El Niño and Infectious Disease

The idea that climatic cycles such as the El Niño/Southern Oscillation significantly affect infectious disease, raised by Rita R. Colwell (Association Affairs, 20 Dec., p. 2025) in the context of cholera outbreaks, is supported by recent findings from veterinary, entomological, and botanical epidemiology (*1*, *2*).

Botanical epidemiologists have found that plant systems have unique advantages for macro-scale, long-term epidemiological studies. Weather and climate are important driving forces affecting plant disease development. For example, the U.S. Department of Agriculture's annual cereal rust survey, a program started in 1917 to monitor rust outbreaks over North America, accumulates time series of disease intensity, yield loss, and races of rust fungi in cereal crops (*3*). With these data, consistent and significant coherence patterns between El Niño and wheat rust intensity have been found in both the Eastern and Western hemispheres (*2*).

Studies of El Niño–disease associations and their underlying mechanisms could lead to the development of early warning systems. Colwell advocates the use of satellite surveillance for predicting cholera outbreaks, while others propose using El Niño forecasts for malaria alerts (*4*). Outbreaks of wheat scab in eastern China can be predicted successfully 4 months in advance by measuring sea surface temperatures in the central Pacific ($R^2 = 0.86$; $P < 0.001$) (*5*); the mechanism for this association is thought to be the El Niño–dependent advance of the summer monsoon through East Asia, whereby increased precipitation causes increased infection by the scab pathogen. El Niño–disease studies are important also in the context of climate change research: infectious diseases driven by multiyear climatic cycles are likely to respond to slow, decadal changes in climate as well.

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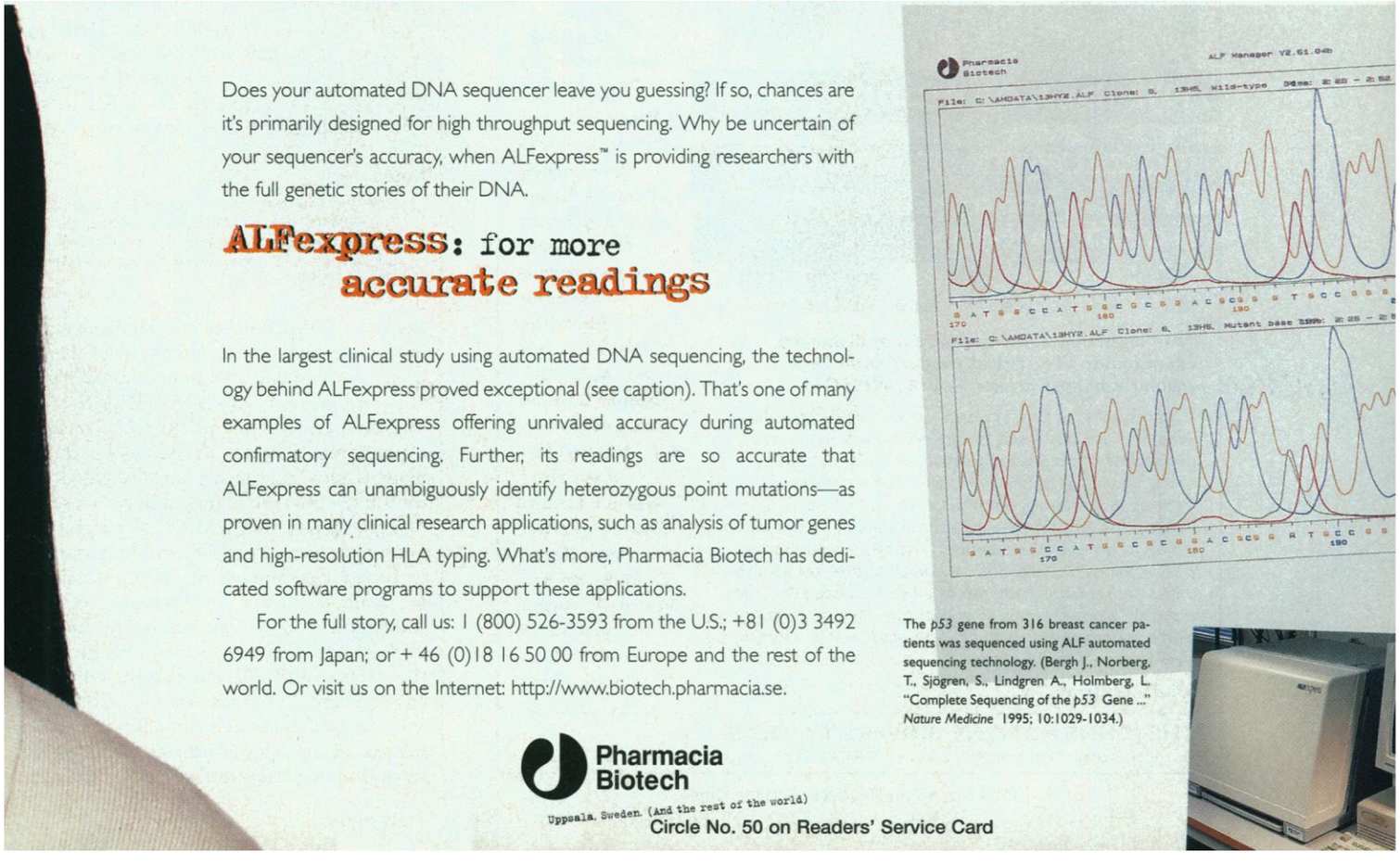
Atmospheric Nitrogen Deposition

David A. Wedin and David Tilman (Reports, 6 Dec., p. 1720) show that increased nitrogen inputs to terrestrial ecosystems might cause smaller increases in the capacity of those ecosystems to store carbon than expected. Their findings are important because nitrogen inputs have increased dramatically over the past decades through fertilizer production, cultivation of nitrogen-fixing legumes, and production of oxides of nitrogen associated with fossil-fuel burning (*1*). However, the simultaneous increase in atmospheric carbon dioxide (CO_2) concentrations caused by burning fossil fuels is likely to at least partially counteract the processes that limited carbon storage in Wedin and Tilman's experiment. CO_2 enrichment generally increases the amount of carbon fixed by plants per unit of nitrogen taken



up from the soil, particularly in carbon-3 (C^3) species (*2*) such as those that invaded their nitrogen-enriched plots. Compared with the C^4 species that thrived before nitrogen was added, the invading C^3 species have relatively lower C-to-N ratios, limiting the amount of carbon stored in response to nitrogen input. However, with elevated CO_2 tending to increase the C-to-N ratio of these C^3 plants, N and CO_2 enrichment in concert would likely cause greater C storage than observed by Wedin and Tilman.

Rising atmospheric CO_2 may also increase N inputs to terrestrial ecosystems, amplifying the direct human impact on the N cycle. CO_2 enrichment often increases the growth of plants housing N-fixing bacteria in their roots, and this stimulation is relatively larger than non-N-fixing plants (*3*). Thus, in addition to the direct anthropogenic stimulation of N inputs to terrestrial ecosystems through agriculture and fossil-fuel burning (*1*), humans may indirectly increase N inputs to terrestrial ecosystms by increasing atmospheric CO_2 concentrations. The interaction between CO_2 and N enrichment, as well as shifts in plant species, will likely influence future C storage by the terrestrial biosphere.

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Atmospheric deposition of N has been fingered as a "major threat" to grasslands in terms of biodiversity loss and disruption of ecosystem functioning in a report by Wedin and Tilman and an accompanying feature by Jocelyn Kaiser (Research News, 6 Dec., p. 1610). But are current observed rates of atmospheric N deposition sufficient to generate the extreme responses seen by Wedin and Tilman? Online data suggests not, at least not in the conterminous United States. Isopleth maps of inorganic N deposition for 1994 and 1995 produced by the National Atmospheric Deposition Program (NADP) show annual rates well below 1 gram of N per square meter throughout the grasslands of the Great Plains (*1*). Indeed, Wedin and Tilman in their site analysis used an average annual deposition rate of 0.6 gram of N per square meter. Study treatments, however, ranged from 1 to 27 grams of N per square meter per year applied in two doses. Clearly, their study shows that too much of a good thing causes problems for ecosystems, just as it does for humans. Yet the intensity of their fertilization episodes likely induced microbial dynamics and plant nitrogen availability that were significantly different from temporally diffuse patterns of wet and dry atmospheric deposition. Perhaps it is premature to raise the spectre of terrestrial eutrophication by means of atmospheric deposition as a major threat to grasslands: it pales in comparison to overgrazing and direct loss of grasslands to cultivation and urbanization.

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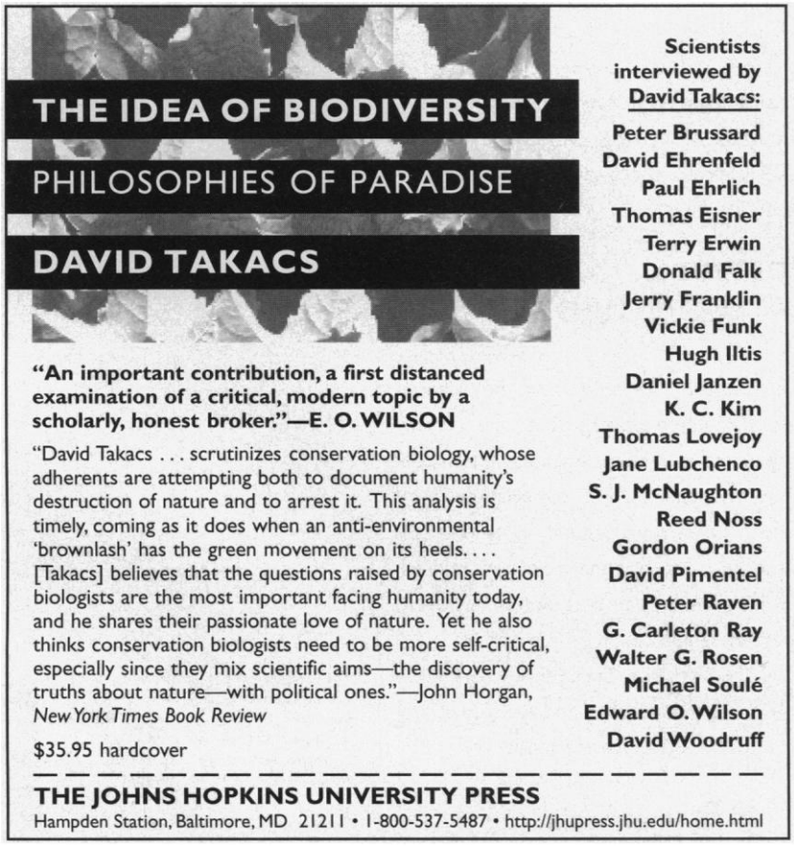
Notes

1. See for example, http://nadp.nrel.colostate.edu/NADP. NADP data before 1994 are not directly comparable because of a change in sampling protocol and are likely to overestimate nitrogen deposition slightly (<2%).

Response: Both Henebry and Hungate *et al.* raise important points; alteration of the N cycle is indeed only one component of global environmental change. As Hungate *et al.* suggest, elevated atmospheric CO_2 concentrations may increase the C-to-N ratio of plant tissues, countering the decreased C-to-N ratios that we observed in response to N loading, and thus affect C sequestration rates. However, most CO_2 enrichment studies have focused on within-species plasticity in tissue chemistry and resource use in short-term studies. Our results are largely explained by between-species differences in these traits. Will CO_2 enrichment (or other environmental changes) lead to significant shifts in species composition? What are the relative magnitudes of intraspecific plasticity and interspecific differences for the key



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plant traits that drive ecosystem functioning? Several new CO_2 enrichment experiments address these questions.

Henebry correctly points out that regional rates of atmospheric N deposition across much of the North American grassland biome are relatively low. We suggest, however, that our higher rates of experimental N addition may predict the longer-term cumulative effects of current regional rates of N deposition on grasslands. Moreover, N deposition increases dramatically across the gradient from the Great Plains to the southern Great Lakes region, where, we believe, it poses a significant threat to native prairie remnants. We strongly agree with Henebry that fragmentation, intensive agriculture, and urbanization are the major current threats to grassland biodiversity. However, even areas that are protected from these threats, but are subjected to elevated N inputs, may still suffer in the long term. In addition, NADP sites are chosen to measure regional atmospheric chemistry and are not located near point sources of atmospheric or surface-water N pollution, such as major transportation corridors, fertilized agricultural fields, or intensive livestock operation. Prairie preserves, especially in the Midwest, are often in precisely such locations and may receive N loads exceeding NADP's regional estimates.

Ecology is ultimately the study of interactions. The strength of our study is its experimental demonstration of interactions between species composition and ecosystem responses to N loading. As suggested by Henebry and Hungate *et al.*, the interactions of N loading and climate change, CO_2 enrichment, habitat fragmentation, and altered disturbance regimes (for example, grazing and fire) remain as critical research questions.

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Grizzly Habitat

The grizzlies described in Bernice Weuthrich's article (News & Comment, 25 Oct., p. 493) are hardly "wayward." They are exploring their natural habitat. The "grazing land" they have chosen to forage on is national forest—agency-managed land of many uses, including (one hopes) habitat for threatened wild mammals. The biggest threat to survival facing large predators such as grizzlies in the United States today is not hunting or poaching, loss of prey, or habitat fragmentation. It is the politically driven approach to their "conservation" through pressure applied to agencies such as the U.S. Fish and Wildlife Service and the U.S. Forest Service.

It is high time for biologists to advocate the protection and restoration of endangered species and their habitat using ecological criteria, rather than criteria related to political pressure. Otherwise, these species will go extinct.

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Corrections and Clarifications

In a Random Samples item "More private funding for Alzheimer's" (3 Jan., p. 35), Marcelle Morrison-Bogorad's affiliation was incorrectly stated. She is at the University of Texas Southwestern Medical Center in Dallas.

Wade Roush's Research News article "Fly sex drive traced to *fru* gene" (13 Dec., p. 1836) should have noted that Don Gailey led a 1991 study on the role of the *fru* gene in regulating a fruit fly muscle.

The Perspective "High anxiety" by David Goldman (29 Nov., p. 1483) contained two errors. The last sentence of the first paragraph should have been deleted. In the next sentence, the long allele should have been described as containing a 44–base pair (not amino acid) insertion.

In Jocelyn Kaiser's 8 November News & Comment article "Panel finds EMFs pose no threat" (p. 910), Richard Luben should have been identified as a biochemist.

In column two of the first page of the report "Promotion of mitochondrial membrane complex assembly by a proteolytically inactive yeast Lon" by M. Rep *et al.*, (4 Oct., p. 103), the numbering of the mutagenized serine residue "Ser^{1040}" was incorrect. It should have been "Ser^{1015}." Throughout the same report, the numbering of the mutated gene "LON S1040A" should have been "LON S1015A."

Letters to the Editor

Letters may be submitted by e-mail (at science_letters@aaas.org), fax (202-789-4669), or regular mail (*Science*, 1200 New York Avenue, NW, Washington, DC 20005, USA). Letters are not routinely acknowledged. Full addresses, signatures, and daytime phone numbers should be included. Letters should be brief (300 words or less) and may be edited for reasons of clarity or space. They may appear in print and/or on the World Wide Web. Letter writers are not consulted before publication.

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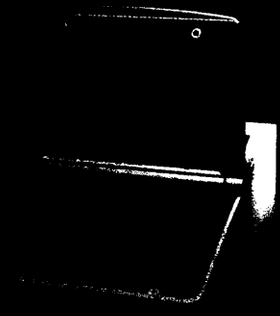

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