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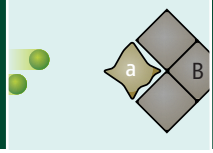

LETTERS

edited by Etta Kavanagh

Finding Good in the Bad and Vice Versa

DONALD KENNEDY'S EDITORIAL "GOOD NEWS—AND BAD" (13 JAN., P. 145) on the intelligent design Dover, Pennsylvania, court decision and the South Korean stem cell scandal overlooks some of the bad news in the good and the good news in the bad. The fraud perpetrated by at least some members of Hwang's research group is indeed bad news. But the good news is that the fraud was caught very soon after publication, and it is being dealt with. This is an example of science working, largely by reason of its openness and institutionalized commitment to testing and debate. Compare this scandal to deception in the business and political realms. Enron's fraud was hidden for years. Abramoff and DeLay similarly operated for years before indictment. Indeed, even supposedly democratic governments classify their fraudulent or other illegal actions, using the cloak of national security, and crimes can remain buried for decades. There is good reason to resist efforts to increase secrecy in any realm of human endeavor. With secrecy, the tail ends up wagging the dog and corruption becomes a way of life.

There is no question that the court decision in the Dover case was a good one. The opinion written by Judge Jones is rigorous and thorough—and yes, quite elegant. There is a substantial dark side to this decision, however, that reflects poorly on the scientific community. How did it come to this court fight? How, in a country as "developed" as the United States, have the school system, the media, and the scientific community failed so miserably to educate the majority of Americans about the nature of science in general and evolution in particular? Too often, scientists do not take their public role seriously enough. If scientists do not respond aggressively to the need to bring the rest of society along and confine themselves to talking to each other, the scientific enterprise will likely find itself uncomfortably out on a limb. Should the United States continue to drift closer to the world's theocracies and away from the preferable if very flawed secular democracies, science and scientists will suffer.

"How, in a country as 'developed' as the United States, have the school system, the media, and **the scientific community failed so miserably to educate** the majority of Americans about the nature of science in general and evolution in particular?"

—Johns

DAVID JOHNS

School of Government, Portland State University, Post Office Box 751, Portland, OR 97207, USA. E-mail: johnsd@pdx.edu

Diversity in Tropical Forests

IN HER ARTICLE "RARE TREE SPECIES THRIVE IN local neighborhoods" (News of the Week, 27 Jan., p. 452) discussing a study by C. Wills *et al.* ("Nonrandom processes maintain diversity in tropical forests," Reports, 27 Jan., p. 527), E. Pennisi states that "Biodiversity may be threatened worldwide, but small pockets of tropical-forest trees are surprisingly becoming more diverse over time." However, the key implication of Wills *et al.*'s study is that, in several tropical forests on two different continents, strong density- and frequency-dependent mortality tends to favor rare over common tree species locally (at a scale of tens to hundreds of square meters), and that this is a key process that helps to maintain (but not increase) tree diversity at a forest-wide scale.

Hence, Wills *et al.* have helped to reveal the mechanisms by which tropical forests maintain their extraordinary biological diversity, but there is nothing in their study to suggest that the number of species in these forests is somehow increasing.

WILLIAM F. LAURANCE

Smithsonian Tropical Research Institute, Apartado 2072, Balboa, Republic of Panama. E-mail: laurancew@si.edu

Genetic Polymorphism of Fc

J. M. WOOF'S PERSPECTIVE "TIPPING THE SCALES toward more effective antibodies" (2 Dec. 2005, p. 1442) presents a good commentary on how Fcγ receptors (FcγR) could contribute to the observed variation in immunoglobulin G (IgG) subclass responses to pathogens and tumor antigens. The author briefly points out the importance of FcγR gene polymorphism in differential binding to human IgG subclasses. I would like to add that the genetic variation in the Fc domain might also contribute to the effector functions of the IgG molecules. Surprisingly, virtually all studies—whether involving interaction of Fc and FcγR or engineering mouse-human chimeric antibodies for immunotherapy—have treated the Fc region as if it were monomorphic. The Fc region of IgG (both human and mice) is not monomorphic. For instance, Fc regions of γ1, γ2, and γ3 chains possess polymorphic determinants (called GM allotypes) coded by genes on chromosome 14 in humans (*1*). It is possible that particular FcγR and Fc (GM) alleles epistatically interact and contribute to specific effector responses mediated by IgG molecules, providing a mechanistic explanation for numerous associations observed between various FcγR and GM alleles and immunity to infectious, autoimmune, and malignant diseases. This would be analogous to the reported interaction between HLA and NK cell inhibitory receptor genes in the resolution of hepatitis C virus infection (*2*).

JANARDAN P. PANDEY

Department of Microbiology and Immunology, Medical University of South Carolina, 171 Ashley Avenue, Charleston, SC 29425, USA.

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