

Letters to the Editor

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Spain's Earth Scientists and the Oil Spill

THE SPANISH COAST OF GALICIA IS CURRENTLY subject to an oil spill that, given its spatial and temporal extent, could become one of the worst spills ever. The Spanish and the local Galician governments have been maintaining for 30 days that, since 13 November, when the tanker *Prestige* ran into problems, every decision implemented throughout this crisis, including the key resolution of transporting the vessel offshore, was guided by the technical advice of Spanish experts. In our opinion, the recurrence and indiscriminate generalization implicit in such a statement entail a serious threat to the credibility of the Spanish Earth sciences community as a whole. Moreover, this crisis is revealing a serious malfunctioning of the national research system. This moves us, as marine and atmospheric scientists, and members of the Spanish Institute of Oceanography (IEO), National Research Council (CSIC), universities, and other research centers, to express the following:

1) Given the well-known winter climatology of the area of the spill, dominated by west-southwesterly winds and a south to north slope current on the sea (*I-II*), the decision to move the vessel from about 43°N, 9.5°W offshore to the southwest was a consequence of poor communication between the government officials dealing with the spill and the scientific and technical communities, rather than a deficit of knowledge. This move was responsible for the spreading (spatial amplification) of the spill, which now extends across about 900 km of shoreline. The position of the sunken ship at about 42°N, 12°W, 145 nautical miles off the south coast of Galicia will probably cause successive oil waves (temporal amplification) to arrive at the Spanish, Portuguese, and/or French coasts. Thirty days after the first spill (*I2*), the Galician coast now faces a third, and possibly not the last, oil wave.

2) Once the oil spill had occurred, the poor coordination of the Spanish authorities has led to a very ineffective use of scientific institutions, resources, and knowledge, reflected in inexplicable delays and overlapping actions. For example, the first draft of a scientific action plan is dated 13 December, 1 month after the beginning of the crisis and 4 days after the first scientific commission was convened.

3) We demand that the Spanish authorities improve the mechanisms and logistics for scientific and technical consultation and refrain from making vague public statements that are seriously, and unfairly, damaging the image of Spanish marine and atmospheric sciences.

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A man walks along an oil-covered beach in Caion, northern Spain, 18 November 2002.

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- This letter was written on 21 December 2002.

Revisiting Coauthor Responsibility

MICHAEL PHILLIPS' CONTENTION THAT CO-authors of fraudulent papers who signed those papers in the belief that the data were honest are themselves victims ("Should coauthors share liability?" Letters, 22 Nov., p. 1554) is a disturbing proposition that one would hope is not widely shared. It is unfortunately saddening rather than shocking that some would hew to such an idea. Certainly, if the scientific community is to retain and warrant the esteem of the public, then the notion that coauthors do not have sufficient knowledge of the details of the study they purport to have participated in to vouch for its integrity simply does not wash. As a practical matter, mandating such responsibility would service two useful purposes: (i) to curb freeloading on papers by individuals with only marginal involvement and (ii) to provide a first line of defense against fraud. For those who do not wish or have the time for this level of involvement, there is a long-standing tradition of acknowledgment short of authorship.

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The Next Generation of Science Policy-Makers

DONALD KENNEDY ("AFTERMATHS," Editorial, 29 Nov., p. 1679) is wise to advocate new means of encouraging science and technology training for future policy-makers in the United States. The National Defense Education Act is an informative model, not least for its expansive view of the critical skills the United States would require. By focusing on educating the next generation of public servants, however, Kennedy addresses only a part of the United States' intellectual deficit and only part of our opportunity.

In an era of dazzling opportunities across many scientific disciplines, our country has been disinvesting (*I*). Federally supported R&D shrank in 2000 to less than 0.7% of the gross domestic product—a level last seen before the Soviets launched Sputnik. A laudable bipartisan effort to double the NIH budget has nourished biomedical research and training, but a steady attrition of investment has weakened the physical sciences and engineering. The Department of Energy's funding for the physical sciences has decreased by 20% since 1993. The number of graduate students has dropped

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proportionately, with U.S. students minorities in many departments.

While continuing to welcome foreign students and sustaining our enormously beneficial position as graduate school to the world, we must take new steps to encourage and support the advanced graduate studies of U.S. citizens in science and engineering. An ambitious new program of graduate fellowships—let us call them Benjamin Franklin fellowships—would show students that our country values science and technology and would spur them toward creative public service. Graduate students we attract during this decade will help shape the world for half a century, so it would be shortsighted to target a few specialties. It would be better by far to attract more of our best students to the most interesting sciences and to inspire them, like Benjamin Franklin, to range over pure and applied science, engineering, and even statecraft!

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1. "Federal Investment in R&D," a project memorandum prepared for the President's Council of Advisors on Science and Technology by the RAND Science and Technology Policy Institute and the AAAS (see www.rand.org/publications/MR/MR1639.0/).

Heat and Biodiversity

SINCE THE TIME OF DARWIN, THE LATITUDINAL gradient of increasing species diversity from the poles to the equator has perplexed biologists and shaped ecological and evolutionary theories. A. P. Allen *et al.* present a model intended to explain this pattern ("Global biodiversity, biochemical kinetics, and the energetic-equivalence rule," Reports, 30 Aug., p. 1545). Their model is a formalization of the "species-energy hypothesis" and predicts that "biodiversity is positively correlated with productivity because more productive environments contain more individuals and can therefore support more species populations above some minimum size required for persistence" (p. 1547). The model predictions are consistent with patterns of increasing species number with increasing mean air or water temperatures for trees, amphibians, marine gastropods, fish, and fish parasites.

In spite of its intuitive appeal, this model suffers from the two fundamental flaws of the species-energy hypothesis. First, "environmental energy" (in this model, mean temperature) does not correspond to the energy actually available to organisms, which is the energy stored in carbon compounds produced by photosynthesis. Although it is true that the tropics tend to be warmer than the temperate zone, higher

temperatures do not necessarily result in higher productivity of plants or animals. The most extensive data set on the net primary productivity of plants compiled to date reveals that the mean annual productivity of tropical forests is the same as that of temperate forests (1). Marine productivity is much higher in the cold high-latitude oceans, where the world's great fisheries are located, than in the warmer tropics (2).

Second, many of Earth's highest diversity areas have low productivity (3). Examples include the mediterranean climate shrublands of South Africa and Australia, which occur on poor soils with low primary productivity; the diversity of bird species with small range sizes in Africa, which is unrelated to net primary productivity (4); and the diversity of aquatic and marine phytoplankton, which is higher in unproductive, nutrient-poor environments than in productive environments. All of these patterns directly contradict the predictions of species-energy theory. Thus, it does not seem likely that a temperature-based species-energy model is the explanation for the latitudinal gradient of species diversity.

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Response

HUSTON SEEMS TO MISINTERPRET BOTH THE substance and the intent of our Report. He suggests that our model is some version of the long-standing species-energy hypothesis and then criticizes it for "suffer[ing] from the two fundamental flaws" of this hypothesis. Huston correctly points out that temperature "does not correspond to the energy actually available to organisms." Indeed, temperature indexes the average kinetic energy of molecules, not the potential for photons, organic compounds, and other energy and materials to be used by, and fluxed through, organisms and ecosystems. Temperature affects the rate of metabolism, but it is not the fuel for metabolism. Our paper argues that temperature affects biodiversity through its fundamental influence on the rates of biochemical reactions, whole-organism metabolism, and ecological interactions. The traditional species-energy hypothesis attributes species richness in large part to productivity, the rate of flux of biologically usable potential

energy. Biodiversity is almost certainly influenced by both kinetics and productivity, but they are not the same thing.

Huston is correct that in some cases, high species diversity occurs in cold or low-productivity environments. Temperature and productivity are often, but by no means always, correlated in nature, so it will be a challenge to understand their separate and interacting effects. Although the rate of biological production is powerfully constrained by temperature, it is also affected by other environmental variables—most notably by the supply of water and nutrients. The ability to predict the kinetic effects of temperature from a basic theoretical perspective should aid in understanding the other environmental factors and ecological processes that also affect biodiversity. Contrary to Huston's assertions, we do not claim that "a temperature-based species-energy model is the explanation for the latitudinal gradient of species diversity." We do claim that the fundamental effect of temperature on rates of biological metabolism and ecological interactions must be an important component of any theory to explain the latitudinal and other major patterns of species diversity.

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Gluten Peptides and Celiac Disease

IN THEIR REPORT "STRUCTURAL BASIS FOR gluten intolerance in Celiac Sprue" (27 Sept., p. 2275), L. Shan *et al.* describe a 33-mer gluten peptide that is resistant to degradation in the gastrointestinal tract and contains several T cell stimulatory epitopes. All Celiac disease (CD) patients tested made T cell responses to this 33-mer peptide. Homologs of the peptide are present in barley and rye, which are toxic to CD patients, but not in oats, rice, and maize, which are considered safe for patients. An enzyme is described that eliminates the T cell-stimulatory properties of the peptide. Although we acknowledge that this is an important step forward, we feel that it is an oversimplification of the problem. There are at least 15 T cell-stimulatory gluten peptides, and most of these are not found in the 33-mer peptide (1). In fact, in 50% of children with CD, we find no responses to sequences in the 33-mer peptide, but we do find responses to other gluten peptides (2). Also, the authors ignore our description of a T cell-stimulatory gluten peptide of which identical homologs exist in barley and rye, but not in oats. This peptide is also not found in the 33-mer

peptide (3). Evidently, responses to peptides other than the 33-mer can be linked to disease development and cereal toxicity.

Finally, we propose a word of caution regarding the "therapy." What is proposed is enzymatic destruction of the peptide, which may prove difficult because gluten is usually present in a food matrix, together with many other compounds. This severely complicates gluten detection and quantification in food, let alone enzymatic degradation and proof that all relevant gluten peptides have been degraded. Moreover, the authors incorrectly state that all gluten peptides described by us (2) will be destroyed by the enzyme. Consequently, this enzyme treatment will fail to remove toxicity completely.

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Response

WE THANK KONING AND VADER FOR THEIR comments. The existence of T cell epitopes outside the 33-mer peptide is explicitly acknowledged by us in Table 2 and in cited references, including a recent publication by the authors. A key point, not mentioned in their Letter, is the substantially enhanced potency of the 33-mer in eliciting T cell responses relative to individual epitopes present in most short peptides (e.g., see Fig. 3 and Table 2). Presumably, these quantitative differences arise because of the multivalency and proteolytic stability of the 33-mer. Therefore, it is inappropriate to conclude that, just because individual epitopes in the 33-mer do not stimulate patient-derived T cells, the 33-mer will also not do so. We are unaware of any published results from the authors in which side-by-side tests have been performed with the 33-mer and other gliadin peptides on T cell lines originally challenged with gluten. Moreover, in apparent contrast to the authors' findings, one of us (L.M.S.) has observed T cell responses to one or more epitopes found in the 33-mer in all adult patients tested so far ($n > 30$).

Koning and Vader also draw attention to the fact that other predictors of cereal toxicity have been proposed in the literature. We do not state or imply that the 33-mer sequence is the only known predictor of cereal toxicity. Our observation that homologs of the 33-mer are found in toxic but not nontoxic cereals was simply intended to rationalize its exceptional inflammatory character.

Finally, Koning and Vader argue that, given the diversity of known immunogenic epitopes in gluten and their assumptions regarding the dynamics of gluten release from food in the small intestine, the complementary action of endogenous proteases and an exogenous prolyl endopeptidase (PEP) “will fail” to detoxify gluten. To our knowledge, every intestinal T cell epitope identified from gluten to date contains one or more Pro residues. Bacterial PEPs have broad tolerance for proline-containing peptides. For example, although the *F. meningoscepticum* PEP (used in our study) prefers Pro[↓]X-Pro motifs, it can also cleave other Pro[↓]X-X motifs (1), especially in shorter peptides, such as most gluten peptides generated by the action of gastric and pancreatic proteases. Other homologous bacterial PEPs will undoubtedly have varying S₂, S₁′, and S₂′ subsite preferences, some of which may be even better suited for gluten detoxification than the *F. meningoscepticum* enzyme. The main conclusions from our study are that (i) the mammalian digestive apparatus is inca-

pable of effective proteolytic cleavage on the COOH-terminal side of internal Pro residues in dietary proteins; (ii) PEPs from other sources have this capability; and (iii) endogenous proteases and an exogenous PEP act in concert to dramatically accelerate the rate of digestion (and consequent detoxification) of Pro-rich dietary proteins such as gliadins. Of course, only a carefully planned clinical study will definitively answer the question of whether such detoxification can provide a Celiac Sprue patient some relief from the severe burden of a strict, lifelong gluten-free diet.

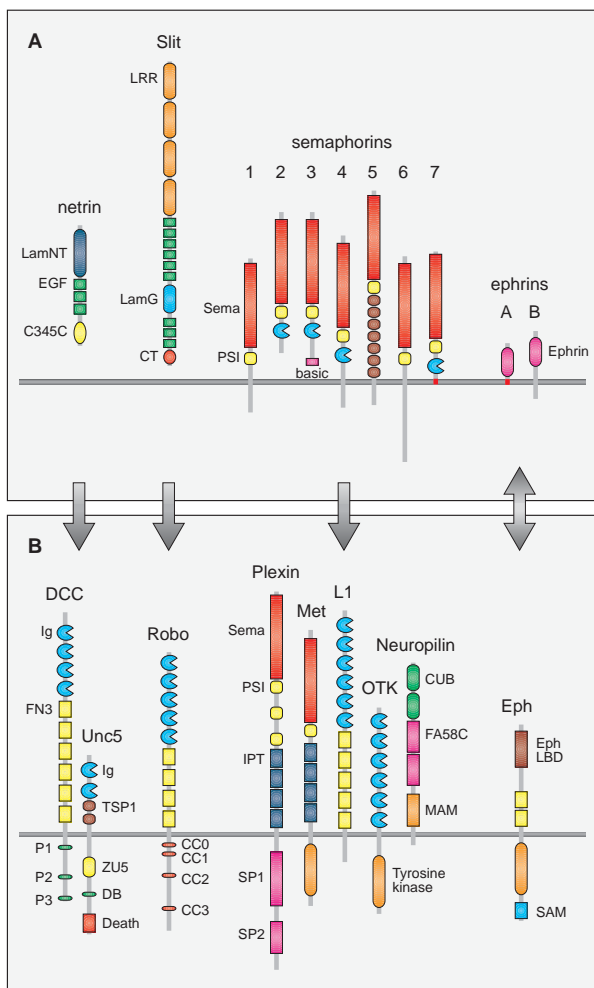
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CORRECTIONS AND CLARIFICATIONS



SPECIAL ISSUE ON POLARITY:

REVIEWS: “Molecular mechanisms of axon guidance” by B. J. Dixon (6 Dec., p. 1959). There were several labeling errors in Fig. 1A. The correct version of the figure appears to the left.

REPORTS: “Regulation of hypoxic death in *C. elegans* by the insulin/IGF receptor homolog DAF-2” by B. A. Scott *et al.* (28 June, p. 2388). One of the *daf-2* strains tested in the paper was incorrectly called *e979*. The strain referred to as *daf-2(e979)* has subsequently been discovered to be *daf-2(m41)*. The strain was obtained from the *Caenorhabditis* Genetics Center (CGC) as *e979*, but the error was on the part of other investigators who originally submitted the strain to the CGC. This discovery does not change any of the conclusions of the authors’ work. In particular, the lack of correlation of life-span and hypoxic death is still maintained ($r = 0.32$, $P = 0.36$). However, the Gly³⁸³ to Glu mutation assigned to the *e979* mutant should in fact be assigned to *m41*, as previously reported [H. Yu, P. L. Larsen, *J. Mol. Biol.* **314**, 1017 (2001)].