icans against any possible hazard from food additives in the food supply. But today, the law may have the paradoxical effect of increasing public health risk. Since that earlier era, there have been enormous advancements in both analytical abilities and our understanding of cancer. It is now recognized that some animal models are not relevant to humans and that substances that induce cancer in animals at high doses should not be presumed to have the same effects in humans at low levels.

In the real world, humans are exposed to small doses of many carcinogens found naturally in foods. The Delaney clause does not apply to these carcinogens in traditional foods, although the body makes no distinction between whether a carcinogen is natural or synthetic. Furthermore, Delaney does not discriminate between potent carcinogens and those that pose a weak or insignificant risk. Thus, enormous resources are being spent to address what can amount to zero risk.

A more enlightened approach to cancer-related regulation focuses on the *mechanisms* by which a substance causes cancer at a particular dose, not solely the cancer endpoint. Such an approach has been recognized by the International Agency for Research on Cancer and by other respected worldwide health authorities.

Delaney also has the unintended and unfortunate outcome of leading individuals to believe that major dietary risks accrue from food additives and pesticides. Yet, scientific consensus overwhelmingly points to consumers' inadequate consumption of fruits and vegetables as a major carcinogenic risk. To the extent that Delaney adversely affects the availability, price, or variety of produce—by unnecessarily restricting pesticide uses or stifling the development of less risky alternatives—it is counterproductive to public health.

The United States is the only nation in the world that regulates carcinogens through a Delaney-type procedure. In more than 20 congressional hearings over 15 years, the National Academy of Sciences, the Food and Drug Administration, the Environmental Protection Agency, and independent health experts have testified on the need to reform Delaney to a negligible risk standard. At a time when resources are becoming ever more limited, Congress can no longer afford inaction. Americans deserve the best public health protection based on sound, scientific policy.

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Transgenic Plants: Effect on the Third World

I read with interest the article by Anne Simon Moffat "Plants as chemical factories" (News, 5 May, p. 659). It is true that, with the introduction of transgenic plants designed to synthesize specific fatty acids, and so forth, the capability of industry

to acquire needed fatty acids for the production of edible oils as well as oleochemicals is greatly improved.

However, there is a dark side to this picture. A number of third-world countries depend on the export of oils produced by their plantations for funds to support their budgets. In the Philippines, coconut oil is a major source of lauric acid for the detergent industries. In Malaysia, now considered a developed country, the very important palm oil industry also supplies lauric acid from the palm kernel oils. Perhaps the marketplace will determine the final outcome of the oils supplied by these countries and those produced by transgenic plants. The immediate impact at present is negligible, but 10 years from now production of oils from the new plants developed by molecular biology may have a severe economic effect on those countries that depend on the sale of their agronomic products for their income.

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Familial Polycythemia

Wade Roush, in his Research News article "An 'off switch' for red blood cells" (7 Apr., p. 27), discusses a recent *Cell* paper from Harvey Lodish's group (*1*) that elaborated on the erythropoietin receptor (EPO-R) signal transduction pathway through Jak2 and hematopoietic phosphatase originally reported by James Ihle's group (*2*) and its possible implication regarding the human disease familial polycythemia. Readers may conclude, we believe incorrectly, that the disease referred to as familial erythrocytosis, originally described as autosomal dominant polycythemia or primary familial and congenital polycythemia (PFCP) (*3*, *4*), is something desirable for the human body and its athletic performance. The observation that a Finnish cross-country skier affected with this disorder won three Olympic gold medals should be tempered by cautious reflection about the pathophysiology of this entity, as such speculation may encourage the practice of "blood doping" (hypertransfusion or parenteral administration of recombinant erythropoietin), which is done by some high-performance athletes.

We originally anticipated that this genetic disease was benign, but more than 18 years of clinical and laboratory follow-up of 10 large families and several sporadic cases of PFCP have indicated that this may not have been correct. The propositus from the original family suffered several cardiac vascular and valvular-acquired problems and died in his 50s of a stroke. His affected son had a myocardial infarction at age 40. Another PFCP propositus suffered an intracranial hemorrhage at the age of 29 (*4*), without having any known risk factors. We are aware of a large Swedish family in which all affected polycythemic patients fare generally poorly and are hypertensive even in childhood. Several affected subjects in the large Finnish family with primary familial polycythemia died at a young age. The quoted example of the Olympic gold medal winner may therefore be a testimony to his superior athletic ability in spite of his primary polycythemia.

Circulation of blood through the organs and tissue oxygen delivery are tightly regulated processes with many feedback controls. At a certain point, elevated red cell mass increases whole blood viscosity with resulting decreased oxygen tissue delivery. Although, by "blood doping," athletes may benefit from an absolute increase in red cell mass, the high hemat-