ECOLOGY

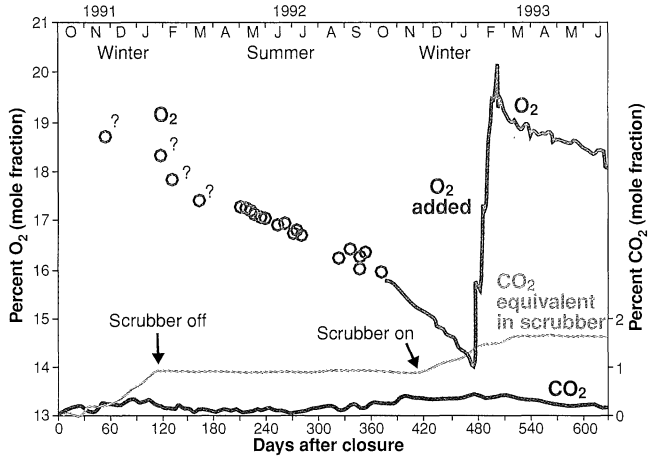
Biosphere 2 and Biodiversity: The Lessons So Far

Joel E. Cohen and David Tilman

On 1 January 1996 (*1*), Columbia University took over scientific management of Biosphere 2, a 3.15-acre closed ecosystem in Oracle, Arizona, containing soil, air, water, plants, and animals. Since then, the facility has been seeking suggestions for its future research mission from a broad range of scientists. In September, Columbia's Wallace Broecker, Biosphere 2's new chief scientist, convened a committee of ecologists, plant physiologists, and population geneticists to propose possible biodiversity experiments at Biosphere 2 (*2*). These have yet to be evaluated, in part because the new director of Biosphere 2, William C. Harris, has just moved to Columbia from the National Science Foundation. Nevertheless, the committee on biodiversity experiments was struck by some fundamental lessons already learned from Biosphere 2.

No existing closed-environment facilities for ecological research approaches the size and sophistication of Biosphere 2: the original airtight footprint covered 13,000 m^2 and enclosed 204,000 m^3. Despite the enormous resources invested in the original design and construction (estimated at roughly $200 million from 1984 to 1991) and despite a multimillion-dollar operating budget, it proved impossible to create a materially closed system that could support eight human beings with adequate food, water, and air for 2 years. The management of Biosphere 2 encountered numerous unexpected problems and surprises, even though almost unlimited energy and technology were available to support Biosphere 2 from the outside. Isolating small pieces of large biomes and juxtaposing them in an artificial enclosure changed their functioning and interactions rather than creating a small working Earth, as originally intended.

The staff of Biosphere 2, and several reports (*3–5*), revealed to the committee numerous examples of surprises that had been encountered since the facility began its first "mission," the widely publicized enclosure of eight Biospherians from 1991 to 1993. By January 1993, 1.4 years after material closure of Biosphere 2, the oxygen concentration in the closed atmosphere fell from 21% to about 14% (see figure). This oxygen level, ordinarily found at an elevation of 17,500 feet, was barely sufficient to keep the Biospherians functioning. Carbon dioxide levels skyrocketed, with large daily and seasonal oscillations. Subsequent analyses discovered that microbial degradation of carbon in the highly fertile soils (needed for food production) consumed the atmospheric oxygen, producing carbon dioxide. Although no one knew it at the time, some of the carbon dioxide combined with the calcium in the concrete used to construct Biosphere 2 to produce calcium carbonate (*4*). The original atmospheric oxygen, in effect, became locked up in the walls of the structure. In early 1993, before the end of the first 24-month "mission," oxygen was added to Biosphere 2's atmosphere from outside. Another atmospheric problem was also unanticipated. The N_2O concentration of the air rose to 79 parts per million after 3 years of closure. At that level, N_2O may reduce vitamin B_{12} synthesis to a level that can impair or damage the brain. These and other such unforeseen problems made the biogeochemical regulation of a closed atmosphere a delicate problem.



Oxygen and carbon dioxide in Biosphere 2. The drop in O_2 concentration in 1992 is much greater than the increase in CO_2, suggesting an unexpected sink for O_2 or CO_2. This sink ultimately proved to be $CaCO_3$ in the concrete walls of Biosphere 2. (The scrubber removed CO_2 from the atmosphere.)

Vines originally introduced as a carbon dioxide sink (such as morning glory, *Ipomoea* aff. *hederacea*) proved to be exceptionally aggressive. The vines required a great deal of hand weeding, which was not entirely successful, to prevent them from overrunning other plants, including food plants. The trunks and branches of large trees became brittle and prone to catastrophic and dangerous collapses. Although some species were expected to go extinct, particularly among the plants, the extremely high fraction of species extinctions (for example, 19 of 25 vertebrate species) was unanticipated (*3*). All pollinators went extinct. Consequently, the majority of the plant species, which depend on insect or vertebrate pollinators for reproduction, had no future beyond the lifetime of the individuals already present. The majority of the introduced insects went extinct, leaving crazy ants (*Paratrechina longicornus*) running everywhere, together with scattered cockroaches and katydids. Despite the relatively small size of the Biosphere 2 ocean compared to the land areas, extinction rates in the ocean appeared to be lower than those on land. Air temperatures in the upper reaches of the glass structure were far higher than anticipated, while light levels were significantly lower. Areas designed to be deserts initially became chaparral or grasslands because of a failure to adjust the rainfall to reduced evaporative demand. Water systems became loaded with nutrients, polluting aquatic habitats. Nutrients had to be removed from the water by passage over plates on which algal mats grew. The algal mats were then harvested manually, dried, and stored within the enclosure. Water chemistry management made it necessary to separate a planned brackish estuary from the ocean.

These surprises left the committee with the impression that Biospherians, despite annual energy inputs costing about $1 million (*5*), had to make enormous, often heroic, personal efforts to maintain ecosystem services that most people take for granted in natural ecosystems. Even these efforts did not suffice to keep the closed system safe for humans or viable for many nonhuman spe-

An enhanced version of this Perspective, with live links, can be seen in *Science* Online on the Web at http://www.sciencemag.org/

J. E. Cohen is at the Rockefeller University, New York, NY 10021–6399 and Columbia University, New York, NY 10027, USA. E-mail: cohen@rockvax.rockefeller.edu
D. Tilman is at the University of Minnesota, St. Paul, MN 55108, USA. E-mail: tilman@swan.lter.umn.edu

cies. Some of the surprises might, in principle, have been foreseen through better linkages with the research community of plant physiologists and ecologists. But several visiting ecologists doubted that a viable closed habitat to support human life could have been assured, even had the best ecological knowledge of the time been brought to bear.

The major retrospective conclusion that can be drawn is simple. At present there is no demonstrated alternative to maintaining the viability of Earth. No one yet knows how to engineer systems that provide humans with the life-supporting services that natural ecosystems produce for free (*5*). Dismembering major biomes into small pieces, a consequence of widespread human activities, must be regarded with caution. Despite its mysteries and hazards, Earth remains the only known home that can sustain life.

There may be a partial analogy between the initial problems of Biosphere 2 and the early, well-publicized flaws of the Hubble Space Telescope. Just as the Hubble telescope's initial images, although fuzzy, produced insights for astronomers, the initial work in Biosphere 2 has already provided insights for ecologists—and perhaps an important lesson for humanity. Now that the Hubble telescope has been improved, it is a major instrument with the potential for observations never possible before. Similarly, research in a retooled Biosphere 2 may well contribute exciting insights into the task of maintaining the viability of Biosphere 1—the Earth.

References and Notes

1. L. Wolfgang, *Science* **270**, 1111 (1995); C. Macilwain, *Nature* **380**, 275 (1996).
2. Committee members: H. A. Mooney, J. A. Berry, J. E. Cohen, R. Dirzo, C. B. Field, L. Graumlich, D. Melnick, S. Naeem, O. E. Sala, and D. Tilman.
3. T. Burgess, B. V. D. Marino, J. Joyce, *Biodiversity Working Group Summary*, internal report of the Biosphere 2 Science and Research Department (11 to 12 August 1995).
4. J. P. Severinghaus *et al.*, *Eos* **75**, 33 (1994); W. S. Broecker, *GSA Today* **6**, 1 (1996).
5. W. F. Dempster, *Tech. Pap. Ser. 932290* (Society of Automotive Engineers, Warrendale, PA, 1993); E. Odum, *Nature* **382**, 18 (1996).

BIOMEDICINE

Does Leptin Contribute to Diabetes Caused by Obesity?

Simeon I. Taylor, Valarie Barr, Marc Reitman

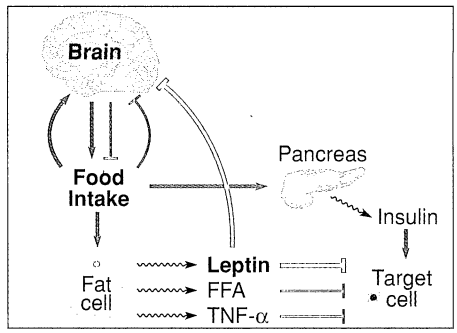
Obesity—an all-too-common public health problem—increases the chances of developing several other diseases including non–insulin-dependent diabetes mellitus (NIDDM) and hypertension. In a report on page 1185 of this issue, Cohen *et al.* (*1*) suggest that secretion of the satiety-inducing peptide leptin may be one way that obesity causes insulin resistance and thus NIDDM.

In 1994, Friedman and his colleagues (*2*) achieved a major breakthrough when they identified and characterized the *obese* gene, mutated in the obese mouse strain *ob/ob*. The *obese* gene encodes leptin, a 16-kD peptide that is secreted by fat cells (adipocytes). (*Leptin* is also the new name assigned to the *obese* gene.) Treatment of *ob/ob* mice with leptin reversed all the manifestations of the *ob/ob* phenotype and also caused weight loss in wild-type mice (*3*, *4*). This dramatic success raised the hope that leptin would be therapeutically useful for human obesity; it is now in the early phases of clinical testing in humans. Despite this enthusiasm, there remain many unanswered questions about leptin action: Which cells and tissues respond to leptin? What are the molecular mechanisms of leptin action? What is the role of leptin in the pathophysiology of human disease?

Mice with the *ob/ob* phenotype are very similar to animals with lesions in the ventromedial hypothalamus, leading to the prediction that leptin acts in the central nervous system to suppress appetite. This hypothesis was supported by the demonstration that intraventricular infusion of leptin is more effective than is intraperitoneal injection in causing weight loss in mice (*4*). However, leptin does more than decrease food intake. Without leptin, animals show decreased physical activity, hypothermia, and infertility. Indeed, leptin and its receptor probably evolved to trigger an array of adaptations to starvation, rather than as a "satiety hormone" to prevent overeating when food is abundant (*5*). Scarcity of food and starvation was a common problem throughout evolution; abundance of food is a relatively recent development.

At least two defects characterize NIDDM: insulin resistance and insulin deficiency. Genetic defects have been identified in patients with quite rare forms of NIDDM (for example, mutations in the insulin receptor gene that cause insulin resistance and mutations in the glucokinase gene that impair insulin secretion). But in most patients with NIDDM, the primary causes of the disease are unknown. Nevertheless, there is a strong association of NIDDM with obesity. In addition, obesity at least in part causes insulin resistance because weight reduction ameliorates insulin resistance. Circumstantial evidence suggests a role for the adipocyte in the genesis of insulin resistance (see figure). One hypothesis is that adipocytes secrete factors that cause insulin resistance. Free fatty acids, produced by hydrolysis of triglycerides stored in adipose tissue, can inhibit glucose utilization by peripheral tissues. Therefore, free fatty acids were among the first candidates proposed to explain the association between increased adiposity and insulin resistance (*6*). More recently, increased tumor necrosis factor–α (TNF-α), also produced by adipocytes, has been invoked as a cause of insulin resistance (*7*).

The new work by Cohen *et al.* (*1*) suggests that secretion of leptin by adipose tissue may be another mechanism whereby increased adiposity causes insulin resistance. Their data suggest the existence of "cross talk" between the signaling pathways downstream from insulin and leptin receptors. According to the usual model of insulin action, insulin binding stimulates phosphorylation of multiple tyrosine residues in the cytoplasmic domain of its receptor; this, in turn, activates the receptor to phosphorylate other substrates such as insulin receptor substrate–1 (IRS-1). Tyrosine phosphorylation of IRS-1 (and other substrates) is required to activate downstream effector pathways. When phosphotyrosines in YXXM motifs in IRS-1 bind the p85 regulatory subunit of phosphatidylinositol 3-kinase (PI 3-kinase), PI 3-kinase is activated—a necessary step for the triggering



Now there are three. Leptin joins free fatty acids (FFAs) and TNF-α as possible mediators of the insulin resistance (and NIDDM) caused by obesity.

The authors are in the Diabetes Branch, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, MD 20892–1829, USA. E-mail: simeon_taylor@nih.gov