Openness in Private-Public Collaboration

As a long-time champion of private-public collaboration, I was pleased to read Eliot Marshall's article "Drug firms to create public database of genetic mutations" (News of the Week, 16 Apr., p. 406). The new initiative is a laudable effort in many ways and an example of the kind of initiative needed to address human health issues of concern to all sectors of society.

Marshall notes the open nature of the effort, which was appropriately viewed by some scientists as a "new model" of public-private collaboration and viewed by some as "absolutely unique." A similar mode of an "open nature" that was initiated in 1976 and continues today involves the Chemical Industry Institute of Toxicology (CIIT). CIIT is a not-for-profit research institute supported principally by dues payments made by more than 30 private companies in the chemical sector. The founding Board of Directors of CIIT wisely put in place operation guidelines calling for all research findings to be analyzed and prepared for publication in a timely manner, irrespective of potential impact on the industry. In addition, the board specified that all manuscripts should be subjected to rigorous internal peer review and submitted to high-quality, peer-reviewed journals. And, most important, in the same manner now being advocated for the new genomic initiative, the results were to be disseminated broadly to all interested parties without giving preference to the sponsors. This open communication of research findings has been the key to CIIT's achievement of a high level of scientific credibility. As a result, the institute's research results are widely used around the world in regulatory proceedings and in nonregulated voluntary actions to limit human health risks of chemicals.

The high level of credibility of the institute has led to the use of CIIT as a cornerstone for an expanded health and environmental research initiative supported through the Chemical Manufacturers Association (CMA) by more than 190 chemical companies. The CMA leadership has adopted the CIIT practices of peer review, complete public disclosure, and availability of results to all interested parties. These policies are essential for the credibility of private-public collaborations in a society that is frequently skeptical of private, and for that matter, government endeavors. Good business sense and the common good can both be served in advancing science and human health when openness is used to counter skepticism.

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Brain Regions and Drug Addiction

Robert F. Service reports on an interesting study by Astrid Nehlig and her colleagues at the recent American Chemical Society meeting (News Focus, 9 Apr., p. 244). Nehlig and her colleagues found that caffeine does not result in increased blood flow to the nucleus accumbens, a brain area known to be involved in many drugs of abuse. In contrast, she found increased blood flow to the caudate-putamen. These results fit well with studies showing that caffeine induces expression of the activity-dependent gene *c-Fos* in the caudate-putamen, with little activation in the nucleus accumbens (*1*). However, a lack of activity in the nucleus accumbens does not indicate that the drug is not addictive. Several other brain regions, including the caudate-putamen, have been linked to drug addiction. Moreover, all drugs of abuse that have been

studied induce expression of *c-Fos* in the caudate-putamen, but not all induce this gene in the nucleus accumbens (*1*). Activation of the nucleus accumbens is neither necessary nor sufficient for addiction.

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Scientists in the Courtroom

The 2 April article "Court views engineers as scientists" by Jeffrey Mervis (News of the Week, p. 21) includes a curious quote from Richard Meserve, the attorney for the U.S. National Academy of Engineering (NAE), which filed a brief in the U.S. Supreme Court case *Kumho* v. *Carmichael*. The attorney states that experts "ought to be embarrassed if a judge finds their testimony not acceptable." This presumes that judges are never biased and never misunderstand the scientific issues. Ironically, in struggling with issues of scientific evidence, some courts have promulgated pseudo-science, for instance, holding that it takes a doubling of epidemiological risk to imply that a toxic substance is more likely than not to have caused an individual's disease. This is scientifically false (*1*). The statement that a scientist should be embarrassed by a negative judge's ruling also presumes that other scientists would agree with the decision, and it presumes that higher courts will not overturn the decision. But most important, the statement ignores the fact that scientists are often barred from testifying for reasons having nothing to do with the reliability of their science. Instead, the judge rules that the expert's science is not relevant to the legal issues in the case.

No judgment about a scientist's worth should be made merely on the basis of a statement that a judge barred his or her expert report from a proceeding. This will only discourage scientists from bringing their knowledge to the courtroom.

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Origin of the Japanese Population

I read with great interest the article by Dennis Normile (News of the Week, 5 Mar., p. 1426) about the origin of the Japanese population. Recently, there has been an increasing interest in the origin of Japanese (*1*) and Chinese (*2*) populations. Relevant to this question, we have identified a splicing mutation that causes glycogen storage disease 1a (GSD1a), 727G RT, in the glucose-6-phosphatase gene. The incidence of the mutation in our local Chinese population is 1 in 385 (*3*). This same mutation has also been identified in the Japanese population with an incidence of 1 in 432 (*4*). The 727G RT mutation accounts for a majority of the GSD1a cases in both Hong Kong Chinese and Japanese. Interestingly, this mutation is absent in other ethnic populations. We have also identified the polymorphism 1176C/T in the 3′ untranslated region of the same gene (*5*). All studied 727G RT mutant alleles are linked to the polymorphic marker 1176C in both Chinese and Japanese populations (seven Chinese and nine Japanese; $P = 0.0000047$) (*6*).

Thus, it is most likely that the 727G RT mutations in Japanese and Chinese populations descend from a single event. This observation supports the notion that Japanese and Chinese GSD1a patients share a common ancestor, providing an interesting complement to the anthropological evidence for the origin of these two populations. Our finding is consistent with other recent molecular genetic findings (*1*) indicating that the Japanese derived from the Chinese population. The ancient mutation we describe in the glucose-6-phosphatase gene will enhance the tracking of the origin of the modern Japanese population by determining its prevalence in Chinese from different provinces of China (*2*).

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A Fidgeter's Calculation

J. A. Levine *et al.* (Reports, 8 Jan., p. 212) fed extra calories to human subjects and attributed the lack of weight gain in some of them to their fidgeting. Since my wife describes me as a consummate fidgetarian, I initially thought that one of life's little mysteries, the fact that I do not gain weight easily, had been solved. However, I am also a muscle biophysicist by trade, so I decided to pursue this