

## LETTERS

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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### Reference

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## 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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### References

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2. M. J. Behrenfeld *et al.*, *Science* **291**, 2594 (2001).
3. M. A. Huston, *Biological Diversity: The Coexistence of Species on Changing Landscapes* (Cambridge Univ. Press, Cambridge, 1994).
4. E. Ravasz, A. L. Somera, D. A. Mongru, Z. N. Oltvai, A.-L. Barabási, *Science* **297**, 1551 (2002).

## 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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3. W. Vader *et al.*, *J. Exp. Med.* **195**, 643 (2002).

## 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.