#### RE: Power of Choice Submission – Stage 3 DSP Review EPR0022

#### To Whom It May Concern,

This submission relates to the aspect of the Draft proposal concerning plans for roll out of a wireless smart grid for utilities. I wish to submit several documents relating to the potential negative public health impact of such a roll out.

The first document that I wish to draw attention to is European Parliament's Resolution 1815 of May last year. In this document, in light of a growing body of scientific research, Parliament officially recognises the harm potential to humans and the environment from non ionising radiation exposure and urges its member states to adopt the precautionary principle in reducing public exposure to wireless microwave radiation. They also urge countries to step up research into new technologies that are free from biological impacts. (See Clauses 4, 6 & 8.1.5) Note regret is also expressed at member states slow response to the warning of scientists.

The second document is Santa Cruz Public Health Department's health assessment report on smart meters, undertaken in January this year. The report identified some key health concerns in relation to wireless utility meters, resulting in calling for a moratorium on installation of the meters in that jurisdiction.

Third document is a 2002 letter from the U.S. EPA asserting that current FCC radiofrequency guidelines offer no protection against 'non thermal' effects.

Whilst the existence of certain non thermal effects was acknowledged at the time of setting of the current standards, such effects were not addressed by the standards due to poor understanding of their mechanism. It was however noted that more research into these effects should be undertaken. Since then a growing body of research has confirmed earlier findings such as blood brain barrier disruption, as well as effects on DNA, immune system, reproduction, cancer promotion and concerns have been raised about impacts for children, pregnant women and the fetus. However standards have not yet been updated to protect against these important non thermal effects and existing guidelines continue to be quoted though scientists warn that they are obsolete as the actual science no longer supports them,

The fourth document, a report by Biomedical Engineer Dr.Karl Maret, documents some of the non thermal research concerns in its relevance to smart meters.

Fifth document is a 2004 patent application by Swiss telecommunications company, Swisscom, which states that as of the date of that application, sufficient research existed to indicate harmful effects to human health from exposure to WLAN devices that remain on standby.

For over a decade, warning scientists have been predicting increases in cancers and neurodegenerative diseases if we fail to address the need for biologically based safety standards. The incidence of certain cancers has certainly increased in the last decade since the widespread propagation of wireless technology. We are now told that 1 in 2 will get cancer and there have also been recent unexplained increases in earlier onset of neurodegenerative disease and large increases in the incidence of autism.

The roll out of smart meters on people's homes as part of a mesh microwave grid with its associated tower infrastructure will only further increase public exposure to non ionising radiation and directly fly in the face of last years urging for caution by European Parliament. Such a plan is also at odds with the World Health Organisation's official recognition of non ionising microwave radiation from wireless devices as a possible carcinogen in May last year.

It is noteworthy that there have been widespread complaints of adverse health effects from people who have had smart meters installed on their homes in overseas countries, however it is also important to note that expert Dr. Blank has found RF radiation at levels many times beneath the current FCC standard elicits an automatic cellular stress response independent of the issue of EHS and whether or not one experiences symptoms.

It would be a gross disservice to the Australian public to risk public health by continuing plans to roll out wireless utility meters in the face of warnings, when such potential for disease and environmental harm could be avoided by the use of fibre optic alternatives. Surely forward thinking plans for progress should take into account important impact for public health, particularly given that this has financial impact in itself.

I have included a tiny sample of papers from a vast body of research relating to health effects of non ionising microwave radiation in order to illustrate just some of the concerns relating to non ionising radiation exposure associated with wireless technology.

Please see list of attachments below:

- 1. Resolution 1815 urging precautionary principle public radiofrequency exposure.
- 2. Santa Cruz Public Health Department report on wireless Smart Meter health issues.
- 3. EPA letter confirming inadequacy of current FCC radiofrequency exposure guidelines to protect against non thermal effects.
- 4. Biomedical Engineer Dr. Karl Maret's report on health impact of radiofrequency from smart meters.
- 5. Swisscom patent application acknowledging harm from RF exposure from WLAN devices operating in standby mode.
- 6. Two papers dealing with effects of cell phone radiation on blood brain barrier and brain function (wherein researchers found maximum disruption to BBB actually occurred at a surprising distance of around 1m from cell phone antenna).
- 7. Research paper on Cell phones and brain tumours.
- Research abstract whole body exposure to mobile phone/DECT base radiation: <u>http://www.ncbi.nlm.nih.gov/pubmed/22263702</u> (No PDF)
- 9. Research paper ROS Formation & Apoptosis via 900MHz Mobile Phone Radiation.
- 10. Oncology research review citing urgent need for review of current standards.

Also see video links with important relevance to Government, featuring the addresses of expert scientists relating to research findings and the inadequacy of current FCC guidelines to protect public health:

- 1. Dr. Ted Litovitz address to U.S. Congress in 2001 re serious inadequacy of current RF safety standards to protect public health: <u>http://www.youtube.com/watch?v=6lAFbQqyVio</u>
- 2. Dr. Martin Blank, Columbia University, speaking on urgent need for standard review: http://www.youtube.com/watch?v=a6wLFeIrCtU
- 3. Dr. Martin Blank, Columbia University, speaking on the cellular stress response: http://vimeo.com/17266941
- 4. Epidemiologist Dr. Devra Davis presenting research to other scientists at NIEHS earlier this year: <u>http://www.youtube.com/watch?v=a6wLFeIrCtU</u>
- I am also including this video featuring Consulting Engineer Rob States, since his video addresses concerns specific to wireless smart meter & grid. <u>http://www.youtube.com/watch?v=FLeCTaSG2-U</u>

Helen Weir (helx1@iprimus.com.au)





#### http://assembly.coe.int

### **Résolution 1815**

27 May 2011

# The potential dangers of electromagnetic fields and their effect on the environment

*Text adopted by the Standing Committee*, acting on behalf of the Assembly, on 27 May 2011 (see Doc. 12608, report of the Committee on the Environment, Agriculture and Local and Regional Affairs, rapporteur: Mr Huss).

1. The Parliamentary Assembly has repeatedly stressed the importance of states' commitment to preserving the environment and environmental health, as set out in many charters, conventions, declarations and protocols since the United Nations Conference on the Human Environment and the Stockholm Declaration (Stockholm, 1972). The Assembly refers to its past work in this field, namely Recommendation 1863 (2009) on environment and health, Recommendation 1947 (2010) on noise and light pollution, and more generally, Recommendation 1885 (2009) on drafting an additional protocol to the European Convention on Human Rights concerning the right to a healthy environment and Recommendation 1430 (1999) on access to information, public participation in environmental decision-making and access to justice – implementation of the Aarhus Convention.

2. The potential health effects of the very low frequency of electromagnetic fields surrounding power lines and electrical devices are the subject of ongoing research and a significant amount of public debate. According to the World Health Organisation, electromagnetic fields of all frequencies represent one of the most common and fastest growing environmental influences, about which anxiety and speculation are spreading. All populations are now exposed to varying degrees of to electromagnetic fields, the levels of which will continue to increase as technology advances.

3. Mobile telephony has become commonplace around the world. This wireless technology relies upon an extensive network of fixed antennas, or base stations, relaying information with radio frequency signals. Over 1.4 million base stations exist worldwide and the number is increasing significantly with the introduction of third generation technology. Other wireless networks that allow high-speed internet access and services, such as wireless local area networks, are also increasingly common in homes, offices and many public areas (airports, schools, residential and urban areas). As the number of base stations and local wireless networks increases, so does the radio frequency exposure of the population.

4. While electrical and electromagnetic fields in certain frequency bands have wholly beneficial effects which are applied in medicine, other non-ionising frequencies, be they sourced from extremely low frequencies, power lines or certain high frequency waves used in the fields of radar, telecommunications and mobile telephony, appear to have more or less potentially harmful, non-thermal, biological effects on plants, insects and animals as well as the human body even when exposed to levels that are below the official threshold values.

5. As regards standards or threshold values for emissions of electromagnetic fields of all types and frequencies, the Assembly recommends that the ALARA or "as low as reasonably achievable" principle is applied, covering both the so-called thermal effects and the athermic or biological effects of electromagnetic emissions or radiation. Moreover, the precautionary principle should be applicable when scientific evaluation does not allow the risk to be determined with sufficient certainty, especially given the context of growing exposure of the population, including particularly vulnerable groups such as young people and children, which could lead to extremely high human and economic costs of inaction if early warnings are neglected.

6. The Assembly regrets that, despite calls for the respect of the precautionary principle and despite all the recommendations, declarations and a number of statutory and legislative advances, there is still a lack of reaction to known or emerging environmental and health risks and virtually systematic delays in adopting and implementing effective preventive measures. Waiting for high levels of scientific and clinical proof before taking action to prevent well-known risks can lead to very high health and economic costs, as was the case with asbestos, leaded petrol and tobacco.

7. Moreover, the Assembly notes that the problem of electromagnetic fields or waves and the potential consequences for the environment and health has clear parallels with other current issues, such as the licensing of medication, chemicals, pesticides, heavy metals or genetically modified organisms. It therefore highlights that the issue of independence and credibility of scientific expertise is crucial to accomplish a transparent and balanced assessment of potential negative impacts on the environment and human health.

8. In light of the above considerations, the Assembly recommends that the member states of the Council of Europe:

8.1. in general terms:

8.1.1. take all reasonable measures to reduce exposure to electromagnetic fields, especially to radio frequencies from mobile phones, and particularly the exposure to children and young people who seem to be most at risk from head tumours;

8.1.2. reconsider the scientific basis for the present electromagnetic fields exposure standards set by the International Commission on Non-Ionising Radiation Protection, which have serious limitations and apply "as low as reasonably achievable" (ALARA) principles, covering both thermal effects and the athermic or biological effects of electromagnetic emissions or radiation;

8.1.3. put in place information and awareness-raising campaigns on the risks of potentially harmful long-term biological effects on the environment and on human health, especially targeting children, teenagers and young people of reproductive age;

8.1.4. pay particular attention to "electrosensitive" persons suffering from a syndrome of intolerance to electromagnetic fields and introduce special measures to protect them, including the creation of wave-free areas not covered by the wireless network;

8.1.5. in order to reduce costs, save energy, and protect the environment and human health, step up research on new types of antennas and mobile phone and DECT-type devices, and encourage research to develop telecommunication based on other technologies which are just as efficient but have less negative effects on the environment and health;

8.2. concerning the private use of mobile phones, DECT phones, WiFi, WLAN and WIMAX for computers and other wireless devices such as baby phones:

8.2.1. set preventive thresholds for levels of long-term exposure to microwaves in all indoor areas, in accordance with the precautionary principle, not exceeding 0.6 volts per metre, and in the medium term to reduce it to 0.2 volts per metre;

8.2.2. undertake appropriate risk-assessment procedures for all new types of device prior to licensing;

8.2.3. introduce clear labelling indicating the presence of microwaves or electromagnetic fields, the transmitting power or the specific absorption rate (SAR) of the device and any health risks connected with its use;

8.2.4. raise awareness on potential health risks of DECT-type wireless telephones, baby monitors and other domestic appliances which emit continuous pulse waves, if all electrical equipment is left permanently on standby, and recommend the use of wired, fixed telephones at home or, failing that, models which do not permanently emit pulse waves;

8.3. concerning the protection of children:

8.3.1. develop within different ministries (education, environment and health) targeted information campaigns aimed at teachers, parents and children to alert them to the specific risks of early, ill-considered and prolonged use of mobiles and other devices emitting microwaves;

8.3.2. for children in general, and particularly in schools and classrooms, give preference to wired Internet connections, and strictly regulate the use of mobile phones by schoolchildren on school premises;

8.4. concerning the planning of electric power lines and relay antenna base stations:

8.4.1. introduce town planning measures to keep high-voltage power lines and other electric installations at a safe distance from dwellings;

8.4.2. apply strict safety standards for sound electric systems in new dwellings;

8.4.3. reduce threshold values for relay antennas in accordance with the ALARA principle and install systems for comprehensive and continuous monitoring of all antennas;

8.4.4. determine the sites of any new GSM, UMTS, WiFi or WIMAX antennas not solely according to the operators' interests but in consultation with local and regional government officials, local residents and associations of concerned citizens;

8.5. concerning risk assessment and precautions:

8.5.1. make risk assessment more prevention oriented;

8.5.2. improve risk-assessment standards and quality by creating a standard risk scale, making the indication of the risk level mandatory, commissioning several risk hypotheses and considering compatibility with real life conditions;

8.5.3. pay heed to and protect "early warning" scientists;

8.5.4. formulate a human rights oriented definition of the precautionary and ALARA principles;

8.5.5. increase public funding of independent research, *inter alia* through grants from industry and taxation of products which are the subject of public research studies to evaluate health risks;

8.5.6. create independent commissions for the allocation of public funds;

8.5.7. make the transparency of lobby groups mandatory;

8.5.8. promote pluralist and contradictory debates between all stakeholders, including civil society (Aarhus Convention).



### **County of Santa Cruz**

#### **COUNTY ADMINISTRATIVE OFFICE**

701 OCEAN STREET, SUITE 520, SANTA CRUZ, CA 95060-4073 (831) 454-2100 FAX: (831) 454-3420 TDD: (831) 454-2123 SUSAN MAURIELLO, J.D., COUNTY ADMINISTRATIVE OFFICER

January 18, 2012

AGENDA: January 24, 2012

Board of Supervisors County of Santa Cruz 701 Ocean Street Santa Cruz, California 95060

SmartMeter Moratorium

Dear Members of the Board:

On December 13, 2011, your Board directed this office to return today with a report on issues associated with the current SmartMeter moratorium ordinance, and information on the possible extension of the moratorium for an additional year. Your Board also directed the Public Health Officer to return with an analysis of the research on the health effects of SmartMeters, and directed County Counsel to return with a report regarding the legality of a public utility refusing service to customers who are willing to pay for service and are willing to have an analog meter.

As your Board is aware, the California Public Utility Commission is considering PG&E's application for modification to PG&E's SmartMeter proposal to include an option for residential customers who do not wish to have a wireless SmartMeter. The item was scheduled on the January 12, 2012 agenda, but the commission anticipates that a vote on the proposal will not happen prior to February 1, 2012.

#### Moratorium Ordinance

Your Board has heard significant amounts of testimony regarding SmartMeters and concerns about their possible impact on health, questions about their accuracy, their inability to recover real-time data, privacy concerns, and the lack of safety standards for chronic long-term exposure to electromagnetic frequency radiation. In addition, PG&E has not presented studies to support their primary justification that the SmartMeter program will encourage customers to more effectively manage their utilization of electricity.

Given the broad concern about SmartMeter technology and your Board's desire to go on record, this office and County Counsel believe that notwithstanding the enforcement challenges, that it is in the best interest of public health, safety, and welfare for your Board to adopt the attached ordinance (Attachment A) implementing a temporary moratorium on the installation of SmartMeters in or on any home, apartment, condominium or business within the unincorporated area of the County. The purpose of the moratorium is to allow additional time to educate the CPUC about these concerns and allow time for adequate study of the impacts resulting from the SmartMeter technology.

SERVING THE COMMUNITY - WORKING FOR THE FUTURE

0249

Ordinance Imposing Temporary Moratorium on Installation of SmartMeters Agenda: January 24, 20012

PG&E, asserting that local governments do not have jurisdiction on the installation of the meters, has ignored the previous Santa Cruz County ordinance as well as similar ordinances adopted in other jurisdictions. PG&E believes that only the California Public Utilities Commission (CPUC) has the authority to stop installation of the meters. Elected representatives, including the Board of Supervisors of Marin County, have acknowledged the limits of their ordinances to actually stop the installation of the meters. However, jurisdictions have adopted their ordinances with statements that such ordinances play an important role by informing the CPUC of significant community concerns.

#### Health Officer Report

The Public Health Officer's report is provided as Attachment B. The report discusses the health risks associated with SmartMeters, the scientific reports and actions the public might take to mitigate potential harm.

#### PG&E Shutoff Update

At the December 13, 2011, meeting, your Board questioned the PG&E representative about the utility company's decision to shut off power to the homes of residents who removed their SmartMeters. Subsequent to that meeting, PG&E restored power to those residences with the intent of charging them based on past electrical bills.

#### Petition

At your January 10, 2012 meeting, your Board was presented with a petition to the California Public Utilities Commission regarding PG&E SmartMeter Opt-out Application, (Petition A.11-03-014). The petition provides the opportunity for local elected officials to urge the Commission to continue Petition A.11-03-014 for further public hearings. The petition is provided as Attachment C. It is recommended that your Board direct the Chair to sign the petition on behalf of the Board and submit it to the PUC.

#### IT IS THEREFORE RECOMMENDED THAT YOUR BOARD:

- Direct the Chair to send a letter to the PUC calling for independent testing and monitoring of SmartMeters in place to determine duty cycles and frequency, especially in the following circumstances
  - Where both gas and electric meters are located closely together
  - Where there is a bank of SmartMeters such as on a multi-family residential building or apartment building
  - Where there is a collector meter on a home that serves the home, plus as many as 5000 other residential units in the area
  - Where a SmartMeter on a home acts as a relay for other local neighborhood meters

- (2) Direct the Chair to send a letter to the PUC and PG&E allowing any Santa Cruz County resident to request removal of a previously installed SmartMeter and the replacement with an analog meter
- (3) Accept and file the report from the Public Health Officer
- (4) Direct the Chair to sign the petition to the California Public Utilities Commission on behalf of the Board urging the Commission to delay consideration of a preliminary decision on PG&E's SmartMeter application until further public hearing and input are completed, and
- (5) Adopt the attached ordinance imposing a temporary moratorium on the installation of SmartMeters within the unincorporated area of Santa Cruz County and direct the Clerk of the Board to place the ordinance on the February 7, 2012 agenda for final consideration.

Very truly yours.

SUSAN A. MAURIELLO County Administrative Officer Attachments:

- A. Proposed Ordinance
- B. Report from Public Health Officer
- C. Petition to CPUC
- cc: PG&E California Public Utilities Commission

Attachment A

0252

#### ORDINANCE NO.

#### AN UNCODIFIED ORDINANCE OF THE COUNTY OF SANTA CRUZ IMPOSING A TEMPORARY MORATORIUM ON THE INSTALLATION OF SMARTMETERS AND RELATED EQUIPMENT IN, ALONG, ACROSS, UPON, UNDER AND OVER THE PUBLIC STREETS AND OTHER PLACES WITHIN THE UNINCORPORATED AREA OF SANTA CRUZ COUNTY

The Board of Supervisors of the County of Santa Cruz find as follows:

WHEREAS, the County of Santa Cruz (the "County"), through its police powers granted by Article XI of the California Constitution, retains broad discretion to legislate for public purposes and for the general welfare, including but not limited to matters of public health, safety and consumer protection; and

WHEREAS, the County of Santa Cruz has a franchise agreement with PG&E that has been in effect since 1955; and

WHEREAS, in addition, the County retains authority under Article XII, Section 8 of the Constitution to grant franchises for public utilities, and pursuant to California Public Utilities Code section 6203, "may in such a franchise impose such other and additional terms and conditions..., whether governmental or contractual in character, as in the judgment of the legislative body are to the public interest;" and

WHEREAS, Public Utilities Code section 2902 reserves the County's right to supervise and regulate public utilities in matters affecting the health, convenience and safety of the general public, "such as the use and repair of public streets by any public utility, the location of the poles, wires, mains, or conduits of any public utility, on, under, or above any public streets, and the speed of common carriers operating within the limits of the municipal corporation;" and

WHEREAS, Pacific Gas & Electric Company ("PG&E") is now installing SmartMeters in Central and Northern California and is installing these meters within the County of Santa Cruz; and

WHEREAS, concerns about the impact and accuracy of SmartMeters have been raised nationwide, leading the Maryland Public Service Commission to deny permission on June 21, 2010 for the deployment of SmartMeters in that state. The State of Hawaii Public Utility Commission also recently declined to adopt a smart grid system in that state. The CPUC currently has pending before it a petition from the City and County of San Francisco, and other municipalities, seeking to delay the implementation of SmartMeters until the questions about their accuracy can be evaluated; and

WHEREAS, major problems and deficiencies with SmartMeters in California have been brought to the attention of the Board of Supervisors of the County of Santa Cruz, including PG&E's confirmation that SmartMeters have provided incorrect readings costing ratepayers untold thousands of dollars in overcharges and PG&E's records outlined "risks" and "issues" including an ongoing inability to recover real-time data because of faulty hardware originating with PG&E vendors; and

WHEREAS, the ebb and flow of gas and electricity into homes discloses detailed information about private details of daily life. Energy usage data, measured moment by moment, allows the reconstruction of a household's activities: when people wake up, when they come home, when they go on vacation, and even when they take a hot bath. SmartMeters represent a new form of technology that relays detailed hitherto confidential information reflecting the times and amounts of the use of electrical power without adequately protecting that data from being accessed by unauthorized persons or entities and as such pose an unreasonable intrusion of utility customers' privacy rights and security interests. Indeed, the fact that the CPUC has not established safeguards for privacy in its regulatory approvals may violate the principles set forth by the U.S. Supreme Court in *Kyllo v. United States* (2001), 533 U.S. 27; and

WHEREAS, significant health questions have been raised concerning the increased electromagnetic frequency radiation (EMF) emitted by the wireless technology in SmartMeters, which will be in every house, apartment and business, thereby adding additional human-made EMF to our environment around the clock to the already existing EMF from utility poles, individual meters and telephone poles; and

WHEREAS, FCC safety standards do not exist for chronic long-term exposure to EMF or from multiple sources, and reported adverse health effects from electromagnetic pollution include sleep disorders, irritability, short term memory loss, headaches, anxiety, nausea, DNA breaks, abnormal cell growth, cancer, premature aging, etc. Because of untested technology, international scientists, environmental agencies, advocacy groups and doctors are calling for the use of caution in wireless technologies; and

WHEREAS, the primary justification given for the SmartMeters program is the assertion that it will encourage customers to move some of their electricity usage from daytime to evening hours; however, PG&E has conducted no actual pilot projects to determine whether this assumption is in fact correct. Nontransmitting time-of-day meters are already available for customers who desire

2

4]

them, and enhanced customer education is a viable non-technological alternative to encourage electricity use time shifting. Further, some engineers and energy conservation experts believe that the SmartMeters program--in totality--could well actually increase total electricity consumption and therefore the carbon footprint; and

WHEREAS, this Board of Supervisors sent a letter to the CPUC on September 15, 2010 expressing concern about reports that SmartMeter technology was interfering with the proper functioning of common household devices and requesting a response from the CPUC; and

WHEREAS, there has been no response by the CPUC to the letter sent by the Board of Supervisors; and

WHEREAS, because the potential risks to the health, safety and welfare of County residents are so great, the Board of Supervisors wishes to adopt a moratorium on the installation of SmartMeters and related equipment within the unincorporated area of the County of Santa Cruz. The moratorium period will allow the Council on Science and Technology and legislative process referenced above to be completed and for additional information to be collected and analyzed regarding potential problems with SmartMeters; and

WHEREAS, there is a current and immediate threat to public health, safety and welfare because, without this urgency ordinance, SmartMeters or supporting equipment will be installed or constructed or modified in the County without PG&E's complying with the CPUC process for consultation with the local jurisdiction, the County's Code requirements, and subjecting residents of Santa Cruz County to the privacy, security, health, accuracy and consumer fraud risks of the unproven SmartMeter technology; and

WHEREAS, the Board of Supervisors hereby finds that it can be seen with certainty that there is no possibility that the adoption and implementation of this Ordinance may have a significant effect on the environment. This Ordinance does not authorize construction or installation of any facilities and, in fact, imposes greater restrictions on such construction and installation in order to protect the public health, safety and general welfare. This Ordinance is therefore exempt from the environmental review requirements of the California Environmental Quality Act (CEQA) pursuant to Section 15061(b)(3) of Title 14 of the California Code of Regulations; and

WHEREAS, there is no feasible alternative to satisfactorily study the potential impact identified above as well or better with a less burdensome or restrictive effect than the adoption of this interim urgency moratorium ordinance; and

WHEREAS, based on the foregoing it is in the best interest of public health, safety and welfare to allow adequate study of the impacts resulting from the SmartMeter technology; therefore it is appropriate to adopt a temporary moratorium that would remain in effect from the date of its adoption until December 31, 2012, unless your Board acts to repeal it prior to that date.

**NOW, THEREFORE BE IT ORDAINED** by the Board of Supervisors of the County of Santa Cruz as follows:

#### **SECTION I**

Moratorium. From and after the effective date of this Ordinance, no SmartMeter may be installed in or on any home, apartment, condominium or business of any type within the unincorporated area of the County of Santa Cruz, and no equipment related to SmartMeters may be installed in, on, under, or above any public street or public right of way within the unincorporated area of the County of Santa Cruz.

#### **SECTION II**

Violations of the Moratorium may be charged as infractions or misdemeanors as set forth in Chapter 1.12 of the Santa Cruz County Code. In addition, violations may be deemed public nuisances, with enforcement by injunction or any other remedy authorized by law.

#### **SECTION III**

This Board of Supervisors finds and determines that: (a) there is a current and immediate threat to the public peace, health, or safety; (b) the moratorium must be imposed in order to protect and preserve the public interest, health, safety, comfort and convenience and to preserve the public welfare; and (c) it is necessary to preserve the public health and safety of all residents or landowners adjacent to such uses as are affected by this interim ordinance as well as to protect all of the citizens of Santa Cruz County by preserving and improving the aesthetic and economic conditions of the County.

#### **SECTION IV**

If any provision of this interim ordinance is held to be unconstitutional, it is the intent of the Board of Supervisors that such portions of such ordinance are severable from the remainder and the remainder is given full force and effect.

0255

41

#### **SECTION V**

This interim ordinance is not subject to the California Environmental Quality Act (CEQA) pursuant to Section 15060(c) (2) – the activity will not result in a direct or reasonably foreseeable indirect physical change in the environment and Section 15060(c) (3) – the activity is not a project as defined in Section 15378 of the CEQA Guidelines, because it has no potential for resulting in physical change to the environment, directly or indirectly.

#### **SECTION VI**

This ordinance shall take effect on the 31<sup>st</sup> day after the date of final passage.

PASSED AND ADOPTED THIS \_\_\_\_\_ day of \_\_\_\_\_, 2012, by the Board of Supervisors of the County of Santa Cruz by the following vote:

AYES:	SUPERVISORS
NOES:	SUPERVISORS
ABSENT:	SUPERVISORS
ABSTAIN:	SUPERVISORS

Chairperson of the Board of Supervisors

Attest:

Clerk of the Board

APPROVED AS TO FORM:

County Counse



Attachment B



## County of Santa Cruz 0257

#### HEALTH SERVICES AGENCY

POST OFFICE BOX 962, 1060 EMELINE AVE., SANTA CRUZ, CA 95061-0962 TELEPHONE: (831) 454-4114 FAX: (831) 454-5049 TDD: (831) 454-4123

Poki Stewart Namkung, M.D., M.P.H. Health Officer Public Health Division

#### Memorandum

Date: January 13, 2012

To: Santa Cruz County Board of Supervisors

From: Poki Stewart Namkung, M.D., M.P.H. PM Health Officer

Subject: Health Risks Associated With SmartMeters

#### <u>Overview</u>

On December 13, 2011, Santa Cruz County Board of Supervisors directed the Public Health Officer to return on January 24, 2012, with an analysis of the research on the health effects of SmartMeters.

#### Background

In order to analyze the potential health risks associated with SmartMeters, the following questions should be asked:

- 1) What is the SmartMeter system and what is the potential radiation exposure from the system?
- 2) What scientific evidence exists about the potential health risks associated with SmartMeters?
- 3) Are there actions that the public might take to mitigate any potential harm from SmartMeters?

SmartMeters are a new type of electrical meter that will measure consumer energy usage and send the information back to the utility by a wireless signal in the form of pulsed frequencies within the 800 MHz to 2400MHz range, contained in the microwave portion of the electromagnetic spectrum. SmartMeters are considered part of 'smart grid' technology that includes: a) a mesh network or series of pole-mounted wireless antennas at the neighborhood level to collect and transmit wireless information from all SmartMeters in that area back to the utility; b) collector meters, which are a special type of SmartMeter that collects the radiofrequency or microwave radiation signals from many surrounding Health Risks Associated With SmartMeters Agenda: January 24, 2012 Page 2 of 8

buildings (500-5000 homes or buildings) and sends the information back to the utility; and c) proposed for the future, a power transmitter to measure the energy use of individual appliances (e.g. washing machines, clothes dryers, dishwasher, etc) and send information via wireless radio frequency signal back to the SmartMeter. The primary rationale for SmartMeters and grid networks is to more accurately monitor and direct energy usage.

The public health issue of concern in regard to SmartMeters is the involuntary exposure of individuals and households to electromagnetic field (EMF) radiation. EMFs are everywhere, coming from both natural and man-made sources. The three broad classes of EMF are:

- extremely low frequency, ELF (from the sun or powerlines)
- radio frequency, RF (from communication devices, wireless devices, and SmartMeters)
- extremely high frequency, known as ionizing radiation (x-rays and gamma rays)

Much of this exposure is beyond our control and is a matter of personal choice; however, public exposure to RF fields is growing exponentially due to the proliferation of cell phones, and wireless fidelity (Wi-Fi) technology. To understand the relationship between EMF from SmartMeters and other sources, it is helpful to view the electromagnetic spectrum:

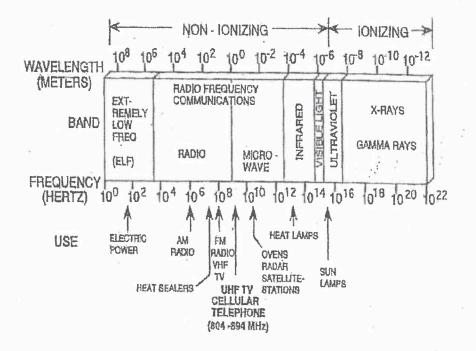


Fig. 1: The electromagnetic spectrum, showing the relation s between ELF and RF fields, wavelength and frequency, and the ionizing and non-ionizing portions of the spectrum.

The Federal Communications Commission (FCC) has adopted limits for Maximum Permissable Exposure (MPE) that are based on exposure guidelines published by the National Council on Radiation Protection and Measurements (NCRP). The limits vary with

#### Health Risks Associated With SmartMeters Agenda: January 24, 2012 Page 3 of 8

Attachment B

0259

the frequency of the electromagnetic radiation and are expressed in units of microwatts per centimeter squared. A SmartMeter contains two antennas whose combined timeaveraged public safety limit of exposure is 655µW/cm² (Sage, 2011). According to the California Council on Science and Technology (CCST) Report (2011), within distances of three to ten feet, SmartMeters would not exceed this limit. However, CCST did not account for the frequency of transmissions, reflection factors, banks of SmartMeters firing simultaneously, and distances closer than three feet. There are numerous situations in which the distance between the SmartMeters and humans is less than three feet on an ongoing basis, e.g. a SmartMeter mounted on the external wall to a bedroom with the bed placed adjacent to that mounting next to the internal wall. That distance is estimated to be one foot. The CCST Report also states that SmartMeters will generally transmit data once every four hours, and once the grid is fully functional, may transmit "more frequently." It has been aptly demonstrated by computer modeling and real measurement of existing meters that SmartMeters emit frequencies almost continuously, day and night, seven days a week. Furthermore, it is not possible to program them to not operate at 100% of a duty cycle (continuously) and therefore it should not be possible to state that SmartMeters do not exceed the time-averaged exposure limit. Additionally, exposure is additive and consumers may have already increased their exposures to radiofrequency radiation in the home through the voluntary use of wireless devices such as cell and cordless phones. personal digital assistants (PDAs), routers for internet access, home security systems. wireless baby surveillance (baby monitors) and other emerging devices. It would be impossible to know how close a consumer might be to their limit, making safety a uncertainty with the installation of a mandatory SmartMeter.

This report will focus on the documented health risks of EMF in general, the relevance of that data to SmartMeters exposure, the established guidelines for RF safety to the public at large, and then provide recommendations to ameliorate the risk to the public's health.

#### Evidence-based Health Risks of EMFs

There is no scientific literature on the health risks of SmartMeters in particular as they are a new technology. However, there is a large body of research on the health risks of EMFs. Much of the data is concentrated on cell phone usage and as SmartMeters occupy the same energy spectrum as cell phones and depending on conditions, can exceed the whole body radiation exposure of cell phones phones (see Attachment B1, Figure 4). In terms of health risks, the causal factor under study is RF radiation whether it be from cell phones, Wi-Fi routers, cordless phones, or SmartMeters. Therefore all available, peer-reviewed, scientific research data can be extrapolated to apply to SmartMeters, taking into consideration the magnitude and the intensity of the exposure.

Since the mid-1990's the use of cellular and wireless devices has increased exponentially exposing the public to massively increased levels of RF. There is however, debate regarding the health risks posed to the public given these increased levels of radiation. It must be noted that there is little basic science funding for this type of research and it is largely funded by industry. An intriguing divide, noted by Genuis, 2011 is that most

Health Risks Associated With SmartMeters Agenda: January 24, 2012 Page 4 of 8 Attachment B

0260

research carried out by independent non-government or non-industry affiliated researchers suggests potentially serious effects from many non-ionizing radiation exposures; most research carried out by independent non-government or non-industry affiliated researchers suggests potentially serious effects from many non-ionizing radiation exposures research funded by industry and some governments seems to cast doubt on the potential for harm. Elements of the controversy stem from inability to replicate findings consistently in laboratory animal studies. However, analysis of many of the conflicting studies is not valid as the methodology used is not comparable. Despite this controversy, evidence is accumulating on the results of exposure to RF at non-thermal levels including increased permeability of the blood-brain barrier in the head (Eberhardt, 2008), harmful effects on sperm, double strand breaks in DNA which could lead to cancer genesis (Phillips, 2011), stress gene activation indicating an exposure to a toxin (Blank, 2011), and alterations in brain glucose metabolism (Volkow, 2011).

In terms of meta-analyzed epidemiological studies, all case–control epidemiological studies covering >10 years of cell phone use have reported an increased risk of brain tumors from the use of mobile phones (Hallberg, 2011). Other studies have pointed to an increasing risk of acoustic neuroma, salivary gland tumors, and eye cancer after several years of cell phone use and the tumors occur predominantly on the same side of the head as the phone is used. The analysis of brain cancer statistics since the mid 20<sup>th</sup> century in several countries reveals that brain tumor formation has a long latency time, an average of over 30 years to develop from initial damage.(Hallberg, 2011). Therefore using studies such as the Interphone Study which looked as shorter latency periods for the development of specific brain cancers will result in inconclusive data.

Another potential health risk related to EMF exposure, whose legitimacy as a phenomen remains contentious, is electromagnetic hypersensitivity (EHS). In the 1950's, various centers in Eastern Europe began to describe and treat thousands of workers, generally employed in jobs involving microwave transmission. The afflicted individuals often presented with symptoms such as headaches, weakness, sleep disturbance, emotional instability, dizziness, memory impairment, fatigue, and heart palpitations. Clinical research to verify the physiological nature of this condition did not begin in earnest until the 1990's and found that the EMF involved was usually within the non-ionizing range of the electromagnetic spectrum. In the early 2000's, estimates of the occurrence of EHS began to swell with studies estimating the prevalence of this condition to be about 1.5% of the population of Sweden (Hilleert et al., 2002), 3.2% in California (Levallios et al., 2002), and 8% in Germany (infas Institut fur angewandte Sozialwissenschaft GmbH, 2003).

In 2004, WHO declared EHS "a phenomenon where individuals experience adverse health effect while using or being in the vicinity of devices emanating electric, magnetic, or electromagnetic fields (EMFs)...Whatever its cause, EHS is a real and sometimes debilitating problem for the affected persons (Mild et al., 2004)."

Currently, research has demonstrated objective evidence to support the EHS diagnosis, defining pathophysiological mechanisms including immune dysregulation in vitro, with

Attachment B

Health Risks Associated With SmartMeters Agenda: January 24, 2012 Page 5 of 8

0261

increased production of selected cytokines and disruption and dysregulation of catecholamine physiology (Genuis, 2011).

Until recently, the diagnosis of EHS has not received much support from the medical community due to lack of objective evidence. In an effort to determine the legitimacy of EHS as a neurological disorder, however, a collection of scientists and physicians recently conducted a double-blinded research study that concluded that "EMF hypersensitivity can occur as a bona fide environmentally-inducible neurological syndrome (McCarty et al., 2011).

#### **Safety Guidelines**

The guidelines currently used by the FCC were adopted in 1996, are thermally based, and are believed to protect against injury that may be caused by acute exposures that result in tissue heating or electric shock. FCC guidelines have a much lower certainty of safety than standards. Meeting the current FCC guidelines only assures that one should not have heat damage from SmartMeter exposure. It says nothing about safety from the risk of many chronic diseases that the public is most concerned about such as cancer, miscarriage, birth defects, semen quality, autoimmune diseases, etc. Therefore, when it comes to nonthermal effects of RF, FCC guidelines are irrelevant and cannot be used for any claims of SmartMeter safety unless heat damage is involved (Li, 2011).

There are no current, relevant public safety standards for pulsed RF involving chronic exposure of the public, nor of sensitive populations, nor of people with metal and medical implants that can be affected both by localized heating and by electromagnetic interference (EMI) for medical wireless implanted devices. Many other countries (9) have significantly lower RF/MW exposure standards ranging from 0.001 to 50  $\mu$ W/cm<sup>2</sup> as compared with the US guideline of 200-1000  $\mu$ W/cm<sup>2</sup>. Note that these recommended levels are considerably lower that the approximately 600  $\mu$ W/cm<sup>2</sup>. (time-averaged) allowed for the RFR from SmartMeters operating in the low 900 MHz band mandated by the FCC based on only thermal consideration.

In summary, there is no scientific data to determine if there is a safe RF exposure level regarding its non-thermal effects. The question for governmental agencies is that given the uncertainty of safety, the evidence of existing and potential harm, should we err on the side of safety and take the precautionary avoidance measures? The two unique features of SmartMeter exposure are: 1) universal exposure thus far because of mandatory installation ensuring that virtually every household is exposed; 2) involuntary exposure whether one has a SmartMeter on their home or not due to the already ubiquitous saturation of installation in Santa Cruz County. Governmental agencies for protecting public health and safety should be much more vigilant towards involuntary environmental exposures because governmental agencies are the only defense against such involuntary exposure to electromagnetic radiation can be found in Attachment B2.

0262

#### References:

Balmori, A. "Electromagnetic Pollution from Phone Masts. Effects of Wildlife." <u>Pathophysiology</u> (2009).

Blackman, C. "Cell Phone Radiation: Evidence from ELF and RF studies supporting more inclusive risk identifiation assessment,." <u>Pathophysiology</u> (2009): doi: 10.1016.

-. "Cell Phone Radiation: Evidence from ELF and RF Studies Supporting More Inclusive Risk Identification Assessment." <u>Pathophysiology</u> (2009).

Blank, M, Goodman R. "Electromagnetic field stress living cells."

Pathophysiology (2009): doi: 10.1016.

Blank, M. "Prefice." <u>Pathophysiology</u> (2009): doi10.1016.

Carpenter, D. and Sage, C. "BioInitiave Report: A Rationale for a Biologicallybased Public Exposure Standard for Electromagnetic Fields." (2007).

Carpenter, David O. "Electromagnetic Fields and Cancer: The Cost of Doing Nothing." (2009).

Carpenter, David O. " Report on the CCST document "Health Impacts of Radiofrequency from Smart Meters"." (n.d.).

Carpenter, David O. Sage Cindy. "Setting Prudent Public Health Policy for Electromagnetic Field Exposures." <u>Reviews on Environmental Health (2008)</u>: Vol. 23 No.2.

Consultants, Sage Associates - Environmental. "Assessments of Radiofrequency Microwave Radiation Emmissions from SmartMeters." (2011).

Davanipour, E. Sobel. "Long Term Exposure to magnetic fields and the risks of Alzheimer's disease and breast cancer." <u>Pathophysiology</u> (2009): doi: 10.1016. De-Kun Li, MD PhD MPH. "Repsonse to CCST." <u>Written Testimony</u> (2009).

Genuis SJ, Lipp CT. "Electromagnetic Sensitivity: Fact or Fiction?" <u>Sci total</u> <u>Environ</u> (2011): doi: 10.1016.

Goldworthy, Andrew. "The Biological Effect of Weak Electronmagnetic Fields." (2007).

Hallberg O, and Morgan J. "The Potential Impact of Mobile Phone Use on trends in Brain and CNS Tumors." <u>Neuro and Neurophysiology</u> (2011).

Hallberg, O et. al.,. "Apparent decreases in Swedish Public Health indicators after 1997-Are they due to improved diagnostic or environmental factors?" Pathophysiology (2009): doi: 10.1016.

Hankin, Norbert EPA. "Response to Janet Newton EMR Network re: Radiofrequency Guidelines." (2002).

Hardell, L. et al.,. "Epidemiological eveidence for an association between use of wireless phones and tumor diseases." <u>Pathophysiology</u> (2009): doi: 10.1016. Hillert, L et al.,. "Prevalence of self-reported hypersensitivity to electric or magnetic fields in a population-based questionnaire survey." <u>Scab J Work</u> <u>Environ Health 28</u> (2002): 33-41.

#### Health Risks Associated With SmartMeters Agenda: January 24, 2012 Page 7 of 8

Hirsch, Daniel. "Comments on the Draft Report by the Council on Science and Technology "Health Impacts of Radio frequency from Smart Meters"." (2011). Hondou, Tsuyoshi. "Passive exposure to Mobile Phones: Enhancement of Intensity by Reflection." (2006).

Huttunen, P. et al., "FM-radio and TV tower signals can cause spontaneous hand movements near moving RF relflector." <u>Pathophysiology</u> (2009): doi: 10.1016.

Infas. "Study on concern and anxiety of the general public with respect to the possible risks due to high frequency electromagnetic fields used." (2004). Johannsson, Ollie Proffessor Dept of Neuroscience, Karolinksa Institute Stockholm, Sweden. "Commentary." (2011).

Khurana, Vini G. et al.,. "Cell phones and brain tumors: A review including the long-term epidemiologic data." <u>Science Direct, Surgical Direct, Surgical</u> <u>Neurology</u> (2009).

Kreutzer, Rick CDPH. "Technical Commentary on CCST Report: Health Impact on Radio Frequencies from SmartMeters." (2011).

Kundi, M., Hutter MP. "Mobile Phone base stations-Effects on wellbeing and health." <u>Pathophysiology</u> (2099): doi:10.1016.

Lai, Henry Dept. of Bioengineering Univ. Of Washington. "Biological Effects of Radiofrequency Radion." (2002).

Levallois, P and et al. "Study of self-reported hypersensitivity to electromagnet fields in California." <u>Environ Health Perspect</u> (2002): 110 (Suppl 4); 619-23. Levis, Angelo G. et al. "Mobile phones and head tumors. The discrepancies in cause-effect relationships in the epidemiological studies-how do they arise?" <u>Environmental Health</u> (2011).

Lotz, W. Gregory. "Letter to Richard Tell in support of RF exposure guidelines." (n.d.): 1999.

Maret, Dr. Karl. "Commentary on the CCST report " Health Impacts of Radio Frequency from Smart Meters"." (2011).

Mauer, Sandy EMF Network. "PG&E SmartMeters violate FCC RF Exposure Complinace Rates." (2010).

McCarty, DE et al.,. "Electromagnetic hypersentivity: Evidence for a novel neurological syndrome." Int. J Neurosci (2011).

Mekaya, MA et al., Dept of Biophysics University Anakara, Turkey. "Pulse modulated 900 Mhz radiation induces hypothyroidism and apoptosis in thyroid cells: a light, electron microscopy and immunohistochemical study." (2010). Mild, Kjell Hansson and Emilie van Dventer Paolo Ravazzani editors Mike Repacholi. "Electromagnetic Hypersensitivity - Proceedings International Workshop of EMF Hypersensitivity Prague, Czech Republic ." (2004). Neutra, Dr. Raymond Richard. "Commentary." (2011).

Organization, Word Health. "IARC Classifies radiofrequency electromagnetic fields as possible carcinogenic to humans." (2011).

0263

Health Risks Associated With SmartMeters Agenda: January 24, 2012 Page 8 of 8

Organization, World Health. "Electromagnetic fields and public health: Base stations and wireless technologies." <u>Fact Sheet 304 Accessed on January 31, 2011</u> (2006):

http://www.who.int/mediacentre/factsheets/fs304/enIndex/Html. —. "Electromagnetic fields and public health: Electromagnetic hypersensitivity." Fact Sheet No. 296 (2011):

http://who.who.int/mediacentre/factsheets/fs296/index.html.

-. "Interphone study reports on mobile phone use and brain cancer." (2010).
 Peevey, Michael. "Ruling and Scoping Memo to PUC: Opt out program and its cost." (2011).

Phillips J.L. et al. "Electromagnetic fields and DNA damage." <u>Pathophysiology</u> (2009): doi: 10.1016.

Pourlis, A.F. "Reproductive and developmental effects of EMF in vertebrate models." <u>Pathophysiology</u> (2009): doi: 10.1016.

Sage, C and Carpenter D. O. "Public health implications of wireless technologies." <u>Pathophysiology</u> (2011): 16: 233-246.

Schüz, Joachim et., al. "Cellular Phones and the Risks of Glioma and Meningioma." <u>American Journal of Epidemiology</u> (2006): doi: 10.1093 . Supervisors, Santa Cruz county Board of. "Temporary Moratorium on the Installation of SmartMeters." (2011).

Techology, California Council on Science and. "Health Impact of Radio Frequency Exposure from Smart Meters." (2011).

Tell, richard. "Summary Discussion of RF Fields and the PG&E SmartMeter System (2005 report and 2008 report)." (2009).

Volkow, N. D et al., "Effects of cell phone radiofrequency signal exposure on brain glucose metabolism." JAMA (2011): 305:808-13.

Yakemenko, I et al.,. "Long Term Exposure to Microwaves Provokes Cancer Growth: Evidences from Radar and Mobile Communications systems." <u>Experiemental Oncology</u> (2011).

Attachment B

#### Attachment B1

#### Figure 4 from Hirsch; 2011

0265

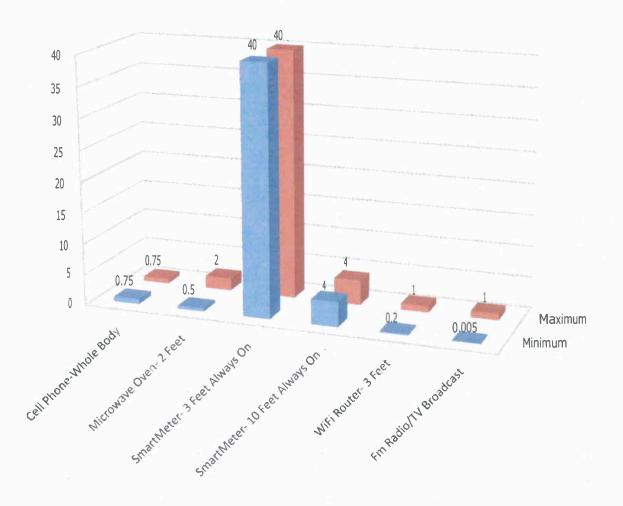


Figure 4. Comparison of Radio-Frequency Levels to the Whole Body from Various Sources in  $\mu$  W/cm<sup>2</sup> over time [corrected for assumed duty cycle and whole body exposure extrapolated fro m EPRI/CCST SmartMeter estimated levels at 3 feet].

0266

Attachment B2

Examples of strategies to reduce electromagnetic radiation.

$\langle \frown$		<b>A</b> 1	001	
(Gen	1110	C I	2011	11
LUTER	IIIS.	- D. I	ZU I	
	uio.	$\nabla \mathbf{v}_{1}$	201	11

Sources of adverse EMR	Considerations to reduce EMR exposure
Cell phones and cordless phones	• Minimize use of cell and cordless phones and <i>use</i> speaker phones when possible
	• Leave cell or cordless phone away from the body rather than in pocket or attached
Wireless internet	at the hip. • Use wired internet
	• Turn off the internet router when not in use (e.g. night-time)
	• Use power line network kits to achieve internet access by using existing wiring and avoiding wireless emissions.
Computers releasing high EMR	• Limit the amount of time spent working on a computer
	<ul> <li>Avoid setting a laptop computer on the lap</li> <li>Increase the distance from the transformer.</li> </ul>
	• Stay a reasonable distance away from the computer
Handheld electronics (electric toothbrus hair dryer, Smart phone, electronic tablet	• Limit the use of electronics and/or revert to using power-free devices
etc.)	<ul><li>Turn devices off before going to sleep</li><li>Minimize electronics in bedrooms</li></ul>
Fluorescent lights	<ul> <li>Consider using alternate lighting such as incandescent (Uncertainty exists about the safety of LED lights)</li> <li>Rely on natural sunlight for reading</li> </ul>
Household power	• Measure levels of EMR and modify
	<ul><li>exposures as possible</li><li>Avoid sleeping near sites of elevated EMR</li><li>Filters can be used to mitigate dirty power</li></ul>
High voltage power lines substations, transmission towers,	• Consider relocating to an area not in close proximity to high voltage power lines
and emitters (cell phone tower, radar, etc.)	<ul> <li>Maintain considerable distance from emitters</li> <li>Consider forms of shielding (shielding)</li> </ul>
	<ul> <li>Consider forms of shielding (shielding paints; grounded metal sheets)</li> <li>Increase size of neutral-wire to substation an</li> </ul>
Utility neutral-to-ground bonded to water pipes	install dielectric coupling in water pipe.

å

.

Petition to the California Public Utilities Commission Re: PG&E SmartMeter Opt-out Application, A.11-03-014

We the undersigned elected officials urge the Commission to delay consideration of President Peevey's preliminary decision until further public hearing and input are completed. The decision, which calls for charging fees to customers who elect to opt out of the SmartMeter program, conflicts with local planning authority, does not protect the health or safety of all residents and imposes a prejudicial financial burden on ratepayers who chose to opt out of the program. We therefore urge the Commission to continue consideration of this matter until further public hearings are completed to ensure the due process rights of all stakeholders.

The order does not provide an empirical basis for the amount of the fees to be charged to opt out customers nor does it consider the net financial impact of PG&E's latest proposal to permit customer retention of analogue meters. Hence the order effectively eliminates a full and fair hearing process for these contested issues of fact to be considered and resolved.

Historically, telecommunications carriers throughout this state have complied with local planning codes which provide notice to residents as to the construction of transmission facilities. Pacific Gas and Electric Company ignored such codes in the deployment of the Smart Meter telecommunications network. Currently many of our jurisdictions have passed ordinances which impose a moratorium on wireless SmartMeters and have petitioned to opt out on a jurisdictional basis. The current order is silent on these issues and effectively discards them without consideration.

The decision also ignores the longstanding controversy and concern about the health impacts associated with electro-magnetic fields. A 1998 California Department of Health Services study commissioned by the California Public Utility Commission itself found that 3.2% of Californians reported hypersensitivity to electro-magnetic fields. A May 2011 study released by the World Health Organization/International Agency for Research on Cancer reclassified RF radiation of the type emitted by wireless equipment throughout the Smart Meter system as "possibly carcinogenic" to humans. President Peevey's order effectively imposes a different rate on many utility customers who need to avoid exposure in violation of California Public Utilities Code section 453(b) which states in pertinent part that "No public utility shall prejudice, disadvantage, or require different rates or deposit amounts from a person because of ancestry, medical condition, marital status or change in marital status, occupation..."

President Peevey's decision does not address these concerns nor does it the financial viability of wired equipment alternatives. In so doing, it eliminates a much anticipated public hearing process.

Signature	Jurisdiction
Signature	Jurisdiction

For all of the foregoing reasons, we respectfully urge the Commission to continue Petition A.11-03-014 matter for further hearings.



#### **Maureen McCarty**

From: Sent: To: Subject: Mark Stone [BDS050@co.santa-cruz.ca.us] Monday, January 09, 2012 1:30 PM Maureen McCarty FW: smart meter opt-out letter and moratorium on smart meters

From: theodora kerry[<u>SMTP:THEKERRY@COMCAST.NET</u>] Sent: Monday, January 09, 2012 1:30:14 PM To: Mark Stone Subject: re: smart meter opt-out letter and moratorium on smart meters Auto forwarded by a Rule

This letter is directed to the whole Board of Supervisors, and, as such, should be included in the public record.

Dear Chairperson Stone,

Having attended the board meeting on Dec. 13, and witnessed the Board's active interrogation of the P.G.&E. rep's woeful defense of her employer's shutting off of electricity to customers who dared to protect their health and that of their children by removing their smart meters, I'm very disappointed to read the agenda for tomorrow's meeting only to find that the expected follow-through re: smart meters was no where to be found. While you did approve a letter to the CPUC expressing your opposition to opt-out charges, many of us need you to go further and protect our right to analog meters, as many health problems have been linked to smart meters that have their wireless component turned off. Despite PG&E's crying "public safety concerns", the analog meters have proven to be safe for decades, unlike the recently installed smart meters which have already been linked to health problems, fires, and overcharging. Unfortunately, the CPUC is supposed to decide this issue as early as Jan.12, leaving you no time to write a stronger letter to the CPUC given that the issue is not on the agenda. While I applaud the strong stance you took with the PG&E's rep at the last meeting, that in itself does little to protect us, your constituents. Even the smart meter moratorium as been little more than window dressing as the Sheriff continues to use his power to protect PG&E contractors, instead of the local citizenry. I reiterate my call for you, the Board of Supervisors, to use your power of the purse strings to make it clear to the Sheriff that he is expected to support the moratorium/citizens, not the profiteering corporations.

Regardless of what you eventually decide, you, like the rest of us, are equally at the mercy of these meters. What you allow to be done unto us by PG&E is also being done unto you.

Theodora Kerry Santa Cruz, CA 95060

41

#### Commentary on the California Council on Science and Technology Report "Health Impacts of Radio Frequency from Smart Meters"

#### By Dr. Karl Maret Dove Health Alliance, Aptos, CA January 30, 2011

This is a commentary on the California Council on Science and Technology (CCST)report, "Health Impacts of Radio Frequency from Smart Meters" published January 2011. I submit that the CCST report, written in response to health concerns expressed by Assembly Members of the California Legislature, contains inaccuracies and minimizes the biological effects and health impacts of non-thermal radiofrequency radiation, such as those produced by wireless technologies including Smart Meters.

For the record, my qualifications to make this commentary are that I hold a Bachelor of Science in Electrical Engineering, a Master of Engineering degree in Biomedical Engineering, and a Medical Doctor degree and have additionally completed a four year postdoctoral fellowship in physiology. I have been interested in the health effects of electromagnetic fields (EMFs) for many years and given lectures about the potential health impacts of non-ionizing radiations, both in Europe and the United States. I am president of a non-profit foundation interested in energy medicine, a sub-specialty within the field of Complementary and Alternative Medicine (CAM) as defined by the National Center for Complementary and Alternative Medicine (NCCAM), a center within the U.S. National Institutes of Health (NIH).

My specific concerns with the report are as follows:

- 1. The minimization of the problem of non-thermal microwave radiation;
- 2. The minimization of the need for lower exposure standards;
- 3. The increase in radiation levels at potential local hotspots through reflection;
- 4. The lack of information about the impact of pulsed radiation from Smart Meters;
- 5. The lack of information on the health impacts of night-time radiation from Smart Meters;
- 6. The lack of modeling or actual measurements of the contribution from Smart Meters to the existing background microwave radiation;
- 7. The lack of health and environmental consideration by the CPUC when the Advanced Metering Infrastructure (AMI) was approved.

Until these issues are more fully addressed it is recommended that the current Smart Meter deployment using radiofrequency radiation (RFR) be halted pending a more unbiased reassessment of the potential health issues associated with these meters, including a reassessment of the Advanced Metering Infrastructure (AMI) program approved by the California Public Utilities Commission (CPUC) without any environmental impact assessment. Further, that the California public be offered the option to opt out of this program, which at present is mandatory for every dwelling.

#### 1. Minimization of Non-thermal Microwave Radiation from Smart Meters

On page 4 of the CCST report it states that "*To date, scientific studies have not identified or confirmed negative health effects from potential non-thermal impacts of RF emissions such as those produced by existing household electronic devices or smart meters.*" This finding minimizes the extensive body of scientific research on the biological effects of non-thermal electromagnetic fields. The biological effects of low-level, non-thermal electromagnetic fields have been researched for over 30 years. Therespected 2007Handbook ofBiological Effects of Electromagnetic Fields edited by Barnes and Greenebaum (1) states on page 377:

"The biophysical lore prevailing until the late 1980s and lingering to this day is that, unless the amplitude and frequencies of an applied electric field were sufficient to trigger an excitable membrane (e.g. heart pacemaker), produce tissue heating or move an ion along a field gradient, there could be no effect. .... However, this position had to be changed as the evidence for weak (non-thermal) EMF bioeffects became overwhelming."

Prof. Arthur Pilla, PhD Professor of Biomedical Engineering, Columbia University

There are numerous reports on the potential health effects of non-thermal electromagnetic fields. Early reports include papers by Frey (1993), Lai (2000) and Hyland (2000), among many others. An international working group has delineated many additional scientific findings (Bioinitiative report, 2007). Special editions of the journal Pathophysiology were specifically dedicated to this topic recently (Pathophysiology, 2009). Recently, the European Journal of Oncology published an entire monograph entitled "Non-Thermal Effects and Mechanisms of Interaction between ElectromagneticFields and Living Matter" outlining non-thermal effects on living systems. This came from the National Institute for the Study and Control of Cancer and Environmental Diseases "Bernardino Mamazzini" (Giuliani &Soffriti, 2010).

The CCST report further states that, "Without a clearer understanding of the biological mechanisms involved, identifying additional standards or evaluating the relative costs and benefits of those standards cannot be determined at this time." I strongly disagree with this conclusion as there is now a large body of scientific literature describing several key mechanisms for the action of weak electromagnetic fields. These include, among others:

- removal of calcium ions bound to cellular membranes, leading to their weakened structure and changed cellular functioning
- change of calcium ion leading to changes in metabolic processes in cells,
- the leakage of calcium ions into neurons generating spurious action potentials,
- fragmentation of DNA in cells seen through the Comet assay
- changes in the blood-brain barrier in animals after microwave exposure
- defined cellular stress response, including the production of heat shock proteins (HSP), that are triggeredelectromagnetically at non-thermal levels that require much less energy than when triggered by heat (so-called thermal considerations)
- activation of specific genes by exposure to non-thermal electromagnetic fields leading to gene transcription form RNA, the first stage in the synthesis of proteins

All these biological effects are well substantiated in the scientific literature and occurred at much lower exposure levels than current FCC standards, but are minimized by the CCST report. It takes many years for definitive health effects to be substantiated beyond all shadow of doubt. Yet the evidence is accumulating that health effects will become more widespread, given sufficient time, from thescientifically researched biological responses to RFR. <u>Until the authors of the CCST report can clearly substantiate their conclusions that the California population will not be adversely affected by the Smart Meter program, a precautionary approach should have been recommended.</u>

The European community has been more concerned about non-thermal radio frequency radiation effects while our government has essentially stopped funding all research in this area (see below). The extensive REFLEX study involving research groups from seven countries found effects on biological systems from cell phone radiation at levels 1/40<sup>th</sup> of the level of accepted safety guidelines promulgated by the International Commission on Non-Ionizing Radiation Protection (ICNIRP) (Adlkofer, 2006). This report focused on a four year international collaboration from cell phones. Even Austrian insurance companies are now accepting the dangers from non-thermal electromagnetic radiation from cell phones (AUVA Report, 2009).

Biological systems often respond in a non-linear manner and there is a large degree of genetic variability as to how animals or people are affected. Non-thermal EMFs might be comparable to the hazards of low levels of toxins found in the environment which can be potent in very low levels at disrupting enzyme systems in the body, but may not be proportionately worse at higher levels.

Dr. Richard Gautier in France offered a full description of active mechanisms for the action of non-thermal EMFs. There are peer-reviewed scientific studies for each step of the processes that can lead to chronic diseases such as cancer, leukemia and neurological diseases. These conditions often require longer time periods to develop and the Precautionary Principle (see later) ought to be applied when adding new sources of microwave radiation such as those from Smart Meters that are active night and day in our homes and places of work.

On page 14 of the CCST report, the statement "*There is currently no definitive evidence linking cell phone usage with increased incidence of cancer*" is another misleading statement that tends to minimize the cancer risk from cell phones. If the authors of the CCST report had looked at other papers from the scientific literature (not mentioned in pages 38-44 of the CCST report), they might come to different conclusions.

There is mounting evidence of various types of tumors being caused from cell phone usage including parotid gland tumor (Czerninski, 2011), meningioma (Hardell et al., 2006), acoustic neuroma (Sato et al. 2011), brain tumors (Hardell&Carlberg, 2009) and testicular tumors (Hardell et al., 2007), to name only some.Considering the increasing number ofscientific papers describing various types of tumors associated with non-thermal radiation from cell phones that are appearing in the medical literature, it is not helpful that non-thermal radiations from Smart Meters, which might potentially add to our long-term susceptibility to serious diseases, be minimized as was done in the report.

#### Page 3 of 14Maret, 2011 – Commentary on CCST Report on Smart Meters

#### 2. The minimization of the need for lower exposure standards

The report states on page 8 that "... given the existing uncertainty about non-thermal effects, there is no generally accepted, definitive, evidence-based indication that additional standards are needed." This statement is misleading since an international collaboration of researchers in this field have called for a reexamination of the current ANSI standard based on the increasing evidence of the adverse effects of low-level electromagnetic fields (Hardell and Sage, 2008) Variousresearch groups have consistently warned that the existing guidelines may be inadequate (Hyland, 2000; Levitt &Lai 2010;Bioinitiative Report, 2007).

Even the International Commission on Non-Ionizing Radiation Protection (ICNIRP) stated in 1998 that "interpretation of several observed biological effects of electromagnetic fields is further complicated by the apparent existence of "windows" of response in both the power density and frequency domains. There are no accepted models that adequately explain these phenomena, which challenge the traditional concept of a monotonic relationship between the field intensity and the severity of the resulting biological effects." (ICNIRP, 1998). In other words, there are windows of sensitive biological response in which potential health effects can occur at much lower exposure levels than currently mandated by the FCC standards.

Already in 1999, the federal government'sRadiofrequency Interagency Work Group (RFIAWG) had "identified certain issues thatwe believe need to be addressed to provide a strong and credible rationale to support RF exposure guidelines." Dr. Gregory Lotz from the Department of Health and Human Services, National Institute for Occupational Safety and Health addressed these specific issues in a letter dated June 17, 1999 to Mr. Richard Tell, then Chair of the IEE SCC28 (SC4) Risk Assessment Work Group. Ironically, it was this same Richard Tell Associates of Las Vegas, NV who wrote the report for PG&E describing the apparent safe exposure limits of the Smart Meter program that was also referenced in the CCST report (Tell, 2005; Tell, 2008).

The Tell Associates report simplified the apparent safety of the Smart Meter radiation by: 1. Only considering a single isolated Smart Meter radiator in free space; 2.Time averaging the pulse RF radiation so that it appeared as a low level of 8.8 uW/cm<sup>2</sup>; 3. Not considering other RF microwave emitters in the home environment; and 4. Considering only ground wave reflections of the microwave emissions and no other reflective surfaces (see below). The report also does not address the concerns of the federal RF Interagency Work Group including among other concerns: 1.The biological basis for local SAR limit; 2. the selection of an adverse effect level; 3. the nature of acute versus chronic exposure; 4. the intensity or pulsed or frequency modulated RF exposure; and 5. the issue of time averaging. These are critical issues which makes the issue of proper exposure guidelines a central issue in this matter. It further casts great doubt on the conclusions of the CCST report that downplays the need for new, lower exposure standards.

Epidemiologic evidence is a major contributor to the understanding of the potential effects of EMF on health. The International Agency for Research on Cancer (IARC) classified EMF as a "possible human carcinogen", or a Group 2B carcinogen; (IARC, 2002) this classification was mostly based on consistent epidemiological evidence. Although the body of evidence is

#### Page 4 of 14Maret, 2011 – Commentary on CCST Report on Smart Meters

always considered as a whole, based on the weight of evidence approach and incorporating different lines of scientific enquiry, epidemiologic evidence, as most relevant, is given the greatest weight.

Several European countries, having taken a deeper look at recent scientific data, are beginning to follow a different approach to the RFR question. They recommend prudent avoidance in siting cell tower antenna installations near schools, hospitals or wherever people congregate. This approach is part of what is called the Precautionary Principle, which has been adopted in many countries, including the U.S., for various applications in international treaties. The Precautionary Principle holds that when questions of safety are concerned, precautions should be taken to protect public health even if scientific data is incomplete, or the mechanisms of action are not understood (Levitt, 2000; Kheifets et al., 2001).

#### 3. The increase in radiation levels at potential local hotspots through reflection

Although it is true that the Smart Meters comply with current U.S. Federal Communications Commission (FCC) guidelines because they operate below the existing power density thresholds, power density is not the only factor determining biological effects from radiofrequency radiation. The power density level safety standards are solely based on thermal considerations, yet it is the non-thermal radiation levels that are the key to potential health impacts. The non-thermal effects occur at lower levels from various emitting radiators now in common use including cell phones, cordless phones, Wi-Fi, Wi-Max, to name only some. Smart Meters add to this cumulative ubiquitous low-level background microwave environment.

RFR can increase to higher levels than anticipated due to surface and ground reflections from the various radiators. (Hondou, 2002; Hondou et al,2006;Vermeeren et al, 2010), even at some distance from the sources. These scientific studies suggest that reflectivity from other metallic surfaces and reflective materials could increase the power density of the RF fields significantly, leading to the development of hot spots in our homes. Richard Tell Associates report commissioned by PG&E in 2005, and updated in 2008, contained calculations of the intensity of RF fields produced by the Smart Meters that included only ground reflections estimated to increase the field strength by 1.6 times (equivalent to a 2.56-fold increase in the power density). In light of recent scientific findings and actual computer modeling studies, the Tell estimate of ground reflectivity may be significantly too low and does not address the development of possible hotspots in the home. If microwave hotspots occurred near sleeping quarters or near a baby's crib, their health impact could be highly significant. Sage Associates report, which made some estimates of Smart Meter impacts through computer modeling, even suggests that under certain assumptions the emissions from Smart Meters and their local reflections might even exceed FCC standards (Sage, 2011).

The CCST report never even acknowledged the need for computer modeling to ascertain the potential riskof higher microwave radiation levels in our homes as a result of Smart Meter installation, alone or in interaction with other microwave emitters. We believe that such modeling is vital if the public is to know the potential for the development hot spots in sensitive living areas. The Richard Tell Associates study carried out for PG&E did not consider other microwave sources in the environment stating, "*The study does not take into* 

#### Page 5 of 14Maret, 2011 – Commentary on CCST Report on Smart Meters

account the potential for RF fields that may be produced by other devices or systems that are not part of the Smart Meter program upgrade. Such devices or systems include cellular telephones, cellular telephone base stations, broadcast radio and TV stations, microwave ovens used in the home or any other source of RF energy."

#### 4. The lack of information about the impact of pulsed radiation from Smart Meters

The is considerable difference between the biological impact of pulsed microwaves, as produced by Smart Meters, compared to continuous waves, such as those produced by microwave ovens. No distinction is made in the safety criteria between continuous and pulsed waves because of the narrow-minded focus on thermal damage alone. Many scientific studies have pointed out that radiofrequency radiation with different modulations and pulse characteristics produce different biological effects even though they may produce the same pattern of different specific absorption rate distribution and tissue heating (Levitt &Lai, 2010).

Peer-reviewed studies have shown that the differences in modulation patterns and waveforms can produce quite different biological effects. They include the works of Arber and Lin (1985); Campisi et al (2010); Huber et al. (2002); Luukkonen et al. (2009); d'Ambrosio et al (2002), among many others. Already Soviet research in the 1960s showed that pulsed waves induced stronger and often inhibitory biological and neurological effects than continuous waves (Osipov, 1965). A review of the hazards to U.Smilitary personnel from high frequency electromagnetic radiation was provided by Pollack (1967) which gives an overview of the extensive Eastern European research in this field.

Marha (1963) described allowable intensities for frequencies above 300 MHz in Czechoslovakia for continuous waves as  $25 \text{ uW/cm}^2$  but limited pulsed waves to only 10  $\text{uW/cm}^2$ . Note that these Czech recommended levels were considerably lower than the approximately 600  $\text{uW/cm}^2$  allowed for the RFR from Smart Meters operating in the low 900 MHz band mandated by the FCC based on only thermal consideration. Also not well known in the West is the Soviet work showing the adverse effect of non-thermal pulsed microwave radiation on cardiac rhythms in animals (Presman&Levitina, 1962).

The CCST report is misleading because it compares the Smart Meter emissions to those of microwave ovens. Microwave ovens produce much higher power output but are <u>not</u> modulated or pulsed in any way. It is imperative to understand that it is the modulation or pulsation pattern that leads to biological effects at non-thermal power levels. Biologically-sensitive amplitude windows have been found at specific frequencies that lead to the selective release of calcium from cell membranes. However, above and below these unique power densities there is no observable effect. Pulses and square waves have the greatest biological impact because they produce rapid changes in voltage across biological membranes. Un-modulated carrier waves have little or no biological effect except if their power is sufficient high, such as in microwave ovens. Comparing the power levels between modulated and un-modulated devices, as the CCST report does, is thus misleading.

The potential health effects from chronic exposure to pulsed, low power density level electromagnetic fields might take several years to appear. These types of radiations produced

#### Page 6 of 14Maret, 2011 – Commentary on CCST Report on Smart Meters

by Smart Meters are of concern for their potential health impacts on the electrically hypersensitive part of the population. In Sweden, electrohypersensitivity(EHS) is an officially recognized functional impairment; however it is not regarded as a disease (Johansson, 2006). Electrical hypersensitivity has been reported by many authors from various industrialized countries over the last 20 years. The CCST report does not consider this segment of our population at all. Yet in the United Kingdom there are excellent resources about this condition, especially the work of Bevington (2010) containing over 700 references.

The ICNIRP, IEEE and ANSI standards that are currently in effect consider only thermal effects of microwave radiation where the energy absorption is fairly linear and thus the protective guidelines are logical. However these energy absorption guidelines would <u>not be appropriate</u> when frequency-specific amplitude windows are involved leading to adverse biologicaleffects that can depend onmodulation patterns, pulse repetition rates, duty cycles, and other frequency spectrum characteristics. With the current PG&E-mandated Smart Meter program having a 20-year life expectancy, Californians will be living with potential health impacts from this unproven technology in our homes for the next two decades.

### 5. The lack of information on the health impacts of night-time radiation from Smart Meters

Another problem that was not addressed in the CCST report is potential health effect of microwave radiation exposure during our sleep which may adversely affect our biological and circadian rhythms (daily physiological regulatory cycles). Smart Meters will pulse intermittently day and night and may have an adverse effect on sleep cycles. We do not use our cellphones during sleep, yet Smart Meters will continue to emit pulsed RFR all night long.

Exposure to microwave/radiofrequency fields affect the neuroendocrine system causing neuroendocrine chemical modulations and behavioral reactions. Already in 1970s it was known that resonant absorption within the cranium may result in the focusing of energy and the production of electromagnetic "hot spots" in the brain (Johnson & Guy, 1972). Microwaves may disturb the critical hormonal regulatory areas including the hypothalamic-pituitary axis through "low intensity" exposure. The body may elicit "different responses relative to the timing of the exposure with respect to circadian rhythm" (Michaelson,1982). At night, while sleeping, the body is principally in a repair mode and the exposure to microwave radiation from Smart Meters may potentially be more damaging than exposure during the day. It is vital that long-term exposure studiesduring the night be carried out to determine if Smart Meter pulsed microwave radiation could have an adverse biological effecton our population.

The European Commission's Scientific Committee on Emerging and Newly Identified Health Risks report on "Health Effects of Exposure to EMF" stated that "*No health effect has been consistently demonstrated at exposure levelsbelow the ICNIRP-limits established in 1998. However, the data base for this evaluationis limited especially for long-term low-level exposure*" (SCENIHR, 2009). In other words, we just don't know what will be the long-term

#### Page 7 of 14Maret, 2011 – Commentary on CCST Report on Smart Meters

effect of consistent low level exposure of RFR such as those imposed by Smart Meters in addition to the other microwave radiation sources now increasingly being used in our homes.

#### 6. The lack of modeling or actual measurements of the contribution from Smart Meters to the existing background microwave radiation

The CCST report is misleading on page 20 where it says that he exposure levels to people living in metropolitan areas is quite low, around 0.005 uW/cm<sup>2</sup>. They base their assertions on an outdated report fromJuly 1986 made by the U.S. Environmental Protection Agency entitled The Radiofrequency Radiation Environment: Environmental Exposure Levels and RF Radiation Emitting Sources, EPA 520/1-85-014. This data is totally outdated since it reflects the situation before the modern cellular telephone networks were put in place.

Already in 2000, in Sweden, the radiofrequency and microwave radiation levels in urban areas were approximately ten times higher than they were in the 1980s—and most of the increase is due to wireless communications, according to Dr. YngveHamnerius of Chalmers University of Technology in Göteborg, Sweden. Hamnerius measured radiation levels in the 30 MHz-2 GHz frequency range at 26 sites across Sweden with varying levels ofurbanization. In cities, the median power density was 0.05 uW/cm2, with a 61% average contribution from GSM cell tower base stations. (Microwave News, July/August 2000). In the U.S. we do not have any up-to-date data since the U.S. Environmental protection Agency has not carried out any research studies for two decades. I have personally measured background microwave radiation levels that are hundreds of times higher in many metropolitan areas than the values described in the CCST report using 1986 EPA data.

This increasing amount of background microwave radiation has become of medical concern in many parts of the world. For example in March 23, 2009 European scientists called for a reassessment of the damaging health impacts of increasing levels of electromagnetic radiation (Electrosensibilité : Appel des scientifiques du 23-03-2009). Similarly, in November 2009 a meeting of international experts on the biological effects of electromagnetic fields met in Stavanger, Norway to discuss the unprecedented global exposures to artificial electromagnetic fields from communication and power technologies. Many scientists at this meeting recommended that lower limits be established for electromagnetic fields and wireless exposures due to the health impacts at much lower exposure levels than are now considered safe.

The United States government essentially stopped all research on RF radiation effects on the environment, including population exposure, in 1996. The Environmental Protection Agency's budget and staffing for RF radiation activities was \$821,000 from 1990 to1995 and only \$25,000 between the years 1996 to 2000 (Levitt, 2000, page 271). Essentially, there was no government money spent in the last 15 years by the EPA to fund a reexamination of the RF exposure limits by the National Council on Radiation Protection and Measurement (NCRP). Our changing microwave environment is thus not being studied by our federal government. If the federal government is not looking after our health concerns concerning low level electromagnetic fields, it is imperative that utilities have their new microwave technologies evaluated by state government research laboratories or public health

#### Page 8 of 14Maret, 2011 – Commentary on CCST Report on Smart Meters

organizations prior to letting this technology be deployed on a largely unaware California public.

What is needed is an up-to-date series of measurements in dense urban environment that measures the combined RFR levels from all radiating emitters and estimates or measures the cumulative effect of Smart Meters and collectors to radiation exposure levels in homes. This must include all RFR emitters that are connected to the MESH and home area networks (HAN) as deployed by PG&E. Only independent assessments or measurements of these radiation levels ought to be considered, not those conducted by companies that have direct or indirect connection to the utilities. Until these studies are available, it is recommended that the Precautionary Principle be adopted.

## 7. The lack of health and environmental consideration by the CPUC when the Advanced Metering Infrastructure (AMI) was approved.

On July 20, 2006, the California Public Utilities Commission (CPUC) issued their final opinion, Decision 06-07-027, authorizing Pacific Gas and Electric to deploy an Advanced Metering Infrastructure (AMI) that would lead to the automation of 5.1 million electric meters and 4.2 million gas meters. The CPUC decision was in response to PG&E's application 05-06-028 filed on July 16, 2005. In Section 7 (Technology) of this CPUC decision, the AMI deployment was described as using Power Line Carrier technology for electric meters and a fixed network system with radio frequency communications channels owned by PG&E for gas meters. The system was to have a useful life of 20 years. In section 15 (Environmental Review) of the Decision, it stated that there is no need for an analysis of PG&E's AMI deployment pursuant to the requirements of the California Environmental Quality Act (CEQA). It appeared that due to the suggested Power Line Carrier technology to be employed, the health or environmental effects were not considered at the time and the CPUC felt under no legal obligation to undertake any environmental review before approving the PG&E application.

On March 12, 2009, the CPUC made another Decision 09-03-026 in response to PG&E's application A.07-12-009 filed on December 12, 2007 to expand the AMI program significantly. Now the CPUC approved the establishment of microwave mesh networks as well as incorporating a Home Area Network (HAN) gateway deviceinto advanced electric meters to support in-home HANapplications; and upgrading PG&E's electric meters to solid state meters, now called Smart Meters. In this decision, which conveniently expanded its 2006 AMI deployment decision, there was absolutely no mention of any environmental or health impact even though a whole new radiofrequency technology infrastructure was now approved for deployment on every home and business in California. We believe that this decision represents a gross degree of negligence by the CPUC in protecting the health and safety of the citizens of California. The CPUC needs to readdress the health and safety issues directly and immediately halt the installation of the Smart Meter program pending clarification of the CCST report.

#### Conclusions

The time needed for a new technology to be developed and rolled out is much shorter than the time needed for research to investigate the possible health effects on the general population. The current Advanced Metering Infrastructure using microwaves in the 900 MHz frequency spectrum approved by the CPUC is going to adversely impact the physiology and ultimately the health of many Californians over the next twenty years, the anticipated life time of the Smart Meters now being deployed. This program is being implemented without widespread public knowledge or approval and without the specific informed consent in writing from every household.

Already the most sensitive members of our society, those who are especially vulnerable by being electrically hypersensitive, are registering health complaints such as headaches, sleep disturbances, cognitive difficulties, dizziness, heart palpitations, to name only a few. Most of these symptoms could also be related to other medical conditions making it difficult to ascribe their appearance specifically to the Smart Meters radiation directly. Although not yet recognized in this country as a state of physiological imbalance, hypersensitivity of human subjects to exposure to electric and magnetic fields has been reported for over 20 years by many authors in many industrialized countries. If only 1% of California's population were to report symptoms of electrical hypersensitivity after Smart Meter installation, over 370,000 people might be adversely affected by RFR.

The dissemination of this Smart Meter technology could have been accomplished without using radiofrequency radiation by using much safer power line, fiber optic or telephone communications technology. For example, a Smart Meter power line communications technology was used by Italian utilities in 27 million households using meters designed in California. In the Netherlands, the population concerned about the security and health issues of Smart Meters was given the options to opt out from having the meters installed. Californians were never given this option. Yet this AMI program, costing utility customers over \$2 billion, represents the largest technology roll-out in the history of Pacific Gas and Electric. Ironically, it is being financed by the rate payers without their direct consent.

This program represents an epidemiological experiment involving our unsuspecting population whose outcome will only be fully known after many years exposure. It is being shepherded through the regulatory process by the CPUC who has not seen fit to study the possible adverse health impacts of this technology before approving its usage. It has never shown any willingness to seriously consider the well-documented non-thermal effects of pulsed microwaves on living systems and will undoubtedly use the misleading CCST report to avoid any questions about future health implications of this technology. Because of the uncertainties of adverse long-term health impacts, the CCST ought to have recommended that a Precautionary Principle be invoked that would allow more time to directly study the effect of this pulsed radiation with both in vitro and in vivo testing in realistic settings of the mesh network, especially in high density Smart Meter environments in our cities.

Additionally, in cities the Subterranean Network Deployment System (SUNDS) is now also being installed by PG&E. This will add even higher microwave exposure levels to the general population. Any description of this new system was conspicuously absent from the CCST report. At a minimum, the utilities and CCST ought to have carried out extensive

#### Page 10 of 14Maret, 2011 – Commentary on CCST Report on Smart Meters

computer modeling to assess the impact of Smart Meter technology in realistic settings, taking into account the other wireless technologies have already been deployed and which have significantly increased the background microwave exposure of the population over the last 20 years.

In summary, we find that the CCST report is incomplete and misleading giving California State regulators a false sense of security while potentially endangering the future health and well-being of Californians. It is requested that the current Smart Meter deployment be halted pending a more comprehensive scientific investigation of the biological response and health impacts of the non-thermal aspects of this technology. All households should be offered full disclosure about possible exposure levels, modulation patterns, peak power levels and interactions with other parts of the microwave spectrum in their home environments. Additionally, those who are sensitive to this radiation must be given the choice to opt out from having this form of RFR imposed upon their residential dwellings.

### References

Adlkofer, F. (2006) Risk Evaluation of Potential Environmental Hazards from Low Energy Electromagnetic Field Exposure using sensitive In Vitro Methods. BIOELECTROMAGNETICS CURRENT CONCEPTS. NATO Security through Science Series, 2006, 2006:331-354. Also known as REFLEX study report.

Arber, S.L., and Lin, J.C. 1985. Microwave-induced changes innerve cells: effects of modulation and temperature. Bioelectromagnetics,**6**(3): 257–270.

AUVA report (2009) Untersuchungathermischer Wirkungenelektromagnetischer Felder imMobilfunkbereich (in German). An English description of the report available at <u>http://www.diagnose-funk.org/assets/2009-7-</u>20 df bp auva-report english.pdf

Barnes, F.S. & B. Greenebaum (eds.) (2007) Biological and Medical Aspects of Electromagnetic Fields. Third edition. CRC Press, Boca Raton, FL.

Bevington, Michael. (2010) Electromagnetic-Sensitivity and Electromagnetic-Hypersensitivity: A Summary. Capability Books, UK ISBN:978-1-872072-20-3 Available from <u>http://www.es-uk.info/</u>

BioInitiative Working Group, Cindy Sage and David O. Carpenter, Editors. (2007) BioInitiative Report: A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF) at www.bioinitiative.org, August 31, 2007.

Campisi, A., Gulino, M., Acquaviva, R., Bellia, P., Raciti, G., Grasso, R., Musumeci, F., Vanella, A., and Triglia, A. 2010. Reactiveoxygen species levels and DNA fragmentation on astrocytesin primary culture after acute exposure to low intensitymicrowave electromagnetic field.Neurosci.Lett.**473**(1): 52–55.

Czerninski, R et al. (2011) Risk of Parotid Gland Tumors in Israel (1970-2006). Epidemiology January 2011 - Volume 22 - Issue 1 - pp 130-131.

d'Ambrosio, G., Massa, R., Scarfi, M.R., and Zeni, O. 2002. Cytogeneticdamage in human lymphocytes following GMSK phasemodulated microwave exposure. Bioelectromagnetics, **23**(1): 7–13

Eberhardt, J.L., B.R. Persson, A.E. Brun, L.G. Salford, and L. O. G. Malmgren. (2008) Blood-Brain Barrier Permeability and Nerve Cell Damage in Rat Brain 14 and 28 Days After Exposure to Microwaves from GSM Mobile Phones. Electromagnetic Biology and Medicine, 27: 215–229.

Frey, Allen H. (1993) Electromagnetic field interactions with biological systems. The FASEB Journal.Feb; Vol 7:272-281.

Gautier, R. Diagram of non-thermal mechanisms available at <u>http://www.next-up.org/pdf/Diagram\_of\_mechanisms\_linked\_to\_EMF\_exposure\_csif.pdf</u>

Giuliani, L. &Soffriti, M eds. (2010) ICEMS Monograph "Non-Thermal Effects and Mechanisms of Interaction between Electromagnetic Fields and Living Matter."National Institute for the Study and Control of Cancer and Environmental Diseases "Bernardino Mamazzini".

Hamnerius, Yngve. Hisresearch quoted inMicrowave News, July/August, 2000, p.3 available on line at <u>http://www.microwavenews.com/news/backissues/j-a00issue.pdf</u>

Hardell, L., M. Carlberg, K. H. Mild. (2006) Pooled analysis of two case-control studies on the use of cellular and cordless telephones and the risk of benign brain tumours diagnosed during 1997-2003. International Journal of Oncology 28: 509-5181

Hardell, I., M. Carlberg, C.-G.Ohlson, H. Westberg, M. Eriksson and K. H. Mild.(2007) Use of cellular and cordless telephones and risk of testicular cancer.Int J Androl. Apr; 30(2):115-22.

Hardell, L. & C. Sage (2008) Biological effects from electromagnetic field exposure and public exposure standards. Biomed Pharmacother.Feb;62(2):104-9.

Hardell, L. & M. Carlberg. (2009) Mobile phones, cordless phones and the risk for brain tumours. International Journal of Oncology 35: 5-17.

Hondou T. (2002) Rising Level of Public Exposure to Mobile Phones: Accumulation through Additivity and Reflectivity. Journal of the Physical Society of Japan, Vol. 71, No. 2, February, 2002, pp. 432–435.

Hondou T Ueda T Sakat Y Tanigwa N Suzuki T Kobayashi T Ikeda K.(2006) Passive Exposure to Mobile Phones: Enhancement of Intensity by Reflection, Journal of the Physical Society of Japan Vol. 75, No. 8, August, 2006.

Huber, R., Treyer, V., Borbe 1y, A.A., Schuderer, J., Gottselig, J.M., Landolt, H.-P., Werth, E., Berthold, T., Kuster, N., Buck, A., and Achermann, P. (2002) Electromagnetic fields, such as thosefrom mobile phones, alter regional cerebral blood flow and sleepand waking EEG. J. Sleep Res. **11**(4): 289–295.

Hyland, G. (2000) Physics and biology of mobile telephony. The Lancet.Vol 356, Nov 25: 1833-1836.

IARC.(2002) Working Group on the Evaluation of Carcinogenic Risks to Humans. Non-ionizing radiation, Part 1: Static and extremely low-frequency (ELF) electric and magnetic fields. *Monographs on the Evaluation of Carcinogenic Risks to Humans*. Lyon: International Agency for Research on Cancer. vol 80.

ICNIRP(1998) "Guidelines for limiting exposure to time-varying electric, and electromagnetic fields (up to 300 GHz) - ICNIRP Guidelines". Health Physics, 74(4): 494-522.

Johansson, O. (2006) Electrohypersensitivity: State-of-the-Art of a Functional Impairment. Electromagnetic Biology and Medicine, 25: 245–258.

Johnson, C.C. & A.W. Guy (1972) Nonionizing electromagnetic wave effects effects in biological materials and systems. Proc IEEE, 60, 692.

Kheifets L, Hester G, Banerjee G. (2001) The Precautionary Principle and EMF: Implementation and Evaluation. *Journal of Risk Research*. 2001;4(2):113-125.

Lai, H. (2000) Biological effects of radiofrequency radiation from wireless transmission towers. in Levitt, B. (ed.) Cell Towers: Wireless Convenience? Or Environmental Hazard? Proceedings of the "Cell Towers Forum", State of the Science/State of the Law, Dec.2, 2000. Chapter 3. New Century Publishing, Sheffield, MA, 2000.

Chapter 3 in

Levitt, B. ed. (2000) Cell Towers: Wireless Convenience? Or Environmental Hazard? Proceedings of the "Cell Towers Forum", State of the Science/State of the Law, Dec.2, 2000. Chapter1. New Century Publishing, Sheffield, MA, 2000.

Levitt, B.B. & H. Lai (2010). Biological effects from exposure to electromagnetic radiation emitted by cell tower base stations and other antenna arrays. Environ. Rev. Vol 18:369-395.

Lotz, Gregory (1999) Letter from Chief, Physical Agents Effects Branch, Division of Biomedical and Behavioral Science, National Institute for Occupational Safety and Health, Robert A. Taft Laboratories, Cincinnati OH dated June 17, 1999 to Mr. Richard Tell, then Chair of the IEE SCC28 (SC4) Risk Assessment Work Group. Available at http://www.emrpolicy.org/litigation/case\_law/docs/exhibit\_a.pdf

Luukkonen, J., Hakulinen, P., Ma<sup>\*</sup>ki-Paakkanen, J., Juutilainen, J.,andNaarala, J. 2009. Enhancement of chemically induced reactiveoxygen species production and DNA damage in human SHSY5Yneuroblastoma cells by 872 MHz radiofrequency radiation.Mutat. Res. **662**: 54–58.

### Page 13 of 14Maret, 2011 – Commentary on CCST Report on Smart Meters

Marha, K., 1963: "Biological Effects of High Frequency Electromagnetic Waves," PracovniLekarstvi, Vol. 15( 9): 387-393. (English transl.: AID Report 66-02, AD 642029, also N67-12957).

Michaelson, S.M. (1982) The Influence of Radiofrequency/Microwave Energy absorption on physiological regulation. Br. J. Cancer 45, Suppl. V: 101-108.

Minecki, L., 1964: "Critical Evaluation of Maximum Permissible Levels of Microwave Radiation, TIArchivZaHigijenuRada I Toksikologiju, Vol. 15(1): 47-55.

Osipov, Yu. a., 1965: Labor Hygiene and the Effect of Radio Frequency Electromagnetic Fields on Workers. Leningrad, Meditsina Publishing House, 220 pp.

Pathophysiology Journal, Special Issue 16: Volumes 1 and 2, 2009. Elsevier Press

Pollack, H. and J. Healer, A Review of Information on Hazards to Personnel from High Frequency Electromagnetic Radiation. Internal Note N-451, Institute for Defense Analysis, Research and Engineering Support Division. IDA/HQ 67-6211, Series B, May 1967. Available at: http://www.magdahavas.com/wordpress/wp-content/uploads/2010/07/Pollack\_19671.pdf

Presman, A. S. and N. A. Levitina, 1962: "Nonthermal Action of Microwaves on Cardiac Rhythm, Communication I. A Study of the Action of Continuous Microwaves," Byull.Eksper.BioI.i Med., Vol. 53(1): 41-44.

Presman, A. S. and N. A. Levitina, 1962: "Nonthermal Action of Microwaves on the Rhythm of Cardiac Contractions in Animals, Report II. Investigation of the Action of Impulse Microwaves," Byull.Eksper.BioI.iMed., Vol. 53 (2): 39-43.

Sage Associates (2011) Assessment of Radiofrequency Microwave Radiation Emissions from Smart Meters. Santa Barbara, CA January 1, 2011. Available at: <u>http://sagereports.com/smart-meter-rf/</u>

Sato, Y., S. Akiba, O. Kubo, and N. Yamaguchi. (2011) A Case-Case Study of Mobile Phone Use and Acoustic Neuroma Risk in Japan. Bioelectromagnetics. Feb;32(2):85-93.

SCENIHR. 2009. Health effects of exposure to EMF, EuropeanCommission, Health& Consumer Protection DG. ScientificCommittee on Emerging and Newly Identified Health Risks(SCENIHR), 19 January 2009.

Tell, Richard A. (2005) Analysis of RF Fields associated with Operation of PG&E Automatic Meter Reading Systems. Richard Tell Associates, N. Las Vegas, NV report prepared for PG&E, April 6, 2005.

Tell, Richard A. (2008) Supplemental Report on an Analysis of Radiofrequency Fields associated with Operation of PG&E SmartMeter Program Upgrade Systems. Richard Tell Associates, Colville, WA for PG&E dated Oct 27, 2008.

Vermeeren G Gosselin MC Gosselin Kuhn S Kellerman V Hadmen A Gati A Joseph W Wiart J Meyer F Kuster N Martens L. The influence of the reflective environment on the absorption of a human male exposed to representative base station antennas from 300 MHz to 5 GHz, Phys. Med. Biol. 55 (2010) 5541–5555.

(19) World Intellectual Property Organization International Bureau



(43) International Publication Date 2 September 2004 (02.09.2004)

РСТ

PCT/CH2003/000138

- (51) International Patent Classification<sup>7</sup>: H04Q 7/32, 7/30
- (21) International Application Number:
- (22) International Filing Date: 24 February 2003 (24.02.2003)

(25) Filing Language: English

- (26) Publication Language: English
- (71) Applicant (for all designated States except US): SWISS-COM AG [CH/CH]; Ostermundigenstrasse 93, CH-3000 Bern 29 (CH).

### (72) Inventors; and

- (75) Inventors/Applicants (for US only): MORENO BLANCA, Ferran [ES/CH]; Ostermundigenstrasse
  93, CH-3050 Bern (CH). BISCHOFF, Jean-Claude [CH/CH]; Le Grand Clos 14, CH-1774 Montagny-les-Monts (CH).
- (74) Agent: BOVARD LTD.; Optingenstrasse 16, CH-3000 Berne 25 (CH).
- (81) Designated States (*national*): AE, AG, AL, AM, AT (utility model), AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA,

### (10) International Publication Number WO 2004/075583 A1

CH, CN, CO, CR, CU, CZ (utility model), CZ, DE (utility model), DE, DK (utility model), DK, DM, DZ, EC, EE (utility model), EE, ES, FI (utility model), FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK (utility model), SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW.

(84) Designated States (regional): ARIPO patent (GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW), Eurasian patent (AM, AZ, BY, KG, KZ, MD, RU, TJ, TM), European patent (AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR), OAPI patent (BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG).

#### **Declaration under Rule 4.17:**

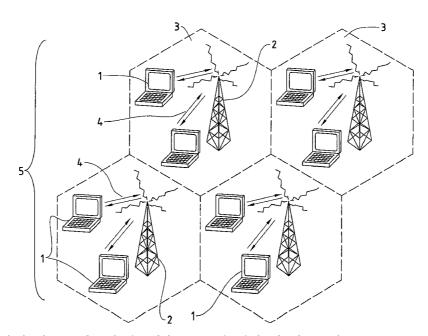
- of inventorship (Rule 4.17(iv)) for US only

### Published:

with international search report

For two-letter codes and other abbreviations, refer to the "Guidance Notes on Codes and Abbreviations" appearing at the beginning of each regular issue of the PCT Gazette.

(54) Title: REDUCTION OF ELECTROSMOG IN WIRELESS LOCAL NETWORKS



(57) Abstract: A method and system for reduction of electrosmog in wireless local networks, one or more mobile network units (1) communicating with a base station (2) of a wireless local network (5). After a predefinable time interval without connecting signal, the base station (2) changes over from the normal transmitting-receiving mode into a sleep mode, in which sleep mode no beacon signals and/or other radio frequency signals are transmitted from the base station (2). If a mobile network unit (1) requires a network connection, it transmits an alert signal, and, upon receiving the alert signal of the mobile network unit (1), the base station transmits beacon signals to the mobile network unit (1) and changes over into the normal transmitting-receiving mode.

200

### **Reduction of Electrosmog in Wireless Local Networks**

This invention relates to a method and system for reduction of electrosmog in wireless local area networks (WLAN), one or more mobile network units communicating with a base station by means of radio frequency 5 signals in a wireless local area network, which base station amplifies the radio frequency signals of the mobile network unit and/or connects the wireless local area network to a wired fixed network by means of bridge functions. In particular, the invention relates to a method and system in which a WLAN

10 The influence of electrosmog on the human body is a known problem. The health risk from mobile radio transmitters, handys and DECT telephones has been an explosive subject among the general public at least since the enormous breakthrough in mobile radio technology in the 1990s. To meet the concerns of science from the legislative side, the permissible limit

comprises a plurality of access points with differing transmission cells.

- values have thus been lowered several times, and technology has been increasingly focused on this problem. The risk of damage to health through electrosmog has also become better understood as a result of more recent and improved studies. When, for example, human blood cells are irradiated with electromagnetic fields, clear damage to hereditary material has been
- 20 demonstrated and there have been indications of an increased cancer risk (Mashevich M., Folkman D., Kesar A., Barbul A., Korenstein R., Jerby E., Avivi L., Department of Human Genetics and Molecular Medicine, Tel-Aviv University, Tel-Aviv, Israel, "Exposure of human peripheral blood lymphocytes to electromagnetic fields associated with cellular phones leads to chromosomal
- instability," Bioelectromagnetics, 2003 Feb., 24(2): 82-90). In this study, for example, human peripheral lymphocytes were exposed to continuous electromagnetic fields of 830 MHz in order to examine whether this leads to losses or gains in chromosomes (aneuploidy). Bigger changes lead to instability of the genome (= the totality of all genes of a germinal cell) and thereby to
- 30 cancer. The human peripheral blood lymphocytes (PBL) were irradiated at different average specific absorption rates (SAR) of 1.6 to 8.8 W/kg over a time period of 72 hours in an exposure system based on a parallel plate resonator in a temperature range of 34.5 to 37.5 °C. The average absorption rate (SAR) and

### **CONFIRMATION COPY**

its distribution in the exposed tissue culture flask were determined by combining the measurement results with a numerical analysis based on a finite element simulation code. A linear increase in the chromosome No. 17 -- an aneuploidy (=numerical chromosome aberration) -- was observed as a function of the SAR,

- 5 demonstrating that this radiation has a genotoxic effect. The SAR-dependent aneuploidy was accompanied by an abnormal mode of replication of the chromosome 17 region engaged in segregation (repetitive DNA arrays associated with the centromere), suggesting that epigenetic alterations are involved in the SAR dependent genetic toxicity. Control experiments (i.e.
- without any radio frequency radiation) carried out in the temperature range of 34.5 to 38.5 °C showed that elevated temperature is not associated with either the genetic or epigenetic alterations observed following RF radiation, these alterations being the increased levels of aneuploidy and the modification in replication of the centromeric DNA arrays. These findings indicate that the
- 15 genotoxic effect of electromagnetic radiation is elicited via a non-thermal pathway. Moreover aneuploidy is to be considered as a known phenomenon in the increase of cancer risk.

Thus it has been possible to show that mobile radio radiation can cause damage to genetic material, in particular in human white blood cells, whereby both the DNA itself is damaged and the number of chromosomes changed. This mutation can consequently lead to increased cancer risk. In particular, it could also be shown that this destruction is not dependent upon temperature increases, i.e. is non-thermal. Based on the scientific studies in the field, and owing to increasing pressure from the public, especially in the industrialized countries, epidemiological studies have been systematized by the World Health Organization (WHO) in the last few years, such as e.g. the currently running WHO Interphone Project, in order to be able to assess more precisely the health risks from electrosmog and work out corresponding guidelines.

<sup>30</sup> Local networks (LAN: Local Area Network) usually consist of socalled nodes which are connected via physical media, such as e.g. coaxial cable, twisted pair or optical fiber cable. These LANs are also referred to as wired LANs (wired fixed networks). In the last few years wireless LANs have

also become more and more popular (e.g. through developments such as the AirPort System of Apple Computer, Inc.). Wireless LANs -- also referred to as WLANs -- are especially suitable for integrating mobile units (nodes), such as e.g. laptops, notebooks, PDAs (Personal Digital Assistants) or mobile radio

- 5 devices, in particular mobile radio telephones, with a corresponding interface, into a local computer network. The mobile nodes have an adaptor comprising a transceiver as well as a control card (such as e.g. infrared (IR) adaptor or a low frequency radio wave adaptor). The advantage of such mobile nodes is that they can be moved freely within the range of the wireless LANs. The mobile
- nodes communicate either directly with one another (peer-to-peer wireless LAN) or send their signal to a base station which amplifies the signal and/or passes it on. The base stations can likewise comprise bridge functions. Via such base stations with bridge functions, so-called access points (AP), the mobile nodes can access the wireless LAN on a wired LAN. Typical network
- 15 functions of an access point comprise the transmission of messages of one mobile node to another, the sending of messages from the wired LAN to a mobile node and the transmission of messages of a mobile node to the wired LAN.
- There exist many different access methods for WLAN in the state of the art which make it possible for a user of a mobile network device to access a wireless local network. One of these access methods, such as e.g. Carrier Sense Multiple Access/Collision Detection (CSMA/CD) or token passing have proved to be highly successful in their industrial application. Today the use of local or wide area networks usually does not have any clearly defined,
- 25 predetermined characteristics anymore. With the growth of heterogeneous multimedia data exchange (e.g. video data streams, etc.) via WLANs, the Quality of Service (QoS) parameter for a particular type of data exchange (or application) has become more and more important. Such parameters comprise, for example, the highest possible bandwidth, lowest possible delay, etc. For
- <sup>30</sup> such accesses, new access methods in the asynchronous or synchronous networks have been developed and can be found in the state of the art.

Together with the growth of the WLAN and the standardization of the access methods and the physical layer specifications for WLANS, such as e.g.

the 802.X physical layer protocols and non- 802.X protocols (e.g. ATM: Asynchronous Transfer Mode Protocol), the security needs of users and service providers of such networks have also become greater and greater. Unambiguous network recognition as well as user identification and/or

- authentication thereby complement one another. Within a WLAN, an AP transmits so-called Service Set IDentifier (SSID) when a mobile network unit tries to integrate itself in the wireless network. An SSID is an unambiguous identification, 32 characters long, which is assigned to the header of data messages sent over the network, and serves as a password for the mobile
- network units. The SSID differs from one WLAN to another. That means that all APs and mobile network units of a particular WLAN must use the same SSID. A network unit which cannot support the unambiguous SSID will not be granted any network access via a base station or respectively an AP. As mentioned, in the 802.X network technology, such as e.g. the 802.11 network technology, the
- 15 network units normally communicate via an access point (AP). In the infrastructure mode, mobile network units can either communicate with one another or with network components of a wired network. An AP with bridge functions, which is connected to a wired network and one or more other access points, is referred to as the Basic Service Set (BSS). Designated as the
- Extended Service Set (ESS) are a plurality of BSS, which form in each case a sub-network. WLANs are usually operated in the infrastructure mode in order to provide access to other services, such as e.g. file server, printer services and/or the worldwide backbone network (Internet). In the 802.X technology, an SSID concerns in each case a Basic Service Set. Thus a mobile unit can only have network access to a BSS if it supports the corresponding SSID. SSIDs are sometimes referred to as network names since the SSIDs unambiguously designate or identify a network.

The physical range of an AP is called the Basic Service Area (BSA). If a mobile node is located within the BSA of an AP, it can communicate with this AP if the AP is likewise within the signal range (Dynamic Service Area (DSA)) of the mobile node. Mobile nodes typically have a signal strength of 100 mwatt to one watt. To connect the wireless LAN to the wired LAN, it is important for the AP to determine whether a particular message (information frame) on the network is intended for a node which lies within the wired LAN or within the

wireless LAN, and to pass on this information, if necessary, to the corresponding node. For this purpose APs have so-called bridge functions, e.g. corresponding to the standard IEEE Std 802.1D-1990 "Media Access Control Bridge" (31-74 ff). With such bridge functions, a new mobile node is registered

- 5 in the wireless LAN, typically in a FDB (Filtering Database) of the AP in whose range the node is located. With each information frame on the LAN, the AP compares the destination address with the addresses (MAC addresses (Media Access Control Addresses)) which it has stored in the FDB, and sends, rejects or passes on the frame to the wired LAN or to the wireless LAN. The range of a
- wireless LAN is limited by factors such as e.g. wavelength of the signal, signal strength, impediments, etc. The radio frequency parameters cannot be selected freely, however. In most countries there are regulations, more or less strict, as mentioned further above, as concerns the low frequency transmission for wireless LANs (e.g. USA (FCC), Switzerland (BAKOM), etc.). This applies in
- particular to the USA, for example. In the USA the regulations are issued by the United States Federal Communications Commission (FCC) (D 15, Title 47, Code of Federal Regulations 1985). Three bandwidths are permitted: 902-928 MHz, 2400-2483.5 MHz and 5725-5850 MHz. Many applications today use the 900 MHz band. The quantity of data which can be transmitted over the 900
- MHz band is limited, however, by the narrow frequency bandwidth in this band. Therefore more and more applications are using the frequency band around 2400 MHz. Future applications will presumably also use the band around 5800 MHz in order to meet the growing demand for high data throughput.

Despite increasingly strict national guidelines with respect to legally specified limits, the impact of electrosmog in WLANs on the human body can be considerable. Moreover it is to be expected that this impact will continue to increase in the future for many people. Two factors in particular are playing a role in this: First, more and more applications require additional, usually higherenergy frequency bands in order to be able to meet the growing need with

30 respect to transmission rate. Second, the need for WLAN expansion in the private sphere as well as in the public sphere, e.g. in airports, railway stations, trains, restaurants, exhibition halls, etc., has by far not yet reached its peak. With the state of the art as a basis, there has been a lot of effort put into providing evidence for the detrimental effects of electrosmog and setting

corresponding limits. Limits and guidelines alone will not suffice, however, to further contain the electrosmog in WLANs since the development in WLANs runs in exactly the opposite direction, as mentioned above. WLANs even represent zones in which people usually spend longer periods of time (place of

- 5 work, Internet, network games, etc.) and are therefore to be considered as particularly problematic with respect to radiation impact. WLANs in the state of the art moreover send base stations, such as access points, so-called beacon signals periodically so that mobile units can recognize the network and authenticate themselves with an access point. These beacon signals comprise
- recognition signals, such as e.g. SSIDs and/or other radio frequency signals with control parameters. Even if no mobile units are located in the WLAN, the beacon signals continue to be transmitted periodically to the APs. This means that even when the WLAN is not being used at all, an underlying stress from electromagnetic radiation remains for persons in the Basic Service Area of an
- 15 access point of the WLAN. For example, in the case of WLANs at places of employment, such as offices, etc., there exists therefore permanent stress from electrosmog from the WLAN on the employees of the company or organization. In the state of the art there exists only the possibility of further reducing the limits for electromagnetic radiation.
- 20 It is an object of this invention to propose a new method and system for reducing electrosmog in wireless local networks which do not have the drawbacks described above. In particular a solution should be proposed which can be managed without any disruptive software and/or hardware adaptations and is thus easily achievable for existing WLAN technologies.
- 25 These objects are achieved, according to the present invention, in particular through the elements of the independent claims. Further preferred embodiments follow moreover from the dependent claims and from the description.
- In particular, these objects are achieved through the invention in that, for reducing electrosmog in wireless local area networks (WLANs), one or more mobile network units communicate with a base station in a wireless local network by means of radio frequency signals, which base station amplifies the

WO 2004/075583

7

radio frequency signals of the mobile network unit and/or connects the wireless local area network to a wired fixed network by means of bridge functions, the base station changes over from the normal transmitting-receiving mode into a sleep mode after a predefinable time interval without connecting signal to a

- 5 mobile network unit, in the sleep mode no recognition signals and/or other radio frequency signals being transmitted from the base station, the base station being ready to receive radio frequency signals, however, when needing a network connection, a mobile network unit transmits an alert signal to the base station, and upon receiving the alert signal of the mobile network unit, the base
- station transmits to the mobile network unit the recognition signals necessary for the connection and changes over into the normal transmitting and receiving mode. The invention as described above has the advantage that electrosmog in WLANs can be greatly reduced during times when there is no network activity. At the same time energy consumption is also reduced since in sleep mode no
- 15 beacon signals or other radio frequency signals are transmitted from the base stations. The whole method and system is achievable in particular without any hardware changes of any kind in the mobile network unit being necessary on the user side, nor on the side of the base stations, and it is therefore simpler and less expensive to achieve compared with other solutions. This means that
- not only are the costs for new hardware saved, but also the costs for installing it. It must also be pointed out that in mobile network units weight and space considerations often play a role too. The present invention requires neither additional hardware space, nor does it result in increased weight of the mobile terminal (network unit). For company-internal WLANs, for example, it also
- <sup>25</sup> further increases security, making it more difficult for the WLAN to be used by unauthorized persons e.g. outside of business hours since no periodic beacon signal is sent anymore by the base station or base stations if they are in sleep mode.

In an embodiment variant, when in need of a network connection, the mobile network unit transmits an alert signal only if it does not receive any recognition signal from a base station. This embodiment variant has the advantage, among other things, that no unnecessary alert signal has to be transmitted if the base station is already in normal transmitting-receiving mode.

This likewise results in a further reduction of electrosmog and at the same time energy saving in the mobile network units.

In another embodiment variant, only the base station in whose basic service area (BSA) the mobile network unit is located changes over into the normal transmitting and receiving mode, the other base stations of the wireless local network remaining in their previous operating mode. This embodiment variant has the advantage, among other things, that the electrosmog can be further reduced since for mobile units which are at times stationary, such as e.g. when working with a laptop at one's place of employment, only the needed base station goes back into the normal transmitting-receiving mode.

In still another embodiment variant, the base stations of the basic service areas (BSAs) bordering on the basic service area (BSA) of the base station in whose BSA the mobile network unit is located likewise change over automatically into the normal transmitting-receiving mode if they were

15 previously in the sleep mode. This embodiment has, among other things, the same advantages as the preceding one, but during a shift of the mobile network unit from one BSA to the next, the base station of the bordering BSA is already in the normal transmitting and receiving mode.

In an embodiment variant, the base station of the wireless local network changes over from sleep mode into the normal transmitting-receiving mode only if a network-specific recognition signal of the alert signal corresponds to a stored recognition signal of the wireless local network. This embodiment has the advantage, among other things, that the user as well as the service provider of the WLAN is given additional security. Through the

additional authentication by means of a network-specific recognition signal, an unauthorized person, such as someone outside the company in the case of company WLANs, cannot even activate the normal transmitting and receiving mode of the WLAN or respectively of the base station.

In an embodiment variant, at least parts of the network-specific recognition signal, such as e.g. supplementary information data, are definable for the wireless local network by a user of the mobile unit and/or by an operator.

This embodiment variant has, among other things, the same advantages as the preceding embodiment variant. The security can be further increased however through the addition of supplementary information data determinable by the user or operator. In an embodiment variant, these data can even be

- 5 supplementary information data freely chosen by the user, whereby, as a borderline case, the supplementary information data could even be empty. As further embodiment variants, an unambiguous identification code of the user can be used as the supplementary information data. For example, this can be an IMSI (International Mobile Subscriber Identification) and/or a MSISDN
- 10 (Mobile Subscriber ISDN) which is stored on a SIM (Subscriber Identification Module) card of the mobile network unit. This has the advantage, among other things, that a particular user can be identified by means of the MSISDN, and, if required, can be correspondingly authenticated, e.g. with a log-in password, etc., without the user having to be registered beforehand in the system, e.g. in a
- 15 database. As an additional embodiment, it is even conceivable for the MSISDN of a mobile radio device of the user to be used as the MSISDN, for example, the mobile radio device being one from which an access request was previously sent to a central unit.
- In a further embodiment variant, the alert signal is transmitted from the mobile unit in a network-independent way for each wireless local network. This embodiment variant has the advantage, among other things, that any mobile network unit can activate possibly available WLANs in a standard way, independently of a specific recognition signal, or at least can receive a beacon signal or similar signal of the network.
- In another embodiment variant, the wireless local network is set up based on the 802.X network technology, the recognition signals containing the corresponding Service Set Identifiers (SSID). This embodiment variant has the advantage, among other things, that a standardized access method and standardized physical layer specifications with the 802.X layer protocols can be used for the WLANs. This allows a cost-effective implementation without it being necessary to depart from the standard methods. At the present time the standards of the Institute of Electrical and Electronics Engineers (IEEE) have taken hold worldwide in the WLAN area. Among the IEEE standards which

have gained acceptance are in particular the IEEE 802 standards for LAN (Local Area Network) technologies.

In another embodiment variant, the wireless local network is set up based on Bluetooth technology. Among other things, this embodiment variant has the same advantages as the preceding one. In particular, Bluetooth is supported by a wide range of well-known hardware and software producers, such as e.g. Ericsson, IBM, Intel, Nokia, Toshiba, etc., which are themselves members of the Bluetooth Special Interest Group, which defines the Bluetooth standard.

10 Embodiment variants of the present invention will be described in the following with reference to examples. The examples of the embodiments are illustrated by the following attached figures:

Figure 1 shows a block diagram illustrating schematically the architecture of an embodiment variant of a method and/or system according to the invention for reducing electrosmog in wireless local networks 5, one or more mobile network units 1 communicating by means of radio frequency signals 4 with a base station 2 of a wireless local network 5, which base station 2 amplifies the radio frequency signals 4 of the mobile network unit 1 and/or connects the wireless local network 5 to a wired fixed network by means of bridge functions.

Figure 2 shows a flow chart presenting schematically the architecture of a method and/or system in a wireless local network 5, whereby a beacon signal is constantly being transmitted from the base stations 2 in order to make a potential user aware of the availability of a WLAN 5.

Figure 3 shows a flow chart presenting schematically the architecture of a method and/or system according to the invention in a wireless local network 5, the WLAN 5 having two different operating modes, such as a normal transmitting - receiving mode and a sleep mode. The figure shows in particular the course of switchover from the sleep mode into the normal transmitting -

receiving mode when a mobile network unit 1 would like to use the wireless local network 5.

Figure 1 illustrates an architecture which can be used to achieve the invention. In this embodiment example, one or more mobile network units 1
communicate by means of radio frequency signals 4 with a base station 2, or respectively an access point, of a wireless local network 5. Wireless local networks 5 are also referred to as WLANs (Wireless Local Area Networks). A WLAN can be composed of one or more such base stations or respectively access points. The base station 2 amplifies the radio frequency signals 4 of the

10 mobile network unit 1 and/or connects the wireless local network 5 by means of bridge functions to a wired fixed network. Base stations 5, or respectively access points, of a WLAN 5 can be connected e.g. via physical media such as, for instance, coaxial cable, twisted pair or fiber optic cable to assigned radius servers. The connection can comprise communication networks, such as, for

example, mobile radio networks, such as a terrestrial mobile radio network, e.g. a GSM or UMTS network, or a satellite-based mobile radio network and/or one or more fixed networks, for instance the public switched telephone network (PSTN) and/or ISDN (Integrated Services Digital Network) or a suitable LAN (Local Area Network) or WAN (Wide Area Network). During log on of a mobile

- 20 network unit 1 of a user in a WLAN 5, an identification code of the user is transmitted for authentication of the user together with supplementary information data, which can be determined by the user, via one of the APs 2 of the WLAN 5 to a central unit and/or radius server. The communication between the central unit and the access points 2 can take place e.g. via a TCP/IP
- 25 interface and/or CORBA interface, an ATM module, a SMS and/or USSD gateway by means of special short messages, for example SMS (Short Message Services), USSD (Unstructured Supplementary Services Data) messages, or other techniques such as MExE (Mobile Execution Environment), via protocols such as GPRS (Generalized Packet Radio Service), WAP
- 30 (Wireless Application Protocol) or another user information channel. The data transfer between the central unit and the access points 2 is initiated and carried out e.g. via transfer modules, implemented through software or hardware, of the central unit as well as of the access points. The mobile network units 1 or so-called mobile nodes can be e.g. laptops, notebooks, PDAs (Personal Digital

Assistants) or mobile radio devices, in particular mobile radio telephones. The mobile nodes are equipped through hardware and software with a corresponding interface in order to integrate them in a local wireless computer network (WLAN). They communicate by means of radio frequency signals with

- 5 the access points 2 of the WLAN 5. The mobile nodes 1 can comprise e.g. an adaptor, which includes a transceiver as well as a control card (such as e.g. an infrared (IR) adaptor or a low frequency radio wave adaptor). The mobile nodes 1 are thereby able to move freely within the range of the wireless LAN 5. The access points 2 of the WLAN 5 can e.g. amplify the radio frequency signals of
- the mobile node 1 as well as comprise bridge functions which make it possible to access nodes 1 of a wired LAN from the wireless local network 5 and viceversa. For transmission of the radio frequency signals, the access points 2 comprise at least one antenna. The antenna can be e.g. a dipole antenna, a loop radiator such as a folded dipole, a Marconi aerial or a ground plane
- 15 antenna, a directional antenna such as e.g. a yagi aerial, a turnstile antenna or a parabolic aerial, an omnidirectional antenna or a fractal antenna system. The radio frequency signals lie typically in the frequency bands reserved for wireless LAN between 800 MHz and 6000 MHz, such as e.g. three frequency bands set by the United States Federal Communication Commission (FCC) in the USA:
- 902-928 MHz, 2400-2483.5 MHz and 5725-5850 MHz (D 15 of Title 47, Code of Federal Regulations). They can also be in the range of 400 MHz, for example, as is common e.g. with electronic, wireless garage openers, or at the WLL (Wireless Local Loop) frequencies auctioned a year ago in Germany and Switzerland, e.g. 26 GHz for wireless local loop methods. It is to be pointed out,
- however, that other frequencies are also possible, without affecting the scope of the invention. Thus, in principle, infrared signals can also be used for the invention such as e.g. IrDA, IR-LAN, etc. The bridge functions of the base station 2 can be achieved e.g. according to IEEE standard 802.1D-1990 "Media Access Control Bridges" pp. 31-47. In the WLAN network recognition and user
- 30 identification and/or authentication complement one another. For network recognition, an AP periodically transmits so-called beacon signals within a WLAN, which signals comprise e.g. Service Set IDentifiers (SSID) and/or other control parameters for integrating a mobile network unit 1 into a wireless network. This applies in particular to the 802.X, such as e.g. the 802.11 network
- 35 technologies, but also to Bluetooth and other network technologies. Beacon

signals are thus transmitted all the time to make potential users or respectively their mobile network units 1 aware of available WLANs 5. In the present invention, however, after a predefined time interval without a connection signal to a mobile network unit 1, the base station 2 switches over from normal

5 transmitting and receiving mode to sleep mode. Understood by "normal transmitting and receiving mode" is the normal operating mode of the AP during which mobile network units 1 can access the APs or not.

In a flow chart, Figure 2 illustrates how a mobile network unit 1 recognizes the WLAN and connects thereto before the user can authenticate himself e.g. with the central unit and/or radius server. As mentioned, the base

- himself e.g. with the central unit and/or radius server. As mentioned, the base station in normal transmitting and receiving mode transmits beacon signals periodically 11. Even when no mobile network units are located in the WLAN, the beacon signals continue to be periodically transmitted from the APs. The SSID can be an unambiguous identification symbol, 32 characters long, which
- 15 is assigned to the header of data messages sent over the network and which serves as a password for the mobile network units. The SSID differs from one WLAN to another. That means that all APs and mobile network units of a particular WLAN must use the same SSID. A network unit which cannot support an unambiguous SSID will normally not be granted any network access via a
- 20 base station or respectively an AP. In the secure access mode (802.X) of the APs, the SSID from base station 2 and mobile network unit 1 must agree. In the non-secure access mode, a mobile network unit 1 can log on with the configured SSID, a blank SSID, or with the SSID set on "any." The beacon signals can be transmitted encrypted or unencrypted. The 802.11 network
- standard uses for encryption purposes WEP (Wired Equivalent Privacy), for example. WEP operates in three modes: no encryption, 40-bit encryption and 128-bit encryption. The 802.11 standard encrypts only the data packets, however, and not the management packets. The SSID is part of the beacon and probe management signal and is not encrypted when WEP is activated. A
- 30 mobile network unit 1 receives the beacon signal 13, and recognizes the WLAN 5 from the beacon. Default SSIDs of WLANs are e.g. "tsunami" - Cisco, "101" – 3Com, "RoamAbout Default Network Name" - Lucent/Cabletron, "Default SSID", "Compaq" - Compaq, "WLAN" – Addtron (a popular AP), "intel" - Intel, "linksys" – Linksys, "Wireless". Thus if a mobile network unit 1 receives a

beacon signal 13, it logs on with the corresponding AP, and carries out the authentication 14 of the user, if necessary, e.g. with the central unit, before it has access to the WLAN 5. If the mobile node 1 does not receive any beacon signal, but nevertheless needs a WLAN connection, it continues to scan for

- 5 beacon signals 15 until it has found an available WLAN. This applies to the normal transmitting and receiving mode. In the normal transmitting and receiving mode the AP automatically transmits a further beacon signal after a predefined time interval 12. In the case that a base station 2 switches over into sleep mode, no recognition signals and/or other radio frequency signals are 10 transmitted anymore from the base station 2, i.e. also no beacon signals, but
- the base station 2 nevertheless remains ready to receive radio frequency signals 4 also in sleep mode.

Figure 3 illustrates the method according to the invention on the side of the AP 2 when the base station 2 is in sleep mode. If a mobile network unit 1 needs a network connection, it transmits an alert signal which is received by the base station 2. If, in the normal transmitting and receiving mode, the base station does not receive any connection signal from a mobile network unit 1, the AP 2 waits for a predefinable period of time 24, if thereafter it still does not receive any connection signal 25, the base station 2 switches over into sleep mode 26, and waits 27 for a connection signal from a mobile node 1. Upon receiving an alert signal from a mobile network unit 1, the base station 2

transmits 22 the recognition signals necessary for the connection and/or beacon signals to the mobile network unit 1 (e.g. beacon signal), and, as described under Figure 2, carries out the authentication of the user of the

- 25 mobile network unit 1. All base stations 2 of a WLAN 5 can always switch together from sleep mode into the normal transmitting and receiving mode, or only those base stations 2 in whose basic service areas 3 the mobile network unit 1 is located, the other base stations 2 of the wireless local network 5 remaining in their previous operating mode. It can make sense in addition for
- the base stations 2 of basic service areas 3 bordering on the basic service areas 3 of the base station 2 in whose BSA the mobile node 1 is located to automatically switch over into the normal transmitting and receiving mode if they were previously in sleep mode. In an embodiment variant, the mobile network unit 1, when needing a network connection, can transmit an alert signal

only when no recognition signal is received from a base station 2, or automatically every time it needs a WLAN, for example. It is furthermore possible for the base station 2 of the wireless local network 5 to switch over from sleep mode into the normal transmitting-receiving mode only when a

- 5 network-specific recognition of the alert signal corresponds with a stored recognition signal of the wireless local network 5. This results in additional protection against unauthorized use of the WLAN. The security of the WLAN 5 can be further increased in that at least parts of the network-specific recognition signal are definable for the wireless local network 5 by the user of the mobile
- unit 1 and/or by an operator. As a special embodiment variant, the MSISDN and/or IMSI of a mobile radio device of the user of the mobile network unit 1 can be used as the supplementary information data. Moreover this can be stored on a SIM (Subscriber Identification Module) card of the mobile network unit. For other embodiments it can be important, however, that the alert signal
- 15 is transmitted from the mobile network unit 1 in a network-independent way. This could be advantageous in particular for WLANs in public buildings, airports, etc. It is important to point out that the method or respectively system according to the invention can be achieved without modification of existing hardware on the side of the base stations 1 and on the side of the mobile
- 20 network units 1, requiring only modification of the corresponding software components. Of course it is also possible to achieve the method and system according to the invention through addition of corresponding hardware modules.

### Claims

 A method for reducing electrosmog in wireless local networks, one or more mobile network units (1) communicating with a base station (2) of a wireless local network (5) by means of radio frequency signals (4), which base
 station (2) amplifies the radio frequency signals (4) of the mobile network unit (1) and/or connects the wireless local network (5) to a wired fixed network by means of bridge functions, wherein

the base station (2) changes over from the normal transmittingreceiving mode into a sleep mode after a predefinable time interval without connecting signal to a mobile network unit (1), in the sleep mode no recognition signals and/or other radio frequency signals being transmitted from the base station (2), the base station being ready to receive radio frequency signals (4), however,

when needing a network connection, a mobile network unit (1) 15 transmits an alert signal to the base station,

upon receiving the alert signal of the mobile network unit (1), the base station (2) transmits to the mobile network unit (1) the recognition signals necessary for the connection and changes over into transmitting and receiving mode.

20 2. The method according to claim 1, wherein, when in need of a network connection, the mobile network unit (1) transmits an alert signal only if it does not receive any recognition signal from a base station (2).

 The method according to one of the claims 1 or 2, wherein only the base station in whose basic service area the mobile network unit (1) is
 located changes over into the normal transmitting and receiving mode, the other base stations (2) of the wireless local network (5) remaining in their previous operating mode.

10

17

4. The method according to claim 3, wherein the base stations (2) of the basic service areas (3) bordering on the basic service area (3) of the base station (2) in whose basic service area the mobile network unit (1) is located likewise change over automatically into the normal transmitting-receiving mode if they were previously in the sleep mode.

5. The method according to one of the claims 1 to 4, wherein the base station (2) of the wireless local network (5) changes over from sleep mode into the normal transmitting-receiving mode only if a network-specific recognition signal of the alert signal corresponds to a stored recognition signal of the wireless local network (5).

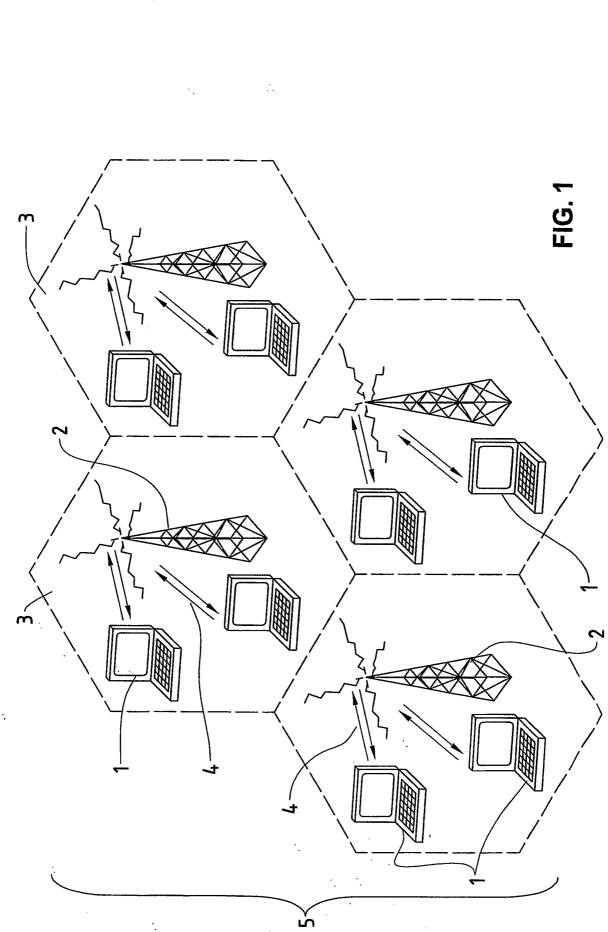
6. The method according to claim 5, wherein at least parts of the network-specific recognition signal are definable for the wireless local network(5) by a user of the mobile unit (1) and/or by an operator.

7. The method according to one of the claims 1 to 6, wherein the alert signal from the mobile network unit (1) is transmitted in a network independent way for every wireless local network (5).

8. The method according to one of the claims 1 to 7, wherein the wireless local network (5) is set up based on the 802.X network technology, the recognition signals containing the respective Service Set Identifier (SSID).

20 9. The method according to one of the claims 1 to 7, wherein the wireless local network (5) is set up based on Bluetooth technology.

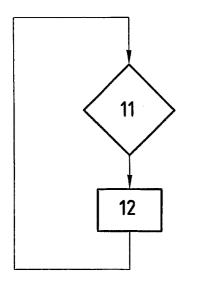
۰.



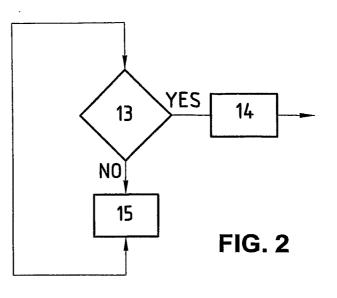
1/2

. :

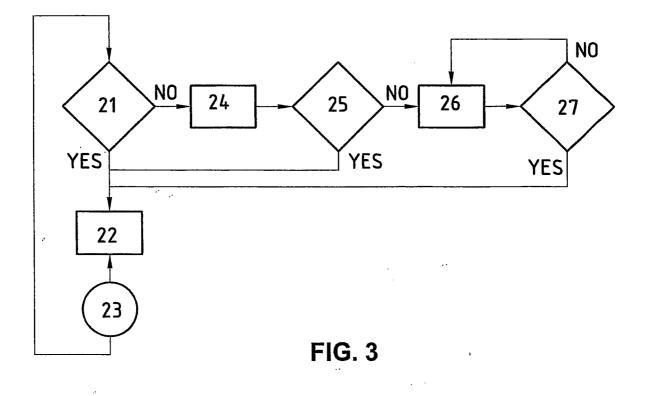
2/2



.•



· .·



### INTERNATIONAL SEARCH REPORT al Application No Internat PCT/CH 03/00138 A. CLASSIFICATION OF SUBJECT MATTER IPC 7 H0407/32 H040 H04Q7/30 According to International Patent Classification (IPC) or to both national classification and IPC **B. FIELDS SEARCHED** Minimum documentation searched (classification system followed by classification symbols) IPC 7 H04Q H04B Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched Electronic data base consulted during the international search (name of data base and, where practical, search terms used) 1.2-EPO-Internal, WPI Data C. DOCUMENTS CONSIDERED TO BE RELEVANT Category ° Citation of document, with indication, where appropriate, of the relevant passages Relevant to claim No. А US 5 884 196 A (LEKVEN ERIC J ET AL) 1 - 916 March 1999 (1999-03-16) abstract figure 2 column 6, line 11 - line 31 WO 02 093778 A (QUALCOMM INC) 1-9 А 21 November 2002 (2002-11-21) abstract paragraph '0009! - paragraph '0010! claim 1 US 6 339 694 B1 (NUCKOLS JEFFREY R ET AL) A 1 - 915 January 2002 (2002-01-15) column 3, line 43 - line 60 abstract \_/\_\_ Further documents are listed in the continuation of box C. Patent family members are listed in annex. X İX ° Special categories of cited documents : 'T' later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention "A" document defining the general state of the art which is not considered to be of particular relevance earlier document but published on or after the international "E" "X" document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to filing date "L" document which may throw doubts on priority claim(s) or which is cited to establish the publication date of another involve an inventive step when the document is taken alone "Y" document of particular relevance; the claimed invention citation or other special reason (as specified) cannot be considered to involve an inventive step when the document referring to an oral disclosure, use, exhibition or document is combined with one or more other such docu-ments, such combination being obvious to a person skilled "0" other means in the art. "P" document published priorito the international filing date but later than the priority date claimed \*& document member of the same patent family Date of the actual completion of the international search Date of mailing of the international search report 14 October 2003 22/10/2003 Name and mailing address of the ISA Authorized officer European Patent Office, P.B. 5818 Patentlaan 2 NL – 2280 HV Rijswijk Tel. (+31–70) 340–2040, Tx. 31 651 epo nl, Fax: (+31–70) 340–3016 Dionisi, M

Form PCT/ISA/210 (second sheet) (July 1992)

# INTERNATIONAL SEARCH REPORT

PCT/CH 03/00138

Α,

C.(Continuation) DOCUMENTS CONSIDERED TO BE RELEVANT								
Category °	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.						
A	WO 02 07464 A (ERICSSON TELEFON AB L M) 24 January 2002 (2002-01-24) page 2, line 5 - line 23 page 15, line 11 - line 15 	1-9						
15 m			300 2					
		4	ηa					
			1 ha					

# INTERNATIONAL SEARCH REPORT

	ling ana	tion on patent family me	mbers	۷.		Application No 03/00138
Patent document cited in search report		Publication date		Patent family member(s)		Publication date
US 5884196 WO 02093778	A	16-03-1999 21-11-2002	AU AU BR CN EP JP KR WO	717244 3569397 9709555 1228230 0903047 2000515334 2000016550 9747149 2002177461	7 A 5 A 7 A2 4 T 9 A2 4 A1	23-03-2000 05-01-1998 11-01-2000 08-09-1999 24-03-1999 14-11-2000 25-03-2000 11-12-1997 28-11-2002
ی ۱۹۹۹ و ۱۹۹۹ - ۱۹۹۹ و ۱۹۹۹ - ۱۹۹۹ و ۱۹۹۹ - ۱۹۹۹ و ۱۹۹۹ و ۱۹۹۹ و ۱۹۹۹ و ۱۹۹۹ و ۱۹۹۹ و ۱۹۹۹ و ۱۹۹۹ و ۱۹۹۹ و ۱۹۹۹ و			US US WO WO WO WO WO US US US	2002173326 2002173326 2002172165 02093788 02098015 02093954 02093954 02093948 02093812 02093778 2003008657 2002173327 2002172169	5 A1 5 A1 5 A1 5 A1 5 A1 6 A1 8 A1 8 A1 8 A1 8 A1 7 A1 7 A1	21-11-2002 21-11-2002 21-11-2002 21-11-2002 05-12-2002 21-11-2002 21-11-2002 21-11-2002 21-11-2002 21-11-2002 09-01-2003 21-11-2002 21-11-2002
US 6339694	B1	15-01-2002	NON	E		
WO 0207464	A	24-01-2002	US AU WO	6584330 7121601 0207464	L A	24-06-2003 30-01-2002 24-01-2002

ſ



ehponline.org

# Nerve Cell Damage in Mammalian Brain after Exposure to Microwaves from GSM Mobile Phones

Leif G. Salford, Arne E. Brun, Jacob L. Eberhardt, Lars Malmgren, Bertil R.R. Persson doi:10.1289/ehp.6039 (available at http://dx.doi.org/) Online 29 January 2003



The National Institute of Environmental Health Sciences National Institutes of Health Department of Health and Human Services

# Nerve cell damage in mammalian brain after exposure to microwaves from GSM mobile phones

Leif G. Salford<sup>1</sup>, Arne E. Brun<sup>2</sup>, Jacob L. Eberhardt<sup>3</sup>, Lars Malmgren<sup>4</sup>, Bertil R.R. Persson<sup>3</sup>

Depts of <sup>1</sup>Neurosurgery, <sup>2</sup>Neuropathology, <sup>3</sup>Medical Radiation Physics and <sup>4</sup>Applied Electronics, Lund University, the Rausing Laboratory and Lund University Hospital, S-22185, Lund, Sweden.

Corresponding author: Leif G. Salford Dept. of Neurosurgery Lund University Hospital S-221 85 Lund, Sweden Phone: +46 46 171270 Fax: +46 46 189287 Email: Leif.Salford@neurokir.lu.se

## **Running Title:**

Nerve Cell damage from GSM Mobile Phones

### Key words:

rats, cns, blood-brain barrier, neuronal damage, microwaves, mobile phones.

### Acknowledgements

We thank BMA Susanne Strömblad and BMA Catarina Blennow at the Rausing Laboratory for excellent technical assistance.

The work was supported by a grant from The Swedish Council for Work Life Research.

### Abbreviations

BBB	Blood-brain barrier
GSM	Gobal System for Mobile Communications
MRI	Magnetic Resonance Imaging
RF	Radiofrequency electromagnetic fields
TEM-cell	Transverse Electromagnetic transmission line cell

# **Outline:**

Abstract: page 3 Introduction: page 4 Material and Methods: page 6 Results and discussion: page 8 References: page 12

# Abstract

The possible risks of radio-frequent electromagnetic fields for the human body, is a growing concern for the society. We have earlier shown that weak pulsed microwaves give rise to a significant leakage of albumin through the blood-brain barrier (BBB). Now we have investigated whether a pathological leakage over the BBB might be combined with damage to the neurons. Three groups of each 8 rats were exposed for 2 hours to GSM mobile phone electromagnetic fields of different strengths. We found, and present here for the first time, highly significant (p<0.002) evidence for neuronal damage in both the cortex, the hippocampus and the basal ganglia in the brains of exposed rats.

### Introduction

The largest human biological experiment ever. So has the voluntary exposure of the brain to microwaves from handheld mobile phones by one fourth of the world's population been called (Salford et al.2001).

Within the near future microwaves will be emitted also by an abundance of other appliances in the cordless office and also in the home. The possible risks of radiofrequency electromagnetic fields (RF) for the human body, is a growing concern for the society. For a review see Hyland (Hyland 2000). Most researchers in the field have dwelled on the question whether RF may induce or promote cancer growth. Some have indicated increased risk (Hardell et al.2002; Repacholi et al.1997) while most studies including our own have shown no effects (Salford et al.1997a) or even a decreased risk (Adey et al.1999)

The possible risks of microwaves for the human body has attracted interest since the 1960-ies, e.g. before the advent of mobile phones, when radar and microwave ovens posed a possible health problem. Oscar and Hawkins early performed studies on effects of RF upon the BBB (Oscar and Hawkins1977). They demonstrated that at very low energy levels (< 10 W/m<sup>2</sup>), the fields in a restricted exposure window caused a significant leakage of <sup>14</sup>C mannitol, innulin and also dextran (same molecular weight as albumin) from the capillaries into the surrounding cerebellar brain tissue. These findings, however, were not repeated in a study using <sup>14</sup>C-sucrose (Gruenau 1982). In a recent in-vitro study it has been shown that EMF at 1.8 GHz increases the permeability to sucrose of the BBB (Schirmacher et al. 2000). Shivers (Shivers et al.1987; Prato et al.1990) examined the effect of MRI upon the rat brain. They showed that the combined exposure to RF, pulsed and static magnetic fields gave rise to a significant pinocytotic transport of albumin from the capillaries into the brain.

Inspired by this work, our group has since 1988 studied the effects of different intensities and modulations of 915 MHz RF in a rat model where the exposure takes place in a TEM-cell during various time periods. In series of more than 1600 animals, we have proven that subthermal energies from both pulse-modulated and continuous RF fields – including

those from real GSM mobile phones - have the potency to significantly open the BBB for the animals' own albumin (but not fibrinogen) to pass out into the brain and to accumulate in the neurons and glial cells surrounding the capillaries (Malmgren1998; Persson et al.1997; Persson and Salford 1996; Salford et al.1992, 1993, 1994, 1997b, 2001) (fig 1). These results are duplicated recently in another laboratory (Töre et al. 2001). Similar results are found by others (Fritze et al.1997).

We and others (Oscar and Hawkins1977; Persson et al.1997) have pointed out that when such a relatively large molecule as albumin may pass the BBB, also many other smaller molecules, including toxic ones, may escape into the brain due to the exposure to RF. We have hitherto not concluded that such leakage is harmful for the brain. It is shown by Hassel, however, that autologous albumin injected into the brain tissue of rats, leads to damage to neurons at the injection site when the concentration of albumin in the injected solution is at least 25% of that in blood (Hassel et al.1994). In the present study, we have investigated whether leakage over the BBB might cause damage to the neurons.

### **Material and Methods**

A Transverse Electromagnetic transmission line cell (TEM-cell) used for the RF exposure of rats was designed by dimensional scaling from previously constructed cells at the National Bureau of Standards (Crawford1974). TEM-cells are known to generate uniform electromagnetic fields for standard measurements. A genuine GSM mobile phone with a programmable power output is connected via a coaxial cable to the TEM-cell. No voice modulation was applied.

The cell is enclosed in a wooden box  $(15 \times 15 \times 15 \text{ cm})$  that supports the outer conductor and central plate. The outer conductor is made of brass-net and is attached to the inner walls of the box. The centre plate, or septum, is constructed of aluminium.

The TEM-cells are placed in a temperature-controlled room and the temperature in the TEM-cells kept constant by circulating room air through holes in the wooden box.

The SAR-distribution in the rat brain has been simulated with the FDTD-method (Martens et a. 1993) and found to vary less than 6 dB in the rat brain.

The rats are placed in plastic trays  $(12 \times 12 \times 7 \text{ cm})$  to avoid contact with the central plate and outer conductor. The bottom of the tray is covered with absorbing paper to collect urine and faeces.

Thirty-two male and female Fischer 344 rats aged 12 - 26 weeks and weighing  $282 \pm$  91 g were divided into 4 groups of each 8 rats. The peak output power from the GSM mobile telephone fed into two TEM-cells simultaneously for 2 hours were 10 mW, 100 mW and 1000 mW per cell, respectively. This exposed the rats to peak power densities of 0.24. 2.4 and 24 W/m<sup>2</sup>, respectively. This exposure resulted in average whole-body specific absorption rates (SAR) of 2 mW/kg, 20 mW/kg and 200 mW/kg, respectively. For further details about exposure conditions and SAR calculations, see (Martens et al. 1993; Malmgren 1998). The fourth group of rats was simultaneously kept for 2 hours in non-activated TEM-cells. The animals were awake during the exposure and could move and turn within the exposure chamber.

The animals in each exposure group were allowed to survive for about 50 days after exposure. They were carefully observed daily for neurological or behavioural abnormalities during this period at the end of which they were anaesthetized and sacrificed by perfusion-fixation with 4% formaldehyde.

The brains were removed from the skull by non-traumatic technique (resection of bone structures at the skull base, followed by a midline incision from the foramen magnum to the

nose) after an extended in situ post mortem fixation time of 30 minutes. Each brain was sectioned coronally in 1-2 mm thick slices, which all were embedded in paraffin and cut at 5 micrometer, stained for RNA/DNA with cresyl violet to show dark neurons. Applying albumin antibodies (Dakopatts), albumin is revealed as brownish spotty or more diffuse discolorations (Figs 1a and b). The microscopical analysis was performed blind to the test situation.

The occurrence of "dark neurons" was judged semi-quantitatively by the neuropathologist as 0 (no or occasional dark neurons), 1 (moderate occurrence of dark neurons) or 2 (abundant occurrence). The Kruskal Wallis one-way analysis of variance by ranks was used for a simultaneous statistical test of the score distributions for the 4 exposure conditions. When the null hypothesis could be rejected, comparisons between controls and each of the exposure conditions was made with the Mann-Whitney non-parametric test for independent samples.

### **Results and discussion**

Controls and test animals alike showed the normal diffuse positive immuno-staining for albumin in hypothalamus, a kind of built-in method control.

Control animals showed either no or an occasional and often questionable positivity for albumin outside the hypothalamus. In one animal a moderate amount of dark neurons were observed while in all the other animals no such change was present.

Exposed animals usually showed several albumin positive foci around the finer blood vessels in white and gray matter. Here the albumin had spread in the tissue in between the cell bodies, and surrounded neurons, which were either free of albumin or in some foci containing albumin. Also scattered neurons, not associated with albumin leakage between the neurons, were positive.

The cresyl violet staining revealed scattered and grouped dark neurons, which were often shrunken and dark staining, homogenised with loss of discernible internal cell structures. Some of these dark neurons were also albumin positive or showed cytoplasmic microvacuoles indicating an active pathological process. There were no haemorrhages and no discernible glial reaction, astrocytic or microglial, adjacent to changed neurons. Changed neurons were seen in all locations, but especially the cortex, hippocampus and basal ganglia, mixed in among normal neurons (fig 2). The percentage abnormal neurons is roughly appreciated to be maximally around 2 %, but in some restricted areas dominated the picture.

The occurrence of dark neurons under the different exposure conditions is shown in figure 3 which shows a significant positive relation between EMF dosage (SAR) and number of dark neurons.

A combined non-parametric test for the 4 exposure situations simultaneously revealed that the distributions of scores differed significantly between the groups (p<0.002).

We present here for the first time evidence for neuronal damage caused by nonthermal microwave exposure. The cortex as well as the hippocampus and the basal ganglia in the brains of exposed rats contain damaged neurons. We realise that our study comprises few animals, but the combined results are highly significant and exhibit a clear dose-response relation.

The observed dark neurons are deemed not to be artefacts for the following reasons. The brains were perfusion fixed in situ and removed atraumatically. The dark neurons were intermingled with normal appearing neurons (see fig 2a,b). Further, the presence of vacuoles in several of the dark neurons is a clear sign that damage occurred in the living animal. We cannot exclude that the neuronal change described may represent apoptotic cell death.

The neuronal albumin uptake and other changes described would seem to indicate a serious neuronal damage, which may be mediated through organelle damage with release of

not only hydrolytic lysosomal enzymes but also e.g. sequestered harmful material, such as heavy metals, stored away in cytoplasmatic organelles (lysosomes).

The time between last exposure and sacrifice is of great importance for the detection of foci of leakage since extra-vasated albumin rapidly diffuses down to, and beyond, concentrations possible to demonstrate accurately immunohistologically. However, the initial albumin leakage into the brain tissue (seen within hours in about 40% of exposed animals in our previous studies) may start a secondary BBB opening, leading to a vicious circle – as we demonstrate albumin leakage even 8 weeks after the exposure.

The reason for our choice of 12 to 26 weeks old rats is that they are comparable to human mobile phone addicted teen-agers with respect to age. The situation of the growing brain might deserve special concern from the society since biological and maturational processes are particularly vulnerable. The intense use of mobile phones by youngsters is a serious memento. A neuronal damage of the kind, here described, may not have immediately demonstrable consequences, even if repeated. It may, however, in the long run, result in reduced brain reserve capacity that might be unveiled by other later neuronal disease or even the wear and tear of ageing. We can not exclude that after some decades of (often), daily use, a whole generation of users, may suffer negative effects maybe already in their middle age.

# **Figure Legends**

Fig. 1 . (a) Slightly enlarged cross section of central parts of the brain of an unexposed control rat, stained for albumin which appears brownish in the central inferior parts of the brain, the hypothalamus, a normal feature. In the left lower corner (arrow) a brown spot representing an occasional focal leakage.

(b) As (a) for an RF exposed rat, stained for albumin, which appears brownish in multiple small foci representing leakage from many vessels.

Fig. 2. (a) Row of nerve cells in a section of the pyramidal cell band of the hippocampus in a RF exposed rat. Among the normal big and pale blue nerve cells there are interspersed black and shrunken nerve cells, so called dark neurons . Microscopical picture stained with Cresyl violet, high magnification

(b) The cortex of an RF exposed rat, showing normal nerve cells pale blue, intermingled with abnormal, black and shrunken " dark neurons " at all depths of the cortex but least in the superficial upper layers. Microscopical picture stained with Cresyl violet, high magnification.

Fig 3. Distribution of scores for the occurrence of "dark neurons" as function of exposure condition. The dotted line connects mean values for each condition. A simultaneous non-parametric comparison of all 4 conditions revealed significant differences (p<0,002). The p-values in the figure depict comparisons between each experimental condition and controls.

## **Reference List**

Adey W, Byus C, Cain C, Higgins R, Jones R, Kean C et al. 1999. Spontaneous and Nitrosourea-induced Primary Tumors of the Central Nervous System in Fisher 344 rats exposed to 836 MHz Modulated Microwaves. Radiat Res 152:293-302.

Crawford M. 1974. Generation of standard EM field using TEM transmission cells. IEEE Trans Electromagn Compat EMC-16:189-195.

Fritze K, Sommer C, Schmitz B, Mies G, Hossman K, Kiessling M et al. 1997. Effect of global system for mobile communication (GSM) microwave exposure on blood-brain barrier permeability in rat. Acta Neuropathol (Berlin) 94:465-470.

Gruenau SP, Oscar KJ, Folker MT, Rapoport SI . 1982. Absence of microwave effect on blood-brain-barrier permeability to [C-14]-labeled sucrose in the conscious rat. Experimental Neurology 75: 299-307.

Hardell L, Hallquist A, Hansson Mild K, Carlberg M, Påhlson A, Lilja A. 2002. Cellular and Cordless telephones and the risk for brain tumours. European Journal of Cancer Prevention 11:377-386.

Hassel B, Iversen E, Fonnum F. 1994. Neurotoxicity of Albumin in-vivo. Neuroscience Letters 167:29-32.

Hyland G. 2000. Physics and Biology of Mobile Telephony. Lancet 356:1833-1836.

Malmgren L. 1998. Radio frequency systems for NMR- imaging-Coil development and studies of non-thermal biological Effects. Series of Licentiate and Doctoral Theses, No. 6, Department of Applied Electronics, Lund University, Lund, Sweden.

Martens L, Van Hese J, De Sutter D, De Wagter C, Malmgren L, Persson BRR, Salford LG. 1993. Electromagnetic field calculations used for exposure experiments on small animals in TEM-cells. Bioelectrochemistry and Bioenegetics 30:73-81

Oscar K, Hawkins T. 1977. Microwave alteration of the blood-brain barrier system of rats. Brain Res 126:281-293.

Persson B, Salford L. 1996. Permeability of the blood-brain barrier in rats induced by continuous wave and pulse-modulated 915 MHz electromagnetic radiation exposure in TEM-cells. (Chiabrera A, Juutilainen J, eds). Brussel:EU DG XIII,66-72.

Persson B, Salford L, Brun A. 1997. Blood-brain barrier permeability in rats exposed to electromagnetic fields used in wireless communication. Wireless Networks 3:455-461.

Prato F, Frappier J, Shivers R, Kavaliers M, Zabel P, Drost D et al. 1990. Magnetic resonance imaging increases the blood-brain barrier permeability to 153-gadolinium diethylenetriaminepentaacetic acid in rats. Brain Res 523:301-304.

Repacholi M, Basten A, Gebski V, Noonan D, Finnie J, Harris A. 1997. Lymphomas in Eµ-*Pim1* Transgenic Mice Exposed to Pulsed 900 MHz Electromagnetic Fields. Radiat Res 147:631-640.

Salford LG, Brun A, Eberhardt J, Malmgren L, Persson B. 1992. Electromagnetic fieldinduced permeability of the blood-brain barrier shown by immunohistochemical methods. In: Interaction Mechanism of Low-Level Electromagnetic Fields in Living Systems (Nordén B, Ramel C, eds). Oxford:Oxford University Press,251-258.

Salford LG, Brun A, Eberhardt J, Persson B. 1993. Permeability of the blood-brain barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50, 200 Hz. Bioelectrochemistry and Bioenergetics 30:293-301.

Salford LG, Brun A, Sturesson K, Eberhardt J, Persson B. 1994. Permeability of the Blood-Brain barrier Induced by 915 MHz Electromagnetic Radiation, Continuous Wave and Modulated at 8, 16, 50, and 200 Hz. Microscopy Research and Technique 27:535-542.

Salford LG, Brun A, Persson B. 1997a. Brain tumour development in rats exposed to electromagnetic fields used in wireless communication. Wireless Networks 3:463-469.

Salford LG, Persson B, Brun A. 1997b. Neurological Aspects on Wireless Communication. In: Non-Thermal effects of RF Electromagnetic Fields. Non-Thermal effects of RF Electromagnetic Fields (Bernhardt JH, Matthes R, Repacholi MH, eds). Munich, Germany:International Commission on Non-Ionizing Radiation Protection,131-143.

Salford LG, Persson B, Malmgren L, Brun A. 2001. Téléphonie Mobile et Barrièrre Sang-Cerveau. In: Téléphonie Mobile - Effets Potentiels sur la Santé des Ondes Èlectromagnétiques de Haute Fréquence. (Pietteur M, ed). Embog, Belgium. 141-152.

Schirmacher A, Winters S, Fischer S, Goeke J, Galla HJ, Kullnick U, et al. 2000. Electromagnetic fields (1.8 GHz) increase the permeability to sucrose of the blood-brain barrier in vitro. Bioelectromagnetics 21: 338-345. Shivers R, Kavaliers M, Teskey G, Prato F, Pelletier R. 1987. Magnetic resonance imaging temporarily alters blood-brain barrier in the rat. Neuroscience Letters 76:25-31.

Töre F, Dulou P-E, Haro E, Veyret B, Aubineau P. 2001. Two-hour Exposure to 2 W/kg, 900 MHz GSM microwaves induces Plasma Protein Extravasation in Rat Brain. In: Proceedings from the 5th International Congress of the European Bioelectromagnetics Association, 6 September 2001 (Hietanen M, Jokela K, Juutilainen, J, eds). Finnish Institute of Occupational Health, Helsinki , 43-45.

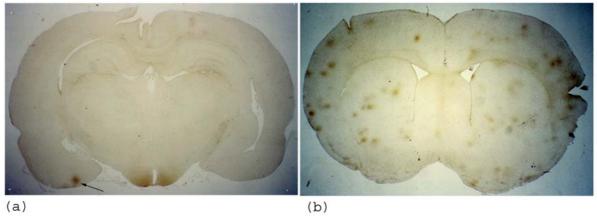


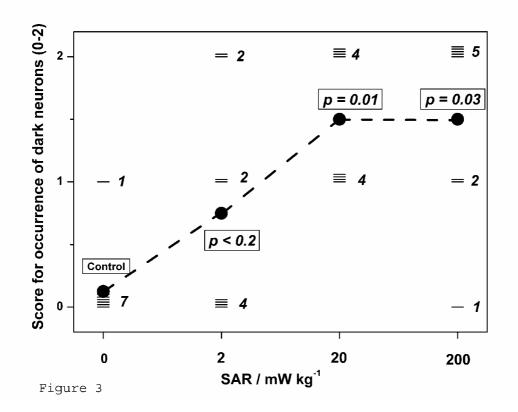


Figure 1

b) a)

Figure 2

16



# The Mammalian Brain in the Electromagnetic Fields Designed by Man-with Special Reference to Blood-Brain Barrier Function, Neuronal Damage and Possible Physical Mechanisms

Leif G. SALFORD,<sup>1,\*)</sup> Henrietta NITTBY,<sup>1</sup> Arne BRUN,<sup>2</sup> Gustav GRAFSTRÖM,<sup>3</sup> Lars MALMGREN,<sup>4</sup> Marianne SOMMARIN,<sup>5</sup> Jacob EBERHARDT,<sup>3</sup> Bengt WIDEGREN<sup>6</sup> and Bertil R. R. PERSSON<sup>3</sup>

<sup>1</sup>Department of Neurosurgery, Lund University, Sweden
 <sup>2</sup>Neuropathology, Lund University, Sweden
 <sup>3</sup>Medical Radiation Physics, Lund University, Sweden
 <sup>4</sup>Applied Electronics, Lund University, Sweden
 <sup>5</sup>Department of Plant Biochemistry, Lund University, Sweden
 <sup>6</sup>Tumour Immunology, Lund University, Sweden

<sup>\*)</sup> Corresponding author. E-mail: Leif.Salford@med.lu.se

Life on earth was formed during billions of years, exposed to, and shaped by the original physical forces such as gravitation, cosmic irradiation, atmospheric electric fields and the terrestrial magnetism. The Schumann resonances at 7.4 Hz are an example of oscillations possibly important for life.<sup>1)</sup>

The existing organisms are created to function in harmony with these forces. However, in the late 19th century mankind introduced the use of electricity, in the early 20th century long-wave radio and in the 1940-ies short-wave radio. High frequency RF was introduced in the 50-ies as FM and television and during the very last decades, microwaves of the modern communication society spread around the world. Today, however, one third of the world's population is owner of the microwave-producing mobile phones and an even larger number is exposed to the cordless RF emitting systems. To what extent are all living organisms affected by these, almost everywhere present radio frequency fields? And what will be the effects of many years of continuing exposure?

Since 1988 our group has studied the effects upon the mammalian blood-brain barrier (BBB) in rats by non-thermal radio frequency electromagnetic fields (RF-EMF). These have been shown to cause significantly increased leakage of the rats' own blood albumin through the BBB of exposed rats, at energy levels of 1W/kg and below, as compared to non-exposed animals in a total series of about two thousand animals.<sup>2)-6)</sup> One remarkable observation is the fact that the lowest energy levels, with whole-body average power densities below 10mW/kg, give rise to the most pronounced albumin leakage. If mobile communication, even at extremely low energy levels, causes the users' own albumin to leak out through the BBB, also other unwanted and toxic molecules in the blood, may leak into the brain tissue and concentrate in and damage the neurons and glial cells of the brain.

In later studies we have shown that a 2-h exposure to GSM 915 MHz, at non-thermal SAR-values of 0.2, 2 and 200 mW/kg, gives rise to significant neuronal damage, seen not only 50 days after the  $exposure^{7}$  but also after 28 days but not after 14 days. Albumin extravasations and uptake into neurons was enhanced after 14 days, but not after 28.<sup>8</sup>

In our continued research, also the non-thermal effects on tissue structure and memory function of long-term exposure for 13 months are studied.<sup>9)</sup> We have also performed microarray analysis of brains from rats exposed to short term GSM both at 1,800 MHz and at 900MHz and have found significant effects upon gene expression of membrane associated genes as compared to control animals.<sup>10),11</sup>

Most of our findings support that living organisms are affected by the non-thermal radio frequency fields. Some other studies agree while others find no effects.

The mechanisms by which the EMFs may alter BBB permeability are not well understood. At low field strengths, the effects on body temperature are negligible and thus heating effects are not involved. A change in the physicochemical characteristics of membranes has been suggested as a cause.<sup>12)</sup>

We have performed experiments to verify a quantum mechanical model for interaction with protein-bound ions. Our results show that controlled frequency and amplitude of ELF EM fields upon spinach plasma vesicles can steer transport over the membrane.<sup>13)</sup> This may be a first proof of a resonance phenomenon where appropriate levels of frequency and amplitude in the right combination have the potency to communicate with the biology of membranes and transport systems. Our study has prompted us to elaborate on magnetic resonance models; the Ion Cyclotron Resonance (ICR) model and the Ion Parametric Resonance (IPR) Model in an attempt to explain the occurrence of resonance frequencies. This is extensively described here under the heading: Mechanisms behind the effects of electromagnetical fields upon biology.

We also bring forward the concept of solitons being active in membranes and DNA/RNAtranscription as a possible mean to understand and prove the biological effects of EMF.

The Nishinomiya-Yukawa International and Interdisciplinary Symposium 2007 raised the question: What is Life? An obvious and simple answer could be: It is DNA!

The DNA strand can be looked upon as an antenna resonating in the microwave band 6GHz with its harmonics and subharmonics.<sup>14)-18)</sup> If this holds true, the dramatic situation might exist, that all living organisms have a receptor for the newly constructed and world-wide man-made microwaves, leading to a direct effect upon the function of DNA - in concordance with our experimental findings!

Our generation invented the microwave emitters. We now have an imperative obligation to further investigate the links between EMF and biology in order to prevent possible detrimental effects of the microwaves.

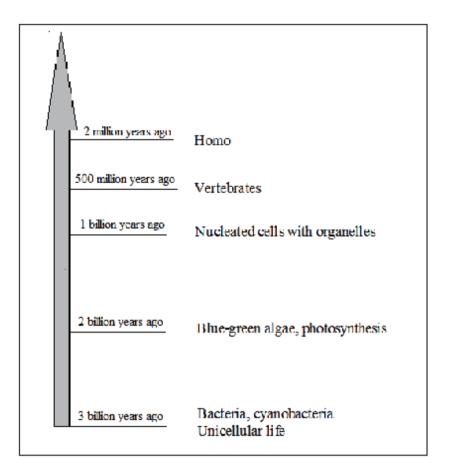


Fig. 1. Time-line for the origin of life (for a more detailed time tree, see Williams 2007).

### §1. Introduction

Our Universe was born in the "Big Bang" approximately 15 billion years ago, our sun and most of the stars were formed 10 billion years later.

Four and a half billion years ago our Earth was formed and already 1.5 billion years after this, the earliest unicellular life/bacteria/cyanobacteria started life on Earth.

Two and a half billion years ago the first photosynthesis by blue-green algae took place and 1 billion years ago the first nucleated cells with organelles emerged. This was followed 500 million years ago by the creation of the first vertebrates and they finally lead to the development of mammals and then, 2 million years ago, the emergence of our own species, Homo.

Since its origin, life on Earth has been exposed to, and shaped by, the original physical forces such as gravitation, cosmic irradiation, atmospheric electric fields and the terrestrial magnetism.

Life has also developed in a multitude of cyclic events occurring with different intervals: Earth's own rotation (1 day), Earth's revolution around the sun (1 year),

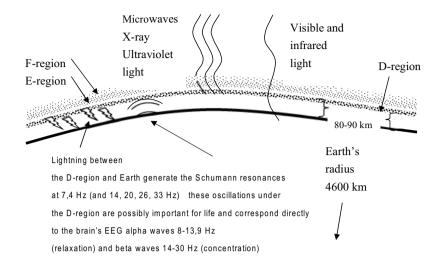


Fig. 2. Ionosphere and Schumann resonances.

the sun's rotation around its own axis (27 days), the synodic period of the moon (29.5 days) and further, the magnetic storms generated by the solar flare generating solar winds with plasma flows which appear 10 times in a month and vary with an eleven year periodicity. These magnetic storms produce alterations of the Earth's geomagnetic field (GMF) lasting from hours to days all around the Earth. The GMF forms an extremely important shield around the Earth, the magnetosphere with its magnetosheath, preventing the solar wind to reach Earth's surface at a harmful level. The protective effect of the magnetosheath can be seen as the solar wind approaches the magnetosphere, where it drops abruptly. A shock wave, known as a bow shock, develops, reminding of the waves in front of a ship travelling through the water, and thus the solar wind deflects around the magnetosphere.

Earth is surrounded by its thin atmosphere reaching only about 180 km above its surface. In parallel with this exists the 3-layered ionosphere (Fig. 2), with its innermost D-region surrounding Earth 80-90 km above its surface. Between 100 and 150 km is the E-region and between 150 and 180 km the F-region. The existence of the ionosphere is an absolute prerequisite for the development and persistence of life.

The enhanced X-rays from solar flares, extreme ultraviolet and all other forms of ultraviolet light are prevented from reaching Earth by the ionosphere whilst visible light and infrared rays pass it.

Ionized particles (mainly protons and electrons) and the enhanced X-rays from solar flares are prevented from reaching Earth by the ionosphere. Short wave ultraviolet radiation is absorbed by the ozone-layer in the stratosphere, whilst longer wave UV-radiation, visible light and infrared rays pass it.

The level of naturally occurring microwaves at the Earth's surface is extremely low. High frequency microwaves are stopped by the ionosphere, especially its Dregion. This function is of importance for the conclusions drawn in this presentation. Natural extremely low frequency electromagnetic fields are formed by electrical discharges in the atmosphere due to the resonance cavity formed by the surface of the Earth and the charged ionosphere resonances occur. These resonance frequencies are named after W. O. Schumann who already 1952 predicted their existence, and were recorded in 1960 by Balser and Wagner.<sup>30</sup>

The Schumann resonances at 7.8, 14, 20, 26, 33, 39, and 45  $Hz^{21)-23}$  are examples of natural oscillating electromagnetic fields of importance. It is possible that these resonances with their frequency predominantly at 7.8 Hz but also at 14-45 Hz, have played — and play — a role in the tuning of the spontaneous frequencies of the mammalian brain, where the frequency during relaxation is around 8 to 14 Hz, and during concentration 14-30 Hz.

Natural extremely low frequency ELF magnetic fields are also generated by the currents in the electrical discharges between clouds and the surface of the Earth.<sup>24</sup>) The daily variation of these ELF magnetic fields is strongly correlated to variations in the atmospheric magnetic field.<sup>25</sup>)

The always present geomagnetic field (GMF) of the Earth is a prerequisite for life. It not only shields us from the solar wind, but also has direct functions for life such as orientation of pigeons,<sup>26)</sup> plant branching, orientation of root branches and shielding of the geomagnetic field causes biological alterations such as decrease of the vital functions in bacteria and effects upon meristem (cf. stemcells in animals) of seedling roots of pea, flax and lentil and electron microscopy reveals changes in the mitochondrial structure.<sup>27)</sup>

Evidence has also been brought forward that we have endogenous internal rhythms in blood pressure and heart rate, which are close to, however not identical to, the period length of the rhythms in the solar wind. So, it has been proposed, that these were installed genetically by natural selection at some time in the distant geological past.<sup>28)</sup> It has also been shown that magnetic storms cause additional biological dysfunctions. Thus, bacterial bioluminescent intensity varies according to the amplitude and duration of the MSs. Further, medical studies correlate MSs with anxiety and irritability and lower attention and accuracy, with an increment of the probability of road accidents<sup>29)</sup> and aviation accidents.<sup>30)</sup> Also, acute attacks of cardiovascular diseases, such as myocardial infarction and stroke, become more frequent.<sup>31)</sup>

We have to conclude that the existing organisms are created to function in harmony with the abovementioned fields and forces which existed when life was born 3 billion years ago. And so was the situation until the generation of our grandparents. They invented the wonders of our modern life. Thus, in the late 19th century mankind introduced the use of electricity. Until then the ELFs, extremely low frequency electromagnetic fields, were represented on Earth principally only by the Schumann resonances. But now Tesla constructed the induction motor, Morse introduced the long-range telegraph, Bell the telephone, Edison developed the commercial electrical networks and electricity spread around the globe. Marconi introduced the wireless receiver 1896 and in the early 20th century long-wave radio and in the 1940-ies short-wave radio appeared.

Compared to the estimated natural background level of natural ELF magnetic fields below 1 pT/Hz ( $10^{-12}$  T/Hz) for which the previous generations of human

beings had been exposed, the average exposure in the modern world is about 100 000 times higher!

### §2. Microwaves

In 1964 Penzias and Wilson discovered the cosmic microwave background (CMB) which fills the whole universe and which originates from the Big Bang. Also ongoing cosmic processes in for example intergalactic gas clouds with temperatures of about 30°K contribute to some cosmic microwaves. But microwaves are heavily attenuated by the ionosphere and the atmosphere. Thus the natural electromagnetic background radiation in radiofrequency and the microwave band is extremely low at the Earth's surface.

The integrated spectral distribution of the microwave background in space results in a power density of about 0.4  $\mu$ W m<sup>-2</sup>. A great deal of this radiation is thus reflected by the Earth's magneto- and ionosphere or is absorbed by water and other molecules in the atmosphere. A rough estimate of the power density of CMB at the Earth's surface varies from  $10^{-21}$  to  $10^{-14}$  Wm<sup>-2</sup> equivalent to  $10^{-15}$ - $10^{-8c}$   $\mu$ Wm<sup>-2</sup>. This level of radiation is extremely low and extremely sensitive measuring equipment is required for its recording.

Thus microwaves had so far been extremely low on Earth's surface, but in the 1950-ies high frequency RF was introduced as FM and television and during the very last decades, microwaves of the modern communication society spread around the world for the first time and now exceed the natural levels by many orders of magnitude (Table I).

Today one third of the world's population owns the microwave-producing mobile phones and an even larger number is exposed to the cordless RF emitting systems ("passive mobile phoning"<sup>5</sup>). To what extent are all living organisms affected by these new, almost everywhere present radio frequency fields? And what will be the effects of many years of continuing exposure?

Table I. Incident energy from a spectrum of sources of electromagnetic energy. These are not actually measured values. They are guideline values set by authorities. (For microwave ovens U.S. Food and Drug Administration since 1971). The actual standard 5 mW/cm<sup>2</sup> = 50 W/m<sup>2</sup> at 5 cm from oven surface, 0.5 mW/m<sup>2</sup> at 50 cm at 2.45 GHz corresponds to 10 W/m<sup>2</sup> = 2W/kg, and 50 W/m<sup>2</sup> = 10 W/kg.

Source	Energy flux density (W/m2)
Natural Background	$< 10^{-14}$
Microwave oven, RF leakage standard	
5  cm for surface	50
50 cm from surface	0.5
Cell telephone (2 GHz) public guideline	10
Cell telephone (850 MHz) public guideline	4.3
RF levels near cellular base antenna (calculated) <sup>*)</sup>	0.05

<sup>\*)</sup>Typical E-field levels in proximity to cellular telephone base stations (< 200 m).<sup>32)</sup>

These questions are extremely important to answer. Our generation and our children are the first to be exposed during a lifetime to the microwaves, which are exponentially increasing underneath the ionosphere which was intended to prevent their access to Earth, at least partially.

Scientists have studied the effects of ELF and MW since the 60-ies, and an abundance of reports have emerged, especially during recent years, many of them demonstrating significant effects upon biology and health, while others have failed to show effects. In this communication we will summarize the results of some of our work in the field since 1988 and also comment to a lesser extent upon the work of other research groups. During recent years, several scientific reports in respected journals have shown significant, but often weak, effects upon cells *in vitro*, experimental animals and also humans (for reference see 33)-35)).

Recent epidemiological studies indicate that long term exposure might increase the risk for some tumour forms (for review see 36)). In a Swedish case-control study it was reported that the use of analogue and digital cellular telephones and cordless phones was correlated to an increased risk for malignant brain tumours. Regarding the use of digital cellular telephones, an odds ratio of 1.9 was observed and with a > 10-year latency period this odds ratio was increased to  $3.6.^{37}$ 

It has also been shown that mobile phone emission modulates (with increase in some cases, and decrease in others) inter hemispheric functional coupling of EEG alpha rhythms.<sup>38)</sup>

The mechanisms through which the electromagnetic fields exert their effect upon cells and organisms are not well understood. This may be part of the reason why the results of different laboratories diverge and it should be pointed out that it is as important to reveal the mechanisms as it is to demonstrate their effects upon biology. In this publication we also dwell at some length at the theoretical models trying to explain the biological effects of EMF in relation to our own experiments on EMF steering of calcium passage over spinach plasma vesicle membranes.

### §3. The Blood Brain Barrier (BBB) of the mammalian brain

Since 1988 our group has studied the effects of RF electromagnetic fields upon the blood-brain barrier (BBB) and we have collected an extensive experimental experience in this field. RF electromagnetic fields have been revealed to cause significantly increased leakage of albumin through the BBB of exposed rats as compared to non-exposed animals — in a total series of about two thousand animals. We have exposed rats to various magnetic and electromagnetic fields, as well 915 MHz continuous wave (CW) as pulse-modulated at various repetition rates (50-200 pulses per s), and we have confirmed these findings in our laboratory in follow-up studies with real GSM-900 and GSM-1800 exposures.<sup>2),3),5)-7),39)</sup>

The mammalian brain is protected from exposure to potentially harmful compounds in the blood by the blood-brain barrier (Fig. 3). Being formed by the vascular endothelial cells of the capillaries in the brain, this hydrophobic barrier maintains and regulates the very sensitively tuned environment within the mammalian brain.

The blood-brain barrier is a highly complex system, in which several kinds of

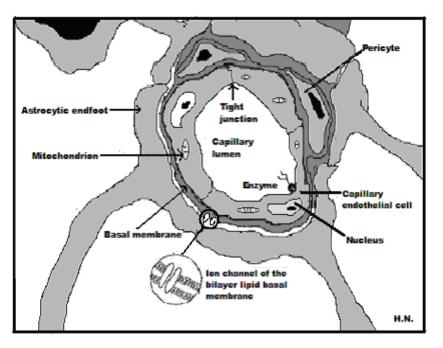


Fig. 3. The blood-brain barrier.

cells exert a wide range of functions. Some of the main characteristics are described below.

- The cell-to-cell contacts between the capillary endothelial cells are sealed with tight junctions, forming a permeability barrier, which is much more selective as compared to the fenestrated sealing of other capillaries.

- The outer surface of the endothelial cells is surrounded by protrusions (end feet) from astrocytes. Thereby, the endothelial cells and the neurons are connected and also, a second hydrophilic barrier is formed. Also, the astrocytes are implicated in the maintenance, functional regulation and repair of the blood-brain barrier.

- A bilayer basal membrane supports the ablumenal surface of the endothelial cells. This membrane might also further restrict the passage of macromolecules into the brain parenchyma.

- Pericytes are other periendothelial accessory structures of the blood-brain barrier. These have capacity for phagocytosis as well as antigen presentation and in fact, they seem to contribute significantly to the immune mechanisms of the central nervous system.<sup>40)</sup>

In addition to these structural properties of the blood-brain barrier, there are also several physiological characteristics of major importance, e.g. the high number of mitochondria within the endothelial cells (five-fold higher as compared to muscular endothelial cells of rats)<sup>41)</sup> and also, the low number of pinocytotic vesicles for nutrient transport through the endothelial cytoplasm. These are properties, which speak in favour for an energy-dependent transcapillary transport system. Of importance in the context of the blood-brain barrier permeability restriction, is also the enzymatic barrier of the cerebral endothelium, which metabolizes drugs and nutrients and thereby prevent their passage into the brain parenchyma.<sup>42)</sup>

Taken together, all these characteristics of the blood-brain barrier guarantee that only those molecules, which are either hydrophobic (such as oxygen, nitric oxygen and steroid hormones), or bind to specific receptors (such as certain amino acids and sugars), can pass freely from the blood circulation out into the brain parenchyma. Additionally, there is also a weight-selectivity, where particles of a larger molecular weight are more effectively excluded from passage over the blood-brain barrier. Also, active transport out from the brain parenchyma and metabolization of certain drugs, made possible by an intact blood-brain barrier, stabilises and optimises the environment surrounding the neurons of the mammalian brain.

In a number of pathological conditions, such as epileptic seizures, sepsis and severe hypertension, the integrity of the blood-brain barrier is disturbed. The sensitively tuned balance within the brain parenchyma is thereby disrupted. This might lead to cerebral oedema, increased intracranial pressure and in the worst case, irreversible brain damage. Also, potentially carcinogenic molecules can gain free access to otherwise protected areas of the mammalian brain. Of importance to remember, is also, that transient openings might be harmful enough to result in permanent tissue damage.<sup>43)</sup>

In conclusion, an intact and fully functioning blood-brain barrier is essential for the proper function of the mammalian brain.

Rectangular pulsed RF were generated by switching the MW generator (900 MHz) on and off with a rectangular pulse train of various repetition frequencies (4-217 Hz). We started our studies on albumin passage over the BBB a repetition frequency of 16 Hz and then with its harmonies of 4, 8 and also 50 Hz, which was felt relevant, as it is the standard voltage of the European power supply, with a carrier wave of 915 MHz. At an early stage also 217 Hz modulation was added as this was the frequency of the then planned GSM system. In all experiments endogenous substances such as albumin and fibrinogen, which occur naturally in the blood circulation, were used for the detection of BBB leakage, which is identified by anti-rat albumin rabbit antibodies and rabbit anti-human fibrinogen.

This work was published in 1994<sup>3)</sup> and 1997<sup>6)</sup> and comprised sham or 915 MHz exposure for in most cases 2 hours (both CWs and pulsed modulated waves). These results, based on 246 rats 1994 and more than 1,000 rats 1997 (the majority EMF exposed and about 1/3 sham-exposed) concluded that there was a significant difference between the albumin extravasation from brain capillaries into the brain tissue between the differently exposed groups and the controls. It is important to point out that though all animals in the 1997 series (and basically all of our experiments) are inbred Fischer 344 rats, only at the most 50% of the identically exposed animals display albumin extravasation (in CW animals and somewhat less in the other exposed animals). Even the sham exposed animals had some albumin leakage though only in seventeen per cent as a mean of all controls and at a lesser extent. The detection of leakage in unexposed animals presumably is due to our very sensitive immune histological methods.

The most remarkable observation was that exposure with whole-body average power densities below 10 mW/kg gave rise to a more pronounced albumin leakage

than higher power densities, all at non-thermal levels. If the reversed situation were at hand, we feel that the risk of cellular telephones, base-stations and other RF emitting sources could be managed by reduction of their emitted energy. The SAR value of around 1 mW/kg exists at a distance of more than one meter away from the mobile phone antenna and at a distance of 150-200 metres from a base station. This has led us to coin the concept passive mobile phoning for all non-users who are exposed.<sup>5</sup>)

The maximally allowed SAR-value for occupational exposure is 10 W/kg, and 2 W/kg is the maximally allowed SAR-value for public exposure. At a frequency of 900 MHz, these values are reached at power densities of  $22.5 \text{ W/m}^2$  for maximally allowed occupational exposure, and  $4.5 \text{ W/m}^2$  for maximally allowed public exposure. That is, 1 W/kg corresponds to  $2.25 \text{ W/m}^2$  at a frequency of 900 MHz.

In many studies of pharmacological effects in connection with RF exposure, response is only seen at a certain dose range, and not at higher or at lower dosages. This is named "the inverted U-function". A similar RF response characteristic has been observed by us, seen as a more pronounced albumin leakage at lower than at higher power densities. According to Adey, this kind of dose response might constitute the basis for window effects observed in connection to RF exposure.<sup>44</sup>)

In the majority of our studies, EMF exposure of the animals has been performed in transverse electromagnetic transmission line chambers (TEM-cells)(for reference see 2),3),5)-7),39),45),46).) These TEM-cells are known to generate uniform electromagnetic fields for standard measurements. In each TEM-cell, two animals can be placed, one in an upper compartment and one in a lower compartment. The experimental model allows the animals, which are un-anaesthetized during the whole exposure, to move and turn around in the exposure chamber, thus minimising the

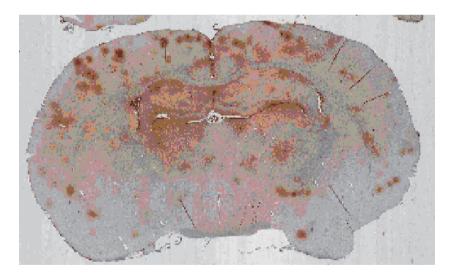


Fig. 4. Pathological leakage around brain capillaries demonstrated by immuno assaying against blood albumin. Fischer 344 male rat (# 3987, weighing 292 g) exposed to 1899 MHz CW microwaves in an anechoic chamber for 2 hours at SAR  $\approx 2mW/kg$ . Ten minutes after this exposure, the animal was anaesthetised and sacrificed.

effects of immobilization induced stress, described by Stagg et al.<sup>47</sup>)

It is important to point out that the position of the animals in upper or lower compartments does not affect the magnitude of observed albumin leakage. Also, we have concluded, with our total series of more than two thousand exposed animals, that there is no difference in the sensitivity to EMF exposure between male and female animals as far as albumin leakage is concerned.

Our initial findings of albumin leakage have been repeated by others,<sup>48)</sup> with 900 MHz exposure of rats for 4 hours at brain power densities ranging from 0.3 to 7.5 W/kg. Another group, working in Bordeaux, and led by Prof Pierre Aubineau, has also demonstrated evidence of albumin leakage in rats exposed for 2 hours to GSM-900 MHz at non-thermal SAR-values of 0.12, 0.5 and 2.0 W/kg, using fluorescein-labelled proteins. The results were presented at two meetings<sup>49)</sup> and are very similar to ours, described above.

Support for our findings that low intensity GSM 900 MHz electromagnetic fields influence the BBB is also found in the *in vitro* proteomic studies on a human endothelial cell line by the group of Leszcynski.<sup>50),51)</sup>

### §4. Neuronal damage

Our consistent findings of albumin passage over the BBB and spread in the surrounding brain tissue with albumin uptake in the cytoplasm of neurons and glial cells brought up the question whether this might lead to neuronal damage.

In a series of experimental situations, neuronal degeneration has been observed in areas with BBB disruption and it has been suggested that BBB leakage is the major reason for nerve cell injury such as that seen in dark neurons.<sup>52)</sup>

It has also been observed after intracarotid infusion of hyperosmolar solutions in rats;<sup>53)</sup> in the stroke-prone hypertensive rat;<sup>52)</sup> and after acute hypertension by aortic compression in rats.<sup>55)</sup> Further, epileptic seizures cause extravasation of plasma into brain parenchyma.<sup>54)</sup> The cerebellar Purkinje cells are heavily exposed to plasma constituents and degenerate in epileptic patients.<sup>55)</sup> This effect may, however, as

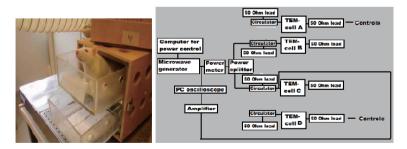


Fig. 5. Left: A rat in the upper exposure tray of a TEM-cell for 915 MHz microwaves. Right: Block diagram of the 4 TEM-cell arrangement used in the experiments in Lund. A microwave power generator is used for feeding the TEM-cells. A power splitter divides the power form the RF generator into equal parts that are fed to each TEM-cell. The output from the cells is terminated in a 50 Ohm dummy load.

well be attributed to hypoxia. It has been postulated that albumin is the most likely neurotoxin in serum.<sup>56)</sup>

In order to seek for neuronal damage in our experimental model, we exposed Fischer 344 rats for 2 hours with non-thermal GSM at SAR values 120, 12 and 1.2 mW/kg.<sup>7)</sup> We made the remarkable observation that a significant (p<0.002) neuronal damage is seen in rat brains 50 days after such an exposure.

It is notable, that we see areas in hippocampus and cortex of exposed animals where the cytoplasm of neurons are filled with autologous albumin while neighbouring neurons display the shrunken and dark state of a "dark neuron" which is a very sick or dying neuron. It may be so that the leakage of albumin out in the neuropil starts a deleterious process whereby more albumin leaks through the endothelium and finally becomes too heavy a burden for the affected neurons. Hassel et al.<sup>57)</sup> have demonstrated that injection of albumin into the brain parenchyma of rats gives rise to neuronal damage. When 25 micro litres of rat albumin is infused into rat neostriatum, 10 and 30, but not 3 mg/ml albumin causes neuronal cell death and severe axonal damage. It also causes leakage of endogenous albumin in and around the area of neuronal damage.

Findings similar to ours in the animals sacrificed late after exposure have been reported in Wistar rats.<sup>58)</sup> Twenty-two female rats were exposed to a 900 MHz electromagnetic GSM near-field signal for one hour a day for seven days. The peak specific absorption rate (SAR) of the brain was 2 W/kg. This resulted in scattered and grouped dark neurons in the cortex, hippocampus and basal ganglia, mixed in among normal neurons with distributions of scores significantly different between the control and the GSM exposure group (p< 0.01).

In continued work we have proven our own finding from 2003 — in a study of 96 non-anaesthetized rats which were exposed or sham exposed for a duration of 2 hours at specific absorption rates (SAR) of 120, 12, 1.2 and now also 0.12 mW/kg. The extravasation of albumin, uptake into neurons and occurrence of damaged neurons were assessed 14 or 28 days later. Albumin extravasation and uptake into neurons was significantly enhanced after 14 days, but not after 28. The occurrence of dark neurons, on the other hand, was significantly enhanced only after 28 days. After 28 days, neuronal albumin uptake was significantly correlated to occurrence of damaged neurons.<sup>8</sup>

In ongoing and recently completed experimental work, we have studied lifelong exposure to GSM 900 as well as the effects of short term exposure to GSM 900 and 1800 in living rats. Lifelong exposure to microwaves seems to be the future of the young generation. Therefore, we have studied male and female Fischer 344 rats, exposed for 2 hours to GSM 900, and sham exposed in our TEM-cells once a week for 13 months. After this they were studied for cognitive functions and compared to cage controls. Significant effects of exposure upon episodic memory function have been demonstrated and published.<sup>9)</sup> In short, the cognitive functions were evaluated in the episodic-like memory test. The GSM-exposed rats had significantly impaired memory for objects and their temporal order of presentation (p=0.02). The detection of a place in which an object was presented, that is the spatial memory function, was not affected by the GSM exposure. In rats, hippocampus is involved in aspects comparable to human declarative memory, and is seems possible that the reduced memory functions that we observed are correlated to hippocampal alterations induced by the mobile phone exposure. Also, temporal order memory, depending on cortical areas such as the perirhinal cortex in the medial temporal lobe, the prefrontal cortex and the interaction between these areas, might explain the reduced temporal order memory of the GSM exposed rats. Finally, after the memory tests had been performed, all animals were sacrificed and the brains are now under examination for albumin leakage, neuronal and glial damage and other signs of pathology.

The possibility that microwaves may affect our DNA has received increased attention since recent epidemiological studies indicate that long term exposure (10 years mobile phone use) increases the risk for developing tumours in the exposed brain hemisphere, both the benign vestibular schwannoma arising from the balance nerve and the highly malignant glioblastoma multiforme.<sup>36),37),59</sup> Regarding the development of vestibular schwannoma, the relative risk seen ten years after the start of mobile phone use, was 1.9 (with confidence interval 0.9-4.1).<sup>59</sup> When only tumours occurring at the same side of the head as the mobile phone had been normally used, the relative risk increased to 3.9 (with confidence interval 1.6-9.5). In a pooled analysis of case-controlled studies on malignant brain tumours, cumulative life use of > 2, 000 hours of mobile phoning revealed an odds ratio of 3.7 (confidence interval of 1.7-7.7).<sup>60</sup>

Studies of gene expression patterns in the living animal may elucidate also other aspects such as effects on genes involved in membrane transport and other basal functions of the living cell *in situ*.

In collaboration with Belyaev and his group we have exposed rats for 6 hours to GSM-900 RFs at SARs of 0.4 mW/kg and investigated the genetic expression from cerebellar tissue. Alterations of genes encoding proteins for BBB functions were observed.<sup>10</sup>

We have now studied whether 6 hours of exposure to the radiation from a GSM mobile phone at 30mW/kg has an effect upon the gene expression pattern in rat brain cortex and hippocampus — areas where we have observed albumin leakage from capillaries into neurons and neuronal damage. Microarray analysis of 31 099 rat genes, including splice variants, was performed in cortex and hippocampus of 8 Fischer 344 rats, 4 animals exposed to GSM for mobile communications electromagnetic fields for 6 hours in an anechoic chamber and 4 controls kept for the same length of time in the same anechoic chamber without exposure. Gene ontology analysis of the differentially expressed genes of the exposed animals versus the control group revealed interesting differences between exposed animals and controls. Genes of interest for membrane transport show highly significant differences.<sup>11</sup> This may be of importance in conjunction with our earlier findings of albumin leakage into neurons around capillaries in exposed animals and has also lead us to look into the mechanisms behind these effects — see below under **DNA Transcription process**, **Solitons and Microwaves**.

## §5. Mechanisms behind the effects of electromagnetic fields upon biology

### 5.1. Interaction of ELF with calcium metabolism

Beyond what is described above, we have also performed experiments where an increase of the  $Ca^{2+}$ -efflux over plasma membranes has been observed in plasma vesicles from spinach exposed to ELF.<sup>13)</sup>

We could show that suitable combinations of static and time varying magnetic fields directly interact with the  $Ca^{2+}$ -channel protein in the cell membrane, and we could quantitatively confirm the model proposed by Blanchard.<sup>61</sup>

Calcium has many important roles in all living organisms. Apart from its structural role in, for example, bone matrix, plant cell walls, and in stabilizing membranes, it plays an essential role in cellular homeostasis, most notably as an intracellular messenger.<sup>62)</sup> The free Ca<sup>2+</sup> concentration in the cytosol is strictly kept at 0.1-0.2  $\mu$ M, which is much lower than that found in the intracellular Ca<sup>2+</sup>-stores or the extra-cellular space. The cytosolic free Ca<sup>2+</sup> ion concentration has influence upon growth and development of the organism and its daily functions as well as death in apoptosis.<sup>62)</sup>

It has been suggested that the mechanism underlying alterations of  $Ca^{2+}$ -fluxes involves inducible changes of both static and time varying magnetic fields.<sup>63</sup> The studies of the effects on  $Ca^{2+}$ -influx over cell membranes are of importance in the perspective of human health, considering the crucial role of  $Ca^{2+}$ -flux played in cellular communications.

The mechanism, by which magnetic fields might interact with biological systems, has been called magnetoreception. Different models try to provide the theoretical framework explaining how this is made possible, and these models are also important for future model-guided investigations of the magnetoreception.

In order to explore the mechanism for possible biological effects of the enhanced ELF radiation environment, we investigated how the transport of  $Ca^{2+}$  ions over the membrane of spinach plasma vesicles varies with frequency and amplitude of ELF magnetic field exposure. Bauréus-Koch et al.<sup>13)</sup> studied the calcium flux through calcium channels in highly purified plasma membranes of spinach (Spinacia oleracea L.).<sup>13)</sup>

A bio-resonance phenomenon was found where appropriate combinations of frequency and amplitude have the potency to affect bio-membranes and their Ca<sup>2+</sup> -ion transport systems at various degrees and directions. With a static magnetic field  $B_{DC} = 37.0 \pm 0.5 \ \mu\text{T}$  we found resonances of  $B_{AC} = 25.9 \pm 0.3 \ \mu\text{T}$  (peak), at the frequencies of 7, 21, 24, and 31 Hz. The Ca2+ -ion efflux ratio at those exposure conditions appears to deviate significantly compared to that of sham exposures.<sup>13</sup>

Three Gaussian peaks with the same width of  $2.5\pm0.4$  Hz could be fitted through the data points with peaks at the frequencies  $20.9\pm0.3$ ,  $25.4\pm0.4$ , and  $30.2\pm0.5$  Hz with a  $\chi^2$  value of 6.0. These frequencies correspond well to the resonance frequencies 20.7 Hz (Mn<sub>ion</sub>, n = 1) 25.2 Hz ( $^{45}$ Ca<sub>ion</sub>, n = 1), and 31.1 Hz (Mn<sub>ion</sub>, n = 1), respectively.<sup>13</sup> With our  $Ca^{2+}$ -efflux studies over plasma membranes as a basis, our research was further extended into the field of magnetic resonance models; mainly the Ion Parametric Resonance (IPR) Model as proposed by Lednev;<sup>64),65)</sup> in an attempt to explain the occurrence of resonance frequencies. In short, Lednev's model considers the polarization of the oscillation of an ion bound to a protein in a combination of static and time-varying magnetic fields.

In our studies of spinach vesicles, the calcium flux was modified at frequencies that corresponded to resonance frequencies for non-hydrated ions of  ${}^{40}\text{Ca}^{2+}$ ,  $\text{Mn}^{2+}$  and  $\text{Mn}^{3+}$ . The resonance frequencies were linearly related to the strength of the static magnetic field applied. The resonance frequency of 24 Hz could be attributed to  ${}^{45}\text{Ca}^{2+}$  (n = 1) or  ${}^{24}\text{Mg}^{++}$  (n = 2). Lednev<sup>64</sup>) predicts an amplitude dependence that follows the Bessel functions.

In our experiments, we concluded that the resonance could be attributed to  ${}^{45}\text{Ca}^{2+}$ . However, as in the experiments performed by Blackman,<sup>66)</sup> a factor of two had to be included in the argument of the Bessel function.

In 1996, Lednev<sup>65)</sup> modified his model, in order to avoid some of the problems identified in the original theory.<sup>67)</sup> In this modified version the amplitude window is described by the square of the Bessel functions. A fit to our data<sup>13)</sup> demonstrates that the factor of two is not required as previously to fit the experimental data to the theory.

Taken together, our experimental results of the interaction of ELF magnetic fields with Calcium bound to proteins in the cell membrane fit extremely well with quantum mechanical interaction models.<sup>61,63,68</sup> Thus, we have shown that ELF magnetic fields interact with Calcium and Manganese ions in plasma membranes at specific frequencies in accordance to a quantum mechanical interaction model.<sup>13</sup>

The search for the mechanisms behind the effect of electromagnetic interactions with biological systems has continued. Another way to address the issue, as compared to our model with the purified membrane system, with theoretical, physical models as a basis, is the biological examination of signalling pathways possibly affected by magnetic fields. As has been shown by Sun et al.,<sup>68)</sup> a possible mechanism for the bioeffects produced by ELF-EMF exposure could be protein tyrosine phosphorylation. 50 Hz power-frequency magnetic fields could activate the stressactivated protein kinase (SAPK),<sup>70)</sup> however, not through the phosphorylation of the upstream kinase of SAPK (SEK1/MKK4).<sup>71)</sup> Noise MF with certain intensity could inhibit the biological effect induced by 50 Hz MF, as seen by the reduced activation of SAPK when noise and 50 Hz exposures were applied simultaneously.<sup>72)</sup> With continued research of this kind, a mosaic of EMF target proteins might crystallize.

### §6. Transmembral transportation — Solitons and microwaves

A major portion of this paper dwells on the passage of albumin from the brain capillaries out into the surrounding brain and the cytoplasm of neurons and astrocytes, and the remarkable observation that it is the lowest energy levels that give rise to the most pronounced albumin leakage.

The mechanisms by which the EMFs may alter BBB permeability are not well

understood. At low field strengths, the effects on body temperature are negligible and thus heating effects are not involved. It has been suggested that physicochemical characteristics of membranes are changed.<sup>12)</sup> One of the great pioneers in the field, Ross Adey discussed the mechanisms behind a possible direct, non-thermal effect of RF radiation upon the central nervous system. He studied amplitude-modulated radiofrequency fields and suggested in 1988 that co-operative processes in the cell membrane might be reactive to the low energy of an electromagnetic field. This oscillating field might result in changes of the membrane potential.<sup>74</sup>

The question might find an answer within a theory which we here by bring forward

### - the possible soliton function in membranes.

The word soliton emanates from John Scott Russell's observation of the solitary wave

In 1834, while conducting experiments to determine the most efficient design for canal boats, this young Scottish engineer made a remarkable scientific discovery, which he described in his "Report on Waves" after his first sighting of a soliton or solitary wave, by Russell called a "Wave of Translation" on the Union Canal near Edinburgh.<sup>73)</sup>

The migration of soliton energy in molecular systems was first considered by Davydov and Kisluka<sup>75)</sup> by the use of a quantum coherent wave theory. Solitons were considered important for energy transfer and storage in biological structures, as described by Davydov<sup>76</sup>) and then by Fröhlich,<sup>77</sup>) as coherent dipolar propagating waves. These applications of quantum field theory to biological systems inspired many theoretical physicists to study biological systems with a special interest focused upon tubulin. This filamentous protein is a fundamental building block of the cvtoskeleton matter.<sup>78),79)</sup> Microtubules are important components of the cytoskeleton, responsible for cellular organization and information processing.<sup>80</sup> Microtubules of the neurons in the brain might be active components of brain functioning and information processing. Endogenous electromagnetic waves are considered to be moving in the cavity of the microtubules, transporting and carrying information. The relevant mechanism of electromagnetic wave interaction has been suggested to be spontaneous breakdown of symmetry in the biological, well ordered structures. Such interaction occurs with the dipole moments of the molecules in the brain microtubules.<sup>79)</sup>

Abdalla et al.<sup>81)</sup> studied the problem of information propagation in the brain microtubules, considering propagation of electromagnetic waves in a fluid of permanent electric dipoles. The problem reduces to sine-Gordon wave equation in one space and one time dimension. The energy balance of the voltage along with the neuronal projection and the microtubule z-axis, results in generation of solitons and propagation of kinks or anti-kinks along the microtubule proto-filaments. The tubulin tails are coupled to the dipoles of nearby water molecules at the microtubule surface and the change of their conformational status at the place of the soliton twist. The standing breather swinging at certain tubulin tail (or breather formed by 2-3 coupled swinging tubulin tails) could catalyze microtubule attachment proteins (MAP) and promote or inhibit the action of kinesin-proteins involved in the microtubule dynamics.<sup>82)</sup> Another interesting result of the work of Abdalla et al.<sup>81)</sup> is the fact that the frequency parameters, which showed up in the model, are compatible with the size of microtubules of brain structures and with the transition period observed for the so called conformational changes of the tubulin dimer protein (namely 1-100 GHz).

The applications of exogenous, electromagnetic waves in this frequency interval, that coincide with that we use for wireless communication, interact with the endogenous electromagnetic wave that might result in biological actions. This may be the mechanism behind our observation of memory impairment in rats exposed to 0.9 GHz microwaves as described above.

Solitons as actors in biology thus have been discussed since the 1970-ies. The effects in biological membranes have recently been brought to the fore by two researchers at the Niels Bohr Institute in Copenhagen, T. Heimburg and AD Jackson in their publication: "On soliton propagation in biomembranes and nerves".<sup>83)</sup> They write: "The lipids of biological membranes and intact biomembranes display chain melting transitions close to temperatures of physiological interest. During this transition the heat capacity, volume and area compressibilities, and relaxation times all reach maxima. Compressibilities are thus nonlinear functions of temperature and pressure in the vicinity of the melting transition, and we show that this feature leads to the possibility of soliton propagation in such membranes. In particular, if the membrane state is above the melting transition, solitons will involve changes in lipid state". The authors discuss solitons in the context of several properties of nerve membranes under the influence of the action potential, including mechanical dislocations and temperature changes.

In a recent paper, the same authors support their hypothesis by pointing out that the Hodgkin-Huxley model for nerve signal transduction never explained the function of anesthesia. The soliton model on the other hand might give an answer. They conclude that anesthetics lower the temperature at which lipids become solid, making it difficult for the soliton waves to form. This should prevent nerves from sending pain signals.

It is known that the action of general anaesthetics is proportional to their partition coefficient in lipid membranes (Meyer-Overton rule). This solubility is, however, directly related to the depression of the temperature of the melting transition found close to body temperature in biomembranes. Heimburg and Jackson proposed a thermodynamic extension of the Meyer-Overton rule, which is based on free energy changes in the system and thus automatically

incorporates the effects of melting point depression. This model accounts for the pressure reversal of anaesthesia in a quantitative manner. Further, it explains why inflammation and the addition of divalent cat-ions reduce the effectiveness of anesthetics.<sup>84)</sup> (Charles Overton was professor of pharmacology at Lund University 1907-1930.)

The statement by Heimburg and Jackson is extremely interesting in reference to an extensive and thorough work on pain perception and electromagnetic fields performed by a research group in London Ontario since the early 1980-ies. (Their work stimulated our group to visit London Ontario and to join in the field in 1988.) In a recent review by the group, "Pain perception and electromagnetic fields", it is concluded that the effects on pain, nociception (pain sensitivity) and opiate-mediated analgesia (pain inhibition) constitute one of the most reproducible and reliable effects of EMFs with observed decrease in pain threshold (Del Seppia et al. 2007). In early studies on the nociception of rodents, the animals were placed on a metal surface at a standard temperature ( $50^{\circ}$ C for mice) and the time taken to respond to the heat stimulus with a stereotypic averse withdrawal was recorded. The exposure to a heterogenous time-varying magnetic field resulted in an enhanced basal nocturnal sensitivity and reduced levels of morphine induced analgesia in mice. Also in connection with geomagnetic storms, mice were similarly less responsive to the analgesic effect of morphine. Further studies, with the land snail Cepaea nemoralis, showed that continuous EMF exposure induced hyperalgesia in a duration-dependent manner (at exposure times ranging from 2 hours to 120 hours). It is also pointed out that the increased pain perception by EMF may be a reason for the increasing prevalence of pain problems in the modern society. (For further discussion of these results, see 84).)

With the solid evidence collected from more than 50 publications, most of them based on studies on the land snail, Cepaea nemoralis but also mice and rats, it is tempting to give the solitons a chance in the search for, and definition of, the physiological mechanisms involved.

Exposure to pulsed magnetic fields (MF) has been shown to have a therapeutic benefit by increasing pain thresholds not only in animals, but also in humans. In a recent study it was concluded that MF exposure does not affect basic human perception, but can increase pain thresholds in a manner indicative of an analgesic response.<sup>85)</sup>

We suggest that soliton models will be considered in studies on the relation between pain, anaesthesia and electromagnetic field exposure. Further those models could be applied to study the effect of EMF field on membrane permeability for various molecules such as calcium and albumin.

It is striking that the soliton theory also may be instrumental in the explanation of how the DNA transcription process is possibly influenced by the Microwaves:

### §7. DNA Transcription process, solitons and microwaves

The Nishinomiya-Yukawa International & Interdisciplinary Symposium 2007 raised the question: What is Life? An obvious and simple answer could be: It is DNA!

The DNA strand can be looked upon as an antenna resonating in the microwave band 6GHz with its harmonics and subharmonics.<sup>14)-18)</sup> If this holds true, the dramatic situation might exist, that all living organisms have a receptor for the newly constructed and world-wide man-made microwaves, leading to a direct effect upon the function of DNA — in concordance with our experimental findings!

Screening of gene expression by microarray technology provides new powerful means for the search for molecular pathways and to elucidate possible molecular markers of response of brain cells to MWs. However, to our knowledge, only two studies have been published on the effects of GSM microwaves upon the gene expression in the CNS after exposure of the whole organism.<sup>10),11)</sup> This material was first presented at the 4th International Workshop, 16-20 October 2006, Crete Greece.<sup>87)</sup>

Those studies are described above and have shown that 6 hours of exposure to GSM 900 MW (at the very low SAR value of 0.4 mW/kg) and 1800 MW (at SAR value 30 mW/kg), to brain cells *in vivo* gives rise to highly significant alterations of gene expressions in cerebellar, cortical and hippocampal cells.

These findings are supported by a series of recent publications where the influence of RF of the type emitted in GSM has been studied in vitro in different cell cultures, proving effects upon gene expression in cultured human cells<sup>88)-90)</sup> and rat neurons<sup>91)</sup> through non-thermal mechanisms.

In the search for a possible mechanism behind these effects of the man-made microwaves upon living organisms, we have explored the effects of microwaves on the DNA/RNA transcription process. In the following we bring forward the possibility of a soliton mechanism in the interaction between microwaves and the DNA/RNA transcription process.

### §8. The DNA transcription process

The first step in genome expression is DNA transcription from the original DNA template contained in the cell, is to make a copy — the RNA messenger — which will then be used as a 'master copy' in determining protein sequences in accordance with the genetic information. The evolutionary advantage of such a messenger is obvious: in this way, the original DNA is opened — and thus less protected — for as small a time as possible.<sup>92)</sup>

In the DNA transcription process, a specialized enzyme (RNA-Polymerase or RNAP) binds to a specific site of the DNA double helix and unwinds it in a local region of 15-20 bases, thus creating a "transcription bubble"; the RNAP and the bubble travel then along the DNA, copying its sequence and producing a RNA-Messenger to be later used to express genes or replicate the local sequence. This process requires a very finely tuned coordination of the motion of RNAP — and production of the RNA-Messenger — with the dynamics of the DNA double chain. In the active phase of the process, the RNAP proceeds along the DNA chain at a speed of several tens or hundreds of base pairs per second. Since each base pair is linked by two or three hydrogen bonds, the energy involved in such a process, even considering only the one to open (and close) the DNA chain, is of the order of at least hundred, if not thousand, H bonds per second. This corresponds to about to a power 300 fW (1 fW = 1 femto-W =  $10^{-15}$  W).

### §9. Solitons hiding in DNA and their role in RNA transcription

In a pioneering paper which appeared in 1980, Englander, Kallenbach, Heeger, Krumhansl and Litwin suggested that nonlinear excitations in the DNA double chain could be instrumental in this process and allow the motion of the transcription bubble to occur at near-zero energy cost. In particular, as the fundamental motion undergone by DNA nucleotides in this process is a roto/torsional one, they suggested

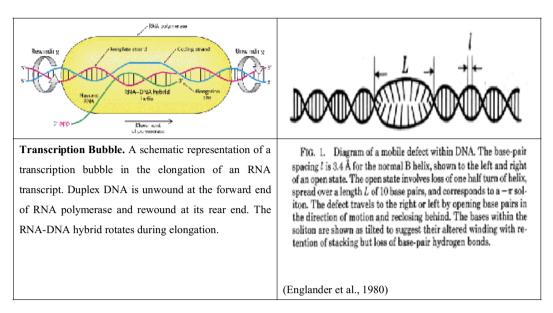


Fig. 6. Solitons in transcription.

modelling the DNA molecule as a double chain of coupled pendulums; the relevant nonlinear excitations would then be (topological) solitons pretty much like those, well known in the sine-Gordon equation<sup>93)</sup> (Fig. 6).

Englander et al.<sup>93)</sup> concluded that precedent for a frequency w, of MHz in double helices implies extended open segments with (L/l) = 10, compatible with the mobile defect model hypothesized (Fig. 7). Experimental indications for processes as fast as GHz exist, but imply very large open structures with (L/1) = 1000. Characteristic attempt frequencies of MHz, on the other hand, seem to be more reasonable in terms of hydrodynamic, melting, and NMR data. The overall activation energy for forming solitons was estimated to 6 kcal/mol which corresponds to  $(L/l) = 100.^{93)}$ The binding energy of individual hydrogen bonds is in the same order of magnitude.

Nonlinear-waves in DNA was suggested by Polozov and Yakushevich<sup>94)</sup> to be involved in the regulation of transcription.<sup>94)</sup> Prohofsky<sup>95)</sup> proposed that the hydrogenbond-stretch (HBS) bands of the double helix appear to be nonlinear enough to support solitary-wave energy concentration. Coupling this fact to predictions of a self-consistent theory of helix melting gives rise to speculations of a mechanism for base pair melting in RNA transcription which is consistent with the known energy needs of that process.<sup>95)</sup>

Guided by the idea of the order parameter of Landau, Zhou and Zhang<sup>96)</sup> analysed the structure and various nonlinear motions in DNA. They argued for the use of four significant variables, i.e., the conformational, rotational, longitudinal and transverse motions. Several sets of nonlinear discrete equations with more reasonable Hamiltonian were established, and their solution of small amplitude (phonons) and large amplitude (soliton or solitary waves) have been given. They speculated in the possible significant implications in duplication, transcription and drug intercalation in DNA.<sup>96</sup>)

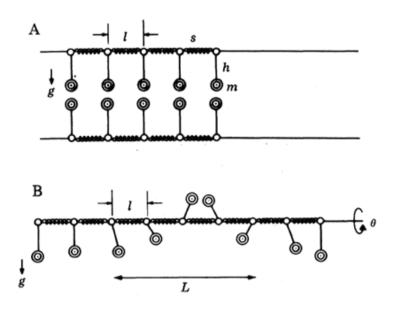


Fig. 7. A mechanical analogue of the DNA double chain, as presented by Englander et al.<sup>92)</sup> Linear chains of the bases (here modelled as pendulums, each with a mass m and length h, with a space in between corresponding to  $\approx 3.4 \text{ \AA}$ ) are connected by sugar-phosphate backbones (modelled as springs). One strand of the DNA double helix is able to undergo torsional oscillations (angle  $\theta$ ) about the sugar-phosphate backbone in the presence of the restoring gravitational force = m \* g. A) The DNA double helix in its ground state.

B) Soliton excitation mode, with large-amplitude excursion of one of the pendulum. The excitation is spread to the group of pendulums within the range of L.

Gaeta<sup>97)</sup> suggested that nonlinear excitations could play a role in the process of DNA transcription, i.e. that the transcription bubble could correspond to a solitary wave travelling along the chain, which the RNAP could then 'surf' in order to access the base sequence with no energy to provide for opening the double helix. He discussed the general idea of providing a simple model for a specific DNA process, and argued that despite the tremendous complexity of the DNA model, this approach is not bound to fail. Recalling the main features of the model proposed by Yakushevich, he mentioned some encouraging achievements and several limitations.<sup>97)</sup>

These limitations, however, more than being inherent to the model, are limitations of the studies conducted so far. It is clear that the model is too simple to be valid as it is. What is needed is to go 'one step further' in the Yakushevich classification of DNA models, but only a more thorough analysis can focus on the detailed refinements which are needed.<sup>98)</sup> In particular, Gaeta<sup>97)</sup> pointed out several directions in which he suggested that it is necessary to generalize the model and to investigate its behaviour, such as considering real nucleic acid base sequences and microwave thermal effects.

### 9.1. Dissociation phase transition in DNA

Bishop, Dauxois, and Peyrard proved the existence of a 'dissociation' phase transition in DNA, considered as a one dimensional system.<sup>99)-103)</sup> Indeed it models DNA as a one-dimensional chain, and by singling out *one* degree of freedom per base — corresponding to 'radial' displacements along the axis joining the two bases of a pair — that is, the degree of freedom thought to be the most relevant for the process under study.

Their theory for DNA melting compares successfully to experimental data on the detailed (spatiotemporal) dynamics of DNA melting. It can predict not only average quantities, as should anyway be the case with a statistical mechanics approach, but a spatiotemporal pattern.<sup>104)</sup>

### 9.2. DNA and microwave absorption

A nontrivial theory for dsDNA phonons and its associated nonlinear modes is provided by the Peyrard-Bishop model<sup>104</sup>) whose Hamiltonian is given by:

$$H_{PB} = \sum_{i=0}^{N} \left(\frac{P_i^2}{2m} + \frac{k}{2}(x_{i+1} - x_i)^2 + V_{H(x_i)}\right),$$
  
$$V_{H(y)} = U_0(\exp(-y/\lambda) - 1)^2,$$

where  $p_i = mv_i$  is the momentum of the ith base pair,

 $x_i$  is the relative coordinate of displacement at each base pair,

 $v_i$  its velocity,

k is the harmonic coupling along each of the chains, and

 $V_H$  refers to the Morse potential representing hydrogen bonds between each base pair.

Fits to experimental data reveal that the well-depth is about normal room temperature (O(10-2 eV)). In a more realistic Peyrard-Bishop-Dauxois model the spring constant k is allowed to vary along the double chain to reflect the requisite stacking energy dependence.<sup>105</sup>)

In the presence of an electric field oscillating in time but spatially homogeneous on the length scale of the dsDNA, we make the following replacement, which follows from standard classical electrodynamics:

$$p_{i} \rightarrow p_{i} - q_{i}A(t)/c,$$
$$A(t) = -\frac{E_{O}c}{\omega_{0}}\sin(\omega_{0}t),$$

where

 $q_i$  is the charge at the ith bond,

A is a component of the vector potential that exhibits solely a time-dependence, c is the speed of light,

 $E_0$  is the amplitude of the incident EM radiation, and

 $\omega_0$  is its frequency.

The charge could be electronic, or it could be a counter-ion adsorbed from the

### 22

aqueous, ionic solvent. We are primarily interested in small perturbations, with a view to estimating at what level they become sinister.

Chivantis describes a dsDNA system, with the following Hamiltonian density, which is the continuum version of the Peyrard-Bishop-Dauxois model.<sup>14),105)</sup>

$$H_{dsDNA} = \frac{1}{2} \left[ (1 - \Lambda(t)) \left( \partial_t \phi(x, t) \right)^2 + c_D^2 (\phi(x, t) \left( \partial_x \phi(x, t) \right)^2 \right] + V_H \left( \phi(x, t) \right)^2 \\ c_D^2 \left( \phi(x, t) \right) = c_0^2 (1 + \rho \exp(-2\alpha \phi(x, t)))$$

where

$$\begin{split} \Lambda\left(t\right) &= \alpha^{2}\sin(\omega_{0}t)^{2} \\ \alpha &= \sqrt{\frac{2\beta Q^{2}\sigma^{2}}{m\omega_{0}^{2}}} < 1 \end{split}$$

 $cD(\phi)$  refers to the extension proposed by Dauxois.<sup>100)</sup>

It causes a stiffening of the backbone as the hydrogen bonds fluctuate. This stiffening reflects the stacking energy dependence of dsDNA. This extension was found to be crucial in understanding the thermal denaturation of dsDNA

It is important to note that the solvent serves to siphon off energy from the disturbance in a very sensitive way. Small changes in the coupling to the solvent bath of phonons affect dramatically the breather modes excited by the EM fields. Experiments where the coupling between the solvent and a DNA molecule is varied will be extremely useful in directing the future development of the understanding of EM effects on the dynamics of DNA.<sup>14</sup>

The free energy needed to melt a GC base pair is generally accepted to be 3.5 kcal/mole and that for an AT base pair 1 kcal/mole. If inflow of this amount of energy occurred, the net energy requirements of transcription would easily be met. The reason to consider this form of energy transfer to the transcription complex is that we believe it would involve the nonlinear hydrogen-bond stretch (HBS) modes. The regime in which the bands of the torsional acoustic (TA) and hydrogen-bondstretch (HBS) modes of DNA interpenetrate each other has been considered by Golo.<sup>16</sup>) He proposes a simple model accommodating the helix structure of DNA and, within its framework, to find a three-wave interaction between the TA and HBS modes. This phenomenon is useful for studying the action of microwave radiation on a DNA molecule. Thus, using Zhang's mechanism of the interaction, he showed that the latter could bring about torsional vibrations that maintain HBS vibrations.

Microwave radiation would maintain the HBS modes and there is no need for long exposures of the sample to radiation.  $\text{Golo}^{16)}$  estimated for the pure experimental system, the critical power density, 100 mW/cm<sup>2</sup>, which is by orders of magnitude larger than that officially prescribed, i.e., at 900 MHz 2W/kg corresponds to 4500 mW/m<sup>2</sup> or 0.45 mW/cm<sup>2</sup>, and at > 2 GHz 10 W/kg corresponds to 10000 mW/m<sup>2</sup> or 1 mW/cm<sup>2</sup>.<sup>16)</sup> The question is, however, if the theoretically derived limit of 100 mW/cm<sup>2</sup> is valid for in vivo exposure conditions. Thus there is still much more research to be done before we might answer that question.

### L. G. Salford et al.

### §10. Conclusion

The first living organisms arose on Earth when it had existed for 1.5 billion years. During the following 3 billion years, life on Earth was formed by, and existed in harmony, with the original physical forces such as gravitation, cosmic irradiation, atmospheric electric fields and the terrestrial magnetism and the cyclic celestial events. This was the world where evolution resulted in Homo sapiens, "the wise man". It took him 200 000 years to reach the level of knowledge where he could dramatically alter the physical forces on Earth. During the last century the levels of ELFs and MWs have been hugely increased in our habitat under the ionosphere.

Even if many studies have seen no effects of the EMFs upon biology, an abundance of scientific reports in respected journals have shown significant, though often weak, effects upon cells *in vitro*, in experimental animals and also in humans.

If the man made EMFs, such as those utilized in mobile communication, even at extremely low SAR values, causes the users' own albumin to leak out through the BBB, which is meant to protect the brain, also other unwanted and toxic molecules in the blood, may leak into the brain tissue. There they concentrate in, and damage, the neurones and glial cells of the brain according to our studies. It cannot be excluded that this, (especially after many years intense use) may promote the development of autoimmune and neuro-degenerative diseases!

It is our generation who invented the microwave emitters. We now have an imperative obligation to further investigate the links between EMF and biology in order to prevent the possible detrimental effects of the microwaves. The concept of solitons as active in membranes and RNA-transcription may be one key to open new paths in the search — a search which must be an imperative not only for researchers but also for states and organisations world-wide.

#### References

- 1) N. Cherry, Natural Hazards 26 (2002), 279.
- 2) L. G. Salford, A. Brun, J. Eberhardt, L. Malmgren and B. Persson, in *Interaction Mechanism of Low-Level Electromagnetic Fields in Living Systems*, ed. B. Nordén and C. Ramel (Oxford University Press, Oxford, 1992), p. 251.
- L. G. Salford, A. Brun, K. Sturesson, J. Eberhardt and B. Persson, Microscopy Research and Technique 27 (1994), 535.
- 4) L. G. Salford, B. Persson and A. Brun, in Non-Thermal effects of RF Electromagnetic Fields, ed. J. H. Bernhardt, R. Matthes and M. H. Repacholi (International Commission on Non-Ionizing Radiation Protection, Munich, 1997), p. 131.
- L. G. Salford, B. Persson, L. Malmgren and A. Brun, in *Téléphonie Mobile Effets Po*tentiels sur la Santé des Ondes Électromagnétiques de Haute Fréquence, ed. M. Pietteur (Embourg, Belgium, 2001), p. 141.
- 6) B. Persson, L. Salford and A. Brun, Wireless Networks 3, 455.
  - M. Peyrard and A. R. Bishop, Phys. Rev. Lett. 62 (1997), 2755.
- 7) L. G. Salford, A. E. Brun, J. L. Eberhardt, L. Malmgren and B. R. R. Persson, Environmental Health Perspectives 111 (2003), 881.
- 8) J. Eberhardt, B. R. R. Persson, L. Malmgren, A. Brun and L. G. Salford, "Blood-brain barrier permeability and nerve cell damage in the rat brain 14 and 28 days after exposure to microwaves from GSM mobile phones", (submitted manuscript).
- 9) H. Nittby, G. Grafström, D. P. Tian, L. Malmgren, A. Brun, B. R. R. Persson, L. G. Salford and J. Eberhardt, Bioelectromagnetics (2008a), published on line Dec. 2007.
- 10) I. Y. Belyaev, C. B. Koch, O. Terenius, K. Roxstrom-Lindquist, L. O. Malmgren, W. H.

Sommer, L. G. Salford and B. R. Persson, Bioelectromagnetics 27 (2006), 295.

- 11) H. Nittby, M. Krogh, G. Grafström, H. Berlin, G. Rehn, J. Eberhardt, L. Malmgren, B. R. R. Persson, B. Widegren and L. G. Salford, "Exposure to global system for mobile communications at 1800 MHz significantly changes gene expression in rat hippocampus and cortex", (submitted manuscript).
- 12) R. Shivers, M. Kavaliers, G. Teskey, F. Prato and R. Pelletier, Neuroscience Lett. 76 (1987), 25.
- 13) C. L. M. Bauréus-Koch, M. Sommarin, B. R. R. Persson, L. G. Salford and J. L. Eberhardt, Bioelectromagnetics 24 (2003), 395.
- 14) S. M. Chitanvis, J. Polymer Science Part B-Polymer Physics 44 (2006), 2740.
- 15) G. S. Edwards, C. C. Davis, J. D. Saffer and M. Swicord, Phys. Rev. Lett. 53 (1984), 1284.
- 16) V. L. Golo, J. Exp. Theor. Phys. **101** (2005), 372.
- 17) E. W. Prohofsky, Bioelectromagnetics 25 (2004), 441.
- 18) C. T. Zhang, Phys. Rev. A **35** (1987), 886; Phys. Rev. A **40** (1989), 2148.
- 19) R. J. P. Williams, J. R. Soc. Interface 4 (2007), 1049.
- 20) M. Balser and C. Wagner, Nature 188 (1960), 638.
- 21) W. O. Schumann, Z. Naturforsch. 7a (1952), 149.
- 22) P. V. Bliokh, A. P. Nicholaenko and Yu. Fillipov, in *IEE Electromagnetic Waves Series* 8 (P. Peregrinus Ltd., Stevenage, 1980).
- 23) D. D. Sentman, Radio Science **22** (1987), 595.
- 24) D. D. Sentman, in *Handbook of Atmospheric Electrodynamics*, vol. I, ed. H. Volland (CRC Press, Boca Raton, 1995).
- 25) R. E. Holzer and D. E. Deal, Nature 177 (1956), 536.
- 26) T. E. Dennis, M. J. Rayner and M. M. Walker, Proc. Biol. Sci. 274 (2007), 1153.
- 27) P. Galland and A. Pazur, J. Plant Res. 118 (2005), 371.
- 28) F. Halberg, G. Cornélissen, P. Regal, K. Otsuka, Z. Wang, G. S. Katinas, J. Siegelova, P. Homolka, P. Prikryl, S. M. Chibisov, D. C. Holley, H. W. Wendt, C. Bingham, S. L. Palm, R. P. Sonkowsky, R. B. Sothern, E. Pales, M. Mikulecky, R. Tarquini, F. Perfetto, R. Salti, C. Maggioni, R. Jozsa, A. A. Konradov, E. V. Kharlitskaya, M. Revillam, C. Wan, M. Herold, E. V. Syutkina, A. V. Masalov, P. Faraone, R. B. Singh, R. K. Singh, A. Kumar, R. Singhs, S. Sundaram, T. Sarabandi, G. Pantaleoni, Y. Watanabe, Y. Kumagai, D. Gubin, K. Uezono, A. Olah, K. Borer, E. A. Kanabrockia, S. Bathina, E. Haus, D. Hillman, O. Schwartzkopff, E. E. Bakken and M. Zeman, Biomed Pharmacother. 58 (2004), Suppl 1, S150.
- 29) P. Volpe, Photochem. Photobiol. Sci. 2 (2003), 637.
- 30) F. I. Komarov, V. N. Oraevski ĭ, Iu. P. Sizov, L. B. Tsirul'nik, Kh. D. Kanonidi, I. B. Ushakov, P. M. Shalimov, M. V. Kimlyk and D. V. Glukhov, Heliogeophysical Factors and Aviation Accidents 43 (1998), 742.
- 31) G. Villoresi, T. K. Breus, L. I. Dorman, N. Iuchi and S. I. Rapoport, Biofizika 40 (1995), 983, (Article in Russian).
- 32) R. Coray, P. Krähenbühl, M. Reiderer, D. Stoll and G. Neubauer, Immissionen in Salzburg. Bundesamt für Metrologie und Akkreditierung (Lindenweg 50, CH-3003 Bern-Wabern, 2002).
- 33) G. Hyland, Lancet **356** (2000), 1833.
- 34) L. G. Salford, H. Nittby, A. Brun, G. Grafström, J. L. Eberhardt, L. Malmgren and B. R. R. Persson, The Environmentalist 27 (2007), 493.
- 35) H. Nittby, G. Grafström, J. L. Eberhardt, L. Malmgren, A. Brun, B. R. R. Persson and L. G. Salford, Electromagnetic Biology and Medicine (2008b). Article accepted for publication.
- 36) M. Kundi K. Mild, L. Hardell and M. O. Mattsson, J. Toxicol. Environ. Health B: Crit. Rev. 7 (2004), 351.
- 37) L. Hardell, M. Carlberg and K. H. Mild, Environmental Research 100 (2006a), 232.
- 38) F. Vecchio, C. Babiloni, F. Ferreri, G. Curcio, R. Fini, C. Del Percio and P. M. Rossini, Eur. J. Neuroscience 25 (2007), 1908.
- 39) L. G. Salford, A. Brun, J. L. Eberhardt and B. R. R. Persson, Bioelectrochem Bioenerg 30 (1993), 293.
- 40) W. E. Thomas, Brain Res. Rev. 31 (1999), 42.

### L. G. Salford et al.

- 41) W. H. Oldendorf, M. E. Cornford and W. J. Brown, Ann. Neurol. 1 (1977), 409.
- 42) J. F. Ghersi-Egea, A. Minn and G. Siest, Life Sciences 42 (1988), 2515.
- 43) T. E. O. Sokrab, B. B. Johansson, H. Kalimo and Y. Olsson, Acta Neuropathology 75 (1988), 557.
- 44) W. R. Adey, in Interaction Mechanism of Low-Level Electromagnetic Fields in Living Systems, ed. B. Nordén and C. Ramel (Oxford University Press, Oxford, 1992), p. 47.
- 45) J. Van Hese, L. Martens, D. De Zutter, C. De Wagter, L. Malmgren, B. R. R. Persson and L. G. Salford, IEEE Transactions on Electromagnetic Compatibility 34 (1991), 292.
- 46) L. Martens, J. Van Hese, D. De Sutter, C. De Wagter, L. O. G. Malmgren, B. R. R. Persson and L. G. Salford, Bioelectrochem Bioenerg 30 (1993), 73.
- 47) R. B. Stagg, L. H. Havel3rd, K. Pastorian, C. Cain, W. R. Adey and C. V. Byus, Radiat. Res. 155 (2001), 584.
- 48) K. Fritze, C. Sommer, B. Schmitz, G. Mies, K. -A. Hossmann, M. Kiessling and C. Wiessner, Acta Neuropathologica 94 (1997), 465.
- 49) F. Töre, P. E. Dulou, E. Haro, B. Veyret and P. Aubineau, Proc. the 5th International congress of the EBEA, Helsinki, Finland (2001), p. 43; Proc. the 24th annual meeting of the BEMS (2002), p. 61.
- 50) D. Leszczynski, S. Joenväärä, J. Reivinen and R. Kuokka, Differentiation 70 (2002), 120.
- 51) R. Nylund and D. Leszcynski, Proteomics 4 (2004), 1359.
- 52) K. Fredriksson, H. Kalimo, C. Nordborg, B. B. Johansson and Y. Olsson, Acta Neuropathologica (Berl) 76 (1988), 227.
- 53) T. S. Salahuddin, H. Kalimo, B. B. Johansson and Y. Olsson, Acta Neuropathologica (Berl) 76 (1988), 1.
- 54) A. Mihày and B. Bozo'ky, Acta Neuropathology 127 (1984a), 251; Acta Neuropathology 65 (1984b), 471.
- 55) T. E. Sokrab, H. Kalimo and B. B. Johansson, Epilepsia **31** (1990), 1.
- 56) S. Eimerl and M. Schramm, Neuroscience Lett. 130 (1991), 125.
- 57) B. Hassel, E. G. Iversen and F. Fonnum, Neuroscience Lett. 167 (1994), 29.
- 58) A. Ilhan, A. Gurel, F. Armuten, S. Kamisifi, M. Iraz, O. Akyol and S. Ozen, Clinica Chimica Acta 340 (2004), 153.
- 59) S. Lőnn, A. Ahlbom, P. Hall and M. Feychting, Epidemiology 15 (2004), 653.
- 60) L. Hardell, M. Carlberg and K. H. Mild, Int. Arch. Occup. Environ. Health 79 (2006b), 630.
- 61) J. P. Blanchard and C. F. Blackman, Bioelectromagnetics 15 (1994), 217.
- 62) G. E. Kass and S. Orrenius, Environ. Health Perspect 107 (1999) (Suppl. 1), 25.
- 63) C. Fanelli, S. Coppola, R. Barone, C. Colussi, G. Gualandi, P. Volpe and L. Ghibelli, FASEB J. 1 (1999), 95.
- 64) V. V. Lednev, Bioelectromagnetics **12** (1991), 71.
- 65) V. V. Lednev, Biophysics **41** (1996), 224.
- 66) C. F. Blackman, J. P. Blanchard, S. G. Benane and D. E. House, Bioelectromagnetics 15 (1994), 239.
- 67) R. K. Adair, Bioelectromagnetics 13 (1992), 231.
- 68) V. V. Lednev, Electricity and Magnetism in Biology and Medicine (1993), p. 550.
- W. J. Sun, Y. N. Yu, H. Chiang, Y. D. Fu and D. Q. Lu, Electro- and Magnetobiology 20 (2001a), 207.
- 70) W. J. Sun, H. Chiang, Y. D. Fu, Y. N. Yu, H. Y. Xie and D. Q. Lu, Electro- and magnetobiology 20 (2001b), 415.
- 71) W. J. Sun, Y. N. Yu, H. Chiang, Y. D. Fu, H. Y. Xie and D. Q. Lu, Electro- and Magnetobiology 21 (2002a), 97.
- 72) W. J. Sun, H. Chiang, Y. Fu, D. Lu and Z. Xu, Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi 20 (2002b), 246, (Article in Chinese).
- 73) W. R. Adey, Prog. Clin. Biol. Res. 257 (1988), 81.
- 74) J. S. Russell, Report on Waves, 14th meeting of the British Association for the Advancement of Science (BAAS, 1844).
- 75) A. S. Davydov and N. I. Kislukha, Phys. Status Solidi B 59 (1973), 465; Phys. Status Solidi B 75 (1976), 735.
- 76) A. S. Davydov, Studia Biophysica 62 (1977), 1.
- 77) H. Fröhlich, Int. J. Quantum Chem. 23 (1983), 1589.

- 78) S. R. Hameroff and R. C. Watt, J. Theor. Biology 98 (1982), 549.
- 79) E. Del Guidice, S. Doglia, M. Milani and G. Vitiello, Nucl. Phys. B 275 (1986), 185.
- 80) L. A. Amos and A. Klug, J. Cell Science 14 (1974), 523.
- 81) E. Abdalla, B. Maroufi, B. C. Melgar and M. B. Sedra, Physica A 301 (2001), 169.
- 82) L. A. Amos and D. Schlieper, Microtubules and maps. Fibrous Proteins: Muscle and Molecular Motors (Elsevier Academic Press Inc., San Diego, 2005).
- 83) T. Heimburg and A. D. Jackson, Proc. Natl. Acad. Sci. USA 102 (2005), 9750.
- 84) T. Heimburg and A. D. Jackson, Biophysical J. 92 (2007), 3159.
- 85) C. Del Seppiaa, S. Ghionea, P. Luschib, K. P. Ossenkopp, E. Choleris and M. Kavaliers, Neuroscience and Biobehavioral Reviews **31** (2007), 619.
- 86) N. M. Shupak, F. S. Prato and A. W. Thomas, Neuroscience Lett. 363 (2004), 157.
- 87) L. G. Salford, M. Krogh, G. Grafstöm, H. Nittby, G. Rehn, H. Berlin, J. L. Eberhardt, L. Malmgren, R. B. R. Persson and B. Widegren, Abstract for the 4th International Workshop: "Biological Effects of Electromagnetic Fields", 16–20 Oct. 2006, Crete, Greece.
- 88) S. Lee, D. Johnson, K. Dunban, H. Dong, X. Ge, Y. C. Kim, C. Wing, N. Jayathilaka, N. Emmanuela, C. Q. Zhou, H. L. Gerber, C. C. Tseng and S. M. Wang, FEBS Lett. 579 (2005), 4829.
- 89) S. Pacini, M. Ruggiero, I. Sardi, S. Aterini, F. Gulisano and M. Fulisano, Oncol. Res. 13 (2002), 19.
- 90) D. Remondini, R. Nylund, J. Reivinen, F. Poulletier de Gannes, B. Veyret, I. Lagroye, E. Haro, M. A. Trillo, M. Capri, C. Franceschi, K. Schlatterer, R. Gminski, R. Fitzner, R. Tauber, J. Schuderer, N. Kuster, D. Leszczynski, F. Bersani and C. Maercker, Proteomics 6 (2006), 4745.
- 91) R. Zhao, S. Z. Zhang, G. D. Yao, D. Q. Lu, J. Huai and Z. P. Xu, Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi 24 (2006), 222, (Article in Chinese).
- 92) W. Saenger, Principles of nucleid acid structure (Springer, Berlin, 1988).
- 93) S. W. Englander, N. R. Kallenbach, A. J. Heeger, J. A. Krumhansl and S. Litwin, Proc. Natl. Acad. Sci. USA 77 (1980), 7222.
- 94) R. V. Polozov and L. V. Yakushevich, J. Theor. Biology 130 (1988), 423.
- 95) E. W. Prohofsky, Phys. Rev. A 38 (1988), 1538.
- 96) G. F. Zhou and C. T. Zhang, Physica Scripta 43 (1991), 347.
- 97) G. Gaeta, J. Biol. Phys. 24 (1999), 81.
- 98) L. V. Yakushevich, Quarterly Reviews of Biophysics 26 (1993), 201; Physica D 79 (1994), 77.
- 99) T. Dauxois and M. Peyrad, Phys. Rev. Lett. 70 (1993), 3935.
- 100) T. Dauxois, M. Peyrad and A. R. Bishop, Phys. Rev. E 47 (1993a), 684.
- 101) T. Dauxois, M. Peyrad and P. A. Bishop, Phys. Rev. E 47 (1993b), R44.
- 102) T. Dauxois, M. Peyrad and C. R. Willis, Physica D 57 (1992), 267.
- 103) M. Peyrard, T. Dauxois, H. Hoyet and C. R. Willis, Physica D 68 (1993), 104.
- 104) M. Peyrard and A. P. Bishop, Phys. Rev. Lett. **62** (1989), 2755.
- 105) N. Theodorakopoulos, T. Dauxois and M. Peyrard, Phys. Rev. Lett. 85 (2000), 6.



Available online at www.sciencedirect.com



SURGICAL NEUROLOGY

Surgical Neurology 72 (2009) 205-215

Neoplasm

www.surgicalneurology-online.com

# Cell phones and brain tumors: a review including the long-term epidemiologic data $\stackrel{\sim}{\asymp}$

Vini G. Khurana, PhD, FRACS<sup>a,b,\*</sup>, Charles Teo, MBBS, FRACS<sup>c</sup>, Michael Kundi, PhD<sup>d</sup>, Lennart Hardell, MD, PhD<sup>e</sup>, Michael Carlberg, MSc<sup>e</sup>

<sup>a</sup>Australian National University, Australia

<sup>b</sup>Department of Neurosurgery, The Canberra Hospital, Garran ACT 2605, Australia <sup>c</sup>The Prince of Wales Private Hospital, Randwick NSW 2031, Australia <sup>d</sup>Institute of Environmental Health, Medical University of Vienna, Vienna A-1095, Austria <sup>e</sup>Department of Oncology, University Hospital, Orebro SE-701 85, Sweden Received 23 December 2008; accepted 21 January 2009

Abstract Background: The debate regarding the health effects of low-intensity electromagnetic radiation from sources such as power lines, base stations, and cell phones has recently been reignited. In the present review, the authors attempt to address the following question: is there epidemiologic evidence for an association between long-term cell phone usage and the risk of developing a brain tumor? Included with this meta-analysis of the long-term epidemiologic data are a brief overview of cell phone technology and discussion of laboratory data, biological mechanisms, and brain tumor incidence.
Methods: In order to be included in the present meta-analysis, studies were required to have met all of the following criteria: (i) publication in a peer-reviewed journal; (ii) inclusion of participants using cell phones for ≥10 years (ie, minimum 10-year "latency"); and (iii) incorporation of a "laterality" analysis of long-term users (ie, analysis of the side of the brain tumor relative to the side of the head preferred for cell phone usage). This is a meta-analysis incorporating all 11 long-term epidemiologic studies in this field.
Results: The results indicate that using a cell phone for ≥10 years approximately doubles the risk

of being diagnosed with a brain tumor on the same ("ipsilateral") side of the head as that preferred for cell phone use. The data achieve statistical significance for glioma and acoustic neuroma but not for meningioma.

**Conclusion:** The authors conclude that there is adequate epidemiologic evidence to suggest a link between prolonged cell phone usage and the development of an ipsilateral brain tumor. © 2009 Published by Elsevier Inc.

Keywords: Acoustic neuroma; Brain tumor; Cell phone; Electromagnetic radiation; Glioma; Incidence; Mechanism; Meningioma; Radiofrequency fields

*Abbreviations:* CBTRUS, Central Brain Tumor Registry of the United States; CDMA, code division multiple access; CI, confidence interval; CNS, central nervous system; EMF, electromagnetic field; EMR, electromagnetic radiation; FCC, Federal Communications Commission; GSM, global system for mobile communication; IARC, International Agency for Research on Cancer; MRI, magnetic resonance imaging; NHL, non-Hodgkin lymphoma; OR, odds ratio; SAR, specific absorption rate; TDMA, time division multiple access; WHO, World Health Organization.

 $<sup>\</sup>stackrel{\leftrightarrow}{}$  There is no author conflict of interest, and no funding was requested or received for this review. The conclusions expressed in this article do not necessarily reflect those of the authors' affiliated institutions and employers.

<sup>\*</sup> Corresponding author. Department of Neurosurgery, The Canberra Hospital, Garran ACT 2605, Australia. Tel.: +61 2 6244 3937; fax: +61 2 6244 2718. *E-mail address:* vgkhurana@gmail.com (V.G. Khurana).

<sup>0090-3019/\$ –</sup> see front matter  $\ensuremath{\mathbb{C}}$  2009 Published by Elsevier Inc. doi:10.1016/j.surneu.2009.01.019

# 1. Background

# 1.1. Cell phone technology

Cell phone technology incorporates base stations, namely, transmission tower antennae, and cell phone handheld units. Cell phone networks were first deployed in Sweden in 1981 via the Nordic Mobile Telephone System (analogue; 450 MHz; first generation or "1G"). The digital system (GSM) started in 1991, representing the second generation of cell phone systems, or "2G." Mass deployment was present in most countries from the mid 1990s (Fig. 1). The latest system currently in mass deployment is based on adaptations of CDMA and TDMA (800 and 1900 MHz; "3G"). Radio waves emitted by modern GSM handsets have a peak power of 1 to 2 W, whereas other digital cellular technologies have power outputs of below 1 W, levels generally regarded as being safe by international regulatory authorities. The 3G has less than 0.25 W of peak power. Through "adaptive power control," the power generated by a cell phone can vary during a conversation according to the amount of interference with the signal, for example, due to the user being in a moving vehicle or within a building or elevator. The output power of the phone is generally set to the highest level during "handovers" between networked base stations as a user moves from one geographic area to another or when signal interference is greatest. The output power of the new 3G is measured for small cells to be, on the average, 0.25 mW, and in a larger cell, about 12 mW. It should be noted that cordless phones operate as transmitters and receivers like GSM cell phones despite shorter signal distances to the home desktop base station. Although such phones have lower peak power than cell phones, user call times tend to be longer. Furthermore, because of adaptive power control of cell phones, the average power output of cordless phones is comparable to cell phones at least in urban areas.

Cell phone base stations or masts emit EMR continuously and at far greater power than cell phones which emit EMR continuously only during calls. Between calls or "at rest" with the "screen asleep" but the power on, cell phones emit a regular pulse of EMR in order for base stations to continuously keep track of the geographic position of the phones in their "cellular network." The GSM antennae are associated with transmitter powers of 10 to 100 W, although 3G antennae use less power-on average 3 W in urban areas. In rural areas, base station power output is much higher because of the vast areas requiring coverage between sparsely distributed base stations, and cell phones rurally are more often at their maximum power output during use in order to maintain good communication [13,37]. Overall, the number of towers has increased tremendously in the past decade and smaller, but even more numerous "microcell" antennae throughout metropolitan environments now enable clear cell phone reception within previously reception-poor locations such as in elevators and building basements.

# 1.2. Electromagnetic field

An EMF is composed of an electric field generated by differences in voltage and a magnetic field generated by the flow of current. The field propagates at the speed of light in waves of a certain length that oscillate at a certain frequency. In the electromagnetic range, gamma rays given off by radioactive materials, cosmic rays, and x-rays are all dangerous to humans and other organisms because of the relatively high-energy "quanta" they carry via highfrequency or short-wavelength waves. Such rays lead to dangerous "ionizing" radiation with an ability to break intermolecular bonds. Cell phone systems also act via EMR

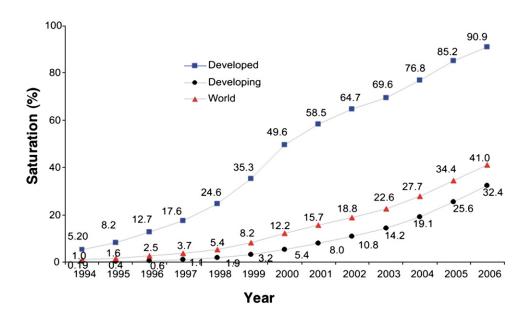


Fig. 1. Worldwide saturation: Cell phone subscribers per 100 inhabitants, 1994 to 2006 (data source: International Telecommunication Union, 2007).

but in the "microwave" or "radiofrequency" range close to that of a microwave oven (although cell phone power output is much less). These systems are supposedly safe because of the lower-energy quanta they carry via relatively lowfrequency or long-wavelength waves, that is "nonionizing" owing to insufficient energy to break intermolecular bonds. This notion, however, has been contested in the scientific literature [27,28,38] and, as detailed below, has led to concerns regarding nonthermal rather than thermal (direct tissue heating) effects of cell phone–related EMR on cells and tissue systems within the near-field of the antenna.

# 1.3. Exposure

The intensity of EMR (power density) varies with the distance from the source according to the inverse square law. The SAR measures the rate at which radiation is absorbed by the human body and is therefore relevant to "exposure." For the head, the FCC has set an acceptable SAR of 1.6 W/kg. In cellular telephony, the SAR depends on several factors, including the antenna type and position, head morphology, the distance between the phone and the head, and the power output of the phone that can vary [3,13]. Exposure of the brain depends on the type of phone and position of the antenna [3] but tends to be highest in the temporal lobe and insular region and overlying skull, scalp, and parotid gland tissues. Irrespective of the type of phone, exposure is highest on the side of the head against which the cell phone is held [3] and appears to be even higher in children owing to thinner scalps and skulls, increased water content of their brain, and lower brain volume [26,65].

### 2. Long-term epidemiologic data

There are currently over 3 billion cell phone users globally, with developed nations already approaching the saturation point (Fig. 2). Users starting as young as 3 years of age are expected to be exposed to near-field cell phone EMR for their entire lifetimes. There has been much controversy regarding health risks associated with cell phones, with confusion partly arising from the relatively short length of participant follow-up in most of the published epidemiologic studies. In studies testing any association between long-term (ie,  $\geq$ 10-year) cell phone use and brain tumor development, the three groups of brain tumors assessed are glioma (specifically, astrocytoma), acoustic neuroma, and meningioma. In this section, the authors focus on all the currently published peer-reviewed epidemiologic studies that have attempted to address whether 10 or more years of cell phone use is associated with the development of intracranial tumors on the same side of the head (ipsilateral) as that preferred for cell phone usage (ie, all long-term studies with a "laterality analysis").

# 2.1. Meta-analysis methodology

In order to be included in the present meta-analysis, studies were required to have met all of the following criteria: (i) publication in a peer-reviewed journal; (ii) inclusion of participants using cell phones for 10 or more years (ie, minimum 10-year latency); and (iii) incorporation of a laterality analysis of long-term ( $\geq 10$ -year) users. The PubMed database was comprehensively searched up to December 1, 2008, using terms including mobile phone, cell phone, brain tumor, neoplasm, incidence, acoustic neuroma, meningioma, glioma, and astrocytoma. If a study had more than one publication on certain epidemiologic aspects, the latest publication giving the most relevant data was used. The present analyses are based on the adjusted ORs in the different studies. It should be reiterated that participant overlap (redundancy) has been avoided in the present metaanalysis by the appropriate handling of pooled versus individual INTERPHONE publications where individual

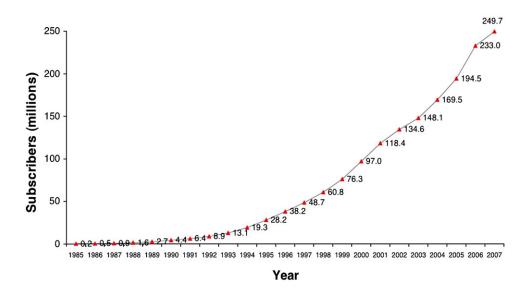


Fig. 2. Number of US cell phone subscribers by year (data source: Cellular Telecommunications Industry Association, 2007).

national data sets were available. Furthermore, there is no overlap of participants in the 2 pooled studies of Hardell [14,18], as well as no overlap in participants between the Swedish studies of Hardell [14,18] and the Swedish arm of INTERPHONE [29,30,35,36] since persons from different parts of Sweden were included in those 2 groups of studies. The present statistical analysis was carried out using a fixed-effects model based on the case-control design of all of the included studies (Stata/SE 10.1 for Windows; StataCorp, College Station, Tex).

# 2.2. Studies included in the meta-analysis fall into two data streams

To the authors' knowledge, there are only 11 published studies examining long-term cell phone use (ie, use for  $\geq 10$  years) and the risk of developing a brain tumor [8,9,14,18,23,29,30,35,36,54,55] (Table 1). These 11 studies fall into two distinct streams of data, namely, (i) the "Hardell group" studies [14,18] from Sweden that were the first case-control studies to report an association between the use of cellular and cordless phones and brain tumors [16] and (ii) the "INTERPHONE group" studies [8,9,23,29,30,35,36,54,55] authored by researchers of the multinational INTERPHONE consortium (see below).

The Hardell studies are comprehensive case-control studies looking at data exclusively from Sweden acquired between 1997 and 2003, whereas the INTERPHONE study is a multinational collective of several comprehensive case-control studies looking at data acquired between 1999 and 2004. Detailed reviews of the methodological aspects of these two data streams, including their limitations pertaining to the extent of subject participation and selection and recall biases, are given elsewhere [4,15,63]. The studies incorporate thousands of cases and controls, although notably far fewer using cell phones for 10 or more years (Table 1), and are briefly summarized below.

# 2.3. The Hardell studies

Since the latter half of the 1990s, Lennart Hardell and his colleagues from Sweden have performed six case-control studies in the area of cellular and cordless phones and tumors [19]. Three of the studies concerned brain tumors; one, salivary gland tumors; one, NHL; and one, testicular cancer. Exposure was assessed by detailed self-administered questionnaires. The Hardell brain tumor studies had approximately 90% case and control participation rates, with cases (n = 2158 participants) and controls (n = 2162 participants) identified from Swedish cancer and population

Table 1

Meta-analysis of epidemiologic studies with results on long-term (>10 or ≥10 years) cell phone use

Study (year) (Ref.)	Countries	Group	Overall			Ipsilateral			Contralateral		
			ca/co	OR	95% CI	ca/co	OR	95% CI	ca/co	OR	95% C
Glioma											
Lonn (2005) [36] <sup>b</sup>	Sweden	Interphone	25/38	0.9	0.5-1.5	15/18	1.6	0.8-3.4	11/25	0.7	0.3-1.5
Christensen (2005) [9] <sup>b</sup>	Denmark	Interphone	14/31	$0.8^{\rm c}$	0.4-1.6	No laterality analysis carried out					
Hepworth (2006) [23] <sup>b</sup>	UK	Interphone	66/112	0.9	0.6-1.3	NA	1.6	0.9-2.8	NA	0.8	0.4-1.4
Schuz (2006) [55]	Germany	Interphone	12/11	2.2	0.9-5.1	No laterality analysis carried out					
Lahkola (2007) [29]	Denmark, UK, Norway, Finland, Sweden	Interphone	143/220	1.0	0.7-1.2	77/117	1.4	1.01-1.9	67/121	1.0	0.7-1.4
Hardell (2006) [18]	Sweden	Hardell	78/99	2.7	1.8-3.9	41/28	4.4	2.5-7.6	26/29	2.8	1.5-5.1
Overall estimate <sup>a</sup> :			233/330	1.3	1.1-1.6	118/145	1.9	1.4-2.4	93/150	1.2	0.9-1.7
Acoustic neuroma											
Lonn (2004) [35] <sup>b</sup>	Sweden	Interphone	14/29	1.8	0.8-4.3	12/15	3.9	1.6-9.5	4/17	0.8	0.2-2.9
Christensen (2004) [8] <sup>b</sup>	Denmark	Interphone	2/15	0.2	0.04-1.1	No laterality analysis carried out					
Schoemaker (2005) [54]	Denmark, UK, Finland, Scotland, Sweden, Norway	Interphone	47/212	1.0	0.7-1.5	31/124	1.3	0.8-2.0	20/105	1.0	0.6-1.7
Hardell (2006) [14]	Sweden	Hardell	20/99	2.9	1.6-5.5	10/28	3.5	1.5-7.8	6/29	2.4	0.9-6.3
Overall estimate <sup>a</sup> :			67/311	1.3	0.97-1.9	41/152	1.6	1.1-2.4	26/134	1.2	0.8-1.9
Meningioma											
Lonn (2005) [36] <sup>b</sup>	Sweden	Interphone	12/36	0.9	0.4-1.9	5/18	1.3	0.5-3.9	3/23	0.5	0.1-1.7
Christensen (2005) [9] <sup>b</sup>	Denmark	Interphone	6/8	1.0	0.3-3.2	No laterality analysis carried out					
Schuz (2006) [55]	Germany	Interphone	5/9	1.1	0.4-3.4	No laterality analysis carried out					
Hardell (2006) [14]	Sweden	Hardell	38/99	1.5	0.98-2.4	15/28	2.0	0.98-3.9	12/29	1.6	0.7-3.3
Lahkola (2008) [30]	Denmark, UK, Norway, Finland, Sweden	Interphone	73/212	0.9	0.7-1.3	33/113	1.1	0.7-1.7	24/117	0.6	0.4-1.0
Overall estimate <sup>a</sup> :	.,		116/320	1.1	0.8-1.4	48/141	1.3	0.9-1.8	36/146	0.8	0.5-1.3

NA, not available, ca/co, number of exposed cases/controls.

<sup>a</sup> Fixed effects model.

<sup>b</sup> Not included in analysis because already part of pooled data.

<sup>c</sup> Crude odds ratio, own calculations.

registries, respectively [14]. Pooled analyses of their results regarding brain tumors are incorporated in the present review. In brief, significantly elevated risks of developing an ipsilateral astrocytoma and acoustic neuroma were found in analogue and digital cell phone and cordless phone users. The OR increased with latency period, particularly more than 10 years, and with cumulative cell phone use more than 2000 hours. Higher ORs were calculated for WHO grade III and IV astrocytomas than for WHO grade I and II astrocytomas. No association was found with salivary gland tumors, NHL, or testicular cancer, but fewer persons in those particular studies were long-term users of cell phones [19]. The aforementioned findings of Hardell [19] suggest specific or differential effects of cell phone radiation on tumor development.

# 2.4. The INTERPHONE study

Following the completion of multinational feasibility studies in the late 1990s, the IARC, a subsidiary of the WHO, commenced the INTERPHONE study. The primary objective of this study, involving 13 nations, was to assess whether radiofrequency radiation exposure from cell phones is associated with tumor risk, specifically, risk of glioma, meningioma, acoustic neuroma and parotid gland tumors. This nonblinded, interview-based, substantially wireless industry-funded case-control study was designed to have enough statistical power to detect a 1.5-fold increase in risk 5 to 10 years from the commencement of cell phone use. The "core protocol" was followed by each of the participating centers [4]. Overall participation rates were relatively low: on average, 53% for controls (n = 7658 participants) in various centres (range, 35%-74%) and 75% (range, 37%-100%) for brain tumor cases (n = 6311 participants) [4,15].

Enrolment in the INTERPHONE study was completed by 2004, although now, almost 5 years later, the publication of the collective INTERPHONE results is still being awaited. In the interim, researchers from the INTERPHONE consortium have published 9 studies incorporating statistically analyzed longterm cell phone usage data pertaining to brain tumors [8,9,23,29,30,35,36,54,55]. All of these publications are listed in Table 1. Only 6 of these 9 INTERPHONE publications involved a laterality analysis [23,29,30,35,36,54]. It should be noted that the Japanese arm [59] of INTERPHONE has been excluded from the present analysis because it did not specifically assess long-term cell phone usage (only 6 meningioma or glioma "cases" and 10 "controls" used cell phones >10 years). It failed to meet the inclusion criteria of the present meta-analysis because that study only reported a laterality analysis of its short-term users (<10 years) [59]. Further, the widely quoted nationwide Danish study [56] involving an assessment of over 420 000 cell phone subscribers is not part of the present analysis because it: (i) was a cohort study comparing incidence in these subscribers with the overall population that, in the meantime, had increased penetration rate of cell phone use from 16% to

80%; (ii) excluded over 200 000 corporate users (ie, those expected to be using cell phones most heavily); (iii) followed users for an average of only 8.5 years; and (iv) did not incorporate any laterality analysis due to using only cell phone subscription data. Finally, other widely referenced US cell phone–brain tumor studies, including those of Inskip [24], Muscat [45], and the Wireless Technology Research Program [5] were not included in the present analysis because they were short-term studies.

### 2.5. Results of the long-term data meta-analysis

Meta-analysis of all available long-term epidemiologic studies reporting an analysis of laterality (Hardell group [14,18] and INTERPHONE group [23,29,30,35,36,54] but excluding those that were already part of pooled analyses that were used instead) gives the following ORs (95% CI) for ipsilateral cell phone use above 10 years (Table 1): glioma (OR, 1.9; CI, 1.4-2.4); acoustic neuroma (OR, 1.6; CI, 1.1-2.4); and meningioma (OR, 1.3; CI, 0.9-1.8). These findings are similar to those in the publication by the Hardell group [16], although a random effects model was used in that publication and indicated a statistically significant elevated odds of developing a glioma or acoustic neuroma on the same side of the head preferred for cell phone use over a duration of exposure of 10 years or more. The authors note that Kan [25], in a meta-analysis of short- and long-term studies in this field, independently found an increased risk of developing a brain tumor with long-term cell phone use (OR, 1.25; 95% CI, 1.01-1.54). However, Kan's meta-analysis is limited by incorporating only 5 long-term epidemiology studies and excluding all of the epidemiologic data from the seminal studies of Hardell [14,18]. To the authors' knowledge, ours is among the first meta-analyses to include all 11 long-term publications, the most recent being the INTERPHONE group's multinational report on meningioma [30].

The authors acknowledge that while there is statistical variance between the different long-term studies for each tumor type, importantly, when all the available long-term data are considered together, there is no decreased risk for contralateral use of cell phones. In short, the meta-analysis shows that long-term cell phone usage can approximately double the risk of developing a glioma or acoustic neuroma in the more exposed (ipsilateral) brain hemisphere and does not protect the less-exposed (contralateral) brain hemisphere against developing a tumor. If the ipsilateral increased odds were caused by recall bias (eg, cases mistakenly reporting more frequently that they used the phone on the same side as the tumor developed), then a decreased risk for contralateral use should be expected but was not found in this meta-analysis. Further, the four publications with the largest numbers of cases and controls that showed elevated OR for ipsilateral glioma and acoustic neuroma did not find an OR <1.0 on the contralateral side [14,18,29,54]. The authors agree with Sadetzki [52] from INTERPHONE Israel

that the side of the head to which an individual prefers to hold a cell phone tends to be related to an individual's handedness, but the concordance is about 60%. The authors reiterate that the risks for the three tumor types analyzed in this work are not the same, that is, the findings of the metaanalysis and its included studies are not "nonspecific." Each of the three tumor types studied is associated with different odds ratios and confidence intervals, and elevated risks of only 2 of the 3 types, namely, glioma and acoustic neuroma, reached statistical significance. These findings may be explained by the different depths and topography of such tumors, and differences in cell types, growth rates, and tumorigenic molecular pathways. As noted in papers from both data streams, there appears to be a statistically significant effect of cell phone usage in terms of tumor type and laterality, latency, and cumulative use of the phone in hours [14,18,29,54].

# 2.6. Limitations of the meta-analysis

The present work attempts to address an important and timely public health concern, namely, does long-term cell phone usage elevate the user's risk of developing a brain tumor? The authors have statistically analyzed all of the published long-term cell phone epidemiologic data to the best of their abilities; however, they also recognize the following limitations of the present meta-analysis. First, in the absence of all of the results of the INTERPHONE study, it is not possible at this time for the authors to assess the homogeneity of long-term associations across each of INTERPHONE's 13 participating nations. The delay in the INTERPHONE study, whose enrolment was completed in 2004, appears to be due to internal difficulties regarding interpretation of the data. Second, the design of each of the studies incorporated into the meta-analysis relies on participants recalling the amount of their use of cell phones through questionnaires and/or telephone interviews, rather than potentially more accurate data acquirable through cell phone company records for study participants. Reliance on recall by a participant regarding time spent using a cell phone (akin to exposure) introduces the potential for recall bias, which can contribute to exposure overestimation or underestimation. Until individual account records are made available to researchers involved in epidemiologic studies comparing tumor incidence among cohorts of heavy versus minimal cell phone users, the results of studies relying on participant memory will continue to be subject to some degree of recall bias [63].

#### 2.7. Exposure overestimation versus underestimation

Recall bias has been proposed by authors of the INTERPHONE study to lead to EMR-exposure overestimation (not underestimation) [63]. However, any overestimation due to recall bias may be countered by exposure underestimation secondary to four key methodological limitations in the INTERPHONE study discussed in detail

elsewhere [15,17,40,41,42] and summarized as follows: in individual INTERPHONE studies, first, the reference group was "never-"/"nonregular" cell phone users, which is appropriate. However, because the published INTER-PHONE studies thus far have not taken into consideration cordless phone use by participants (a risk factor for intracranial tumors [19]), the reference group cannot be described as unexposed to near-field EMR. Second, in the analysis of laterality, persons who developed tumors on the opposite side of the head to the preferred side for cell phone usage were classified as "unexposed" to cell phone EMR. Hence, the INTERPHONE reference (unexposed) category contains subjects using cell phones regularly but reporting use on the other side of the head to the diagnosed tumor. Although exposure to microwaves from cell phone use is substantially lower on the contralateral side [3], the discrepancy is less pronounced for regions of the brain (ventricular and subventricular) where glioma may originate. Third, in the INTERPHONE study, which compared regularly exposed to unexposed individuals, the definition of a "regular" cell phone user is relatively minimalistic, namely, a person who uses a cell phone more than once a week for more than 6 months [4,41,42]. Fourth, the INTERPHONE study's participation rates for cases and controls was low (on average 53% for controls and 75% for cases [4]) compared with the Hardell studies (about 90% each) [14]. In the context of the aforementioned methodological issues, any statistically significant elevated risk in INTERPHONE studies may be expected to be an underestimate of the true risk.

# 3. Laboratory data

Science Magazine has recently acknowledged that there are several peer-reviewed studies from laboratories in at least 7 countries including the United States, showing that cell phone or similar low-intensity EMF can (contrary to expectations of non-ionizing sources) break DNA or modulate it structurally [27]. Although the literature is inconsistent in terms of experimental reproducibility [33,39,50,53,60,62,68], many independent laboratory investigations have suggested adverse biologic effects of cell phone radiation [7,11,12,27,31,32,43,47,50,51,58,64] reviewed in detail elsewhere [28,38,44,62]. An excess of malignant tumors was found in animals exposed for 1 to 2 years to radiofrequency radiation at levels comparable to current standards [7,51], while increased levels of DNA damage via "strand-breakage" have been reported in rat brain cells [31,32] and in human fibroblasts and rat granulosa cells [11] after exposure to cell and cordless phone radiofrequency radiation. Decreases in cell growth rate and survival were found in hamster ovarian cells exposed to radiofrequency radiation over brief time periods but at high specific absorption rates [58], whereas increased DNA fragmentation and cell death and altered reproductive frequency were seen in fruit flies exposed to cell phone radiation [47,64]. In

human and other species' cells, significant gene and protein changes induced by cell phone radiation have been reported, with altered expression, structure and/or function in molecular pathways subserving the heat-shock response [50,64], immune response [50], cellular metabolism [50], and genomic stability [43]. Further, using transcranial magnetic stimulation technology in a double-blind study in humans, local brain hyperexcitability was found during exposure to a GSM cell phone operating for 45 minutes, although that data could not be directly extrapolated to human disease [12].

It should be noted that the induction of stable DNA alterations does not require a DNA-damaging or genotoxic agent. Agents that interfere with epigenetic activities, for example, the processing of these damages, cell cycle control, or apoptosis of the deviating cell, will increase the likelihood of malignant transformation [28]. In this context, expression of genes related to cell death or apoptotic pathways were recently found to be dysregulated in primary cultured neurons and astrocytes following 2-hour exposure to a working GSM cell phone rated at a frequency of 1900 Mhz [67]. Finally, the precise mechanism by which GSM cell phone (nonionizing) EMR can cause or promote neoplasia remains unidentified; however, it has been proposed that the mechanism is unlikely to be related to local heating (thermal effects; the basis of current public and occupational EMF exposure standards [2]) but rather a "nonthermal" interaction between incoming microwaves and exquisitely sensitive oscillatory electrical processes found in living tissues. This interaction that has been referred to as "oscillatory similitude" is akin to the reception of a clock radio being susceptible to interference from a nearby cell phone [22]. It is possible that the phenomenon of oscillatory similitude may lead to genetic or epigenetic damage through increased local production of reactive oxygen species or "free radicals" [2].

### 3.1. Why has the laboratory data been inconsistent?

One key problem with the design of all laboratory studies, both for and against a molecular link between cell phone EMR and brain tumor development, is that such studies fail for understandable reasons to be carried out in larger mammals over time frames consistent with brain tumor development, that is, more than 10 years. Another shortfall of experimental design is failure to take into account the cumulative effects of multiple, varying longterm exposure sources (cell phones, cordless phones and their base stations, high-voltage power lines, WiFi systems, and TV and radio antennae). Finally, naturally occurring genetic variations between individuals (gene polymorphisms) may account for differences in susceptibility to developing brain tumors in humans. Polymorphic genes implicated in brain tumor susceptibility include those subserving immune responses [57], cell cycle control [49] and DNA repair [1,34]. In this context, Yang et al [66] have recently shown that polymorphisms in DNA repair genes appear to enhance susceptibility to leukemia from the lowfrequency EMF of high-voltage power lines. Further, Nylund and Leszczynski [46] have shown that different human endothelial cell lines exposed to the same 1 hour of GSM 900 MHz EMR at a SAR of 2.8 W/kg showed varying degrees of gene and protein expression alterations. They therefore concluded that the cell response to cell phone radiation might be genome and proteome dependent, stating, "It is likely that different types of cells and from different species might respond differently to cell phone radiation or might have different sensitivity to this weak [GSM EMR] stimulus. Our findings might also explain, at least in part, the origin of discrepancies in replication studies between different laboratories" [46].

# 3.2. BioInitiative report

In August 2007, an international working group of scientists, researchers and public health policy professionals (The BioInitiative Working Group) released its report on EMF and health [2]. It raises evidence-based concern about the safety of existing public limits that regulate how much EMF is allowable from power lines, cellular phones, base stations, and many other sources of EMF exposure in daily life. The BioInitiative report [2] provides detailed scientific information on health impacts when people are exposed to electromagnetic radiation hundreds or even thousands of times below limits currently established by the FCC and International Commission for Non-Ionizing Radiation Protection in Europe. The authors reviewed more than 2000 scientific studies and reviews and conclude that (i) the existing public safety limits are inadequate to protect public health, and (ii) from a public health policy standpoint, new public safety limits and limits on further deployment of risky technologies are warranted based on the total weight of evidence [20].

As reviewed in sections 1, 15, and 17 of the BioInitiative report [2], there are several hundred studies that support the existence of low-intensity, non-thermal effects of cell phone radiation on biological systems. The consequences are mostly adverse: DNA single- and double-strand damage, changes in gene transcription, changes in protein folding, heat shock protein generation, production of free radicals, and effects on the immune system. However, that there are also therapeutic effects demonstrated (eg, bone healing and wound healing) from other frequencies and intensities of EMF also gives support to the fact that the human body senses react to and can be differentially affected by low-intensity EMF. This divergent sensitivity is unlikely to be explained by thermal effects alone [20].

# 4. Clinical implications

Taken together, the long-term epidemiologic data suggest an increased risk of being diagnosed with an ipsilateral brain tumor related to cell phone usage of 10 years or more. The data achieve statistical significance for glioma and acoustic neuroma, but not for meningioma. The authors wish to

Table 2 Age-adjusted incidence of primary CNS tumors in the sequential reports of CBTRUS<sup>a</sup>

	CBTRUS Rej	CBTRUS Report							
	2002-2003	2004-2005	2005-2006	2007-2008					
Diagnosi	s year								
1995	13.4 <sup>b</sup>	NA	NA	NA					
1996	14	NA	NA	NA					
1997	14.2	13.5	NA	NA					
1998	14.5	13.9	14.2	NA					
1999	14	14.1	14.5	NA					
2000	NA	14.2	14.8	15.2					
2001	NA	14.7	15.3	15.9					
2002	NA	NA	15.2	16.2					
2003	NA	NA	NA	17					
2004	NA	NA	NA	18.2					

<sup>a</sup> Incidence is the number of cases per 100 000 population age-adjusted to the US population 2000 standard.

<sup>b</sup> Latest published incidence for each year of diagnosis is rendered in boldface. Changes in incidence within and between years have been attributed by CBTRUS mainly to better surveillance and delayed reporting (*late ascertainment*; see text for details) [6].

reiterate that the current long-term epidemiologic data are consistent in determining an increased risk of brain tumors associated with ipsilateral long-term cell phone usage. That is, findings of the laterality analysis of the Hardell group are consistent with those of the INTERPHONE group when the long-term data are specifically assessed [14,18,29,54]. The authors of the present review recognize that the results are subject to the effects of variations in subject participation rates and selection and recall biases; however, they conclude that the currently available long-term epidemiologic evidence points to the aforementioned adverse health effects. Furthermore, the findings pertaining to brain tumors are strengthened by the long-term data recently reported by Sadetzki et al [52], head of INTERPHONE Israel. Sadetzki et al [52] have found significantly elevated odds for the development of ipsilateral parotid gland tumors among heavy cell phone users, effects observed to be dosedependent. Findings from the unrelated publications of Hardell et al [14,18] on brain tumors and Sadetzki et al on parotid tumors, two groups that comprehensively assessed cell phone users in a "dose-dependent" manner, suggest an effect of tumor type and laterality, latency (time to tumor development), and exposure (or "EMR dose," ie, cumulative cell phone use in hours).

### 4.1. Tumor Incidence data from CBTRUS

The CBTRUS maintains a comprehensive and unique record of age-adjusted incidence of primary CNS tumors. In its recently published 2007-2008 Statistical Report [6], which collected data from 2000-2004 from 15-19 state registries in the US, an age-adjusted incidence of 18.2/100,000 population was noted in 2004. According to its 2002-2003 Statistical Report, which collected data from 1995-1999 from 12 state registries, the incidence was 13.4/100,000 population in 1995. The change in incidence rates (Table 2) since 1995 is shown in Fig. 3.

Given that CBTRUS reports CNS tumor incidence ageadjusted to the 2000 US standard population and that the period of these reports is well embedded within the MRI era

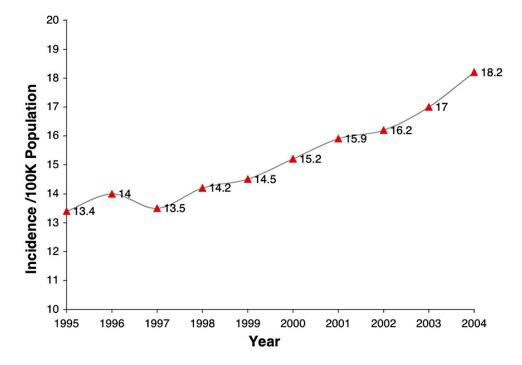


Fig. 3. Age-adjusted incidence of primary CNS tumors by year; US population 2000 standard (data source: CBTRUS 2008) [6].

of the United States, the observed increase in incidence of approximately 36% in less than a decade is not explained by an ageing population (because these figures were ageadjusted to the same standard population) or by "better detection." However, the change may in part be due to the effect of delay in data accrual or reporting referred to as "late ascertainment" [10] (Personal Communication, Lloyd Morgan, Director of CBTRUS; April 23, 2008). Alternatively, as stated in the CBTRUS 2007-2008 Report [6], it may also be due in part to the influence of increased surveillance of nonmalignant tumors resulting from US Public Law 107-260, which was passed in 2002 and instituted beginning in 2004. For these latter reasons, it follows that the 2004 incidence may be an underestimation of the current true incidence in 2008, as observed in changes in yearly incidence between the consecutive Statistical Reports of CBTRUS (Table 2 and Fig. 3) [6]. Although the authors recognize that the current CBTRUS data suggest that malignant brain tumor age-adjusted incidence overall has not increased [6,21], the most recent data are already at least 4 years outdated. On the other hand, a statistically significant increase in benign brain tumor incidence is reported in the most recent publications of CBTRUS [6,48], specifically pilocytic astrocytoma; nerve sheath tumors, and pituitary tumors in people 0 to 19 years old; and nerve sheath tumors, meningioma and pituitary tumors in people 20 to 64 years old. Although no firm conclusions can be drawn regarding the reasons for such changes, following and identifying reasons for any future changes in brain tumor incidence is imperative from a public health perspective, given the high morbidity and mortality associated with these lesions [61].

# 5. Conclusion

The authors believe that the aforementioned epidemiologic and laboratory findings underscore the need for reassessment by governments worldwide of cell phone and also mast radiation exposure standards and the usage and deployment of this technology. If the epidemiologic data continue to be confirmed, then in the absence of appropriate and timely intervention and given the increasing global dependence on cell phone technology especially among the young generation, it is likely that neurosurgeons will see increasing numbers of primary brain tumors, both benign and malignant. The earliest observation of this phenomenon may be commencing as noted in the latest statistical report of the CBTRUS [6].

# References

- Bethke L, Webb E, Murray A, et al. Comprehensive analysis of the role of DNA repair gene polymorphisms on risk of glioma. Hum Mol Genet 2008;17:800-5.
- [2] BioInitiative Working Group. In: Sage C, Carpenter DO, editors. BioInitiative report: a rationale for a biologically-based public exposure standard for electromagnetic fields (ELF and RF); 2007. 2008. http://www.bioinitiative.org.

- [3] Cardis E, Deltour I, Mann S, et al. Distribution of RF energy emitted by mobile phones in anatomical structures of the brain. Phys Med Biol 2008;53:2771-83.
- [4] Cardis E, Richardson L, Deltour I, et al. The INTERPHONE study: design, epidemiological methods, and description of the study population. Eur J Epidemiol 2007;22:647-64.
- [5] Carlo GL, Jenrow RS. Scientific progress—wireless phones and brain cancer: current state of the science. MedGenMed 2000;2:E40.
- [6] CBTRUS. Statistical reports (2002-3, 2004-5, 2005-6, 2007-8). Primary brain tumors in the United States, 1995-2004 (years of data collected in sequential reports). Central Brain Tumor Registry of the United States. http://cbtrus.org/reports/reports.html.
- [7] Chou CK, Guy AW, Kunz LL, et al. Long-term, low-level microwave irradiation of rats. Bioelectromagnetics 1992;13:469-96.
- [8] Christensen HC, Schuz J, Kosteljanetz M, et al. Cellular telephone use and risk of acoustic neuroma. Am J Epidemiol 2004;159:277-83.
- [9] Christensen HC, Schuz J, Kosteljanetz M, et al. Cellular telephones and risk for brain tumors. Neurology 2005;64:1189-95.
- [10] Clegg LX, Feuer EJ, Midthune DN, et al. Impact of reporting delay and reporting error on cancer incidence rates and trends. J Natl Cancer Inst 2002;94:1537-45.
- [11] Diem E, Schwarz C, Adlkofer F, et al. Non-thermal DNA breakage by mobile-phone radiation (1800 MHz) in human fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro. Mutat Res 2005; 583:178-83.
- [12] Ferreri F, Curcio G, Pasqualetti P, et al. Mobile phone emissions and human brain excitability. Ann Neurol 2006;60:188-96.
- [13] Hardell L, Carlberg M, Hansson Mild K. Use of cellular telephones and brain tumour risk in urban and rural areas. Occup Environ Med 2005; 62:390-4.
- [14] Hardell L, Carlberg M, Hansson Mild K. Pooled analysis of two casecontrol studies on the use of cellular and cordless telephones and the risk of benign brain tumours diagnosed during 1997–2003. Int J Oncol 2006;28:509-18.
- [15] Hardell L, Carlberg M, Hansson Mild K. Methodological aspects of epidemiological studies on the use of mobile phones and their association with brain tumours. Open Environ J 2008;2:54-61.
- [16] Hardell L, Carlberg M, Soderqvist F, et al. Meta-analysis of long-term mobile phone users and the association with brain tumours. Int J Oncology 2008;32:1097-103.
- [17] Hardell L, Hansson Mild K. Mobile phone use and risk of glioma in adults. Results are difficult to interpret because of limitations. Letter. BMJ 2006;332:1035.
- [18] Hardell L, Hansson Mild K, Carlberg M. Pooled analysis of two casecontrol studies on use of cellular and cordless telephones and the risk for malignant brain tumours diagnosed in 1997–2003. Int Arch Occup Environ Health 2006;79:630-9.
- [19] Hardell L, Hansson Mild K, Carlberg M, et al. Tumour risk associated with use of cellular telephones or cordless desktop telephones. World J Surg Oncol 2006;4:74.
- [20] Hardell L, Sage C. Biological effects from electromagnetic field exposure and public exposure standards. Biomed Pharmacother 2008;62:104-9.
- [21] Hoffman S, Propp JM, McCarthy BJ. Temporal trends in incidence of primary brain tumors in the United States, 1985-1999. Neuro Oncol 2006;8:27-37.
- [22] Hyland GJ. Physics and biology of mobile telephony. Lancet 2000; 356:1833-6.
- [23] Hepworth S, Shoemaker MJ, Muir KR, et al. Mobile phone use and risk of glioma in adults: case-control study. BMJ 2006;332:883-7.
- [24] Inskip PD, Tarone RE, Hatch EE, et al. Cellular-telephone use and brain tumours. N Engl J Med 2001;344:79-86.
- [25] Kan P, Simonsen SE, Lyon JL, et al. Cellular phone use and brain tumor: a meta-analysis. J Neurooncol 2008;86:71-8.
- [26] Kheifets L, Repacholi M, Saunders R, et al. The sensitivity of children to electromagnetic fields. Pediatrics 2005;116:303-13.
- [27] Khurana VG. Cell phone and DNA story overlooked studies. Letter. Science 2008;322:1325.

- [28] Kundi M, Mild KJ, Hardell L, et al. Mobile telephones and cancer—a review of epidemiological evidence. J Toxicol Environ Health 2004;7: 351-84.
- [29] Lahkola A, Auvinen A, Raitanen J, et al. Mobile phone use and risk of glioma in 5 North European countries. Int J Cancer 2007;120:1769-75.
- [30] Lahkola A, Salminen T, Raitanen J, et al. Meningioma and mobile phone use—a collaborative case-control study in five North European countries. Int J Epidemiol 2008;37:1304-13.
- [31] Lai H, Singh NP. Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. Bioelectromagnetics 1995; 16:207-10.
- [32] Lai H, Singh NP. Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells. Bioelectromagnetics 1997;18:446-54.
- [33] Lee JS, Huang TQ, Kim TH, et al. Radiofrequency radiation does not induce stress response in human T-lymphocytes and rat primary astrocytes. Bioelectromagnetics 2006;27:578-88.
- [34] Liu Y, Zhou K, Zhang H, et al. Polymorphisms of LIG4 and XRCC4 involved in the NHEJ pathway interact to modify risk of glioma. Hum Mutat 2008;29:381-9.
- [35] Lonn S, Ahlbom A, Hall P, et al. Mobile phone use and the risk of acoustic neuroma. Epidemiology 2004;15:653-9.
- [36] Lonn S, Ahlbom A, Hall P, et al. Long-term mobile phone use and brain tumor risk. Am J Epidemiol 2005;161:526-35.
- [37] Lonn S, Forssen U, Vecchia P, et al. Output power levels from mobile phones in different geographical areas; implications for exposure assessment. Occup Environ Med 2004;61:769-72.
- [38] Maisch D. Mobile phone use: it's time to take precautions. J Australas Coll Nutr Environ Med 2001;20:3-10.
- [39] Malyapa RS, Ahern EW, Straube WL, et al. Measurement of DNA damage after exposure to electromagnetic radiation in the cellular phone communication frequency band (835.62 and 847.74 MHz). Radiation Res 1997;148:618-27.
- [40] Milham S. Mobile phone use and risk of glioma in adults: case-control study. Letter to the Editor. Br J Cancer 2006;94:1351.
- [41] Morgan LL. Cellular phones, cordless phones, and the risks of glioma and meningioma (INTERPHONE study group, Germany). Letter to the Editor. Am J Epidemiol 2006;164:292-6.
- [42] Morgan LL. Mobile phone use and risk of glioma in adults. Study has many flaws. Letter. BMJ 2006;332:1035.
- [43] Mashevich M, Folkman D, Kesar A, et al. Exposure of human peripheral blood lymphocytes to electromagnetic fields associated with cellular phones leads to chromosomal instability. Bioelectromagnetics 2003;23:82-90.
- [44] Moulder JE, Foster KR, Erdreich LS, et al. Mobile phones, mobile phone base stations and cancer: a review. Int J Radiat Biol 2005;81: 189-203.
- [45] Muscat JE, Hinsvark M, Malkin M. Mobile telephones and rates of brain cancer. Neuroepidemiology 2006;27:55-6.
- [46] Nylund R, Leszczynski D. Mobile phone radiation causes changes in gene and protein expression in human endothelial cell lines and the response seems to be genome- and proteome-dependent. Proteomics 2006;6:4769-80.
- [47] Panagopoulos DJ, Chavdoula ED, Nezis IP, et al. Cell death induced by GSM 900-MHz and DCS 1800-MHz mobile telephony radiation. Mutat Res 2007;626:69-78.
- [48] Propp JM, McCarthy BJ, Davis FG, et al. Descriptive epidemiology of vestibular schwannomas. Neuro Oncol 2006;8:1-11.
- [49] Rajaraman P, Wang SS, Rothman N, et al. Polymorphisms in apoptosis and cell cycle control genes and risk of brain tumors in adults. Cancer Epidemiol Biomarkers Prev 2007;16:1655-61.
- [50] Remondini D, Nylund R, Reivinen J, et al. Gene expression changes in human cells after exposure to mobile phone microwaves. Proteomics 2006;6:4745-54.
- [51] Repacholi MH, Basten A, Gebski V, et al. Lymphomas in Eu-Pim1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields. Radiation Res 1997;147:631-40.

- [52] Sadetzki S, Chetrit A, Jarus-Hakak A, et al. Cellular phone use and risk of benign and malignant parotid gland tumors—a nationwide casecontrol study. Am J Epidemiol 2008;167:457-67.
- [53] Sakuma S, Komatsubara Y, Takeda H, et al. DNA strand breaks are not induced in human cells exposed to 2.1425 GHz band CW and W-CDMA modulated radiofrequency fields allocated to mobile radio base stations. Bioelectromagnetics 2006;27:51-7.
- [54] Schoemaker MJ, Swerdlow AJ, Ahlbom A, et al. Mobile phone use and risk of acoustic neuroma: results of the Interphone case-control study in five North European countries. Br J Cancer 2005;93:842-8.
- [55] Schuz J, Bohler E, Berg G, et al. Cellular phones, cordless phones, and the risks of glioma and meningioma (Interphone Study Group, Germany). Am J Epidemiol 2006;163:512-20.
- [56] Schuz J, Jacobsen R, Olsen JH, et al. Cellular telephone use and cancer risk: Update of a nationwide Danish cohort. J Natl Cancer Inst 2006; 98:1707-13.
- [57] Schwartzbaum JA, Ahlbom A, Lonn S, et al. An international casecontrol study of interleukin-4Ralpha, interleukin-13, and cyclooxygenase-2 polymorphisms and glioblastoma risk. Cancer Epidemiol Biomarkers Prev 2007;16:2448-54.
- [58] Takashima Y, Hirose H, Koyama S, et al. Effects of continuous and intermittent exposure to RF fields with a wide range of SARs on cell growth, survival, and cell cycle distribution. Bioelectromagnetics 2006;27:392-400.
- [59] Takebayashi T, Varsier N, Kikuchi Y, et al. Mobile phone use, exposure to radiofrequency electromagnetic field, and brain tumour: a casecontrol study. Br J Cancer 2008;98:652-9.
- [60] Thorlin T, Rouquette JM, Hamnerius Y, et al. Exposure of cultured astroglial and microglial brain cells to 900 MHz microwave radiation. Radiat Res 2006;166:409-21.
- [61] Thuppal S, Propp JM, McCarthy BJ. Average years of potential life lost in those who have died from brain and CNS tumors in the USA. Neuroepidemiology 2006;27:22-7.
- [62] Vijayalaxmi, Prihoda TJ. Genetic damage in mammalian somatic cells exposed to radiofrequency radiation: a meta-analysis of data from 63 publications (1990-2005). Radiat Res 2008;169:561-74.
- [63] Vrijheid M, Armstrong BK, Bedard D, et al. Recall bias in the assessment of exposure to mobile phones. J Expo Sci Environ Epidemiol 2008:1-13 [Electronic publication ahead of print].
- [64] Weisbrot D, Lin H, Ye L, Blank M, et al. Effects of mobile phone radiation on reproduction and development in *Drosophila melanogaster*. J Cell Biochem 2003;89:48-55.
- [65] Wiart J, Hadjem A, Wong MF, et al. Analysis of RF exposure in the head tissues of children and adults. Phys Med Biol 2008;53:3681-95.
- [66] Yang Y, Jin X, Yan C, et al. Case-only study of interactions between DNA repair genes (hMLH1, APEX1, MGMT, XRCC1 and XPD) and low-frequency electromagnetic fields in childhood acute leukemia. Leuk Lymphoma 2008;49:2344-50.
- [67] Zhao TY, Zou SP, Knapp PE. Exposure to cell phone radiation upregulates apoptosis genes in primary cultures of neurons and astrocytes. Neurosci Lett 2007;412:34-8.
- [68] Zook BC, Simmens SJ. The effects of 860 MHz radiofrequency radiation on the induction or promotion of brain tumours and other neoplasms in rats. Radiation Res 2001;155:572-83.

# Commentary

The authors have provided the most comprehensive study and analysis to date of this topic, which, until the last year or so, has remained controversial—most studies denying a relation between cell phone use and a risk of brain tumor development. The sentinel work of Hardell et al (noted well in this article) has now alerted the medical community, and the warning in lay publication by Khurana [1] has brought

# Research Article

# Reactive Oxygen Species Formation and Apoptosis in Human Peripheral Blood Mononuclear Cell Induced by 900 MHz Mobile Phone Radiation

# Yao-Sheng Lu,<sup>1, 2</sup> Bao-Tian Huang,<sup>1</sup> and Yao-Xiong Huang<sup>1</sup>

<sup>1</sup> Department of Biomedical Engineering, Jinan University, Guangzhou 510632, China <sup>2</sup> Department of Electronic Engineering, Jinan University, Guangzhou 510632, China

Correspondence should be addressed to Yao-Xiong Huang, tyxhuang@jnu.edu.cn

Received 12 February 2012; Accepted 10 April 2012

Academic Editor: Marcos Dias Pereira

Copyright © 2012 Yao-Sheng Lu et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

We demonstrate that reactive oxygen species (ROS) plays an important role in the process of apoptosis in human peripheral blood mononuclear cell (PBMC) which is induced by the radiation of 900 MHz radiofrequency electromagnetic field (RFEMF) at a specific absorption rate (SAR) of  $\sim$ 0.4 W/kg when the exposure lasts longer than two hours. The apoptosis is induced through the mitochondrial pathway and mediated by activating ROS and caspase-3, and decreasing the mitochondrial potential. The activation of ROS is triggered by the conformation disturbance of lipids, protein, and DNA induced by the exposure of GSM RFEMF. Although human PBMC was found to have a self-protection mechanism of releasing carotenoid in response to oxidative stress to lessen the further increase of ROS, the imbalance between the antioxidant defenses and ROS formation still results in an increase of cell death with the exposure time and can cause about 37% human PBMC death in eight hours.

# 1. Introduction

Mobile phones have been widely used in popular telecommunication and medical telemetry systems. The tremendous use of mobile phone has drastically increased the amount of radiofrequency electromagnetic field (GSM RFEMF) exposure in our daily lives. To ensure telecommunication in anywhere, various kinds of mobile phone relay stations or devices need to be placed inside or near living/working and residential areas. It makes people have the possibility to be exposed to the RFEMF radiation almost every moment. Thus there is a major concern about the effects of RFEMF radiation exposure on human health. Despite previous studies, our knowledge on these effects is still inadequate and strong debates continue [1–5].

Among the various health effects of GSM RFEMF exposure, the formation of reactive oxygen species (ROS) and increased oxidative stress are those proposed mechanisms that can explain the link between RFEMF radiation and possible harmful effects on human health. It was found that RFEMF could induce ROS formation in animal brain, cortical neurons, spleen, blood serum, and human semen [6– 10]. The purpose of this study was to investigate the extent of ROS formation and oxidative DNA damage as well as cell apoptosis caused by RFEMF on human peripheral blood mononuclear cell (PBMC). PBMC cells are a critical component in the immune system to fight infection and adapt to intruders. They also play significant roles in neurodegenerative diseases and aging [11–14]. Therefore, investigation of whether and how oxidative stress activates in PBMC under the exposure of RFEMF radiation can help to further clarify its effects on human health.

In this study, isolated fresh human peripheral blood mononuclear cells were exposed to the radiation of 900 MHz GSM RFEMF at a specific absorption rate (SAR) of 0.4 W/kg for 1 h, 2 h, 4 h, 6 h, and 8 h. The specific absorption rate was chosen to mimic the situation that people usually may absorb in an environment within a distance of 20 meters from mobile phone relay stations, or occupationally in an equipment room of microwave communication, or around a To detect the intracellular ROS activation in the exposed cells, fluorescent dye DCFH was used as the probe in flow cytometry. The caspase-3 activity of the cells was assessed by colorimetric assay, while the cell apoptosis was analyzed by flow cytometry with FITC-Annexin V/Propidium Iodide (PI) double staining. To assess DNA damage of human PBMC and reveal the mechanism of the effect of RFEMF radiation, confocal Raman microspectroscopy was also employed.

# 2. Material and Methods

2.1. Sample and Reagents. Study on blood of volunteers (providing informed written consent) was proved by Jinan University Animal Care and Use Committee conforming to the Chinese Public Health Service Police on Human Care and Use of Laboratory Animals.

Normal peripheral blood was obtained from healthy nonsmoking adult volunteers aging  $25.3 \pm 0.8$  by venipuncture and poured into heparinized tubes. The blood samples were anticoagulated with heparin lithium. After centrifugation, the peripheral blood monocytes in the middle cloud layer were taken out, washed twice repeatedly, and then resuspended. The cell survival rate was >98% estimated by Trypan blue staining.

Annexin V/PI double-staining kit was purchased from Bender Company, USA. The fluorescent dye DAPI was from Roche, USA. The mitochondrial membrane potential detection kit (JC-1), ROS detection kit, Bradford protein concentration assay kit, and caspase-3 colorimetric assay kit were all purchased from Beyotime Institute of Biotechnology, China.

2.2. Exposure of Human PBMC Samples to RFEMF. 200  $\mu$ L of PBMC samples with cell density of 1.5 × 10<sup>6</sup>/L was placed in each well of a culture plate. Then they were exposed to the radiation emitted by a VS401A RF RFEMF emitter (Shenzhen Weikete Technology Company, Ltd. China) at a specific absorption rate of 0.43 W/kg at 37°C for 1 h, 2 h, 4 h, 6 h, and 8 h. The radiation distributed uniformly on the sample and the SAR was determined using the conductivity of the PBMC sample  $\sigma$ , the RFEMF electric field strength *E* at the determined point, and the mass density of the sample  $\rho_{\rm m}$  in the follwoing form: SAR =  $\sigma E^2/\rho_{\rm m}$ . In the experiment,  $\sigma$  was found to be 0.229 ± 0.001 (S/m), *E* was 43.42 (V/m), and  $\rho_{\rm m}$  was 1.011 ± 0.006 (g/mL). Therefore, SAR was estimated to be 0.43 W/kg.

2.3. Cell Apoptosis Detection.  $5\,\mu$ L FITC-Annexin V and  $10\,\mu$ L PI were added to  $100\,\mu$ L cell suspension with cell concentration of  $1 \times 10^6$ /mL. The mixture was incubated for 15 minutes in dark at room temperature. Then they were washed with binding buffer twice and adjusted again to the cell concentration of  $1 \times 10^6$ /mL. The cell apoptosis was analyzed using an FACS Aria flow cytometry (BD company, USA) within 1 hour.

2.4. ROS Detection. The exposed cells were collected and the supernatant was removed by centrifugation. Thereafter the cells were resuspended and  $5 \times 10^5$  cells were collected. They were centrifuged again to remove the supernatant and then added into  $500 \,\mu$ L diluted DCFH-DA. The mixture was incubated for 20 minutes at  $37^{\circ}$ C and then washed twice. The samples were later analyzed with flow cytometer within 1 hour. An Ar<sup>+</sup> laser with 488 nm wavelength was used as the excitation light and 525 nm was the receiving wavelength to obtain the proportion of the fluorescent cells.

2.5. Caspace-3 Activity Detection. The caspace-3 activity of the exposed cells was evaluated using the caspase-3 colorimetric assay kit and the assessment was performed according to the manufacturer's recommendations. The ratio of the OD value of the sample and that of the control group were taken to evaluate the caspase-3 activity.

2.6. Mitochondrial Membrane Potential Determination.  $(10-60) \times 10^5$  exposed cells were resuspended and mixed with the JC-1 staining working solution. The mixture was incubated at 37°C for 20 minutes and then centrifuged for 3-4 minutes to remove the supernatant. The mixture was washed twice with buffer solution and then the cells were resuspended with the buffer solution. The fluorescence of the cells was imaged using a Nikon TE300 inverted fluorescence microscope.

2.7. DNA Damage Detection by Raman Spectroscopy. The Raman spectra of PBMC were recorded by a JY RAM INV system using 514.2 nm excitation line from an  $Ar^+$  ion laser through an inverted Olympus optical microscope with a ×60 objective. The acquisition band was  $600 \sim 1800 \text{ cm}^{-1}$  with a spectrum resolution of  $1 \text{ cm}^{-1}$ . At least 35 cells were measured for each group of the exposed PBMC sample.

*2.8. Data Processing.* The PBMC cells were from the blood samples of 6 volunteers (3 males and 3 females). Each sample contained 10000 cells. All data were averaged from the results of five parallel samples; each sample was detected three times.

The final result is denoted by  $x \pm s$ . SPSS 13.0 was used for statistical analysis of the data, in which P < 0.05 was regarded as significantly different.

# 3. Results

3.1. ROS Activation. The flow cytometric results of human PBMCs' ROS activation are shown in Figure 1. Figure 1(a) indicates the histograms of ROS-positive cells, and Figure 1(b) shows the histograms of mean DCF fluorescence intensity (indication of ROS level). Figure 2 shows how the ROS-positive cells and the ROS level vary with radiation time. We can see that just 1 h radiation can activate ROS in PBMC (P < 0.05, versus control). The ROS level continuously rose in the period from the 2nd h to the 6th h. After 6 h exposure, both the number of ROS-positive cells and ROS level reached their maximum and then declined.

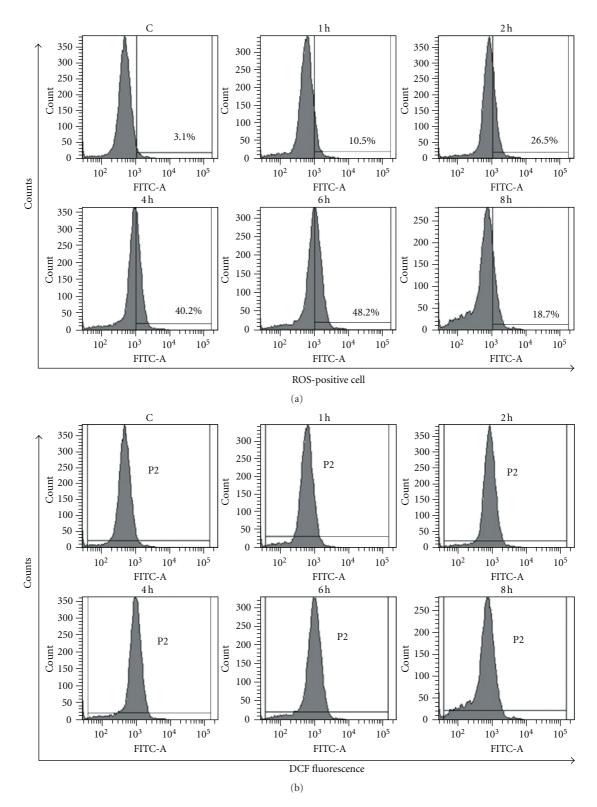


FIGURE 1: The flow cytometric results of human PBMCs' ROS activation. (a) Histograms of ROS-positive cell percentage. (b) Histograms of mean DCF fluorescence intensity.

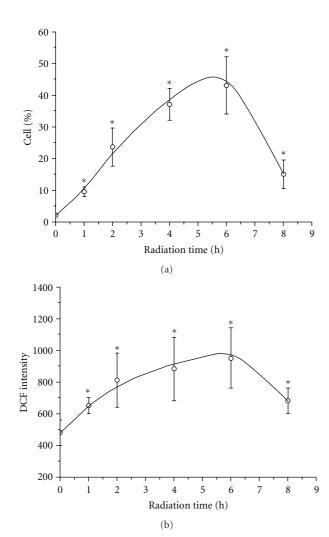


FIGURE 2: The number of ROS-positive cells (a) and DCF intensity (b) versus radiation time.

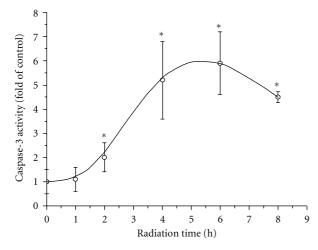


FIGURE 3: The variation of caspacse-3 activity in human PBMCs with radiation time.

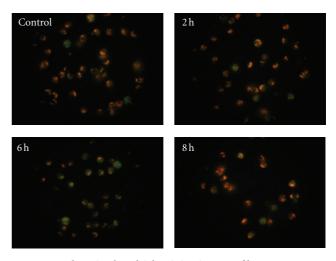


FIGURE 4: The mitochondrial staining images of human PBMCs.

3.2. Caspace-3 Activity. The variation of caspacse-3 activity in human PBMCs with radiation time is shown in Figure 3. Within the first 2 h radiation, the change of caspase-3 content was not evident (P > 0.05 versus control). However, when the cells were radiated longer than 2 h, the caspase-3 activity became significantly increased (P < 0.05 versus control). The activity of caspase-3 at the 6th h was 6 times as that of control group. But at the 8th h, the caspase-3 activity declined significantly compared with that at the 6th h (P < 0.05).

3.3. *Mitochondrial Potential.* Figure 4 illustrates the mitochondrial staining of human PBMCs. The PBMCs in the control group (not exposed to electromagnetic radiation) emitted bright orange-red fluorescence with few emitting green fluorescence. The red fluorescence intensity of the cells weakened while the proportion of the green fluorescence cells increased in the images taken from the 2nd h to the 6th h, indicating a decline of mitochondrial potential in the cells during the period. However, it slightly went up at the 8th h.

3.4. Human PBMC Apoptosis. Figure 5 shows the flow cytometric analysis of apoptosis in human PBMC using FITC-annexin V and PI double staining, and Figure 6 illustrates the apoptotic rates of the exposed cells. It can be seen that neither early apoptosis (Annexin V+/PI-) nor late apoptosis (Annexin V+/PI+) was evident (P > 0.05versus control) in the 1st h. When the exposure lasted longer than 2h, the apoptotic rates increased evidently. The early apoptotic rate increased to  $12.2\% \pm 3.3\%$  and  $21.5\% \pm 5.2\%$ (P < 0.05 versus control), respectively, at the 2nd h and 4th h. At the same time, the late apoptotic rate increased to  $2.2\% \pm 0.8\%$  and  $5.0\% \pm 1.6\%$  (*P* < 0.05 versus control). Compared with the early apoptotic rate at the 4th h, there was no significant increase (P > 0.05 versus the 4th h) at the 6th h, whereas the late apoptotic rate began to decrease at the time. At the 8th h, the early apoptotic rate decreased from  $21.2\% \pm 4.9\%$  (at the 6th h) to  $10.4\% \pm 5.0\%$  (P < 0.05 versus the 6th h). Figures 6(a) and 6(b), respectively, show the detail

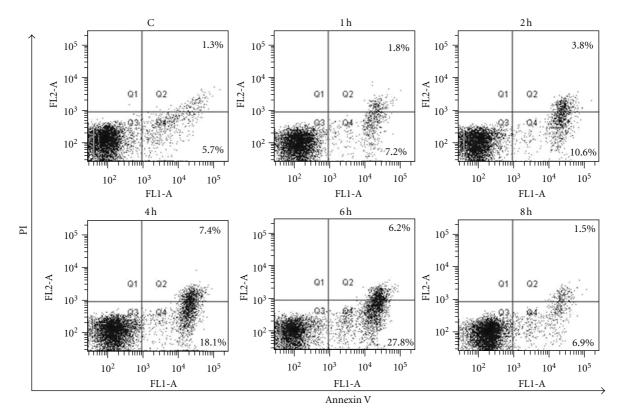


FIGURE 5: The flow cytometric analysis of apoptosis in human PBMCs using FITC-annexin V and PI double staining. Quadrant analysis of the gated cells in FL-1 versus FL-2 channels was from 10,000 events. Annexin V+PI- (lower right quadrant) areas stand for early apoptotic cells, and Annexin V+PI+ (upper right quadrant) areas stand for late apoptotic or necrotic cells.

information about the variations of early and late apoptotic rates of human PBMC with radiation time.

from the 6th h to the 8th h. At the 8th h, about 37% of the exposed cells had died.

3.5. Raman Spectra. Two kinds of Raman spectra were obtained from the exposed cells. One contains weak signal of carotenoid but the other one contains strong signal of carotenoid. Both of them are shown in Figure 7, with peaks at 1157 and  $1525 \text{ cm}^{-1}$  being the bands of carotenoid. The spectra with strong signal of carotenoid were observed only in the samples being exposed longer than one hour but not found in the control group. The proportion of the spectra with strong signal of carotenoid thereafter increased with radiation time and become 60% of all the observed ones in the 4th h. This indicates that the carotenoid releasing in the exposed cells was a reaction to the exposure.

Besides the bands assigned to carotenoid, the bands assigned to DNA (787, 1258, and 1579 cm<sup>-1</sup> in Figure 7(a); 787, 952, 1491, and 1581 cm<sup>-1</sup> in Figure 7(b)) and the bands assigned to protein (1004, 1450, 1616, 1661, and 1678 cm<sup>-1</sup>) also change evidently with radiation time.

3.6. *Cell Counting.* Figure 8 illustrates the result of cell counting on the exposed human PBMC as a function of radiation time. We can see that the number of cells constantly decreases within six hours and then significantly reduces

# 4. Discussion

From the results we can see obviously that cell apoptosis can be induced in human peripheral blood mononuclear cell (PBMC) by the radiation of 900 MHz GSM RFEMF at a specific absorption rate of  $\sim 0.4$  W/kg when the exposure lasts longer than two hours. Using the data about ROS activation, caspacse-3 activity, mitochondrial potential and the Raman spectra of DNA and proteins, we can figure out the mechanism of the cell apoptosis as follows. The exposure to the radiation of 900 MHz GSM RFEMF can induce a series changes in the protein, lipid, and DNA structure. These changes include (1) broken carbon-hydrogen bond of lipid and protein (indicated by the intensity decrease at  $1130 \text{ cm}^{-1}$  in the Raman spectra), (2) damage of the protein side chain (Phe, Tyr, indicated by the intensity decrease at  $1616 \text{ cm}^{-1}$  in the Raman spectra), (3) destruction of the protein secondary structure such as reducing  $\alpha$ -helix and  $\beta$ sheet and increasing random coil (indicated by the intensity decreases at  $1264 \text{ cm}^{-1}$  and  $1678/1680 \text{ cm}^{-1}$  in the Raman spectra), and (4) DNA damage (indicated by the intensity decreases at 952 cm<sup>-1</sup> and 1491 cm<sup>-1</sup> and intensity increase at 1579 cm<sup>-1</sup> in the Raman spectra). All these changes influence the stability of the protein conformation, so that

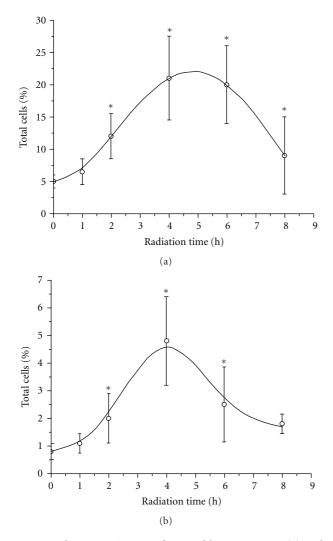
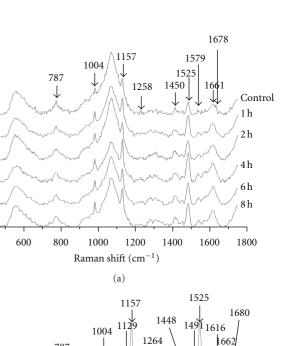


FIGURE 6: The apoptotic rates of exposed human PBMCs:(a) Early apoptotic cells and (b) late apoptotic cells.

the proteins cannot perform their normal function to get rid of the excess ROS. The imbalance between ROS formation and antioxidant defenses results in oxidative stress in human PBMC, thus inducing mitochondrial permeability transition pore (mPTP) opening [20, 21]. The opening of mPTP declines the mitochondrial potential, thereby triggering the caspacse-3 activity and finally inducing cell apoptosis [22– 26]. The apoptosis was mainly early apoptosis, with less than 6% of the cells being late apoptosis.

This is the so-called mitochondrial pathway of apoptosis and has been demonstrated step by step by our experimental results. As described previously, the ROS activation was induced by DNA damage and the disturbance on protein and lipid conformation, suggesting that DNA, protein, and lipid probably are the targets of the GSM RFEMF radiation on human PBMC. On the other hand, human PBMC seems to have a self-protection mechanism of releasing carotenoid in response to oxidative stress to inhibit the further increase of ROS. However, it cannot stop the process of cell death if the exposure continues. The number of cells even decreased



Intensity (cnt)

400

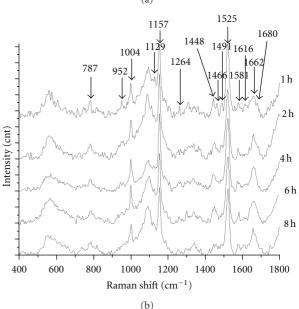


FIGURE 7: The Raman spectra of PBMC which were exposed for different time. (a) Raman spectra with weak signal of carotenoid. (b) Raman spectra with strong signal of carotenoid.

faster in the period from the 6th h to the 8th h as shown in Figure 8. A possibility is that the amount of releasing carotenoid was not enough to against the excessive ROS generation. Another possibility is that, besides cell apoptosis, human PBMC has another cell death process induced by the GSM RFEMF exposure. It is oncosis [27–29] and was proved by our experiment of cell morphological observation on the exposed human PBMC (data not shown). Therefore, the cell number continuously decreased from the 6th h to the 8th h even though the number of apoptotic cells had already decreased in the period. We will not discuss the mechanism of the cell oncosis in human PBMC here but will leave it for a future paper. Finally, we strongly ask for more concern on the possible hazardous health effects of exposure to the radiation of GSM RFEMF emitted from the mobile phone

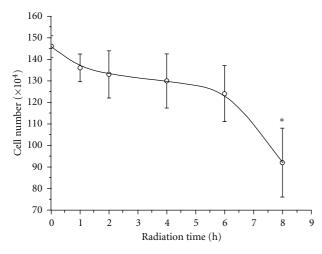


FIGURE 8: The cell counting of the exposed human PBMC versus radiation time.

relay stations or devices as it can cause 37% human PBMC death in eight hours.

# 5. Conclusions

We have demonstrated that cell apoptosis can be induced in human PBMC by the radiation of 900 MHz GSM RFEMF at a specific absorption rate of ~0.4 W/kg when the exposure lasts longer than two hours. The apoptosis is induced through the mitochondrial pathway and mediated by activating ROS and caspase-3, and decreasing the mitochondrial potential. The activation of ROS is triggered by the conformation disturbance of lipids, protein, and DNA induced by the exposure to GSM RFEMF. Although human PBMC has a self-protection mechanism of releasing carotenoid to inhibit further increase of ROS, if the exposure continues, the imbalance between the antioxidant defenses and ROS formation still results in an increase of cell death with the exposure time. These findings not only clarify the effect of GSM RFEMF on human health but also reveal its mechanism. We hope that it will help people to realize the possible hazardous health effects of exposure to GSM RFEMF radiation emitted from the mobile phone relay stations or devices in their living/occupational environment.

# References

- L. Hardell and C. Sage, "Biological effects from electromagnetic field exposure and public exposure standards," *Biomedicine and Pharmacotherapy*, vol. 62, no. 2, pp. 104–109, 2008.
- [2] V. G. Khurana, C. Teo, M. Kundi, L. Hardell, and M. Carlberg, "Cell phones and brain tumors: a review including the longterm epidemiologic data," *Surgical Neurology*, vol. 72, no. 3, pp. 205–214, 2009.
- [3] N. Salama, T. Kishimoto, and H. O. Kanayama, "Effects of exposure to a mobile phone on testicular function and structure in adult rabbit," *International Journal of Andrology*, vol. 33, no. 1, pp. 88–94, 2010.

- [4] A. Abdus-salam, T. Elumelu, and A. Adenipekun, "Mobile phone radiation and the risk of cancer; a review," *African Journal of Medicine and Medical Sciences*, vol. 37, no. 2, pp. 107–118, 2008.
- [5] D. Krewski, B. W. Glickman, R. W. Y. Habash et al., "Recent advances in research on radiofrequency fields and health: 2001–2003," *Journal of Toxicology and Environmental Health— Part B*, vol. 10, no. 4, pp. 287–318, 2007.
- [6] A. Agarwal, N. R. Desai, K. Makker et al., "Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an in vitro pilot study," *Fertility and Sterility*, vol. 92, no. 4, pp. 1318–1325, 2009.
- [7] G. N. De Iuliis, R. J. Newey, B. V. King, and R. J. Aitken, "Mobile phone radiation induces reactive oxygen species production and DNA damage in human spermatozoa in vitro," *PLoS ONE*, vol. 4, no. 7, Article ID e6446, 2009.
- [8] D. Sokolovic, B. Djindjic, J. Nikolic et al., "Melatonin reduces oxidative stress induced by chronic exposure of microwave radiation from mobile phones in rat brain," *Journal of Radiation Research*, vol. 49, no. 6, pp. 579–586, 2008.
- [9] W. Stankiewicz, M. P. Dabrowski, R. Kubacki, E. Sobiczewska, and S. Szmigielski, "Immunotropic influence of 900 MHz microwave GSM signal on human blood immune cells activated in vitro," *Electromagnetic Biology and Medicine*, vol. 25, no. 1, pp. 45–51, 2006.
- [10] S. Dasdag, H. M. Bilgin, M. Z. Akdag, H. Celik, and F. Aksen, "Effect of long term mobile phone exposure on oxidativeantioxidative processes and nitric oxide in rats," *Biotechnology and Biotechnological Equipment*, vol. 22, no. 4, pp. 992–997, 2008.
- [11] C. Colombo, M. Cosentino, F. Marino et al., "Dopaminergic modulation of apoptosis in human peripheral blood mononuclear cells," *Annals of the New York Academy of Sciences*, vol. 1010, pp. 679–682, 2003.
- [12] M. Jenny, M. Klieber, D. Zaknun et al., "In vitro testing for anti-inflammatory properties of compounds employing peripheral blood mononuclear cells freshly isolated from healthy donors," *Inflammation Research*, vol. 60, no. 2, pp. 127–135, 2011.
- [13] A. Prigione, B. Begni, A. Galbussera et al., "Oxidative stress in peripheral blood mononuclear cells from patients with Parkinson's disease: negative correlation with levodopa dosage," *Neurobiology of Disease*, vol. 23, no. 1, pp. 36–43, 2006.
- [14] H. Zhu, M. Belcher, and P. van der Harst, "Healthy aging and disease: role for telomere biology?" *Clinical Science*, vol. 120, no. 10, pp. 427–440, 2011.
- [15] J. P. Gupta, "Microwave radiation hazards from radars and other high power microwave generators," *Defence Science Journal*, vol. 38, no. 3, pp. 287–292, 1988.
- [16] L. Puranen and K. Jokela, "Radiation hazard assessment of pulsed microwave radars," *Journal of Microwave Power and Electromagnetic Energy*, vol. 31, no. 3, pp. 165–177, 1996.
- [17] R. C. Petersen, "Electromagnetic radiation from selected telecommunications systems," *Proceedings of the IEEE*, vol. 68, no. 1, pp. 21–24, 1980.
- [18] International Commission on Non-ionizing Radiation Protection, ICNIRP Guidelines, 1998.
- [19] S. M. Mann, Exposure to Radio Waves Near Mobile Phone Base Stations, National Radiological Protection Board, Oxon, UK, 2000.
- [20] X. H. Cao, S. S. Zhao, D. Y. Liu et al., "ROS-Ca<sup>2+</sup> is associated with mitochondria permeability transition pore involved in

surfactin-induced MCF-7 cells apoptosis," *Chemico-Biological Interactions*, vol. 190, no. 1, pp. 16–27, 2011.

- [21] D. B. Zorov, C. R. Filburn, L. O. Klotz, J. L. Zweier, and S. J. Sollott, "Reactive oxygen species (ROS)-induced ROS release: a new phenomenon accompanying induction of the mitochondrial permeability transition in cardiac myocytes," *Journal of Experimental Medicine*, vol. 192, no. 7, pp. 1001– 1014, 2000.
- [22] D. G. Breckenridge and D. Xue, "Regulation of mitochondrial membrane permeabilization by BCL-2 family proteins and caspases," *Current Opinion in Cell Biology*, vol. 16, no. 6, pp. 647–652, 2004.
- [23] D. R. Green and J. C. Reed, "Mitochondria and apoptosis," *Science*, vol. 281, no. 5381, pp. 1309–1312, 1998.
- [24] N. Mohamad, A. Gutiérrez, M. Núñez et al., "Mitochondrial apoptotic pathways," *Biocell*, vol. 29, no. 2, pp. 149–161, 2005.
- [25] D. Spierings, G. McStay, M. Saleh et al., "Connected to death: the (unexpurgated) mitochondrial pathway of apoptosis," *Science*, vol. 310, no. 5745, pp. 66–67, 2005.
- [26] S. P. Verma and A. Singhal, "Low levels of the pesticide,  $\delta$ -hexachlorocyclohexane, lyses human erythrocytes and alters the organization of membrane lipids and proteins as revealed by Raman spectroscopy," *Biochimica et Biophysica Acta*, vol. 1070, no. 1, pp. 265–273, 1991.
- [27] S. Levin, "Apoptosis, necrosis, or oncosis: what is your diagnosis? A report from the cell death nomenclature committee of the society of toxicologie pathologists 1," *Toxicological Sciences*, vol. 41, no. 2, pp. 155–156, 1998.
- [28] G. Majno and I. Joris, "Apoptosis, oncosis, and necrosis: an overview of cell death," *American Journal of Pathology*, vol. 146, no. 1, pp. 3–15, 1995.
- [29] S. Scarfi, M. Magnone, C. Ferraris et al., "Ascorbic acid pretreated quartz stimulates TNF-*α* release in RAW 264.7 murine macrophages through ROS production and membrane lipid peroxidation," *Respiratory Research*, vol. 10, no. 1, article 25, 2009.





# LONG-TERM EXPOSURE TO MICROWAVE RADIATION PROVOKES CANCER GROWTH: EVIDENCES FROM RADARS AND MOBILE COMMUNICATION SYSTEMS

I. Yakymenko<sup>1,2</sup>\*, E. Sidorik<sup>1</sup>, S. Kyrylenko<sup>3</sup>, V. Chekhun<sup>1</sup> <sup>1</sup>R.E. Kavetsky Institute of Experimental Pathology, Oncology and Radiobiology of NAS of Ukraine, Vasylkivska str. 45, Kyiv 03022, Ukraine <sup>2</sup>Bila Tserkva National Agrarian University, Soborna pl. 8/1, Bila Tserkva 09117, Ukraine <sup>3</sup>Masaryk University, Kamenice 5, A6, Brno 625 00, Czech Republic

In this review we discuss alarming epidemiological and experimental data on possible carcinogenic effects of long term exposure to low intensity microwave (MW) radiation. Recently, a number of reports revealed that under certain conditions the irradiation by low intensity MW can substantially induce cancer progression in humans and in animal models. The carcinogenic effect of MW irradiation is typically manifested after long term (up to 10 years and more) exposure. Nevertheless, even a year of operation of a powerful base transmitting station for mobile communication reportedly resulted in a dramatic increase of cancer incidence among population living nearby. In addition, model studies in rodents unveiled a significant increase in carcinogenesis after 17-24 months of MW exposure both in tumor-prone and intact animals. To that, such metabolic changes, as overproduction of reactive oxygen species, 8-hydroxi-2-deoxyguanosine formation, or ornithine decarboxylase activation under exposure to low intensity MW confirm a stress impact of this factor on living cells. We also address the issue of standards for assessment of biological effects of irradiation. It is now becoming increasingly evident that assessment of biological effects of non-ionizing radiation based on physical (thermal) approach used in recommendations of current regulatory bodies, including the International Commission on Non-Ionizing Radiation Protection (ICNIRP) Guidelines, requires urgent reevaluation. We conclude that recent data strongly point to the need for re-elaboration of the current safety limits for non-ionizing radiation using recently obtained knowledge. We also emphasize that the everyday exposure of both occupational and general public to MW radiation should be regulated based on a precautionary principles which imply maximum restriction of excessive exposure.

Key Words: non-ionizing radiation, radiofrequency, tumor, risk assessment, safety limits, precautionary principle.

# INTRODUCTION

Electromagnetic radiation (EMR) became one of the most significant and fastest growing environmental factors due to intensive development of communication technologies during the last decades. Currently, according to expert estimations, the level of electromagnetic radiation from artificial sources exceeds the level of natural electromagnetic fields by thousand folds. The active development of mobile communication technologies over the world will only raise this level further. In this connection the problem of possible adverse effects of anthropogenic EMR on human health and particularly strictest assessment of possible carcinogenic effects of EMR is extremely important.

In August 2007 an international working group of renowned scientists and public health experts released a report on electromagnetic fields (EMF) and human

Received: March 21, 2011. \*Correspondence: Fax: +380456351288; E-mail: yakymenko@btsau.net.ua Abbreviations used: 8-OH-dG — 8-hydroxi-2-deoxyguanosine; EGF — epidermal growth factor; EMF — electromagnetic field; EMR — electromagnetic radiation; ERK — extracellular-signalregulated kinase; GSM — Global System for Mobile communication; ICNIRP — International Commission on Non-Ionizing Radiation Protection; MW — microwaves; NHL — Non-Hodgkin lymphoma; ODC — ornithine decarboxylase; OER — observed expected ratio; OR — odds ratio; ROS — reactive oxygen species; SAR — specific absorption rate; SIR — standardized incidence ratio; SMR — standardized mortality ratio; WHO — the World Health Organization. health [1]. It raised a serious concern about safety limits for public electromagnetic irradiation from power lines, cell phones, radars, and other sources of EMF exposure in daily life. The authors concluded that the existing public safety limits were inadequate to protect public health. Moreover, very recently a vast number of new extremely important studies in this field have been published. Importantly, nowadays the problem is discussed on highest political level over the world. It appears that the most sound political document in Europe is a European Parliament Resolution from April 2, 2009 (*www.europarl.europa.eu*), where the direct appeals to activate the research and business strategy for effective solving of the problem over the member states were indicated.

In this review we would like to analyze the results of studies on specific biological effects of microwaves (MW), both epidemiological and experimental that deal with cancer promotion by long term low intensity microwave irradiation of human/animal beings. We will concentrate on unequivocal studies and will not analyze ambiguous data. For additional analysis of microwave risks we can recommend recently published reviews [2–10].

# MICROWAVES OF RADARS AND MOBILE COMMUNICATION SYSTEMS

Microwaves are non-ionizing electromagnetic radiation. That means MW is a type of electromagnetic radiation which does not carry enough energy for ionization of atoms and molecules under normal conditions and unlike the ionizing radiation this kind of radiation generally has not enough energy for breaking the intermolecular bonds or for breakaway of electrons from atoms or molecules. MW comprise a part of radiofrequency range. Radiofrequency radiation (RF) refers to electromagnetic waves with a rate of oscillation of electromagnetic fields in the range from 30 kHz to 300 GHz. As any other electromagnetic waves, the radio waves are pulses of electric and magnetic fields. These fields regenerate each other as they move through the space at the speed of light. MW have frequencies from 300 MHz to 300 GHz. As MW have the highest frequency among other RF, it carries the highest energy and produce most thermal effect upon interaction with the matter.

The main sources of radiofrequency radiation during a long period in previous century were broadcasting systems. In some cases, for example, in military and aviation the most powerful local sources of radiofrequency radiation were and still are radars (RAdio Detection And Ranging). However, the situation changed dramatically for general population during recent decades; and currently the most prevailing sources of RF in nearest human environment are mobile communication systems. It is important that both radars and systems for mobile communication use the same microwave part of radiofrequency spectrum.

**Radar systems** are type of powerful sources of pulsed MW which generally effect only certain groups of military or service staff or population living nearby. Radars are detection systems which use MW to determine both moving and fixed objects like aircraft, ships, missiles, etc. Depending on the tasks they use different frequencies of MW, from 1GHz to 12 GHz.

**Mobile communication systems** are undoubtedly the most source of MW in human environment over the world nowadays. Starting from the first commercial mobile phone networks in Japan, Europe and USA since 1979–1983 the number of active users of mobile telephony increased globally to over five billion. In developed countries the number of cellular phone users today is over the point of saturation. It means that many people use more than one cell phone. The initial age of youngest users of cell phone is estimated as three years old [5].

Mobile communication technology utilizes MW for connection of cell phones and base transmitting stations. Phone refers to as mobile because it is free from wire connection and it refers to as cellular/cell because technology utilizes cellular network principle. All area is covered by many base transmitting stations, each station operates in one cell (part of area) and cell phone automatically changes the station when moves from one cell to another. In GSM (Global System for Mobile communication) standard, which covers about 80% of all services over the world the frequencies of electromagnetic waves used are about 850; 900; 1850; or 1900 MHz, which belongs to the microwave range. The useful information (sounds or images) is transferred by modulation of electromagnetic wave frequency. In GSM standard TDMA (Time Division Multiple Access) principle is realized. This means a parttime access of each consumer to the logical channel with frequency of channel rotation about 217 Hz. Thus, both base transmitting stations and cell phones emit MW modulated according to the digital standard.

# SAFETY LIMITS FOR MICROWAVE RADIATION

The main international recommendations on safety levels of non-ionizing electromagnetic radiation is Guidelines for Limiting Exposure to Time-Varying Electric, Magnetic, and Electromagnetic Fields (up to 300 GHz) of International Commission on Non-Ionizing Radiation Protection [11]. The document gives recommended safety limits in all ranges of EMR both for occupational and general public exposure. "Basis for limitation exposure" is dramatically important for understanding the imperfection of this document. Accordingly, the document directly states that "Induction of cancer from long-term EMF exposure was not considered to be established, and so these guidelines are based on short-term, immediate health effects such as stimulation of peripheral nerves and muscles, shocks and burns caused by touching conducting objects, and elevated tissue temperatures resulting from absorption of energy during exposure to EMF." However, the basic assumption of that is questioned nowadays by numerous data sources.

According to that document a few parameters of EMR energy are recommended to be restricted. Among them the two parameters are used the most often: 1) Specific Absorption Rate (SAR) in W/kg, which indicates the EMR energy absorbed per mass unit of human tissue per second; and 2) power density or intensity of incident radiation in  $W/m^2$  (or  $\mu W/cm^2$ ) which indicates the amount of electromagnetic energy which falls on a unit of surface (under the right angle) per second. SAR safety limit for general public exposure indicated in Guidelines as 2 W/kg (for head and trunk) for the microwave range. To that, this limit is accepted by industry as mandatory for every commercial cell phone over the world, and real value of SAR of each cell phone model must be indicated in technical specification of the model. Unfortunately, SAR is rather sophisticated index for measurement. Moreover, only models of adult human head are currently used by industry for calculation of SAR, while real SAR values depend on a geometry and structure of tissues and, for example, was shown to be much higher for a child head than for the adult one [12-14].

Power density, or intensity of radiation, is much more direct and simple index as compared to SAR, although it does not estimate the specificity of interaction of EMR and the matter. Occupational exposure limits in microwave range according to ICNIRP are  $10-50 \text{ W/m}^2$ . Public exposure limits for microwaves according to ICNIRP recommendation were set to  $2-10 \text{ W/m}^2$  (or  $200-1000 \mu \text{W/cm}^2$ ) depending on frequency. For example, for GSM–900 MHz standard IC-NIRP safety limit will be calculated as  $450 \,\mu$ W/cm<sup>2</sup> [11].

It is important to note that ICNIRP recommendations have no legal validity, as it is only a recommendation. Each country has their own national legislation in the field of electromagnetic safety, and national limits are rather different in different countries. Some countries such as the USA and Germany conformed national EMR limits to ICNIRP recommendation. Other countries have much tougher national limits as compared with ICNIRP guidelines. For example, for GSM-900 MHz standard MW safety limits are: in Italy, Russia and China — 10  $\mu$ W/cm<sup>2</sup>, in Switzerland — 4  $\mu$ W/cm<sup>2</sup>, in Ukraine — 2.5  $\mu$ W/cm<sup>2</sup> [1]. As we can see, some countries, including Ukraine, have extremely strict national safety limits. Such national positions are explained first of all by long-term national research traditions in a field of electromagnetic biology, and on experience in studying the non-thermal biological effects of this kind of radiation. On the other hand, some countries like Switzerland follow a strict precautionary principle (Better protect than sorry).

# RADAR RADIATION AND CANCER PROMOTION

Substantial military and occupational data indicate a significant effect of pulse microwaves on cancer development and other pathological conditions in human. Accordingly, a statistically significant increase in immature red blood cells among workers exposed to a radar was reported [15]. In addition, radar-exposed workers had significantly lower levels of leukocytes and thrombocytes than workers distant from MW sources.

Among Polish soldiers (128 thousand personnel subjects aged from 20 to 59 years), soldiers of 20–29 years old exposed to radar microwaves during 1970–1979 had cancer incidence rates 5.5 folds higher than non-exposed soldiers [16]. The greatest rise of cancer cases was detected in blood-forming organs and lymphatic tissues: by 13.9 folds for chronic myelocytic leukemia and 8.6 folds for myeloblastic leukemia. The level of mortality among all exposed personnel was significantly higher than in unexposed: for colorectal cancer (observed-expected ratio, OER 3.2; 95 %), for cancer of esophagus and stomach (OER 3.2; 95 %), cancer of blood-forming system and lymphatic tissues (OER 6.3; 95 %) [17].

Almost two times more cases of cancer were indicated in the high-exposed American naval personnel served during the Korean War (1950–1954) as compared with the low-exposed subjects among 40 thousands of personnel [18]. Death rates for aviation electronic technicians, the group with the highest exposure rate, were significantly higher than those for the other personnel during the following years up to 1974 [15].

A very substantial increase in cancer incidence was also detected in commercial airline pilots. Thus, the standardized incidence ratio (SIR) for malignant melanoma cases was 10.2; 95.5 % for pilots of commercial airlines in Iceland [19]. Significantly increased risks of acute myeloid leukemia (SIR 5.1), skin cancer, excluding melanoma (SIR 3.0) and total cancer (SIR 1.2) were observed also among Danish male jet pilots [20]. These data have been explained as a result of excess cosmic ionizing radiation or even excessive sun radiation during a leisure time. However, analysis of brain cancers among US Air Force personnel has revealed that non-ionizing radiation and particularly MW had significant effect on cancer development (odds ratio, OR 1.38; 95%), whereas ionizing radiation had negative association with cancer cases (OR 0.58; 95%) [21]. To that, standardizing mortality ratio (SMR) for brain tumors was 2.1; 95 % among German male cockpit crew members (6,017 people) [22]. Cancer risk was significantly raised (risk ratio 2.2; 95%) among cockpit crew members employed for 30 years as compared to those employed for less than 10 years. In addition, Non-Hodgkin's lymphoma (NHL) was also increased (SMR 4.2; 95%) among male cabin crew members (20,757 people). Importantly, any increase in cancers associated with ionizing (cosmic) radiation was not detected in this cohort study.

In another report, six incident cases of testicular cancer occurred within a cohort of 340 police officers between 1979 and 1991 in Seattle, Washington, observed/expected ratio was 6.9; p<0.001 [23]. Occupational use of hand-held radar was the only shared risk factor among all six officers, and all had a routine habit of keeping the radar gun directly in close proximity to their testicles. Similarly, in Ontario, Canada risk assessment among police officers exposed to radar devices for speed measurement (1,596 females and 20,601 males) revealed an increased risk among men for testicular cancer (SIR 1.3) and for melanoma (SIR 1.45; 95 %) [24].

In another study, eighty seven persons working with radars (and 150 matched control) were divided into risk groups according to frequencies of MW (200 KHz to 26 GHz) and power density (8  $\mu$ W/cm<sup>2</sup>to 300  $\mu$ W/cm<sup>2</sup>) [15]. Three specific radiation cataracts in persons working with extremely high MW exposure were identified. Lens changes were associated with level of exposure in different risk groups.

Other occupational studies revealed the highest risk ratio (2.6) for acute myelogenous leukemia in radio and radar operators among all occupational groups studied [25]. In addition, excessive risk for breast cancer was detected (SIR 1.5) among Norwegian female radio and telegraph operators (2,619 women) with potential exposure to radio frequency (405 kHz – 25 MHz) [26].

# RADIATION FROM MOBILE COMMUNICATION SYSTEMS AND CANCER PROMOTION

**Cell phones.** A significant increase of risk of particular brain tumors in long-term (10 years or more) users of cell phones and cordless phones has been detected in series of epidemiological studies of Swedish oncologist Prof. L. Hardell with colleagues [27–33]. It is important that for a short-term use of cell phones similar effects were absent or less evident [4].

The risk of development of high-grade glioma has increased in more than 3 times (OR 3.1; 95 %) for bilateral users of cell phones and in more than 5 times (OR 5.4; 95%) for ipsilateral users after 10 years of using [34].

The risk of development of acoustic neuroma for bilateral users of cell phones was OR 2.9; 95% and OR 3.5; 95% for ipsilateral users after 10 years of using [29].

Notably, the highest risk of brain tumors has been detected in the youngest users of cell phones (20–29-yr) among all analyzed age groups (20–80 years old), with OR 5.91; 95% for ipsilateral use of cell phones. The highest risk was associated with more than 5-year using period in the 20–29-yr age group for analog cell phones (OR 8.17; 95%) [28].

International multiyear Interphone project conducted under the management of the World Health Organization and substantially supported by industry, was an interview-based case-control study with 2708 glioma and 2409 meningioma cases and matched controls, conducted in 13 countries using a common protocol [35]. The results of study were rather controversial. For example, authors were forced to declare "a reduced odds ratio related to ever having been a regular mobile phone users was seen for glioma (OR 0.81; 95 %) and meningioma (OR 0.79; 95 %), possibly reflecting participation bias or other methodological limitations." However, significantly increased risks of tumors development in "heavy" users of cell phones (with more than 1640 hours of using during less than four years) have been revealed in this study: for meningioma OR 4.8; 95 %, for glioma OR 3.77; 95% as compared with the matched controls [35]. One thousand and six hundred forty hours per four years means about one hour per day of a cell phone use. In this connection we can point to our data [36] that indicates amount of time which Ukrainian students (like students in other countries?) spend talking via cell phones every day. Our findings indicated that more than a half of them spend over one hour per day, and more than a quarter of them spend over two hours per day talking via cell phones every day.

Parotid gland, like a human brain, is another potential target for cell phone MW radiation during cell phone talks without hands-free devices. Thus, a study done by an Israeli team has indicated an association between a cell phone use and parotid gland tumors [37]. This study comprised 402 benign and 58 malignant cases of parotid gland tumors diagnosed in Israelis at age over 18 years in 2001–2003. The risk of parotid malignant tumors in intensive users of cell phones (for users with more than 5,479 hours of a use during less than five years) were OR 2.26; 95%. Recently new data have been published that totally a 4-fold increase of parotid malignant tumors in Israel during 1970–2006 took place, whereas other salivary glands tumors had been almost on a stable level during that period of time [38]. Previously, a Finnish study has revealed the OR 5.0; 95% for salivary gland cancer among all Finland digital cell phone subscribers compared with control population after one-two years of a cell phone use [39].

The odds ratio for Non-Hodgkin's lymphoma of T-cell, cutaneous and leukemia types has been found for analogue-cell-phone users as 3.4; 95%; for digital-phone users 6.1; 95%; and for cordless-phone users 5.5; 95% by L. Hardell group [40]. An American study indicated OR 1.6; 95% for NHL in users of cell phones with a period of use over eight years [41].

Uveal melanoma (in analysis of 118 cases with uveal melanoma and 475 controls in Germany) has been indicated to have odds ratio 4.2; 95% for people probable/certain exposed to cell phone radiation [42].

Testicular cancer (seminoma) risk had odds ratio 1.8; 95% for men keeping a cell phone during "stand by" in ipsilateral trousers pocket [43]. The results have been based on 542 cases of seminoma in Sweden.

**Base transmitting stations.** During the last decades more than one and half million base transmitting stations for mobile communication have been installed over the world. However, the World Health Organization suggested a priority to study effects mainly of cell phones, while discouraging studies on the effects of transmitting stations (with an exception of years 2003–2006 when WHO recommended studies of possible effects of radiation of transmitting stations as well) [44]. This is probably the main reason why only a few publications on this particular problem can be found to date [45–49].

The comparison of cancer cases among people living up to 400 m from base transmitting station and people living further than 400 m from station during 1994–2004 was carried out in Germany [48]. A total increase of cancer cases among people living nearby to transmitting station over the control population was 1.26 times during the first five-year period (1994–1998), and 3.11 times during the second fiveyear period (1999–2004) of operation of the station. Particularly, in the second period the increase of cancer cases was statistically significant both as compared with the population from more distant area and with the expected background incidence.

Population (n=622) living in the area nearby (up to 350 m) the cell phone base transmitting station (850 MHz, 1500 watt of full power) during one year of operation and matched individuals (n=1222) from other area have been compared In Israel [47]. There were 4.15 times more cases of cancer in transmitted station area than in the rest of a city. Relative cancer rates for females were 10.5 for close to station area, 0.6 for control area and 1 for the whole town. Cancer incidence of women in close to base station area was significantly higher (p<0.0001) as compared with the control area and the whole city. Keeping in mind that very significant increase in a number of cancer cases took place during only one year period, the authors of the study suggested that MW could provoke latent cases of cancer in inhabitants of the area nearby transmitting station.

French and Spanish researchers also revealed that inhabitants living near base station for mobile communication (up to 300 m) developed significantly higher rates of many subjective symptoms of health like headache, fatigue, sleep disorder, depression as compared with the matched control from distant area [49, 50].

# RODENT MODEL OF CANCER PROMOTION BY MICROWAVES

A highly representative research has been carried out at the University of Washington, Seattle commissioned by US Air Force [51]. The experimental rats (100 animals) were exposed during 24 months at 21.5 hours per day to 2,450-MHz pulsed microwaves at 800 pps with a 10 µs pulse width. The pulsed microwaves were square-wave modulated at 8 Hz. An average SAR was 0.4 W/kg for a 200-g rat. It was a model of long-term irradiation of Air Force pilots to pulsed microwaves of radar systems. Totally 155 indexes of metabolisms were checked out during the study. As a result, the most expressive effect of long-term MW irradiation of animals was a dramatic increase in a level of cancer cases. In total, 3.6 folds more cancer cases were detected in irradiated animals than in matched control. Lymphoma cases were diagnosed in the irradiated animals 4.5 times more often than in the control group. In addition, benign tumors of adrenal were detected seven folds more often in the irradiated animals than in the control.

In the next study under US Air Force contract, 200 female C3H/HeJ mice were exposed for 21 months (22 h/day, 7 days/week) to a horizontally polarized 435 MHz pulse-wave (1.0 ps pulse width, 1.0 kHz pulse rate) RF radiation environment with an incident power density of 1.0 mW/cm<sup>2</sup> (SAR 0.32 W/kg), while 200 mice were sham-exposed [52]. Although under the conditions of this study, an exposure of mice prone to mammary tumors did not affect the incidence of mammary tumors, when compared with the controls, some other tumor cases increased markedly. For example, bilateral cases of ovary epithelial stromal tumor raised by five folds; multiple cases of hepatocellular carcinoma, raised 3 folds, and adrenal gland tumor cases (total) raised 1.63 folds.

In the third published study of this series [53] the same prone-mammary tumor mice were irradiated during 20 months to continuous wave 2450 MHz MW radiation with SAR from 0.3 to 1 W/kg (20 h/day, 7 days/week). A hundred mice were exposed, while 100 mice were used as sham-exposed. As a result, the exposed mice had higher level of mammary tumors (1.27 folds), and higher total level of all types of tumor (1.38 folds) as compared with sham-exposed; the difference between groups was statistically insignificant. Meanwhile, multiple mammary tumor cases occurred in exposed mice twice more frequently than in sham exposed.

In other study mice with high incidence of spontaneous breast cancer and mice treated with 3,4-benzopyrene (BP) were irradiated to continuous wave 2,450 MHz microwaves in an anechoic chamber at 5 or 15 mW/cm<sup>2</sup> (2 hours daily, 6 sessions per week, 3 months) [54]. Irradiation with MW at either 5 or 15 mW/cm<sup>2</sup> resulted in acceleration of development of BP-induced skin cancer. Microwaves-exposed mice with high incidence of spontaneous breast cancer developed breast tumors earlier than control. Authors indicated that the promotion of cancer development and lowering of natural antineoplastic resistance was similar in mice exposed to MW at 5 mW/cm<sup>2</sup> and chronically stressed by confinement, but level of cancer cases in animals exposed to 15 mW/cm<sup>2</sup> was significantly higher as compared to chronically stressed by confinement control.

And in well-known study of M. Ripacholi *et al.* (1997) transgenic mice moderately predisposed to develop lymphoma spontaneously have been used for exposure to MW of 900 MHz, with pulse repetition frequency of 217 Hz, incident power densities of 2.6–13 W/m<sup>2</sup>, and average SAR of 0.13–1.4 W/kg [55]. One group of mice (101 females) has been exposed for two 30-min periods per day during 18 months. Another group of mice (100 females) has been a sham-exposed control. Lymphoma risk was significantly higher, more than twice, in the exposed mice than in the matched control (OR 2.4; 95 %). In particular, follicular lymphoma was the major contributor to the increased tumor incidence.

# MICROWAVES AND CELL METABOLISM

**Free radical species,** including reactive oxygen species (ROS), is an intrinsic feature of cell metabolism [56–58]. But disturbance of redox balance, uncontrolled activation of free radical processes, overproduction of ROS and/or suppression of antioxidant defense in cell often are the important signals of some hazardous changes in cell metabolism [59, 60]. That is why data indicated oxidative effect of some factor is extremely important in risk-assessment research.

A significant increase of ROS and nitrogen oxide generation in cells under non-thermal intensities of MW has been detected both in vivo [61-67] and in vitro [68-72]. Possibilities of mitochondrial and membrane NADH oxidase dependent ways of ROS generation in exposed cells have been suggested [71, 72]. Accordingly, it was found that the first step in MW (875 MHz, 0.07 mW/cm<sup>2</sup>) interaction with model cells (Rat1 and HeLa) was mediated in the plasma membrane by NADH oxidase, which can rapidly (during the minutes) generate ROS [72]. ROS directly stimulate matrix metalloproteinases and allow them to cleave and release heparin-binding epidermal growth factor (EGF). This secreted factor activates the EGF receptor, which in turn activates the extracellularsignal-regulated kinase (ERK) cascade and thereby induces transcription and other cellular pathways. On the other hand, on the model of purified human

spermatozoa exposed to MW (1.8 GHz, SAR from 0.4 W/kg to 27.5 W/kg) a significant overproduction of ROS in mitochondria was detected, along with a significant reduction in motility and vitality of spermatozoa [71]. All observed effects were significantly correlated with SAR levels, suggesting that significant effects of MW exposure occurred under non-thermal levels of MW.

Therefore, MW can induce cellular oxidative stress, which in turn can cause cancer stimulation [57, 59]. To that, it is known nowadays that in addition to damage via oxidative stress, ROS in cells can play a role of a secondary messenger for certain intracellular signaling cascades which can induce oncogenic transformation [60].

**DNA damage** in cells exposed to low-intensive microwaves both *in vivo* and *in vitro* was demonstrated during the last years in more than 50 independent studies [73]. The most often method used for detection of DNA damage after the MW exposure was alkaline Comet Assay. A statistically significant increase of both single strand and/or double strand breaks of DNA has been detected in humans [74, 75], animal models [76–79] and cell cultures [76, 80–83] exposed to low intensity microwaves.

Recently, an oxygen damage of DNA in human spermatozoa through formation of 8-hydroxi-2-deoxyguanosine (8-OH-dG) under non-thermal microwaves irradiation *in vitro* has been demonstrated [71].

Consequently, as DNA mutation is a critical step in carcinogenesis and increased level of 8-OH-dG takes place in many tumors [60], the possibility of MW to initiate oxidative damage of DNA is extremely dangerous signal for risk-assessment studies.

**Ornithine decarboxylase (ODC)** significantly changes its activity under conditions of non-thermal microwave exposure [84–88]. It was one of the first markers of carcinogenesis revealed to be activated under the low intensity microwaves exposure. ODC is involved in processes of cell growth and differentiation, and its activity is raised in tumor cells. Although overexpression of ODC is not sufficient for transformation of normal cells into tumorigenic ones, an increased activity of the enzyme was shown to promote the development of tumors from pre-tumor cells [89].

# **DISCUSSION AND CONCLUSIONS**

In this review we presented evidences for carcinogenic effects of low intensity microwaves. Both epidemiological and experimental data led us to a conclusion that at least under certain conditions the exposure to long term low intensity MW can lead to tumorigenesis. Supporting evidences come from statistically significant epidemiological data based either on longterm analysis, e.g., on mortality of US Navy personnel in 20 years after expose during the Korean War [15], or on relatively short, one year exposure, e.g., by base transmitting station for mobile communication in Israel [47]. In the latter case we fully agree with the authors that MW exposure most likely results in acceleration of pre-existed cancer development. It is of note here that the same conclusion was drawn in epidemiological research on fast increase cancer incidence among adult population in Colorado exposed to extremely low frequency radiation [90].

The main shortcoming of the most epidemiological data, both in military studies and in mobile communication risk assessment, is a lack of a strict dose measurement of exposure. We strongly suggest that in the forthcoming epidemiological studies the correct measurement of intensity and dosage of exposure should be obligatory. The example of a large-scale epidemiological research employing personal MW dosimeters can be found in recent studies in Germany [91–94]. On the other hand, we also realize that the levels of the MW exposure in contemporary epidemiological studies, at least in those which deal with mobile communication systems, were within the official "safety limits" set by appropriate national standards and ICNIRP recommendations. Therefore, taking into account the reviewed data, we conclude that the relatively long-term (e.g., 10 years) exposure to microwaves emitted from mobile communication devices operating within "safety limits" set by current regulating bodies can be considered as a potential factor for promotion of cancer growth. Indeed, in the most studies on rodents the intensity of MW exposure was appropriately measured, and in majority of them the MW intensity was below ICNIRP safety limits. Nevertheless, majority of these studies to a greater or lesser extent demonstrated obvious carcinogenic effects after long term exposure (up to 24 months). This further emphasizes that at least under certain conditions the exposure to both pulsed and continuous MW with intensities below the current official "safety limits" can indeed promote cancer development.

In addition, experimental evidences of involvement of typical markers of carcinogenesis like overproduction of reactive oxygen species or formation of 8-OH-dG under conditions of MW exposure further indicate potential danger of this type of radiation for human health. It is important to emphasize here that experimental data, especially obtained in studies *in vitro* often reveal significant biological effects even after short-term (e.g., only a few minutes) [72] and/or extremely weak intensity of exposure to MW (by several orders of magnitude lower than in ICNIRP recommendations) [95]. Taking these data into account we strongly suggest that currently used "thermal" assessment of potential hazards of MW exposure is far from being appropriate and safe.

Taken together, we state here that nowadays there is enough convincing data to appropriately assert that the long-term exposure to low intensity electromagnetic microwaves can indeed promote cancer development. To that, the official recommendations by ICNIRP and safety limits set by many national regulatory bodies for technical devices emitting microwave radiation, first of all for mobile communication systems, must be re-assessed according to the recent alarming data; and additional studies for unprejudiced risk assessment must be carried out. At present, we strongly suggest for a wide implementation of precautionary principle for everyday microwave exposure that implies maximum restriction of excessive exposure.

# **CONFLICT OF INTEREST**

The authors declare no conflict of interest.

# ACKNOWLEDGMENTS

This study was supported by National Academy of Sciences of Ukraine (Grant No 2.2.5.349); and received a financial contribution from the European Community within the Seventh Framework Programme (FP/2007–2013) under Grant Agreement No. 229603; and was also co-financed by the South Moravian Region via SoMoPro programme.

### REFERENCES

1. **Hardell L, Sage C.** Biological effects from electromagnetic field exposure and public exposure standards. Biomed Pharmacother 2008; **62**: 104–9.

2. Breckenkamp J, Berg G, Blettner M. Biological effects on human health due to radiofrequency/microwave exposure: a synopsis of cohort studies. Radiat Environ Biophys 2003; 42: 141–54.

3. Ahlbom A, Green A, Kheifets L, *et al.* Epidemiology of health effects of radiofrequency exposure. Environ Health Perspect 2004; **112**: 1741–54.

4. Morgan LL. Estimating the risk of brain tumors from cellphone use: Published case-control studies. Pathophysiology 2009; 16: 137–47.

5. Khurana VG, Teo C, Kundi M, *et al.* Cell phones and brain tumors: a review including the long-term epidemiologic data. Surg Neurol 2009; **72**: 205–15.

6. Hardell L, Carlberg M, Hansson Mild K. Epidemiological evidence for an association between use of wireless phones and tumor diseases. Pathophysiology 2009; 16: 113–22.

7. **Kundi M.** The controversy about a possible relationship between mobile phone use and cancer. Environ Health Perspect 2009; **117**: 316–24.

8. Leszczynski D, Xu Z. Mobile phone radiation health risk controversy: the reliability and sufficiency of science behind the safety standards. Health Res Policy Syst 2010; 8: 2.

9. Yakymenko I, Sidorik E. Risks of carcinogenesis from electromagnetic radiation of mobile telephony devices. Exp Oncol 2010; **32**: 54–60.

10. Yakymenko I, Sidorik E, Tsybulin O. Metabolic changes in living cells under electromagnetic radiation of mobile communication systems. Ukr Biokhim Zh 2011; 83: 5–13.

11. **ICNIRP.** Guidelines for limiting exposure to timevarying elecrtic, magnetic and electromagnetic fields (up to 300 GHz). Health Phys 1998; **74**: 494–522.

12. Gandhi O, Lazzi G, Furse C. Electromagnetic absorption in the human head and neck for mobile telephones at 835 and 1900 MHz. Microwave Theory and Techniques 1996; **44**: 1884–97.

13. **de Salles AA, Bulla G, Rodriguez CE.** Electromagnetic absorption in the head of adults and children due to mobile phone operation close to the head. Electromagn Biol Med 2006; **25**: 349–60.

14. Christ A, Gosselin MC, Christopoulou M, *et al.* Agedependent tissue-specific exposure of cell phone users. Phys Med Biol 2010; **55**: 1767–83. 15. **Goldsmith JR.** Epidemiological evidence relevant to radar (microwave) effects. Environ Health Perspect 1997; **105**: 1579–87.

16. Szmigielski S. Polish epidemiological study links RF/MW exposures to cancer. Microwave news 1985; 5: 1–2.

17. Szmigielski S. Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. Sci Total Environ 1996; 180: 9–17.

18. **Robinette CD, Silverman C, Jablon S.** Effects upon health of occupational exposure to microwave radiation (radar). Am J Epidemiol 1980; **112**: 39–53.

19. **Rafnsson V, Hrafnkelsson J, Tulinius H.** Incidence of cancer among commercial airline pilots. Occup Environ Med 2000; **57**: 175–9.

20. **Gundestrup M, Storm HH.** Radiation-induced acute myeloid leukaemia and other cancers in commercial jet cockpit crew: a population-based cohort study. Lancet 1999; **354**: 2029–31.

21. Grayson JK. Radiation exposure, socioeconomic status, and brain tumor risk in the US Air Force: a nested casecontrol study. Am J Epidemiol 1996; **143**: 480–6.

22. Zeeb H, Hammer GP, Langner I, *et al.* Cancer mortality among German aircrew: second follow-up. Radiat Environ Biophys 2010; **49**: 187–94.

23. Davis RL, Mostofi FK. Cluster of testicular cancer in police officers exposed to hand-held radar. Am J Ind Med 1993; 24: 231–3.

24. Finkelstein MM. Cancer incidence among Ontario police officers. Am J Ind Med 1998; 34: 157–62.

25. **Savitz DA, Calle EE.** Leukemia and occupational exposure to electromagnetic fields: review of epidemiologic surveys. J Occup Med 1987; **29**: 47–51.

26. **Tynes T, Hannevik M, Andersen A**, *et al*. Incidence of breast cancer in Norwegian female radio and telegraph operators. Cancer Causes Control 1996; **7**: 197–204.

27. Hardell L, Mild KH, Carlberg M. Case-control study on the use of cellular and cordless phones and the risk for malignant brain tumours. Int J Radiat Biol 2002; **78**: 931–6.

28. Hardell L, Mild KH, Carlberg M, *et al.* Cellular and cordless telephone use and the association with brain tumors in different age groups. Arch Environ Health 2004; **59**: 132–7.

29. Hardell L, Mild KH, Carlberg M, *et al.* Tumour risk associated with use of cellular telephones or cordless desktop telephones. World J Surg Oncol 2006; **4**: 74.

30. Hardell L, Hansson Mild K. Mobile phone use and risk of acoustic neuroma: results of the interphone case-control study in five North European countries. Br J Cancer 2006; **94**: 1348–9; author reply 52–3.

31. Hardell L, Carlberg M, Soderqvist F, *et al.* Long-term use of cellular phones and brain tumours: increased risk associated with use for > or =10 years. Occup Environ Med 2007; **64**: 626–32.

32. Hardell L, Carlberg M, Hansson Mild K. Case-control study on cellular and cordless telephones and the risk for acoustic neuroma or meningioma in patients diagnosed 2000–2003. Neuroepidemiology 2005; **25**: 120–8.

33. Hardell L, Carlberg M. Mobile phones, cordless phones and the risk for brain tumours. Int J Oncol 2009; **35**: 5–17.

34. Hardell L, Carlberg M, Mild KH. Case-control study of the association between the use of cellular and cordless telephones and malignant brain tumors diagnosed during 2000-2003. Environ Res 2006; 100: 232–41.

35. Cardis E, Deltour I, Vrijheid M, et al. Brain tumour risk in relation to mobile telephone use: results of the INTER-

PHONE international case-control study. Int J Epidemiol 2010; **39**: 675–94.

36. **Yakymenko I, Sidorik E, Tsybulin O**, *et al.* Potential risks of microwaves from mobile phones for youth health. Environment & Health 2011; **56**: 48–51.

37. Sadetzki S, Chetrit A, Jarus-Hakak A, *et al.* Cellular phone use and risk of benign and malignant parotid gland tumors — a nationwide case-control study. Am J Epidemiol 2008; **167**: 457–67.

38. Czerninski R, Zini A, Sgan-Cohen HD. Risk of parotid malignant tumors in Israel (1970-2006). Epidemiology 2011; 22: 130–1.

39. Auvinen A, Hietanen M, Luukkonen R, *et al.* Brain tumors and salivary gland cancers among cellular telephone users. Epidemiology 2002; **13**: 356–9.

40. Hardell L, Eriksson M, Carlberg M, *et al.* Use of cellular or cordless telephones and the risk for non-Hodgkin's lymphoma. Int Arch Occup Environ Health 2005; **78**: 625–32.

41. Linet MS, Taggart T, Severson RK, *et al.* Cellular telephones and non-Hodgkin lymphoma. Int J Cancer 2006; **119**: 2382–8.

42. Stang A, Anastassiou G, Ahrens W, *et al.* The possible role of radiofrequency radiation in the development of uveal melanoma. Epidemiology 2001; **12**: 7–12.

43. Hardell L, Carlberg M, Ohlson CG, *et al.* Use of cellular and cordless telephones and risk of testicular cancer. Int J Androl 2007; **30**: 115–22.

44. **Kundi M, Hutter HP.** Mobile phone base stations-Effects on wellbeing and health. Pathophysiology 2009; **16**: 123–35.

45. Abdel-Rassoul G, El-Fateh OA, Salem MA, *et al.* Neurobehavioral effects among inhabitants around mobile phone base stations. Neurotoxicology 2007; **28**: 434–40.

46. Hutter HP, Moshammer H, Wallner P, *et al.* Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations. Occup Environ Med 2006; **63**: 307–13.

47. Wolf R, Wolf D. Increased incidence of cancer near a cell-phone transmitted station. In: Columbus F., editor. Trends in cancer prevention: Nova Science Publishers, Inc, 2007: 1–8.

48. Eger H, Hagen K, Lucas B, *et al.* Einfluss der räumlichen Nähe von Mobilfunksendeanlagen auf die Krebsinzidenz. Umwelt-Medizin-Gesellschaft 2004; **17**: 273–356.

49. Santini R, Santini P, Danze JM, *et al.* Study of the health of people living in the vicinity of mobile phone base stations: 1. Influences of distance and sex. Pathol Biol 2002; **50**: 369–73.

50. Navarro E, Segura J, Portoles M, *et al.* The Microwave Syndrome: A Preliminary Study in Spain Electromagn Biol Med 2003; **22**: 161–9.

51. Chou CK, Guy AW, Kunz LL, *et al.* Long-term, low-level microwave irradiation of rats. Bioelectromagnetics 1992; **13**: 469–96.

52. Toler JC, Shelton WW, Frei MR, *et al.* Long-term, low-level exposure of mice prone to mammary tumors to 435 MHz radiofrequency radiation. Radiat Res 1997; **148**: 227–34.

53. Frei MR, Jauchem JR, Dusch SJ, *et al.* Chronic, low-level (1.0 W/kg) exposure of mice prone to mammary cancer to 2450 MHz microwaves. Radiat Res 1998; **150**: 568–76.

54. Szmigielski S, Szudzinski A, Pietraszek A, et al. Accelerated development of spontaneous and benzopyrene-induced skin cancer in mice exposed to 2450-MHz microwave radiation. Bioelectromagnetics 1982; **3**: 179–91.

55. **Repacholi MH, Basten A, Gebski V, et al.** Lymphomas in E mu-Pim1 transgenic mice exposed to pulsed 900 MHZ electromagnetic fields. Radiat Res 1997; **147**: 631–40.

56. Kamata H, Hirata H. Redox regulation of cellular signalling. Cell Signal 1999; 11: 1–14.

57. Halliwell B, Whiteman M. Measuring reactive species and oxidative damage in vivo and in cell culture: how should you do it and what do the results mean? Br J Pharmacol 2004; 142: 231–55.

58. Nemoto S, Takeda K, Yu ZX, *et al.* Role for mitochondrial oxidants as regulators of cellular metabolism. Mol Cell Biol 2000; **20**: 7311–8.

59. Valko M, Leibfritz D, Moncol J, *et al.* Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem Cell Biol 2007; **39**: 44–84.

60. Valko M, Rhodes CJ, Moncol J, *et al.* Free radicals, metals and antioxidants in oxidative stress-induced cancer. Chem Biol Interact 2006; **160**: 1–40.

61. Ferreira AR, Bonatto F, de Bittencourt Pasquali MA, *et al.* Oxidative stress effects on the central nervous system of rats after acute exposure to ultra high frequency electromagnetic fields. Bioelectromagnetics 2006; **27**: 487–93.

62. Grigoriev YG, Grigoriev OA, Ivanov AA, *et al.* Confirmation studies of Soviet research on immunological effects of microwaves: Russian immunology results. Bioelectromagnetics 2010; **31**: 589–602.

63. Irmak MK, Fadillioglu E, Gulec M, *et al.* Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits. Cell Biochem Funct 2002; **20**: 279–83.

64. **Ozgur E, Guler G, Seyhan N.** Mobile phone radiationinduced free radical damage in the liver is inhibited by the antioxidants N-acetyl cysteine and epigallocatechin-gallate. Int J Radiat Biol 2010; **86**: 935–45.

65. **Ozguner F, Altinbas A, Ozaydin M, et al.** Mobile phone-induced myocardial oxidative stress: protection by a novel antioxidant agent caffeic acid phenethyl ester. Toxicol Ind Health 2005; **21**: 223–30.

66. **Ozguner F, Oktem F, Ayata A**, *et al*. A novel antioxidant agent caffeic acid phenethyl ester prevents long-term mobile phone exposure-induced renal impairment in rat. Prognostic value of malondialdehyde, N-acetyl-beta-D-glucosaminidase and nitric oxide determination. Mol Cell Biochem 2005; **277**: 73–80.

67. Sokolovic D, Djindjic B, Nikolic J, *et al.* Melatonin reduces oxidative stress induced by chronic exposure of microwave radiation from mobile phones in rat brain. J Radiat Res (Tokyo) 2008; **49**: 579–86.

68. Agarwal A, Desai NR, Makker K, *et al.* Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an in vitro pilot study. Fertil Steril 2009; **92**: 1318–25.

69. Luukkonen J, Hakulinen P, Maki-Paakkanen J, *et al.* Enhancement of chemically induced reactive oxygen species production and DNA damage in human SH-SY5Y neuroblastoma cells by 872 MHz radiofrequency radiation. Mutat Res 2009; **662**: 54–8.

70. **Zmyslony M, Politanski P, Rajkowska E**, *et al.* Acute exposure to 930 MHz CW electromagnetic radiation in vitro affects reactive oxygen species level in rat lymphocytes treated by iron ions. Bioelectromagnetics 2004; **25**: 324–8.

71. **De Iuliis GN, Newey RJ, King BV**, *et al.* Mobile phone radiation induces reactive oxygen species production and DNA damage in human spermatozoa in vitro. PLoS One 2009; **4**: e6446.

72. Friedman J, Kraus S, Hauptman Y, *et al.* Mechanism of short-term ERK activation by electromagnetic fields at mobile phone frequencies. Biochem J 2007; **405**: 559–68.

73. **Ruediger HW.** Genotoxic effects of radiofrequency electromagnetic fields. Pathophysiology 2009; **16**: 89–102.

74. Gandhi G, Anita. Genetic damage in mobile phone users: some preliminary findings. Indian J. Hum. Gent. 2005; 11: 99–104.

75. Yadav AS, Sharma MK. Increased frequency of micronucleated exfoliated cells among humans exposed in vivo to mobile telephone radiations. Mutat Res 2008; **650**: 175–80.

76. Lai H, Singh NP. Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. Bioelectromagnetics 1995; **16**: 207–10.

77. Lai H, Singh NP. Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. Int J Radiat Biol 1996; **69**: 513–21.

78. Ferreira AR, Knakievicz T, Pasquali MA, *et al.* Ultra high frequency-electromagnetic field irradiation during pregnancy leads to an increase in erythrocytes micronuclei incidence in rat offspring. Life Sci 2006; **80**: 43–50.

79. **Kesari KK, Behari J, Kumar S.** Mutagenic response of 2.45 GHz radiation exposure on rat brain. Int J Radiat Biol 2010; **86**: 334–43.

80. **Diem E, Schwarz C, Adlkofer F**, *et al.* Non-thermal DNA breakage by mobile-phone radiation (1800 MHz) in human fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro. Mutat Res 2005; **583**: 178–83.

81. **Paulraj R, Behari J.** Single strand DNA breaks in rat brain cells exposed to microwave radiation. Mutat Res 2006; **596**: 76–80.

82. Wu W, Yao K, Wang KJ, *et al.* Blocking 1800 MHz mobile phone radiation-induced reactive oxygen species production and DNA damage in lens epithelial cells by noise magnetic fields. Zhejiang Da Xue Xue Bao Yi Xue Ban 2008; **37**: 34–8.

83. Schwarz C, Kratochvil E, Pilger A, *et al.* Radiofrequency electromagnetic fields (UMTS, 1,950 MHz) induce genotoxic effects in vitro in human fibroblasts but not in lymphocytes. Int Arch Occup Environ Health 2008; **81**: 755–67.

84. **Paulraj R, Behari J, Rao AR.** Effect of amplitude modulated RF radiation on calcium ion efflux and ODC

activity in chronically exposed rat brain. Indian J Biochem Biophys 1999; **36**: 337–40.

85. **Byus CV, Kartun K, Pieper S**, *et al.* Increased ornithine decarboxylase activity in cultured cells exposed to low energy modulated microwave fields and phorbol ester tumor promoters. Cancer Res 1988; **48**: 4222–6.

86. Litovitz TA, Krause D, Penafiel M, *et al.* The role of coherence time in the effect of microwaves on ornithine decarboxylase activity. Bioelectromagnetics 1993; **14**: 395–403.

87. Litovitz TA, Penafiel LM, Farrel JM, *et al.* Bioeffects induced by exposure to microwaves are mitigated by superposition of ELF noise. Bioelectromagnetics 1997; **18**: 422–30.

88. **Hoyto A, Juutilainen J, Naarala J.** Ornithine decarboxylase activity is affected in primary astrocytes but not in secondary cell lines exposed to 872 MHz RF radiation. Int J Radiat Biol 2007; **83**: 367–74.

89. Clifford A, Morgan D, Yuspa SH, *et al.* Role of ornithine decarboxylase in epidermal tumorigenesis. Cancer Res 1995; **55**: 1680–6.

90. Wertheimer N, Leeper E. Adult cancer related to electrical wires near the home. Int J Epidemiol 1982; 11: 345–55.

91. **Roosli M, Frei P, Bolte J**, *et al.* Conduct of a personal radiofrequency electromagnetic field measurement study: proposed study protocol. Environ Health 2010; **9**: 23.

92. Heinrich S, Thomas S, Heumann C, et al. Association between exposure to radiofrequency electromagnetic fields assessed by dosimetry and acute symptoms in children and adolescents: a population based cross-sectional study. Environ Health 2010; **9**: 75.

93. Milde-Busch A, von Kries R, Thomas S, *et al.* The association between use of electronic media and prevalence of headache in adolescents: results from a population-based cross-sectional study. BMC Neurol 2010; **10**: 12.

94. Thomas S, Heinrich S, Kuhnlein A, *et al.* The association between socioeconomic status and exposure to mobile telecommunication networks in children and adolescents. Bioelectromagnetics 2010; **31**: 20–7.

95. **De Pomerai D, Daniells C, David H**, *et al*. Non-thermal heat-shock response to microwaves. Nature 2000; **405**: 417–8.