"There's no reason that biodiversity can't be a big-ticket item," Cracraft told SCIENCE NEWS. "We need to shed physics envy and promote systematics envy and biodiversity envy."

Although the effort began as a strategy for U.S. science, it quickly took on an international focus, Cracraft says. It calls for the building of museums and training of systematists in Mexico, Brazil, Indonesia, Colombia, and other "species-rich" countries. According to the agenda, about 80 percent of the land plants and animals live in countries with barely 6 percent of the scientists who know how to determine what these organisms are and how they are related.

"What we're trying to do is to get other countries to realize they must understand biodiversity in a sophisticated way," Cracraft says.

Such knowledge is worth the investment, he and others argue. They cite the value of biodiversity in providing disease-resistant plant and animal varieties, new genes for improving domesticated animals and crops, and new compounds for the pharmaceutical industry. In addition, colorful coral reef fishes, giant saguaro cacti, magnificent elephant herds, and many other plants and animals lure millions of tourist dollars to otherwise destitute regions.

Furthermore, for countries involved in the Biological Diversity Convention (SN: 6/20/92, p.407), the more species they document, the more aid they may warrant for those resources, says Brian Groombridge of the World Conservation Monitoring Center in Cambridge, England.

Thomas Eisner/Cornell Univ.

Systematists have learned that Lake Placid Scrub Mint differs from closely related mints, each a potential source of insect repellent.

As part of this push, researchers recognize the need to get information out of museums and into the hands of policymakers and others. Thus, like the global climate change programs and the Human Genome Project, the plan calls for compiling databases on computer. Ideally, anyone could gain access to pictures and sounds as well as verbal descriptions electronically, says Cracraft, who coordinated development of the agenda.

That access would help countries make better conservation decisions, says Groombridge. His not-for-profit organization supplies diversity information to governments and engineering companies planning development projects or assessing natural resources. At the systematics symposium, he cited the need for more extensive cataloging of more kinds of organisms. His organization's review of newly reported species indicates that about the same number of new ones is described in each plant and animal group each year. "The way I interpret this is we have a bottleneck in taxonomists," Groombridge says. "This serves to highlight the need for more and continuing [growth in] systematics, not less."

"We're the only ones trained to inventory and analyze species diversity and to understand the phylogenetic relationships," adds Cracraft.

Often, however, young U.S.-trained systematists leave the discipline for lack of faculty or museum positions, complains Hugh Iltis, a plant taxonomist at the University of Wisconsin-Madison. "There are no jobs; yet there are huge genera that are not described." *— E. Pennisi*

Gene finding gives clues to DNA repair

In the mid-1960s, as a postdoctoral student, James E. Cleaver thought a lot about the genetics of radiation sensitivity in cells. One day, he read a newspaper report about xeroderma pigmentosum (XP), a rare disease that renders people ultrasensitive to sunlight. Are people with XP, Cleaver wondered, somehow unable to repair the genetic damage caused by exposure to the sun's ultraviolet rays?

"It was the kind of [hypothesis] that if I was right, I had a living out of it, and if I was wrong, nobody would have noticed," recalls Cleaver, now a geneticist at the University of California, San Francisco. He proposed the connection between XP and faulty DNA repair and proved it. Now, 25 years after Cleaver's initial report, two teams of scientists have independently flushed out the defective gene that causes a particularly severe form of the disease, XP-G. The researchers report their work in the May 13 NATURE.

Defects in this gene, and in seven others linked to different forms of XP, interfere with normal DNA repair. Usually, these genes serve as a blueprint for enzymes that recognize and cut out sections of cells' damaged DNA. Other cell mechanisms clear away the wreckage and replace the damaged sections of genetic code.

People with XP suffer various symptoms, depending on which defective gene they carry. In people with XP-A, for example, DNA repair is almost completely knocked out, causing brain deterioration and many skin tumors. In contrast, people with less severe forms of the disease can avoid many of its serious symptoms by simply avoiding exposure to sunlight. About one person in 100,000 has the disease, says Richard D. Wood, a biochemist at Clare Hall Laboratories in South Mimms, England.

DNA repair, the researchers emphasize, has proved one of the most fundamental aspects of cell life. It counteracts the constant assault on cells by chemicals, radiation, and other environmental causes of genetic damage. Unfortunately for people with XP, "mutations in these [repair] genes can cause important developmental defects, such as mental retardation, immune-system diseases, and sensitivity to cancer-producing compounds," explains Stanford University molecular biologist Philip C. Hanawalt.

In one of the new studies, Wood and graduate student Anne O'Donovan found that extracts from normal cells, containing functioning repair enzymes, turned DNA repair back on in XP-G cell extracts. The scientists then isolated the particular enzyme that reversed the defect and mapped its gene to chromosome 13.

A team of researchers at the University Medical Center in Geneva, Switzerland, came upon the XP-G gene while studying an entirely different disease, systemic lupus erythematosus (SLE). Searching for a protein linked to SLE, these researchers by chance discovered a gene capable of restoring normal DNA repair function to XP-G cells, report Daniel Scherly and colleagues.

These two reports herald the final stage of a 25-year effort to identify and copy, or clone, the defective DNA-repair genes that cause XP, Wood explains. To date, five genes have been isolated. By next year, Wood predicts, investigators will find the remaining three. At that point, research will shift toward puzzling out the function of each repair gene.

Cancer treatment could benefit from increased intimacy with DNA-repair biology, says Wood. Typically, cancer drugs kill tumor cells by attacking their DNA. If researchers could find a way to selectively shut down tumor cells' repair machinery, cancer drugs could kill tumor cells much faster than they kill normal cells, he says. *— D. Pendick*