SWEET TRUTH

What do scientists really know about saccharin? And what does it mean for the regulators?

By Elizabeth M. Whelan and William R. Havender

Saccharin has come to mean much more than an artificial sweetener. For seven years, the Food and Drug Administration (FDA) has been trying to ban its use, acting under laws that give the agency authority to ban any food additive that has been found to induce cancer in humans or animals. And for seven years Congress, prompted by heated consumer opposition to a ban, has prevented the FDA from taking saccharin off the market.

Thus *saccharin* has come to stand for issues of much wider significance than merely whether Americans can quaff diet pop or indulge a sweet tooth. Regulators have viewed it as a test case whose resolution will have a precedent-setting impact on food-safety regulation and the role of regulatory agencies in general. Scientists have viewed it as central to determining the role of science (and hence of scientists) in setting public policy. Consumers have seen it as entailing a serious issue of regulators' power over their lives. And students of public policy have seen this issue as making manifest disagreements over principles of decisionmaking in a free society.

Indeed, these wider considerations probably are more important in determining the attitudes of the various players than are concerns about the actual level of measured risk. Still, the nominal arguments—the ones ostensibly dominating public debate—are almost always couched in terms of science and the law: What is the validity and proper role of high-dose animal tests in predicting human cancer risk? How can long, apparently safe human experience with saccharin be incorporated into a wise regulatory decision? What are the applicable laws and precedents? And so on.

Discussions of value questions, if they are to be profitable, must to some degree be constrained by facts. In this case, a survey of the pertinent science will set bounds within which questions of value can be usefully examined. So it is important to look at the relevant scientific considerations before turning to the value issues raised so poignantly by America's long-running saccharin controversy.

The FDA moved in 1977 to ban saccharin because studies linked high doses of saccharin and the development of cancer in rats. Four independent tests have now shown this, and there are no tests of suitable design that have failed to yield this outcome.

Yet there are two central matters of controversy in the attempt to interpret this finding in terms of human risk. The first of these concerns the large dose used, which in the four tests was the equivalent of a human drinking approximately 1,000 cans of diet pop every day of his life.

The central question is whether such a high dose (just slightly below the dose that would actually poison the animals to death) might itself predispose the animals to develop cancer through weakening, or overwhelming, their resistance. Things might happen in such stressed animals that do not happen at all, or at least not in proportion, at the much lower doses typical of human use. If so, this would have an important bearing on the degree of risk imputed to humans.

For a long time, scientists didn't know

Illustration by Bruce Jones

much that could throw light on this question. But in the last several years they have produced a wealth of pertinent, new information concerning high-dose tests for both cancer-causing agents in general and saccharin in particular.

The most important body of this information has come from tests for the incidence of animal cancer at different doses of various substances. (These dose-response tests still constitute only a small fraction of animal cancer tests, because they are large and difficult to do.) The largest study, which involved 24,000 mice, was carried out at the government's National Center for Toxicological Research. A potent cancer-causing chemical (2-acetylaminofluorene) was fed to the mice in their diet at doses ranging from 30 parts per million (ppm) to 150 ppm.

A significant tumor response was seen only in two organs, the liver and the bladder. Even casual inspection of the figures for bladder tumors showed that the results were compatible with the existence of a "threshold"—that is, a "no effect" level, or "safe dose," below which tumors would not occur or would be unlikely. This stands in contrast to the regulators' normal assumption that the dose-response relationship is linear, with the risk of cancer declining exactly proportionally as the dose level is reduced. In fact, in this study, the incidence of bladder tumors declined much more rapidly in the low-dose range; and using the linear assumption to estimate the risk of bladder cancer at low doses, based on the incidence at high doses, would overestimate the *actual* measured risk by more than tenfold.

The liver tumors, on the other hand, looked on simple inspection to decrease steadily in proportion to dose over the tested range. But even here, close statistical analysis showed this not to be the case; the incidence of cancer in fact decreased more rapidly in the low-dose range. This means that estimating the cancer risk at much *below* the tested range by assuming a linear relationship would overestimate the risk by many times.

Thus, both of the sites showing cancer response in this enormous test were incompatible with the assumption of a linear dose-response relationship. For both sites, things do seem to occur at high doses that do not occur in proportion at low doses. The significance of this finding is that it contradicts a key assumption—that cancer risk is proportional to dose—in estimating the likely degree of human risk from saccharin use on the basis of animal tests.

This finding is by no means isolated. One scientist recently examined the results of *every* reported animal test with a design suitable for assessing dose-response relationships (suitable tests have multiple doses and adequately high numbers of animals). It was found that 31 tests, involving 15 chemicals, met this standard, and all but 4 of the 31 tests had dose-response curves incompatible with the linear assumption.

The results of a new test on saccharin reported in May 1983 further confirm the nonlinear, "threshold" concept. This test used the same two-generation design as the previous three tests on saccharin that had produced tumors. It also used the most-sensitive species, strain, and sex, based on the earlier studies (Sprague-Dawley male rats). The important difference is that this study was specifically designed as a dose-response study, in contrast to the earlier three studies. As in those studies, tumors of the bladder were seen at the higher doses. But the results were dramatically incompatible with the linear hypothesis that cancer incidence is directly proportional to dose. In fact, the results showed that a 100-fold decrease in dose would be accompanied by a 1-million-fold decrease in tumor risk, rather than the 100-fold decrease predicted under the linear hypothesis.

As more and more data accumulate, these strongly suggest, both for high-dose tests in general and for saccharin in particular, that estimating cancer risks at low doses from the results of experiments using high doses tends to overestimate the true risk by many orders of magnitude. This is a *very* significant finding with respect to saccharin. In its estimates of human risks, the FDA relied on the twin assumptions of linear extrapolation from high to low dose and of equal human and rat susceptibility. The agency also used liberal estimates of saccharin consumption by people. Even on these assumptions, the FDA's maximum estimated risks were in the low thousands of cases of bladder cancer annually. Decreasing a number that is already so small by even a *few* orders of magnitude wipes it out.

Predictions of cancer risk from animal tests to humans have never been validated, even qualitatively, and this is the second major area of controversy in interpreting the saccharin-cancer link in rats. This is true of animal tests generally and especially, because of their unusual two-generation design, of the *saccharin tests*. This assertion may well startle the reader, but it is true and bears explanation.

What is the record of rat and mouse cancer tests in predicting cancer generation in humans? The usual response to this question is to reverse its terms and focus on how many human carcinogens have been shown to be animal carcinogens. This is in fact a *very* different question, but the answer to it is that of all the chemicals with a well-defined nature that have been shown to cause cancer in humans, all but one or at most two of them have also been shown to cause cancer in animals.

It is frequently assumed that if the correlation in this direction is so good, then the correlation in the reverse direction is likely to be pretty good, too—in other words, that practically all chemicals shown to be carcinogens in animals must be able to cause cancer in humans as well. This reasoning, however, is logically incorrect; just because all lima beans are vegetables does not mean that all vegetables are lima beans. The crucial question, then, is, How often does a chemical that causes cancer in laboratory animals turn out *not* to be a carcinogen in humans? In short, how often are predictions from animals to humans wrong?

No one knows. This is because a vital piece of information is missing: a list of chemicals that have been thoroughly examined in studies of the incidence of human diseases (epidemiological studies) and have shown indisputably negative results. Just as in animal cancer tests, all epidemiological studies are limited by their size and design and can only potentially detect cancer incidences larger than the statistical limit set by their design. Failing to find a positive result thus will set a "cap," or upper bound, on the possible incidence, but it can never establish that the incidence is perfectly zero. So there are no chemicals that have been adequately studied in humans and that everyone agrees to be completely incapable of causing cancer. This lack means that there is no way to *test* whether a positive outcome in an animal test can lead to a false inference of carcinogenicity in man. For in any likely such instance, one can always argue that the human data, no matter how extensive and no matter how apparently negative, are still "consistent" with a small positive effect below the statistical power of the studies to detect. Thus, we have no way of discovering how often animal tests make *falsely* positive predictions vis-à-vis humans.

And how often have animal tests made *correctly* positive predictions in humans? There are, so far, out of the hundreds of

chemicals that have by now given positive results in at least one animal test, just *seven* substances for which animal data *preceded* the showing of carcinogenicity in humans. But there was no way to know *prospectively* that it would be *these* seven, out of the hundreds of positive animal carcinogens, that would be vindicated by later human data. Obviously, one cannot cite just these few favorable instances to argue that animal tests are in general excellent predictors of cancer risk in humans. The upshot, then, is that we really have no way of knowing how many of the chemicals that are carcinogenic to animals are also carcinogens in humans.

The fact that we have no way to measure the frequency of falsely positive predictions takes on very serious overtones in the light of the policy decision of the regulatory agencies. That decision has been to resolve all questions of scientific uncertainty in the interpretation of animal cancer tests on the basis of "prudence"—that is, by assuming the worst in each contested instance. Such choices include: use of the Maximum Tolerated Dose (MTD) in animal tests so as not to miss weak carcinogens; the assumption that risk and dose have a linear relationship; taking the results in the most-sensitive species and strain and sex as the basis for estimating human risk; ignoring the experience of decades of safe use by humans or ignoring other, negative animal evidence; counting nonlethal tumors as though they were as serious as lethal ones (on the theory that some benign tumors can progress to malignancy and one cannot divine which, or how many, will do so); and many other, similar decisions.

The compounded effect of these choices is to push the decision boundary in the evaluation of an animal test in the direction of drawing a positive judgment, and this is justified by the "prudent" desire to be on the "safe" side. But, as discussed, we cannot know the effect that this shift of decision boundary will have on the frequency of falsely positive judgments. As a result, we simply don't know if *all* we are doing is detecting more and more weak carcinogens; it could just as well be that the truly dangerous substances are being swamped by falsely positive judgments and that we will as a consequence lose all hope of making discriminant risk decisions. This is not without its ill consequences. For the substances that are being removed from use on such "prudent" grounds are all substances that serve some useful human purpose.

There are indirect ways of getting at the problem of making risk decisions. For example, if the decision framework were excessively biased toward getting positive results, then one would predict that an unexpectedly large proportion of the chemicals would come out positive. This is just what we see. Of 252 chemicals tested and reported by the National Cancer Institute (NCI) and the National Toxicology Program (NTP), 42 percent were judged to be carcinogens in at least one species tested. This high proportion is quite unexpected, because the effort to identify and banish human carcinogens has been based on the belief that these substances are relatively *uncommon*; if a substantial fraction of *all* of the thousands of chemicals one comes into contact with each day is carcinogenic, then this approach would be hopeless.

Of course, the high frequency of positive results in the NCI/NTP series could be explained away by saying that these chemicals had been preselected as likely to be carcinogens in the first place and that NCI/NTP had been so successful in their preliminary divination that about half of their candidates turned out in fact to be carcinogens. This may be correct. But the disconcerting fact is that *we don't know*! It could just as well be that the conditions of conducting animal tests (particularly the high doses used), combined with the decision criteria used for interpreting the results of these tests (chosen largely on the basis of "prudence"), are such as to yield a high proportion of falsely positive judgments. In other words, it could just as well be that in human use, most of the chemicals would not be capable of causing a significant amount of cancer.

Allied to this is the finding that many *normal* constituents of the human diet—such as sugar, Vitamins A and D, pepper, and a mixture of egg yolks and milk—are turning out the be carcino-

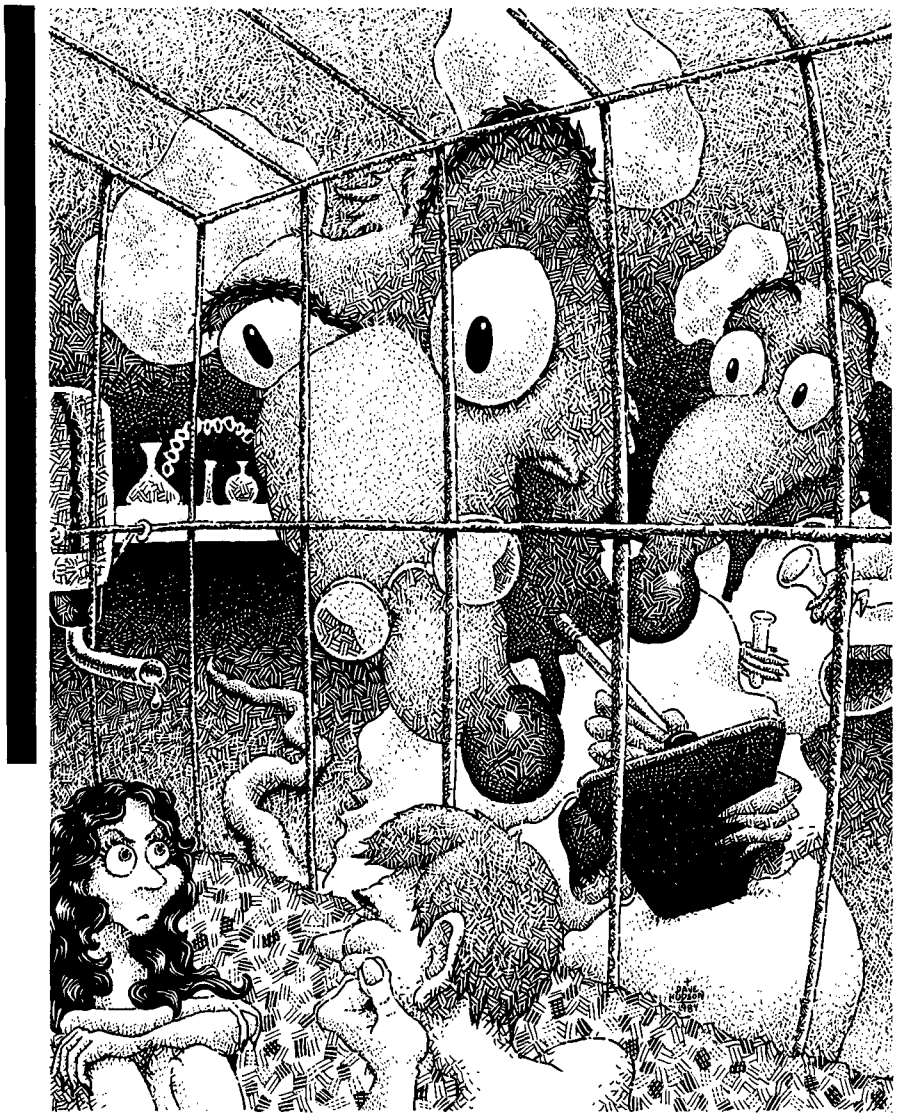

Illustration by Dave Hudson

genic on the basis of at least one animal test. It is not impossible that these animal test results *are* validly implicating these common foods in human cancer. But since such constituents (with the possible exception of pepper) have been part of the human diet for millennia, this would seem to be a very strange conclusion. It seems more likely that experimental conditions (such as dose) and type of species tested are such that these findings don't apply to humans.

A fact making these considerations even more pertinent to the saccharin tests is that no single-generation test of saccharin (where exposure started only after weaning) has been decisively positive; a significant incidence of tumors is only replicably seen in two-generation experimental designs, where the *parent* generation of the test rats is also dosed at the maximum tolerated level, so that exposure starts at *conception.* The predictive difficulty is that we are faced with two conflicting sets of data: the one-generation tests are overwhelmingly negative, while all of the two-generation tests have been positive. Which set should form the basis for inferring human risk?

From a strictly scientific point of view, given available evidence, there is no way to know. Regulators usually resolve this uncertainty by means of "prudence"; they just *assume* that two-generation tests are more sensitive at picking up weak carcinogens. They thus ignore the possibility that by so notching the decision boundary yet another step in the direction of prudence, they may just be raising the frequency of judgments that are actually falsely positive vis-à-vis humans.

Impinging on the matter of predictive validity is the question of whether there exist biochemical singularities in one species of animal that render invalid predictions of carcinogenicity to other species, such as humans. As discussed, we can't directly answer this as concerns predictions from rodents to man. But we can ask how well results with one animal species predicts results for another. If such predictions are pretty good across several species for a given chemical, then one could reasonably argue that the substance is affecting a basic aspect of metabolism that is common to most mammals and hence, it could reasonably be guessed, to humans as well. Many carcinogens show just such behavior: aflatoxin is a carcinogen in mice, rats, fish, ducks, marmosets, tree shrews, and monkeys; 4-aminobiphenyl is a carcinogen in mice, rats, rabbits, and monkeys; asbestos is carcinogenic in mice, rats, hamsters, and rabbits; DES is carcinogenic in rats, hamsters, and dogs. It comes as no great surprise, then, to learn that all of these are also carcinogenic in humans.

But many other animal carcinogens do not show this behavior. Of 190 chemicals tested in the NCI series (tested in rats and mice simultaneously, using similar experimental designs), 54 of the 98 chemicals that were positive in at least one of the test species were positive in *only* one of them. That is, about half of them did not produce cancers in the other species *even* when tested with similar high doses, chronic exposure, and numbers of animals at risk, and when the same set of decision criteria was used to evaluate the results. Considering the taxonomic closeness of these two species and the similar experimental procedures, such a high rate of discordance is surprising, and it suggests that sensitivity of individual species may be stronger than usually assumed. This casts a long doubt on the validity of inferences made about human risk from such animal tests, especially if the predictions are being made to the very different conditions of low-dose exposure (which, as we have seen, is tricky in the extreme), or to a taxonomically widely different species, as humans in fact are.

How often does a chemical that causes cancer in laboratory animals turn out not to cause cancer in humans? No one knows.

Thus, when tests of similar design and statistical thoroughness involving different species disagree, this fact does imply that carcinogenesis cannot be generalized in trying to make a decision about humans. Conversely, concordance among several species strengthens such predictions. The fact that the incidence of bladder tumors in saccharin tests has been high only in *male* rats, never in females tested in parallel, must be seen in this context.

Let us now sum up the animal information about saccharin. It is a carcinogen. However, it is the weakest carcinogen ever detected in an animal test (this is based on the size of the dose needed to induce tumors). The tumors that did develop did not spread, were not lethal, and did not visibly impair the health of the animals (their life spans were normal; the tumors were only detected in microscopic examination after death from natural causes). Further, saccharin's tumor-producing activity is *highly* dose-dependent. Even if one rejects the notion of a "safe dose" in favor of a non-threshold model, the best predictive model projects a risk more than 10,000-fold *smaller* than had earlier been estimated on the assumption of a linear relationship. (And that overestimation itself predicted only one or two thousand cases of bladder cancer per year in the United States.) Moreover, tumors in response to saccharin have been seen in significant numbers only in two-generation tests, the predictive validity of which, in comparison with contradictory one-generation data, has never been established. And carcinogenicity for saccharin has never been demonstrated in any other species than the rat.

Superimposed on all this is the fact that animal tests in general have not been convincingly validated for the purpose of making qualitative human cancer-risk predictions. The frequency of false positives in such predictions is unknown and unmeasureable, but a bothersomely large fraction of chemicals tested under high-dose conditions and evaluated by "prudent" decision criteria is coming out positive. Nor do animal tests have an impressive record of successful qualitative predictions between species even *within* a similar experimental design or to a taxonomically close relative.

All of these considerations point in the same direction: saccharin presents a risk to humans that in all likelihood is negligible, if not nonexistent.

What do available data for humans reveal about saccharin's carcinogenicity? A large number of investigations of various designs have been conducted on artificial sweetener use (saccharin could not usually be distinguished from cyclamate in these studies). Overall, these studies have had persuasively negative results. Two studies do, however, merit special mention.

One was conducted by Canadian researchers and did claim to find, on the basis of a statistically significant but weak result (a shift of only six cases among the several hundred studied would have rendered the result insignificant) a 60 percent elevation of the risk of

bladder cancer among normal male users of artificial sweeteners. This was a startling finding, since up until that time all other studies had been negative.

In an effort to resolve this inconsistency, one of the largest epidemiology studies ever conducted on a food additive was commissioned by the National Cancer Institute in the United States. Some 10 percent of all the new bladder cancer cases in the United States in the year of the study were identified (slightly over 3,000 cases) and matched with some 9,000 controls. If the Canadian claim of a 60 percent increase in risk among men (or women, for that matter) were correct, this much-larger study would certainly be able to detect it with statistical confidence. The study, however, was negative; there was no elevation of risk among typical users of artificial sweeteners.

While invalidating the Canadian study, this result does not, as discussed earlier, prove that saccharin causes no cancer in humans whatever. But it does set an upper bound on the maximum number of such cases that might exist. Were there an excess of bladder cancer cases as large as 3,000 in the United States each year due to the use of artificial sweeteners, then it would in all probability have been detected in this giant study. Not detecting any such excess, therefore, means that if saccharin in its past patterns of use does cause bladder cancer, it does not cause a number significantly larger than about 3,000 cases annually.

So much for what science can tell us. It is within these boundaries that value considerations can be dealt with.

A word or two about saccharin's *benefits* is in order at this point. The salient fact is that there is little or no convincing evidence of an "analytical" sort—that is, accessible to outsiders, such as "experts" and regulators—that any of the health benefits claimed for saccharin actually exist. These benefits include weight reduction or maintenance, management of diabetes, and reduction of tooth decay. It is highly plausible that some such health benefits may exist for some people, but evidence to confirm or refute this is lacking.

It would be a mistake, however, just because of this state of affairs, to *assume* that no such health benefits exist. They might; and because saccharin's cancer risk to humans is at most very, very low (there is *no* uncertainty about this point, since it is based on the epidemiology results), these benefits would not have to be very large to completely offset saccharin's risks. A few thousand deaths avoided per year for all reasons combined (diabetic crises avoided, heart attacks avoided because of successful weight management, cancer avoided because of obesity reduction—yes, obesity per se is a risk factor predisposing one to develop cancer) would be sufficient for this purpose. It is difficult even to imagine in principle how one might design a study that could decisively rule out the possibility that health benefits of this small magnitude exist as a result of the ways that diet products are currently consumed. For this reason, the strategy of using prudence as a decision rule for dealing with uncertainty fails in this instance; faced with the choice to ban saccharin or not, a prudent regulator could not know which alternative would in fact favor public safety. As a matter of fact, the benefits could be quite a bit larger than just a few thousand lives saved each year, because there aren't any studies of benefits of sufficient rigor to set an upper bound on the size of the possible benefits. On the other hand, since the risks are no more than a few thousand cases of bladder cancer, prudence might well come down on the side favoring saccharin's availability.

Faced with the choice to ban saccharin or not, a prudent regulator could not know which alternative would favor public safety.

There is a series of considerations that lead, irresistibly in our opinion, to a specific policy recommendation about saccharin. It is that individuals be allowed to make their own decisions about saccharin use.

The first consideration is that this is not a situation similar to air pollution or acid rain or the contamination of the land with pesticides, where externalities—side effects imposed on unwilling recipients—prevent individuals from taking independent action to control their own fates. Instead, it is predominantly a *user-risk* situation, where users are generally the only persons exposed and can choose whether or not to take the risk. (There are a few exceptions to this generalization that will be dealt with below).

Second, there are, at least hypothetically (because the existence of *any* risks or benefits is speculative), great deviations from the average in the risk-benefit circumstances of individuals. Pregnant and lactating women may run an elevated risk from saccharin, since their fetuses or newborn infants might be more sensitive to the effects of saccharin. Children in general might be at elevated risk, because they have such a large life expectancy ahead that an induced cancer would have ample time to develop. By the same token, older people would probably have a reduced risk, since their remaining life expectancy is sufficiently short that a cancer induced by saccharin would have little chance to become manifest. Adult women probably have a less-than-average risk, because *all* of the rat tests to date have found no significant tumor incidence for females, and all of the epidemiology studies (including the Canadian investigation) have found no measurable risk to normal women users.

Variation on the benefit side is likely as well. People who are obese are at elevated risk for diabetes, heart disease, cancer, and other conditions; so they are in a position to derive more benefit from using saccharin than would a person of normal weight. Diabetics, too, are in a position to benefit more than the average. And since there is a tendency for people to gain weight and develop diabetes concurrently as they get older, their potential for reaping a health benefit increases in step with their decreasing vulnerability (because of shortening life expectancy) to its risks.

This variability in individual circumstances makes it difficult to see how a ban on saccharin's availability could, in its uniformity, be superior to a policy that makes allowance for such variations. One could in principle try to take a "utilitarian" position—adding up all the health benefits and risks to derive an overall sum for the society as a whole, and, depending on whether the sum is positive or negative, deciding whether to ban or not. But this suffers from the distributional flaw, which has been exhaustively discussed in the political science and philosophical literature, that *one* person's health benefits get needlessly sacrificed to save *someone else* from risk. Such sacrifices are wholly unnecessary in a situation where the risks and the benefits are born predominantly by the same individual. To be blunt about it, a person who is in a position to benefit from saccharin and is coercively prevented from

obtaining these benefits in a peaceful manner *has his rights violated*—precisely because this is primarily a user-risk situation, and so his obtaining these benefits does not impose harm on others.

The third consideration leading to a user-decides policy is that the knowledge of these particular, individuated risk-benefit circumstances necessarily exists in a dispersed form across the social order. There is no hope of gathering all knowledge of local risk-benefit variations—which are specific for each individual—into a central decisionmaking office. Not only the knowledge is dispersed, but so is the motivation to use it optimally. And, as Austrian school economist and political thinker F. A. Hayek argues in a parallel context, the best way to assure that this necessarily dispersed knowledge is effectively used, rather than just lost, is to decentralize the decisionmaking authority out to where the knowledge of local circumstances exists. In this case, that means letting each person make the decision about the use of saccharin for himself.

The fourth consideration is the political philosophy that underlies the Declaration of Independence and our Constitution. Suffice it to say that, based on these documents, citizens' self-autonomy has been the norm in large areas of civic life. Customarily, individuals have been left to decide what risks they are willing to take. Why treat saccharin in a manner that deviates from that custom or establishes dramatic new precedents?

The discussion to this point establishes that saccharin does not have any distinguishing features sufficient to justify treating it in an unusual way. Quantitatively, its risks—at most, about 3,000 cases of cancer a year—are absolutely minor compared to other risks that we freely permit people to decide for themselves. We let people drive cars, climb mountains, ride motorcycles, live in earthquake zones, and so on; and if risk decisions of such magnitude can be entrusted to them, there is no reason to make an exception for saccharin. Qualitatively, the same is true, too. The salient qualitative feature of saccharin's hypothesized risk is that it is cancer. But we freely allow people to make decisions for themselves about smoking, drinking, sunning themselves, when to have their first child, the number of their sexual partners, and how obese they become—all of which are proven risk factors for cancer. And given the fact that we permit people to make decisions for themselves about *proven* factors of such magnitude, there is no reason for treating saccharin, whose risk to humans is entirely hypothetical, differently.

The fifth consideration, an extension of the previous one, deals with the exceptions to the general rule that the costs and benefits in this case are virtually all borne by the same person. Certain exceptions are obvious: pregnant or lactating women may influence a fetus or infant by their decision to use saccharin. And other groups in society, such as children, orphans, or the senile elderly have decisions made for them by others. Again, however, traditional ways to delegate decisionmaking already exist to cope with these situations, and there is nothing about the hypothesized risk of saccharin that would call for any change in these customs. Primary responsibility for a fetus's well-being has always been delegated to the woman carrying it; we allow her ultimately to decide whether to smoke, drink, take drugs (medicinal and otherwise), wrestle, ride horses, jog, stay up late at night, and so on. Seen against this background, there is no basis not to permit her to make the final decision about saccharin, too. Similarly for lactating mothers. Decisions concerning older children, again, have been traditionally, in the main, delegated to their parents. And for the senile, or orphans, decisionmaking has customarily been delegated to guardians, relatives, doctors, nursing-home administrators, and so on; nothing now known about saccharin's risks suggest the need for any change in these arrangements.

The sixth consideration is that the argument for locating ultimate decisionmaking power concerning this issue in the individual does *not* depend on the assumption that he will always make "informed" risk-benefit judgments of this "complex" issue. What, after all, is complex about it? *Whatever* decision an individual makes regarding saccharin, he cannot hurt himself by much; the worst he could do to himself is absolutely trivial compared to many other decisions he has to make in the course of a day. Rules of thumb, whims, habits, folklore, and so on, are quite likely to lead to decisions about saccharin use that won't be demonstrably inferior to "informed" decisions in terms of effects on the individual. And this means that we don't have to presuppose vast amounts of erudition or reasoning skill on the part of the decisionmakers. Any citizen able to lead an independent life can be presumed capable of deciding whether or not to use saccharin. We don't require people to prove that they can make "informed" decisions before we let them choose a spouse, occupation, doctor, religion, or politician, nor to buy insurance or take out a mortgage, so there is no reason to get pompous about letting them make the decision to drink diet pop.

Seventh, precisely because the decision need not be well-informed, there seems to be no need for exaggerated or special methods for imparting information to consumers about the risks and benefits of saccharin—as long as the risks remain as small as now is the case. The existing channels for spreading new information—consumer groups, the media, etc.—seem adequate to impart a sufficient amount of knowledge for this minor decision.

Eighth, there is an important dimension to this matter that transcends the issue of whether or not saccharin poses a serious health risk. Is it the proper role of government ever to regulate such substances, whatever risks their use might pose for particular individuals? Such regulatory policies assume that the judgment of regulators and "experts" ought to prevail over the choices of individuals, who are deemed too ill-informed or too foolhardy to make wise decisions about such matters.

One could rebut this assumption by invoking the argument that only individuals (perhaps in consultation with their doctors, family, etc.) have sufficient knowledge of their particular circumstances to assess the risks and benefits of a given course of action. But the issue goes beyond whether individuals are capable of making wise decisions. Ought governments to *prevent* individuals from making bad decisions or from taking risks? Is it the government's role to protect individuals from themselves?

In the tradition of individual rights and limited government, it is the business of government to protect individuals from being harmed by others. It is not the business of government to prevent individuals from pursuing actions that may result in harms only to themselves. Such restrictions erode freedom of choice and individual responsibility, essential ingredients of a free society. ◻

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BIPARTISAN B·A·L·O·N·E·Y

An industrial policy will mean wise regulation by independent experts? History tells otherwise.

BY MARVIN N. OLASKY

Some of Gary Hart's "new ideas," like the former Democratic presidential candidate himself, seem on their way to becoming footnotes in American political history. But others, like the establishment of a "grand coalition" of economic and political leaders—representing both major political parties—who would unite to create greater governmental economic authority, are on display in this year's Democratic Party platform.

While many eyes are on vice-presidential nominee Geraldine Ferraro—the campaign season's big attention-getter—the party of the people is demanding appointment of an "economic cooperative council" and endorsing "industrial strategies to create a cooperative partnership of labor, capital and management." And the House Banking Committee already has endorsed legislation to create an economic council, which would include businessmen, labor leaders, and academicians.

The fashionable buzzword describing the notion of a single governmental economic authority had been, of course, "national industrial policy." Now, as the election campaigns gear up, Hart's "grand coalition" may become a front-runner among the linguistic candidates to popularize this latest version of central planning. But while the phrases in which the idea is couched may be of recent vintage, the idea itself, like many others in all the election-year rhetoric, is actually quite old—at least as old as Plato's *Republic*, with its ideal of "philosopher kings."

The reasons behind the popularity of such a Platonic concept in this notably Aristotelian age become more apparent if we compare the "grand coalition" idea not only to its predecessors among the ancient Greeks, but to its American ancestors, as well. After all, the *United States Government Manual* lists 57 such small-scale "grand coalitions"—bipartisan boards designed to oversee entire industries or activities and remove their particular attempts to pursue happiness from the supposedly dreary intervention of the populace and its elected representatives. These baby-grand coalitions are called "independent agencies."

Their charters vary widely and include everything from the trivia of the American Battle Monuments Commission to the judgments of the Federal Trade Commission or the Securities and Exchange Commission, which can make or break companies. But this bureaucratic Heinz 57 has two common denominators. First, all of these agencies are outside the ordinary executive-branch chain of command used to manage the Cabinet-level departments (State, Defense, etc.). Second, all of these agencies are more or less patterned after the first major independent agency, the Interstate Commerce Commission. The ICC was created almost 100 years ago, largely upon the suggestions of early public-relations "experts" whose views were similar in some ways to those of our latter-day speechwriters.

To understand the similarities, we need a quick look at the political economy of a century ago. The 1880s are sometimes looked back upon as a time of great national optimism and excitement concerning the industrial and mechanical advances that were becoming evident almost every year. Such reminiscences are correct, but economic advance also was accompanied by social pressure—and pressure created worry about the growth of the first genuinely national business enterprises. There especially was concern about the development of the "steel horses" and steel lines—the railroads—which provided the transportation base for America's rapid economic growth.

Into this thicket of worry swashbuckled the railroad entrepreneurs of the time, fulfilling societal needs by laying more track faster than anyone would have imagined possible a few years before and, through their efficiency, opening up new lands for small farmers and prairie immigrants. But they also raised concerns about a possible centralization of economic power. The railroad industry had grown fast, going from 30,000 miles of track during the Civil War to 141,000 miles in 1882. With such a great leap noticeable to all, the railroads quickly became the focal point for concern about rapid industrialization and the new organizational forms that accompanied it.

Naturally, the political and economic powers of the time attempted to make the new economic structure work to their advantage by obtaining special privileges from the state. For instance, some Chicago merchants became irate because they could not get special rates from the Chicago, Burlington and Northern line. Chicago businessman William H. Beebe told a Senate investigating committee that he did "not lean very much toward paternal legislation on the part of the Government," but felt that *in this case*, "regulation by a commission or by some other governmental agency would be beneficial." Similarly, when Pittsburgh merchants were unable to get a special deal from the railroads, the Pittsburgh Chamber of Commerce called for federal rail regulation.

Political pressure from those generally favoring statist solutions also grew during the 1880s. As labor upheavals resonated through the decade, with some demonstrations leading to violence, there were claims that the American flag was unraveling stripe by stripe, and only strong governmental hands could sew it together.

The initial response of railroad-industry leaders was not a principled defense of their business, but a series of not-so-subtle attempts to swing public sentiment to their side. William K. Acker-