U.S.–China Collaboration

While we appreciate the interest shown in our collaborative project in China (Research News, 4 May, p. 553), I would like to clear up three possible misunderstandings.

First, the arrangements we have made with Taiwan and with China represent two bilateral agreements with Cornell that will lead to joint compilation of data. This is not a research project "between the two countries," but two bilateral projects.

Second, mortality study undertaken in the 1970s at the Chinese Academy of Sciences was conducted by Li Junyao and his colleagues. Since then, Li has been one of the four principal investigators on our collaborative project.

Third, there was quite naturally early skepticism on both sides of the Pacific about the logistics of effecting sample shipment, analytical reliability, data reliability, and so forth for our project.

But whatever difficulties have been experienced or even perceived did not come for the most part from the Chinese side. We have found our Chinese colleagues' interest and willingness to participate in a forthright and scholarly manner to be exceptional. We could not have hoped for a more forthright and generous collaboration.

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Moffat states that in the traditional Chinese diet "animal fat provides only 15% of the calories," while noting that in a "typical U.S. diet, animal fat provides 40 to 45% of the calories."

Data collected for the U.S. Department of Agriculture (USDA) Nationwide Food Consumption Survey for 1985 (*1*) indicates that fat consumption by adults in the United States was 36 to 37% of total calories consumed. Having been reported as over 40% in both 1965 and 1977, this figure represents a decrease in fat calories estimated by this survey. The USDA reports that about 50% of our fat intake is from animal products, slightly more than 30% coming from meats (red meat, poultry, fish, and mixtures).

Careful interpretation of epidemiological information is needed when it is suggested that a single environmental component is the cause of an effect, in this case, that meat consumption is the cause of the differences in disease susceptibility. In China, the isolation of populations, both nutritionally and genetically, makes interpretation of differences among these subgroups difficult and also makes direct comparison with the U.S. population questionable.

These epidemiological studies are the beginning of research aimed at improving our understanding of the relationships between diet and disease, not the end. Much of the public view that individual foods are the cause of chronic disease comes from misuse of epidemiological observations. A great deal of basic and applied research is needed to establish the existence of a sound relationship between a diet component and susceptibility to a disease.

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Anne Simon Moffet describes the opportunity China provides for epidemiological study. A photo on page 553 shows children lined up in front of a balance beam scale, having, according to the caption, their "heights" measured. The clever surveyors must know how high the correlation is between weight and height and decided to measure just one of these values. But the article closes with the statement that "This study . . . offers the Chinese an opportunity to learn from our mistakes." Is this one of them?

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Response: Unfortunately, yes. During editing, "heights" was substituted for "weights."
—Eds.

Support for Systematics

Ward Watt's defense (Letters, 6 Apr., p. 18) of Paul Ehrlich's mid-century work in systematics complements today's research in phylogenetic systematics. Unfortunately, as exemplified in other recent *Science* articles on biodiversity (*1*), there is still little understanding of how to use such reconstructions of shared evolutionary history and trait inheritance as a basis for comparative biology. This does not augur well for biology's "golden" interdisciplinary age (News & Comment, 1 Dec. 1989, p. 1115). If systematics is to meet E. O. Wilson's prediction (*2*) and guide this pluralization, there must be an understanding that systematics matters to biology because it embodies the process theories of organisms' existence.

The most remarkable collective property of organisms is not their diversity, but their many shared traits through which that diversity is expressed. Through evolutionary modification, certain traits of species have become the inherited, homologous traits of their descendants in a historical hierarchy of clades of descendent taxa, each nested within a more inclusive, temporally prior, clade, with every taxon sharing certain primitive and derived traits. Systematics tries to identify these traits and reconstruct the hierarchy of relationships. So systematics is not only taxonomy—the description of organisms in an ordered system of words—or only the collection and identification of organisms. It is, most generally, the study of how to best compare the results of evolution (*3*).

Yet biologists today are being asked to support systematics for only the service-industry tasks of identification and enumeration (*4*). This is a needlessly restrictive and nonevolutionary approach, for without a phylogenetic context, species might as well have been created yesterday one by one (*5*) and biodiversity studies, for all their breadth, are arcane exercises in splitting, lumping, and pigeon-holing. With a phylogenetic context, biological research becomes more efficient: kinship is distinguished from overall similarity; nested clades eliminate redundant explanations; a biological "law" may be restricted to a speciose clade, therefore, check outside the clade before relying on the law. And research can *save money*. For example, if a model species proves impractical (for, say, genomic sequencing, drug production, or even species preservation), the homologous trait(s) of interest may be present in the sister species, which might be a continent away; conversely, the expense of species assays can be reduced by avoiding clades that have never yielded the trait(s) of interest. Systematics can provide the evolutionary basis for these and other comparative decisions.

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2. E. O. Wilson, *BioScience* **39**, 242 (1989).
3. A. B. Smith and C. Patterson [*Evol. Biology* **23**, 127 (1988)] demonstrate how poor systematic method-

ology can distort comparisons and produce spurious measures of biodiversity.
4. R. J. O'Hara, D. R. Maddison, and P. F. Stevens [*Science* **241**, 275 (1988)] concur.
5. K. S. Thomson [*Am. Scientist* 77, 264 (1989)] notes the damage that data-centered terminology has inflicted on systematics.

EPA and Asbestos Removal

Environmental Protection Agency (EPA) Administrator William K. Reilly's letter disclaiming EPA responsibility for the removal of asbestos from buildings (1 June, p. 1064) is self-indicting. He clearly states that EPA only requires asbestos removal when building demolition or renovation activities threaten to release significant amounts of asbestos fibers into the air; he also seems to say that it is not the fault of EPA if, in his words, "a number of building owners are removing asbestos from their buildings . . . due to forces (for example, concerns about property devaluation, insurance, and liability) that may be unrelated to health risks."

Over the years there have been numerous EPA press releases pointing to the danger of asbestos and the need to protect public health. Reilly should reread those releases and then reaffirm the extent of EPA's involvement in creating the "forces" that have incited the need for asbestos removal.

EPA alone bears the responsibility for the "killer" image of asbestos. The American public, after spending billions of dollar removing it, now deserves to hear the facts with which the EPA can prove its claims about the danger of asbestos. The disclosure of that proof is long overdue.

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British Radiation Study

The conundrum resulting from Martin J. Gardner's recent report (*1*) of paternal irradiation and childhood leukemia (News & Comment, 6 Apr., p. 24) begs for resolution by synthesis rather than refutation of either side. The missing factor may be the dietary habits (particularly the dietary fat intake) of the British and Japanese populations studied.

There is a dramatic difference in the incidence of certain cancers in the Japanese compared with that in Western populations, and a possible explanation involves both the lower intake of total fat and the higher percentage of ω-3 fatty acids in the Japanese diet. Animal studies have demonstrated a marked effect of oil seed ω-6 fatty acids as tumor promoters when provided in the range similar to that in the current Western diet (*2*) and a countervailing effect of the long-chain ω-3 fatty acids obtained from cold water fish (*3*). Differences in dietary fatty acid composition can affect membrane content of highly unsaturated essential fatty acids, their eicosanoid products, and the expression of the *ras* oncogene (*3*).

At the time of exposure to the radiation from nuclear weapons used at Hiroshima and Nagasaki, the population of those cities were consuming only 10 to 15% of calories as fat, with ratios of ω-6 to ω-3 of about 1 to 1. In recent decades the Western diet has consisted of 30 to 40% fat calories with an ω-6 to ω-3 ratio of more than 10 to 1 (*4*). These dramatic differences in total fat and the ratios of metabolically distinct fatty acid families could be a factor in the differences in the post-exposure incidence of leukemia in the two populations. The diet of the father or the child may thus amplify or suppress the oncogenic initiating effect of the ionizing radiation.

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3. R. A. Karmali, in *Dietary Omega 3 and Omega 6 Fatty Acids; Biological Effects and Nutritional Essentiality*, C. Galli and A. P. Simopoulos, Eds. (Plenum, New York, NY, 1989), pp. 351–359.
4. O. Adams, in *ibid.*, pp. 33–41.

Usefulness of the Human Genome Project

The crisis in funding by the National Institutes of Health (NIH) of research R01 grants has renewed the dialog about the usefulness of the Human Genome Project. As long as there was adequate funding for basic research, this discussion was based on more theoretical grounds (for example, whether the human, mouse, yeast, and so forth, genome should be sequenced and how). However, with the drastic cutbacks in NIH funding, the discussion has become more personal as many investigators are questioning their survival in academic science without NIH grants.

Both the Reagan and Bush administrations have been committed to providing a certain amount of money for biological research. What has become apparent is that basic research funds are being removed from the general allotment of federal funds for biological research and are being funneled into the Human Genome Project, which results in a contraction of basic science research funding.

With this point in mind I recently reviewed the research in my own lab in the context of what we would have done differently if the mouse genome had been sequenced before the start of our project. Specifically, we have recently completed the cloning and sequencing of the coding sequences for the murine complement receptor Crry and Cr2 genes. If we had had the 60 to 80 kilobases of mouse genomic sequence that contains these genes before we had started our work, what would we have done differently? Because one cannot look at a piece of genomic DNA and determine coding sequences, cDNAs have to be isolated and sequenced for the analysis of any gene. The most time-consuming and laborious steps in this project were the production and screening of the spleen and liver cDNA libraries and the subsequent sequencing of the cDNA clones. The sequence of the mouse genome would not have aided in this step of the project other than to provide confirmatory sequence information.

The only information that the full genomic sequence would have provided, in the context of our study, would have been the intron-exon border junctions. The Cr2 gene covers about 50,000 base pairs in the genome and probably contains between 15 to 20 exons. We could easily determine the intron-exon organization of this gene on our own for the price of the oligonucleotides needed to sequence across the junctions. These oligonucleotides would cost about $500. The mammalian genome contains about 5×10^9 base pairs of DNA, which, on the basis of our $500-for-50,000-base-pairs estimate, means that we as a scientific community can obtain the pertinent sequences from the human genome for $50 million, or the equivalent of one-quarter of next year's projected funding for the Human Genome Project.

Obviously the Human Genome Project must be considered as two distinct projects: (i) the mapping of the genome and (ii) determining its DNA sequence. The former project is laudatory and worth the money and time invested. The latter project is not cost-effective because the pertinent coding sequences must be obtained from the analysis of messenger RNA transcripts and cannot be deduced solely from the analysis of the genomic sequence.

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