The Future of Farming and Conservation

The Research Article by R. E. Green et al.

“Farming and the fate of wild nature” (28 Jan., p. 550) adds to the already burgeoning literature on agroecosystems and conservation. The authors are to be commended for their attempt to develop a model that could be of use in decision-making about agricultural development and wildlife conservation. Unfortunately, this work contains some critical errors, conceptual flaws, and missing literature that invalidate its conclusions. We list here a few such problems.

1) The assertion is made that existing agroenvironmental schemes depend on farmers receiving large amounts of financial compensation from the government. Although a popular vision in Europe, this is not true throughout the world. A growing number of wildlife-friendly schemes rely on premium prices paid by consumers for environmental services (e.g., organic certification or the Smithsonian Bird Friendly Certification for coffee). This also brings up an issue of which the authors seem unaware, the hidden costs of conventional intensive agriculture, which have been widely exposed and documented over the past 20 years (1–5).

2) Given the extensive literature on the effects of pesticides and fertilizers on non-target organisms and wildlife outside farming areas (6–8), ignoring this in the construction of the model is not justified and could be very misleading to policy-makers.

3) The unwarranted assumption is made that wildlife-friendly agriculture (which includes a wide variety of practices and techniques) reduces yield. However, there is accumulating evidence that organic farming can produce as much, if not more in some cases, as conventional agriculture (9, 10).

4) Most conservation biologists have gone beyond the simplistic idea that there is “wild habitat” and “agricultural land.” Most land is subjected to some sort of human interference, and the goal for conservation is to preserve as much biodiversity as possible in landscapes that include mosaics of different types of land use.

5) Almost all landscapes are currently fragmented, with patches of more or less native vegetation interspersed among a matrix of different land-use systems, including agriculture. Metapopulation and metacommunity structures are likely to exist among those patches (11–13) and organisms need to migrate from patch to patch to maintain these structures. If the so-called high-productivity agriculture includes monoculture and/or the use of biocides, this may result in the death of many organisms attempting that interpatch migration, which is necessary to avoid what almost all agree will be the inevitable extinctions from the patches that, on their own, are almost certainly too small to sustain most populations.

6) Green et al.’s assumptions about food needs and how to meet them are largely political. Not all experts agree on how future food needs should be met—many focus on equity and food security rather than production.

Although the issue of agriculture and biodiversity preservation is certainly an important one, Green et al. present an analysis that is misleading to those not familiar with recent literature.

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References

Response

Vandermeer and Perfecto raise several points that require clarification. They assert that we assume that wildlife-friendly farming is always associated with yield penalties. We explicitly say the opposite. We state in our section describing wildlife-friendly farming that it is clearly beneficial if it does not involve yield penalties. We also explore a very wide range of shapes of species-specific density-yield functions in our model, including ones in which density increases or remains constant with respect to yield, at least over some interval. Our paper is an argument for the need to know, for a wide range of organisms and farming systems, what the relationships between species density, farming methods, and yield actually are. Vandermeer and Perfecto imply that the answers are already known and that, for many farming systems, species densities and province-wide species persistence are neutral to increases in yield. However, the literature they cite does not provide the quantitative evidence required to establish this, and the widespread need for agri-environment payments and price premiums suggests that yield penalties are instead commonplace. Moreover, price premiums can only be levied on produce that is sold to wealthy consumers, so they are not applicable to the rising productivity needed to meet food demand from poorer nations.

Vandermeer and Perfecto are concerned that we concentrate on food production rather than distribution. Hunger is undeniably linked to equity of access, but given that food demand is likely to at least double by 2050, it is essential to consider how production can be increased.

Vandermeer and Perfecto raise important points about our model not addressing the external effects of intensive farming on wildlife in nonfarmed areas, and its movement between habitat patches. We agree that these are serious limitations, and we raised them at some length in our paper as needing further research. However, as we pointed out, both high-yielding and low-yielding agriculture can have negative effects on biodiversity in nonfarmed land, and those effects associated with low-yielding agriculture, while they may be lower per unit area of farmed land, can have a severe effect if they require farming of a larger area. We are producing an extended version of our model, which incorporates negative externalities and effects of habitat fragmentation, and hope that this will suggest practical ways of better quantifying the relevant effects.

In sum, we maintain that the key to identifying how best to reconcile food production and biodiversity conservation is through explicit models guiding systematic data collection, rather than by qualitative inference.

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Fossil Horses and Rate of Evolution

In his discussion of the evolution of fossil horse teeth (“Fossil horses—evidence for evolution,” Perspectives, 18 Mar., p. 1728), B. J. MacFadden does not mention the interesting suggestion of J. B. S. Haldane (1) that these fossil data could be used for measuring the unit of evolutionary rate, for example, a darwin, for an increase or decrease of size by a factor of e per million years, or an increase or decrease of 1/1000 per 1000 years. The horse rates would range around 40 millidarwins. Haldane wrote that the unit for the character may be a unit increase in the natural logarithm of a variate, or alternatively one standard deviation of the character in a population at a given horizon.

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Reference

Response
Dronamraju correctly points out that quantitative rates of morphological evolution have been calculated from the fossil record. Haldane (1) originally proposed the “darwin” (d) to compare morphological rates of evolution in dinosaurs, fossil horses, and fossil humans. Since Haldane’s time, over a half-century ago, more key fossils have been found and the precision of geological dating has vastly improved. Gingerich (2) analyzed rates of morphological change in many fossil groups ranging from invertebrates to mammals. He found relatively high rates (~1 d) within adaptive radiations of some primitive mammals. Using tooth measurements in ancestral-descendant pairs of fossil horses from North America, I (3) found that morphological rates of evolution vary widely in different variates; for example, tooth crown height evolves more rapidly (~0.1 d) than tooth length (~0.03 d) in some extinct grazing horses, an example of mosaic evolution (4).

With some important caveats (2), Haldane’s method is an effective way to compare morphological rates evidenced from the fossil record, including the Family Equidae.

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References

HIV and Smallpox

In their Perspective “HIV: experiencing the pressures of modern life,” D. Nolan et al. depict the complex interaction of this pathogen with us humans as its host (4 Mar., p. 1422). The authors conclude that HIV is highly adaptable. As an example of a less adaptable organism, they mention vaccinia virus as the cause of smallpox. Although under certain conditions, vaccinia virus infection may cause lesions similar to those of smallpox in humans (1), smallpox in
humans was caused by variola virus, which was eradicated in the late 1970s (2).

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References

Response
GRUTERS AND OSTERHAUS ARE CORRECT IN pointing out that the virus responsible for smallpox was variola. Vaccinia is a relatively nonpathogenic virus that has served as the basis for smallpox vaccination since Jenner’s first fortuitous discovery. Knowledge of the diversity and adaptability of naturally occurring variola virus is limited, with only two of the 200 variola proteins characterized (1), although it is presumed that the high degree of homology between these related poxviruses—as well as their lack of diversity—have contributed to the success of this global vaccination strategy. The variola virus may also be relevant to the genetics of the chemokine receptor 5 (CCR5)-HIV interaction (2), as variola encodes a high-affinity secreted chemokine-binding protein that binds CC-chemokine receptors (1). This virus, now extinct in its natural form, may have been responsible for enriching (3, 4) or maintaining (5) the CCR5Δ32 mutation within Northern European populations, thus providing their descendants more than a thousand years later with relative protection against HIV infection and disease progression (2, 6).

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References

How Similar Are Poxviruses?
IN HIS ARTICLE "UNNOTICED AMENDMENT bans synthesis of smallpox virus" (News of the Week, 11 Mar., p. 1540), M. Enserink refers to the October 2004 Act to reform the intelligence community of the United States, which outlawed, inter alia, “knowing production, engineering, synthesis, acquisition, possession, usage or threatening to use variola virus,” specified in the bill as “a virus that can cause human smallpox or any derivative of variola virus that contains more than 85% of the gene sequence of the variola major or variola minor virus.” In connection with the Act, Peter Jahrling from USAMRIID points out that many poxviruses, including vaccinia virus, have genomes more than 85% identical to variola major, and suggests that overzealous interpretation of the Act would put a lot of poxvirologists in jail.

It is undisputable that many poxviruses share high degree of similarity of their nucleotide sequences; however, this similarity is pertinent only to specific portions of their genomes and not to the overall genomes. Alignments of variola major virus strain Bangladesh-1975, camelpox virus strain CMS, and vaccinia virus strain Copenhagen were shown to share nucleotide identity 91% throughout the conserved central region of their genomes (1). Nevertheless, for the interpretation of the Act, the global alignment of the whole genomes would be more appropriate than the local alignment of their conserved portions. Global alignment by Lalign program with scoring matrix dna.mat and gap penalties -14/-4 (2) shows that variola major virus...
(NC_001611) shares identity of only 28.2% with variola minor Garcia-1966, 28% with vaccinia virus WR, and 27.9% with Camelpox virus CMS.

**CORRECTIONS AND CLARIFICATIONS**

**News Focus:** "Ibogaine therapy: A ‘vast, uncontrolled experiment’ " by B. Vastag (15 Apr., p. 345). The article incorrectly described work by Deborah Mash’s group at the University of Miami. The brain bank she runs is sponsored by the National Parkinson Foundation and is primarily devoted to that disease. Her research on cocaine and alcohol did not demonstrate brain damage, but that the two drugs combine to form a lethal metabolite. Ibogaine has been shown to slow the heart only in cocaine-dependent patients with depleted blood volume. Mash holds no patent on ibogaine but has patents on the use of its metabolite, noribogaine. This metabolite, not ibogaine, was described as acting like ‘super-sticky long-acting Prozac.’ Mash says she has not published all her data on noribogaine because they are proprietary. In addition, Dorit Ron and colleagues at the University of California, San Francisco conducted research on mice addicted to alcohol, not opiates. Robert Burke and colleagues at Columbia University did the work on mice overproducing glial cell line–derived neurotrophic factor and suggested that GDNF maintains and possibly repairs dopamine receptors. Kenneth Alper is not at Columbia but is associate professor of psychiatry and neurolgy at the New York University School of Medicine.

**ScienceScope:** "Alaskan coral preserved" (18 Feb., p. 1027). The image credit was incorrect. It should have been "Alberto Lindner/NOAA."

**News Focus:** "RNAi shows cracks in its armor" by J. Cousin (12 Nov. 2004, p. 1124). The story inadvertently omitted mention of Sumedha D. Jayasena at Amgen. His team helped explain how small interfering RNA molecules unwind, which plays a role in how they target genes.

**TECHNICAL COMMENT ABSTRACTS**

**COMMENT ON “The Involvement of the Orbitofrontal Cortex in the Experience of Regret”**

David M. Eagleman

Camille et al. (Reports, 21 May 2004, p. 1167) hypothesized that regret is useful for steering decision-making. They sought to show that patients with lesions of the orbitofrontal cortex perform more poorly on a gambling task because they do not experience regret. However, choices in the task design raise questions about their interpretation.

Full text at www.sciencemag.org/cgi/content/full/308/5726/1260b

**RESPONSE TO COMMENT ON “The Involvement of the Orbitofrontal Cortex in the Experience of Regret”**

Giorgio Coricelli, Nathalie Camille, Pascale Pradat-Diehl, Jean-René Duhamel, Angela Sirigu

We recently showed that in contrast to normal subjects, patients with orbitofrontal lesions do not experience the emotion of regret. Eagleman argues that the task parameters we used may have provoked frustration, rather than regret. We demonstrate that this alternative explanation fails to consider several of our findings and is therefore implausible.

Full text at www.sciencemag.org/cgi/content/full/308/5726/1260c