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.

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6. On page 1895 of our report, last paragraph, line 17, we were discussing a paper by C. A. Clarke *et al.* [reference (*4*) above], page 1895, first column, third paragraph, lane 17.
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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.