200 B.C., the second was from 2100 to 2000 B.C., and the third was from 2000 to 1100 B.C. If the Super Collider is our Stonehenge, can we expect it to be completed in 3692 A.D.?

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Lederman is apparently a great believer in the economic wisdom of the ancients, to judge from his estimate of prehistoric inflation and cost accounting at Stonehenge. The estimated cost of the Super Collider is $8 billion. If the cost of Stonehenge were adjusted for inflation by even as little as 1% per year, a present value of $8 billion would imply an original cost of $0.000000045.

Let the new Administration in Washington note: The secret of keeping inflation down while rebuilding our nation's infrastructure lies not in Reaganomics or Clintonomics, but in the pre-druid past!

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Pitohui: How Toxic and to Whom?

I read the report "Homobatrachotoxin in the genus *Pitohui*: chemical defense in birds?" by John P. Dumbacher *et al.* (30 Oct., p. 799) with great excitement. As with many important findings, the defensive chemical was discovered serendipitously. While holding individual hooded pitohuis (*Pitohui dichrous*), the authors appear to have picked up the chemical on their hands, and touching their hands to oral and nasal epithelium, they experienced "numbness, burning, and sneezing." Unfortunately, the bioassay used in the study to further explore the defensive value of homobatrachotoxin is ecologically irrelevant. In brief, subcutaneous injections of either homobatrachotoxin or crude homobatrachotoxin-containing extracts from different body parts were made into the hindquarters of laboratory mice. These injections produced convulsions and death in many of the mice. I have three concerns with this bioassay.

First, why was this chemical applied to the hindquarters rather than the oral cavity? If natural predators respond orally to homobatrachotoxin like humans do, then the irritating oral effect alone would effectively deter many predators. Second, in the event that a predator actually swallowed pitohui tissue, the homobatrachotoxin would have to cross the gastrointestinal wall, which is a major barrier to absorption for many xenobiotics. For example, laboratory mice are substantially more sensitive to subcutaneous (and intraperitoneal) doses than they are to oral doses of many drugs and poisons (*1*). To rationalize the use of subcutaneous injections, the authors need to demonstrate that homobatrachotoxin readily crosses the gastrointestinal wall. Third, why was the laboratory mouse used as the test species? The authors state that the most likely natural predators of pitohuis are "snakes, raptors, and potentially some arboreal marsupials." Given this assemblage of predators, a placental mammal seems to be an inappropriate model. There are large species differences in sensitivity to poisons, even among placental mammals (*1*). Despite the fact that batrachotoxin, which is structurally related to homobatrachotoxin, polarizes nerve and muscle cells in several species of mammal and a mollusk, the key issue is whether the concentration of homobatrachotoxin is high enough to repel the pitohui's natural predators during an attack. I suggest that until a more ecologically relevant bioassay is employed, the jury is still out on the defensive function of pitohui homobatrachotoxin.

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Response: It was not our intention to use the mouse bioassay "to further explore the defensive value of homobatrachotoxin." Instead, we used it in the process of purifying the toxic substance from the skin and feathers of hooded pitohuis. This led to its identification as homobatrachotoxin, a neurotoxin previously thought to be made only by poison-dart frogs in tropical South America. The mouse bioassay also provided a quantitative measure of concentrations of toxin between different tissues and between different species of pitohuis, as it had in earlier studies of poison-dart frogs.

Birds avoided by predators or having unpalatable flesh have been described in the literature (*1*) and have been suggested as plausible examples of chemical defense in birds. These suggestions were based either on "bioassays" with hornets, domestic cats, or humans or on ancedotal accounts. No previous study has identified any toxic substances or performed repeatable experiments showing that a live bird could repel potential predators. Instead, it has been suggested that diet (fish, decaying flesh, or insects) contributed to making the bird flesh distasteful. Our study demonstrates a toxin in the *skin* and *feathers* of pitohuis, external tissues well suited for chemical defense. Additionally, batrachotoxins, including homobatrachotoxin, appear to serve as a chemical defense in *Phyllobates* frogs (*2*). Pitohuis are defended from human hunters in many areas of New Guinea; they recognize pitohuis as undesirable and do not hunt them.

We agree with Glendinning that field studies with potential predators are needed to demonstrate the defensive value of homobatrachotoxin. We are also aware that

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.