

Thinking about
reasoning

1794

What happens
in classrooms?

1795



LETTERS | BOOKS | POLICY FORUM | EDUCATION FORUM | PERSPECTIVES

LETTERS

edited by Etta Kavanagh

Wildlife Population Increases in
Serengeti National Park

IN THEIR BREVIA "EFFECTIVE ENFORCEMENT IN A CONSERVATION area" (24 Nov. 2006, p. 1266), R. Hillborn *et al.* report that antipoaching efforts led to increased wildlife populations in the Serengeti National Park. Although these results are compelling, we are not convinced that the documented population increases are a result of these efforts.

First, the data presented demonstrate correlation, but not causation. There may be other reasons for the population recovery, but the authors did not consider alternative hypotheses. Documented increases in buffalo numbers may be explained as a recovery to postdrought conditions rather than the results of antipoaching efforts. This is a plausible alternative hypothesis, because large herbivore populations can be regu-

lated by food availability (1–3) and experience higher rates of predation during drought periods (2, 4).

Second, the authors show an exponential increase in the number of patrols per day after 1993, but it is unclear what level of patrolling is necessary to realize population recovery. For example, between 1985 and 1993, the buffalo population increased with number of patrols. However, the number of patrols during this period was much lower than during other periods of population increase. It is important to identify how much patrolling is actually needed to minimize poaching such that limited conservation funds are used efficiently.

Finally, the authors argue that community-based conservation could not explain the declines in poaching because the populations were already increasing before community-based programs were in place. Yet community-driven management programs adjoining the park are important for wildlife populations to thrive (5). It is possible that an alternative factor, such as postdrought recovery, could have been the impetus for population recovery and that community-based conservation programs then led to a reduction in poaching.

JULIE K. YOUNG, LEAH R. GERBER, CATERINA D'AGROSA

Department of Ecology, Evolution and Environmental Science, School of Life Sciences, Arizona State University, Box 874501, Tempe, AZ 85287–4501, USA.

References

1. N. Georgiadis, M. Hack, K. Turpin, *J. Appl. Ecol.* **40**, 125 (2003).
2. P. J. Funston, M. G. L. Mis, *S. Afr. J. Wildl. Res.* **36**, 9 (2006).
3. M. G. L. Mills, H. C. Biggs, I. J. Whyte, *Wildl. Res.* **22**, 75 (1995).
4. A. J. Loveridge, J. E. Hunt, F. Murindagomo, D. W. Macdonald, *J. Zool.* **270**, 523 (2006).
5. S. Thirgood *et al.*, *Ann. Conserv.* **7**, 113 (2004).



Elephants in Serengeti National Park, Tanzania.

Response

YOUNG *ET AL.* ARGUE THAT CHANGES IN THE intensity of poaching are only one possible explanation for the change in abundance of elephants, buffalo, and rhino in the Serengeti park. They do not appear to question that poaching rates increased dramatically after 1977, when enforcement was reduced. We agree that there are other possible explanations, but within the strict word limit for Brevia, we had little scope for discussion of these factors. In particular, we suspect that the reduction in world price for elephant ivory and rhino horn due to the CITES (Convention on International Trade in Endangered Species of Fauna and Flora) bans contributed to making poaching on these species less profitable. Further, we agree that changes in rainfall can

influence year-to-year abundance, as seen by the 1993 drought's impact on buffalo.

However, we formulated a hypothesis about the time trend in poaching intensity from the history of arrests and antipoaching efforts and then tested that hypothesis using the trends in abundance of the three species. This is a reasonably strong test of hypothesis, but clearly not totally definitive. The evidence that poaching pressure in the 1990s is considerably less than in the 1980s is very strong. The annual mortality rates after 1977 from poaching using abundance data alone were 58% for rhinos, 30% for elephants, and 15% for buffalo. These populations could not have recovered if these levels of poaching had continued.

Thus, the only significant question is what caused the dramatic decrease in poaching by

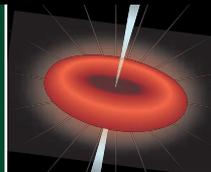
1990. There were no significant community development programs in place until the late 1990s, and community development programs simply cannot explain why poaching had declined by 1990. Young *et al.* argue that the increase in buffalo after the drought could be due to density-dependent factors, ignoring the fact that poaching rates must have been much lower than the 8% maximum rate of increase observed in the 1960s and 1970s. Density dependence does not explain why poaching rates declined so much. Although a multitude of factors, including antipoaching efforts, price of ivory and rhino horn, rainfall, community development projects, and local villagers' cash income and demands for cash, all undoubtedly contribute to poaching and changes in population abundance, the data we present

CREDIT: HAL BERAL/CORBIS



Origin of domestication

1797



Stellar fireworks

1798

provide undeniable evidence that the poaching mortality rates both increased and decreased, and the timing of these increases and decreases is best explained by the changes in antipoaching efforts.

RAY HILBORN,¹ GRANT HOPCRAFT,^{2,3}
PETER ARCESE⁴

¹School of Aquatic and Fishery Sciences, Box 355020, University of Washington, Seattle, WA 98195, USA. ²Frankfurt Zoological Society, Post Office Box 14935, Arusha, Tanzania. ³Community and Conservation Ecology, University of Groningen, Post Office Box 14, 9750AA, Haren, Netherlands. ⁴Center for Applied Conservation Research, University of British Columbia, Vancouver, BC V1T 1Z4, Canada.

HIV-Malaria Interactions: Don't Forget the Drugs

THE ADVERSE EFFECT OF CO-INFECTION WITH HIV and malaria is becoming increasingly apparent. The importance of these interactions is illustrated by the mathematical modeling of L. J. Abu-Raddad *et al.* ("Dual infection with HIV and malaria fuels the spread of both diseases in sub-Saharan Africa," 8 Dec. 2006, p. 1603), which predicts significant increases in the prevalence of both diseases due to an interaction between them. Theoretical models suggested that the effect of antimalarial chemotherapy on co-infected individuals would be a decline in both HIV and malaria prevalence. These findings assume that the effect of antimalarial chemotherapy on HIV infection is a shorter duration of raised HIV viral load after malaria infection. Although this is an important consideration, a number of studies have demonstrated direct effects of antimalarial drugs on HIV replication (1) and inhibition of *Plasmodium falciparum* development by HIV protease inhibitors (PIs) (2–4). Some HIV PIs act synergistically in vitro in combination with antimalarials (5). These direct drug effects have the potential to alter the complex interaction between malaria and HIV in co-infected individuals. Although HIV PIs are not currently recommended for first-line antiretroviral (ARV) therapy, they are likely to assume a greater role in ARV therapy in malaria-endemic regions as thermostable formulations of PIs are made available at significantly reduced cost (6), and as the need to

combat ARV-induced drug resistance increases (7). We endorse the view of Abu-Raddad *et al.* that further studies are required to explore these interactions, particularly with respect to the effect of interventions with potential efficacy against both pathogens.

KATHERINE T. ANDREWS, MICHELLE L. GATTON,
TINA S. SKINNER-ADAMS, JAMES S. MCCARTHY,
DONALD L. GARDINER

Queensland Institute of Medical Research, Queensland 4029, Australia.

References

1. A. Savarino, J. R. Boelaert, A. Cassone, G. Majori, R. Cauda, *Lancet Infect. Dis.* **3**, 722 (2003).
2. T. S. Skinner-Adams, J. S. McCarthy, D. L. Gardiner, P. M. Hilton, K. T. Andrews, *J. Infect. Dis.* **190**, 1998 (2004).
3. K. T. Andrews *et al.*, *Antimicrob. Agents Chemother.* **50**, 639 (2006).
4. S. Parikh *et al.*, *Antimicrob. Agents Chemother.* **49**, 2983 (2005).
5. T. S. Skinner-Adams, K. T. Andrews, L. Melville, J. McCarthy, D. L. Gardiner, *Antimicrob. Agents Chemother.* **51**, 759 (2007).
6. See "Abbott statement regarding new initiatives to expand access and affordability to lopinavir/ritonavir in the developing world," 14 Feb. 2007 (available at www.abbott.com/global/url/pressRelease/en_US/60.5:5/P_ress_Release_0341.htm).
7. S. H. Eshleman *et al.*, *J. Infect. Dis.* **192**, 30 (2005).

Response

ANDREWS *ET AL.* INDICATE THOUGHTFUL AND important considerations regarding interventions targeting HIV and malaria and their interactions. Indeed, we only considered one effect of chemotherapy: that of reducing the malaria infectious period and the duration of heightened HIV viral load. We concur with Andrews *et al.* that detailed modeling studies of the impact of single and synergistic interventions warrant further consideration, such as highly active antiretroviral therapy (HAART) (1, 2), HIV protease inhibitors (3), malaria prophylaxis (2), antimalarials (4), and insecticide-

Letters to the Editor

Letters (~300 words) discuss material published in *Science* in the previous 3 months or issues of general interest. They can be submitted through the Web (www.submit2science.org) or by regular mail (1200 New York Ave., NW, Washington, DC 20005, USA). Letters are not acknowledged upon receipt, nor are authors generally consulted before publication. Whether published in full or in part, letters are subject to editing for clarity and space.

impregnated bednets (2), in addition to behavioral interventions. This issue assumes particular importance with the expansion of HAART in sub-Saharan Africa as combinations of therapy may become logistically feasible. Moreover, there is evidence for dual beneficial effects of a number of antivirals and antimalarials (2–5). Finally, data on the biological effects of therapy combinations at the individual level would be of great utility to explore the epidemiological and population level consequences of intervention efforts.

LAITH J. ABU-RADDAD,^{1,2} PADMAJA PATNAIK,³
JAMES G. KUBLIN⁴

¹Statistical Center for HIV/AIDS Research and Prevention, Fred Hutchinson Cancer Research Center, Seattle, WA 98109, USA. ²Center for Studies in Demography and Ecology, University of Washington, Seattle, WA 98195, USA. ³Department of Epidemiology, School of Public Health, University of North Carolina at Chapel Hill, NC 27599, USA. ⁴International Health Program, University of Washington, Seattle, WA 98109, USA.

References

1. J. S. Montaner *et al.*, *Lancet* **368**, 531 (2006).
2. J. Mermin *et al.*, *Lancet* **367**, 1256 (2006).
3. K. T. Andrews *et al.*, *Antimicrob. Agents Chemother.* **50**, 639 (2006).
4. A. Savarino, J. R. Boelaert, A. Cassone, G. Majori, R. Cauda, *Lancet Infect. Dis.* **3**, 722 (2003).
5. S. Parikh *et al.*, *Antimicrob. Agents Chemother.* **49**, 2983 (2005).

Coal-Fired Power Plants: Imprudent Investments?

GRANGER MORGAN'S EDITORIAL "DON'T grandfather coal plants" (17 Nov. 2006, p. 1049) wisely suggests not "grandfathering" (i.e., not exempting from regulations) carbon emissions from coal-fired power plants. This is not just a matter of good policy, but it is also sensible in light of a widespread and longstanding principle of utility law.

In most of the United States, state public utilities commissions decide whether costs incurred by utilities can be passed along to ratepayers or whether they will be borne by investors. For decades, commissions have based their decisions on the prudence and usefulness of decisions to build or run power plants and negotiate power contracts. A prudence review occurs after the fact, but seeks to take into account the information available at the time of the action taken. It "determines whether a utility's management decisions... were reasonable in light of all the circumstances that existed at the time the actions in question were taken" and then decides whether costs should be allowed in rates (1). In a highly relevant example, many utility commissions ordered major disallowances of nuclear-plant investments, years after allowing the initial construction, and the United States Supreme

Court rejected investors' efforts to overturn those regulatory decisions (2).

When utilities calculate the life-cycle risks involved in constructing a new coal-fired power plant, the likelihood of federal carbon dioxide regulation is already clearly foreseeable. Thus, as the Coalition for Environmentally Responsible Economies points out, wise investors are already demanding that corporations calculate and inform potential investors about the costs of carbon regulations (3). Morgan's Editorial is only one of many indicia that those future liabilities are currently "foreseeable." Imagine utility investors ignoring this possibility, investing in coal technology that does not allow carbon control, and later requesting a rate increase when forced to retrofit or retire the plant. Public utility commissions could well find such decisions imprudent. That would result in the utility's investors footing the bill for expensive retrofits or even more expensive stranded costs (costs that investors cannot recover either from markets or from ratepayers).

Legislators may or may not explicitly forbid grandfathering, but, regardless of that, investors should recognize that utilities that

rush to add coal-fired capacity may face not only future compliance costs, but also a reality in which such imprudent costs are paid by investors, and not by ratepayers.

MICHAEL DWORKIN, SHANNA VALE,
ELLEN CRIVELLA

The Institute for Energy and the Environment at Vermont Law School, South Royalton, VT 05068, USA.

References

1. Vermont Public Service Board, Docket No. 5983 (1998), and, more generally, "Abandoned Nuclear Plant Recovery," 83 ALR 4th 183 (1991).
2. See, e.g., *Duquesne Light Co. v. Barasch*, 488 U.S. 299 (1989).
3. Coalition for Environmentally Responsible Economies (CERES), "Best Practices in Climate Change Risk Analysis for the Electric Power Sector" (CERES, Boston, MA, Oct. 2006), p. 22.

Response

IN MY EDITORIAL, I SUGGESTED THAT ONE approach to emission constraints would be to mandate controls only on plants constructed after carbon regulations go into effect "while exempting existing plants for some extended period on the grounds that firms would otherwise face large 'stranded costs.'" I suggested that this might be a factor in the current rush to build new conventional coal plants and

noted that "[s]ome investors may be counting on this or on the hope that such costs could be passed on to electricity rate payers." In concluding, I observed that while "[a] state-by-state approach is not optimal," it could be undertaken in such a way as to "place future liability on investors, not rate-payers, and thus send a clear message to those planning new plants..."

In their Letter, Dworkin (who is the former Chairman of the Vermont Public Service Board and one of the United States' leading thinkers on utility regulation) and co-authors persuasively elaborate this argument. The message is clear. Unless investors are confident that they will face sympathetic politically appointed state regulators for decades to come, they run a considerable financial risk when they choose today to build a conventional coal plant in the face of what is now compelling evidence of the need to limit future emissions of carbon dioxide, and with technical options, now available, that could control emissions.

M. GRANGER MORGAN

Department of Engineering and Public Policy, Carnegie Mellon University, Pittsburgh, PA 15213-3890, USA.

IBC's 2nd Annual International Conference and Exhibition

Drug Discovery and Development Partnering, Licensing and R&D Innovation Summit

Building Your Drug Candidate Pipeline through Global Alliances, Compound Acquisitions and Innovative R&D

April 25-27, 2007 • Tower Hall Funabori • Tokyo, Japan

The ONLY international conference in Japan providing multiple speaker case studies of successful international alliances and R&D strategies PLUS themed networking events to help you find partners and meet new companies from Japan, USA, Europe and Asia.

250+ attended
our 2006 event!

- Benefit from Case Studies on How to Successfully Bring Your Products to Market in Japan and Globally
- Examine Japan's Industry Evolution and Marketing/Partnering Opportunities to Enhance Your Global Strategies
- Meet New Companies at the Drug Candidate/Technology Showcase and Alliance Strategy Session
- Learn How to "Globalize" Your R&D Efforts in Asia and Around the World
- Find Partners in Themed Sessions on Antibodies/Biologics, RNAi and PGx

In Association with:



Produced by the Organizers of:



www.IBCLifeSciences.com/Japan

Tel: (+65) 6835-5136 • Fax: (+65) 6733-5087 • E-mail: enquiry@ibcasia.com.sg