



A Smithsonian Jewel: Biological Collections

ONE OF THE "JEWELS IN THE CROWN" OF THE Smithsonian Institution—the national biological collections and their associated systematics research programs—is overlooked in the News Focus article about Smithsonian science ("Turmoil behind the exhibits" by E. Pennisi, 13 Jul., p. 194). At a time when the demands for biodiversity information are greater than ever (1), the Smithsonian is one of the primary sources of such information, data, and knowledge.

The biological collections include 83 million biological specimens (complemented by 40 million fossils, plus smaller living collections), forming one of the two greatest collections of biodiversity in the world. Seventy Smithsonian research systematists work with these collections, along with 40 systematists in associated federal agencies and more than 50 research associates. This represents a unique federal partnership, begun in 1881, in which the U.S. Departments of Agriculture, Commerce, Defense, and Interior co-locate their systematics researchers and identification services with the Smithsonian. These federal partners contribute \$6.5 million annually toward our overall programs, which represents a massive cost-share for the Smithsonian.

Over the last 10 years, the manner in which systematics products and identification services are paid for and distributed has changed dramatically (2). Many of the world's museums have been forced by financial problems to increasingly focus on provincial issues of greatest interest to the people who "pay the bills" (3). Meanwhile, world productivity in systematics research appears to be declining (4). Thus, the Smithsonian and its federal partners are becoming more and more important to provide the infrastructure to sup-

port systematics and identification services. Collectively, our biologists hosted over 900 scientific visitors for more than 10,000 visitor days, made 1433 outgoing loans totaling 166,695 specimens for research, and made more than 50,000 identifications in 2000. Over half the articles in major U.S. journals in systematic botany cite the use of Smithsonian specimens (5) and although there are 34 insect collections in North America with more than 1 million specimens, some 20% of the insect specimens loaned for research by North American museums come from the Smithsonian (6). Our role in graduate education is highlighted by hosting eight National Science Foundation Partnerships in Enhancing Expertise in Taxonomy (PEET) grants in collaboration with several universities.

We continue to add specimens at almost half a million annually (7), and external systematists are constantly upgrading the information content of our collections. We have begun increasing accessibility of our data by digitizing some 3.7 million records, representing 9% of the collection (8). Collections of specimens represent a vital information base for protecting the world's environment for future generations, and we are committed to put in place the infrastructure needed to efficiently manage and disseminate appropriate data, while maintaining the collections into perpetuity.

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5. Brittonia, Fieldiana, Novon, Phytologia Systematic Botany, and Systematic Botany Monographs, surveyed for 1990 to 2000.
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8. Some specimens are cataloged in lots, so the number of data records is less than the number of individual specimens.

Geneticists' Views on Embryonic Stem Cells

THE MEDIA COVERAGE OF THE CONTROVERSY over embryonic stem cell research cites the arguments of many individuals or selected groups, but what opinions do specialists in human genetics research hold about the issue? Last year I conducted a survey of U.S. members of the American Society of Human Genetics (1). The survey questions covered many ethical issues in the field of human genetics, several of which dealt specifically with embryonic stem cell research.

Of the more than 1200 respondents, 87% agreed with the 1999 ruling by the U.S. Department of Health and Human Services that exempted human embryonic stem cells from the Congressional ban on the use of federal funds for human embryo research. The ruling determined that the ban did not apply to human embryonic stem cells primarily because, by themselves, they do not have the capacity to develop into human beings. Most of these scientists (71%) favored discontinuance of the ban itself. But whether embryonic stem cell research was considered ethically acceptable depended on the source of the embryos. Significant majorities responded that it was acceptable to use embryos retrieved from

Letters to the Editor

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The Smithsonian's National Museum of Natural History holds one of the world's premier biological collections.

aborted fetal tissue and embryos originally created for fertility treatment (77 and 59%, respectively); however, 73% of respondents viewed creation of human embryos for research purposes as ethically unacceptable.

Thus, although the respondents are generally opposed to the creation of embryos for research, most appear to view the potential medical benefits as such that we should take advantage of the existence of unused embryos that would ultimately be discarded.

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References and Notes

1. The survey was part of a study sponsored by the Richard Lounsbery Foundation. A 12-page survey was sent to 3632 U.S. members of the American Society of Human Genetics, and 1236 surveys were returned, 1229 of which met all eligibility criteria. A copy of the group of questions from the survey relevant to this discussion are available from June Goldner, research consultant for the survey, at goldner@bestweb.net

Parasitic Plants Major Problem to Food Crops

IN THE PLANT PATHOLOGY SPECIAL ISSUE, THE News articles and Reviews discuss such seed plant pathogens as bacteria, fungi, viruses, nematodes, and insects (22 Jun., pp. 2269–2289). Strikingly absent from the discussion topics were parasitic angiosperms, plant pathogens that are plants themselves.

Such parasitic plants are present in most terrestrial ecosystems, but those feeding on food crops are particularly infamous (1). Species of the root parasitic genera *Striga* and *Orobanch* are notorious for the devastation they cause in less developed countries. *Striga* is estimated to infest more than two-thirds of the 73 million hectares in cereals and legumes in Sub-Saharan Africa, affecting the daily lives of some 300 million Africans in 25 countries (2). *Orobanch* destroys a broad spectrum of host crops, including legumes, vegetables, and sunflowers, in the Mediterranean and Middle East regions. Although developed countries have not escaped the ravages of parasitic plants—dwarf mistletoe (*Arceuthobium* spp.) destroy up to 3.2 billion board feet of lumber per year in western U.S. forests (3)—lesser developed



Orobanch crenata damages broad-beans in Syria.

countries are most affected.

The International Parasitic Plant Society (<http://www.ppws.vt.edu/IPPS/>) was inaugurated this year to promote the study of parasitic plants toward the aim of ultimately enhancing world food security. Funding research aimed at controlling parasitic weeds falls outside the agenda of most federal agencies, so it has largely been international agencies and foundations, notably the U.S. Agency for International Development and the Rockefeller Foundation, that pay for these studies. Their efforts have resulted in significant advances toward ameliorating the *Striga* problem, most notably through breeding efforts for developing host resistance.

With the emergence of genomic data from model species such as *Arabidopsis* and rice, novel approaches for controlling these pests can be envisioned. But the first step is bringing the problem to light.

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The Brain's Susceptibility to Amyloid Plaques

AMONG THE MOST CHALLENGING MYSTERIES of Alzheimer's disease is the identification of factors that render the brain particularly susceptible to the extracellular deposition of β -amyloid ($A\beta$). $A\beta$ peptides are thought to be toxic and are the central component of neuritic plaques, which are preferentially localized to brain regions that are critical for memory, cognition, emotional state, and personality.

Recent research has begun to provide new clues concerning the biological basis for the vulnerability of the brain to these abnormalities, and this information has relevance to developing therapies for Alzheimer's. $A\beta$ is generated from cleavages of the β -amyloid precursor protein (APP) by β site APP-cleaving enzyme 1 (BACE1) (1, 2) and by an enzyme activity termed γ -sec-

retase. In vivo, BACE1 has been found to be the principal β -secretase necessary to cleave APP to generate $A\beta$ (3). In contrast, APP can also be cleaved within the $A\beta$ sequence by putative " α -secretases" or by BACE2 to release the ectodomain (the amino-terminal soluble fragment) of APP (4); these cleavages within the $A\beta$ domain of APP preclude the formation of $A\beta$. In Alzheimer's, why is the brain but not any other organ (such as the pancreas, for example) particularly vulnerable to $A\beta$ deposition?

BACE1 and BACE2 are expressed ubiquitously, but levels of BACE1 messenger RNA (mRNA) are particularly high in brain and pancreas, whereas the levels of BACE2 mRNA are relatively low in all tissues, except in the brain, where it is nearly undetectable. Because BACE1 is the principal β -secretase in neurons and BACE2 limits the secretion of $A\beta$ peptides, we propose that BACE1 is a pro-amyloidogenic enzyme, whereas BACE2 is an anti-amyloidogenic enzyme.

In this scenario, the relative levels of BACE1 and BACE2, in concert with the abundance of APP in neurons, are major determinants of $A\beta$ formation. Under this model, the secretion of $A\beta$ peptides would be expected to be highest in neurons and brain, as compared with other cell types or organs, because neurons express high levels of BACE1 coupled with low expression of BACE2. If the ratio of the level of BACE1 to BACE2 is a critical factor that selectively predisposes the brain to the formation of $A\beta$, Alzheimer's disease would be predicted to involve the brain rather than heart or pancreas.

Seemingly inconsistent with this hypothesis is the observation that there are very high levels of BACE1 mRNA in the pancreas (1). It appears that some of this mRNA is alternatively spliced to generate a BACE1 isoform that is incapable of cleaving APP (5); thus, $A\beta$ is not deposited in the pancreas. Taken together with the observations that the pancreas has low levels of BACE1 protein (6), as well as low BACE1 enzymatic activity (2), these results are consistent with the view that a high ratio of BACE1 to BACE2 activity is a major determinant of selective vulnerability of the brain to formation of $A\beta$ plaques.

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