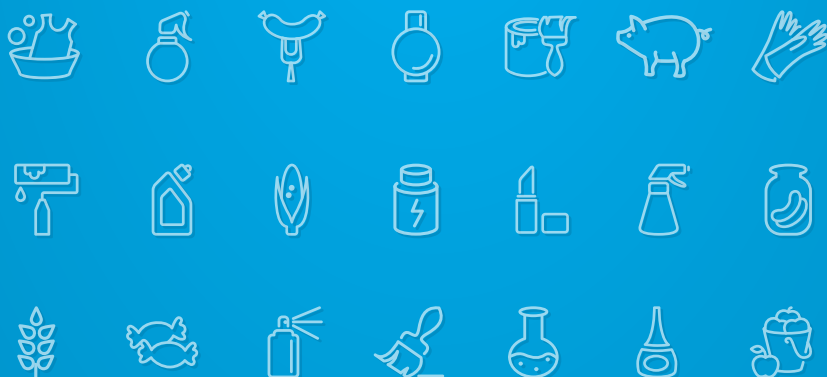


A Consumer's Guide to **CHEMICAL RISK**

Deciphering the "Science" Behind Chemical Scares



Angela Logomasini, Ph.D.

COMPETITIVE ENTERPRISE INSTITUTE | 2014

A Consumer's Guide to Chemical Risk

**Deciphering the “Science”
Behind Chemical Scares**

Angela Logomasini

**Competitive Enterprise Institute
2014**

Executive Summary

A constant barrage of news headlines suggests that synthetic chemicals—even some naturally occurring ones—are responsible for nearly every public health problem imaginable, sowing fear and confusion among consumers. This publication is designed to reduce both the confusion and fears about chemicals by providing consumers with some insights on the science and the politics behind the headlines.

When it comes to studies attempting to link chemicals and health ailments, the absolute risks are largely theoretical and generally too low to measure. In fact, many of the alarmist new stories report on studies where the findings are very weak and often inappropriate for drawing any conclusions. Accordingly, rather than panic about scary headlines, consumers can seek answers to some fundamental questions such as:

- **Is the association strong and statistically significant?** Most of the studies cited in the news are merely statistical analyses that assess whether two factors occur at the same time. They do not prove cause-and-effect relationships, which can only be inferred when the association is very strong. Accordingly, if researchers report a “weak” or “suggestive” association, consumers should be suspicious of the claims.
- **Is the sample any good?** Ideally, a randomly selected, large sample of a population provides the best chance of good data, but locating and developing such samples is difficult and expensive. Accordingly, researchers often work with less-than-ideal samples and existing databases that offer imperfect data, or both. Accordingly, a study with 1,000 randomly selected subjects is more reliable than one with 100 subjects cherry-picked from a database.

- **Are there serious confounding factors?** The possibility that a factor other than the two variables in question is responsible for the result is always present. While scientists attempt to apply “controls” in studies in an effort to negate the impact of such confounding factors, it does not always work. Consumers should be suspicious when there are other factors that more likely contributed the result.
- **What is the potential for recall bias among study participants?** Some studies require interviewing subjects about their personal behavior, sometimes expecting them to recall chemical exposures dating back decades. The subject’s failure to recall the facts accurately can so undermine the validity of the data that the final study results are completely off the mark. Consumers should be wary of studies that rely on this type of subjective data collection.
- **Does the language used by researchers suggest bias?** Good researchers will strive to keep their biases in check, while still working toward finding something interesting. Yet, others add “spin” to weak and meaningless “findings” to garner publication and media interest—and more funding. For example, researchers trying to prove that trace chemicals can make us fat have captured headlines by labeling these chemicals as “obesogens.” Their science may be weak, but their marketing it this way garners lots of media coverage.
- **Is the study relevant to humans?** Tests on rodents involve administering massive amounts of chemicals to animals bred to be highly susceptible to cancer, and many form tumors as a result. Despite what headlines may suggest, such tests are not particularly relevant to risks associated with human exposures to trace levels of chemicals.

- **Is the exposure significant enough to matter?** Many substances that are helpful or benign at low levels can sicken or kill at high levels. Accordingly, if the study involved high exposures, consumers should question whether it is relevant to trace exposures through consumer products.
- **Is the study peer-reviewed and published?** Peer review is designed for an industry to self-regulate to reduce fraud and poor quality research. While it alone is not sufficient to assure a study is completely sound, consumers should be very skeptical of claims from studies that have not undergone peer review.
- **Can other researchers reproduce the study results and have they done so?** Science is a long process of discovery that brings us closer to an answer as an issue is examined time and again. Part of that process involves repeating specific studies to see whether different scientists or teams of scientists can reproduce results of their peers' or even their own research. If data are unavailable or other researchers have not been able to reproduce the result, the study is less compelling and may be discredited.

In addition, consumers should be alerted to sensationalist hype by simply paying attention to certain terms used by those in the alarmist trade. For example, many times, activists will call for bans or hype risks about products that contain trace amounts of chemicals that are “classified carcinogens.” Government and scientific bodies around the world have developed such classification systems to indicate that *at some exposure level* and *under some circumstance* a chemical *might* increase cancer risk. Such listings do not mean that the chemicals cause cancer in

humans exposed to trace amounts found in consumer products.

Similarly, some environmental activists raise fears about chemicals by claiming that most cancers are related to “environmental factors.” It is true that cancer researchers blame “environmental factors” as causing most cancers, but they define these factors as anything *but* genetics. According to the landmark research conducted by Richard Doll and Richard Peto, environmental factors include tobacco, dietary choices, infections, natural radiation, and reproductive behavior. Trace chemicals in consumer products are not a demonstrated cancer source.

Consumers need not panic about any particular study, nor completely dismiss studies because of uncertainty. Rather, we should focus less on any single study and more on what the larger body of research indicates about a particular issue. In addition, we should remain wary of headlines and anyone claiming to have changed the nature of the debate with a single study.

INTRODUCTION

Will these chemicals make me fat? That sounds like a weird question, but some consumers may actually have such worries, thanks to a constant barrage of news headlines claiming that synthetic chemicals—and even some naturally occurring chemicals—are responsible for nearly every public health problem imaginable, from obesity to heart disease to diabetes and cancer. Lately, many of these news stories have focused on the chemical bisphenol A (BPA). For that reason, this guide uses many BPA studies as examples. But BPA is just the latest victim of the hype, and the data do not warrant the alarmism.

Consumers may be skeptical of many of the allegations, but nonetheless remain fearful. A 2013 poll produced for the Independent Women's Forum, for example, showed that while 60 percent of women are skeptical of the groups leading the anti-chemical crusades, 79 percent said they were concerned about the health impacts of chemicals.¹ Perhaps if consumers had access to better information to more critically evaluate headlines and scientific research, those fears would subside.

This guide is designed to help consumers decipher the real meaning behind the many terms and creative phraseology that the media and anti-chemical activists regularly employ. First, it reviews some of the general concepts that underlie sound science. Second, it provides an overview of the common terminology that activists use to mislead consumers about risk, providing some examples of the same.

A large percentage of studies condemning chemicals are based on statistical analyses that attempt to correlate health effects with chemical

Epidemiologists use statistics to compare risks between groups in search of a discovery as to whether two factors often happen at the same time.

exposures among both humans and animals. The tests involving human subjects comprise the field of epidemiology. In contrast, animal testing seeks to find associations between chemical exposures and health effects in animals, and then employs the additional step of extrapolating those risks to humans. This paper commences with an overview of both areas of research.

EPIDEMIOLOGY OVERVIEW

So you hear that a chemical doubles cancer risks among a certain segment of the population. What does that really mean? Such findings often come from epidemiological studies that measure the relative risk of belonging to one of two or more groups. Epidemiologists use statistics to compare risks between these groups in search of an association—a discovery as to whether two factors often happen at the same time. These studies by themselves do not establish a cause-and-effect relationship, but the stronger the association, the more compelling the case is for a potential cause-and-effect relationship.

Types of Epidemiological Studies. Researchers employ four main types of epidemiological studies:

1. Clinical;

2. Cohort;
3. Case-control; and
4. Ecological.

Clinical studies provide the most reliable results, followed by cohort studies, case-control studies, and then ecological studies. Clinical studies rely on experimental data collected through controlled experiments, while others rely on observational data from existing sources—such as polling subjects on health history, census data, or other databases.

Clinical studies are the most reliable because researchers have greater control of the data and can simultaneously study people exposed to the substance being studied and “control groups” of people exposed to little or none of the substance. When it comes to chemicals, such as pharmaceuticals, clinical studies involve administering known amounts of the drugs to human volunteers under controlled conditions, observing the effects, then comparing results to a control group with no or significantly lower exposures. For example, a clinical drug trial might include assessing the health of carefully selected volunteers both before and after administering the drug. These results would then be compared to a similar group given a placebo.

Clinical trials to assess the danger of non-pharmaceutical chemicals are limited in scope because of the ethical issues associated with tests involving human subjects. Thus, human testing is limited to low-risk, low-impact exposures, such as assessing the potential of skin irritations from insect repellents.

Estimating exposure to chemicals requires interviewing study participants who may express their own biases or may not recall the facts accurately.

studies.

Given the ethical limitations of human testing, most non-drug-related studies rely on observational data that estimates existing chemical exposures and disease prevalence. “Cohort studies” are a type of observational study that follows a group of people, known as a “cohort,” over a number of years, measuring their exposures to chemicals found in their environment (rather than administering chemicals) and assessing the health of each study participant. By tracking a population over years, often decades, researchers examine whether highly exposed individuals are more likely to develop health ailments. Because cohort studies track exposure starting at the present and into the future, these studies are also classified as *prospective*

Many complications arise in drawing conclusions from these studies, because of confounding factors, which are discussed in greater detail below. In addition, estimating exposure to chemicals requires interviewing study participants who may express their own biases or may not recall the facts accurately.

Another type of observational research is case-control studies, in which researchers identify individuals with certain diseases and attempt to

correlate those illnesses with prior exposure to chemicals. Researchers may have to rely on individual recollections related to chemical exposures that may have occurred decades earlier. Such accounts are often unreliable and further weaken the findings. Because these studies attempt to quantify past exposures, they are classified as *retrospective* studies.

Finally, the weakest of all are ecological studies. These are observational studies that compare large populations rather than individually track participants divided into groups. An example of an ecological study would be a comparison of health statistics in Europe against those in the United States. While the populations are large, the ability to control confounding factors is very limited because there are just too many of them.

It is difficult to draw conclusions about individuals within ecological study populations based on statistical associations that researchers may discover between the two groups. Drawing such conclusions is called an *ecological fallacy*. For example, if a study finds that Europeans have lower heart disease rates, it would be an ecological fallacy to assume a specific European would have a lower risk of heart disease than a random American. "Ecological studies can provide useful clues, but conclusions about individuals are in general only weakly supported by data on groups," explains the late David Freedman, professor of statistics at the University of California, Berkeley.²

Relative Risk. Researchers measure the strength of associations by assessing the *relative risk* of a chemical. This involves comparing groups of individuals with high exposures to groups of individuals with low or

low or no exposures. In the observational studies, if the group (or groups) with higher exposure to a chemical experiences more health ailments, researchers report an association between the chemical and the illnesses they discover. They then engage in calculations to express the strength of that association numerically as a risk ratio. If the risk ratio is 1, then the study reports no difference between the groups. If the level is higher than 1, the research indicates a statistical association between health problems and the chemical exposure. When the number is less than one, the research indicates an association between high chemical exposures and better health—what drug researchers seek when conducting drug trials.

Steve Milloy explains it well in *Junk Science Judo*:

A relative risk of 1.0 means there is no difference in the rate of disease between two study populations. A relative risk of 2.0 means that the study population with the exposure of interest has double the rate of disease (100 percent more) than the non-exposed population. A relative risk of 3.0 means the rate is three times as high (200 percent more) ... and so forth.³

However, relative risk numbers reveal only the strength of an association, not actual risk levels. This is different from what researchers refer to as “absolute risk,” which is based on actual incidence of something within a single group. For example, if one out of 1,000 Americans contracts a disease annually, that individual’s absolute risk would be 0.1 percent or expressed as a ratio of 0.01. If we compare risks associated with a smaller population with twice the level of illnesses than experienced in

our sample of 1,000 Americans, we find a relative risk of two, which indicates 100 percent more illnesses in this second group than in the larger group. In this situation, the absolute risk of being in the smaller group increases to 0.2 percent or 0.02.

While a 100 percent increase in illnesses might sound like a large increase, studies finding a relative risk of 2 or even 3 are not particularly compelling and are considered weak associations. Mount Sinai School of Medicine epidemiologist Paolo Boffetta explains: “Although any measure of risk would follow a continuous distribution and there are no pre-defined values that separate ‘strong’ from ‘moderate’ or ‘weak’ associations, relative risks below 3 are considered moderate or weak.”⁴

It is true that when risks are high, relatively small increases may translate into a large number of people facing a higher risk. But what if the absolute risk of the first group is extremely low? Being two, three, four times or even 10 times more likely to suffer effects of a negligible risk may still mean that an individual's absolute risk remains negligible.

Sarah Williams of the United Kingdom's nonprofit cancer research institute, Cancer UK, notes:

Recently, the *Guardian* looked at a “70% increase” in cancer among women after the Fukushima Daiichi nuclear disaster. This was actually drawn from statistics showing an increase in absolute risk from 0.77 per cent to 1.29 per cent (this is indeed a 70 per cent increase). But, as reported by the *Wall Street Journal* ... the absolute increase is ‘tiny’ – about 0.5 per cent.⁵

In *Statistics Explained*, Professor Steve McKillup of Central Queensland University uses the lottery as an example of how even seemingly big increases in relative risk (in this case chance of winning) do not dramatically change probability. If buying a lottery ticket gives you a one in 10 million chance of winning, you can increase your chances 10 times simply by purchasing 10 tickets. Yet the probability that you will win is still very low (one in 1 million).⁶ Similarly, the risk of being killed by a lightning strike is very low at about 0.25 per 1 million people. So for example, people in Kansas face a risk that is more than double, but that is still only 0.57 percent per million.⁷

When it comes to studies attempting to link chemicals and health ailments, the absolute risks are largely theoretical and generally too low to measure. That is one reason not to panic when studies report an association showing that one group of individuals exposed to certain chemicals suffered two or three times more health problems than people in other groups.

Statistical Significance. When researchers do find an association, they then try to determine whether the finding is statistically significant. Statistical significance is the attempt to quantify the probability that research findings were merely accidental rather than the result of a real relationship between two variables in the study. It is usually expressed as a percent and is referred to as the p-value, with “p” standing for “probability.”

A p-value of 1 or $p=0.01$, suggests a 1 percent probability that the results occurred by chance, and that the researcher is 99 percent confident in the results (confidence interval of 99 percent). Likewise, $p=0.05$ suggests a 5 percent probability that the results are due to mere chance and 95 percent confidence that they are correct, and so on. Studies with a p-value less than 0.05 are usually considered significant, while those above that mark are not.

The fact that a study's findings are statistically significant does not by itself establish a cause-and-effect relationship. Even statistically significant findings may occur by mere chance, failure to control confounding factors, researcher bias, and many other causes.

In fact, some researchers point out that small changes in the p-value can move a study from significant to insignificant pretty easily.⁸ One study title highlights an ironic conclusion: "The Difference Between 'Significant' and 'Not Significant' is not Itself Statistically Significant."⁹ Indeed, the 0.05 cutoff is itself arbitrary and prone to be abused. A study of psychology research papers, for example, showed that a large portion reported p-values just under the 0.05 cutoff for significance, indicating that researchers regularly work the data to push their findings into the sig-

Even statistically significant findings may occur by mere chance, failure to control confounding factors, researcher bias, and many other causes.

nificance category, thus increasing their chances of publication in a peer-reviewed journal.¹⁰

Philosophy professor Mark Battersby of Capilano University in British Columbia explains how using the term “significant” can confuse some people about the importance of study results:

It is unfortunate that statisticians chose such a loaded term as “significant” to describe what is merely a probabilistic judgment that difference between the two sample groups is unlikely to be the result of chance. Many results that are statistically significant simply aren't significant or important in any ordinary sense. And sometimes the lack of statistical significance is more medically or humanly important.¹¹

CHALLENGES TO EPIDEMIOLOGICAL RESEARCH

In addition to the task of finding an association and then proving it statistically significant, researchers face a number of other challenges to establishing the validity of their findings. The following provides an overview of some of these concerns.

Random Samples. Finding reliable data—a representative sample of the population under examination—is one of the researcher's most challenging tasks. Ideally, a randomly selected large sample of a population provides the best chance of good data, but locating and developing such samples is difficult and expensive. Accordingly, researchers often work with less-than-ideal sample sizes, existing databases that offer imperfect data, or both.

For example, many studies on the chemical BPA rely on data from the National Health and Nutrition Examination Survey (NHANES),¹² a Centers for Disease Control and Prevention (CDC) program that assesses national health trends. CDC collects health data from a different group of volunteers every year via physical exams and interviews. In addition to recording the volunteers' health ailments, the data also measure BPA in urine and blood. Numerous BPA studies pull the data from various years to see if there are correlations between certain illnesses and levels of BPA in the volunteers' urine. But the data involve a one-time measurement of BPA, which varies considerably in the body over just hours. These data tell us nothing about overall exposure and hence are inappropriate for drawing conclusions about BPA risk. Yet there are

dozens of studies that rely on BPA data from NHANES data published in peer-reviewed journals, reporting statistically significant associations. While these studies make headlines, they do not offer much scientific insight.¹³

Using data from the NHANES and other databases offers researchers myriad opportunities for “data mining”—selectively pulling data out for statistical studies with the hope of finding meaningful relationships. Reasons for researchers manipulating such data include the desire to generate positive and interesting results or to increase publication possibilities. Rather than producing a truly random sample, data mining may involve excluding certain participants as “outliers” or pulling out select data subsets from the larger database. In that case, the sample may reflect the researcher’s bias instead of constituting a truly random sample. The problem can be so bad that some have referred to it as “data torturing.”¹⁴

Sample Size. The size of the study sample is also very important, with smaller samples more likely to discover associations by mere accident. A study that includes more than 1,000 participants exposed to high levels of a chemical is stronger than one assessing exposure among a sample size of only 100. Furthermore, one small study is not made stronger if other studies with small samples report the same results. Small-scale studies are still produced, largely because of the cost associated with collecting more data, but they are of limited value.

Researchers may attempt to increase the strength of the smaller studies by grouping the data from many different studies to run a new analysis. These so-called meta-analyses may add value to a field of science, but have their own pitfalls. Selection of the studies for which data are pooled raises a host of issues from researcher bias when selecting studies, varied data quality, difficulty finding truly heterogeneous data worthy of pooling, biases toward using only studies with positive findings, and more. As Battersby notes: “So meta-analyses, which tend not to include negative data, may be biased towards finding a correlation or finding a greater degree of correlation than a total aggregation of the research would justify.”¹⁵

Meta-analyses also provide opportunities for scientific abuse, as some researchers may try to validate weak research by pooling it with a selection of other poorly designed studies. If the data and designs of the studies going into the analysis are not sound, a meta-analysis will not fix those problems.

Confounding Factors. Statistics only measure associations rather than cause-and-effect relationships. Therefore, the possibility that a factor other than the two variables in question is responsible for the result is always present. For example, a statistical analysis might find that people who spend a great deal of time in bars have a higher cancer rate. Does that mean that going to a bar can give you cancer? Probably not. Instead, it might mean that smoking rates are higher among people who spend time in bars. Hence, smoking, not going to bars, would be the problem.

*Recall bias can
so undermine
the validity of
the data that
the final study
results are
completely off
the mark.*

While scientists attempt to apply “controls” in studies in an effort to negate the impact of confounding factors, they do not always succeed. Some researchers admit it is not really possible. For example, a study reporting an association between the chemical bisphenol A and obesity admitted that it could not control for the fact that the obese children had elevated BPA exposures because they probably had a higher caloric intake. BPA exposure comes largely from packaging for processed food, which tends to have higher calories. In that case, it is more likely that the real association they measured was between higher calories and obesity, with BPA being largely irrelevant.¹⁶ But that is not what we read in the headlines.¹⁷

Recall Bias. Some observational studies, particularly retrospective case-control studies, require interviewing subjects about their personal behavior, sometimes expecting them to recall chemical exposures dating back decades. Many times, individuals suffering from a health ailment might exaggerate their exposure level since its natural to look for an explanation for illnesses the causes of which we really do not understand. Other times, individuals simply cannot recall well enough for the data to be particularly useful, but researchers may lead them to provide it anyway.¹⁸ According to the literature, recall bias is most significant when a disease under investigation is serious (such as with cancer), the

subject believes the risk factor is high, news reports have exaggerated risks about the substance being studied, or the chemical in question is not socially acceptable (such as with illegal drug use).¹⁹ Recall bias can so undermine the validity of the data that the final study results are completely off the mark.

Researcher Bias. We all would like to believe that researchers' motives are unbiased and pure, but the reality is that incentives and personal opinions can have a huge impact on study design and results. Theoretically, researchers are supposed to try to prove a "null hypothesis." That means that rather than try to prove their theory, they act as a sort of devil's advocate, attempting to show no effect. In that case, biases may be kept in check and positive associations should be more robust. But researchers need to get published and attract research dollars, which often depends on producing studies with positive associations that are statistically significant. Accordingly, they may work the data to generate positive associations.

Good researchers will strive to keep their biases in check, while still working toward finding something interesting. Yet, others add "spin" to weak and meaningless "findings" to garner publication and media interest—and more funding. In a *Nature* magazine editorial, Arizona State University professor Dan Sarewitz points to such biases among pharmaceutical industry researchers working for drug approvals.²⁰ But the problem is also prevalent within the politically driven field of government-funded²¹ chemical and environmental policy, where funding needs and political biases play a big role.²²

Unlike their counterparts in industry, government and university researchers are rarely held accountable for their mistakes.

Unlike their counterparts in industry, government and university researchers are rarely held accountable for their mistakes. For example, if a drug harms the public, pharmaceutical companies pay dearly and can be driven out of business. In contrast, government and tenured academic researchers continue their work even when useful products are removed from commerce because of their research claims.

Sometimes government-funded research is driven by political agendas. For example, politicians have funded BPA research *ad nauesum* because activist claims and headlines have generated fear among consumers. The additional funding has not discovered much of anything new, but the resulting weak and largely inconclusive research continues to generate yet more headlines and fears. A similar situation has arisen because of press coverage related to triclosan, the anti-bacterial chemical found in soap.²³ Is there a good reason for more government funding for research on triclosan rather than on finding cures for cancers? Not really. Triclosan has been well studied and used safely for decades, with enough research to indicate that its benefits outweigh the alleged risks.²⁴

RODENT STUDIES

When environmentalists and government agencies label chemicals as carcinogens, they often point to animal tests, particularly rodent tests. However, they usually exaggerate the importance of these tests and downplay the limitations. Tests on rodents involve administering massive amounts of chemicals to animals bred to be highly susceptible to cancer. Researchers then extrapolate the possible effects of such chemicals on humans, who may be exposed to small amounts of the same chemical over their lifetimes.

First, we should ask: Are the impacts on rodents relevant to humans? As researchers Sir Richard Doll and Richard Peto note in their seminal work on cancer causes, some chemicals found to be carcinogenic in humans have not produced cancerous tumors in rodent experiments.²⁵ In fact, for many years, cigarette smoke failed to produce malignant tumors in laboratory animals even though tobacco may be the leading cause of cancer in the United States. These discordant effects of chemicals in animals and humans underscore the difficulty of relying on animal results to estimate human risks.²⁶

Second, researchers question whether the extremely high doses administered in the lab are relevant even to low-level exposures in the real world. Researchers Bruce Ames and Lois Swirsky Gold of the University of California, Berkeley, demonstrate why we need not be concerned about low-level exposure to “rodent carcinogens,” noting that such chemicals pose no more risk than that posed by the many natural, unregulated substances that are common and accepted parts of a healthy diet.²⁷

Ames and Gold report that 212 of 350 synthetic chemicals and 37 out of 77 natural substances tested by various agencies were found to be carcinogenic at the massive doses given to rodents when employing the same methodology.²⁸ As these tests show, it is the dose that makes the poison. We safely consume thousands of natural chemicals every day at much higher levels than synthetic chemicals that have been labeled carcinogens because they caused cancer in rodents when administered in massive doses. For example, humans consume thousands of natural pesticides, which plants naturally produce as a biological defense mechanism.²⁹

BASIC PRINCIPLES PROMOTING GOOD SCIENCE

Now that we are more aware of the challenges to science, let us look at some factors that make more compelling science.

Exposure matters. When researchers find a strong association that is statistically significant, we need to consider whether the exposures in the study are relevant to real-world exposures. If so, that makes it a more compelling association for drawing conclusions. Traditionally, scientists have contended, “The dose makes the poison.” Indeed, many substances that are helpful or benign at low levels can sicken or kill at high levels. In addition, the duration of exposure matters.

For example, a 70-year-old individual who smoked a pack of cigarettes a day for 30 years has a much higher cancer risk than a 70-year-old who only smoked that much for a year or two in his 20s, all other factors being equal. Likewise, there is a big difference between intermittent trace exposures to a chemical among consumers and high-level, long-term exposures to that chemical among workers in an environment where the chemical is present at substantial levels. And the exposures to workers in the past may not even be relevant to workers today, as vastly improved workplace practices limit exposure to levels that are thousands of times lower.

Yet we often see headlines and websites suggesting any exposure level to certain chemicals is dangerous.³⁰ This idea emerged in the latter part of the 20th century, when many researchers abandoned the idea that every chemical has a threshold exposure level below which risk is negligible. Those who abandoned the “threshold theory” contended that

*The low-dose
linearity model
ignores the
human body's
ability to
create defense
mechanisms
against
chemicals at
low doses.*

many chemicals can have adverse effects at any level and that risks increase linearly with any dose above zero. On the basis of those assumptions, regulatory policy around the world has focused on ways to regulate chemicals to reduce exposure to as close to zero as possible.

However, there is considerable evidence that the original research on linearity was fraudulent. Dr. Edward Calabrese of the University of Massachusetts documents how scientist Hermann J. Muller and his colleagues intentionally covered up research that contradicted Muller's "discovery" of linearity, which had earned him a Nobel Prize.

As Calabrese explains, Muller proclaimed in his Nobel Prize speech, "that one could no longer consider the possibility of a threshold dose response for germ-cell mutagenicity," yet he "made these public claims while knowing that the most extensive and relevant testing supported a threshold interpretation."³¹

Evidence continues to grow in support of the threshold theory. Many chemicals are safe under a given threshold or exposure level, with each chemical having its own threshold. The low-dose linearity model ignores

the human body's ability to create defense mechanisms against chemicals at low doses. That means low-level exposures might help us fight off cancer and other illnesses. Scientist Jay Lehr notes that studies have found cases in which people exposed to low levels of radiation actually experienced less incidence of leukemia than the general population, whereas highly exposed individuals experienced elevated rates of leukemia.³² This idea that chemicals may be beneficial at low levels but dangerous at high levels is called hormesis.³³ It makes sense. Consider the fact that many vitamins and minerals are essential at trace-exposure levels, but they can be dangerous or even deadly when consumed at high levels.

Peer Review. Studies gain greater validity after they undergo peer review. In peer review, a panel of scientists reviews a study to validate or refute its findings, usually before publication, assessing the quality of the data and methodology. Peer review is designed for an industry to self-regulate in order to reduce fraud and poor quality research. However, peer review has serious limitations. Statistician William Briggs is so skeptical of peer review that he describes it as “the weakest filter of truth that scientists have.” “Yet,” he continues, “civilians frequently believe that any work that has passed peer review has received a sort of scientific imprimatur. Working scientists rarely make this mistake in thinking.”³⁴ In fact, plenty of studies that have passed peer review were later discovered to be fraudulently produced or simply full of enough errors to require retraction.³⁵ Hence, peer review is helpful, but it alone is not sufficient to assure a study is completely sound.

No single study settles any scientific issue.

Reproducibility. No single study settles any scientific issue. Rather, science is a long process of discovery that brings us closer to an answer as an issue is examined time and again. Part of that process involves repeating specific studies to see whether

different scientists or teams of scientists can reproduce results of their peers' or even their own research. This process reduces the probability that an accidental association is taken as gospel on an issue. In fact, many "groundbreaking" studies have eventually been debunked because other researchers could not produce the results.

Weight of the Evidence. One tool designed to help synthesize the state of science on an issue involves consideration of the weight of the evidence. In that case, rather than mere peer review of a single study, scientists attempt to consider the full body of research on an issue, emphasizing the best quality studies—those of significant size, most significant findings, and best methodologies—and assessing the most likely conclusions. When it comes to setting regulatory standards for science affecting public policy, it makes sense to call for best available, peer-reviewed science that focuses on a weight-of-the-evidence test.

National Cancer Institute (NCI) researchers offer some insights on what factors help make research on an issue most compelling. A summary of their key points is as follows:³⁶

- **Strength of the association.** Is the relationship strong or weak? For example, did a high percentage of subjects exposed to a chemical develop cancer compared to a low number in the control group, or was the percentage so low that the number could be attributed to mere chance? As noted, relative risk numbers of 2, 3, and even 4 are not particularly compelling, despite headlines suggesting otherwise.
- **Consistency of the association.** Do many studies find this association or is the finding an aberration?
- **Reproducibility.** If other researchers conduct a study with the same data, do they consistently come up with the same conclusions?
- **Dose-response relationship.** Is the dose that humans experience large enough to pose a serious risk?
- **Order of events.** The association must be temporally correct—that is, the alleged cause must precede the effect. For example, if an illness occurs before exposure to a chemical, the chemical cannot be blamed for the illness.
- **Biological plausibility.** The finding is stronger if there is a good biological explanation as to the link.

TRICKS AND TERMS OF THE ALARMIST TRADE

Armed with the information above, consumers are better equipped to tackle misuse of science by activists, researchers, and reporters who benefit from sensationalizing scientific research. The following offers additional insights that can help consumers better assess questionable terminology and phraseology employed by those within the alarmist trade

Tendentious Language: “Linked to,” “Suggests,” “Consistent with”

Many of the headlines we see these days are not simply driven by environmental activists, but by well-schooled research study authors one would expect to be more careful. They craft the words in their study abstracts so that even studies that find little can appear more significant when reported in the news media. Certain words and phrases work well for this effect.

Many studies condemn products by asserting the chemicals are “**linked to**” various health ailments. But this phrase simply means that a researcher found some statistical association, which does not prove causation and is not considered particularly important if the relative risk number is low. For example, in 2010 numerous headlines touted a study that claimed that breast cancer is “linked to” synthetic fibers³⁷ — “Chemical Exposure Could Triple Breast Cancer Risk,”³⁸ “Study Links Chemical Exposure to Breast Cancer,”³⁹ “Chemical Exposure: Science Takes it Seriously Where Breast Cancer Is Concerned.”⁴⁰

Reuters reported:

In a study in *Occupational and Environmental Medicine*, a *British Medical Journal* title, the researchers found that women

exposed to synthetic fibers and petrol products during the course of their work appeared to be most at risk.

“Occupational exposure to acrylic and nylon fibers, and to polycyclic aromatic hydrocarbons may increase the risk of developing postmenopausal breast cancer,” they wrote. But some experts commenting on the study expressed caution, saying such links can crop up by chance. “In a study of this sort positive associations often occur simply by chance,” said David Coggon, a professor of occupational and environmental medicine at Britain’s Southampton University. “They carry little weight in the absence of stronger supportive evidence from other research.”

The Canadian scientists conceded their findings could be due to chance, but also said they were consistent with the theory that breast tissue is more sensitive to harmful chemicals if the exposure occurs when breast cells are still active—in other words, before a woman reaches her 40s.⁴¹

As the Reuters story’s one critic points out, a relative risk level of 3 (three times more likely) is not particularly strong, as it has a high probability of being merely accidental. The Canadian researchers basically admit that reality but then move on to rationalize their claims with yet another tricky phrase: Their findings are “**consistent with**” a theory about breast cancer tissue being sensitive to the chemicals. The authors are mincing words to increase confusion about the study result, which merely reports a weak association that is at best consistent with a mere *theory*.

Likewise, “**suggestive**” findings represent nothing much at all. Yet researchers use the term all the time to generate interest in studies that yield findings too weak to really matter. For example, in one study of the chemical bisphenol A, researchers conclude: “Our study suggests that BPA could be a potential new environmental obesogen. Widespread exposure to BPA in the human population may also be contributing to the worldwide obesity epidemic.”⁴² Their relative risk number was very low: 2.32. And this “finding” was only discovered within a subset of their sample: girls aged 9-12. The entire sample included 1,326 male and female school-age children (grades 4 to 12) living in Shanghai, China. The study measured the BPA levels in these children’s urine and correlated that with obesity levels.

It looks like they had to work the data to produce a subset with a positive relative risk and that the risk number is low enough to leave a good probability that it is a mere statistical accident. The authors rationalize their claim by noting: “Other anthropometric measures of obesity showed similar results.”⁴³ And they note that, although they could not find any association between BPA and obesity in boys, the “gender difference of BPA effect was *consistent* with findings from experimental studies and previous epidemiological studies.” [Emphasis added] But the mere existence of “other studies” with “similar” or “consistent” results does not make a study any more compelling.

There are many additional reasons to doubt this study, such as the fact that there is considerable evidence that the human body passes BPA

quickly out of the body before it can have any effects.⁴⁵ And there are plenty of studies that contradict this one. For example, another study found a link between BPA and obesity in non-Hispanic white boys, but not girls or other boys, which is inconsistent with the finding among girls and not boys in the Shanghai study.⁴⁶ Those researchers also cherry-picked a subset of their data to find an association.⁴⁷

Another recent example is a study by Tufts University researchers, featured in a *USA Today* video, produced to promote breast cancer awareness month.⁴⁸ The authors write: “Our findings suggest that developmental exposure to environmentally relevant levels of BPA during gestation and lactation induces mammary gland neoplasms in the absence of any additional carcinogenic treatment. Thus, BPA may act as a complete mammary gland carcinogen.”⁴⁹ Interestingly, they use the word “suggest” because they did not actually *find* anything. The authors originally included a bolder statement in the study in the advance publication version. These researchers were forced to revise their paper after *Forbes* journalist Trevor Butterworth pointed out that the data did not support those claims.⁵⁰ Butterworth documents⁵¹ the changes to the report that downgrade the researchers to claims to nothing more than “suggestive” results. Yet, the report still made headlines despite the findings not being particularly compelling and despite the existence of other more robust studies on the topic.⁵²

Judging the Probability of False Findings

Epidemiologist John P. A. Ioannidis wrote in in the online journal *PloS Online*: “There is increasing concern that most current published research findings are false.”⁵³ He explains that many study findings not only suffer from practical limitations, such as study size, but that the risk of false positives is higher when researchers have financial interests in the outcome or merely personal bias.⁵⁴ He notes, “[C]laimed research findings may often be simply accurate measures of the prevailing bias.” Thus, he offers the following rules or corollaries as he calls them, for critical thinkers to consider when reviewing studies or even the headlines they generate:

Corollary 1: The smaller the studies conducted in a scientific field, the less likely the research findings are to be true.

Corollary 2: The smaller the effect sizes in a scientific field, the less likely the research findings are to be true.

Corollary 3: The greater the number and the lesser the selection of tested relationships in a scientific field, the less likely the research findings are to be true.

Corollary 4: The greater the flexibility in designs, definitions, outcomes, and analytical modes in a scientific field, the less likely the research findings are to be true.

Corollary 5: The greater the financial and other interests and prejudices in a scientific field, the less likely the research findings are to be true.

Corollary 6: The hotter a scientific field (with more scientific teams involved), the less likely the research findings are to be true.⁵⁵

Sometimes researchers even make claims using unpublished research that has not undergone peer review. For example, recent news headlines indicated that pregnant women should worry about the chemical bisphenol A because a “new study” says it increases the risk of miscarriage. But there was no published study to validate these claims. The only part of this research that was available was an abstract produced for a presentation at a conference hosted by the American Society for Reproductive Medicine.⁵⁶ Fortunately, the abstract provides enough information to allay fears, despite the headlines.⁵⁷

Based on the abstract, we can see that the study examined 114 women in the early stages of pregnancy, who were in a high-risk group for miscarriage. The researchers took “spot samples” of the women’s blood and measured BPA in their blood serum. Sixty-eight of these women suffered from miscarriages; the rest carried their babies to term.⁵⁸

The researchers then measured the BPA in the serum to see if those who miscarried had more BPA in their blood serum than the others. They then divided these 68 women into four groups based on the BPA exposure levels, ranging from those with the lowest to those with the highest. Using these data, the researchers calculated that those in the group with the highest BPA levels in their serum had an 80 percent higher risk of miscarrying than did those in the lowest-exposed group.⁵⁹

Sounds pretty clear, right? Not so fast. These findings are not as meaningful as they might appear at first sight.

It is virtually impossible to prove absolute safety of anything—not even a glass of water.

First, all the researchers found was a weak association. They report a 1.83 risk ratio for the highest risk group, which is low and suggests that the result may have arisen by accident or researcher bias.

Second, the sample size was very small, which greatly increased the probability that the weak association was little more than accidental. Larger samples by definition are more representative of the larger popula-

tion. Accordingly, if this sample were 10 or 20 times larger, a weak association would have greater meaning.

Third, researchers measured the BPA in the blood serum only once. Since BPA levels in the body can fluctuate considerably over time, one-time measures cannot reveal which women actually had higher exposures. Accordingly, the data going into this research were not good enough to draw conclusions.

Finally, the researchers and news organizations that cited these findings failed to note the many other studies that contradict them. This study would be much stronger if it were consistent with the larger body of research, particularly the larger, best-designed studies, but it is not. Scientific panels around the world have reviewed the full body of BPA research repeatedly, and they all concluded that current consumer exposure to BPA is simply too low to have any adverse public health impacts.⁶⁰

Despite the obvious weaknesses of their claims, the researchers and others spun the issue to grab headlines. Lead researcher Ruth B. Lathi of Stanford University notes in *USA Today*, that although she and her colleagues did not show that BPA is dangerous, “it’s far from reassuring that BPA is safe.”⁶¹ But that comment may be safe to say because it is virtually impossible to prove absolute safety of anything—not even a glass of water.

Lathi also recommends avoiding plastic food packaging, not cooking in plastic food containers, and not leaving bottled water in the sun. Never mind that *BPA is not used for most of these products*, a fact she does not mention. There is no BPA in the lightweight flexible plastic that makes single-use bottled water or plastic food storage containers such as GladWare, which is what Lathi seems to be suggesting we avoid. She also says to limit eating canned food.

BPA is used to make hard-clear plastics, such as the five-gallon water jugs used in office water coolers and safety goggles, and in resins that line canned goods to prevent rust and the development of deadly pathogens. Can Lathi assure us that removing those resins will be safe? Certainly not, yet her rhetoric advances policy in that direction.

Others in the scientific community have fanned the flames as well. Dr. Linda Giudice, president of the American Society for Reproductive Medicine, told reporters that although this research proves nothing, it adds to the “biological plausibility” that BPA affects fertility and health.⁶² It is true that if something also has a biological explanation (plausibility), researchers can make a stronger argument for a cause-and-

effect relationship, particularly if their study discovers a reasonably strong association. But using biological plausibility to rationalize a weak association is itself pretty weak.

In a press release referencing this unpublished study, Giudice notes: “Many studies on environmental contaminants’ impact on reproductive capacity have been focused on infertility patients and it is clear that high levels of exposure affect them negatively. These studies extend our observations to the general population and show that these chemicals are a cause for concern to all of us.”⁶³

This cryptic comment has no real relevance to the research at hand, even though Giudice placed it directly under the abstract in the press release. After all, what chemicals is she talking about? Who knows? It is clear that the *high-level chemical exposures* to which she refers have nothing to do with *extremely low, easily metabolized trace levels* of BPA. But her comment worked well for the alarmists in the media accounts alleging BPA-induced miscarriage risk.⁶⁴

Often times, activists will lump together groups of such largely inclusive studies with weak associations and even worse studies with merely “suggestive” findings to claim that somehow, all these studies together allow us to draw a stronger conclusion. Hogwash. Something that proves nothing much alone does not become stronger when added to a list of other studies that also find nothing much. Instead, conclusions are drawn when we find many substantial studies have strong associations.

Classified Carcinogens

Many times, activists will call for bans or hype risks about products that contain trace amounts of chemicals that are “classified carcinogens.” Government and scientific bodies around the world have developed such classification systems to indicate that *at some exposure level and under some circumstance* a chemical *might* increase cancer risk. Such listings do not mean that the chemicals cause cancer to humans exposed to trace amounts found in consumer products. In some cases, these assessments list chemicals as “carcinogens” because they are associated with cancer among workers exposed to very high amounts over decades. These studies are of limited relevance to workers today who employ safety measures, and even less relevant to consumer exposures. A large number of chemicals end up on cancer lists simply because they cause tumors in rodents exposed to massive amounts, which also has little relevance to human exposures.

The Environmental Working Group (EWG), for example, recently applauded McDonald's for eliminating coffee Styrofoam cups, which are made with the chemical styrene, because EWG notes, “The International Agency for Research on Cancer classifies styrene as a known carcinogen.”⁶⁵ Similarly, a group called to California Clean Water Action warns: “The International Agency for Research on Cancer (IARC) has determined that styrene is a known lab-animal carcinogen and a possible human carcinogen, particularly in the occupational setting, with the strongest evidence coming from reinforced plastics workers.”⁶⁶

If you are alarmed about those claims, you should stop eating pickles on your burgers and drinking coffee because both pickles and coffee are also IARC-listed “possible human carcinogens.” In fact, the “possible” category indicates a very low risk. It is akin to saying, “There isn’t much evidence of harm, but we cannot prove otherwise.” In fact, IARC could not find significant risk even among workers exposed to relatively high levels of styrene.

Most recently, controversy erupted when the U.S. National Toxicology Program (NTP) at the National Institutes of Health reclassified styrene as “reasonably anticipated to be a human carcinogen.”⁶⁷ But this conclusion should raise more alarm bells about the politicization of science at the NTP than about styrene.⁶⁸ The report notes that data on styrene-related human cancers among workers exposed to high amounts of the substance is “limited”—that is, very weak. That is the same conclusion drawn by IARC. The only other real “hard” data relate to cancers among “some strains of mice,” but not rats, whose stomachs were injected with massive amounts of the chemical.

Humans—even plastics workers—do not experience such high doses or exposure routes in real life, which makes extrapolation from rodents to humans a very tenuous exercise. So be wary when you hear that something should be banned, regulated, or simply feared because it ended up on some government list.

Environmental Factors

Some environmental activists also raise fears about chemicals by claiming that most cancers are related to “environmental factors.” For example, actress and Cancer Schmancer founder Fran Drescher recently made this argument, calling on consumers to “detox” their homes of synthetic chemicals. In response to another article on the topic, she comments: “With 90 percent of cancers being environmental- and lifestyle-related, not genetic, I find it hard to believe that you would try to dissuade folks from detoxing their homes.”⁶⁹

It is true that cancer researchers blame “environmental factors” as being the cause of most cancers, but they define these factors as anything *but* genetics. According to the landmark research conducted by Richard Doll and Richard Peto, environmental factors include tobacco, dietary choices, infections, natural radiation, and reproductive behavior.⁷⁰ Trace chemicals in consumer products are not a demonstrated cancer source.⁷¹

According to Doll and Peto, pollution, including exposure to chemicals via consumer products, accounts for only about 2 percent of all cancer cases.⁷² Tobacco use accounts for about 30 percent and dietary choices for 35 percent of annual cancer deaths. Bruce Ames and Lois Swirsky Gold have come to similar conclusions, noting that smoking causes about a third of all cancers. They underscore the importance of diet by pointing out that the quarter of the population eating the fewest fruits and vegetables had double the cancer incidence than those eating the most.

Finally, they conclude: “There is no convincing evidence that synthetic chemical pollutants are important as a cause of human cancer.”⁷³

Interestingly, Drescher links to a study that contradicts her point. It highlights the Doll and Peto study findings on cancer causes,⁷⁴ and does not list trace chemicals used in consumer products as important carcinogens.

“**Hazardous**”

What about “**hazardous** chemicals? They sure do sound scary! And environmental activists are aware of that, which is why they use the terms regularly. Greenpeace claims: “Hazardous chemicals are substances that are dangerous to people, wildlife, and the environment at any stage of their lifecycle, from production to use to disposal.”⁷⁵

Green activist groups are working to phase out such hazardous products, and some have even assembled online databases that highlight the risks associated with certain chemicals that are considered “hazardous.” For example, the Environmental Working Group’s Skin Deep Database, which supposedly helps consumers understand the risks associated with chemicals used in cosmetics, is simply a mishmash of studies and other sources that report weak associations or suggestive findings, along with rodent studies of limited relevance to human exposures. They do not offer a sound scientific assessment of the body of research related to a chemical’s actual risk level.

Another coalition, SaferChemicals.org, is pushing for the phase-out of 100 so-called hazardous products as part of its “Mind the Store”

campaign. It notes on its website:

The Hazardous 100+ List of Chemicals of High Concern represents a small subset of all inherently hazardous chemicals of concern to which humans and the environment may be exposed in certain consumer products. Scientists have established links between exposures to many of these chemicals and chronic diseases and health conditions, including cancer, infertility, learning and developmental disabilities, behavioral problems, obesity, diabetes, and asthma.

Here green groups condemn chemicals for their hazardous profiles and potential “links” to myriad health problems—no studies needed. They developed their list of 100 “hazardous” chemicals based on government **“chemicals of concern”** lists,

_____ which are developed not by comprehensive scientific risk review processes, but by politicians and regulators responding largely to media hype generated by environmental activists.

The lists include such substances as formaldehyde (which the human body itself produces through respiration), bisphenol A, styrene, and more.

“Chemicals of concern” lists are developed not by comprehensive scientific risk review processes, but by politicians and regulators responding largely to media hype generated by environmental activists.

All the chemicals listed are approved as safe at the trace levels found in consumer products by various government agencies around the globe. Many of these chemicals have been used for decades without any evidence of health problems.

The focus on *hazard* rather than *risk* downplays, if not largely ignores, the benefits from these products. Hazard is the *potential* for harm at some level or under some specific circumstance. A risk assessment considers the *probability* that something will happen.

Consider the hazardous qualities associated with plain old water. It can kill you if you drink too much and experience “water intoxication,” which can swell your brain cells.⁷⁷ We all know that the risk of water intoxication is low from taking a few sips of water, but the risk level increases as an individual continues to consume the water at higher and higher levels. The hazard posed by the water remains the same in all instances while the risk changes with the exposure level.

Likewise, we have many “hazardous” chemicals in our homes—everything from cleaning supplies to bug spray to olive oil, which makes floors slippery when spilled. Each represents a hazard, but the risk depends on how we use them. Fortunately, we can benefit from each of these products while mitigating their associated risks.

We all assess risks every day. We assess the risks of staying in bed versus the risks and benefits of getting up. We know the hazards associated with taking a shower, driving, and even eating (don't forget the choking hazard). We manage these hazards by using a bath mat, driving

carefully, and chewing our food well enough so that we can enjoy the benefits associated with swallowing it.

In the realm of public policy, “hazard assessment” is only one step in the risk assessment process whereby researchers consider risks associated with actual or estimated exposures. Yet thanks to environmental hype surrounding chemical hazards, current regulatory trends are moving toward using hazard as a justification for regulating or even banning the use of certain products, which would threaten our ability to enjoy the benefits of those products.

For example, the U.S. Environmental Protection Agency’s (EPA) Design for the Environment (DfE) program calls on companies to eliminate certain chemicals from their products voluntarily, largely on the basis of hazard rather than actual risk.⁷⁸ Similarly, the agency program to list chemicals on “concern lists” under the Toxic Substances Control Act embodies the idea that the use of certain “hazardous” chemicals is a problem regardless of the actual risk from exposure.⁷⁹ Many relatively safe and beneficial products may eventually disappear as a result.

Toxic Chemicals

Sometimes green activists attack chemicals as “**toxic**” rather than “hazardous,” but the concept is nearly the same. Demonizing certain substances as “toxic” suggests that no level is safe. These claims might technically be true while actual risk of harm remains extremely low given real-world exposures. But that reality does not prevent activists from producing alarming rhetoric. For example, activists attack the use

of pesticides to control the deadly mosquito-transmitted West Nile Virus, exclaiming that the chemicals are “toxic” or not 100 percent safe.

One anti-pesticide group explained that it “opposes toxic methods to eradicate adult mosquitoes,” in part because “[t]he U.S. Environmental Protection Agency says that it is illegal to call these pesticides safe.”⁸⁰ Pesticides are indeed designed to be toxic—to bugs! That does not mean that low-level exposures to humans are much of a concern, despite suggestions to the contrary.

By suggesting that it is not legal or appropriate to call a pesticide “safe,” activists create the impression that these products should be feared, even when used appropriately. In reality, it is not illegal for anyone to assert that a pesticide is safe when used appropriately. Federal law regulates how manufacturers may label or advertise their pesticide products; it is not designed to suggest these products pose unacceptable risks. In fact, EPA labeling mandates are designed to ensure the products pose negligible risks when used according to label directions.⁸¹

Similarly, consider the following misleading comment by Dr. Philip Landrigan of the Mount Sinai School of Medicine regarding one of the key pesticides used for mosquito control: “Malathion is as bad as it gets... There is no such thing as a safe pesticide. It’s a matter of looking at benefits.”⁸² What does “as bad as it gets” really mean? Nothing, considering the pesticides used for spraying pose insignificant risks to humans. All he is saying is that it is the “worst risk” among negligible risks. However, Dr. Landrigan is known for making anti-pesticide claims, so the scary implications of his statement may well be intentional.

Endocrine Disruptors

Activists also claim that some man-made chemicals found in consumer products mimic human hormones and thereby cause a host of health problems, including developmental issues. In reality, trace chemicals found in consumer products and in the environment do not have enough potency to have any such effects.

Yet environmentalists and others hype the risks, sometimes pointing to the use of the drug diethylstilbestrol (DES), which was once in fact associated with higher incidences of reproductive problems. Between 1940 and 1970, many women took DES to prevent miscarriages. The relevance of these cases to low-level environmental exposures to synthetic chemicals is highly tenuous, as many researchers have pointed out. Toxicologist Stephen Safe notes: “DES is not only a potent estrogen, but it was administered at relatively high doses.... In contrast, synthetic environmental endocrine-disrupting compounds tend to be weakly active.”⁸³

Yet anti-chemical activists continue to use the DES story to hype risks about chemicals whose estrogenic potency is nowhere near that of this potent drug! For example, in a June 2013 BPA study already cited in this paper, researchers toss in a reference to DES as if it were relevant, noting: “BPA, like DES, acts on estrogen receptors which could lead to obesity in a gender-specific and dose-response manner.”⁸⁴

Similarly, in April 2011, the *New England Journal of Medicine* published a commentary that alluded to the DES situation as evidence that chemicals in the environment posed a risk—despite the low dose and weak po-

gency. Steve Milloy exposes the absurdity of this suggestion, pointing out: “DES was designed to be a hormone and it was. It was not some treatment that inadvertently acted like a hormone or disrupted hormones. It functioned like it was intended. So DES is not an ‘endocrine disrupter’—especially as the enviros use the term.”⁸⁵ Not surprisingly, when the National Research Council examined the issue in detail in 1999, it reported that it lacks data showing that “hormonally active” compounds cause any adverse impacts.⁸⁶

If weakly active trace chemicals can impact our endocrine systems, then we should really fear Mother Nature, who produces a host of such “disrupters” far more potent than synthetic chemicals.⁸⁷ For example, soy and nuts naturally contain such substances at levels that are tens of thousands of times higher than levels from man-made chemicals and are far more potent.⁸⁸ If such endocrine mimicking chemicals were a problem, these foods would be wreaking havoc on human health. But they are not. Instead, these “superfoods”⁸⁹ contribute to people today living longer, healthier lives than ever before.⁹⁰

Obesogens

The nation’s obesity problem has given new impetus and spin to the endocrine disrupter theory. To boot, researchers misuse the field of epigenetics, which involves the study of how environmental factors—that is, factors other than DNA—can influence how genes express themselves and how those changes can be passed down from one generation to the next. For example, a person’s diet or even stress levels may influence gene expression and those traits may be inheritable.

It is not yet clear how trace exposure to synthetic chemicals might impact gene expression, and some researchers are drawing conclusions too quickly. Generating headlines and perhaps potential funding, some researchers have gone as far as coining the buzzword “**obesogens**,” which is more akin to marketing phraseology than scientific terminology. Allegedly, these so-called obesogens include endocrine disrupters that not only affect our health by changing our hormone levels today; they change our gene expression in ways that make future generations more prone to obesity.⁹¹

*The buzzword
“obesogens”
is more
akin to
marketing
phraseology
than scientific
terminology.*

The obesogens theory is highly speculative, with the underlying research suffering from many confounding factors and limitations,⁹² and results have not been reproducible.⁹³ But using loaded phraseology, researchers have been able to capture provocative headlines among a wide range of sources from mommy blogs⁹⁴ to *The Atlantic*,⁹⁵ building momentum for their research efforts. As a result, many consumers may now believe that they are basically pre-programmed to become fat, discouraging them from taking positive action to prevent or reverse obesity. Some may even focus on following the foolish “Non-Obesogen Diet Plan,” as *New York Times* columnist Nicolas Kristof calls it, rather than addressing real issues related to overeating.⁹⁶ Meanwhile, there is a far more compelling body of evidence that obesity is related to overeating and lack of exercise.⁹⁷

Unfortunately, in addition to misdirecting consumers about how they can address obesity, this kind of framing fosters the outrageous, generating headlines like: “The Chemicals Behind the Shrinking of Boys’ Genitals.”⁹⁸ Never mind the fact that there is no compelling body of evidence to support this theory.

Biomonitoring the “Body Burden”

Green activists also attack chemicals simply because they might be found in the human body or in urine. For example, a coalition of groups that includes Greenpeace USA and Pesticide Action Network North America hosts a website called “The Body Burden,” which suggests we should fear the appearance of trace chemicals in our bodies.⁹⁹ But there is no compelling body of evidence showing that the existence of trace chemicals in the human body is a problem. Relatively high exposures to certain chemicals, such as lead or arsenic, are another matter. A 2009 report issued by the Centers for Disease Control and Prevention, which monitors such chemicals in the human body, explains:

The presence of an environmental chemical in people’s blood or urine does not mean that it will cause effects or disease. The toxicity of a chemical is related to its dose or concentration, in addition to a person’s individual susceptibility. Small amounts may be of no health consequence, whereas larger amounts may cause adverse health effects. The toxicity of a chemical is related to its dose or concentration in addition to a person’s susceptibility.¹⁰⁰

Accordingly, the fact that the human body may contain trace levels of chemicals should not raise alarm, particularly when we see no adverse effects. Even primitive humans had chemicals in their bodies due to exposure to different chemicals coming from such activities as burning wood to cook food and heat homes. Today we find chemicals in the human body related to our lifestyles. The main difference is that people today live decades longer. And that is due in large part to chemicals that are used to provide health care, store food, and keep drinking water safe and clean.

The precautionary principle grants regulators arbitrary power to regulate based on political rather than scientific or risk-based grounds.

Green Policy Framing: Precautionary Principle, Green Chemistry, and Beyond

In addition to using the label “toxic,” environmental activists market their hazard-based policies under a number of catchphrases that sound very reasonable despite the unreasonable policy implications.

Top of the list is their call for the implementation of the **precautionary principle**, a politically appealing concept that at first appears to be a common-sense policy of “better safe than sorry.” This “principle” holds that new technologies should be proven safe before placing them in use. However, the precautionary principle is impossible to

meet in practice because one cannot prove a negative—and nothing in life is 100 percent safe. Once the precautionary principle is accepted as a matter of policy, it encourages policy makers to make regulations as stringent as possible and even to ban certain technologies on the basis that they *might* pose some safety risks to somebody sometime in the future. In essence, it grants regulators arbitrary power to regulate based on political rather than scientific or risk-based grounds.

One step beyond the precautionary principle is the wrongheaded notion that somehow regulators know better than private parties how to safely and efficiently meet consumer needs. Not only will they protect us from alleged dangers, regulators will get involved in product design. This concept is embodied in the marketing term “**green chemistry.**” One might think that this phrase simply means employing science to develop safer products, but market-driven chemistry performs that function already, because companies have many strong incentives to make safe and effective products—far more incentives than do regulators whose focus is political.¹⁰¹

The Environmental Protection Agency defines it thus: “Green chemistry is the design of chemical products and processes that reduce or eliminate the use or generation of hazardous substances.”¹⁰² Note the word “hazardous.” It focuses on replacing risk *management* with risk *elimination*, which is impossible.

But the term has an even broader political meaning. A definition found on an “educational” green chemistry website run by the group Beyond Benign more clearly focuses on the greens’ agenda: “Green Chemistry

is a revolutionary philosophy that seeks to unite government, academic, and industrial communities by placing more focus on environmental impacts at the earliest stage of innovation and invention.”¹⁰³ So, green chemistry is a philosophy—which suggests a way of life, one where product design and development become a political process guided by bureaucrats and “stakeholders.”

So who really knows best when it comes to manufacturing and product design, government or industry? Green chemistry advocates appear to pick government. Yet, bureaucrats lack situation-specific expertise as well as the technical information possessed by tens of thousands of product engineers working in thousands of businesses. That much information cannot be aggregated by government to design a product, let alone guide an entire industry.

Moreover, government regulators are not accountable to consumers. When products fail or prices skyrocket, people do not blame Uncle Sam. They blame business. That in turn leads to yet more regulations on business—not the mitigating of green demands many businesses hope to achieve if they play the green chemistry game.

Other articulations of this concept include advocacy of “**safer substitutes**” or the replacement of existing chemicals with so-called “**inherently safer chemicals**.” But by definition, government-driven product substitution means that consumers lose access to safe and effective products we would otherwise choose because they both enrich our lives and make them safer. And these second-best substitutes might not only fail to meet our needs, but could actually reduce safety.

For example, if activists succeed in removing many flame retardant¹⁰⁴ products from the market, more people might die in fires. If we eliminate disinfectants such as triclosan,¹⁰⁵ more people may get sick. If we take the preservative formaldehyde out of cosmetics, more consumers might suffer health effects from applying rancid products on their skin.¹⁰⁶ And if retailers refuse to sell containers lined with bisphenol A-based resins, inferior replacement products may lead to more incidences of food-borne illnesses.¹⁰⁷

MORE REASONS TO DOUBT MOST ALARMIST STUDIES ABOUT CHEMICALS

Trace chemicals are unlikely to have much health effects at all for the reasons discussed in this paper. For example, environmentalists often call for regulation on the grounds that man-made chemicals used in consumer products pose a serious cancer risk. Yet, as noted, all pollution—including exposure to chemicals via consumer products—accounts for only 2 percent of all cancer cases, if that much.¹⁰⁸ Lifestyle choices, such as our diets and smoking behavior, should be our real concern.

In fact, in its “Annual Report to the Nation on the Status of Cancer,” the National Cancer Institute reports that since 1975 cancer incidence has continued to decline among men and declined for women until 2006, after which rates stabilized.¹⁰⁹ In addition, thanks to earlier detection and treatment, mortality has continued to decline for both sexes.

NCI data also challenge activist attempts to link breast cancer to chemicals. It reports that breast cancer has stabilized after “sharply decreasing” following the reduction of hormone replacement therapy, and survival trends are very positive. A woman with breast cancer in the late 1970s had a 75 percent chance of surviving five or more years, while today she has a 90 percent chance, according to NCI data.¹¹⁰

While stable for other groups of women, breast cancer incidents increased for black and Asian and Pacific Islander women in recent years. NCI identifies the likely causes as “reproductive factors and postmenopausal hormone replacement therapy, obesity after menopause, weight gain

throughout life, and alcohol consumption.”¹¹¹ Chemicals are not listed among these likely causes.

The American Cancer Society posts a similar list of breast cancer causes on its website and also notes, “[A]t this time research does not show a clear link between breast cancer risk and exposure to things like plastics, certain cosmetics and personal care products, and pesticides.”¹¹²

CONCLUSION: IS SCIENCE EVEN USEFUL?

So with all the limitations and challenges to science, we might question whether we can trust any scientific study. Alas, science is not perfect. And heaped atop all the uncertainty present in scientific research, our views on certain issues can quickly become upset when headlines suggest that “new information” has transformed our understanding. This is frustrating for consumers who one day are told, for example, that margarine is better for health than butter only to learn a decade later that margarine has trans fats that might be worse than the saturated fats in butter—at least for *some* people!

In *Making Sense of Uncertainty: Why Uncertainty is Part of Science*, a paper published by the UK-based group Sense About Science, various scientists explain that uncertainty is a natural and necessary part of science.¹¹³ While each experiment or research study presents many uncertainties, each plays a role in building a body of research that gains meaning over time. Rarely, they note, does any single study act as a “game changer.” Rather, each study opens avenues for additional study and possible refinement of our understanding.

This discovery process that we call science has brought forth advances of monumental importance, including control of many infectious diseases that once plagued humanity, from smallpox to tuberculosis. And while debates about food safety are likely to continue, there is ample evidence of numerous and reproducible research demonstrating some important and useful principles, such as: eating lots of fruits and veg-

etables improves health, and calorie control can help people lose weight or maintain a healthy weight.

Consumers need not panic about any particular study, nor completely dismiss studies because of uncertainty. Rather, we should focus less on any single study and more on what the larger body of research indicates about a particular issue. In addition, we should remain wary of headlines and anyone claiming to have changed the nature of the debate with a single study. Armed with information about what makes one study's finding more meaningful and robust, we can avoid being fooled by headlines that exaggerate the results of a handful of studies.

NOTES

- 1 The Polling Company, Inc./WomanTrend, *National Online Survey of Women*, produced for the Independent Women's Forum, May 17, 2013, <http://iwf.org/files/IWF-Alarmism-Executive-Summary.pdf>.
- 2 David A. Freedman, "The Ecological Fallacy," Department of Statistics University of California, Berkeley, August 1, 2002, <http://www.stat.berkeley.edu/~census/ecofall.txt>.
- 3 Steven J. Milloy, *Junk Science Judo: Self-Defense Against Health Scares & Scams* (Washington, D.C.: Cato Institute, 2001), p. 102.
- 4 Paolo Boffetta, "Causation in the Presence of Weak Associations," *Critical Reviews in Food Science and Nutrition* Vol. 51, supp. 1 (December 4, 2010): pp. 13–16, <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3024843>.
- 5 Sarah Williams, "Absolute Versus Relative Risk—Making Sense of Media Stories," Science Update Blog (project of hosted by the nonprofit Cancer Research UK), March 15, 2013, <http://scienceblog.cancerresearchuk.org/2013/03/15/absolute-versus-relative-risk-making-sense-of-media-stories>.
- 6 Steve McKillup, *Statistics Explained: An Introductory Guide for Life Scientists* (New York: Cambridge University Press, 2012), p. 73.
- 7 Mark Battersby, *Is That a Fact? A Field Guide for Evaluating Statistical and Scientific Information* (Ontario: Broadview Press, 2009) pp. 203, 231.
- 8 Ronald P. Carver, "The Case Against Statistical Significance Testing," *Harvard Educational Review*, Vol. 48, No. 3 (1978): pp. 378-399, http://scholasticadministrator.typepad.com/thisweekineducation/files/the_case_against_statistical_significance_testing.pdf.
- 9 Andrew G. Elman and Hal S. Tern, "The Difference Between 'Significant' and 'Not Significant' is not Itself Statistically Significant," *The American Statistician* Vol. 60, No. 4 (November 2006): pp. 238-331, <http://www.stat.columbia.edu/~gelman/research/published/signif4.pdf>.
- 10 E.J. Masicampo and D.R. Lalande, "A Peculiar Prevalence of p Values Just Below .05," *Quarterly Journal of Experimental Psychology*, Vol. 65, No. 11 (August 2, 2012), pp. 2271-2279, <http://www.tandfonline.com/doi/abs/10.1080/17470218.2012.711335#preview>.
- 11 Battersby.
- 12 For more information see: National Health and Nutrition Examination Survey, Centers for Disease Control and Prevention, pages last updated August 26, 2013, <http://www.cdc.gov/nchs/nhanes.htm>.
- 13 Judy S. LaKind, Michael Goodman, and Daniel Q. Naiman, "Use of NHANES Data to Link Chemical Exposures to Chronic Diseases: A Cautionary Tale," *PLoS ONE*, Vol. 7 No. 12 (December 2012), p. 51086, <http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0051086>.

- 14 James of Mills of the National Institute of Child Health and Human Development lamented in an article in the *New England Journal of Medicine* back in 1993: “If you torture your data long enough, they will tell you whatever you want to hear’ has become a popular observation in our office. In plain English, this means that study data, if manipulated in enough different ways can prove whatever the investigator wants to prove.” James L. Mills, “Data Torturing,” *New England Journal of Medicine*, Vol. 329, No. 16 (October 14, 1993), pp. 1196-1199.
- 15 Battersby, p. 164.
- 16 Angela Logomasini, “Medical Junk Science: Canned Veggies May Make Kids Fat,” OpenMarket.org, October 3, 2012, <http://www.openmarket.org/2012/10/03/medical-junk-science-canned-veggies-may-make-kids-fat/>.
- 17 For more background on confounding factors see: “What are Confounding Factors and How Do They Affect Studies? Statistical Assessment Service (STATS), website, accessed August 20, 2013, http://stats.org/in_depth/faq/confounding_factors.htm.
- 18 For example, Geoffrey Kabat of the Statistical Assessment Service at George Mason University points out how recall bias rendered the results of a number of retrospective studies linking brain cancer and cell phones completely wrongheaded when compared to prospective cohort studies of cell phone users. See Geoffrey Kabat, “Yet Another Large Study Discredits the Alleged Link Between Cellphones and Brain Cancer,” *Forbes* (online), September 4, 2013, <http://www.forbes.com/sites/geoffreykabat/2013/09/04/yet-another-large-study-discredits-the-alleged-link-between-cellphones-and-brain-cancer>.
- 19 Eman Hassan, “Recall Bias Can Be a Threat to Retrospective and Prospective Research Design,” *The Internet Journal of Epidemiology*, Vol. 3, No. 2 (2006), <http://archive.ispub.com/journal/the-internet-journal-of-epidemiology/volume-3-number-2/recall-bias-can-be-a-threat-to-retrospective-and-prospective-research-designs.html#e-3>.
- 20 Daniel Sarewitz, “Beware the Creeping Cracks of Bias,” *Nature*, May 9, 2012, <http://www.nature.com/news/beware-the-creeping-cracks-of-bias-1.10600>.
- 21 Tibor R. Machan, “Bias in Government Science,” *Mises Daily*, June 4, 2000, <http://mises.org/daily/437>.
- 22 Michael H. Huesemann, “The Inherent Biases in Environmental Research and Their Effects on Public Policy,” *Futures*, Vol. 34 (2002): pp. 621–633
- 23 Michelle Yeomans. “Scientists Receive New Grant to Extend Research on Triclosan,” *CosmeticsDesign.com*, August 20, 2013, <http://www.cosmeticsdesign.com/Formulation-Science/Scientists-receive-new-grant-to-extend-research-on-triclosan>.

- 24 For more details see Angela Logomasini, "'Shocking' Truth about Government and Soap," Openmarket, May 13, 2013, <http://www.openmarket.org/2013/05/13/shocking-truth-about-government-and-soap>.
- 25 Richard Doll and Richard Peto, "The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today," *Journal of the National Cancer Institute*, Vol. 66, No. 6 (1981): pp. 1191–1308.
- 26 Ibid.
- 27 Bruce N. Ames and Lois Swirsky Gold, "Too Many Rodent Carcinogens: Mitogenesis Increases Mutagenesis," *Science* 249, no. 4976 (August 31, 1990): p.1487.
- 28 Ibid.
- 29 Ibid.
- 30 For example, the Environmental Working Group annually releases its "Shoppers Guide to Pesticides" (<http://www.ewg.org/foodnews>), which sounds alarm bells about trace pesticides found on produce, despite the fact that the residue levels meet U.S. Environmental Protection Agency and Food and Drug Administration safety standards and are too low to have any public health significance. Yet headlines suggest these trace exposures matter thanks to EWG's annual report. For example, see Ashleigh Schmitz, "Meet the 'Dirty Dozen': Produce with the Most Pesticides," Fox News, May 3, 2013, <http://www.foxnews.com/health/2013/05/03/meet-dirty-dozen-produce-with-most-pesticides>; and "Which Foods Are Laced with Carcinogens? Now You Can Find Out," The Daily Green, June 19, 2009, <http://www.thedailygreen.com/environmental-news/latest/pesticide-residue-foods-47061902>.
- 31 Edward J. Calabrese, "How the US National Academy of Sciences Misled the World Community on cancer risk assessment: new findings challenge historical foundations of the linear dose response," *Archives of Toxicology* (August 2013) 2013; DOI: 10.1007/s00204-013-1105-6.
- 32 Jay Lehr, Ph.D., "Good News About Radon: The Linear Nonthreshold Model Is Wrong," JunkScience.com, May 1996, <http://www.junkscience.org/news/lehr.html>.
- 33 Mark P. Mattson, "Hormesis Defined," *Aging Research Reviews*, Vol.7, No. 1 (January 2008): pp. 1-7, <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2248601/pdf/nihms39393.pdf>.
- 34 William M Briggs, "A Case Of Failed Peer Review: Dust And Death," blog post on wmbriggs.com, October 20 2011, <http://wmbriggs.com/blog/?p=4587>.
- 35 Many examples are available on Retractionwatch.com. It is worth noting that retractions of scientific studies may indicate failure of peer review, but Retractionwatch.com bloggers explain it in a more positive light: Retraction reflects a "self-correcting" element of science, they say.

Logomasini: A Consumer's Guide to Chemical Risk

- 36 National Cancer Statistics Branch, National Cancer Institute, *Cancer Incidence and Survival among Children and Adolescents: United States SEER Program 1975-1995* (Washington, D.C.: National Institutes of Health, 1999).
- 37 France Labrèche et al., "Postmenopausal Breast Cancer and Occupational Exposures," *Occupational and Environmental Medicine*, Vol. 67, No. 4 (April 2010): pp. 263-269.
- 38 "Study: Chemical Exposure Could Triple Breast Cancer Risk," Fox News, April 01, 2010, <http://www.foxnews.com/story/2010/04/01/study-chemical-exposure-could-triple-breast-cancer-risk/>.
- 39 Kate Kelland, "Study Links Chemical Exposure to Breast Cancer," Reuters, April 1, 2010, <http://www.reuters.com/article/2010/04/01/us-cancer-breast-chemicals-idUSTRE62U5Y120100401>.
- 40 Britta Aragon, "Chemical Exposure: Science Takes it Seriously Where Breast Cancer Is Concerned," Cinco Vidas Blog, September 29, 2010, <http://cincovidas.com/chemical-exposure-science-takes-it-seriously-where-breast-cancer-is-concerned/>.
- 41 Kelland.
- 42 De-Kun Li et al., "Urine Bisphenol-A Level in Relation to Obesity and Overweight in School-Age Children," *PLOS ONE* 8, no 6 (June 12, 2013): e65399, <http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0065399>.
- 43 Ibid.
- 44 Ibid.
- 45 Justin G. Teeguarden, et al., "Twenty-Four Hour Human Urine and Serum Profiles of Bisphenol A During High-Dietary Exposure," *Toxicological Sciences*, Vol. 123, No.1 (2011): pp. 48-57, http://www.maine.gov/dep/ftp/bep/ch882citizen_petition/Exhibit14/Tab%20-%20T-1%20Teeguarden%20et%20al%202011.pdf.
- 46 R. Bhandari, J. Xiao, and A. Shanka, "Urinary Bisphenol A and Obesity in U.S. Children," *American Journal of Epidemiology*, Vol. 177 No. 11, (June 1, 2013): pp. 1263-1270, <http://www.ncbi.nlm.nih.gov/pubmed/23558351>.
- 47 For more details see: Geoffrey Kabat, "How Abysmal Scientific Research Is Used To Scare America's Parents." *Forbes* (online), August 20, 2013, <http://www.forbes.com/sites/geoffreykabat/2013/08/20/how-abysmal-scientific-research-is-used-to-scare-americas-parents>.
- 48 "Concerns about BPA and Breast Cancer," *USA Today* video, October 8, 3013, <http://www.usatoday.com/videos/news/health/2013/10/08/2923855>.
- 49 Nicole Acevedo, et al, "Perinatally Administered Bisphenol A as a Potential Mammary Gland Carcinogen in Rats," *Environmental Health Perspectives*, Vol. 121, No. 9 (September 2013): pp. 1040-1046, <http://ehp.niehs.nih.gov/wp-content/uploads/121/9/ehp.1306734.pdf>.

- 50 Trevor Butterworth, "Taxpayer-Funded Journal Walks Back BPA Cancer Claim after Statistical Meltdown," *Forbes* (online), September 26, 2013, <http://www.forbes.com/sites/trevorbutterworth/2013/09/26/taxpayer-funded-journal-walks-back-bpa-cancer-claim-after-statistical-meltdown>.
- 51 Trevor Butterworth, "BPA And Breast Cancer: When Academics Spin Statistics," *Forbes* (online), July 30, 2013 <http://www.forbes.com/sites/trevorbutterworth/2013/07/30/bpa-and-breast-cancer-when-academics-spin-statistics/>
- 52 Trevor Butterworth, "USA Today Spins Breast Cancer Scare Out Of Retracted Study Claim As New EPA Study Dismisses Risk," *Forbes* (online), October 9, 2013, <http://www.forbes.com/sites/trevorbutterworth/2013/10/09/usa-today-spins-breast-cancer-scare-out-of-retracted-study-claim-as-new-epa-study-dismisses-risk/>.
- 53 John P. A. Ioannidis, "Why Most Published Research Findings Are False," *PLOS Medicine*, Vol. 2, No. 8 (August 2005), pp. 696-701, <http://www.plosmedicine.org/article/fetchObject.action?uri=info%3Adoi%2F10.1371%2Fjournal.pmed.0020124&representation=PDF>.
- 54 Ibid.
- 55 Ibid.
- 56 The abstract was published: R. B. Lathi et al., "Maternal Serum Biphenol-A (BPA) Level Is Positively Associated With Miscarriage Risk." *Fertility & Sterility*, Vol. 100, No. 3 suppl 1 (September 3, 2013), S19, <http://download.journals.elsevierhealth.com/pdfs/journals/0015-0282/PIIS001502821300962X.pdf>.
- 57 For example, see Rachael Rettner, "BPA Exposure Linked to Higher Rate of Miscarriage, LiveScience.com, October 14, 2013, <http://www.livescience.com/40391-bpa-higher-rate-miscarriage.html>; Ryan Jaslow, "BPA Exposure May Increase Miscarriage Risk in Pregnant Women," CBS News, October 14, 2013, http://www.cbsnews.com/8301-204_162-57607385; and Daniel Beekman, "BPA, Chemical Found in Canned Foods and Plastics, Tied to Higher Rate of Miscarriage," *New York Daily News*, October 14, 2013, <http://www.nydailynews.com/life-style/health/bpa-raises-miscarriage-risk-study-article-1.1484731>.
- 58 R. B. Lathi et al.
- 59 Ibid.
- 60 For an overview of these many reviews, see: <http://www.factsaboutbpa.org/scientific-assessments>.
- 61 Marilyn Marchione, "BPA's Possible Role in Miscarriages Examined," *USA Today*, October 14, 2013, <http://www.usatoday.com/story/news/nation/2013/10/14/bpa-miscarriages/2977079>.

- 62 Ibid.
- 63 ASRM Office of Public Affairs, "Effects of BPA and Phthalates on Conception and Pregnancy," ASRM Press Release, October 14, 2013, http://www.asrm.org/Effects_of_BPA_and_Phthalates_on_Conception_and_Pregnancy.
- 64 Rebekah Marcarelli, "BPA Miscarriage: Could Food Packaging Interfere With Fertility?" HNGN Headlines & Global News, October 15, 2013, <http://www.hngn.com/articles/14944/20131015/bpa-miscarriage-food-packaging-interfere-fertility.htm>.
- 65 Ashley McCormack, "McDonald's Makes the Switch from Foam to Paper Hot Cups," Enviroblog, September 30, 2013, <http://www.ewg.org/enviroblog/2013/09/mcdonald-s-makes-switch-foam-paper-hot-cups>.
- 66 Clean Water Action, "Health Effects and Regulation of Styrene (CASRN 100-42-5)," undated fact sheet, http://www.cleanwateraction.org/files/publications/ca/cwa_fact_sheet_styrene_2011.pdf.
- 67 National Toxicology Program, *Report on Carcinogens*, Twelfth Edition (Washington, D.C.: U.S. Department of Health and Human Services, 2011), <http://ntp.niehs.nih.gov/?objectid=03C9AF75-E1BF-FF40-DBA9EC0928DF8B15>.
- 68 Richard Belzer, "The Report on Carcinogens: What Went Wrong and What Can Be Done to Fix It," *Issue Analysis* 2012 No. 1, (Washington D.C.: Competitive Enterprise Institute, 2012), <http://cei.org/sites/default/files/Richard%20B%20Belzer%20-%20The%20Report%20on%20Carcinogens.pdf>.
- 69 Fran Drescher, "Err on the Side of Caution: Avoid Harsh Chemicals and Pesticides," *Huffington Post*, March 8, 2013, http://www.huffingtonpost.com/fran-drescher/cancer-prevention_b_2838649.html.
- 70 Doll and Peto.
- 71 "Chemicals, Cancer, and You" (brochure), The Agency for Toxic Substances and Disease Registry, undated, <http://www.atsdr.cdc.gov/emes/public/docs/Chemicals,%20Cancer,%20and%20You%20FS.pdf>.
- 72 Doll and Peto.
- 73 Bruce N. Ames and Lois Swirsky Gold, "Environmental Pollution, Pesticides, and the Prevention of Cancer: Misconceptions," *FASEB Journal*, Vol. 11, No. 13, 1997, pp. 1041–1052, <http://www.fasebj.org/content/11/13/1041.full.pdf+html>.
- 74 Preetha Anand et al., "Cancer is a Preventable Disease that Requires Major Lifestyle Changes," *Pharmaceutical Research*, Vol. 25, No. 9 (September 2008): pp. 2097–2116, <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2515569/>.
- 75 "What are Hazardous Chemicals?" undated Greenpeace.org Web page, <http://www.greenpeace.org/eastasia/campaigns/toxics/science/hazardous-chemicals/>.
- 76 Safer Chemicals, Healthy Families, "What is the Hazardous 100+?", accessed

Logomasini: A Consumer's Guide to Chemical Risk

- online October 24, 2013, <http://mindthestore.saferchemicals.org/methodology>.
- 77 D.J. Farrell and L. Bower, "Fatal Water Intoxication," *Journal of Clinical Pathology*, Vol. 56, No. 10 (October 2003): pp. 803–804, <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1770067>.
- 78 U.S. Environmental Protection Agency "Design for the Environment," EPA website, undated, accessed December 19, 2013, <http://www.epa.gov/dfe/pubs/about/index.htm>.
- 79 EPA "Essential Principles for Reform of Chemicals Management Legislation," online document last updated April 28, 2010, accessed January 5, 2012, <http://www.epa.gov/oppt/existingchemicals/pubs/principles.html>.
- 80 Asael Sala, Pesticides Being Proposed for Aerial Spraying Over South Sacramento County Among the Most Toxic," *Sacramento Press*, June 11, 2012, <http://sacramentoexpress.com/2012/06/11/pesticides-being-proposed-for-aerial-spraying-over-south-sacramento-county-among-the-most-toxic>.
- 81 EPA explains on its website: "Pesticide product labels provide critical information about how to safely handle and use pesticide products," Pesticide Product Labels, page last updated on December 11, 2013, accessed December 18, 2013 <http://www.epa.gov/opp00001/regulating/labels/product-labels.htm>.
- 82 Julie Deardorff and Karen Mellen, "Metro Spraying Targeting Hosts of Nile Virus," *Chicago Tribune*, August 21, 2002.
- 83 Stephen Safe, "Endocrine Disrupters: New Toxic Menace?" in *Earth Report 2000* (New York: McGraw- Hill, 2000), p. 192.
- 84 De-Kun Li et al.
- 85 Steve Milloy, "Earth to Harvard Med Faculty: DES is not an 'Endocrine Disrupter,'" *Junkscience.com*, April 20, 2011, <http://junkscience.com/2011/04/20/earth-to-harvard-des-is-not-an-endocrine-disrupter>.
- 86 National Research Council, *Hormonally Active Agents in the Environment* (Washington, DC: National Academies Press, 1999).
- 87 For details see, Jonathan Tolman, *Nature's Hormone Factory*, (Washington, D.C.: Competitive Enterprise Institute, 1996), http://cei.org/PDFs/hormone_factory.pdf; and Angela Logomasini, "Endocrine Disrupters" in *The Environmental Source*, Washington, D.C.: Competitive Enterprise Institute, 2008, <http://cei.org/sites/default/files/Angela%20Logomasini%20-%20Environmental%20Source%20Chemical%20Endocrine.pdf>.
- 88 Ibid.
- 89 Jenny Stamos Kovac, "Beans: Protein-Rich Superfoods," *WebMD the Magazine*, March 1, 2007, <http://www.webmd.com/diet/features/beans-protein-rich-superfoods>.
- 90 Angela Logomasini, "Chemicals and Health in Perspective," *Safe Chemical Policy.org*, April 12, 2012,

Logomasini: A Consumer's Guide to Chemical Risk

- <http://safechemicalpolicy.org/chemicals-and-health-in-perspective>.
- 91 See Angela Logomasini, "What will Really Make us Fat in the New Year," Fox News, December 29, 2010, <http://www.foxnews.com/opinion/2010/12/29/really-make-fat-new-year>.
- 92 Richard M. Sharpe and Amanda J. Drake, "Obesogens and Obesity—an Alternative," *Obesity*, Vol. 21 (2013): pp.1081-1083.
- 93 University of Missouri, "Previous Studies on Toxic Effects of BPA Couldn't be Reproduced, says MU Research Team," Press Release, January 2, 2013, <http://munews.missouri.edu/news-releases/2013/0102-previous-studies-on-toxic-effects-of-bpa-couldn%E2%80%99t-be-reproduced-says-mu-research-team>.
- 94 Rachel Sarnoff, "3 Tips to Avoid Obesogens—And Obesity," Mommy Greenest (blog), July 25, 2013, <http://www.mommygreenest.com/3-tips-to-avoid-obesogens-and-obesity>.
- 95 Kristin Wartman, "What's Really Making Us Fat?" *The Atlantic*, March 8 2012, <http://www.theatlantic.com/health/archive/2012/03/whats-really-making-us-fat/254087>.
- 96 Nicholas D. Kristof, "The All-New Non-Obesogen Diet Plan!" *New York Times* blog, January 19, 2013, http://kristof.blogs.nytimes.com/2013/01/19/the-all-new-non-obesogen-diet-plan/?_r=0.
- 97 For example, see Lisa R. Young and Marion Nestle, "The Contribution of Expanding Portion Sizes to the US Obesity Epidemic," *American Journal of Public Health*, Vol. 92, No 2 (February 2002): pp. 246–249, <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1447051>.
- 98 Dr. Mercola, "The Chemicals Behind the Shirking of Boys' Genitals, Food Consumer," September 1, 2013, http://www.foodconsumer.org/newsite/Non-food/Environment/the_chemicals_behind_the_shrinking_of_boy_s_genitals_0901130736.html.
- 99 Chemical Body Burden website, <http://www.chemicalbodyburden.org/whatisbb.htm>.
- 100 *Fourth National Report on Human Exposure to Environmental Chemicals* (Atlanta: Centers for Disease Control and Prevention, 2009), http://www.cdc.gov/exposurereport/pdf/FourthReport_ExecutiveSummary.pdf.
- 101 For more on this topic see: Angela Logomasini and Daniel J. Murphy, *Green Chemistry's March of the Ostriches* (Washington, D.C.: Competitive Enterprise Institute, 2011), <http://cei.org/issue-analysis/green-chemistrys-march-ostriches>.
- 102 U.S. Environmental Protection Agency, "Green Chemistry," webpage accessed October 31, 2013, <http://www2.epa.gov/green-chemistry>.
- 103 What is Green Chemistry? Beyond Benign, undated website, accessed, November

- 8, 2013, <http://www.beyondbenign.org/greenchemistry/greenchem.html>.
- 104 Angela Logomasini, "Flame Retardant Risks Overblown," OpenMarket.org, July 18, 2012, <http://www.openmarket.org/2012/07/18/flame-retardant-risks-overblown>.
- 105 Angela Logomasini, "'Shocking' Truth about Government and Soap."
- 106 Angela Logomasini, "Of Mice, Mushrooms, And Formaldehyde," OpenMarket.org, October 18, 2012, <http://www.openmarket.org/2012/10/18/of-mice-mushrooms-and-formaldehyde>.
- 107 Angela Logomasini, "BPA resin replacements may be more harmful, *The Hill Congress Blog*," December 27, 2012, <http://thehill.com/blogs/congress-blog/energy-a-environment/274643-bpa-resin-replacements-may-be-more-harmful>.
- 108 Doll and Peto, "The Causes of Cancer."
- 109 Simard A. Jemal et al., "Annual Report to the Nation on the Status of Cancer, 1975-2009, Featuring the Burden and Trends in HPV-Associated Cancers and HPV Vaccination Coverage Levels." *Journal of the National Cancer Institute*, Vol. 105, No. 3 (February 2013), <http://jnci.oxfordjournals.org/content/early/2013/01/03/jnci.djs491.full>.
- 110 Cancer Advances in Focus: Breast Cancer, National Cancer Institute website, accessed November 8, 2013, <http://www.cancer.gov/cancertopics/factsheet/cancer-advances-in-focus/breast>.
- 111 Jemal et al.
- 112 "Breast Cancer Overview," American Cancer Society, Web document last revised October 24, 2013, <http://www.cancer.org/cancer/breastcancer/overviewguide/breast-cancer-overview-what-causes>.
- 113 *Making Sense of Uncertainty: Why Uncertainty is Part of Science*, (London: Sense About Science, June 27, 2013), http://www.senseaboutscience.org/data/files/resources/127/SAS012_MSU_ONLINE.pdf.

About the Author

Angela Logomasini is a Senior Fellow at the Competitive Enterprise Institute, where she conducts research and analysis on environmental regulatory issues and manages the consumer education website, SafeChemicalPolicy.org. She is co-editor of the CEI book, *The Environmental Source*. Her articles have been published in the *Wall Street Journal*, *New York Post*, *Washington Times*, and other papers. Logomasini also makes regular media appearances. She has appeared on dozens of radio shows, including NPR's "The Diane Rehm Show," CNN Radio, and Radio America. Television appearances include Fox Business's "STOSSEL," ABC's "Nightline," C-SPAN, CNN, and more.

She served as Legislative Assistant to Senator Sam Brownback (R-Kan.) from 1996 to 1998, advising the senator on energy and environmental issues. Before that, she was Environmental Editor for the Research Institute of America. From 1989 to 1994, Logomasini worked for Citizens for a Sound Economy (CSE), serving as Director of Solid Waste Policy for a CSE affiliate, Citizens for the Environment, and as a policy analyst covering various economic issues.

Angela Logomasini has a Ph.D. in politics, which she earned at the Catholic University of America.

The Competitive Enterprise Institute promotes the institutions of liberty and works to remove government-created barriers to economic freedom, innovation, and prosperity through timely analysis, effective advocacy, inclusive coalition-building, and strategic litigation.



COMPETITIVE
ENTERPRISE
INSTITUTE

1899 L Street, NW
12th Floor
Washington, DC 20036
cei.org