

10 April 2005
Lloyd Morgan
2022 Francisco Street
Berkeley, CA 94709
bilovsky@aol.com

David Heim
Deputy Editor, Consumer Reports Magazine
101 Truman Avenue
Yonkers, NY 10703
heimda@consumer.org

Dear Mr. Heim,

Some time ago now we exchanged e-mails regarding cellphones, their potential to create human harm, and the most recent research on the subject. Below is my long over-due response.

Your first query was regarding the peer-reviewed status of the Hardell and Hansson-Mild study that for the first time, includes risks by age group. The answer is that the presentation by Hardell and Hansson-Mild has been accepted for publication in a peer-reviewed journal (Arch Environ Health), but it is still "in-press". Here is the URL: (http://www.cost281.org/documents.php?node=77&dir_session=) where the study and presentation can be accessed on-line (it is also attached).

I will also endeavor to discuss the Hardell and Hansson-Mild data and its implications. However, as introduction, I think that it would be useful if I first provide you with some background to assist an understanding of the interpretive issues to be found in the Hardell and Hansson-Mild study and the studies that preceded it, and, second, offer, also as background material, a short historical synopsis of the cell phone scientific literature that was instrumental in convincing me of the harmful potential of continued cell phone use. I will end my discussion with some brief reflections on precautionary measures that I believe are critical.

Thank you in advance for your time and attention in this important matter. I fear, but hope that I am wrong, that we are at an historical moment, watching the tide recede prior to a health emergency tsunami that is poised to drown us in future health tragedies of unknown dimensions.

Sincerely,

A handwritten signature in black ink that reads "Lloyd Morgan". The signature is written in a cursive, flowing style.

Lloyd Morgan

Cellphones and Human Health

Introduction

It is my contention, *based solely on my reading of the scientific literature* to date, that human exposure to cellphones poses a major health threat. Yet, everywhere we hear sanguine reports of cellphone use. (The latest was a cheery story on CBS TV's Good Morning show, April 1st, 2005, regaling, without a mention of potential harm, the “wonderful” new cellphone products being developed by several corporations for 8-11 year olds!) What is to be made of these differing views?

The thesis of this discussion is that the divergent views on the health effects of cellphone exposure lie not in “the truth” of the scientific findings but rather in the pre-existing interpretive “booby traps” unique to studies involving brain tumors, and indeed unique to studies of cellphone use, especially at this stage of exposure. To therefore make sense of what can sometimes be seen as dueling studies and conflicting claims, this discussion will try to provide a working understanding of the interpretive “booby traps” which lay in wait. This examination will then be followed by a reinterpretation of the scientific literature in hopes of providing a fresh look—and a more accurate picture of what may befall us with near-universal exposure to cellphones in our future.

Interpretive Booby Traps of Cellphone Studies

1. Difficulty in Interpreting the “Numbers”

Actual harm caused by exposure to an activity/substance is generally measured by counting how much “bad stuff”, or cases of harm, is resultant. However, in many circumstances—most especially where the “harm” is manifested as brain tumors—this measurement is not so simple.

One issue in relying uncritically on the number of brain tumors developed after exposure is the rarity of brain tumors. In the broad brush, if there are too few cases, then the study will have little statistical power to determine whether, or not, a risk exists.

Rare diseases are difficult for epidemiology, as a relatively large study is required to have any statistical power to resolve an increased risk. Brain tumors, both benign and malignant, are indeed a rare disease with an incidence in the United States of 141 cases per million people per year. If only malignant brain tumors are considered, as is the case in many cellphone studies of brain tumors, then the US incidence is 73 cases per million people per year.¹ Acoustic neuromas, the most common brain tumor associated with

¹ 2004-2005, Primary Brain Tumors in the United States, Statistical Report, 1997-2001, *Year Data Collected*, p. 9; Central Brain Tumor Registry of the United States (www.cbtrus.org), 2004.

cellphone use is even more rare. The US incidence rate of acoustic neuromas is 54% of all nerve sheath tumors.² The incidence rate for acoustic neuromas is a mere 6 per million people per year.³

Generally, for statistical meaningfulness, it is felt that a sample of no less than 30 subjects is required. Though a given study may select a far larger number than 30, what is important is the number for the sub-groups of interest. For example, Christensen et al. (see Study 8 below) started with 141 cases. After excluding cases for various reasons, there were 106 cases. When they had determined how many cases actually used a cellphone there were 45 cases. When these cases were further divided into years of use, there were 27, 17, and 2 cases for 1-4 years, 5-9 years and >10 years respectively. Not surprisingly, when sub-divided into the years of use, all of the results were so far from statistical significance that there was no meaningful result.

So we see from this example, that a negative finding of harm in a scientific study with too small of a sample, must actually be interpreted as meaningless. It tells us nothing. It does not say there is no risk nor does it say there is a risk.

2. Cohort vs. Case-Controlled Studies

In interpreting cellphone studies it is not only important to examine the amount of raw numbers, but it is also vital for a meaningful interpretation to understand the context in which these numbers were obtained.

An example of the role that number context plays in cellphone studies can be found in the differences that lay in two epidemiological research models: cohort studies versus case-controlled studies. A cohort study uses a large population (the cohort), which is then used to examine how many cases are observed, versus expected, to determine whether there is an excess risk or not. For rare diseases such as brain tumors, there are rarely enough cases to determine if there is a risk or not. As an example of this difficulty to determine risk from a cohort study, there is a Danish cohort study, discussed later as Study 3. In this study the size of the cohort (Danish cellphone subscribers) is 420,095. Yet, because of the rareness of brain tumors, they only observed 11 temporal lobe brain tumors compared to 12.8 expected. The result is again so far from statistical significance that no meaningful conclusion can be drawn.

Case-controlled studies, on the other hand, are a better model design to study rare diseases. Case-control studies select cases, and then find controls to match each case. The importance of this approach is that the study starts by finding a sufficient number of cases (hopefully) to provide sufficient statistical power to determine whether or not there

² 2004-2005, Primary Brain Tumors in the United States, Statistical Report, 1997-2001, *Year Data Collected*, p. 7; Central Brain Tumor Registry of the United States, 2004.

³ Calculated result using Table 8, p.32 in 2004-2005, Primary Brain Tumors in the United States, Statistical Report, 1997-2001, *Year Data Collected*; Central Brain Tumor Registry of the United States, 2004.

is a risk above a defined level.⁴ Each control is commonly matched (a matched case-control study) to each case by various attributes such as age, gender, ethnicity, income and so on. With the selection of the cases and the controls, a common questionnaire is answered by each case and each control.

Questionnaires are not practical for cohort studies because of the size of the cohort, so less is known about the cohort than is known about the cases and controls of a case-control study. The Danish cohort study (Study 3) was accomplished using data linkages between the cellphone company's records and the Danish cancer registry information. No member of the cohort was contacted.

3. Latency and Duration of Exposure

Another interpretive difficulty is assessing the harm within the context of the latency time between exposure to a possible carcinogen and the diagnosis of the tumor. When analyzing a cellphone study it is essential to understand the length of time that a subject has been using a cellphone. A study may have a reasonable number of brain tumor cases, and the context of these cases may make these numbers robust, but when we consider some minimum time of use, the number of brain tumor cases is reduced, often substantially.

With all cancers, there is a significant latency time between exposure to a carcinogen and tumor diagnosis. It is generally considered that the latency time between exposure and diagnosis spans several decades.⁵ What is alarming is that cellphone studies are reporting much shorter latency times. One cellphone study reports median latency times of 7 years for analog phones, 5 years for cordless phones, and 3 years for digital phones;⁶ another study reports a latency time for acoustic neuromas from cellphone exposure of 10-years or greater.⁷ If we believe that average latency times are on the order of several decades, then we can assume that these initial findings of tumors that reflect only one decade, are just the tip of the iceberg. If these early indications of a risk for brain tumors are true, then what will be the increased risk when cellphone exposures have existed for multiple decades?

4. Ages of Subjects

It is also important to recognize that the ages of the subjects in cellphone studies warrant interpretive attention. That is, the risk of a brain tumor is much higher in younger, than in older people. This age-relatedness of increased cancer risks to exposure to carcinogens was first established in the study of exposure to ionizing radiation (as opposed to the non-ionizing radiation of cellphones) in atomic bomb survivors. It is generally accepted that

⁴ Statistical power is a formal and calculable term. For example, a paper may state, "The statistical power of this study provides an 80% confidence of finding a 2-fold risk or higher, if there is a risk."

⁵ As but one example of such reports, see Ionizing radiation and chronic lymphocytic leukemia.; Environ Health Perspect. 2005 Jan;113(1):1-5.

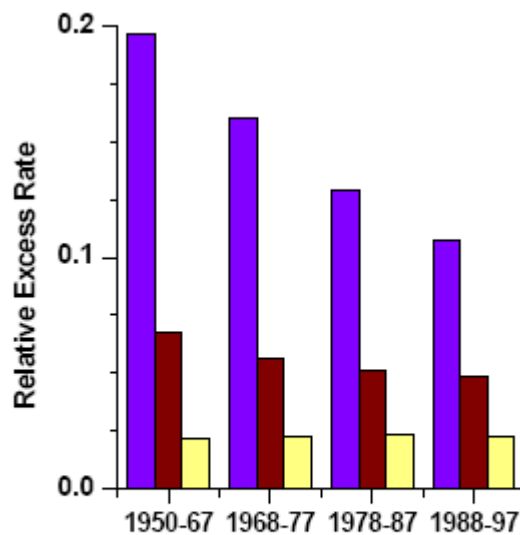
⁶ European Journal of Cancer Prevention 2002, 11, 1-10

⁷ Epidemiology 2004;15: 653-659)

the reason the risk from a carcinogen is higher for younger people is because the young have more cells dividing.

Figure 1 below shows the relative cancer rate excess among atomic bomb survivors. The graph clearly shows that age at exposure is a strong function of the relative excess rate of solid cancers for ionizing radiation.

Figure 1
Relative Excess Rate of Solid Cancers by Age at Exposure from Atomic Bomb Survivors (Ionizing Radiation)



(LSS Report 13, Preston et al, 2003)

Age at exposure
0 - 19
20 - 39
40+

While an argument may be made that there are no published studies linking exposure to non-ionizing cellphone radiation with higher carcinogen-risk for younger age groups, the Hardell and Hansson-Mild study (Study 10) does just this. It shows far higher risk for the youngest age group than for older age groups.

Any indication of increased risk for younger groups suggests that the risk of cellphone use by children may be higher yet. Study 10 should be read as a “canary in the mine” warning. Indeed, scientists and policy makers in the U.K. have been so alarmed at the

first studies finding increased harm to youth that an advisory warning has been instituted for cellphone use by children.⁸

5. Tumor Location Analysis

Another interpretive risk for which we need to be mindful is failure to pay close attention to both the location of the tumor and the location of the cellphone exposure in relation to the tumor. Brain tumors that are counted for a study that are beyond the cellphone's radiation "plume" (e.g., brain stem or cerebellum tumors) may result in an inaccurate interpretation of the true dangers, if any, of cellphone use. On the other hand, when the location of cellphone exposure (i.e., the side of the head where the cellphone is held) is not indicated, the ability to measure the risk between location of exposure and location of tumors is unfortunately diluted. Ideally a study should only report temporal lobe and acoustic neuroma tumors occurring on the same side of the head where the cellphone was predominantly used, since this is where the cellphone radiation plume is strongest.

6. Differing Wireless Phone Technology: Does It Matter?

Yet another interpretive trap to be wary of is proffered arguments denying harm based on differing wireless phone technologies. Along these lines it has lately become fashionable to dismiss any positive finding of harm to the fact that the analysis finding harm is based on "old" technologies, while pointing to the fact that the "newer" technologies show less, or no findings of harm.

For example in Studies 5 and 6, an elevated risk of harm is found for users of analog cellphones (an older technology) but less risk of harm for cordless phones (an intermediate technology) and the least for digital cell phones (newest technology). The higher risk of harm that is evident for users of analog phones is brushed away as a non-issue because, "Analog cellphones are not used any more, anyway". However easy this brush off may be, the facts of the matter deserve a deeper examination.

Firstly, the differences in technology between analog and digital cellphones or cordless phones are arbitrary and are based on engineering terminology—concepts that have not been shown to have a biological basis. These engineering differences between analog and digital cellphones or cordless phones are changes to modulation techniques (how information is created by differing signals), and in changes to carrier frequencies (different frequencies change "channels" to receive, or send, differing signals).

Secondly, cellphone technology is constantly changing, but the changes continue to be focused on the engineering changes described above. Therefore, assuming that modulated radiation may be the factor of harm, then the "different" exposures provided by each new cellphone technology rather than being altered, remain constant.

Thirdly, what does change, and is reflected in all cellphone studies (and is the true relevant factor concerning differing technologies), is the length of time of use. As discussed in Interpretive Booby Trap 3, "Latency Time", one of the most important

⁸ Associated Press report at <http://www.forbes.com/home/feeds/ap/2005/01/11/ap1752648.html>

factors in any study concerning exposure to substances that may cause cancer or tumors, is the length of time of that exposure. It makes perfect sense that analog cellphones (oldest used) show consistently highest risk of harm, as opposed to digital cordless phones (next oldest, with a risk between analog and digital cellphones) and last, digital cellphones (newest and showing least risk): analog cellphones have been used the longest and have been used by more people the longest. The other two phone technologies studied have a lower latency time, and have been used by a smaller population. It is therefore clear that when a study shows findings of harm from exposure to cellphones, regardless of its technology, old or new, if the technology of greatest length of exposure shows increased harm—it is cause for great concern. And there is no reason to believe that as the newer technologies are used for an equivalent period of time, say 10 years plus, that similar factors of risk will not be seen.

A last point on the significance of differing wireless phone technologies is that of the difference in “power” between analog, and digital cellphones and digital cordless phones.

It is true that analog cellphones are almost 10 times more powerful than digital cellphones or cordless phones (cordless phones radiate about the same power as digital cellphones). But the extra power is not the same as extra “dose” with which it is often confused.

In fact there are good reasons to postulate that it is pretty much the same absorbed exposure, independent of the radiated power. First, while the older analog phones radiate more power, they also were housed in a larger, thicker casing so that they were held further from the head. Applying the inverse square law (radiated power decreases by the square of the distance from the source, i.e., the cellphone), the higher power of analog phones is nullified by the further distance with which the analog cellphone was held as opposed to the slimmer digital phones that are held much closer.

To illustrate the impact of the inverse-square relationship, if one person uses a digital phone $\frac{1}{16}$ inch from the ear and the next person uses an analog cellphone $\frac{1}{4}$ inch from their ear, the absorbed power will be less for the analog cellphone (79%) as compared to the 10-fold lower power digital phone.

Second, there has been a tendency to use cordless phones for much longer conversations, since the presumption (erroneous) is that they are “safe” in comparison to cellphones. This is particularly true in the case of children, where parents have felt more comfortable providing cordless phones, rather than cellphones, for their children’s use at home.

Third, precisely because analog cellphones generate more power, they also generate increased heat and the anecdotal evidence is that many users of these first wireless phones correspondingly kept their phones a tiny bit further from their ear. Thus, how the cellphone is held relative to ones’ ear tends to have more to do with absorbed power than does the actual power generated by the cellphone.

7. Harm Standards: Do They Make Sense?^{9,10}

Another interpretive dilemma regarding cellphone studies concerns the very standard that the cellphone scientific community demands for a finding of harm. This scientific community actually demands the meeting of a two-tier test as the level of certainty required for causal evidence linkage. The level of certainty required for this cumulative test is 95%-99%—a nearly impossible scientific hoop to jump through.

The first requirement that must be met is a finding of “95% confidence level” for each individual scientific study before a claim of “concern” of harm will be accepted. The general meaning of “95% confidence level” is that there is only a 5% chance that a given result happens by chance; or, conversely, that there is a 95% chance of the result being a true result.

To further compound the difficulty of producing an accepted finding of harm, scientists studying the biological effects of cellphone exposure must satisfy the second-tier requirements that include, but are not limited to, the following: multiple replications of each finding at the cellular (in-vitro), the animal (in-vivo), and human epidemiological levels (and again, all these studies must meet the 95% confidence level). Additionally, these combined results must then reach a consensus finding of harm by both the independent and the cellphone industry components of the scientific community (see discussion in *The Role of “Industry”*, below).

As can be seen, these multiple standards require not only virtual certainty in a complex world where not much can be found to be absolutely certain, but also endless funding and time.

To fully comprehend the scientific trap that we have created by using this impossible set of standards it is instructive to examine the case of tobacco. It is now readily accepted by both the lay and scientific communities that tobacco is carcinogenic and therefore “causes” cancer. Yet scientists conducted decades of animal studies without a single “positive” finding. Indeed, today the evidence of harm for tobacco could not possibly reach the standard for concern for which cellphones are now held. However, with only about 10% of the smoking population being diagnosed with lung cancer, we would be negligent, indeed, to not at least call for a cautious use of tobacco.

How do other communities that shape public policy verify that harm has occurred?

⁹ This discussion is based in part on a presentation by Cindy Sage during the Bioelectromagnetics Society’s meeting, Washington DC, June 2004.

¹⁰ Part of this discussion, and the next section (*The Role of “Industry”*) are also based on comments by Dr. Michael Kundi in the March/April 2004 Bioelectromagnetics Newsletter.

American Legal System

For almost all civil cases, the standard required for evidentiary findings of proof in the legal system is preponderance of the evidence; in other words, is the “existence of a particular fact more probable than its nonexistence”.¹¹ This standard of proof, then, really only requires the barest tipping point beyond equipoise, or, approximately 51%.

The legal system also has two other standards: clear and convincing proof (which asks for a “substantially greater degree of belief concerning the existence of a fact”);¹² and for criminal cases, beyond a reasonable doubt (which calls for proof that is “inconsistent with any other rational conclusions”).^{13,14} Although the degree of certainty that is required for these two stricter legal standards is not usually expressed as exact numerical percentages, a reasonable guess could interpret these standards as somewhere between 70% and 80%—a requirement of certainty that is far from the 95%-99% certainty required of cellphone science.

Finally, as to legal standards of proof, it is important to note that the legal community has long recognized the fact that in special circumstances there may be a need to create new standards. And one factor that has been used to determine the need for such a revision is to ensure “*the most desirable result in terms of public policy*”.¹⁵ Why is there not such a rational option in the scientific community where lives are potentially at stake?

California Environmental Quality Act

The California Environmental Quality Act (CEQA) requires a “threshold of significance” for a given detrimental environmental effect. Court cases have interpreted this “threshold of significance” to be somewhere between a 10% and 30% chance of a detrimental environmental effect. So again, instead of requiring a near certainty of environmental damage, as the scientific community demands, the environmental community’s threshold of 10%-30% can be expressed as requiring only a standard of a “potential” for significant impact.

Some Thoughts on a New Standard For Cellphones

Why do we accept lower thresholds for the findings of legal liability and environmental damage, than we accept for the acknowledgement of human health hazards? One must query if this high standard is related to the economic needs of industry rather than to sound public policy promoting human health. Whatever the motives, it is essential that while we are reviewing the cellphone literature we view these studies that are being published with a more sensible standard, for the standard being applied verily militates against a finding of harm—even when cases of harm exist.

¹¹ West’s California Evidence Code, p. 72.

¹² Ibid.

¹³ Black’s Law Dictionary, p. 1381.

¹⁴ The citation in West’s Annotated Codes, Evidence Code, Sections 1-599, p. 561, 1995, sub-section 11 “Clear and Convincing Evidence”, and sub-section 12 “Absolute Certainty” make it clear that even for this more stringent standard of proof it “does not mean absolute certainty”, “it is not equivalent to...unanswerable evidence”, and “conclusive proof is never necessary”.

¹⁵ West’s California Evidence Code, p. 73.

Perhaps if there were a true intention to require such a high standard of “scientific certainty” for cellphone research, it would be wise to also add into the hopper for scientific “weight” the “risk factor” for cellphone use. Risk factors (also known as “odds ratio”, “OR”, “relative risk”, or “RR”) are routinely calculated to determine the correlation between the exposure to a substance/activity and health effects. (See Figure 2, below, which compares cellphone risk to the known causes of cancer: asbestos, second-hand tobacco smoke, X-rays, sunlight and radon, a naturally occurring radioactive gas.).

Figure 2

Some examples of known cancer risks relative to cellphone brain tumor risk				
Cancer Type	Risk Factor	Relative Risk	Comments	References
Lung	Asbestos +	2.55 6.08	Insulation workers 10-14 yr employment Insulation workers 30-34 yr employment	Selicoff et al. (1979)
Brain	Wireless phones	4.2*	Median use 7 yrs, tumor on same side of head as phone use, analog cellphone	Hardell et al. (2003)
		3.9*	≥10 years use, tumor on same side of head as phone use, analog cellphone	Lonn et al. (2004)
		8.19	20-29 year old group, >5 years use, analog cellphone	Hardell & Hansson-Mild (2004) ¹
		4.30*	20-29 year old group, >5 years use, cordless phone	
Breast	Second-hand smoke +	3.1*	Women exposed to tobacco smoke for at least 1 hr/day for at least 1 consecutive year	Morabia et al. (1996)
Brain	X-rays +	2.5*	Child tumors: >5 full-mouth dental X-rays	Preston-Martin et al. (1982)
Melanoma	Sunlight +	2.3* ?	Women in San Francisco who have never used sunscreen	Holly et al. (1995)
Lung	Second-hand smoke +	1.19*	U.S. nonsmokers	EPA (1992b)
		1.38*	U.S. nonsmokers, highest exposed groups	
Lung	Home radon +	1.20	U.S. nonsmoking women, highest exposure	Alavanja et al. (1994)
		1.20	Sweden, non-smokers, highest exposure	Pershagen et al. (1994)
		1.80*	Sweden, all subjects, highest exposure	

* Finding is statistically significant
 ? There was a statistically significant dose-response
 + Factor is generally considered to be known cause of cancer, although not necessarily of all types, and under conditions in these examples.

¹ Presented at European Cooperation in the Field of Scientific and Technical Research (COST), Potential Health Implications from Mobile Communication Systems, Budapest, 10 May 2004. In-press Arch Environ Health.

Adapted from Table 3.9, Bonneville Power Administration, December 1996. Electrical and Biological Effects of Transmission Lines: A Review

For example, the risk factor for developing lung cancer from second-hand smoke is 1.38 for highest exposed non-smokers. This risk factor translates to a 38% higher risk of developing lung cancer for those persons exposed to second-hand smoke than for those persons who are not exposed to second-hand smoke.

Because of this high risk factor it makes sense that a correlation between exposure to second-hand smoke and the risk of developing lung cancer is accepted as common knowledge. But then it should also stand to reason that, with a risk factor (using data from two of the studies discussed, below) of developing brain tumors from cellphone exposure ranging from 3.9 to 8.19, the connection between harm and cellphone use would also be accepted knowledge. Amazingly, for contested knowledge, these numbers

translate (for the youngest group for which data has been collected in the Hardell and Hansson-Mild study) to a 719% higher risk of developing a brain tumor from cellphone exposure than for those not exposed to cellphones.

As we read each new cellphone study with ever-increasing evidence of harm, but accompanied by contradictory conclusions of “no risk”, it is important to consider that our steadfast adherence to a scientific *certainty standard* legitimizes a glaring public policy paradox. For the demand for scientific certainty where cellphones are concerned, is in effect a declaration that we are more willing to condemn countless persons to carcinogenic exposure—risking death, even for small children—than we are to condemn one person to death in a capital criminal case with a sole requirement of “beyond a reasonable doubt”.¹⁶

8. *The Role of “Industry”*¹⁷

The final and most pervasive interpretive trap is, in elephant-sitting-in-the-middle-of-the-room fashion, the trap that no one dare speak its name: the role of the cellphone industry in cellphone science. And yet, this trap is now almost the entire story when it comes to cellphone research and any attempts to ensure safe use of cellphones.

To be fair, the cellphone industry, as one can well imagine, has much to be concerned about—basically their economic bottom line—if they did not become fully engaged in cellphone research: they are concerned about manufacturing changes that they may be asked to make; concerned about potential lawsuits (remember—the standard is lower for findings of liability than for findings of health risk); and ultimately they are concerned about a potential loss of their ever increasing revenues.

Despite the fact that the cellphone industry’s intimate involvement with all aspects of cellphone science poses a glaring conflict of interest, this industry that has so many self-interest agendas, has managed to infiltrate all aspects of cellphone science and now controls, to a large extent, the three major areas in cellphone science: the funding, the science, and the creation of cellphone use policy.

Funding

The cellphone industry pours large amounts of money into all aspects of this field and consequently, controls the vast majority of the research by funding their own studies, hiring key researchers from Universities and government, and putting financial pressure on Universities and government to nix research in the field or fund only studies designed to be incapable of finding a risk (e.g., using too few cases, or with too short of a latency time). They also use their funding prowess for PR obfuscation efforts that control the reports in the mass media (CBS is owned by Viacom and cellphone advertising is a major source of media revenue).

¹⁶ From testimony given to the California Public Utilities Commission by Dennis Zell, April 4, 2005.

¹⁷ Information in this section is a compilation of information from Dr. Michael Kundi (Institute of Environmental Health, Vienna, Austria), Dr. Louis Slesin (Publisher, Microwave News) and attendance for 10 years to scientific meetings in the field.

Science

Not only does the cellphone industry control the research by being a major funder of the research it has also made sure that its personnel are placed on safety standard committees and even into the World Health Organization's EMF study projects. The cellphone industry also controls the science by controlling the interpretation of the findings and how it is presented—if it is presented, to the public. Again, as far as controlling cellphone science interpretation many of the cellphone corporations, particularly Motorola, have their own “team” that travel to all the major meetings throughout the year, insisting on a negative “spin” of each presentation that points to a finding of harm.

Policy

The cellphone industry controls the policy arm of cellphone science by ensuring that all policy bodies (for example, the World Health Organization) are led by or have a majority of cellphone industry personnel and consultants as their staff. This is even true of scientific organizations: the Bioelectromagnetics Society (BEMS), which ought to be comprised of independent scientists has a large contingent of its members who are Motorola employees or Motorola paid consultants. Not only are the science labs, governmental offices, and science interest groups associated with cellphone science becoming operatives of the phone industry, but the media, both in-house and public is now so controlled by the phone industry that it is very difficult to find a way to disseminate any other information other than the cellphone industry's official line of “no harm”.

The most chilling involvement of the cellphone industry is a growing campaign to stop all cellphone research. At the 2003 BEMS meeting, Joe Elder, a Motorola employee (formerly employed at the U.S. Environmental Protection Agency (EPA) where he was in charge of the EPA's RF health review), actually presented a slideshow—in the time slotted for the sharing of scientific findings—that called for not only the cessation of all new cellphone studies, but the immediate halt to all on-going research, pinned on the justification that “it is clear that there is nothing to be found”.

A Reexamination of the Scientific Literature Regarding Cellphone Use

To complete the picture of the genuine risks that exposure to cellphones presents, it is important to now reexamine the scientific literature as stand alone evidence, as well as in the light of the interpretive traps discussed, above.

To date there are now thousands of studies that examine the biological consequences of exposures to cellphones. These studies range from those involving various species of animals in a cellular state (in vitro studies) to live (in vivo) animal studies. As of Spring 2005 there now is also a large body of epidemiological studies—the “in vivo”, if you will, study of human populations focused on determining what, if any, biological responses they have experienced with exposure to cellphones.

For the purposes of this discussion on cellphone studies, the focus will be limited to this epidemiological research. And because the epidemiological data confirms that by far the strongest risk associated with cellphone studies is that of acoustic neuromas, all but one of the ten studies highlighted, below, will be epidemiological studies that link acoustic neuromas with cellphone exposure. The one exception will be the Hardell & Hansson-Mild study that looks at brain tumor data by age groups.

These studies show that from the beginning, rather than a cause for sanguinity, there was cause to be concerned.

1. Board Presentation: CTIA, February 1999.

The earliest report that cellphone use increases the risk of acoustic neuromas was made to the cellphone industry's own trade organization, the Cellular Telecommunications Industry Association (CTIA). The person giving the presentation, Dr. George Carlo,¹⁸ was probably as knowledgeable about the status of the research on the risk of brain tumors from cellphone use as anyone on the planet. He ran the cellphone industry's \$25M research program for 6 years. The presentation he gave to the full board of the CTIA was sobering. He told the board, "[T]he risk of acoustic neuroma . . . was 50 percent higher in people who reported cellphone use for six years or more; moreover, that relationship between the amount of cellphone use and this tumor appeared to follow a dose-response curve."¹⁹

2. NCI-Inskip, Cellular-Telephone Use and Brain Tumors, Dec. 2000

The earliest scientific report of a risk of acoustic neuromas as a result of cellphone use came from a paper entitled "*Cellular-Telephone Use and Brain Tumors*" conducted by a National Cancer Institute team.²⁰ The impetus for this study was a January 21, 1993 Larry King interview of a grieving husband, David Raynard, who had just filed suit on behalf of his wife Susan, alleging that her death from a brain tumor was the result of cellphone use. Because of the public alarm raised by the Larry King interview, the National Cancer Institute (NCI) pushed for an epidemiology study of the risk of brain tumors from cellphone use. In spite of concerns that it was too soon for a study because cellphones had not been in use long enough, the study proceeded.

The resultant NCI-Inskip et al. study presented some 20 findings of the risk of acoustic neuromas from cellphone use. Not one of the 20 results reached the "statistical significance" level (i.e., $\geq 95\%$ confidence, or equivalently $p \leq 0.05$). The reason for the lack of statistical significance? There were too few cases of acoustic neuromas.

This study included only 25 cases of acoustic neuromas (enrollment in the study was between June 1994 and August 1998, a time when it was still rare for a cellphone to be

¹⁸ Dr. George Carlo, Chief Scientist, Wireless Technology Research (established by CTIA) oversaw multiple "scientific" studies funded by the CTIA.

¹⁹ Page 205, *Cell Phones, Invisible Hazards in the Wireless Age* by Dr. George Carlo and Martin Schram, Carroll & Graf Publishers, Inc., 2001.

²⁰ Inskip PD, et al.; *Cellular-Telephone Use and Brain Tumors*. N Engl J Med 344(2):79-86, 2001.

used).²¹ Taken by itself, this study gave little reason for concern, at least concern for short-term use of a cellphone.

However, the study did show for exposures of >3 years, or exposures >100 hours, or use from 1993 or before, a possible increased risk of acoustic neuromas, but with large p-values, it was hard to make a case for increased risk (see the Warning in footnote 18). However, taken in the context of all the studies that were to follow, this study was an early warning, that there might be reason for concern. The odds ratios, with p-values, number of cases and exposure time is summarized in Table 1 below:

Table 1, Risk of Acoustic Neuroma from Cellphone Use

AN ²² Odds Ratio ²³	Calculated p-value ²⁴	Number of Acoustic Neuroma Cases	Exposure Time
1.4	0.438	10	> 3 years
1.9	0.261	5	> 5 years
1.4	0.445	9	> 100 cumulative hours
1.0	NA	1	>500 cumulative hours
1.2	0.733	6	Use began during 1992 or before
1.3	0.764	2	Use began before 1990

Note: while none of the p-values comes close to “significance”, if we ask ourselves the question, what is the chance that 5 of the results would show increased risk and none would show decreased risk, then, using a binomial probability calculation, the conclusion is that there is an increased risk (with a binomial analysis we cannot say the size of the increased risk) and the probability is 97%, a “statistically significant” result.

²¹ In June 1994 there were only 19 million subscribers, by August 1998 this had grown to about 65 million. Three years earlier (an implied, but very short latency time) between June 1991 and August 1991 the number of subscribers was 6 million and 7 million respectively. At the end of 2004 there were 180.5 million subscribers. Abstracted from CTIA'S SEMI-ANNUAL WIRELESS INDUSTRY SURVEY RESULTS, June 1985 - December 2004

²² AN: Acoustic neuroma

²³ Odds Ratio: Odds ratios >1 indicates an increased risk. Odds ratios <1 indicates a decreased risk. If there is no risk, it can be expected that roughly 50% of the odds ratios will be >1 and the other 50% will be <1.

²⁴ p-value is a common term used by statisticians. It refers to the probability that a finding is due to chance. p-values, here and in all the following reports of p-values, are calculated from the published 95% confidence intervals. Such calculations are subject to rounding error. Common practice is to term a p-value ≥ 0.05 as “statistically significant”. Subtracting 1 from the p-value given the “confidence”, thus p=0.05, or p=0.01, or p=0.001 is equivalent to 95%, 99% and 99.9% confidence.

WARNING: I present calculated p-values throughout this discussion. Results with p-values larger than 0.05 require caution and results with p-values larger than 0.10 require extreme caution. An example of a severe misinterpretation result would be to interpret getting 3 heads on three consecutive tosses of a coin (p=0.125) means that there is a 12.5% chance that the coin is fair and a 87.5% chance that the coin is biased. Such a result does imply that more coin tosses are required (equivalent to more brain tumor cases) to determine if the coin is a fair coin.

3. Cellular Telephones and Cancer—a Nationwide Cohort Study in Denmark, Feb 2001²⁵

The next study, as was the case with the previous NCI-Inskip study, was interpreted to put any concerns about the risk of brain tumors at bay.

Though this study did not report on acoustic neuromas directly it did include a few cases of nerve sheath tumors. It is part of this discussion only because an acoustic neuroma is a nerve sheath tumor. This Danish cohort study was published just a few weeks after the NCI-Inskip study, sponsored in part by two large Danish cellular telephone operators, TeleDanmark and Sonofon. This study reported, “. . . [No] association between use of these telephones and tumors of the brain or salivary gland, leukemia, or other cancers.” Because this study relied on a cohort of 420,095 subscribers from 1982 to 1995, it purported to be the definitive answer to the question of risk of cancers from the use of cellphones. However, use of this cohort study, as a definitive answer was illusory. As discussed above, cohort studies for rare diseases are quite limited. This is true, not only because few cases can be expected for rare diseases, but also because there were few cellphone users who would have used a cellphone long enough to meet a cancer’s expected latency time.

The Table 2 illustrates how few cellphone users were included in the study’s early years.

Table 2, % of Cellphone User by Year Groups

Cellphone Users	% Of Total	Years
3,819	0.9	1982-1984
7,866	1.9	1985-1987
21,292	5.1	1988-1990
96,959	23.1	1991-1993
290,159	69.1	1994-1995

The average use of analog cellphone in this study use was only 3.5 years and users of the digital cellphone averaged only 1.9 years. Yet this study is often cited as “proof” that there should be no concern that cellphones cause cancer.

It is not surprising that this study with so few cellphone users in the early years of the cohort and the extreme rarity of acoustic neuroma had almost nothing to report. However, it did report 7 cases of nerve sheath tumor with 10.9 expected. Acoustic neuromas are a nerve sheath tumor, but it is unclear whether any of these 7 cases were acoustic neuromas. If they were, based on the US incidence data, there would only be 3, perhaps 4, acoustic neuromas; a number too small to draw any conclusions.

4. Muscat, et al.; Handheld cellular telephones and risk of acoustic neuroma, Apr. 2002²⁶

This study also reported no risk of acoustic neuromas.

²⁵ Johansen et al., Journal of the National Cancer Institute, Vol. 93, No. 3, February 7, 2001

²⁶ Muscat et al., Neurology 2002;58:1304-1306

The abstract reports, “The relative risk was 0.9 (p = 0.07) and did not vary significantly by the frequency, duration, and lifetime hours of use. In patients who used cellular telephones, the tumor occurred more often on the contralateral than ipsilateral side of the head.” Unless one reads further, this study says there is no problem (remember, a result <1 indicates the possibility of a decreased risk).

However this acoustic neuroma study, *funded by the cellphone industry*, found a non-significant increased risk of acoustic neuroma when the cellphone was used for > 3 years (see Table 3, below).

Table 3, Risk of Acoustic Neuroma for Greater Than 3 Years of Cellphone Use

AN Odds Ratio	Confidence Level	Number of Patients	Exposure Time
1.7	0.361	12	> 3 years

Of the 90 acoustic neuroma patients in the study, only 18 patients (20%) reported using a cellphone “regularly.” The average cellphone use for patients was 4.1 years. Yet for ≥3 years (12 patients), there was a 70% increased risk of acoustic neuroma, albeit with such a large p-value, caution is important. With only 18 patients using a cellphone “regularly” even the finding reported in the abstract was not significant. Because there were only 12 patients using a cellphone for greater than 3 years, the confidence level of the risk is further diminished (i.e., the p-value was increased). It is disingenuous that the abstract of this *cellphone industry study* reports a “non-significant” decreased risk—without even a mentioning of a non-significant and low potential of a 70% increased risk.

While this Muscat study reports that, “There was no evidence of a trend in the odds ratio with increasing levels of exposure,” the very scientist who managed this study, Dr. George Carlo, reported that, “Muscat’s analysis concluded that the increased risk was statistically significant after patients had used cell phones for six years.”²⁷

5. Cellular and cordless telephones and the risk for brain tumours²⁸, August 2002

This study presents a strong reversal of what was reported in the previous 3 studies. And, as you will see in the studies that follow, the risk of acoustic neuromas from cellphone use now becomes very clear.

A few months after the cellphone industry funded-Muscat study, an independent group from Sweden’s National Institute for Working Life (equivalent to the US OSHA) and from Örebro University reported a strong risk of acoustic neuromas with cellphone exposure.

It is important to recognize that this study’s results *probably understated the risk of brain tumors* because it excluded 37% of potential cases. If the patient had died or was unable to fill out the questionnaire they were excluded from the study. Also, all acoustic

²⁷ Dr. George Carlo and Martin Schram, Cell Phones, Invisible Hazards in the Wireless Age, p170; Carrol & Graf Publishers, Inc. 2001.

²⁸ European Journal of Cancer Prevention: Volume 11(4) August 2002 pp 377-386

neuroma cases were histologically confirmed. If there was only an MRI diagnoses of an acoustic neuroma the case was excluded. Histological confirmation can only be done with surgery or biopsy. As a result histological confirmation *could severely underestimate the risk* of acoustic neuromas as radiation, rather than surgery, is common.

The median time for cellphone use to diagnosis of a brain tumor was 7, 5, or 3 years with analog, cordless and digital phones respectively. Table 4 summarized the risk of acoustic neuroma from cellphone exposure.

Table 4
Risk of Acoustic Neuroma by Phone Technology and Cellphone Time of Use

AN Odds Ratio	Calculated p-value	Number of Cases	Exposure Time	Comments
3.5	0.000163	38	<1 year	Analog phones
3.7	0.000298	26	>5 years	
3.5	0.115	7	>10 years	
1.03	0.910	30	<1 year	Cordless phones
1.8	0.232	11	>5 years	
2.0	0.556	2	>10 years	
1.2	0.524	23	<1 year	Digital phones; Study period ended less than 10 years after digital phones introduced.
2.0	0.556	2	>5 years	
		0	>10 years	

With this study, for the first time there was evidence of an extraordinary report of the risk of acoustic neuromas from exposure to cellphones. This evidence should be viewed as especially compelling because the p-values were very small, equivalent to an over 99.9% confidence level that the results were not due to a chance finding.

Note also that that all results for acoustic neuroma risks show an increased risk, albeit often with a low confidence level. It is important to realize once again that the lack of confidence is the result of too few cases of cellphone use for any length of time.

6. Further aspects on cellular and cordless telephones and brain tumours,²⁹ February 2003

Six months later additional results from Study 5 were published. Not only had the risk of acoustic neuromas increased to over 4-fold, the statistical significance had also increased. Additionally there were now sufficient cases to report results of the risk of tumors on the same side of the head where the cellphone was used.

Not only was the risk increasing, but, for the first time, there was sufficient data available to show trend data. This study found not only an increased risk for acoustic neuroma, but also an increasing risk of acoustic neuromas with each additional year of cellphone use.

²⁹ Int J Oncol 2003 Feb;22(2):399-407

For every year of cellphone use the risk of acoustic neuroma increased by between 5% and 29% per year, depending on the phone technology used (which, as was discussed in the Interpretive Booby Traps Section, is relevant to use time, rather than to inherent technology differences). Table 5 summarized the results.

Table 5
Risk of Acoustic Neuroma on Same Side of Head as Cellphone Use and Risk Change Per Year by Phone Technology

AN Odds Ratio	Calculated p-value	Comments
4.4	0.0000603	Analog phone
4.2	0.00291	Analog phone, AN on same side as phone use
1.4	0.203	Cordless phone
1.3	0.437	Cordless phone, AN on same side as phone use
1.4	0.203	Digital phone
1.5	0.286	Digital phone, AN on same side as phone use
AN Odds Ratio, Increase Per Year	Calculated p-value	Comments
1.29	0.000718	Analog phones
1.05	0.476	Digital phones
1.10	0.0934	Cordless phones

The results of this study are very similar to study 9, “Mobile Phone Use and the Risk of Acoustic Neuroma, November 2004.” Study 9 effectively replicates this study’s findings.

7. Vestibular schwannoma³⁰, tinnitus and cellular telephones³¹, March 2003

With previous studies now consistently pointing to a strong risk of acoustic neuromas linked to cellphone exposure, researchers in this study turned their focus to discerning if the incidence rate of acoustic neuromas exhibited a change from pre-cellphone years to cellphone use years. To study if such a change existed, the researchers of this study examined the yearly change in the incidence rate of acoustic neuroma for 2 time periods, 1960-1979 (before cellphones) and 1980-1998 (cellphone use begins in 1981).

In the first period there was a small annual *decrease* in incidence rate (-0.64% per year) of acoustic neuroma and in the second period there was a large annual *increase* in the incidence rate (+2.16% per year).

Also examined, for the 2 time periods, was the annual change in “benign”³² brain tumors (excluding acoustic neuromas) and malignant brain tumors. They found a shift in the annual change between the two time periods, with the shift in the *opposite direction*.

³⁰ Vestibular schwannoma is another name for acoustic neuroma.

³¹ Neuroepidemiology 2003 Mar-Apr;22(2):124-9

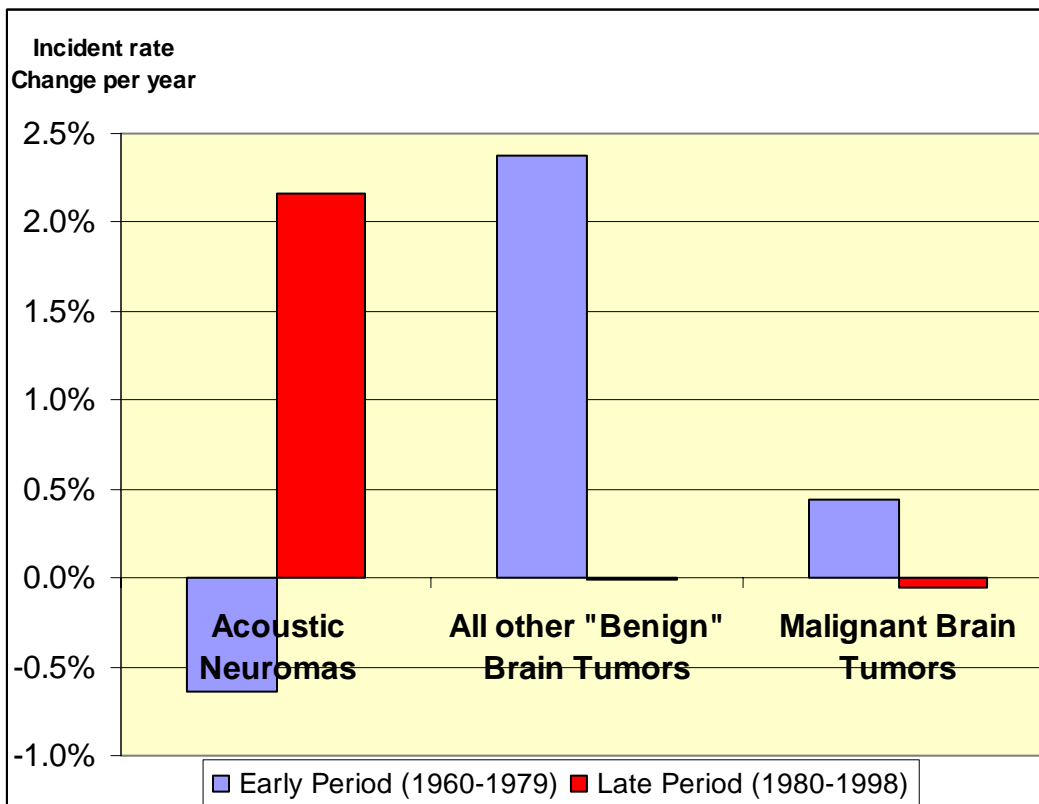
³² In the context of brain tumors, I use quotes around the term “benign” with irony as the dictionary defines benign as harmless.

Acoustic neuromas went from decreasing to increasing. "Benign" and malignant brain tumors went from increasing to decreasing.

In the case of "benign" brain tumors, the 1960-1979 incidence change per year was +2.38% per year and the 1980-1998 incidence drops sharply to nearly no annual change (-0.01% per year). The results for malignant brain tumors are similar but less pronounced; 1960-1979 it increases by +0.44% per year and 1980-1998 it has decreased to -0.06% per year.

Once again, the insights found in this study are intriguing. Cellphones did not exist in the early period but were introduced at the beginning (1981) of the late period when the striking rise in acoustic neuromas also began. Some argue that the change in the number of acoustic neuromas reflects, instead of the introduction of cellphones, the introduction of better diagnostic tools; specifically CT scans which began in 1974 and MRI that was first used in 1984. However, if this were true then logically we would also see a similar change (increased incident rate change) in "benign" and malignant brain tumors, but instead we see a decrease. Although not definitive, this study adds to the "weight" of the evidence that there is indeed an adverse connection between cellphones and harm. Figure 3 shows the change per year for the 3 tumor types.

Figure 3
Annual Changes in Brain Tumor Incidence for Early and Late Periods



This study also analyzed the risk of acoustic neuromas from cellphone use by phone technology for various lengths of time. Again, we find a large increased risk of acoustic neuromas. Again, we see the number of cases is highest for analog phones, less for cordless phones, and lowest for digital phones, consistent to the first Section’s discussion on Technology Differences. Table 6 summarizes the results of this study.

Table 6
Risk of Acoustic Neuroma for Years of Cellphone Use and Phone Technology

AN Odds Ratio	Calculated p-value	Cases	Time from First Cellphone Use	Comments
3.45	0.000219	38	>1 year	Analog phones; median latency, 8 years
3.71	0.00170	26	>5 years	
3.50	0.110	7	>10 years	
1.03	0.908	30	>1 year	Cordless phones, median latency 4 years; risk increases with increasing time of exposure
1.83	0.224	11	>5 years	
2.00	0.564	2	>10 years	
1.21	0.530	23	>1 year	Digital phones; median latency, 3 years
2.00	0.564	2	>5 years	
-	-	0	>10 years	

8. Christensen et al., Cellular Telephone Use and Risk of Acoustic Neuroma³³, February 2004

This study does not show a risk of acoustic neuroma for longer durations of cellphone use. Perhaps with only 2 cases using a cellphone for >10 years, the lack of an increased risk for acoustic neuroma is the result of too little data?

Further, not one of the reported results is “statistically significant.” Though there is a small, but statistically meaningless, increased risk of acoustic neuromas (given a p-value=0.7) with digital phone use. There were a total of 45 cases and 97 controls. Table7 summarized the results.

Table 7, Risk of Acoustic Neuroma by Duration of Cellphone Use

AN Odds Ratio	Confidence Level	Duration of Use	Comments
0.90	0.708	“Regularly”	45 cases
0.86	0.638	1-4 years	23 cases
0.68	0.305	≥5 years	19 cases
0.86	0.706	5-9 years	17 cases
0.22	0.0684	≥10 years	2 cases
1.58	0.520	Not reported	Phone type not known, 5 cases
0.26	0.0213	Not reported	Analog cellphone, 4 cases

³³ Christensen et al. Cellular Telephone Use and Risk of Acoustic Neuroma, American Journal of Epidemiology; Vol. 159, No. 3 (2004).

1.11	0.733	Not reported	Digital cellphone, 36 cases
0.99	0.974	≤167.5 hours	25 cases
1.01	0.981	>167.5 to 654 hours	12 cases
0.66	0.392	>654 hours	8 cases
1.03	0.927	≥5 years but ≤81.7 hours	10 cases
0.73	0.510	≥5 years and ≥81.7 hours	9 cases

It is interesting to consider again that only 2 cases (1.9% of the total cases) in this study used a cellphone for 10 years or more as we examine study 9 that shows a risk of acoustic neuroma with over 10 years of exposure.

9. Mobile Phone Use and the Risk of Acoustic Neuroma³⁴, November 2004

In many ways this study can be seen as the tipping point between *there may be a risk* to *there is a risk*. First it replicates Study 6. Of far more import, given this replication, is the criticism lobbed by Study 9’s team leader, Anders Ahlbom, in regards to Study 6.

In an Stockholm newspaper editorial ‘EXPERTS WHO TALK RUBBISH,’³⁵ Ahlbom railed about the, “Deliberate exaggeration of the importance of the results obtained...[:] no greater risk had been found on the whole, but according to the scientist, the location of these tumours had shifted to the side of the skull where the telephone is applied. This kind of interpretation seems bizarre in biological terms and is probably based on chance findings.” One can only wonder if Ahlbom would have wished to withdraw his vitriolic attack once his team reported the same results (see Comments, Table 8).

The study, *partially funded by the cellphone industry*, diagnosed acoustic neuromas from 1999-2002. It has the longest time horizon yet. Because digital cellphones were not introduced in Sweden until 1991, it is the first study to be able to see a 10-year horizon clearly. Yet it reports, “Slightly less than 6% of the population used mobile phones in 1990, 23% in 1995, and over 80% today.” So, at best, for a 10-year horizon, there were only 6% of Swedes using a cellphone. Nevertheless, its findings are alarming.

Unlike Study 4 above, Study 9 did not require histologically confirmed diagnoses and, it also allowed radiological diagnoses (MRI or CAT scan). This made a big difference in the numbers: thirty-nine percent of the cases were diagnoses by histology, the remainder by radiological techniques.

The findings were as follows: “...[no indication of] an increased risk of acoustic neuroma related to short-term mobile phone use after a short latency period. However, our data **suggest an increased risk of acoustic neuroma associated with mobile phone use of at least 10 years’ duration.**” [bold emphasis is ours]

Yet an examination of the data (Table 8, below) indicates that even for cellphone use of *less* than 10 years, there is a possible increased risk of acoustic neuromas, albeit not a “statistically significant” risk.

³⁴ Epidemiology 2004;15: 653–659

³⁵ Svenska Dagbladet/Brännpunkt, Sweden’s leading daily newspaper, on Monday, 3 September 2001

Table 8, Cellphone Use and Risk of Acoustic Neuroma by Duration of Use

AN Odds Ratio	Confidence	Years of Use	Comments
3.9	0.00224	≥10 years since first regular use	Tumor on same side as phone use Odds ratios increase as the years of use increase. No trend was reported
1.4	0.305	5-9 years since first regular use	
0.8	0.386	<5 years since first regular use	
1.9	0.0904	≥10 years since first regular use	Without regard to which side of the head the phone was on.
1.1	0.729	5-9 years since first regular use	Odds ratios increase as the years of use increase. No trend was reported
0.8	0.350	<5 years since first regular use	
AN Odds Ratio	Confidence	Cum No. of Calls	Comments
1.2	0.524	≥7350 calls	Odds ratios increase as the number of calls increase. No trend was reported.
0.9	0.646	625-7349 calls	
0.8	0.476	<625 calls	
AN Odds Ratio	Confidence	Cum Hrs of Use	Comments
1.1	0.739	≥450 hours	Odds ratios increase, then flat as cumulative hour of use increase.
1.1	0.667	30-449 hours	
0.7	0.194	<30 hours	

When they examine the risk of acoustic neuromas by phone technology we see an apparent higher risk for analog phones used for 10 years or more, but roughly the same risk for analog or digital phones for 5-9 years of ≥5 years of use. Table 9 summarized these results.

Table 9, Cellphones Technology and Risk of Acoustic Neuroma

AN Odds Ratio	Confidence	Years of Use	Comments, Types of Phones
1.8	0.162	≥10 years since first “regular use”	Analog phones (insufficient data for digital phones)
1.3	0.505	5-9 years since first “regular use”	Analog phones
1.2	0.507	≥5 years since first “regular use”	Digital phones

The consistent theme that Study 9 illustrates is that the higher the duration of use, the more the risk. This is true, not only for years of use, but also for number of calls, or number of hours. However, the authors of Study 9 contradict their own findings by reporting in their summary that they, “found no association between acoustic neuroma and amount of use measured as cumulative number of hours or total number of calls.” Perhaps this is because the trend did not meet the threshold of 95% confidence, but their reasons for their contradictory conclusion is never explained.

10. Hardell and Hansson-Mild, Mobile and cordless telephones and the association with brain tumours in different age groups³⁶

This study shows that age at exposure matters and for the young, it matters a lot.

The stunning data found in Study 10, has had the effect of breaking loose, if ever so slightly, the many layers of denial surrounding the harmful health effects of cellphone exposure. For the first time, not only have many in the scientific community started expressing alarm, but as well, a number of public media outlets as disparate as Public Radio (November 2004) and O Magazine (April 2005) have changed their “all good” attitude towards cellphones to a “wait and see”.

Another first for Study 10 is that it links the risk of harm to the age of the cellphone user. As discussed in the first Section on the interpretive trap of age, when reading cellphone studies the presence of risk differences in age groups is critical to an understanding of the degree of risk, if cellphones are shown to be tumorigenic.

Looking at all forms of brain tumors, they found an 8-fold risk of brain tumors in the youngest, 20-29 year old age group. Further, this is very close to 95% confidence. When all ages are combined there is a much smaller risk (1.31 fold), implying that the youngest carry by far the largest proportion of the risk. For older age groups, only 50-59 years old showed a near statistically significant risk of 1.56.

Of particular import is to note that cordless phones (very similar to digital cellphones, but in use for a longer period) also shows a dramatically higher risk for the youngest age group.

Table 10, illustrates the major findings of this study.

³⁶ Hardell and Hansson-Mild, in-press, Arch Environ Health

Table 10
Risk of Brain Tumor from Cellphone Exposure
20-29 Age Group versus All ages from 5 years of cellphone use

Age Group	Risk of BT ³⁷ Odds Ratio	Calculated p-value	Phone Technology	Cellphone Use (years)
20-29	8.17	0.0517	Analog	5
All ages	1.31	0.0282		
20-29	4.30	0.0201	Cordless	
All ages	1.40	0.0113		
20-29	0.84 ³⁸	0.907	Digital	
All ages	1.11	0.571		

As you can see, the 20-29 year age group is at greatest risk, except for digital phones where there is insufficient data to draw any conclusion

The importance of Study 10, thus, is that not only is there an indisputable connection of harm to cellphone exposure, but that this harm creates a stronger risk for younger brains, perhaps, as posited before, because they are still forming.

Study 10 is important to view within the context of the new cellphone industry push to market cellphones to children. The CBS news report³⁹ of this industry’s campaign, mentioned at the top of this discussion, is quite sobering when the actual statistics are absorbed: “ in 2003 one third of US kids aged 11-17 had their own cellphones; by the end of 2003, estimates grew to 40%. [By] 2004 nearly half of the kids in this age group had cellphones.” This same show went on to report/advertise-for-free that several toy makers were beginning their marketing of specialized cellphones for kids 8-12. Imagine what is in store for us when the cellphone studies include exposure risks to these tender ages some many years hence.

Sensible Recommendations

As we can see from the proceeding discussion, when the scientific literature regarding human exposure to cellphones is interpreted within its own context, which also includes interpretive booby traps to be avoided, we have the makings of a very serious health epidemic on our hands.

So what is to be done? Outright banning of cellphones at this juncture seems both unwarranted and too extreme. However, a growing, worldwide community of concerned scientists and health advocates are beginning to call for the application of caution in the use of cellphones. This caution is recommended in the form of invoking the “Precautionary Principle”: the implementation of low-cost mitigations—e.g., akin to the health warning labels that must now be added to all tobacco advertising or packaging. As

³⁷ BT: Brain tumor

³⁸ Results based on one case and one control.

³⁹ Good Morning America, Cellphones for Kids, April 1, 2005

of 2003, the following groupings have now invoked the Precautionary Principle with respect to the use of mobile phones and the siting of mobile phone antennas:⁴⁰

- Government of Italy (1998)
- Advisory Board on Non-Ionizing Radiation to the Czech National Institute of Public Health (1999)
- Government of Switzerland (1999-2000)
- U.K. Independent Expert Group on Mobile Phones, also known as the Stewart Committee (2000)
- Advisory Committee to the Director-General of Health of France, also known as Zmirou Committee (2001)
- British Medical Association (2001)
- German Academy of Pediatrics (2001)
- German Radiation Protection Commission (2001)
- Advisory Panel to the Spanish Ministry of Health (2001)
- German Federal Office for Radiation Protection (2002)
- Russian National Committee on Non-Ionizing Radiation Protection (2002)
- City of Paris (2003)
- World Health Organization (WHO) International EMF Project (2003)

Below are some simple, specific, and sensible recommendations to go along with the invocation of the Precautionary Principle:

1. New cellphone models should only be manufactured to include a headset and without a speaker for the ear. The cost of this would be close to zero, as it requires adding one item and deleting another.
2. Children should be banned from using a cellphone. In the UK, this is already a formal recommendation⁴¹ as it is for the Russian Academy of Sciences.⁴²
3. Cellphone owners need to be educated to only carry the cellphone in the “off” position if they want to minimize their exposures. When turned on they can see who has called, and return the calls.
4. Cellphone owners with older models need to be educated to only use their cellphone with a headset.

In giving serious consideration to these low-cost responses to the potential harm from cellphone use, it is important to remember two things. First, many parallel recommendations have been implemented for tobacco use, despite the fact that most of the scientific studies showing risk of tobacco exposure never reached the high standards now imposed for cellphone exposure risk—and tobacco becomes carcinogenic for probably no more than 10%-15% of the population.

⁴⁰ Microwave News, May/June 2003, p. 10.

⁴¹ Associated Press report at <http://www.forbes.com/home/feeds/ap/2005/01/11/ap1752648.html>

⁴² Vladimir Binhi, Russian Academy of Sciences

Finally, it is also vital to contemplate that while we are busy enforcing an impossibly high risk-of-harm bar, we are all engaged, with our now near-universal cellphone use, in the world's largest human health experiment—without the necessary informed consent.

All rights reserved per Sections 107 and 108 of the United States Copyright Act. Permission is required to reproduce any portion or the entirety of this publication in any form or by any means.

Requests: Lloyd Morgan (bilovsky@aol.com)