

# WHY THE PRECAUTIONARY APPROACH IS NEEDED FOR NON-IONISING RADIATION DEVICES

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## ABSTRACT

There is now sufficient evidence on the actual and potential adverse health effects of radio frequency (RF) devices like mobile phones and Wi-Fi to require the application of the Precautionary Approach to the management of the health risks.

In 2011, the International Agency for Research on Cancer (IARC, a WHO agency) classified radiofrequency electromagnetic fields as a possible human carcinogen.

The Precautionary Approach is enshrined in various pieces of Australian legislation from public health to fisheries; however, it is yet to be applied in national approaches to radiation.

The Precautionary Approach can provide a means of moving forward in the face of uncertainty, reducing the risk of possible adverse health effects and promoting research to better understand these health effects. The “ALARA” principle can be considered a historical version of the Precautionary Approach.

This paper examines the evidence for health effects and how the Precautionary Approach can be implemented and discusses the wider issues around its use.

### Key Words

Electromagnetic Radiation, EMR, EME, EMF, RF, Microwaves, Wi-Fi, Mobile Phones, Health, Cancer, Precautionary Approach.

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## 1. THE DIFFERENCE BETWEEN THE PRECAUTIONARY PRINCIPLE AND APPROACH

The terms “Precautionary Approach” and “Precautionary Principle” are often used interchangeably, but there is a difference<sup>1</sup>. In legal terms, a principle (of law) is a fundamental statement and binding. There is some hesitation in using the term “principle” to guide the management of risk when there are scientific uncertainties. The concept of “approach” is

more flexible and implies a way of focussing on risks in a prudent manner. In international law, the terms are often interchanged<sup>2</sup> but there are subtle differences, which are discussed below. The term “Precautionary Approach” rather than “Precautionary Principle” will be used, as the effective differences are small.

These concepts were explored in an unpublished White Paper<sup>3</sup> by one of us (DB) for SafeWork Australia, to facilitate a move towards adopting the Precautionary Approach to extend the “Code of Practice on Risk Management” in workplaces<sup>4</sup>, when there was scientific uncertainty about the risk.

### 1.1 The concept of precaution

The concept of precaution is reflected in sayings like “an ounce of prevention is worth a pound of cure” and “look before you leap”. It is countered by other sayings like “nothing ventured, nothing

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gained”, and “he who hesitates is lost”.

The principal shift in the management of risk in the face of uncertainty is the change from asking “What level of harm is acceptable?” to a precautionary approach which asks, “How can we prevent harm?”. It is also a shift from “damage control” and “polluter pays” to “pre-damage control” or prevention<sup>5</sup>.

### 1.2 Precautionary Principle

There are various forms of the definition of the Precautionary Principle internationally, which can be classified as weak, moderate or strong, depending on the degree of onus on the regulator to act and whether there is a reversal of the “burden of proof”<sup>5</sup>.

- The **weak version** is the least restrictive and allows preventive measures to be taken in the face of uncertainty, but does not require them. The 1992 Rio Declaration is an example of the “weak” version. Many “weak” versions explicitly consider the cost-effectiveness of precautionary measures.
- In **moderate versions** of the principle, the presence of an uncertain threat is a positive basis for action, once it has been established that a sufficiently serious threat exists.
- The **strong versions** of the principle differs from the weak and moderate versions in reversing the burden of proof and in responsibility for paying the cost.

For a significant health hazard, the creators of the hazard could be expected to accept most of the cost in adopting the Precautionary Approach, particularly where large populations are involved.

### 1.3 Precautionary Approach

A simple and general definition of the Precautionary Approach could be “A risk management framework in the face of scientific uncertainty”.

However, the successful implementation of the Precautionary Approach is complex and requires widespread trust in the process to make it workable and acceptable. The approach has to be seen to be fair and reasonable. For this to be evident, a high degree of transparency and openness is required. Use of the Precautionary Approach is effectively a social license to continue business in the face of the uncertainty of a known risk.

### 1.4 ALARA and ALARP

As Low As Reasonable Achievable (ALARA) and As Low As Reasonable Practicable (ALARP)

are historical Precautionary Approaches, but lack the same formal structures that surround the comprehensive implementation of the Precautionary Approach.

The ALARP principle was used in the UK Health and Safety at Work etc. Act 1974 and required the “*Provision and maintenance of plant and systems of work that are, so far as is reasonably practicable, safe and without risks to health*”. The meaning of “reasonably practicable” has been the subject of UK case law since 1949 – risks must be averted unless there is a large imbalance between the costs and benefits of doing so. ALARA is widely used with ionizing radiation.

### 1.5 Trigger Points

The trigger points for invoking the Precautionary Principle can be variable depending on the perceived or likelihood of risk<sup>6</sup>. There are two main factors that trigger the precautionary approach. These are:

- the **strength of evidence** and
- the potential **cost of doing nothing**.

As in the case of many toxic agents, the delay between disease and full biological explanation can occur over many decades; for example;

- Asbestos (1898–1999): 101 years
- Water contaminated with cholera, Dr John Snow (1854–1883): 29 years.

## 2. CURRENT AUSTRALIAN EMF-RF REGULATION

The Australian Communications Media Authority (ACMA) is the radio communications regulator. The *Radiocommunications Act 1992* Section 162 (3) (f)<sup>7</sup> imposes upon ACMA the regulatory responsibility to provide health and safety protection to persons who operate, work with or use wireless equipment via the establishment of standards.

Currently, the ACMA collects revenue on behalf of the Australian Government through broadcasting, radiocommunications and telecommunications taxes, charges and licence fees. It also collects revenue from price-based allocation of the RF spectrum. When government health and safety protection responsibilities are considered, there are obvious conflicts of interest in those roles.

Another major problem which is often overlooked is that when ACMA chose subsections of the ARPANSA RPS3 (based on ICNIRP 1998 RF Guidelines) as the “standard” to provide health and safety protection, it specifically excluded 5.7 (e) covering precautionary measures because “(i) inclusion of the precautionary principle in the ACMA regulatory instruments would place a regulatory burden on industry which would require strong justification. The ACMA does not discern that justification.”

### 3. THE EVIDENCE FOR HEALTH EFFECTS OF EMF-RF

The current ICNIRP Guidelines were developed in 1998, well before the explosion in mobile phone use. The guidelines are largely based on thermal effects. Non-thermal effects are effectively ignored, because in 1998 there was no substantiated evidence of health effects on humans.

The ICNIRP 2002 statement<sup>8</sup> under “People being protected” notes that:

*“Different groups in a population may have differences in their ability to tolerate a particular NIR exposure. For example, children, the elderly, and some chronically ill people might have a lower tolerance for one or more forms of NIR exposure than the rest of the population.”*

However, ICNIRP has made no attempt to take a Precautionary Approach for these at-risk groups, instead placing the onus of responsibility on “relevant authorities in each country”.

The ARPANSA Review TR-164 for the period 2000–2012 has been determined to be a flawed document. The authors not only failed to use the available evidence in their own database but they completely ignored the overwhelming evidence in the scientific literature associated with oxidative stress effects<sup>9</sup>.

The supposition in TR-164 that microwave radiofrequency transmissions cannot cause cancer is flawed. It is argued that a photon of this wavelength does not have enough energy to directly break ionic bonds like an X-ray, and therefore could not possibly cause damage or mutations in DNA. This simplistic approach sounds like good physics, but it isn’t good biology. Ionising radiation is only one way of causing the mutations in DNA which can produce cancer.

Another common pathway shared by all causes is that they produce an inflammatory response in the body which increases the activity of free radicals (reactive oxygen species or ROS). These free radicals produce oxidative damage in the tissues<sup>10</sup>.

#### 3.1 Evidence ORSAA Database – Human epidemiological studies

The ORSAA database contains 252 epidemiological studies that are specific to mobile phones as shown in Figure 1. Some of these studies are country specific, while others are international studies. Other smaller studies are often occupationally based. These studies have been conducted to explore the relationship between mobile phones and health.

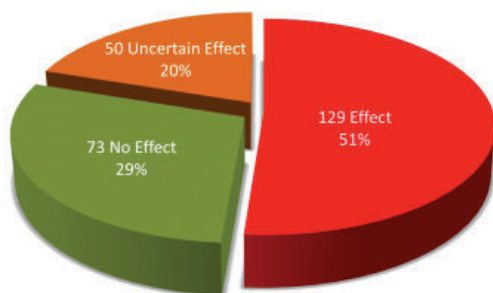
One of the problems with studies that have demonstrated an association is that use of cumulative hours is a crude measure of exposure, because while a mobile phone user may have a low number of annual hours, they still may have had large exposure because of poor signal strength.

Other flaws with mobile phone epidemiology are<sup>11</sup>:

1. Selection bias
2. Recall bias
3. Insufficient latency time
4. Definition of “regular” cellphone user
5. Inclusion of business users with controls
6. Inclusion of cordless phone users with controls
7. Exclusion of young adults and children
8. Brain tumour risk from cell phones radiating higher power levels in rural areas not investigated
9. Exposure to other transmitting sources are not considered
10. Exclusion of brain tumour types
11. Tumours outside the cellphone’s radiation plume are treated as exposed
12. Exclusion of brain tumour cases because of death or participants too ill to respond.

Despite all these flaws, some flaws diluting the ability of studies to detect significant effects, there are studies which do show a positive statistical association between cumulative hours used and

certain rare nerve tumours found in the brain and ear. Figure 1 shows the analysis of all 252 epidemiological studies in the ORSAA database.



**Figure 1**  
Summary of the evidence for UHF studies

In the INTERPHONE study (2000-2004) [12] some 6100 cancer patients were interviewed. These patients had rare nerve sheath tumour types (Glioma and Acoustic Neurinoma), as summarized in Table 1.

The Interphone study showed an increase in the risk of glioma in the group with the longest duration of use ( $\geq 1640$  h) (OR=1.40; 95% CI 1.03 to 1.89), higher for ipsilateral use and temporal tumours. This association was not observed for meningiomas (OR=1.15; 95% CI 0.81 to 1.62)<sup>12</sup>.

A multi-centre case-control study by French scientists called CERENAT<sup>13</sup> was carried out in four areas in France in 2004–2006. Data about mobile phone use was collected through a detailed questionnaire delivered face-to-face. No association with brain tumours was observed for casual mobile phone use. However, a statistically significant association was found in the heaviest users. In these users, time since first use was mostly 5–9 years (49%) or 10 years and more (40%), corresponding to approximately 54 min/day.

Another possible source of bias with epidemiological studies relates to when mobile phone use is measured via self-reporting. However, the COSMOS study<sup>14</sup> investigated the validity of self-reported mobile phone use and found a

(surprising) reasonable agreement between self reported use and the actual hours of use as obtained from the mobile phone operators.

Interestingly in the COSMOS study, a number of questions on health effects revealed that 14% reported experiencing symptoms around using a mobile phone and 59% reported some level of concern about mobile phones and health (ranging from 36% showing a little concern, up to 1% showing extreme concern).

### 3.2 Bradford Hill system of causation

Within the ORSAA database we have incorporated Bradford Hill's "nine indexes of causation"<sup>15</sup>. Hill's criteria (i.e. indices) for causation are a group of minimal conditions necessary to provide adequate evidence of a causal relationship between an incidence and a possible consequence. For EMF, the Bradford Hill index "Analogy" is not applicable and is thus omitted. For each epidemiological study in the database, the number of Hill indices that are satisfied within that particular paper are noted. With the ability to compile reports, the ORSAA database provides a summary of the most frequently noted indices (Figure 2). Of the eight indices used, four are regularly present within the epidemiology studies; e.g., plausibility.

This application of the Bradford Hill system of causation has not previously been incorporated in this manner to assist in determining whether causation is likely.

### 3.3 Recognition of causation

Recognition of the possibility of causation between RF-EMF and health effects occurred in May 2011 when the WHO's International Agency for Research on Cancer (IARC) classified RF-EMR as a **Group 2B possible human carcinogen**<sup>16</sup>.

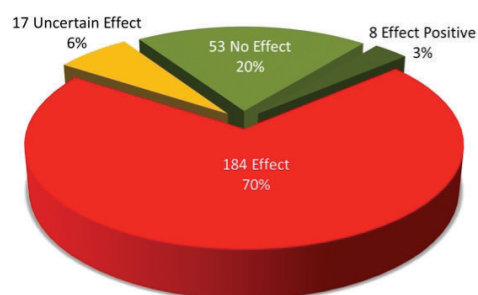
Some researchers<sup>17</sup> believe that the evidence for glioma and acoustic neuroma (Vestibular Schwannoma) being caused by RF-EMF emissions from wireless phone is strong. Furthermore, according to the IARC Preamble<sup>16</sup>, the classification should be upgraded to Group 1, i.e., "the agent is

Patients interviewed	Type of tumour	Organ
2708	Glioma	Brain
2409	Meningioma	Brain
1100	Acoustic Neurinoma (Vestibular Schwannoma)	Acoustic nerve
400	Parotid gland	Salivary gland

**Table 1**  
Summary of Interphone and Patients studied



**Figure 2**  
**Summary of Bradford Hill Indices for**  
**Epidemiological studies for UHF studies**  
**Source: ORSAA.org website**



**Figure 3**  
***In vivo* animal studies for ≤ 2 W/kg**

carcinogenic to humans”. A revision of the current guidelines for RF-EMF exposure is needed.

### 3.4 *In vivo* human UHF studies

The *in vivo* studies in both the ORSAA and ARPANSA databases are predominantly animal studies, along with a very limited number of human studies, such as EEG or ECG monitoring of human female volunteers (with foetal and neonatal exposure) and some blood or saliva testing from provocation studies. Table 2 summarises the effects shown by such studies, as reported by the ORSAA database.

### 3.5 *In vivo* animal UHF studies

The bio-effects animal studies for those with Specific Absorption Rate (SAR) exposures ≤ 2 W/kg show that 70% of all studies are “Effect” studies and far outweigh the “No Effect” studies (Figure 3). Furthermore, 3% of the studies are noted as “Effect Positive” studies. The authors of these studies have noted that exposures can lead to potential therapies being required, e.g. for conditions like reversing Alzheimer’s disease, anti-inflammation protective, adaptive response for cancer treatment, or bone healing.

Bio-effects data from *in vivo* animal experiments using exposures below the “member of the public”

exposure limit (≤ SAR 2 W/kg) reveal some interesting patterns in the bio-effects. For example, in Table 3, the bio-effect “DNA damage” for short-term experiments is ranked sixth while for longer-term studies, the bio-effect “DNA damage” moves up the ranking of effects. Unfortunately, most of the experiments are short-term and this trend would need more detail investigation.

### 3.6 *In vivo* long-term animal UHF studies

The US Food and Drug Administration (FDA) nominated the need for toxicology and carcinogenicity testing of cell phone RFR emissions in 1999. At that time, animal experiments were deemed crucial, because meaningful human exposure data from epidemiological studies were not available. A decade later this work was commissioned by the US National Toxicology Program (NTP), an inter-agency program run by the US Department of Health and Human Services to co-ordinate, evaluate, and report on toxicology within public agencies. The NPT is headquartered at the US National Institute of Environmental Health Sciences (NIEHS).

The NPT study was a 2-year study on rats and mice<sup>18</sup> and the most comprehensive study conducted on the long term health risks of wireless technology, using the following design characteristics:

Organ or fluids sampled	Studies			Top 6 major Bio-Effect categories
	Effect	No Effect	Uncertain Effect	
Saliva; Blood (Haemoglobin, chromosomes & lymphocytes); Sperm; Skin; Auditory system; Core Temperature; Pituitary Hormones; Urine; Faeces; EEG studies’ ECG studies	51	10	7	<b>DNA damage</b> Biochemical changes Altered Enzyme Activity Cell Irregularities/ Damage/ Oxidative Stress Cardiovascular/ Vascular Effects

**Table 2**  
***In vivo* testing from Human Epidemiological studies – UHF Studies**



Cumulative Exposure range (h)	Number of studies			First Six (6) Bio-Effect categories (Number of Effect studies in parenthesis)
	Effects	No Effects	Uncertain	
<b>Group 1</b> ≤ 100 h (≤ 4.2 d)	366 (79%)	85 (18%)	14 (3%)	<ol style="list-style-type: none"> <li>Biochemical changes (167)</li> <li>Altered Enzyme Activity (144)</li> <li>Oxidative Stress (122)</li> <li>Cell Irregularities/ Damage (82)</li> <li>Neuro-behavioural Effects/ Cognitive Effects (53)</li> <li><b>DNA damage</b>/ Mutagenic /Genotoxic (42)</li> </ol>
<b>Group 2</b> 100 to ≤ 750 h (4.2 d to 1 month)	76 (76%)	21 (21%)	3 (3%)	<ol style="list-style-type: none"> <li>Biochemical changes (35)</li> <li>Altered Enzyme Activity (33)</li> <li>Oxidative Stress (29)</li> <li>Cell Irregularities/ Damage (18)</li> <li>Apoptosis (Programmed cell death) (12)</li> <li><b>DNA damage</b>/ Mutagenic /Genotoxic (11)</li> </ol>
<b>Group 3</b> >750 ≤8700 h	19	11	1	<ol style="list-style-type: none"> <li>Biochemical changes (7)</li> <li>Altered Enzyme Activity (6)</li> <li><b>DNA damage</b>/ Mutagenic /Genotoxic (4)</li> <li>Oxidative Stress (4)</li> <li>Sperm effects (4)</li> <li>Apoptosis (Programmed cell death) (3)</li> </ol>
<b>Group 4</b> >8700 h (≥ 1 year)	9	2	3	<ol style="list-style-type: none"> <li><b>DNA damage</b>/ Mutagenic /Genotoxic (3)</li> <li>Cell Irregularities/ Damage (3)</li> <li>Altered Enzyme Activity (2)</li> <li>Sperm effects (2)</li> <li>Tumour Promotion (2)</li> <li>Altered Enzyme Activity (2)</li> </ol>

**Table 3**  
*In vivo* UHF All animal studies

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- Double the usual number of animals required for this type of study,
  - Three exposure groups: 1.5 W/kg, 3W/kg, and 6 W/kg,
  - Exposure intensity at low non-thermal or non-heating levels,
  - Near-field study,
  - Three independent panels for determining whether the abnormal tissues were cancerous,
  - The NTP solicited unprecedented review from multiple external scientists to critically review all aspects of the data analysis and study findings.
- The results of the study were insensley interpreted as follows:
- Evidence of Carcinogenic activity was rated as:
    - 1) Clear evidence
    - 2) Some evidence
    - 3) Equivocal evidence of carcinogenic ( demonstrated by studies that are interpreted as showing a marginal increase of neoplasms which may be test agent related)
    - 4) No evidence
    - 5) Inadequate study.
  - Exposure to GSM- or CDMA-modulated cell phone RFR at 1,900 MHz did not increase the incidence of any non-neoplastic lesions in male or female B6C3F1/N mice.
  - Rat exposures resulted in different outcomes for males and females. Some occurrences of rare nerve sheath tumours were statistically significant and others were not. However, rare nerve tumours, malignant Schwannoma tumours were found in the hearts of **male** rats. These cell types are also found in the nerves of the human ear.

- Significant positive trends were found for gliomas in **male** rats exposed to CDMA-modulated RF radiation and for heart schwannomas tumours in **male** rats exposed to GSM or CDMA-modulated RF.
- Malignant gliomas in the brain were present in exposed rats compared to controls; however, this finding was not statistically significant and was thus labelled as equivocal evidence.

A similar far-field animal study has been completed by the Ramazzini Institute in Italy [19]. An interim paper shows the same rare nerve tumours have been found in **male** rats as in the NPT study. However, these rare nerve tumours were also present in the control **male** rats, which was not the case in the NPT study. These results are less convincing than the NPT study.

Similar the findings of both studies, gliomas in humans are more common in men than in women<sup>20</sup>.

The question is: *Does rat research inform human health risk?* The answer is *It does inform*, which is a given in common practice whereby:

- Rats are the preferred animal models for carcinogenicity studies.
- Regulatory agencies currently rely on rodent carcinogenicity bioassay data to predict whether or not a given chemical poses a carcinogenic threat to humans.

#### 4. RF EXPOSURE PRODUCES OXIDATIVE STRESS

Chronic inflammation can cause cancer. Cigarette smoke can cause cancer. Toxins and autoimmune disease can cause cancer. One common pathway

shared by these causes is that they produce an inflammatory response in the body which increases the activity of free radicals (reactive oxygen species (ROS) /reactive nitrogen species (RNS)). These free radicals produce damage in the tissues.

This oxidative activity is the tool that the human body uses to break down foreign bacteria (DNA fragments etc.) so that it can be digested by the immune system. While free radicals are an important defensive weapon, an excess of oxidative activity can lead to damage of human tissue. Such excesses have been associated with many chronic conditions including autoimmune disease, heart disease, and some forms of cancer. Every week another article is published suggesting that taking antioxidants may be protective against some of these problems.

Though immune cells such as activated phagocytes also produce ROS to kill invading pathogens, which is part of the natural immune defence mechanisms, in environmental toxic exposures it appears that ROS precedes the inflammation (i.e. oxidative stress is the trigger for inflammation). This is further complicated by disruption of the signal transduction pathways, as carefully maintained ROS/RNS are important messengers in keeping a cell's homeostasis. Table 4 illustrates how the long-term bio-effect of oxidative stress is linked to both cardiovascular disease, neurodegenerative diseases and cancer<sup>21-24</sup>.

#### 5. CONCLUSIONS

The evidence that there are health effects from long term exposure to wireless devices like mobile phones is strong. Therefore, using the moderate version of the *burden of proof* described above, further research is needed to clarify the risks. In addition, RF Standards need to be strengthened to provide biological protection against a range

Top Health Burdens	RF-EMR bio effects with evidence
Cardiovascular Disease	Cardiac and vascular effects, <b>oxidative stress</b> and effects on voltage-gated Ca <sup>2+</sup> channels
Cancer	DNA damage, altered cell metabolism, altered gene expression, <b>oxidative stress</b> , inflammation
Neurodegenerative diseases	Neuronal damage (evidence of functional and histopathological changes), <b>oxidative stress</b> , metabolic changes, blood brain barrier damage
Mental illness	Neurobehavioural changes including anxiety, cognitive impairment, changes in neurotransmitters
Allergies	Serological evidence of elevation of IgE antibodies and Th2 cytokines, lowering of cytotoxic activity of white blood cells, mast cell degranulation

Table 4  
Chronic Diseases in Modern Society – Does Anthropogenic EMR have a Role?

of bio-effects noted in well conducted research at a non-thermal level. Radiation protection authorities need to be more forthright with their advice and should be recommending a precautionary approach to government, industry and the general public. For example:

- Mobile phones need to be designed to a higher safety standard which considers non-thermal biological effects.
- Stronger recommendations need to be adopted and publicised to ensure safer use by the general public, most especially children and other sensitive individuals.

Currently, it is up to the user to apply a Precautionary Approach to the use of mobile phone and other wireless technology. The awareness of the risk of potential health effects is almost non-existent. Warnings are buried deep with in the mobile phone menus or phone manuals that no one reads as is the case with long worded software agreements. Some companies send intermittent SMS messages to targeted users.

As radiation protection scientists we suggest the following: Given the strong evidence for harmful effects reported above, and the possible cost of doing nothing, we believe the trigger point for adopting a more Precautionary Approach to this new RF-EMR technology has been reached. We welcome debate and invite responses to this suggestion.

## 6. REFERENCES

1. Recuerda, M.A., Dangerous Interpretations of the Precautionary Principle and the Foundational Values of the European Union Food Law: Risk Versus Risk. *Journal of Food Law and Policy*, Vol. 4, No. 1, 2008, 2008.
2. Peel, J., Precaution – A Matter of Principle, Approach or Process? *Melbourne Journal of International Law*, 2004. **5**(2): p. 483.
3. Bromwich, D.W., The Precautionary Approach and its Application to Workplace Health and Safety (unpublished report). 2012: Canberra. p. 21.
4. SWA, How to Manage Work and Safety Risks. Code of Practice. 2011, Safe Work Australia: Canberra. p. 30.
5. Cameron, L., Environmental Risk Management in New Zealand – Is There Scope to Apply A More Generic Framework? 2006, New Zealand Treasury: Wellington.
6. Gee, D., Late Lessons from Early Warnings: Towards realism and precaution with EMF? *Pathophysiology*, 2009. **16**(2): p. 217-231.
7. Commonwealth Government, Radiocommunications Act – Section 162 The ACMA's power to make standards. 1992: Australia.
8. ICNIRP, ICNIRP Statement – General Approach to Protection Against Non-Ionizing Radiation Protection, *Health Physics*, 2002. **84**(4): p. 540-548.
9. Leach, V.A. and S. Weller, Radio Frequency Exposure Risk Assessment and Communication: Critique of ARPANSA TR-164 Report. Do We Have a Problem? *Radiation Protection in Australasia*, 2017. **34**(2): p. 9-18.
10. Barnes, F. and B. Greenbaum, Some Effects of Weak Magnetic Fields on Biological Systems: RF fields can change radical concentrations and cancer cell growth rates. *IEEE Power Electronics Magazine*, 2016. **3**(1).
11. Morgan, L.L., Estimating the risk of brain tumors from cellphone use: Published case-control studies. *Pathophysiology* 2009. **16**(2-3): p. 127-147.
12. The INTERPHONE Study Group, Interphone. Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. *International Journal of Epidemiology*, 2010. **30**(3): p. 687-894.
13. Coureau, G., *et al.*, Mobile phone use and brain tumours in the CERENAT case-control study. *Occup Environ Med* 2104. **71**: p. 514-522.
14. Toledano, M.B., *et al.*, An international prospective cohort study of mobile phone users and health (COSMOS): Factors affecting validity of self-reported mobile phone use. *International Journal of Hygiene and Environmental Health*, 2018. **221**(1): p. 1-8.
15. Hill, A.B., The Environment and Disease: Association or Causation? *Proceedings of the Royal Society of Medicine*, 1965. **68**(5): p. 295–300.
16. WHO, IARC Classifies Radiofrequency Electromagnetic Fields as Possibly Carcinogenic to Humans. Press Release. 2011, International Agency for Research on Cancer: Lyon, France.
17. Hardell, L. and M. Carlberg, Using the Hill viewpoints from 1965 for evaluating strengths of evidence of the risk for brain tumors associated with use of mobile and cordless phones1), in *Reviews on Environmental Health*. 2013. p. 97.
18. National Toxicology Program, TR-595: Toxicology and Carcinogenesis Studies in Hsd:Sprague Dawley SD Rats Exposed to Whole-Body Radio Frequency Radiation at a Frequency (900 MHz) and Modulations (GSM and CDMA) Used by Cell Phones. 2017, US Department of Health and Human Services.
19. Falcioni, L., *et al.*, Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8 GHz GSM base station environmental emission. *Environmental Research*, 2018.
20. Ostrom, Q.T., *et al.*, The epidemiology of glioma in adults: a “state of the science” review. *Neuro-*



- Oncology*, 2014. **16**(7): p. 896-913.
21. Kaszuba-Zwolińska, J., *et al.*, Electromagnetic field induced biological effects in humans. *Przegl Lek*, 2015. **72**(11): p. 636-41.
  22. Jauchem, J.R., Effects of low-level radio-frequency (3kHz to 300GHz) energy on human cardiovascular, reproductive, immune, and other systems: a review of the recent literature. *Int. J. Hyg. Environ. Health*, 2008. **211**(1-2): p. 1-29.
  23. Valko, M., *et al.*, Free radicals and antioxidants in normal physiological functions and human disease. *Int J Biochem Cell Biol*, 2007. **39**(1): p. 44-84.
  24. Loh, M., Exposure to Environmental Hazards and Effects on Chronic Disease, in *Environmental Determinants of Human Health. Molecular and Integrative Toxicology*, J. Pacyna and E. Pacyna, Editors. 2016, Springer: Cham. p. 27-49.

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