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LETTERS

Colon Cancer Screening

In the article "Gene defect identified in common hereditary colon cancer" (Research News, 10 Dec., p. 1645), Francis Collins, director of the National Center for Human Genome Research at the National Institutes of Health (NIH), is quoted as saying, "This is a circumstance where a strong case can be made for presymptomatic DNA testing in the general population."

Hereditary nonpolyposis colon cancer (HNPCC) is a dominantly inherited condition of late onset with a high penetrance. Case finding after family investigation therefore offers an efficient way of recognizing most of the people at risk in the community. Case finding will be much easier than with Huntington's disease or myotonic dystrophy because colon cancers are subjected to pathological examination, and it should be simple to combine this with an assessment of mutations in DNA extracted from a blood sample to identify those patients with colon cancer resulting from mutations in the gene on chromosome 2. (Of course improvement in mutation detection methods will be needed to cope with the volume of work.) Subsequent studies among family members of those shown to have chromosome 2 mutations should yield positive results in 50% of those tested.

If one uses the figures quoted in the article, the first step of this procedure would have a 15% yield and the second step a 50% yield, compared with the 1 in 200 frequency of positive tests to be expected in whole community screening. Only a high mutation rate, unlikely in a disease of postreproductive onset, could negate this logic. A great additional advantage is that all testing would be performed on persons who already have the disease or who are concerned about the disease because a close relative is suffering from it. In this country there is a high level of concern about the possibility of overzealous application of genetic testing, especially on a population-wide basis, and while I share Collins' enthusiasm, one must keep in mind how best to establish efficient preventive health measures without alarming the general public.

David M. Danks
Director, Murdoch Institute for Research into Birth Defects, Royal Children's Hospital, Flemington Road, Parkville, Melbourne 3052, Australia

Response: Danks is quite right to raise concern about the quotation attributed to me, which could easily be misinterpreted. While presymptomatic screening of the general population for HNPCC may not be completely out of the question in the long term, I agree it would be premature to consider such screening at the present time, given the current state of our ignorance. In fact, the National Advisory Council on Human Genome Research (which I chair) has recently issued a statement (*1*) that presymptomatic testing for MSH2 mutations should at present be offered only to high-risk individuals, and even then only in a closely monitored research environment. The joint NIH–Department of Energy Working Group on Ethical, Legal, and Social Implications of the Human Genome Project has endorsed this statement.

Research studies are urgently needed to clarify the technical issues surrounding HNPCC mutation detection, to assess the penetrance of mutant alleles, and to illuminate the complexities of patient education and genetic counseling. A Request for Applications has just been issued by the National Center for Human Genome Research, the National Cancer Institute, the National Institute of Nursing Research, and the National Institute of Mental Health to solicit research projects designed to clarify some of these issues (*2*).

Certainly the initial clinical consequences of testing are likely to be most profound in individuals with a strong family history of colon cancer. But it would be surprising if HNPCC alleles of lower penetrance were not also uncovered. An individual carrying such an allele might still be at considerably elevated risk from colon cancer (and could thus greatly benefit from increased medical surveillance), but might not necessarily have a dramatic family history or a relative who came to pathologic and DNA examination.

These complex scientific issues, as well as questions of cost-effectiveness and the feasibility of large-scale genetic testing and counseling, will have to be satisfactorily addressed before consideration of general population screening for colon cancer. The reasoned and thoughtful input of many groups—geneticists, oncologists, primary care physicians, ethicists, genetic counselors, health economists, and espe-

cially consumers—will be necessary if we are to negotiate these difficult waters successfully.

Francis S. Collins
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Plutonium's Existence

The article "No easy way to shackle the nuclear demon" (Science after the Cold War, 4 Feb., p. 629) by Gary Taubes states that plutonium is an element that did not exist on the planet before it was made in the laboratory in 1940. Plutonium (^{244}Pu) has existed in nature at low concentrations for eons (*1*). Primordial ^{239}Pu would have long since decayed into ^{235}U. Yet in uranium-bearing minerals small quantities of ^{239}Pu are formed by the action of natural neutrons on ^{238}U to form ^{239}U, which forms ^{239}Np through beta decay. After another beta emission, the ^{239}Np becomes ^{239}Pu (*2*). In fact, we now know the isotope ^{239}Pu was formed in natural reactors in western Africa almost 2 billion years ago (*3*).

Plutonium (later shown to be ^{238}Pu) was first isolated in trace quantities by Glenn T. Seaborg and his co-workers at the University of California, Berkeley, on 24 February 1941 (*4*). It was not until spring 1941 that ^{239}Pu, the fissile stuff of nuclear bombs, was isolated and identified at Berkeley. The element with atomic number 94 was not named plutonium until March 1942.

The article also states that all the isotopes of Pu are fissionable. However, of the 16 isotopes of Pu (*5*), only ^{239}Pu is fissile and used in nuclear bombs. The shorter-lived ^{241}Pu is fissile, but not of importance to nuclear bombs.

Finally, the article refers to tritium as being "short-lived." Actually, its 12.35-year physical half-life, although short by comparison with that of ^{239}Pu, is relatively long compared with the life-span of human subjects. For example, after 30 years, about 19% of tritium atoms still remain.

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Fusion Confusion

James Glanz, in his article "DOE lifts the veil of secrecy from laser fusion" (News & Comment, 17 Dec., p. 1811), refers to "Project Matterhorn" as the early name of the U.S. magnetic fusion program. That was the name of the (originally classified) magnetic fusion program at Princeton University, which became the Princeton Plasma Physics Laboratory. "Project Sherwood" was the overall name for the U.S. fusion program (*1*), which in the 1950s and 1960s was virtually all based on magnetic confinement.

The Princeton fusion program inherited its name (and some key players) from an earlier Project Matterhorn, which under the direction of John Archibald Wheeler studied some