

**PRE-FILED TESTIMONY  
OF LENNART HARDELL, MD, PhD  
MPUC Docket No. 2011-00262**

1   **Q.    Please state your name and address.**

2   A.    Lennart Hardell, MD, PhD  
3         Professor  
4         Department of Oncology  
5         Orebro University Hospital  
6         S-70185 Orebro  
7         Sweden

8   **Q.    Briefly state your occupation, educational background and current employment.**

9   A.    I am a professor of oncology at Orebro University Hospital specializing in the  
10         epidemiological research studying cancer risks related to exposure to environmental  
11         toxins, including electromagnetic radiation. I have been a specialist in oncology since  
12         1979. My work as an oncologist has included medical treatment of cancer patients and  
13         radiotherapy. In parallel with that work I have performed epidemiological research  
14         mostly on risk factors for cancer. Attached as Exhibit A is my curriculum vitae (C.V.).

15   **Q.    Are you a member of any professional organizations? If so, please list.**

16   A.    This is listed in my C.V.

17   **Q.    Have you authored any papers or journal articles?**

18   A.    I have published more than 300 scientific articles in peer-reviewed scientific journals,  
19         chapters in books, and commentaries. In addition I have published 149 abstracts for  
20         scientific meetings and shorter communications. A full list is shown on Exhibit A.

21   **Q.    Briefly describe your work and experience related to the study of health risks**  
22         **related to electromagnetic fields and radiofrequency electromagnetic fields in the 30**  
23         **MHz to 300 GHz range (RF-EMF). Identify any studies or published writings on**  
24         **the subject.**

1 A. In 1995 we published our evaluation of cancer risks associated with exposure to  
2 extremely low frequency electromagnetic fields (ELF-EMF) in a peer-reviewed scientific  
3 journal as supplement (Hardell et al 1995).

4 After that I have participated in and been the lead investigator and author of a  
5 large number of scientific studies on use of mobile phones and cordless phones and the  
6 risk for certain malignant diseases (brain tumors, salivary gland tumors, testicular cancer,  
7 non-Hodgkin lymphoma, malignant melanoma). This has resulted in more than 80  
8 publications that are listed in Exhibit A. I have also on numerous occasions been invited  
9 to participate in scientific meetings to present the results from our studies in this area. I  
10 am regularly referee for journals on submitted scientific articles. I was invited to be part  
11 of the expert panel at IARC in May 2011 on the evaluation of scientific evidence of the  
12 carcinogenic effects of RF-EMF. Recently I was invited to submit an article on this topic  
13 to the publication Late Lessons from Early Warnings, volume II; European Environment  
14 Agency, Copenhagen, as well as the BioInitiative 2012 Report, and to different journals. I  
15 have also supervised several medical dissertations within epidemiology, mostly on risk  
16 factors for cancer.

17 **Q. Are you familiar with other peer-reviewed epidemiological studies addressing the**  
18 **risk of cancer, disease, or other adverse health effects resulting from the exposure to**  
19 **RF-EMF, meaning radio frequency radiation at levels below which thermal effects**  
20 **are known to occur?**

21 A. Yes. Since this is one of my specialties I have studied and scrutinized most of the peer-  
22 reviewed studies that have been published on the subject since the 1990's. It has also  
23 been part of being a scientific expert, etc see above.

1 **Q. Have you performed any meta-analyses of different studies on the subject? Briefly**  
2 **describe the work you have produced conducting meta-analyses and the**  
3 **conclusions.**

4 A. Yes, my co-workers and I have performed three meta-analyses of the risk for brain  
5 tumors associated with use of wireless phones.

6 The first one was published in 2007 in Occupational Environmental Medicine  
7 (Hardell et al 2007a). Of the 16 case-control studies that were published on this topic, 11  
8 gave results for 10 years use or more (latency period = time from first use of the mobile  
9 phone until the brain tumor was diagnosed). An association with acoustic neuroma was  
10 found in four studies in the group with at least 10 years use of a mobile phone. Six  
11 studies gave results for malignant brain tumors in that latency group. All gave increased  
12 odds ratios (OR), especially for ipsilateral exposure, that is the phone had been used on  
13 the same side of the brain as the tumor appeared. In the meta-analysis, ipsilateral cell  
14 phone use gave OR = 2.4, 95% confidence interval (CI) = 1.1 - 5.3, for acoustic neuroma  
15 and OR = 2.0, 95 % CI = 1.2 - 3.4, for glioma using a tumor latency period of 10 years  
16 and more.

17 The next overview including meta-analysis was published in Pathophysiology in  
18 2009. In summary the review yielded a consistent pattern of an increased risk for glioma  
19 and acoustic neuroma after >10 years mobile phone use, but not for meningioma. We  
20 concluded that current standards for exposure to microwaves during mobile phone use  
21 were not adequate for long-term exposure and needed to be revised (Hardell et al 2009).

22 Our most recent meta-analysis was published Dec 20, 2012 in Pathophysiology  
23 (Hardell et al 2012). We gave an overview of current epidemiological evidence for an

1 increased risk for brain tumors including a meta-analysis of the Hardell group and  
2 Interphone results for mobile phone use. These studies with results for longest duration of  
3 use served as important bases for IARC classification of RF-EMF as possible human  
4 carcinogen, see discussion below.

5 In contrast to the Hardell group studies, results for cordless phones are lacking in  
6 Interphone and the other studies in this area. Cordless phones emit similar RF-EMF  
7 emissions as mobile phones. Thus, including such use in the ‘unexposed’ group would  
8 obscure the possibility to identify an increased risk for cancer associated with use of  
9 wireless phones (mobile phones and cordless phones). It is like studying the risk for lung  
10 cancer among smokers and disregarding use of one type of cigarettes (e.g. one brand) and  
11 to include that group in the non-smoking group. Since also use of cordless phones emit  
12 RF-EMF emissions with increased risk for brain tumors in the Hardell group studies,  
13 excluding such use from the exposed groups (instead included in the ‘unexposed’ group)  
14 results in lower risk estimates and diminishes the possibility to find an increased risk.

15 The 2012 meta-analysis gave for glioma in the most exposed part of the brain, the  
16 temporal lobe, OR = 1.71, 95 % CI = 1.04-2.81 in the  $\geq 10$  years ( $>10$  years in the  
17 Hardell group) latency group. Ipsilateral mobile phone use  $\geq 1,640$  hours in total gave  
18 OR = 2.29, 95 % CI = 1.56-3.37. The results for meningioma were OR = 1.25, 95 % CI  
19 = 0.31-4.98 and OR = 1.35, 95 % CI = 0.81-2.23, respectively. Regarding acoustic  
20 neuroma ipsilateral mobile phone use in the latency group  $\geq 10$  years gave OR = 1.81, 95  
21 % CI = 0.73-4.45. For ipsilateral cumulative use  $\geq 1,640$  hours OR = 2.55, 95 % CI =  
22 1.50-4.40 was obtained. Also, use of cordless phones increased the risk for glioma and  
23 acoustic neuroma in the Hardell group studies. This meta-analysis confirmed previous

1 findings of an increased risk for glioma and acoustic neuroma associated with use of  
2 wireless phones (Hardell et al 2012).

3 In summary, there is sufficient evidence to conclude that RF-EMFs are bioactive  
4 and have a potential to cause health impacts. There is a consistent pattern of increased  
5 risk for glioma and acoustic neuroma associated with use of wireless phones (mobile  
6 phones and cordless phones). The current safety limits and reference levels are not  
7 adequate to protect public health. New public health standards and limits are needed.

8 **Q. What information is there about glioma patient survival and is that relevant to the**  
9 **causal association between glioma and cell phone RF-EMF exposure?**

10 A. A carcinogenic effect of RF-EMF emissions would be strengthened if exposure might  
11 negatively correlate with survival of glioma patients. The Hardell group analysed  
12 survival of glioma patients in the study period 1997-2003. Decreased survival of glioma  
13 cases (glioblastoma multiforme) with long-term and high cumulative use of wireless  
14 phones was found (Hardell, Carlberg 2012). Thus, this is an additional finding that adds  
15 to the biological relevance of a causation of glioma by exposure to RF-EMF. The results  
16 show an increased risk for glioma for wireless phone use but also worse outcome of the  
17 disease.

18 It should be added that a poorer survival among children with acute lymphoblastic  
19 leukaemia exposed to ELF-EMF has been reported in two studies (Foliart et al 2006,  
20 Svendsen et al 2007). These findings certainly strengthen a causal association between  
21 exposure to ELF-EMF and childhood leukaemia.

1 **Q. Experts testifying for CMP, Drs. Bailey and Shkolnikov, emphasized the importance**  
2 **of a Danish cohort study on mobile phone users and cancer risks. Are you familiar**  
3 **with that study?**

4 A. Yes.

5 **Q. Are the results in the Danish cohort study reliable and based on sound**  
6 **epidemiological methods?**

7 A. No. This was a record linkage study partly funded by two Danish telecom-operating  
8 companies, TeleDenmark Mobil and Sonofon, with no individual exposure data. It has  
9 produced four articles that we have made a thorough review of (Söderqvist et al 2012a).  
10 We concluded that its many limitations - embedded in the study design from the very  
11 beginning and mainly related to poor exposure assessment - cloud the findings of the four  
12 reports to such an extent that render them uninformative, at best. At worst, they may be  
13 used in a seemingly solid argument against an increased risk— and the reader may not  
14 understand that the Danish cohort study is a textbook example of a study design that  
15 precludes the possibility to find an increased risk, as we have discussed in our review.  
16 The term *business bias* may apply to the Danish cohort study, a terminology recently  
17 discussed by Levis et al (2012). There is always a potential of conflicts of interest when  
18 a study is sponsored by the industry (Hardell et al 2007b)

19 The Danish cohort study was included in the IARC evaluation of RF-EMF as  
20 human carcinogen, but the conclusion was that “phone provider, as a surrogate for mobile  
21 phone use, could have resulted in considerable misclassification in exposure assessment.”  
22 (Baan et al 2011). Thus, the Danish cohort study was considered to be uninformative as  
23 to cancer risks from mobile phone use.

1 **Q. Is there a potential for adverse health effects from RF-EMF radiation from mobile**  
2 **phone base stations?**

3 A. Yes. By searching PubMed, we identified a total of 10 epidemiological studies that  
4 assessed for putative health effects of mobile phone base stations. Seven of these studies  
5 explored the association between base station proximity and neurobehavioral effects and  
6 three investigated cancer. We found that eight of the 10 studies reported increased  
7 prevalence of adverse neurobehavioral symptoms or cancer in populations living at  
8 distances < 500 meters from base stations. None of the studies reported exposure above  
9 accepted international guidelines, suggesting that current guidelines may be inadequate in  
10 protecting the health of human populations (Khurana et al 2010). The results and  
11 discussion by the Salford group on non-thermal effects on the blood-brain-barrier and  
12 neuronal death in their experimental studies are most relevant in this context (Salford et  
13 al 2012), see also discussion below.

14 **Q. Based on all of the epidemiological evidence available, please state your opinion**  
15 **about the extent of the association between exposure to RF-EMF and cancer.**

16 A. There is a consistent pattern of an increased risk of glioma (malignant brain tumor) and  
17 acoustic neuroma (benign tumor on the 8<sup>th</sup> cranial nerve) associated with use of wireless  
18 phones (both mobile phones and cordless phones). The increased risk is highest for  
19 tumors on the same side of the head (ipsilateral) as the phone has been used during long-  
20 term and highest cumulative use in hours over the years. The conclusion is based on all  
21 studies, especially those that provide results for at least 10 years latency period (time  
22 from first use until tumor diagnosis) for use of wireless phones.

1           It should be noted that this association was not found for meningioma. Patients  
2           with that tumor type were included in the same studies on glioma and acoustic neuroma.  
3           A systematic bias in the studies that would explain the results for glioma and acoustic  
4           neuroma would also have been the case for meningioma. Thus, different results in the  
5           same studies according to tumor type give strength to the findings of an increased risk for  
6           glioma and acoustic neuroma.

7           In summary the strengths of evidence areas follow, using the Hill criteria from  
8           1965 (Hill 1965). For more details see also Carlberg, Hardell (2012) and Hardell et al  
9           (2011, 2012).

- 10       1. **Strength:** Ipsilateral use (tumor in the brain on same side as where wireless phone  
11           was used) finds the highest risk, especially for localisations with highest exposure  
12           (temporal lobe). Cumulative hours of use finds the highest risk
- 13       2. **Consistency:** Similar results have been found in different studies, e.g. the Hardell  
14           group and Interphone.
- 15       3. **Specificity:** Regions of brain which absorb the highest wireless phone radiation (e.g.,  
16           temporal lobe) have the highest risk. Risk pattern differs according to brain tumor  
17           type.
- 18       4. **Temporality:** Those with most years since first use have the highest risk; i.e. an  
19           effect of latency time.
- 20       5. **Biological gradient:** There is a clear dose-response effect, i.e. higher cumulative use  
21           in hours of wireless phones gives a higher risk with statistically significant trend.
- 22       6. **Plausibility:** Laboratory studies show toxic effects from RF-EMF on DNA that may  
23           lead to an increased risk for brain tumors. These non-thermal effects are mediated via



1 the formation of reactive oxygen species (ROS).

2 7. **Coherence:** Several studies show by now an increasing incidence of brain tumors,  
3 especially of the type that would be expected based on epidemiological results  
4 (glioblastoma multiforme), in the most exposed parts of the brain (temporal and  
5 adjacent lobes).

6 8. **Experiment:** In the Hardell group studies it was shown that the group of subjects that  
7 only used the mobile phone in a car with a fixed external antenna (no exposure) did  
8 not have an increased risk for brain tumors in contrast to users without such devices  
9 (Hardell et al 2002). The EMF-RF toxic effects on DNA mediated by ROS can be  
10 prevented by antioxidants.

11 Sir Austin Bradford Hill (1965) noted that: “However, before deducing ‘causation’ and  
12 taking action we shall not invariably have to sit around awaiting the results of that  
13 research. The whole chain may have to be unravelled or a few links may suffice. It will  
14 depend on circumstances.” p 295. And “If we are wrong in deducing causation from  
15 associations no great harm will be done....All scientific work is incomplete...That does  
16 not confer upon us a freedom to ignore the knowledge we already have, or to postpone  
17 the action that it appears to demand at a given time.” p 300.

18 Using the Hill criteria on use of wireless phones and brain tumor risk infers causation of  
19 the association found in epidemiological studies. Most of these criteria are fulfilled.

20 **Q. Have you reviewed the joint testimony of William H. Bailey, Ph.D. and Yakov**  
21 **Shkolnikov, Ph.D., dated September 19, 2012, and their testimony dated**  
22 **November 16, 2010, which was presented jointly with Linda S. Erdreich, Ph.D.?**

23 **A. Yes.**

1   **Q.   In their testimony, Dr. Bailey and Dr. Shkolnikov cite a report by the ICNIRP**  
2       **Committee, which concluded, “the trend in the accumulated evidence is increasingly**  
3       **against the hypothesis that mobile phone use causes brain tumors.” Do you agree**  
4       **with that conclusion?**

5   **A.**   No, this is not a statement based on up-to-date knowledge of the scientific literature; see  
6       above, e.g. regarding meta-analysis of the brain tumor risk. Furthermore, it should be  
7       noted that the International Agency for Research on Cancer (IARC) at WHO evaluation  
8       of the carcinogenic effect of RF-EMF on humans took place during a 24 – 31 May 2011  
9       meeting at Lyon in France. The Working Group consisted of 30 scientists and  
10      categorised the radiofrequency electromagnetic fields from mobile phones, and from  
11      other devices that emit similar non-ionising electromagnetic fields (RF-EMF), as Group  
12      2B, i.e. a ‘possible’, human carcinogen (Baan et al 2011, IARC in press).

13           The IARC decision on mobile phones was based mainly on two sets of case-  
14      control human studies; the Hardell group of studies from Sweden and the IARC  
15      Interphone study. Both provided complementary and supportive results on positive  
16      associations between two types of brain tumors; glioma and acoustic neuroma, and  
17      exposure to RF-EMF from wireless phones. I was an invited expert and the results from  
18      our research group were included in the evaluation as foundation for classification of the  
19      carcinogenic potential to humans from RF-EMF exposure. The Danish cohort study on  
20      mobile phone ‘users’ was considered to be uninformative. Besides our studies only  
21      Interphone gave results for long-term use of mobile phones (also cordless phones in our  
22      study group). The other studies were judged to be uninformative as to long-term cancer  
23      risks.

1   **Q.   Dr. Bailey and Dr. Shkolnikov testified that: “Despite the substantial increase in**  
2       **mobile phone use since the mid-1990s, rates of brain cancer incidence have**  
3       **generally remained consistent over time.” Do you agree with that conclusion?**

4   **A.**   No, see our discussion about brain tumor incidence in our recent article in  
5       Pathophysiology (Hardell et al 2012). In fact several studies have shown increasing  
6       incidence of brain tumors.

7           In Denmark a statistically significant increase in incidence rate per year for brain  
8       and central nervous system tumors (combined) was seen during 2000-2009; in men +2.7  
9       %, 95 % CI = +1.1 to 4.3 % and in women +2.9 %, 95 % CI = +0.7 to 5.2 %  
10       (NORDCAN). Furthermore, updated results for brain and central nervous system tumors  
11       have been released in Denmark. The age-standardized incidence of brain and central  
12       nervous system tumors increased with 40 % among men and 29 % among women during  
13       2001-2010 (Sundhedsstyrelsen, 2010). A more recent news release based on the Danish  
14       Cancer Register stated that during the last 10 years there has been an increasing number  
15       of cases with the most malignant glioma type, glioblastoma multiforme (astrocytoma  
16       WHO grade IV), especially among men ([http://www.cancer.dk/Nyheder/nyhedsartikler](http://www.cancer.dk/Nyheder/nyhedsartikler/2012kv4/Kraftig+stigning+i+hjernesvulster.htm)  
17       [/2012kv4/Kraftig+stigning+i+hjernesvulster.htm](http://www.cancer.dk/Nyheder/nyhedsartikler/2012kv4/Kraftig+stigning+i+hjernesvulster.htm)).

18           Zada et al (2012) studied incidence trends of primary malignant brain tumors in  
19       the Los Angeles area during 1992-2006. The overall incidence of primary malignant  
20       brain tumors decreased over the time period with the exception of glioblastoma  
21       multiforme (astrocytoma WHO grade IV). The annual age adjusted incidence rate of that  
22       tumor type increased statistically significant in the frontal lobe of the brain with Annual  
23       Percentage Change (APC) +2.4 % to +3.0 % ( $p \leq 0.001$ ) and temporal lobe APC +1.3 %

1 to +2.3 % ( $p \leq 0.027$ ) across all registries. In the California Cancer Registry the  
2 incidence of glioblastoma multiforme increased also in cerebellum, APC +11.9 % ( $p <$   
3 0.001). That means that the incidence increased most in the parts of the brain with highest  
4 absorption of RF-EMF emissions during use of wireless phones (Cardis et al 2008) and  
5 especially for the tumor type, glioblastoma multiforme, with high risk from such  
6 emissions (Hardell et al 2012).

7 Of interest is also the report by de Vocht et al (2011) from England that showed  
8 for the time period 1998 to 2007 a statistically significant increasing incidence of brain  
9 tumors, the majority glioma, in the temporal lobe for men and women ( $p < 0.01$ ), and  
10 frontal lobe for men ( $p < 0.01$ ). That means an increasing risk for a tumor type in a  
11 specific area of the brain that would be predicted based on current knowledge on the risk  
12 pattern.

13 Deltour et al (2012) reported increasing glioma incidence rates in Denmark,  
14 Finland, Norway, and Sweden for the time period 1979-2008. APC increased for men  
15 with +0.4 %, 95 % CI +0.1 to 0.6 % and for women with +0.3 %, 95 % CI +0.1 to 0.5 %.  
16 A study from Australia for the time period 2000-2008 showed that APC for malignant  
17 brain tumors increased statistically significant +3.9 %, 95 % CI +2.4 to 5.4 % (Dobes et  
18 al 2011). An increase was seen among both men and women. The APC for benign  
19 tumors increased with +1.7 %, 95 % CI -1.4 to +4.9 %, thus not statistically significant.

20 From urban Shanghai an increasing incidence of brain and nervous system  
21 tumors for the time period 1983-2007 was reported with APC +1.2 %, 95 % CI +0.4 to  
22 1.9 % in males and APC +2.8 %, 95 % CI +2.1 to 3.4 % in females (Ding and Wang  
23 2011).

1           We reported increasing incidence of astrocytoma WHO grades I-IV during 1970-  
2           2007 in Sweden. In the age group > 19 years the annual change was +2.16 %, 95 % CI  
3           +0.25 to 4.10 % during 2000-2007, for further details see Hardell and Carlberg (2009).

4           In summary, it should be noted that studies on tumor type and anatomical  
5           localisation show an increasing incidence of brain tumors that would be expected based  
6           on increased risk from RF-EMF emissions.

7   **Q. Dr. Bailey and Dr. Shkolnikov discuss the IARC 2B classification. They emphasize**  
8   **a finding by the IARC that there is only “limited evidence” for cancer resulting**  
9   **from RF exposure. Can you explain what is meant by “limited evidence” in this**  
10   **context and its implications for assessing potential risks to humans from RF**  
11   **exposure?**

12   **A.**   A summary of the evaluation has been published by Baan et al in Lancet Oncology 2011  
13           (Baan et al 2011) and the whole Monograph is to be published (IARC in press).

14           The conclusion at the IARC meeting was that RF-EMF is a ‘possible’ human  
15           carcinogen, Group 2B of evidence. There was a very large majority among the 30  
16           participating independent experts for that decision; almost all voted for Group 2B in the  
17           summary evaluation. I was one of the 30 invited experts. The classification system can  
18           be found in the IARC Preamble.

19           (<http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf>) .

20           IARC evaluates the ‘**hazard**’ of potential carcinogens, i.e. ‘an agent that is  
21           capable of causing cancer under some circumstances’, while a cancer **risk** is an estimate  
22           of the carcinogenic effects expected from an exposure to a cancer hazard. The IARC

monographs are an exercise in evaluating cancer hazards, despite the historical presence of the words ‘risks’ in the title.

IARC has categorised nearly 1 000 potentially carcinogenic **hazardous** agents, which it has studied over the past 40 years, into 5 classifications. These are differentiated by strengths of evidence.

In descending order of strengths of evidence they are:

- **Group 1**, which are ‘**established**’ human carcinogens, such as asbestos, diesel engine exhaust, tobacco, and X-rays (109 agents);
- **Group 2A**, which are ‘**probable**’ carcinogens, such as perchloroethylene (65 agents);
- **Group 2B**, which are ‘**possible**’ carcinogens, such as other traffic fumes, lead, DDT and now radiofrequency electromagnetic fields including mobile phones (275 agents);
- **Group 3**, where the agent is ‘**not classifiable**’ because the evidence is inadequate and does not permit another classification (503 agents); and
- **Group 4**, where the agent is ‘**probably not carcinogenic to humans**’, based on fairly strong evidence **against** a cancer effect in both humans and animals (1 agent).

Source: <http://monographs.iarc.fr/ENG/Classification/index.php>

It should be noted that most of the agents (n=503) were evaluated as ‘not classifiable’ Group 3 and one as ‘probably not carcinogenic to humans’, Group 4. Thus classification of RF-EMF as a carcinogen (possible) is based on scientific literature. It is not an agent that cannot be classified according to its potency to be a human carcinogen. This is an important distinction regarding the carcinogenic evidence of exposure to RF-EMF, i.e. belongs to the group of agents in Group 1 or 2 with carcinogenic potential.

More discussion can be found in our chapter in Late Lessons from Early Warnings, part 2, published by European Environment Agency, Copenhagen, Denmark (Hardell et al 2013).

1 **Q. Have there been any court cases in Europe finding that cancer was caused by EMF**  
2 **exposure?**

3 A. Yes. An increased risk for leukemia among children has been reported in a small town  
4 near Rome because of the Vatican Radio broadcaster's high-powered transmitters. That  
5 is the type of malignant disease that would be expected based on scientific knowledge  
6 and the IARC evaluation of ELF-EMF as a Group 2B carcinogen to humans (IARC  
7 2002). Italy's supreme Court has ordered Vatican Radio to compensate the victims  
8 ([http://www.independent.co.uk/news/world/europe/vatican-radio-is-told-to-pay-out-over-](http://www.independent.co.uk/news/world/europe/vatican-radio-is-told-to-pay-out-over-cancer-risk-scare-2228541.html)  
9 [cancer-risk-scare-2228541.html](http://www.independent.co.uk/news/world/europe/vatican-radio-is-told-to-pay-out-over-cancer-risk-scare-2228541.html))  
10 (<http://www.magdahavas.com/study-finds-vatican-radio-causes-cancer>).

11 The first case on compensation of a patient who developed a neuroma after long-  
12 term wireless phone use has now been established in court. The Italian Supreme Court  
13 affirmed a previous ruling that the Insurance Body for Work (INAIL) must grant  
14 worker's compensation to a businessman who had used wireless phones for 12 years and  
15 developed a neuroma in the brain ([www.applelettrosmog.it](http://www.applelettrosmog.it); [www.microwavenews.com](http://www.microwavenews.com)).  
16 He had used both mobile and cordless phones for five to six hours per day preferably on  
17 the same side as the tumor developed. The neuroma was located in the trigeminal  
18 Gasser's ganglion in the brain (5<sup>th</sup> cranial nerve). It is the same type of tumor as the  
19 acoustic neuroma in the 8<sup>th</sup> cranial nerve located in the same area of the brain where an  
20 increased risk of acoustic neuroma has been found among wireless phone users. The  
21 Italian case fulfils the criteria for a causal association; more than 10 years use of wireless  
22 phones, high cumulative exposure on the same side as the tumor appeared, and a tumor

1 type that would be predicted based on previous research on use of wireless phones and  
2 brain tumor risk. No further appeal of the Supreme Court decision is possible.

3 **Q. Does the IARC 2B classification of carcinogenicity apply to all sources of RF-EMF?**

4 A. Yes. In an email by Dr Baan at IARC dated 29 Aug 2011 it was stated that: “So the  
5 classification 2B, possibly carcinogenic, holds for all types of radiation within the  
6 radiofrequency part of the electromagnetic spectrum, including the radiation emitted by  
7 base-station antennas, radio/TV towers, radar, Wi-Fi, smart meters, etc.”, see Exhibit C.  
8 This is an important message; the IARC decision includes “the whole radiofrequency  
9 region of the electromagnetic spectrum.” The frequency range 30 kHz – 300 GHz is also  
10 defined in the publication by Baan et al (2011).

11 **Q. Dr. Bailey and Dr. Shkolnikov testified that: “a careful scientist cannot conclude  
12 that studies and reports have identified a true, non-thermal effect.” Do you agree,  
13 assuming a definition of non-thermal effect as biological effects occurring below RF-  
14 EMF intensities at which thermal effects are known to occur?**

15 A. There are by now a vast majority of scientific studies published in peer-reviewed  
16 scientific journals showing non-thermal effects. The brain tumor risk as discussed above  
17 is one non-thermal risk.

18 We used serum transthyretin (TTR) as a marker of cerebrospinal fluid and blood-  
19 brain barrier damage in a cross-sectional study on 313 subjects (Söderqvist et al 2009a).  
20 We found a statistically significant positive  $\beta$  coefficient for TTR for time since first use  
21 of mobile phones and desktop cordless phones combined ( $P = 0.03$ ), i.e. increased serum  
22 concentrations of TTR. The electromagnetic field parameters were similar for the phone  
23 types. In a provocation study on 41 persons exposed for 30 min to an 890-MHz GSM



1 signal with specific absorption rate of 1.0 Watt/kg to the temporal area of the brain, we  
2 found statistically significant increased serum TTR 60 min after exposure (Söderqvist et  
3 al 2009b).

4 The lipocalin type of prostaglandin D synthase or  $\beta$ -trace protein (BTP) is  
5 synthesized in the choroid plexus, leptomeninges and oligodendrocytes of the central  
6 nervous system and is secreted into the cerebrospinal fluid. BTP is the key enzyme in the  
7 synthesis of prostaglandin D2, an endogenous sleep-promoting neurohormone in the  
8 brain. Exposure to RF-EMF has in some studies been associated with disturbed sleep.  
9 We studied the concentration of BTP in blood in relation to emissions from wireless  
10 phones (Hardell et al 2010, Söderqvist et al 2012b). The concentration of BTP decreased  
11 in the youngest age-group in the study, 18-30 years, with increasing number of years of  
12 use of a wireless phones. Also cumulative use in hours decreased the concentration of  
13 BTP. EMF emissions may down-regulate the synthesis of BTP in the brain. This  
14 mechanism might be involved in sleep disturbances reported in persons exposed to RF-  
15 EMF fields.

16 Another example of non-thermal effect is the finding that exposure to RF-EMF  
17 induces significant sperm DNA damage (Aitken et al 2005) and subsequent sperm  
18 apoptosis (De Iuliis et al 2009). Studies have shown a positive association between  
19 mobile phone use and impaired male reproduction (Agarwal et al 2009).

20 Specifically, the post-meiotic phase during DNA synthesis is the most vulnerable  
21 phase to environmental toxins, because during that phase the cells have lost part of their  
22 cytoplasm containing the abundant antioxidant enzymes that protect from reactive  
23 oxygen species (ROS) oxidation (Olsen et al 2005). Studies have shown that RF-EMF

1 may stimulate ROS generation both in vivo (Avci et al 2012) and in vitro (Lu et al 2012).  
2 Increased generation of ROS is considered to be one of the primary mechanisms that are  
3 involved in the bio-effects that are mediated by RF-EMF exposure (Friedman et al 2007).

4 In a recently published study, it was demonstrated that RF-EMF exposure induced  
5 the formation of oxidative base damage in a mouse spermatocyte-derived cell line (Liu et  
6 al 2013). This was mediated by ROS production. These results suggest that RF-EMF  
7 radiation emitted during mobile phone use may produce genotoxicity in the form of DNA  
8 base damage, see Graphical Abstract with Legend, Dr Liu with courtesy, Exhibit D.

9 To further elucidate the central role of ROS in RF-EMF exposure-induced DNA  
10 base damage, the authors used  $\alpha$ -tocopherol pre-treatment to antagonize the oxidation of  
11 ROS;  $\alpha$ -tocopherol is an important lipophilic antioxidant that can inactivate harmful  
12 ROS. The protective role of  $\alpha$ -tocopherol pre-treatment confirmed that ROS are involved  
13 in RF exposure-induced DNA base damage (Liu et al 2013). The mode of action for RF-  
14 EMF-induced genotoxicity involved the induction of oxidative DNA base damage.  
15 These findings support the novel idea that low energy RF-EMF that is insufficient to  
16 directly induce DNA strand breaks may nonetheless produce genotoxic effects in the  
17 form of DNA base damage.

18 Another recently published study showed that 2.45 GHz low-level RF-EMF  
19 radiation induced oxidative stress and suppressed implantation or pregnancy in mice. It  
20 was also concluded that it might lead to deformity of the embryo in case pregnancy  
21 continues. The oxidative stress may lead to DNA strand breakage in the brain according  
22 to the authors and thus be a mechanism for causation of brain tumors. The effects were

1 non-thermal at power density =  $0.033549 \text{ mW/cm}^2$ , and specific absorption rate (SAR) =  
2  $0.023023 \text{ W/kg}$  (Shahin et al 2013).

3 Antioxidants such as melatonin, vitamin C and vitamin E can alleviate the ROS  
4 oxidation and apoptosis that are induced by RF-EMF in an animal model (Oral et al 2006,  
5 Ozguner et al 2006).

6 The results in the study by Liu et al (2013) and Shahin et al (2013) are important  
7 findings to further elucidate the mechanisms for RF-EMF genotoxicity. These effects are  
8 clearly non-thermal. In summary these and other studies show that oxidative stress is an  
9 important mechanism for adverse health effects from RF-EMF emissions.

10 Non-thermal effects of electromagnetic fields on living systems have been further  
11 discussed in a monograph from the Ramazzini Institute (Giuliani, Soffritti 2010).  
12 [http://www.icems.eu/papers/ramazzini\\_library5\\_part1.pdf](http://www.icems.eu/papers/ramazzini_library5_part1.pdf);  
13 [http://www.icems.eu/papers/ramazzini\\_library5\\_part2.pdf](http://www.icems.eu/papers/ramazzini_library5_part2.pdf)

14 **Q. Is the blood-brain-barrier (BBB) a predictor of low-dose, or “non-thermal”, adverse**  
15 **health effects?**

16 A. Yes. In the BioInitiative Report 2012 leading researchers in this area, Dr Salford, Dr  
17 Nittby and Dr Persson conclude: “The intense use of mobile phones, not least by  
18 youngsters, is a serious memento. A neuronal damage may not have immediately  
19 demonstrable consequences, even if repeated. It may, however, in the long run, result in  
20 reduced brain reserve capacity that might be unveiled by other later neuronal disease or  
21 even the wear and tear of ageing. We cannot exclude that after some decades of (often),  
22 daily use, a whole generation of users, may suffer negative effects such as autoimmune

1 and neuro-degenerative diseases maybe already in their middle age”. (Salford et al 2012,  
2 page 45). They continue with:

3 “One remarkable observation, which we have made in our studies  
4 throughout the years, is that exposure with whole-body average power  
5 densities below 10 mW/kg gives rise to a more pronounced albumin  
6 leakage than higher power densities, all at non-thermal levels. These very  
7 low SAR-values, such as 1 mW/kg, exist at a distance of more than one  
8 meter away from the mobile phone antenna and at a distance of about  
9 150–200 m from a base station.

10 Further, when a mobile phone operating at 915 MHz (and its  
11 antenna) is held 1.4 cm from the human head, the very low SAR levels of  
12 10 mW/kg exist in deep-lying parts of the human brain such as the basal  
13 ganglia, and the power density of 1 mW/kg and less is absorbed in  
14 thalamus bilaterally.

15 With this information as a background, it is difficult to recommend  
16 safety limits as the function of existing mobile systems might not allow for  
17 limits that produce SAR levels below 1 or 0,1 mW/kg in the human brain,  
18 which are reported to cause a pathological leakage of the BBB and to  
19 neuronal damage.” (Salford et al 2012, Page 45)

20 Thus, these very low RF-EMF levels giving neuronal damage (dark neurons) and BBB-  
21 leakage must be considered in the context of exposure from smart meters. Note the  
22 oxidative stress found in the Shahin et al (2013) study at power density 0.033549  
23 mW/cm<sup>2</sup>.

1  
2 **Q. Do you agree with their testimony that the authors of the BioInitiative Report used**  
3 **flawed methods and failed to follow “the standard, scientific methods for developing**  
4 **exposure limits.”**

5 A. No. This is a statement that has no scientific credibility and is unfounded; see e.g. the  
6 BioInitiative Report 2012 ([www.bioinitiative.org](http://www.bioinitiative.org)). In the Preface  
7 ([http://www.bioinitiative.org/report/wp-content/uploads/pdfs/seci\\_2012\\_Preface.pdf](http://www.bioinitiative.org/report/wp-content/uploads/pdfs/seci_2012_Preface.pdf)) it  
8 is stated that:

9 “Today, the BioInitiative 2012 Report updates five years of  
10 science, public health, public policy and global response to the growing  
11 health issue of chronic exposure to electromagnetic fields and  
12 radiofrequency radiation in the daily life of billions of people around the  
13 world.

14 The BioInitiative 2012 Report has been prepared by 29 authors  
15 from ten countries, ten holding medical degrees (MDs), 21 PhDs, and  
16 three MsC, MA or MPHs. Among the authors are three former presidents  
17 of the Bioelectromagnetics Society [BEMS], and five full members of  
18 BEMS. One distinguished author is the Chair of the Russian National  
19 Committee on Non-Ionizing Radiation. Another is a Senior Advisor to the  
20 European Environmental Agency. As in 2007, each author is responsible  
21 for their own chapter.

22 The great strength of the BioInitiative Report  
23 ([www.bioinitiative.org](http://www.bioinitiative.org)) is that it has been done independent of

1 governments, existing bodies and industry professional societies that have  
2 clung to old standards. Precisely because of this, the BioInitiative Report  
3 presents a solid scientific and public health policy assessment that is  
4 evidence-based.” ([http://www.biointitiative.org/report/wp-](http://www.biointitiative.org/report/wp-content/uploads/pdfs/seci_2012_Preface.pdf)  
5 [content/uploads/pdfs/seci\\_2012\\_Preface.pdf](http://www.biointitiative.org/report/wp-content/uploads/pdfs/seci_2012_Preface.pdf))

6 It should be stressed that the different chapters are written by world leading experts in the  
7 relevant scientific areas.

8 **Q. Are there peer-reviewed studies reporting results that could support a plausible**  
9 **explanation for any mechanisms by which cancer could be caused by RF exposure?**

10 A. Yes. The energy level associated with exposure is too low to cause direct DNA strand  
11 breaks and DNA crosslinks. However, DNA damages can be caused by cellular  
12 biochemical activities such as free radicals. Several studies indicate that RF-EMFs  
13 increase free radical activity in cells (Lai, Singh 1997, Phillips et al 2009). One  
14 mechanism is probably mediated via the Fenton reaction. Hydrogen peroxide is  
15 converted into hydroxyl free radicals that are potent cytotoxic molecules. This reaction is  
16 catalyzed by iron. High levels of iron are found in metabolic active cells such as cancer  
17 cells and cells undergoing abnormal proliferation, but also in brain cells. Glia cells might  
18 turn cancerous due to DNA damage, see also discussion above.

19 **Q. Are children and adolescents more sensitive than adults to the toxic effects from RF-**  
20 **EMF exposure?**

21 A. Yes. Children have smaller head and thinner skull bone than adults. Their brain tissue  
22 has also higher conductivity and these circumstances give higher absorption from RF-  
23 EMF than for adults (Cardis et al 2008, Christ et al 2010, Gandhi et al 2012). The

1 developing brain is more sensitive to toxins (Kheifets et al 2005) and it is still developing  
2 until about 20 years of age (Dosenbach et al 2010). The greater absorption of RF energy  
3 per unit of time, the greater sensitivity of their brains, and their longer lifetimes with the  
4 risk to develop a brain tumor and other diseases leaves children at a higher risk than  
5 adults from mobile phone radiation.

6 We studied the risk for glioma in different age groups. Highest risk was found for  
7 first use of mobile phone or cordless phone before the age of 20 years. Thus, mobile  
8 phone use yielded for glioma OR = 3.1, 95 % CI = 1.4-6.7 and cordless phone OR 2.6, 95  
9 % CI = 1.2-5.5. The risk increased further for ipsilateral mobile phone use in the  
10 youngest age group to OR = 4.4, 95 % CI = 1.3-15, and to OR = 4.3, 95 % CI = 1.4-13  
11 for cordless phone use (Hardell et al 2012).

12 Also for acoustic neuroma the risk was highest in the youngest age group with OR  
13 = 5.0, 95 % CI = 1.5-16 for use of mobile phone increasing to OR = 6.8, 95 % CI = 1.4-  
14 34 for ipsilateral use.

15 CEFALO was a multicenter case-control study on the risk for brain tumors among  
16 children and adolescents aged 7–19 years. A statistically non-significant increased risk  
17 among regular users (one call per week for at least 6 months) of mobile phones was  
18 found; OR = 1.36, 95 % CI = 0.92-2.02. This OR increased somewhat with cumulative  
19 duration of subscriptions and duration of calls (Aydin et al 2011a). No data for long-term  
20 use were given; the longest latency period was 5 years. Interestingly, further support of a  
21 true association was found in the results based on operator-recorded use for 62 cases and  
22 101 controls, which for time since first subscription >2.8 years yielded a statistically

1 significant OR of 2.15, 95 % CI = 1.07-4.29, with a statistically significant trend  
2 (p=0.001).

3 Use of cordless phones was not well assessed. The authors stated that such use  
4 was covered only in the first 3 years of use. No explanation was given for this most  
5 peculiar definition. Wireless phone use was not considered, that is use of both mobile  
6 phones and cordless phones as the relevant exposure category, as used by the Hardell  
7 group and adopted by IARC (Baan et al 2011). Instead Aydin et al (2011a) included use  
8 of cordless phones in the ‘unexposed’ category when risk estimates were calculated for  
9 mobile phone use. Similarly, when use of cordless phones was analysed mobile phone  
10 use was regarded as ‘no exposure’. Thus, an increased risk was potentially concealed.

11 Aydin et al discussed recall bias – that people tend to overestimate their number  
12 of calls – and interestingly they showed that controls overestimated their number of calls  
13 more than cases (Aydin et al 2011b). It was concluded that it was unlikely that a false  
14 positive result occurred in CEFALO and that the OR was underestimated for heavy users.  
15 Thus the results in the study were biased towards unity (no risk) for several reasons, e.g.  
16 including use of cordless phones in the ‘unexposed’ group and overestimated use of  
17 mobile phones among the controls. The true OR would thus have been higher than that  
18 presented in the study. The authors summarised that they “did not observe that regular  
19 use of a mobile phone increased the risk for brain tumors in children and adolescents.”  
20 This statement is not correct. The results indicate an increased risk, in spite of low  
21 exposure, short latency period and limitations in study design and analyses, as we have  
22 discussed elsewhere (Söderqvist et al 2011).



1 Another example of the higher sensitivity of adolescents is our finding of  
2 decreased BTP concentration in blood among long-term wireless phone users in the age  
3 group 18-30 years, but not among older persons, see Hardell et al ( 2010) and Söderqvist  
4 et al (2012b).

5 **Q. Should peak power densities or average power densities of RF-EMF be considered**  
6 **as to the health effects?**

7 A. This is a question that has not been well studied. First it must be emphasized that a  
8 carcinogenic hazard in general exists regardless of dose for epigenetic toxic effects, i.e.  
9 no threshold. Dr Baan at IARC notes in a March 30, 2012 email that with lower dose “the  
10 hazard still exists”. *See Exhibit E.*

11 As to ionizing radiation it is well known that the toxic effect of a specific  
12 radiation dose is higher if given as a single shot in radiotherapy than if the same dose is  
13 fractionated over several days (lower average dose over time).

14 By analogy peak density of RF-EMF may more accurately represent the radiation  
15 exposure to the body than power density that is a calculated average dose during a  
16 specified time, i.e. single peaks of radiation may have toxic effects and multiple peaks of  
17 radiation may have cumulative effects that are not accurately represented by averaged  
18 values. Thus, peaks of density should not be recalculated as average dose over time  
19 when the risk is estimated, instead the peak density should also be considered. The peak  
20 density is of special concern regarding e.g. the foetus (pregnant women), children,  
21 adolescents, sick and disabled.

22 Regarding ionizing radiation there is no homeostasis; that is that the body does  
23 not adapt to the exposure. The effects are permanent when a given part of the body is

1 irradiated, the given radiation dose makes additional radiotherapy impossible (depending  
2 on dose) – otherwise more serious tissue damage would occur. Regarding RF-EMF there  
3 are no well-done studies that show homeostasis in the human body for non-thermal  
4 effects. On the contrary studies show accumulating adverse health effects as exemplified  
5 by the brain tumor risk depending on cumulative dose over time (dose-response). Our  
6 results of decreased BTP concentration among long-term users of wireless phones (18-30  
7 years of age) is another example of a cumulative effect and not homeostasis. If the  
8 human body would adapt to the RF-EMF exposure no adverse health risks would occur,  
9 which is not the case.

10 **Q. Dr. Bailey and Dr. Shkolnikov testified that: “The weight of the evidence does not**  
11 **support the idea that significant biological or adverse health effects can occur” from**  
12 **EMF-RF exposure. Do you agree with this conclusion?**

13 A. No. On the contrary the weight of evidence shows an increased risk for certain types of  
14 brain tumors, see discussion above. The results are in accordance with what would  
15 biologically be plausible; see above the Hill (1965) criteria of evidence. On 23 January  
16 2013 concern of adverse health effects by e.g. use of mobile phones were expressed at a  
17 meeting at the EU parliament in Brussels while Late Lessons from Early Warnings,  
18 Volume II was launched.

19 ([http://www.eea.europa.eu/acl\\_users/credentials\\_cookie\\_auth/login\\_form?came\\_from=ht](http://www.eea.europa.eu/acl_users/credentials_cookie_auth/login_form?came_from=http%3A//www.eea.europa.eu/publications/late-lessons-2)  
20 [tp%3A//www.eea.europa.eu/publications/late-lessons-2](http://www.eea.europa.eu/publications/late-lessons-2)).

21 Furthermore, several studies show other non-thermal effects in humans that can  
22 be linked to certain outcomes like fertility and disturbed sleep as examples, see also the  
23 BioInitiative 2012 Report ([www.bioinitiative.org](http://www.bioinitiative.org)).

1   **Q.    In your opinion, could a careful scientist familiar with the body of knowledge on the**  
2       **subject reliably conclude that there are no risks of adverse health effects from the**  
3       **exposure to RF in the 2.4 GHz range?**

4    A.   No. The body of evidence shows risk of impaired health from the RF-EMF exposure and  
5       2.4 GHz is included in the IARC evaluation 30 kHz – 300 kHz. Hill’s analytic  
6       framework for assessing when the association is strong enough to infer causation and  
7       warrant action has been discussed above. This is particularly relevant when the question  
8       is whether a person is exposed to an agent of possible harm, such as RF-EMF, in the  
9       home or in his/her own living environments without consent.

10     Sir Bradford Hill in his famous article on causation provides a helpful framework for  
11     assessing the risk and he offers some very insightful comments that are useful in this  
12     context (Hill 1965). To answer those scientists who insist that every positive study be  
13     replicated, he states that “Once again looking at the obverse of the coin there will be  
14     occasions when repetition is absent or impossible and yet we should not hesitate to  
15     draw conclusions”. Hill, p. 297.

16     To those who insist we wait until the exact causal mechanism is established, he states:  
17     “It will be helpful if the causation we suspect is biologically plausible. But this is a  
18     feature I am convinced we cannot demand. What is biologically plausible depends  
19     upon the biological knowledge of the day”. P. 298.

20     To those who insist on more in vivo or in vitro evidence, he states: “Nevertheless,  
21     while such laboratory evidence can enormously strengthen the hypothesis and, indeed,  
22     may determine the actual causative agent, the lack of such evidence cannot nullify the

1 epidemiological observations in man”. P. 298 “What I do not believe - and this has  
2 been suggested - is that we can usefully lay down some hard-and-fast rules of evidence  
3 that must be obeyed before we accept cause and effect.” P. 299

4 **Q. What is the message from the European Environment Agency (EEA) in**  
5 **Copenhagen regarding emerging technologies such as wireless networks currently**  
6 **in use?**

7 A. On 23 January 2013 EEA published their ‘Late Lessons from Early Warnings, Volume  
8 II’ at a public meeting in the EU parliament in Brussels. Among the Key  
9 recommendations are:

- 10 • Policy makers should respond to early warnings more rapidly, the report says,  
11 particularly in cases of large scale emerging technologies. It proposes that those  
12 causing any future harm should pay for the damage.
- 13 • Risk assessment can also be improved, the report says, by embracing uncertainty  
14 more broadly and acknowledging what is not known. For example, ‘No evidence  
15 of harm’ has often been misinterpreted to mean ‘evidence of no harm’ when the  
16 relevant research was not available.
- 17 • The report calls for new forms of governance involving citizens in choices about  
18 innovation pathways and risk analysis. This would help to reduce exposure to  
19 hazards and encourage innovations with broader societal benefits. Greater  
20 interaction between business, governments and citizens could foster more robust  
21 and diverse innovations at less cost to health and the environment  
22 (<http://www.eea.europa.eu/pressroom/newsreleases/the-cost-of-ignoring-the>) .

23  
24 One chapter deals with mobile phones and brain tumors as an emerging issue (Hardell et  
25 al 2013). The whole volume can be downloaded from the EEA website  
26 (<http://www.eea.europa.eu/publications/late-lessons-2>).

27 **Q. Based on your own research and scientific knowledge what is your opinion about**  
28 **the risks associated with smart meters?**

A.           There is accumulating scientific evidence on the potential of exposure to RF-EMF to give adverse health consequences and these effects are clearly non-thermal at low intensity. Certain groups of humans such as children, adolescents, elderly and the sick and disabled are more sensitive than others. The unborn child (foetus) may be at especially high risk. Exposure to RF-EMF from smart meters is without consent in contrast to use of wireless phones that are used by the individual's own choice. I have not performed a detailed study of smart meter technology, and ultimately this is more of a policy question than a scientific one. But, in my opinion, using the Hill criteria on association or causation, there is sufficient evidence to warrant actions that would prevent or avoid exposure, and the utility should be required to prove that smart meter radiation is safe without relying on safety standards that protect only against thermal effects.

Dated this \_\_\_31st\_\_\_ day of January, 2013.

  
\_\_\_\_\_  
Lennart Hardell

# Hardell Exhibit A

## C.V. Lennart Hardell

### Education, appointments

Medical degree from University of Uppsala, Sweden, October 27, 1971.  
Authorization to practice the medical profession, National Board of Health and Welfare, Stockholm, November 5, 1971.  
Specialized in internal medicine, October 18, 1976.  
Specialized in oncology, June 20, 1979.  
Medical dissertation, University of Umeå, May 15, 1981.  
Qualified as an Associate Professor of Oncology, University of Umeå, December 9, 1983.  
Research fellow at School of Public Health, University of California, Berkeley, USA, August 20, 1984 - August 19, 1985.  
Consultant at the Department of Oncology, Örebro Medical Center, Örebro, Sweden August 1, 1991.  
Qualified for appointment as a Professor of Oncology in Århus, Denmark, 1992.  
Research appointment at the Swedish Medical Research Council July 1, 1994 – June 30, 2000 in molecular genetic epidemiology.  
Professor in Oncology, Örebro University, Örebro May 1, 2001.

### Research, academic assignments

Head of a research team since the 1980's investigating mostly environmental risk factors for cancer  
Supervised eight researchers for their medical dissertations.  
Served in several expert panels for medical dissertations.  
Chairman of Örebro County Council Radiation Protection Committee July 1, 1995 – December 31, 1998.  
Vice chairman in Örebro County Council Research Committee January 27, 1992 – December 31, 1998.  
Vice secretary of Örebro County Council Ethical Committee January 27, 1992 – December 31, 1998.  
Deputy of Ethical Committee at Akademiska sjukhuset, Uppsala, Sweden until December 31, 1998.  
Member of Research Committee at the Medical Center, Örebro.  
Member of Educational Committee at Örebro Medical Center, Örebro.  
Faculty member of International Journal of Oncology.  
Member of editorial board of American Journal of Industrial Medicine.  
Continuously referee for a large number of medical journals.  
Partner in EU research project: Occupational risk factors for rare cancer of unknown etiology.  
Member of Collegium Ramazzini 2003.

Member of Swedish expert group on 'Exposure to electromagnetic fields and the risk of malignant diseases - an evaluation of epidemiological and experimental findings'. 1995.  
Member of IARC expert committee on the evaluation of the carcinogenic effect of RF-EMF on humans, 24 – 31 May 2011.

Invited to a large number of scientific meetings since the 1980's to present results from my group's scientific studies,

.

Published 348 research papers and 149 abstracts, short reports

### Awards

Receiver of the Fernström Award, University of Umeå, October 9, 1982.  
Receiver of Cancer- and Allergy Fund Environmental Research Prize, April 24, 1997, Stockholm.  
Miljöpartiet de Gröna (Green Environmental Party) Research Prize, September 21, 2001.  
European Journal of Cancer Prevention Research Prize November 2005  
Acta Oncologica Lecture Prize 2006-03-22  
Prize winner Swedish Cooperation (Änglamark) 2007

## Publications

1. Hardell L. Maligna mesenkymala mjukdelstumörer och exposition för fenoxisyror - en klinisk observation. *Läkartidningen* 1977;71:2753-2754.
2. Hardell L. Kemiska substanser och maligna mesenkymala tumörer. Svensk förening för radiobiologi. *Förhandlingar* 1978;9:18-21.
3. Hardell L, Sandström A. Case-control studie: Maligna mesenkymala mjukdelstumörer och exposition för fenoxisyror eller klorfenoler. *Läkartidningen* 1978;75:3535-6.
4. Hardell L, Sandström A. Case-control study: Soft-tissue sarcomas and exposure to phenoxyacetic acids or chlorophenols. Paper presented at the conference on pesticides and human health. Society for Occupational and Environmental Health, Washington D.C., USA, December 10-13, 1978
5. Hardell L, Sandström A. Case-control study: Soft-tissue sarcomas and exposure to phenoxy acids or chlorophenols. *Br J Cancer* 1979;39:711-7.
6. Hardell L. Malignant lymphoma of histiocytic type and exposure to phenoxyacetic acids or chlorophenols. *Lancet* 1979;I:55-6.
7. Eriksson M, Hardell L, Berg NO, Möller T, Axelson O. Case-control studie över maligna mesenkymala mjukdelstumörer och exposition för kemiska substanser. *Läkartidningen* 1979;76:3872 -5.
8. Hardell L. Cancer epidemiology in Northern Sweden. *Nordic Council Arct Med Res Rep* 1980;26:94-7.
9. Hardell L. The clofibrate problem: A different viewpoint. *Lancet* 1980;II:1081 2.
10. Hardell L. Direct testimony before the United States Environmental Agency. Washington D.C. Exhibit No 762. 1980;September 29-30.
11. Eriksson M, Hardell L, Berg No, Möller T, Axelson O. Soft-tissue sarcomas and exposure to chemical substances: A case-referent study. *Br J Ind Med* 1981;38:27-33.
12. Hardell L, Eriksson M, Lenner P. En fall-kontroll studie: Maligna lymfom och exposition för kemiska substanser, särskilt organiska lösningsmedel, klorfenoler och fenoxisyror. *Läkartidningen* 1980;77:208-10.
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14. Hardell L, Eriksson M, Lenner P, Lundgren E. Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols and phenoxy acids: A case-control study. *Br J Cancer* 1981;43:169-76.
15. Hardell L, Eriksson M. Soft-tissue sarcomas, phenoxy herbicides and chlorinated phenols. *Lancet* 1981;II:250.
16. Hardell L. Relation of soft-tissue sarcoma, malignant lymphoma and colon cancer to phenoxy acids, chlorophenols and other agents. *Scand J Work Environ Health* 1981;7:119-30.

17. Hardell L. Direct testimony before the United States Environmental Protection Agency. Washington D.C. Exhibit No 956. 1981;February 9.
18. Hardell L, Eriksson M. Fenoxisyror, klorfenoler och cancer. Läkartidningen 1981;78:2862-3.
19. Hardell L. Epidemiological studies on soft-tissue sarcoma and malignant lymphoma and their relation to phenoxy acid or chlorophenol exposure. Umeå University Medical Dissertations. New Series No 65 - ISSN 0346-6612, 1981.
20. Hardell L. Fall-kontroll studier: Maligna mesenkymala mjukdelstumörer, maligna lymfom och exponering för fenoxisyror eller klorfenoler. NJF-seminar 1980; November 12 - 13. Nordisk jordbruksforskning 1981;1:58-9.
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**LENNART HARDELL**  
**EXHIBIT B**

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**LENNART HARDELL  
EXHIBIT B**

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## Hardell Exhibit C

-----Original Message-----

From: Robert Baan <[BaanR@iarc.fr](mailto:BaanR@iarc.fr)>

Date: Mon, 29 Aug 2011 09:47:10

To: connieahudson@yahoo.com<[connieahudson@yahoo.com](mailto:connieahudson@yahoo.com)>

Cc: COM ([com@iarc.fr](mailto:com@iarc.fr))<[com@iarc.fr](mailto:com@iarc.fr)>

Subject: EMF Class 2B Classification

Dear Dr Hudson,

Thank you for your message, which was forwarded to me, and to which I would like to respond as follows.

The IARC Working Group classified "Radiofrequency Electromagnetic Fields" (RF-EMF) as possibly carcinogenic to humans (Group 2B).

The information that formed the main basis for this evaluation was found in epidemiological studies on cell-phone use, where a slightly increased risk for glioma (a malignant form of brain cancer) and acoustic neuroma (a non-cancerous type) was reported among heavy users.

There were some indications of increased cancer among radar-maintenance workers (occupational exposure), but no reliable data from studies among, e.g., people living close to base-station antennas, radio/TV towers, etc (environmental exposure).

Although the key information came from mobile telephone use, the Working Group considered that the three types of exposure entail basically the same type of radiation, and decided to make an overall evaluation on RF-EMF, covering the whole radiofrequency region of the electromagnetic spectrum.

In support of this, information from studies with experimental animals showed that effects on cancer incidence and cancer latency were seen with exposures to different frequencies within the RF region.

So the classification 2B, possibly carcinogenic, holds for all types of radiation within the radiofrequency part of the electromagnetic spectrum, including the radiation emitted by base-station antennas, radio/TV towers, radar, Wi-Fi, smart meters, etc.

An important point is the radiation level. The exposure from cellular phones (personal exposure) is substantially higher and much more focused (usually on the brain) than exposures from radio/tv towers, antennas, or Wi-Fi.

I hope this is useful.

Thank you for your interest in our work.

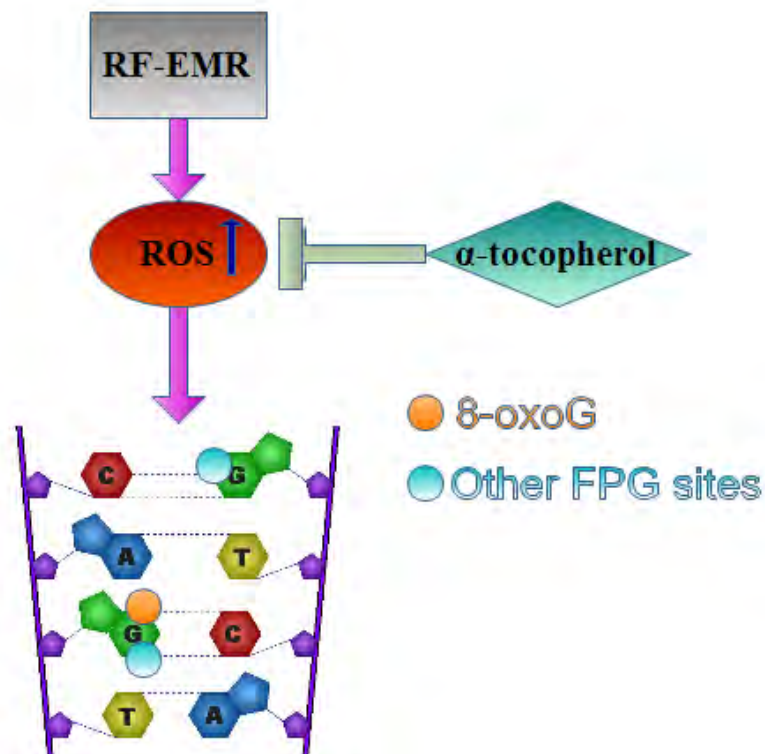
Sincerely yours,

Robert A Baan PhD  
The IARC Monographs  
IARC, Lyon, FRANCE

## Hardell Exhibit D

### Proposed model for the effects of RF-EMR on DNA integrity in germ cell line.

Though the use of a mouse spermatocyte-derived cell line, we demonstrated that RF exposure might increase ROS production and subsequently induce the formation of oxidative base damage as evaluated by FPG-comet assay and 8-oxoG formation. The increase of ROS and the protective role of  $\alpha$ -tocopherol pretreatment confirms that ROS are involved in RF exposure-induced DNA base damage. However, such exposure can't lead to detectable DNA strand breakage. These results may suggest that RF-EMR emitted during mobile phone use may produce genotoxicity in the form of DNA base damage other than DNA strand breaks.



## **Hardell Exhibit E**

**FROM: Robert A Baan PhD**  
**Responsible Officer, Monograph 102 on RF-EMF**  
**The IARC Monographs IARC,**  
**Sent: Friday, March 30, 2012 10:21 a.m.**  
**Subject: IARC's RF classification**

Dear Mrs. Atzmon,

The IARC Monographs classification of Radiofrequency Electromagnetic Fields (RFEMF) covers the entire radiofrequency segment of the electromagnetic spectrum (30 kHz-300 GHz). Within this spectrum, the electromagnetic fields around (or the radiation emitted by) mobile telephones represent the most intense and most widespread exposure situation, for which a small increase in risk for glioma and acoustic neuroma has been found in the group of 'heavy users'. Because there were also some indications of increased cancer risks from studies on occupational exposures to different frequency-ranges (in the military, in the plastic-ware industry), the IARC Working Group did not want to restrict the overall evaluation to “RF-EMF from mobile phones” or to “RF-EMF from mobile phones that were used in the late 1990s” or to “RF-EMF from mobile phones that were used in the INTERPHONE study”, since many other devices emit the same type of RF radiation, e.g., basestation antennas, radio/tv antennas, WiFi stations, smart meters, etc. Therefore, all these fall under the same broad evaluation of “Radiofrequency Electromagnetic Fields”.

This is what the Working Group discussed and decided last year. Of course, because the exposure levels for many of these other devices and exposure situations are so much lower than the exposure to someone who has a functioning cell phone against her/his ear, the risk will be considerably less (although the hazard still exists).

I hope this is sufficiently clear to be useful. Thank you for your interest in our work.

Sincerely yours,

Robert A Baan PhD  
Responsible Officer, Monograph 102 on RF-EMF The IARC Monographs IARC,  
Lyon, FRANCE