Editorial & Letters

EDITORIAL

Science Policy in Canada

Canada's 1998 budget overturns 3 years' worth of funding cuts to its three research granting councils and provides a turnaround for basic science. How did we get here? Partly through realizing the benefits of Canada's now strong economy and its first balanced budget in 29 years. Partly through a wrenching process of cuts and prioritization occurring over several years.

After the 1993 election, the Chrétien government faced competing challenges. Expenditures had to be reduced. Canada was spending about $160 billion each year, with revenues of about $120 billion. Yet science efforts were vital to economic growth. How to proceed?

As Secretary of State for Science, Research and Development, I worked with John Manley, Minister of Industry, to focus initially on business R&D, an area where Canada has lagged. We emphasized the information superhighway and environmental and space technologies to help businesses, while also facilitating basic science and science culture development. Examples included an academic–industry consortium, CANARIE, to build and use broadband networks; SchoolNet, Computers for Schools, and a Community Access Program to help schools and rural communities connect to and use the Internet; and a digital collections initiative (for libraries and historical material). Despite the deficit, the Canadian Space Program had to be renewed, so $1 billion of new funding over ten years was provided in 1994. Efforts in space were refocused emphasizing economic benefits and Canadian science's strengths.

A second thrust addressed science-industry links and venture capital. Much mature university and federal laboratory science had commercial applications. A new tax-advantaged, labor-sponsored venture capital fund was established for medical science (the Canadian Medical Discovery Fund), as well as a broader Science and Technology Growth Fund. In both cases, the fund was linked to reviews of the science involving the granting councils, in order to provide an evaluation using expert knowledge. Regional economic development agencies like Western Economic Diversification provided new high-tech loan investment funds. University-industry links were promoted with a Technology Partnership Program and new Networks of Centres of Excellence (NCEs)(national targeted university-industry consortia) in applied health, sustainable forestry, technology-based learning, and advanced materials.

In early 1996, after extensive consultations, a new strategy called Science and Technology for the New Century was created, and a major new technology development fund (Technology Partnerships Canada) was implemented. It provided $150 million in year 1, increasing to $250 million by year 3, or $2.3 billion over 10 years.* The momentum continued in the 1997 budget: The NCE program received significant long-term funding (about $50 million per year, or $1 billion over the next 20 years). To lower industry R&D costs, tax benefits were maintained, and for small businesses the Industrial Research Assistance Program was continued. A Health Services Research Fund ($65 million) was established in 1996 to improve the application of science to health care delivery. The aptly named Data Liberation Initiative made Statistics Canada information readily available to university researchers.

The 1995 budget decreased basic science funding less than than that of other programs (10 to 15% compared to 20% in most areas, and 40% for most government economic support programs).† The '96 and '97 budgets could not reverse this trend, given a potential domino effect on other reductions. But the '97 budget really set the stage for science, providing $800 million for university research infrastructure. The 1998 Canadian budget reverses all previous cuts for basic science while instituting new incremental funds. There is a net increase of $400 million for the Medical Research Council, Natural Sciences and Engineering Research Council, and the Social Sciences and Humanities Research Council by the year 2001, as well as $2.5 billion in new scholarships and other initiatives to help postsecondary education students.

Where next? A clear plan and funding approach is needed for international science participation. The National Advisory Board on Science and Technology (NABST) recommended a more vigorous research effort to support social programs.‡ Along with continued growth in research council funding, consideration should be given to a Canadian National Institutes of Health. Science and technology investments remain vital to national success.

Jon Gerrard

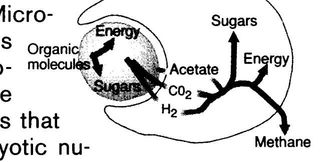
The author is a professor of pediatrics and child health at the University of Manitoba in Winnipeg, Manitoba, and the former Secretary of State for Science, Research and Development for Canada in the Chrétien Cabinet

* W. Kondro, *Lancet* **347**, 256 (1996). †D. Powell, *Science* **267**, 1418 (1995). ‡ NABST, *Healthy, Wealthy and Wise*, Government of Canada, Ottawa (1995).

LETTERS

Origins

Coral researchers propose that both parrotfish bites and a fungus are causing damage to coral in Central and South America, the Caribbean, and Florida. Microbiologists offer support for the hypothesis that the eukaryotic nucleus "originated in a methanogen" (above, drawing showing possible exchanges of molecules, including hydrogen, binding microbes together in an early complex cell). Other letters discuss "the earliest Americans," brain function, the early Black Sea, fertilizer use, seal research, and hemophilia.



Coral Disease

Last year, it was reported that rapid-wasting disease (RWD) killed scleractinian corals at rates as high as 7.5 centimeters of tissue in 24 hours (Random Samples, 27 June 1997, p. 1979). An international group of scientists representing diverse disciplines is collaborating to investigate what we now believe to be two different, but related, syndromes that we term parrotfish white spot biting (PWSB) and rapid-wasting syndrome (RWS). Both of these have been seen in South and Central America, throughout the Caribbean and in Florida (*1*).

More recent observations have shown that the condition initially described as RWD spreads less rapidly than previously thought and that the large white dead zones are actually bite lesions where coral tissue and skeleton have been forcibly removed by parrotfish (A. Bruckner and R. Bruckner, Letters, 27 Mar., p. 2023). PWSB is a phenomenon of corals related to bites by *Sparisoma viride*, the stoplight parrotfish. *S. viride* inflicts overlapping bite marks on the coral, with deep excavation, and frequently returns to the same coral to inflict additional damage. Large mid-phase and terminal-phase males exhibit this biting behavior most often (*2*). The lesions are most commonly found on all morphotypes of *Montastraea* species and on *Colpophyllia natans* (which previously has not been documented as a grazing target of *S. viride*). Some corals fail to recover from the bite damage, because algae have colonized areas of skeleton that were exposed by the *S. viride* bites, preventing recovery. According to data collected by one of us (J.M.C.) during January 1998, there has been a dramatic decline in attacks by *S. viride*. Horizontal and vertical transects (measured by J.M.C. and T.J.G.) reveal a significant decrease in the quantity of corals attacked. Lesions were less evident during our most recent visits to Bonaire. Horizontal 15-meter belt and video transects (measured by J.M.C. and T.J.G.) show that during February 1997, there were 0.5 cases per meter of RWS lesions, compared with 0.075 cases per meter during March 1998.

RWS, the term now used for RWD, is characterized by a fungal invasion of the coral. A distinctive fungus takes up residence within and on top of the coral tissue, causing cell damage. The coral-bite lesions appear to be connected to the fungal invasion characteristic of RWS. Sometimes the fungus resides on coral that has been bitten by *S. viride*, which explains why the two syndromes were originally thought to be one disease. In fact, PWSB has been documented at the exact location where two of us (T.J.G. and J.M.C.) first described RWD in February 1997 (*3*).

We are jointly engaged in field observations and experiments, laboratory experiments, and microbiological studies to determine the origin of the RWS fungus and to resolve the association of PWSB and RWS. The fungus associated with RWS has been examined in the laboratories of Ray Hayes, Garriet Smith, and Steve Golubic. It was initially hypothesized that the fungus might be endolithic to the corals. However, an investigation by Golubic (*4*) showed that the fungus is not present within normal coral tissue. There are many possible mechanisms for the fungal invasion of the corals. These include a secondary infection, perhaps spread to the corals by *S. viride*, or transport of the fungus by runoff, or the fungus could be an opportunistic infection aggravated by environmental degredation. All that is clear at this time is that RWS is the result of a complex process.

More work is needed to characterize the spatial and temporal scales of PWSB and RWS. It is possible that the fungus present in RWS samples may be present before the damage inflicted by the parrotfish and that it expands opportunistically when the coral is stressed by the bite. Diseases and putative disease syndromes other than RWS may also be increasing in frequency and extent (*5*), but none of these others seem to be related to parrotfish-inflicted lesions on living corals.

James M. Cervino, *Global Coral Reef Alliance, 124-19 9th Avenue, College Point, NY 11356, USA, E-mail: nidaria@earthlink.net*; **T. J. Goreau**, *Global Coral Reef Alliance, E-mail: goreau@bestweb.net*; **R. L. Hayes**, *Howard University College of Medicine, Washington, DC 10059, USA*; **L. Kaufman**, *Boston University Marine Program, Department of Biology, Boston University, 5 Cummington Street, Boston, MA 02215, USA*; **I. Nagelkerken**, *Caribbean Marine Biological Laboratory, Piscadera Baai, Curacao, Netherlands Antilles*; **K. Patterson**, **J. W. Porter**, *Institute of Ecology, University of Georgia, Athens GA 30602–2602, USA*; **G. W. Smith**, *Biology Department, University of South Carolina, Aiken, SC 29801, USA*; **C. Quirolo**, *Reef Relief, Post Office Box 430, Key West, FL 33041, USA*

References

1. J. M. Cervino and G.W. Smith, *Ocean Realm* (Summer 1997), p. 33; T. J. Goreau *et al.*, *Rev. Biol. Trop.*, in press.
2. P. Frydl, *Int. Rev. Hydrobiol.* **64**, 737 (1979).
3. H. Bruggemann, M. van Oppen, A. Breeman, *Mar. Ecol. Prog. Ser.* **106**, 41 (1994); J. M. Van Rooij, E. de Jong, F. Vaandrager, J. J. Videle, *Environ. Biol. Fishes* **47**, 81 (1996).
4. S. Golubic, personal communication.
5. R. Hayes and N. Goreau, *Rev. Biol. Trop.*, in press.

■

Origin of the Eukaryotic Nucleus

In support of William Martin and Miklós Müller's hypothesis that the eukaryotic nucleus originated in a methanogen (G. Vogel; Research News, 13 Mar., p. 1633) (*1*), we note that, whereas most eukaryotes use histones to compact their nuclear DNA into nucleosomes, the only prokaryotes that have histones and nucleosomes are the Euryarchaeota, the division of the Archaea that includes the hydrogen-consuming methanogens (*2*). At the high DNA concentrations present in the nucleus, DNA molecules spontaneously aggregate if not hindered by histone packaging, and it has been proposed that it was the availability of the nucleosome system of DNA packaging that facilitated nuclear expansion and therefore eukaryotic evolution (*3*). Perhaps eukaryotic evolution continued beyond the ancient hydrogen-based metabolic symbiosis postulated by Martin and Müller because the hydrogen-consuming partner fortuitously also had a histone-based system of genome packaging.

Kathleen Sandman
John N. Reeve
Department of Microbiology, Ohio State University, Columbus, OH 43210, USA
E-mail:reeve.2@osu.edu