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April 13, 2011

Do Cellphones Cause Brain Cancer?

By SIDDHARTHA MUKHERJEE

On Jan. 21, 1993, the television talk-show host [Larry King](#) featured an unexpected guest on his program. It was the evening after Inauguration Day in Washington, and the television audience tuned in expecting political commentary. But King turned, instead, to a young man from Florida, David Reynard, who had filed a tort claim against the cellphone manufacturer NEC and the carrier GTE Mobilnet, claiming that radiation from their phones caused or accelerated the growth of a [brain tumor](#) in his wife.

“The [tumor](#) was exactly in the pattern of the antenna,” Reynard told King. In 1989, Susan Elen Reynard, then 31, was told she had a malignant astrocytoma, a brain [cancer](#) that occurs in about 6,000 adults in America each year. To David Reynard, the shape and size of Susan’s tumor — a hazy line swerving from the left side of her midbrain to the hindbrain — uncannily resembled a malignant shadow of the phone (but [tumors](#), like clouds, can assume the shapes of our imaginations). Suzy, as she was known, held her phone at precisely that angle against her left ear, her husband said. Reynard underwent surgery for her cancer but to little effect. She died in 1992, just short of her 34th birthday. David was convinced that high doses of radiation from the cellphone was the cause.

Reynard v. NEC — the first tort suit in the United States to claim a link between phone radiation and brain cancer — illustrated one of the most complex conceptual problems in cancer epidemiology. In principle, a risk factor and cancer can intersect in three ways. The first is arguably the simplest. When a rare form of cancer is associated with a rare exposure, the link between the risk and the cancer stands out starkly. The juxtaposition of the rare on the rare is like a statistical lunar eclipse, and the association can often be discerned accurately by observation alone. The discipline of cancer epidemiology originated in one such a confluence: in 1775, a London surgeon, Sir Percivall Pott, discovered that scrotal cancer was much more common in chimney sweeps than in the general population. The link between an unusual malignancy and an uncommon profession was so striking that Pott did not even need statistics to prove the association. Pott thus discovered one of the first clear links between an environmental substance — a “carcinogen” — and a particular subtype of cancer.

The opposite phenomenon occurs when a common exposure is associated with a common form

of cancer: the association, rather than popping out, disappears into the background, like white noise. This peculiar form of a statistical vanishing act occurred famously with tobacco [smoking](#) and lung cancer. In the mid-1930s, smoking was becoming so common and lung cancer so prevalent that it was often impossible to definitively discern a statistical link between the two. Researchers wondered whether the intersection of the two phenomena was causal or accidental. Asked about the strikingly concomitant increases in lung cancer and smoking rates in the 1930s, Evarts Graham, a surgeon, countered dismissively that “the sale of nylon stockings” had also increased. Tobacco thus became the nylon stockings of cancer epidemiology — invisible as a carcinogen to many researchers, until it was later identified as a major cause of cancer through careful clinical studies in the 1950s and 1960s.

But the most complex and most publicly contentious intersection between a risk factor and cancer often occurs in the third instance, when a common exposure is associated with a rare form of cancer. This is cancer epidemiology’s toughest conundrum. The rarity of the cancer provokes a desperate and often corrosive search for a cause (“why, of all people, did I get an astrocytoma?” Susan Reynard must have asked herself). And when patients with brain tumors happen to share a common exposure — in this case, cellphones — the line between cause and coincidence begins to blur. The association does not stand out nor does it disappear into statistical white noise. Instead, it remains suspended, like some sort of peculiar optical illusion that is blurry to some and all too clear to others. (A similarly corrosive intersection of a rare illness, a common exposure and the desperate search for a cause occurred recently in the saga of [autism](#) and vaccination. Vaccines are nearly universal, and autism is relatively rare — and many parents, searching to explain why their children became autistic, lunged toward a common culprit: childhood vaccination. An avalanche of panic ensued. It took years of carefully performed clinical trials to finally disprove the link.)

The Florida Circuit Court that heard Reynard v. NEC was quick to discern these complexities. It empathized with David Reynard’s search for a tangible cause for his wife’s cancer. But it acknowledged that too little was known about such cases; “the uncertainty of the evidence . . . the speculative scientific hypotheses and [incomplete] epidemiological studies” made it impossible to untangle cause from coincidence. David Reynard’s claim was rejected in the spring of 1995, three years after it was originally filed. What was needed, the court said, was much deeper and more comprehensive knowledge about cellphones, brain cancer and of the possible intersection of the two.

Allow, then, a thought experiment: what if Susan Reynard was given a diagnosis of astrocytoma in 2011 — but this time, we armed her with the most omniscient of lawyers, the most cutting-edge epidemiological information, the most powerful scientific evidence? Nineteen years and several billion cellphone users later, if Reynard were to reappear in court, what would we now

know about a possible link between cellphones and her cancer?

To answer these questions, we need to begin with a more fundamental question: How do we know that anything causes cancer?

The crudest method to capture a carcinogen's imprint in a real human population is a large-scale population survey. If a cancer-causing agent increases the incidence of a particular cancer in a population, say tobacco smoking and lung cancer, then the overall incidence of that cancer will rise. That statement sounds simple enough — to find a carcinogen's shadow, follow the trend in cancer incidence — but there are some fundamental factors that make the task complicated.

The most important of these is life expectancy, which is growing almost everywhere. The average life expectancy of Americans has increased — from 49 in 1900 to 78 in 2011. Several cancers are strongly, often exponentially, age-dependent. An aging population will seem more cancer-afflicted, even if the real cancer incidence has not changed.

But what if we make an “age adjustment” for the population and shrink or expand the cancer incidence to match the changes in age structure? To ask whether cellphones increase the risk of brain cancer, then, we might begin by turning to this question: Has the age-adjusted incidence of brain cancer increased in the recent past?

The quick answer is no. Brain cancer is rare: only about 7 cases are diagnosed per 100,000 men and women in America per year, and a striking increase, following the introduction of a potent carcinogen, should be evident. From 1990 to 2002 — the 12-year period during which cellphone users grew to 135 million from 4 million — the age-adjusted incidence rate for overall brain cancer remained nearly flat. If anything, it decreased slightly, from 7 cases for every 100,000 persons to 6.5 cases (the reasons for the decrease are unknown). In 2010, a larger study updated these results, examining trends between 1992 and 2006. Once again, there was no increase in overall incidence in brain cancer. But if you subdivided the population into groups, an unusual pattern emerged: in females ages 20 to 29 (but not in males) the age-adjusted risk of cancer in the front of the brain grew slightly, from 2.5 cases per 100,000 to 2.6. These cancers appear in the frontal lobe — a knuckle-shaped area immediately behind the forehead and the eye. It is difficult to imagine that cellphones caused these frontal-lobe tumors: how, or why, would a phone's toxicity have skipped over the area nearest to it and caused a tumor in a distant site? Most epidemiologists and biologists do not find such a tissue-skipping mechanism plausible and most doubt that there is any causal link between frontal tumors and phones.

But a populationwide survey, you might argue, has its limits. The carcinogenic effect of a phone might be so subtle that it never registers in such a survey. A phone may cause cancer after a

long lag time — say, 20 years — and it may be too early to look for an effect in a general population. The survey data could be incomplete or of poor quality, thus limiting an epidemiologist's ability to ever find a discernible link.

Epidemiologists, fortunately, possess a more powerful alternative to uncover a link between a risk factor and cancer. Consider the classic studies that finally revealed the association between tobacco and lung cancer. In the late 1940s, Sir Richard Doll and Sir Austin Bradford Hill, working in London, and Ernst Wynder and Evarts Graham, working in St. Louis, began investigating whether tobacco smoking increased the risk of lung cancer.

Working independently, Doll and Hill, and Wynder and Graham, devised remarkably similar kinds of surveys to reveal a possible link. Using hospital records, they identified a “case” group (a cohort of men with lung cancer) and a matched group of men without lung cancer (a “control” group).

The case group and the control group were asked the same questions, including how much and how often they smoked. By comparing the responses of lung-cancer-afflicted men and nonafflicted men, the two teams of researchers stumbled on a striking association: men with lung cancer had a much longer and deeper history of smoking compared with men without lung cancer.

What if you perform a similar case-control study with cellphones — comparing men and women suffering from brain cancer (cases) and men and women without brain cancer (controls) — looking at their past cellphone use? In 2010, an enormous study, called Interphone, tried to accomplish this task. Setting up the study took years: Interphone recruited participants in 13 countries, ran for a decade and included 5,117 brain-tumor cases and 5,634 controls. The study was coordinated by the [World Health Organization](#) and financed primarily by the [European Union](#) and cellphone companies, although by agreement industry representatives did not have privileged access to results before publication.

Trials like Interphone are undertaken in the hope that they cleanse the field of doubts. In fact, Interphone achieved just the opposite effect: it ignited even more puzzling questions. Over all, the study found little evidence for an association between brain tumors and cellphones. But when the two cohorts — cancer and no cancer — were subdivided according to the frequency of cellphone use, bizarre results emerged. To start with, there was an apparently *decreased* risk of brain tumors in regular phone users, compared with rare users or nonusers. In other words, regular cellphone use seemed to reduce the risk of brain tumors. In stark contrast, very high cellphone use (measured as a user's cumulative call time) seemed to increase the risk of a particular subtype of brain tumor. Needless to say, it is biologically implausible that these results are simultaneously true: how can regular cellphone use protect against cancer while

frequent phone use increases risk? To most epidemiologists, including the authors of Interphone, the results point to a systemic flaw in the trial.

Similar case-control studies have examined other kinds of brain tumors, including a rare nonmalignant tumor called an [acoustic neuroma](#). Here, too, the trials have been contradictory. Multiple studies found no association with cellphone use. In contrast, one study from Sweden found an increased risk in people who used their phones for more than 10 years.

How can trials that seem so similar at face value arrive at such disparate and contradictory results? The most likely common problem is bias — built into the very structure of these trials. In a case-control trial, patients are asked to remember their risk of exposure after the fact. In the Interphone study, for instance, participants were asked to recall the extent of their phone use years or even decades in their past. And memory, we now know, is a terribly slippery entity. A patient's memory of his or her past is a particularly charged and malleable thing; burned into David Reynard's memory, poignantly, is the shape of the cellphone in his wife's hand and the imprint of the cancer on her brain.

In fact, our memories turn out to be systematically fragile, especially when we are summoning our past to understand illness. In 1993, a Harvard researcher named Edward Giovannucci set out to measure this phenomenon. Giovannucci identified a cohort of women with [breast cancer](#) and an age-matched cohort without cancer, and asked each group about its previous dietary habits. The survey produced a reliable and reasonable trend: women with breast cancer were more likely to have consumed diets high in fat.

But the women in Giovannucci's study had also completed a dietary survey before their diagnosis of breast cancer. How did a woman's memory of her diet compare with the actual diet that she recorded before her cancer diagnosis?

Giovannucci's study illustrates the insidious nature of "recall bias." In women with no cancer, there was no change between the actual and remembered diet. But women with breast cancer typically recalled a much-higher-fat diet than they actually consumed. The diagnosis of breast cancer had not just changed a woman's present and the future; it had altered her sense of her past. Women with breast cancer had (unconsciously) decided that a higher-fat diet was a likely predisposition for their disease and (unconsciously) recalled a high-fat diet. It was a pattern poignantly familiar to anyone who knows the history of this stigmatized illness: these women, like thousands of women before them, had searched their own memories for a cause and then summoned that cause into memory.

It is very likely that similar effects undid the Interphone trial: some men and women with brain cancer recalled a disproportionately high use of cellphones, while others recalled

disproportionately low exposure. Indeed, 10 men and women with brain tumors (but none of the “controls”) recalled 12 hours or more of use every day — a number that stretches credibility. In a substudy of Interphone, researchers embedded phones with special software to track phone usage. When this log was compared with the “recalled” usage, there were wide and random variations: some users underreported, while others overreported use.

The trouble is that even the largest, longest, best-designed retrospective studies that rely on memory are likely to be riddled by recall bias. Typically, it is not the *failure* of memory that produces this bias, but its **hyperactivity** — its desire to explain the uncertainty of the present with the certainty of the past.

There are certainly methods in epidemiology to counteract the biases created by selective memory: Interphone researchers could have initially identified a cohort of high-volume cellphone users and of nonusers, and followed them over time to determine who developed or did not develop cancer. Such a study — called a “prospective trial” — would certainly erase the biases of memory. But it would be logistically impossible to perform. Since the rates of brain cancer are small, about 6.5 cases per 100,000 persons, a trial of this design would need to follow an enormous cohort of cellphone users — hundreds of thousands of participants — to record even a few cancers. And where on earth would you find the nonusers for the study? In most nations, cellphone usage is so common that finding 500,000 people who will not use phones for a decade is hard to imagine.

There are yet more powerful epidemiological methods that seem even more far-fetched. A trial that forcibly randomizes men and women to use cellphones or restrict phone use — a “randomized trial” — would certainly guarantee the most bias-free result, but would trespass inviolable ethical and practical concerns. Another study might try to minimize a person’s biased memory of exposure by collecting actual data on phone use from phone networks (scanning phone minutes and call logs from real bills), but this would violate privacy laws. Thus far, individual call logs — even anonymized logs — have not been made public to researchers.

What if we moved the studies from humans to animals? Benzene, benzopyrenes, methylcholanthrene and some aniline derivatives (among many other chemicals) were first discovered as cancer-causing agents using mice, rats and rabbits. Decades before Doll and Hill’s elaborate studies on tobacco smokers in London, an Argentine biologist, Angel Roffo, “painted” rabbits with a grey-black solution containing distilled cigarette tar and demonstrated that the smoky residue caused cancer.

Might an animal experiment identify the carcinogenicity of cellphone radiation that Interphone missed? Prototypical animal studies for carcinogens involve exposing one group of animals to the suspected agent and comparing it to the unexposed group. But as the 16th-century

physician Paracelsus reminds us, “It is the dose that makes the poison.” Determining the appropriate amount to test and delivering it to the right part of an animal’s body is often crucial to the experiment.

At face value, testing “radiation,” which is measured in standardized doses, would seem to make this simple. But all radiation is not created equal. The word “radiation” refers to energy that emanates from a source — but the spectrum of radiant energy is broad. On the highly energetic end of the spectrum is ionizing radiation — like X-rays or cosmic rays — that are so powerful they can tear away electrons from atoms and molecules and penetrate barriers like the skull and the brain. On the way into — and through — the body, they deposit powerful bursts of energy, generate corrosive chemicals, ruffle up DNA, kill cells and, most notably, mutate growth-controlling genes to cause cancer.

Nonionizing radiation lies on the other end of the energy spectrum. These rays can warm cells, boil water and stimulate chemical reactions, but they cannot strip electrons away from atoms or damage DNA. They have no capacity to mutate genes directly and thereby no simple and direct means of initiating cancer. Radiation from microwaves, from cellular phones and from light bulbs are examples of nonionizing radiation.

All of this makes cellphone radiation a relatively unlikely culprit as a mutation-causing agent. Nonetheless, biologists have exposed mice and rats to chronic nonionizing radiation (comparable to that emitted by phones) to determine whether it causes cancer. In rats prone to developing breast cancer, there was no acceleration of breast cancer. In another experiment, rats were treated with a chemical carcinogen in utero (to “prime” them to develop brain tumors) and then exposed to radiant energy comparable to cellphone radiation for two hours per day, four days a week, for 22 months. The experiment revealed no increased incidence of brain tumors in rats. Nor was there any accelerated growth in previously established brain tumors. From 1997 to 2004, six independent experiments on mice and rats studied the effects of chronic radiation on brain cancer. No experiment revealed an increased risk of brain cancer.

But radiant energy need not penetrate the brain and mutate genes to have a biological effect on it. A cellphone user might experience changes in physiology that have nothing to do with the ionizing capacity of radiation. Might a cellphone leave a physiological mark on the brain through a yet unknown mechanism?

A recent study by Nora Volkow, published in The Journal of the American Medical Association (JAMA) and reported in this newspaper on March 30, has raised this unusual possibility. Volkow is an innovative brain researcher who is director of the National Institute on Drug Abuse in Bethesda, Md. She recruited 47 people and placed an “active” phone next to one ear (the phone was on — generating radiation, but silent, so that Volkow could eliminate the effects

of sound and conversation). She then used a specialized brain scanner capable of detecting alterations in glucose. Glucose — a sugar — is the metabolic fuel for the brain. When parts of the brain are activated, brain cells begin to metabolize glucose at an increased rate. Volkow's scanner was equipped to detect even marginal changes in glucose metabolism.

When Volkow compared subjects with phones turned on with subjects who had their phones turned off, she found a striking pattern: there was a telltale sign of increased brain-glucose activity in the area of the brain immediately adjacent to the antenna of the phone.

But as Volkow points out, there is still a long conceptual leap from “increased brain-glucose activity” to “brain cancer.” Our brains are constantly altering the metabolism of sugar — the flux of glucose changes when we remember Grandma's house in Texas or listen to Bach or smell roses. When human beings dream during sleep, the increase in glucose metabolism in some parts of the brain is just as striking as the increase found in Volkow's study with phones. “It's not a dramatic increase,” she says. “When our eye responds to a visual cue, glucose metabolism in the brain increases much more dramatically” — and, surely, we do not think that visual stimulation causes cancer. Her study proves, importantly, that cellphone radiation has a biological effect on the brain. But whether this effect is consequential — whether it causes cancer or, for that matter, protects against it — is entirely speculative.

The most exquisite — and arguably the most sensitive — means to identify a carcinogen is to study the effects of the substance not on humans or animals but on cells. In the 1970s, a Berkeley biochemist named Bruce Ames devised a cellular test to do just that. Ames's test is based on a series of simple principles. Normal cells in the body grow through cell division, or mitosis, which is carefully regulated by genes. Certain genes accelerate growth, while other genes dampen or stop it. Cancer originates when the “accelerator” genes are permanently activated or when the “brake” genes are permanently damaged. Since genes are encoded by DNA, chemicals that mutate DNA — mutagens — can alter the growth-controlling genes and thereby cause cancer. Ames devised a special strain of bacterial cells that act as a “sensor” for mutations and therefore can also detect mutagenic chemicals. Chemical mutagens are so commonly carcinogenic that versions of the Ames test represent the gold standard by which most carcinogens are found.

Cellphone radiation is not a chemical, of course, but the rules about mutagenicity still apply (X-rays, for instance, are known to cause cancer and are detectable by Ames's test). Laboratory experiments that link phone radiation to DNA mutation using a version of the Ames test have been largely contradictory. In 2005, a panel of experts, including a biomedical engineer, an epidemiologist, a genetic toxicologist and a radiation biologist, published a review of nearly 1,700 scientific papers on the cellular effects of radiation emitted by phones. In the review of

more than 50 experiments linking phone radiation to DNA damage in animal or bacterial cells, evidence of damage has been negative in more than two-thirds of the studies. Since nonionizing radiation cannot directly affect the structure of DNA, experiments linking phone radiation to DNA damage are generally unconvincing. The most striking study linking cellular phone radiation to DNA damage, published in 2005 by researchers from the Medical University of Vienna, has recently been embroiled in even deeper scientific controversy: researchers studying the data intensively have argued that the original study is fraudulent.

But it is possible for something to be a carcinogen without directly damaging DNA. Some chemicals might activate growth pathways or survival pathways in cancer cells (eventually damaging DNA and mutating genes — but indirectly). Exogenous [estrogen](#), for instance, activates growth pathways in breast cells and can cause breast cancer but doesn't damage DNA. Others may provoke inflammation, creating a physiological milieu in the body that allows malignant cells to grow and survive. Yet others — the class of substances that we know least about — might not damage DNA directly but chemically modify genes so that their regulation is changed. These substances are like the dark matter of the carcinogenic world: they are barely visible to our current tests for carcinogens and thus lie at the boundaries of the knowable universe. Cellphones and their radiation have been tested for many of these properties — for instance, their ability to chemically modify DNA without causing mutations — but evidence linking this form of radiation to such cellular changes remains largely negative.

In the expert panel's 2005 review, the authors summarized the evidence: "There is little theoretical basis for anticipating that [RF](#) energy [from cellular phones] would have significant biological effects at the power levels used by modern mobile phones and their base station antennas. The epidemiological evidence for a causal association between cancer and RF energy is weak and limited. Animal studies have provided no consistent evidence that exposure to RF energy at nonthermal intensities causes or promotes cancer. Extensive in vitro studies have found no consistent evidence of [DNA damage] potential, but in vitro studies assessing the epigenetic potential of RF energy are limited. Over all, a weight-of-evidence evaluation shows that the current evidence for a causal association between cancer and exposure to RF energy is weak and unconvincing."

The word "carcinogen," it is believed, was first coined by the surgeon James Paget in an obscure passage of a lecture on surgical pathology in 1853. Paget asked if there is "one material for cancer, one carcinogen," that "may form different but closely allied compounds?"

Our vision of carcinogenesis has become vastly more complex since 1853. We now know that there is no "one cancer." Breast, lung, prostate and blood cancer share a similarity — the uncontrolled growth of cells — but the specific genes and behaviors of these cancers are far from

identical.

Nor is there “one material for cancer” — one archetypal carcinogen. Agents that cause cancer are chemically diverse and cancer-specific. Estrogen can provoke cancer in the breast, but destroys prostate-cancer cells; vinyl chloride is exquisitely carcinogenic to the liver but not to the skin; chlorine and nitrogen mustard are both poison gases, but only one causes leukemia.

Notably, there is also no “one test” for carcinogens. Scientific studies to capture the association between an agent and cancer cast an astonishingly wide net. On one end of that spectrum lie populationwide human trials involving hundreds of thousands of men and women. On the other end are precise laboratory experiments that plumb the molecular depths of cells and genes. The tests range from the telescopic to microscopic, from statistics to biochemistry — from observations of chimney sweeps to bacteria on a petri dish. Often one test must be corroborated by another. Asbestos and tobacco were identified by case-control studies and validated in animal models. Estrogens were implicated by studies on human and animal physiology and then found to be carcinogenic in prospective human trials.

Finding a carcinogen, in short, is not like solving a mathematical equation, with a single formula and solution. It is more like solving an epic detective case, with individual pieces of evidence that, taken together, suggest a common culprit.

But thus far, this extraordinarily wide-cast net has yet to find solid proof of risk for cellphone radiation: not a single trial or test that has attributed carcinogenic potential has been free of problems. Populationwide studies have failed to demonstrate an increased incidence; retrospective trials have been contradictory and riddled with biases; animal studies negative; human physiological experiments inconclusive; cellular studies inconsistent and weak. What is clearly needed, experts agree, is a single, definitive, unbiased study — “one trial,” to borrow Paget’s terminology. Logistically speaking, the simplest such human trial is a case-control study that compares cancer patients with healthy patients, using phone-log data that companies have thus far been reluctant to provide. The simplest animal study involves subjecting rats and mice to long-term exposure to cellphone radiation. The National Toxicology Program has begun such a study. Cellphone radiation will be turned on and off for 10-minute stretches for 20 hours each day. This experiment — the closest we will get to making mice use actual cellphones — is likely to be published in 2014.

It is possible, of course, that even these sophisticated experiments will be unable to determine the risk. The lag time of cancer development with phone use may be 50 or 70 years — and cellphones have been around for only three decades or so. Yet even a slow-lagging cancer is unlikely to arise at a single point in time after exposure. Like most biological phenomena, cancer risk typically rides a statistical curve, with some patients developing cancer early, others

peaking in the middle and yet others trailing off decades later. Thus far, no such statistical curve has been evident for brain cancer.

Might the cellphone industry have already performed such experiments and conspired to keep real data on brain cancers from us — just as the tobacco industry conspired to obfuscate real data on tobacco and carcinogenesis in the 1950s? It's possible, but there are important differences in comparing these trials with the tobacco studies. With smoking, despite active attempts by the industry to stifle data, the epidemiological trials were incontrovertibly positive, human physiological data markedly suggestive and animal studies (including Roffo's painted-rabbit experiment) striking.

As we await the definitive trial, then, it's probably wise to also start thinking differently about the cause of Susan Reynard's cancer. When a suspected cause for a devastating illness begins to slip away, there is often frustration and turmoil, paranoia and nihilism. In a short story by [Lorrie Moore](#), the mother of a toddler with cancer rattles off a list of potential causes of her child's illness — “giant landfills, agricultural run-off”; “lurid water”; “toxic potatoes”; “Joe McCarthy's grave.”

The trouble with this kind of grasping is that it is indiscriminate. In truth, many substances of modern life do not — cannot — cause cancer. Some do, and it's absolutely critical to identify and reduce exposure to them. Others don't, and it's absolutely worthwhile identifying these, so that we can focus on the real carcinogens around us. If we lump everything into the category of “potentially carcinogenic,” from toxic potatoes to McCarthy's grave, then our scientific language around cancer begins to degenerate. The effect is like crying “wolf” about cancer: the public progressively numbs itself to real environmental toxins and becomes disinvested in finding bona fide carcinogens.

To keep ourselves on the right path on environmental carcinogens, then, we need not just standards to rule carcinogens “in” but also standards to rule them “out.” The final, definitive trials on phone radiation may settle this issue — but, as of now, the evidence remains far from convincing. Understanding the rigor, labor, evidence and time required to identify a real carcinogen is the first step to understanding what does and does not cause cancer.

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