Cellphones and Human Health

A Review of the Scientific Literature and Commentary By Lloyd Morgan and Diana Bilovsky

Introduction

It is our contention, *based solely on our 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. (One of 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 central 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 preexisting interpretive "booby traps" unique to studies involving brain tumors, and indeed unique to studies of cellphone use, especially at this stage of cellphone 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 <u>rarity</u> of brain tumors. In the broad brush, if there are too few brain tumor 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"¹ 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.² Acoustic neuromas, the most common brain tumor associated with

¹ In the context of brain tumors, I use quotes around the term "benign" with irony as the dictionary defines benign as harmless.

² 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 (<u>www.cbtrus.org</u>), 2004.

cellphone use is even more rare. The US incidence rate of acoustic neuromas is 54% of all nerve sheath tumors.³ The incidence rate for acoustic neuromas is a mere 6 per million people per year.⁴

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. Yet Study 8 summarized its findings by reporting, "The results of this . . . study . . . do not support an association between cell phone use and risk of acoustic neuroma."

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 casecontrolled 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, it is likely that there will be scarcely enough cases to determine if there is a risk or not. As an example of this difficulty in determining risk from a cohort study, it is important to examine the Danish cohort study, discussed later as Study 3. In this study the size of the cohort (Danish cellphone subscribers) is 420,095. However, 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. Yet it provides a good example of the irrelevance of the results derived from a cohort study approaching a half million people.

³ 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.

⁴ 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.

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 is a risk above a defined level.⁵ 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. For all brain tumors (malignant and "benign" combined) the latency time ranges from 20 to 40 years.⁶ For the most common "benign" brain tumor, meningioma, the latency time is thought to be between 30 and 40 years.^{7,8}

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 cellphones, 5 years for cordless phones, and 3 years for digital cellphones;⁹ another study reports a latency time for acoustic neuromas from cellphone exposure of 10-years or greater.¹⁰ If we believe that average latency times are on the order of several decades, then we can assume that

⁵ "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 or higher risk, if there is a risk."

⁶ "Brain cancer is often described as having a lengthy latency or development period, sometimes in excess of 20 years." Massachusetts Department Of Public Health, Bureau of Environmental Health Assessment, Community Assessment Program; Assessment of Brain and Central Nervous System Cancer Incidence in Needham, MA 1987-1998; December 2002

⁷ Sadetzki S, Flint-Richter P, Ben-Tal T, Nass D: Radiation-induced meningioma: a descriptive study of 253 cases. J Neurosurg 97: 1078-1082, 2002

⁸ Shintani T, Hayakawa N, Hoshi M, Sumida M, Kurisu K, Oki S, Kodama Y, Kajikawa H, Inai K, Kamada N: High incidence of meningioma among Hiroshima atomic bomb survivors. J Radiat Res (Tokyo) 40: 49-57, 1999

⁹ European Journal of Cancer Prevention 2002, 11, 1–10

¹⁰ Epidemiology 2004;15: 653–659)

these initial findings of tumors that reflect only about 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 increased risk of a brain tumor is much higher in younger, than in older people after exposure to a carcinogen. 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 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 risk of developing cancer for the survivors of the 1945 atomic bombs compared to people not exposed to the bomb.¹¹ In this example the relative risk is the increased risk cause by the ionizing radiation from the atomic bomb. Thus a relative risk of 1.0 means that the cancer rate would be the same if there were no exposure to the atomic bomb. The graph clearly shows that <u>age at exposure is a strong function of the relative excess rate of solid cancers</u> for ionizing radiation.

While an argument may be made that, lacking parallel studies linking exposure to nonionizing cellphone radiation with higher carcinogenic risk for younger groups (as we have with ionizing radiation), our fears for risk-association with youth are groundless. However, Hardell et al. (Study 10) does just this: provides us with the first study linking younger age at exposure to increased brain tumor risk from non-ionizing exposures. Its findings show far higher risk for the youngest age group exposed to non-ionizing radiation than for older age groups, the same as we have come to expect with ionizing radiation exposure.

Because the rate of cellular growth is so much higher in children than in younger adult groups any indication of increased risk for younger adult 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.¹²

¹¹ Preston et al., Studies of Mortality of Atomic Bomb Survivors. Report 13: Solid Cancer and Noncancer Disease Mortality: 1950–1997; RADIATION RESEARCH 160, 381–407 (2003), Adapted from FIG. 1. Excess cancer rates by calendar periods and age-at-exposure categories: absolute and relative to background rates.

¹² Associated Press report at <u>http://www.forbes.com/home/feeds/ap/2005/01/11/ap1752648.html</u>

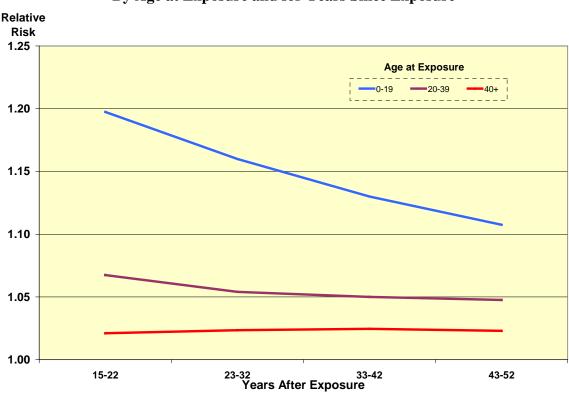


Figure 1 Relative Risk of Solid Cancers from Atomic Bomb Exposure By Age at Exposure and for Years Since Exposure

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 emphasize 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 digital 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 only on differing engineering terminology—concepts that have not been shown to have a differing biological basis. These engineering differences between analog and digital cellphones or cordless phones are changes to modulation techniques (how information is created by changing the 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 factors in any study concerning exposure to agents that may cause 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 or more, that similar factors of risk will not be seen.

A few more notes concerning the length of use time and cellphone type: Cordless phone conversations usually last for much longer than do cellphone conversations. Partially this is a result of the cost of cellphone use time and partially this is the result of the quality of the connection. And, as far as the use time for cellphones, we must consider the fact that parents often provide young children with cordless phones even when they do not allow these same children to have cellphones. This may be because while there has been a general public perception that there might be a problem with cellphones, cordless phones have heretofore been considered safe.

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.

The power absorbed by a cellphone user's brain is considered the "dose" and is measured using a factor called SAR, or Specific Absorption Rate. The units for SAR are watts per kilogram.¹³ In other words, the power absorbed per weight of brain tissue. We might think that cellphones radiating the same power (Watts) would have similar SAR values. This is not the case. SAR values were measured for 16 different digital cellphones, each radiating the same power. The SAR values ranged from 0.28 W/kg to 1.33 W/kg, a factor of 4.75.¹⁴ We might think that for a given phone's SAR value that the specific SAR we receive would be the same. But this is not the case either. The way someone holds the cellphone is also important: "... when the phone is slightly tilted towards the head . . . the value can go from 0.2 to 3.5 W/kg"¹⁵—a factor of 17.5. Therefore, to imbue undo significance to the amount of power absorbed by our brain (AKA SAR) when using a cellphone is missing the bigger picture. SAR varies to such an extent that the specific power that a phone radiates becomes a secondary if not tertiary factor. In fact, when examining the aggregate power differences between the analog and digital cellphone and the different ways of holding the cellphone, the only conclusion that can be drawn is that of "a rather large uncertainty in the actual SAR determination for a specific situation, with a factor of 100 from the nearness to the base station and at least a factor of 10-50 depending on make and model and personal style of use."¹⁶

7. Harm Standards: Do They Make Sense?^{17,18}

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

¹³ Interestingly, the ionizing radiation dose is SA, or Specific Absorption. SAR is just the SA received per

second (the Rate). ¹⁴ Kuster, N. (1997), Swiss Tests Show Wide Variation in Radiation Exposures from Cell Phones; Microwave News, November/December 1997

¹⁵ Hansson-Mild et al, "Exposure" and "Dose" in Mobile Phone Health Studies; Mobile Communication and Health, Medical, Biological and Social Problems, Moscow, Sept. 2004.

¹⁶ Ibid

¹⁷This discussion is based in part on a presentation by Cindy Sage during the Bioelectromagnetics Society's meeting, Washington DC, June 2004.

¹⁸ 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.

finding of risk happens by chance; or, conversely, that there is a 95% chance of the finding of risk 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?

American Legal System

For almost all civil cases, the standard required for evidentiary findings of proof in the legal system is <u>preponderance of the evidence</u>; in other words, is the "existence of a particular fact more probable than its nonexistence".¹⁹ 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: <u>clear and convincing proof</u> (which asks for a "substantially greater degree of belief concerning the existence of a fact);²⁰ and for criminal cases, <u>beyond a reasonable doubt</u> (which calls for proof that is "inconsistent with any other rational conclusions").^{21, 22} 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

¹⁹ West's California Evidence Code, p. 72.

²⁰ Ibid.

²¹ Black's Law Dictionary, p. 1381.

²² 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".

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*".²³ 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.

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, on the next page, 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.).

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 higher 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

²³ West's California Evidence Code, p. 73.

exposure ranging from a factor of 3.9 to 8.19, the connection between harm and cellphone use would also be accepted knowledge. Amazingly, these risk factor 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, where cellphones are concerned, the demand for "scientific certainty" 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".²⁴

| 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) | |
| | | 4.2* | Median use 7 yrs, tumor on same side of head as phone use, analog cellphone | Hardell et al. (2003) | |
| Brain | Wireless | 3.9* | ≥10 years use, tumor on same side of head as phone use, analog cellphone | Lonn et al. (2004) | |
| | phones | 8.19 | 20-29 year old group, >5 years use, analog cellphone | Hardell & Hansson-Mild | |
| | | 4.30* | 20-29 year old group, >5 years use, cordless phone | (2004) ¹ | |
| 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* 1.38* | U.S. nonsmokers U.S. nonsmokers, highest exposed groups | EPA (1992b) | |
| | | 1.20 | U.S. nonsmoking women, highest exposure | Alavanja et al. (1994) | |
| Lung | Home radon ⁺ | 1.20 1.80* | Sweden, non-smokers, highest exposure Sweden, all subjects, highest exposure | Pershagen et al. (1994) | |

Figure 2

* 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 Presentated 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

²⁴ Paraphrase from testimony given to the California Public Utilities Commission by Dennis Zell, April 4, 2005, reported by Cindy Sage.

8. "No Known Mechanism" Argument

Perhaps the silliest "booby trap" is the argument that the data must be wrong because "no known mechanism" exists to explain the results of the data. Stefan Lönn, the lead author of Study 9, is one of many who have expressed this sentiment. In reference to Study 9 he recently wrote, "... despite extensive experimental research, there is still no known mechanism than can explain how cell phone use might lead to an increased risk of cancer."²⁵

Such an argument seems ever so reasonable. Without a known mechanism, how can a finding be true? Yet, such an argument turns science on its head. The essence of science is to examine experimental data. Yes, the data may have inaccuracies, and, yes experiments need replication to provide confidence that some factor in a given experiment may be unknowingly distorting the data, but given accurate and replicated data, science has never required a known mechanism to validate the data.

Such an argument is tantamount to denying that science is a valid process. It is equivalent to saying, when the known mechanism for the cosmos was Newtonian mechanics, that the precession of the orbit of Mercury around the sun "cannot be". As we all know, a mechanism was eventually found: Einstein's theory of relativity, which did provide a known mechanism for the long known *scientific truth* that Mercury's orbital precession around the sun existed, albeit for some time without a known mechanism.

9. The Role of "Industry"²⁶

The final and most pervasive interpretive trap is, in elephant-sitting-in-the-middle-of-theroom 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 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 the ever-increasing revenues of a \$100 billion industry.²⁷

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

²⁵ National Brain Tumor Foundation, Search, Spring 2005, Issue No. 63

²⁶ 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.

²⁷ The Wall Street Journal, Cellphone Makers Say Radio Waves Are Found to Be Save, April 27, 2005, page D4.

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 through funding their own studies, hiring key researchers from Universities and government, sponsoring scientific meetings 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 (e.g., CBS is owned by Viacom and cellphone advertising is a major source of media revenue).

Science

Not only does the cellphone industry control the research by being a major source of funds 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 the world attending 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 instead is comprised of a large contingent of cellphone industry employees or consultants including representatives from Motorola, Nokia, Vodafone and the industry's trade association, the Cellular Telephone Industry Association (CTIA). 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 newsletters and public journalism—is now so controlled by the phone industry that it is very difficult to find a way to disseminate any other information about cellphone exposure findings—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 electromagnetic 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".²⁸

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 electromagnetic fields and cellphones. These studies range from cellular studies using various cell types from humans and other animals (in-vitro studies) to live (in-vivo) animal studies—typically rodents but other species, as well, including fruit flies. 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 large human populations 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 exposure is that of acoustic neuromas (tumors on the nerve from the ear to the brain), all but one of the ten reports highlighted, below, will be reports that link acoustic neuromas with cellphone exposure. The one exception will be the Hardell & Hansson-Mild study (Study 10) that looks at aggregate 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. <u>Board Presentation:</u> 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,²⁹ 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."³⁰

²⁸ Elder's EPA report concluded: "Biological effects occur at an SAR of about 1 W/kg; some of them may be significant under certain environmental conditions."

²⁹ Dr. George Carlo, Chief Scientist, Wireless Technology Research (established by CTIA) oversaw multiple "scientific" studies funded by the CTIA.

³⁰ Page 205, Cell Phones, Invisible Hazards in the Wireless Age by Dr. George Carlo and Martin Schram, Carroll & Graf Publishers, Inc., 2001.

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.³¹ The impetus for this study was a January 21, 1993 Larry King TV 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 a p-value³², the chance of no risk, \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 used).³³ 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 32). 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:

³¹ Inskip PD, et al.; Cellular-Telephone Use and Brain Tumors. N Engl J Med 344(2):79-86, 2001.

 $^{^{32}}$ p-value is a common term used by statisticians. It refers to the probability of risk is due to chance. p-values, in Table 1 and in all the following reports of p-values, are calculated from the published 95% confidence intervals. Such calculations are subject to rounding errors. 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: Calculated p-values are presented 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.

³³ In June 1994 there were 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.

| AN ³⁴ Odds Ratio ³⁵ | Calculated p-value | 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 |

Table 1, Risk of Acoustic Neuroma from Cellphone Use

3. Cellular Telephones and Cancer—a Nationwide Cohort Study in Denmark, Feb 2001³⁶

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 nerve sheath tumors may include acoustic neuromas. 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.

| Percent of Cellphone User in 3-Year Segments | | | | | | | |
|--|------------|-----------|----------------|--|--|--|--|
| Cellphone Users | % Of Total | Years | Latency | | | | |
| 3,819 | 0.9 | 1982-1984 | 11 to 13 years | | | | |
| 7,866 | 1.9 | 1985-1987 | 8 to 10 years | | | | |
| 21,292 | 5.1 | 1988-1990 | 5 to 8 years | | | | |
| 96,959 | 23.1 | 1991-1993 | 2 to 4 years | | | | |
| 290,159 | 69.1 | 1994-1995 | 0 to 1 year | | | | |

 Table 2

 Percent of Collabora User in 3 Year Segments

³⁴ AN: Acoustic neuroma

³⁵ 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.

³⁶ Johansen et al., Journal of the National Cancer Institute, Vol. 93, No. 3, February 7, 2001

The average use of analog cellphones in this study 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. 2002^{37}

This study also reported no risk of acoustic neuromas.

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. 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).

| I able 5, R | Table 5, Kisk of Acoustic Neuronia for Greater Than 5 Years of Cemptone Use | | | | | |
|------------------|---|--------------------|---------------|--|--|--|
| AN Odds Ratio | Calculated p-value | Number of Patients | Exposure Time | | | |
| 1.7 | 0.361 | 12 | > 3 years | | | |

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

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 \geq 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 mention of a non-significant 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.

³⁷ Muscat et al., Neurology 2002;58:1304-1306

George Carlo, reported that, "Muscat's analysis concluded that the increased risk was statistically significant after patients had used cell phones for six years."³⁸

5. <u>Cellular and cordless telephones and the risk for brain tumours³⁹</u>, 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 Örebro University and the National Institute for Working Life (equivalent to the US OSHA) 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 neuroma cases were histologically confirmed. If there was only an MRI diagnoses of an acoustic neuroma the case was excluded. As a result, it is likely that the histological confirmation in this study severely underestimated the risk of acoustic neuromas because 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.

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

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

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.

³⁸ Dr. George Carlo and Martin Schram, Cell Phones, Invisible Hazards in the Wireless Age, p170; Carrol & Graf Publishers, Inc. 2001.

³⁹ European Journal of Cancer Prevention: Volume 11(4) August 2002 pp 377-386

Note also 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 when there is a lack of confidence it is often the result of too few cases.

6. <u>Further aspects on cellular and cordless telephones and brain tumours</u>,⁴⁰ 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 improved. 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. To wit: 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 Vear 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 | Calculated p-value | Comments | |
| Ratio, Increase | | | |
| Per Year | | | |
| 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.

⁴⁰ Int J Oncol 2003 Feb;22(2):399-407

7. <u>Vestibular schwannoma⁴¹</u>, tinnitus and cellular telephones⁴², 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 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 the incidence rate of "benign" 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*. 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 rate change was +2.38% per year, and the 1980-1998 incidence dropped sharply to nearly no annual change (-0.01% per year). The results for malignant brain tumors were similar but less pronounced: between 1960-1979 malignant brain tumors incidence rate increased by +0.44% per year; by 1980-1998 it had 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 scans, 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 the opposite: 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, below shows the changes per year for the 3 tumor types.

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.

⁴¹ Vestibular schwannoma is another name for acoustic neuroma.

⁴² Neuroepidemiology 2003 Mar-Apr;22(2):124-9

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

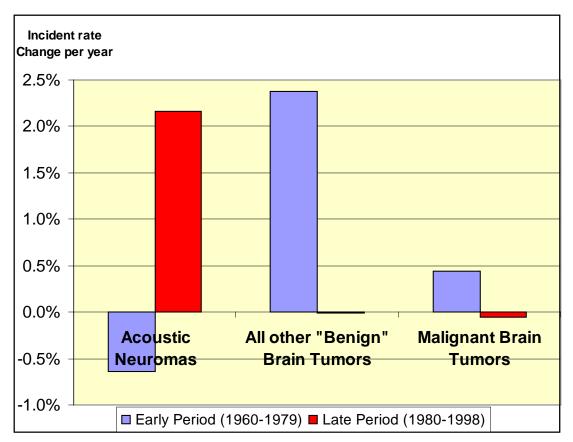


Table 6

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

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

8. <u>Christensen et al., Cellular Telephone Use and Risk of Acoustic Neuroma⁴³</u>, 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", making any determination of risk or lack of risk nearly meaningless. Yet the study summarizes its findings, "The results of this . . . study . . . do not support an association between cell phone use and risk of acoustic neuroma."

Once again, we see that the reason for "not statistically significant" results is the lack of an inadequate number of cases: a total of 45 cases, and, when examined for cellphone use of greater than a year, only 23 cases! Table7 summarized the results.

| AN Odds | Calculated | Duration of Use | Comments |
|---------|------------|--------------------------------------|-------------------------------|
| Ratio | p-value | | |
| 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 |
| 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 | \geq 5 years but \leq 81.7 hours | 10 cases |
| 0.73 | 0.510 | \geq 5 years and \geq 81.7 hours | 9 cases |

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

It is interesting to consider again that only 2 cases in this study used a cellphone for 10 years or more as we juxtapose it with the next report (Study 9) that <u>found</u> an acoustic neuroma risk with 12 cases of over 10 years of cellphone exposure.

9. <u>Mobile Phone Use and the Risk of Acoustic Neuroma⁴⁴</u>, 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*. In part because it replicates Study 6 and in part because its findings of risk for greater than 10 years of cellphone use are so strong.

⁴³ Christensen et al. Cellular Telephone Use and Risk of Acoustic Neuroma, American Journal of Epidemiology; Vol. 159, No. 3 (2004).

⁴⁴ Epidemiology 2004;15: 653–659

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" that is, 10 years previously. 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. When the risk of acoustic neuromas is examined 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 \geq 5 years of use. Table 9, below, summarized these results.

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. As the number of hours increases, the odds ratio increases, and then goes flat. However, the authors of Study 9 contradict their own findings by reporting in their summary that the study, "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.

| AN Odds | Calculated | Cases | Years of Use | Comments |
|---------|------------|-------|-----------------------|--------------------------------|
| Ratio | p-value | | | |
| 3.9 | 0.00224 | 12 | \geq 10 years since | |
| | | | first regular use | Tumor on same side as phone |
| 1.4 | 0.305 | 17 | 5-9 years since | use |
| | | | first regular use | Odds ratios increase as the |
| 0.8 | 0.386 | 22 | <5 years since | years of use increase. No |
| | | | first regular use | trend was reported |
| 1.9 | 0.0904 | 14 | >10 years since | Without regard to which side |
| | | | first regular use | of the head the phone was on. |
| 1.1 | 0.729 | 30 | 5-9 years since | |
| | | | first regular use | Odds ratios increase as the |
| 0.8 | 0.350 | 44 | <5 years since | years of use increase. No |
| | | | first regular use | trend was reported |
| AN Odds | Calculated | Cases | Cum No. of | Comments |
| Ratio | p-value | | Calls | |
| 1.2 | 0.524 | 28 | ≥7350 calls | Odds ratios increase as the |
| 0.9 | 0.646 | 38 | 625-7349 calls | number of calls increase. No |
| 0.8 | 0.476 | 18 | <625 calls | trend was reported. |
| AN Odds | Calculated | Cases | Cum Hrs of | Comments |
| Ratio | p-value | | Use | |
| 1.1 | 0.739 | 21 | <u>></u> 450 hours | Odds ratios increase, and then |
| 1.1 | 0.667 | 44 | 30-449 hours | are flat, as cumulative hours |
| 0.7 | 0.194 | 17 | <30 hours | of use increase. |

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

Table 9, Cellphones Technology and Risk of Acoustic Neuroma

| AN Odds | Calculated | Cases | Years of Use | Comments, Types of |
|---------|------------|-------|-----------------------|-----------------------------|
| Ratio | p-value | | | Phones |
| 1.8 | 0.162 | 14 | \geq 10 years since | Analog phones (insufficient |
| | | | first "regular | data for digital phones) |
| | | | use" | |
| 1.3 | 0.505 | 13 | 5-9 years since | Analog phones |
| | | | first "regular | |
| | | | use" | |
| 1.2 | 0.507 | 29 | \geq 5 years since | Digital phones |
| | | | first "regular | |
| | | | use | |

10. Hardell and Hansson-Mild, Mobile and cordless telephones and the association with brain tumours in different age groups 45^{45}

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 National Public Radio (November 2004) and O Magazine (April 2005) have changed their "all good" attitude towards cellphones to a "wait and see".

But the most significant import of 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 at exposure", if cellphones are shown to be tumorgenic, then when reading cellphone studies the presence of risk differences in age groups is critical to an understanding of the degree of risk.

Looking at all forms of brain tumors, Hardell et al. found an 8-fold risk of brain tumors in the youngest—the 20-29 year old age group. Further, this very alarming finding is quite 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 importance is to note that cordless phones (very similar to digital cellphones, but in use for a longer period) also show a dramatically higher risk for the youngest age group.

Table 10, illustrates the major findings of this study.

| 20-29 Age Group versus All ages from 5 years of cellphone use | | | | |
|---|---------------------------------|------------|------------|-------------|
| Age Group | Risk of BT ⁴⁶ | Calculated | Phone | Cellphone |
| | Odds Ratio | p-value | Technology | Use (years) |
| 20-29 | 8.17 | 0.0517 | Analog | |
| All ages | 1.31 | 0.0282 | | |
| 20-29 | 4.30 | 0.0201 | Cordless | 5 |
| All ages | 1.40 | 0.0113 | | |
| 20-29 | 0.84^{47} | 0.907 | Digital | |
| All ages | 1.11 | 0.571 | | |

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

⁴⁵ Hardell and Hansson-Mild, in-press, Arch Environ Health, see (<u>http://www.cost281.org/documents.php?node=77&dir_session</u>=) to view a PowerPoint presentation of this report.

⁴⁶ BT: Brain tumor

⁴⁷ Results based on one case and one control.

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 significance of Study 10, thus, is that not only is there a 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 report 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 8-12.⁴⁸ And, that's not all: New campaigns by various corporations are exploring an expansion of the cellphone market to preschoolers!⁴⁹ Imagine what is in store for us when the cellphone studies include exposure risks to these tender ages some many years hence.

Some 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 the 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 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:⁵⁰

- 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)

⁴⁸ Good Morning America, Cellphones for Kids, April 1, 2005

⁴⁹ "A Way to Calm a Fussy Baby: 'Sesame Street' by Cell Phone", The Wall Street Journal, April 18, 2005.

⁵⁰ Microwave News, May/June 2003, p. 10.

- 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) [Rescinded 3 months later after cellphone industry pressure was applied.]⁵¹
- City of San Francisco (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. Cellphone owners with models made prior to implementation of recommendation 1, need to be educated to only use their cellphone with a headset.
- 3. Children should be banned from using a cellphone. In the UK, this is already a formal recommendation⁵² as it is for the Russian Academy of Sciences.⁵³
- 4. All cellphone owners need to be educated to carry the cellphone in the "off" position to minimize their exposures. Thus functioning as an answering machine, the cellphone can be periodically turned on for accessing messages and returning calls.

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.

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—an experiment that has failed to provide us with the necessary informed consent.

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⁵¹ "WHO Flip-Flops on EMFs, Precautionary Principle Now Revoked", Microwave News, Vol. XXIII, No. 3, May/June 2003, page 1.

⁵² Associated Press report at <u>http://www.forbes.com/home/feeds/ap/2005/01/11/ap1752648.html</u>

⁵³ Vladimir Binhi, Russian Academy of Sciences