ic planning and priorities. These resources should not be treated as an entitlement. There needs to be more discussion regarding the challenges we face in evaluating this proposal; some of the basic issues about how this systemwide program must change to meet these challenges. None of the three campuses involved can assume that past history will dictate the use of these resources. In reality we must be prepared to develop new directions to meet the challenges within the university and to continue to meet our mission-oriented responsibilities in agricultural and environmental sciences for the state of California.

Barbara O. Schneeman
Dean, College of Agriculture and Environmental Sciences, University of California, Davis, CA 95616–8571, USA

I, and other colleagues, were disappointed by the one-sided article about the reorganization of the College of Natural Resources (CNR) at the University of California (UC), Berkeley. Vice President for Agriculture and Natural Resources Kenneth Farrell heads the three Agricultural Experiment Stations (AES) divisions in the UC system, but the funds are controlled by the UC Office of the President. The CNR appointments (full-time employees) vary, but are roughly 75% for AES research and 25% for instruction. Unfortunately, some UC Berkeley administrators and AES faculty appear to have forgotten this ratio and to have helped erode the AES research mission.

The article implies that dissenters were tools of agribusiness and that Farrell was opposed to the new ecosystems-environmental thrust, but it is safer to say that he was ignored by UC Berkeley, which is seeking to appropriate resources he controls for legally mandated AES responsibilities and to reallocate them for the development of biotechnology at Berkeley. Although biotechnology has made tremendous scientific breakthroughs and changes are needed in the AES, ravaging CNR for the "promise" of patent revenues will have important future costs. I can only hope that this new direction does not make the university a tool of the biotechnology industry and that the resolutions of important environmental issues are not subverted for the sake of biotech profits.

Last, the method of reorganization was anathema to Berkeley traditions of faculty self-governance; all authority has been centralizing in the CNR dean's office, and a top-down administration has been imposed.

Andrew Paul Gutierrez
Department of Environmental Science, Policy and Management, University of California, Berkeley, CA 94720, USA

I would like to comment on Barinaga's article "A bold new program at Berkeley runs into trouble." I am confident that an accurate analysis of the budgets of most land-grant universities would show that a substantial amount of funding has been refocused into high-profile–high-tech programs and away from traditional production-related agricultural research. However, this redistribution of funds has, in general, remained cloaked in the mantle of traditional agriculture (for example, filling a plant breeding position with a basic plant molecular biologist who works with *Arabadopsis*), so that the fiefdoms of the U.S. Department of Agriculture (USDA) and experiment station administrators are not disrupted. It appears that the Berkeley faculty are being punished for their direct frontal assault on the policy prerogatives of the old boy network that still controls the distribution of most USDA research funds.

Alan H. Goldstein
California State University, Los Angeles, CA 90032, USA

Response: Farrell's characterization of my article is far from the mark. Since he doesn't say what he thinks my "bias" is, I can't respond specifically, but in fact my article was not written out of any preconception about the situation at Berkeley. On the contrary, it was based on more than two dozen interviews—not only with Berkeley faculty, as Farrell seems to imply—but with faculty and administrators at UC Davis and other top land-grant colleges around the country, with members of an outside committee that reviewed Berkeley's program, and with the director of the Board on Agriculture at the National Research Council in Washington, D.C. The views of that large sample are accurately and fairly represented in the story. Indeed, one piece of evidence that my article was not biased is the fact that the point of view Farrell expresses in his letter was represented, both in his words and in those of a Berkeley professor, in the article.—***Marcia Barinaga***

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Blood Type and the Risk of Gastric Disease

In their 17 December report (p. 1892), T. Borén *et al.* (*1*) present compelling evidence that *Helicobacter pylori* did not bind to the Lewis^b (Le^b) blood group antigen in the presence of blood group A determinant, which suggests that individuals with blood type O may have increased *H. pylori* receptors, making them more susceptible to

duodenal ulcer, gastric ulcer, and gastric cancer. On the other hand, Borén *et al.* may go too far in stating that these three diseases are associated with blood group O. Several studies have shown that there is a 30% or 1.3-fold increased risk of developing a duodenal ulcer for individuals with blood group O as compared with blood groups A, B, and AB (*1–4*). There is a 50% or 1.5-fold increase risk of duodenal ulcer for individuals with nonsecretory blood group antigens (nonsecretors) (*2–4*). Individuals with both blood group O and nonsecretor status have a 150% or 2.5-fold increased risk of developing a duodenal ulcer (*2–4*). There are conflicting reports with regard to the association of blood group O and gastric ulcer, although the preponderance of evidence suggests no clear association (*2, 4*). Because the association of *H. pylori* with gastric ulcer is weaker than it is with duodenal ulcer (*5*), the argument that blood group O may be associated with both duodenal ulcer and *H. pylori* receptor binding is not diminished. Finally, even though there is growing epidemiologic evidence for an association between *H. pylori* and gastric cancer, factors other than the binding of *H. pylori* to Le^b antigen must be involved, as blood group A has been reported to be more prevalent in patients with gastric carcinoma (*6*) than blood group O.

Ann E. Hallstone
Gastrointestinal Section,
Veterans Administration Medical Center,
150 Muir Road (111 G),
Martinez, CA 94553, USA
Edith A. Perez
Hematology/Oncology Section,
University of California,
Davis, CA 95817, USA

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Response: The purpose of our investigation (*1*) was to identify host cell receptors for the gastric pathogen *H. pylori*, and we identified the Le^b and H blood group antigens as receptor structures for *H. pylori* adherence. The Le^b and H-1 antigens are carbohydrate structures typical for individuals of blood group O phenotype. Antigens of the blood groups A and B are made

byadding a terminal GalNAcα- or Galα- residue, respectively, to the H or Le^b antigens. We demonstrated that the A-Le^b antigen is not a receptor for *H. pylori*. However, differences in the activity or efficiency of the "substituting" transferase result in variation of substitution (A-Le^b antigen or B-Le^b antigen) between individuals and consequently in different amounts of available Le^b in individuals with blood groups A and B (*2*).

Individuals with blood group O run a higher risk of developing peptic-duodenal ulcers (*3*, *4*) and have a higher incidence of gastric ulcers (*5*). Taken together, the data suggest that the number of available receptors for *H. pylori* could be generally lower in individuals with blood groups A and B. Hallstone and Perez agree that individuals with blood group O run an approximate 1.5- to 2.0-fold increase of developing ulcers. Unfortunately, we used the term gastric ulcer instead of ulcer (*6*).

A reduced amount of Le^b antigen, that is, functional *H. pylori* receptors, could explain the higher incidence of ulcers in individuals with blood group O. However, the difference will not be absolute because of the presence of available Le^b in individuals with blood groups A and B and because of the influence of other suggested receptors such as sialic acid (*7*), phospholipids (*8*), and sulfatide (*9*). Furthermore, other factors, like the vacuolating cytotoxin produced by some *H. pylori* strains (*10*), will affect bacterial virulence.

It is, however, a possibility that the initial adherence and subsequent attachment of bacteria to the cell surfaces is such an important part of the pathogenic process that differences in numbers of available receptors will reflect the subsequent development of disease.

We agree with the comment by Hallstone and Perez that individuals with blood group O are not overrepresented in the population suffering from gastric carcinoma. In our introduction (*1*, p. 1892), we pointed out the intriguing data by D. Forman *et al.* (*11*), indicating that there seems to be a high correlation between the presence of *H. pylori* in gastric tissue and the prevalence of gastric carcinomas. Gastric carcinomas, however, have a multifactorial etiology (*12*) and are not only governed by bacterial adherence.

Thomas Borén
Department of Molecular Microbiology, Washington University School of Medicine, 660 South Euclid Avenue, St. Louis, MO 63110–1093, USA
Per Falk
Department of Molecular Biology and Pharmacology, Washington University School of Medicine

References and Notes

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Corrections and Clarifications

In the News & Comment article "Scientists return to the elementary-school classroom" by Joseph Alper (6 May, p. 768), the name of Jan Tuomi, director of outreach at the National Science Resources Center, was misspelled. Her proper e-mail address is "jtuomi@nas.edu"

The numbers in the y-axis of figure 2 (p. 227) in the article "Modulated magnetic phases in rare earth metallic systems" by T. Chattopadhyay (8 Apr., p. 226) were incorrect. They should have been 0.16, 0.18, 0.20, 0.22, 0.24, 0.26, and 0.28, respectively.

In the News & Comment article "Report calls for smaller clinical center" by Jon Cohen (25 Mar., p. 1678), the name of Paul Marks of Memorial Sloan-Kettering Cancer Center in New York City was misspelled.

In the Research News article "Is marine biodiversity at risk?" by Elizabeth Culotta (18 Feb., p. 918), the two stomatopods in the lower picture on page 919 were misidentified. *Haptosquilla stoliurus* is on the right and *Gonodactylus viridis* is at left.

Figure 2C (p. 827) of the Report "Prevention of vertebrate neuronal death by the *crmA* gene" by V. Gagliardini *et al.* (11 Feb., p. 826) was incorrectly printed. The correctly printed figure is shown below.