

**Evidence of brain cancer from
occupational exposure to pulsed
microwaves from a police radar.**

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Abstract

Since the first evidence that RF radiation damages chromosomes in 1959, many independent studies have identified broken DNA stands, chromosome aberrations and altered gene expression in animal cells, human cells and in living animals and humans from EMR exposure. This confirms that RF/MW radiation is genotoxic with a safe exposure level of zero. Scores of epidemiological studies show that EMR increases brain tumors, including 16 studies with dose-response relationships and at least six specifically identifying astrocytomas. Exposure to RF/MW is consistently associated with headaches, fatigue, loss of concentration and memory loss. These symptoms have been called "The Radiofrequency Sickness Syndrome" or "Microwave Syndrome". These symptoms are now shown with cell phone use in a significant dose-response manner. Cellphone use has also been associated with increases brain tumor in 4 studies and eye cancer in one study. Police traffic radar is also shown to be genotoxic through studies associating it with increases testicular cancer. This is a consistent and coherent set of studies confirming that microwaves, radar and police radar is genotoxic and when exposing an officer's head over many months, will produce a significantly increased risk of producing an Astrocytoma brain tumor. All of these effects occur for exposures well within existing standards. The standards are based on tissue heating and ignore the evidence of genotoxicity, cancer and neurological effects.

1. Introduction:

1.1 A traffic radar caused this officers brain tumor:

A 32 year old police officer has been chronically exposed over years to a pulsed microwave signal from a Doppler radar used for traffic speed detection. The officer has developed an astrocytoma tumor in his left frontal lobe on the side of his head exposed by the radar. Documents obtained claim that the maximum exposures comply with safety standards, Kustrom Signals Inc. This is accepted. The United States occupational standard for microwave exposure is 10 mW/cm². The Kustrom report also states that the maximum exposure of the officer's seat was 0.021 mW/cm², 250 times lower than the safety standard, p56. The Battelle report for a Kustrom KR 10 SP radar reports an average leakage of 0.139 mW/cm², in the range 0.055 to 0.400 mW/cm².

Dr Virginia Weaver is of the opinion that this situation could not have caused this officer's brain tumor. It is my expert scientific opinion that the scientific evidence soundly and comprehensively challenges this opinion with extensive evidence of causal microwave genotoxicity and strong epidemiological evidence of EMR induced brain tumors.

Since brain tumor is quite rare at this young age, microwaves and radar are causally related to chromosome aberration and DNA-strand breakage, and consistently cause brain tumors, including Astrocytoma, it is my expert opinion that the chronic exposure to the traffic radar to his head has caused his brain cancer.

For example, digital mobile phone radiation uses a signal that is similar to radar, i.e. a pulsed microwave signal. Mobile phone radiation severely breaks DNA strands, $p < 0.0001$, at an exposure level of 0.0024 W/kg (0.0012 mW/cm²), Phillips et al. (1998). Compared to the US occupational safety standard of 4 W/kg (10 mW/cm²), this is 1666 times lower and 8.8 times higher than the lowest estimate of the radar head exposure. For this and many other reasons, the meeting the US standard is totally irreverent to this case.

The US safety standard is based on avoiding tissue heat. The extremely significant ($p < 0.0001$) DNA strand breakage is shown to occur over 2000 times lower than microwaves produce measurable heat. Since this is one of over 20 studies showing that RF/Microwaves damage DNA, and at least 4 show dose-response relationships, microwaves are causally genotoxic. This means that RF/MW is carcinogenic. This is confirmed by many studies particularly showing that exposed people get leukaemia and brain tumors. A substance that is genotoxic damages the DNA in cell-by-cell. Therefore a genotoxic substance, such as microwaves, has no safe threshold, UKRCEP (1998). Any exposure above zero exposed cells with quantum microwaves damaging cell-by-cell. Despite the body's highly efficient DNA-strand repair mechanisms, mistakes are made and cancer develops, usually over a few years to decades. Continuing day-by-day exposure to a genotoxic radiation signal accelerated the cancer promotion and progression to a tumor. This happened to Officer Wayne Dixon and produced his astrocytoma brain tumor.

To confirm this I will summarize the studies showing that radar exposure causes cancer. I will present strong and consistent evidence that RF/MW radiation causes DNA damage and hence is causing cancer through being genotoxic. That electromagnetic radiation across the spectrum confirms that EMR causes brain tumors and microwaves cause astrocytomas. I will also give evidence that shows why brain tumors occur because brains are sensitive to EMR across the spectrum and causes many neurological effects, diseases and mortality.

1.2 Radar is carcinogenic at very low to high exposure:

Radar exposure is significantly linked to cancer in military personnel and in residents of cities close to US Air Force bases. Lester and Moore tested the hypothesis that microwaves from radar caused cancer at the level of exposure experienced in residential situations. They confirmed that it radar does significantly elevate cancer in two studies covered by three papers, Lester and Moore (1982a,b) and Lester (1985). The 1982 study used the fact that Wichita, Kansas had an airport radar to the east and to the west of the city. They reasoned that the undulating hill pattern produced people living on the ridges residentially exposed to two radars, people living on east or west facing slopes exposed to one radar and people in the valleys exposed to no radar. In this order the cancer incidence rates were 470, 423 and 303 per 100,000 person-yrs. The rate ratios of 1.55, 1.42 and 1.0 respectively, Lester and Moore (1982). This dose-response strongly supported their hypothesis.

They then predicted that counties with Air Force Bases (AFBs), because they all had radar sets operating almost continuously, should have higher cancer rates than those counties without AFBs. They found that the counties with AFBs had significantly higher cancer

rates, Lester and Moore (1982b). Polson and Merritt (1985) criticized this study. For example, they pointed out quite reasonably that some AFBs were close to a city that was in another county. Lester (1985) compared the cancer rates of cities in proximity to AFBs with radars and found that this significantly strengthened their result. He thus concluded that they confirmed their observation that cities near AFBs (and radars) had significantly higher cancer rates in the period 1950-69, Lester (1985).

Two large military studies confirm that radar and radio radiofrequency/microwave (RF/MW) exposures significantly increase overall cancer and death rates. Robinette et al. (1980) studied the US Navy technical staff who had been exposed to radar and radio while they served on ships in the Korean War. Health and mortality records from about 20 years were used. Over 40,000 naval personnel were studied. Occupational groups were rank according to their work in relation to RF/MW exposures. A 5 % sample from the three "high exposure" groups, were studied in detail to carry out a job matrix exposure assessment. When this selected sample had their health records matched to their hazard index, a significant dose-response for all mortality resulted, trend $p=0.03$. This exposure survey revealed a strong exposure gradient between the three "high exposure" groups, Electronics Technician (ET), Fire Control Technician (FT) and Aviation Electronics Technician (AT). Their mean radar hazard indices were 1610, 2870 and 3700 respectively. Hence a good exposure dichotomy can be obtained by comparing the high exposure AT group with the lower exposure ET group, using the data from Table 5 in Robinette et al. (1980).

From Table 5 the high exposure AT group has 3273 members, 198 of who had died within the defined period, 1950-1974. The low exposure ET group had 13078 members of whom 441 had died in the same period. This gives:

$$RR = 1.79, 95\% CI: 1.52-2.11, \text{Chi Squared} = 49.97, p < 0.0000001$$

For all cancer mortality:

$$RR = 1.66, 95\%CI: 1.06-2.60, \text{Chi Squared} = 5.03, p = 0.025$$

These are extremely and highly significant mortality effects from radar exposure. The sample was too small to detect significant increases in other individual cancers, except lymphoma/leukaemia. It does confirm that radar exposure significantly increases mortality and malignant tumors. When low, intermediate and high exposure groups were combined, dose-response increases of mortality occurred, Figure 1.

Dose-response mortality for all death, all diseases, all cancer and lymphoma/leukaemia are shown in figure 1. The smallest group, Aviation Electrician's Mate (AE), 1412 members, has been omitted. This was because of the probable misclassification of this group as a "low" exposure group when low exposure is based on groups who operate the radars and radios when the AE group is obviously a repairer group and therefore is a "high exposure" group. However, its exposure hazard index was not assessed in the 5 % survey and therefore it cannot be ranked like the other three high exposure groups.

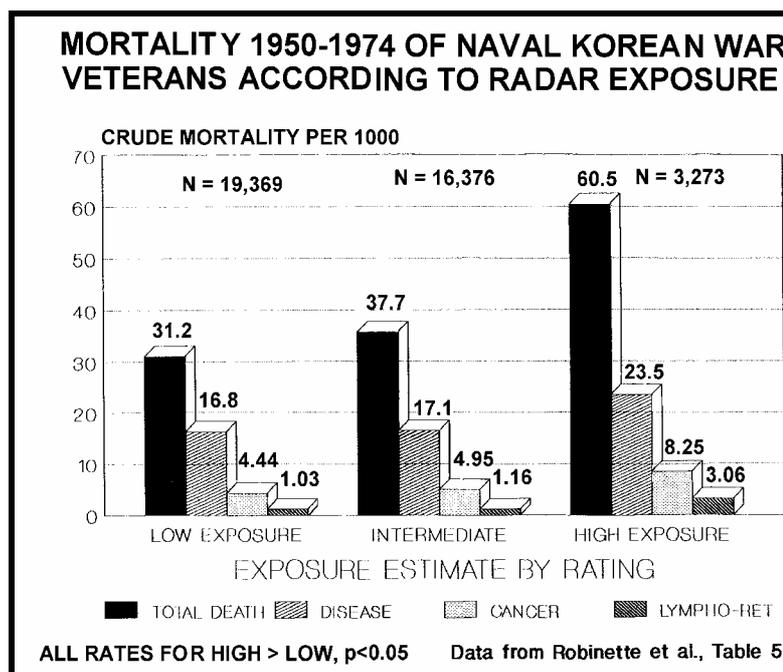


Figure 1: Naval occupations grouped by exposure category, showing dose response increases in mortality for all mortality, all disease, cancer and Lymphatic/Leukaemia. Low exposure (RM+RD), Intermediate exposure (ET+FT), High exposure (AT).

The Polish Military Study, Szmigielski (1996), involves a larger sample and has a clear and distinct process to add people to the exposed group through reported and recorded RF/MW exposures. The remaining "unexposed" Polish military personnel form the control group involving 1.5 million p-yrs. The exposed group includes over 55,000 p-yrs.

This study found extremely significant increases in Leukaemia/Lymphoma, RR = 6.31, 95%CI: 3.12-14.21, p<0.001, with the highest rate ratio of RR = 13.9, 95%CI: 6.72-22.12, p<0.00001, for Chronic Myelocytic Leukaemia. The sample size was large enough to have a reasonable individual cancer site breakdown. For Brain and CNS tumors, RR = 1.90, 95%CI: 1.08-3.47, p<0.05.

While these military personnel had occasional high exposures. Between 80 and 85% of them had intermittent exposures of less than 0.2mW/cm². Their hourly mean exposures were likely to be less than 10 % of this and daily mean exposures are less than a third of the hourly mean, i.e. less than 0.0067mW/cm². This is likely to be somewhat less than the exposure of a police officer who has worked day after day for several years, with a radar less than 3 feet from his head and pointed forward past his head.

In 1977 Dr Milton Zaret reported a cluster of 2 astrocytomas in a group of 18 workers who were servicing microwave communication equipment, Zaret (1977). Compared to the rate of astrocytomas in male workers in the 35-50 year old age range, this is extremely significant. RR = 74.1 (15.0-367), p<0.0001, n=2.

In 1987 the National Cancer Institute published a study of microwave exposed workers in electrical and electronic industries on the east coast of the United States, Thomas et al. (1987). They found that workers who were jointly exposed to solder fumes and RF/microwave radiation had a dose response increase in Astrocytoma with years of exposed work:

Overall incidence of Astrocytic brain tumours: RR=4.9 (1.9-13.2)

Dose-response trend with years of employment.

		Duration Employed (yr)			
		Unexposed	<5	5-19	≥ 20
	RR	1.0	3.3	7.6	10.4
Solder fume adjusted by a factor of 2	RR	1.0	1.65	3.8	5.2
		(trend p<0.05)			

This police officer was on traffic duty for hundreds of days in traffic. Hence he was continually exposed to toxic traffic fumes such as benzene. When he is also chronically exposed to a pulsed microwave radar signal released near to his head height, then his risk is the same as shown here to produce an astrocytoma brain tumor.

These cited studies show that RF/MW exposure of people does significantly increase the risk of brain cancer, including specific risk of developing an astrocytoma.

Over 75 studies have reported increases in brain cancer over 157 human groups exposed to electromagnetic radiation across the EMR spectrum. Over 50 of the studies show statistically significant increases in brain and CNS cancer in 66 groups. To date 16 studies have been published showing dose-response increases. The vast majority of these studies involve mean exposures that are over 1000 times lower than the 10mW/cm² standard and well below the exposure range of the police officer exposed to the traffic radar.

This evidence taken together gives overwhelming evidence of a causal association between chronic low level exposure to RF/MW radiation, including that produced by police traffic radar, and brain tumors, including Astrocytomas.

2. Methods:

In this report the sequence of evidence will be as follows:

- The scientific principles used in this report will be laid out.
- The evidence that radiofrequency/microwave (RF/MW) electromagnetic radiation (EMR) is genotoxic will be presented.
- Evidence that the protective effect of melatonin is reduced by EMR exposure is presented.
- Epidemiological evidence of EMR causing cancer in is outlined and summarised. It is contended that the brain is a very electromagnetically sensitive organ that is vulnerable to electrical interference and cellular damage from electromagnetic radiation. Evidence that EMR exposure of the brain causes neurological effects is consistent with and supportive of the vulnerability of the brain to brain tumors. The large body of epidemiologic studies on Brain tumors will be especially considered. This evidence is consistent with and confirms that EMR is genotoxic and carcinogenic.

- Consideration of police radar exposures will also be considered and they confirm, as expected since microwaves and radar are genotoxic and carcinogenic, that they have been observed to cause cancer (testicular) in police officers using traffic radars.
- The evidence is then summarized and conclusions are drawn.

3. Scientific Principles:

3.1 Biostatistical principles:

3.1.1 Significance:

Statistical tests are used to test the null hypothesis. The null hypothesis is that any observed effects are entirely due to sampling errors (i.e. due to chance). The tests are set up to evaluate whether the null hypothesis is true or is reliably rejected. A threshold of $p=0.05$ is typically chosen for acceptance or rejection of the null hypothesis.

For $p=0.05$, there is only a 5% chance that it is random and 95% that it is not random. If $p=0.05$ or less, then null hypothesis is rejected because the result is not caused by chance. The smaller the p-value is the stronger is this conclusion.

Hence $p \leq 0.05$ shows the result is significant so it could be causal.

$p \leq 0.01$ shows the result is highly significant it is probably causal.

$p \leq 0.001$ shows the result is extremely significant and it supports a causal relationship.

3.1.2 One- and two-tail t-test:

In the analysis of variance the common test of significance uses the t-test, using the Student t distribution. A study is looking for a single-direction effect such as is this exposure increasing the risk of brain cancer? In this case a single-tail test is appropriate. If the study is open to the possibility that the exposure could increase or decrease the incidence of a disease then it is appropriate to test a two-directional effect using a two-tail test. A two-tail test has higher thresholds for t-values than the one-tail test.

For example, for 5 degrees of freedom the t-value of a two-tail test for $p=0.05$ is 2.571, for a one-tail test the t-value for $p=0.05$ is 2.015. If the t-value was 2.571 for a one-direction effect then $p=0.025$, half the value and twice as significant compared with the two-tail assessment.

For determining the significance of fitted trends in dose-response relationships the data falls on both sides of the fitted line and a two-tail test is appropriate.

Some authors favour a position that a non-significant elevation shows "no effects". This is not true since, as Hill (1965) shows, a consistent elevation of respiratory disease, never achieving statistical significance because of the small sample size, was concluded to be causal for cardroom workers in a cotton mill exposed to dust. To maintain this "no effect" position some researchers choose to apply a two-tailed test to a one-direction effect to

half the level of significance (double the p-value). For example with the data given above for 5 degrees of freedom, a t-value of 2.5 would be significant from a one-tail test and not for a two-tail test.

3.1.3 Danish Cellphone study:

For example, Johansen et al. (2001) investigated the Danish population using cellphones with an *a priori* assumption that cellphones could produce cancer, especially brain tumor. They used a two-tail t-test to evaluate a one-direction effect. Throughout the paper not a single significant increase of cancer is shown by using the two-tail test. Some would have been significant if a one-tail test was used.

The level of individual cellphone usage was not investigated. Period of ownership was used. For over 67 % of the cellphone users this was 2 years or less. This does not give sufficient time for cancer to develop for most of the study group.

The effects would have been even higher and more significant if a more appropriate reference group was used. They also used more general population cancer rates to calculate Specific Incidence Rates (SIR). In doing so the brain tumor rate of people who had cell phones for less than 1 year was 70 % of the reference rate, SIR =0.7. Some of the SIRs for particular cancers are <0.5. This indicates that the chosen reference group had much higher cancer rates than the cellphone group. An appropriate reference group would have less than half of cancer rates than the group chosen as a reference group. This was confirmed by Danish neurologist, Dr Albert Gedde, who criticized the paper saying that the observed brain tumor rate was significantly higher than the average Danish rate.

For example, the given data for female cancer had 515 cases with 497.6 expected, giving SIR = 1.03, 95%CI: 0.95-1.13. If the reference group had half the expected cancer rate to bring it closer to the age distribution of the cell phone users then the expected cases would be 249, and SIR = 2.02, 95%CI: 1.74-2.34. $p < 0.0000001$.

Despite the fact that the group consisted of people who had used phones for less than 3 years, the elevated rates of a number of cancers is quite serious. The authors also state that no dose-responses in brain tumors were observed. That is incorrect. As a function of the duration of using a GSM phone the brain tumor rate was SIR =0.7 for <1 yr, SIR = 0.9 for 1-2 years and 1.2 for ≥ 3 yrs. If the <1 year group is used as an internal reference group then the SIRs would be 1.0, 1.29 and 1.71, respectively. If the reference group had half the assumed cancer rate then the SIRs would be 1.4, 1.8 and 2.4 respectively.

The direct relevance of the Danish study to this case is the results of exposing a large group of people to a pulsed microwave signal primarily in the region of the head. The women have elevated cancers of the Pharynx (SIR = 2.43, 95%CI: 0.65-6.72) and Esophagus (SIR = 1.53, 95%CI: 0.31 - 4.46). Cellphones also expose other body parts, especially when carried on the belt. Women have elevated cancer of the cervix (SIR = 1.34, 95%CI: 0.95-1.85) and men of the testis (SIR = 1.12, 95%CI: 0.97-1.30), and together they experienced a dose-response increase in brain cancer.

3.1.4 Statistical errors in medical studies:

Gantz (1997) reviewed the studies of assessments of statistical methods used in medical studies. Between 40 % and 70 % contained statistical errors, particularly in their use of t-test and Chi Squared interpretation, Gore, Jones and Rytter (1977).

3.1.5 Comparative 2x2 Analysis:

The standard table of the 2x2 analysis compares the exposed and non-exposed groups who do and do not have the disease.

	Exposed	Unexposed
Disease	a	b
No Disease	c	d

Here a is the number of people who were exposed and do have the disease while b are those who were not exposed and do have the disease, etc.. The Odds Ratio (OR) is the ratio of odds of being exposed and having disease to those of being exposed who have no disease: $OR = (a/c)/(b/d) = ad/bc$

The Relative Risk or Rate Ratio (RR) is the ratio of occurrence of disease in exposed to non-exposed populations: $RR = [(a/(a+b))] / [(c/(c+d))]$.

An indication of significance is given by considering the 95% confidence interval:

$$95\% \text{ Confidence Interval} = \exp (\log_e (OR) \pm 1.96 \sqrt{[1/a + 1/b + 1/c + 1/d]})$$

$$95\% \text{ Confidence Interval} = \exp (\log_e (RR) \pm 1.96 \sqrt{[1/x_o + 1/x_e]})$$

where x_o is the non-exposed number of people ($b + d$) and x_e is the exposed number of people ($a + c$).

If the lower limit of the 95% confidence interval is ≥ 1.0 then it is significant.

The Chi Squared statistic (χ^2) is given by:

$$\chi^2 = (ad-bc)^2 N/[(a+c)(a+b)(b+d)(c+d)]$$

The probability is obtained from a table of χ^2 values and p-values in texts. For a 2x2 table the number of degrees of freedom is 1. The thresholds for significance of χ^2 are 3.84 for $p=0.05$, 5.02 for $p=0.025$, 6.63 for $p=0.01$ and 7.88 for $p=0.005$.

This shows that the width of the confidence interval is dependent on the size of the Odds or Rate ratios and closely dependent on the size of the study samples. Small samples produce wide widths and large sample a narrow width.

For example, a study samples two groups of 1000. One is exposed and one is non-exposed, i.e. treated in the same way in all respects except the exposure. The biological effect is measured in 20 of the exposed samples and 10 of the unexposed (sham exposed) sample. The statistical analysis using the Woolf method from Silman (1995) is

$$n=20 \quad OR = 2.02 \quad RR = 2.0 \quad 95\%CI: 0.94 - 4.25 \quad \chi^2 = 3.38 \quad p = 0.066$$

For a situation where there are $n = 20$ cases, the rate of the effect is doubled and the result is not statistically significant, $p > 0.05$. The statistical package used the Mantel-Haenszel Chi Squared estimate.

Halving and doubling the sample size yields:

$n = 10$ OR = 2.01 RR = 2.0 95%CI: 0.69 - 5.83 $\chi^2 = 1.68$ $p = 0.195$

$n = 40$ OR = 2.04 RR = 2.0 95%CI: 1.18 - 3.40 $\chi^2 = 6.87$ $p = 0.0088$

The actual effect is the same in every case doubling the Relative Risk. The exposure doubles the effect. For $n=20$ the sample is just below the selected level for statistical significance. By halving the sample size the p-value is highly non-significant and by doubling the sample size the p-value is highly significant.

Sir Austin Bradford Hill, Hill (1965), sets out the guidance for assessing the causal effect. He asks the question ***"is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?"*** Sir Austin is very aware of the problem of statistics and sample size. He states: ***"No test of significance can answer these questions. Such tests, can and should, remind us of the effects that the play of chance can create, and they will instruct us in the likely magnitude of those effects. Beyond that they contribute nothing to the 'proof' of our hypothesis."***

3.2 Epidemiological Principles:

3.2.1 Setting Standards:

Setting standards for protection of public and occupational health are based on using studies involving human exposures to disease agents, epidemiological studies. In the absence of epidemiological studies, animal and cell-lines studies can be used to determine whether an agent is genotoxic. However, human studies are much more powerful when considering public health protection standards.

Eminent Epidemiologist, Professor Abraham Lilienfeld states, Lilienfeld (1983),

"The proper study of man is man."

This is paraphrased as:

"Public Health Standards should be based on Public Health Studies"

In consideration of the question: ***"is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect"***, Sir Austin Bradford Hill set out very helpful guidance, Hill (1965). This is primarily based on epidemiological studies. For example, the exposure must precede the effect. It is helpful if there is consistency and specific effects. However, these are not necessary because some agents cause several effects and some effects are caused by several agents.

Current Western EMR standards are based on the biological effect of tissue heating and ignoring or inappropriately dismissing epidemiological evidence. In Sir Austin's assessment, biological mechanisms play a very minor role. Of "Biological Plausibility" he

states: ***"It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day"***. This statement ranks biological plausibility as the weakest evidence in considering causality.

Strength of Association can be helpful in assessing a causal effect. However, just because an effect is small doesn't exclude it from being causal. However, highly significantly strong relationships, without feasible confounders, point strongly to a causal effect. The strongest single assessment indicating a causal effect is the biological gradient or dose-response increase in effect with increased exposures.

3.2.2 Dose-response trends:

Dose-response relationships are the strongest epidemiological evidence, Hill (1965). In his paper Sir Austin states: ***"the clear dose-response curve admits of a simple explanation and obviously puts the case in a clearer light."***

A WHO document, Beaglehole, Bonita and Kjellstrom (1993) states: ***"The demonstration of a clear dose-response relationship in unbiased studies provides strong evidence for a causal relationship between exposure or dose and disease"***.

This is generally interpreted as accepting a dose-response relationship as a causal effect of a disease agent, Beale et al. (1997). The dose-response is also valuable in determining acceptable exposure levels for public and occupational protection standards.

3.2.2 Healthy worker effect:

The healthy worker effect is a well established feature of occupational epidemiological studies. Workers usually exhibit lower overall sickness and death rates than the general population. The effect is much stronger in uniformed services because of the physical requirements of the jobs and the selection processes. Fire and Police Officers are in this category. Hence occupational exposures, such as to radar, must be related to carefully chosen reference groups or internal cohorts.

3.3 Genotoxicity:

The protection or repair of DNA molecules is of paramount importance and damaged cells need to be repaired "at all costs", Elliott and Elliott (1997). A substance is defined as "genotoxic" if it damages DNA, thereby causing mutations or cancer, Dorland's (1994). DNA is folded into chromosomes that contain thousands of genes. Hence any evidence that a substance causes chromosome aberrations, micronuclei formation, DNA strand breaks or altered genetic activity (especially proto-oncogenes) is direct evidence of genotoxicity.

Because chromosomes are visible in microscopes, chromosome aberrations are visible evidence of genotoxicity. In the past decade assay methods to detect single and double DNA strand breakage have been developed. Because DNA damage is common and repair is paramount, biological systems have highly developed DNA repair mechanisms. An earlier assay searched for evidence of chemical changes associated with DNA repair as an indication of DNA damage, Meltz (1995). Hence induced DNA repair is evidence of DNA damage and the over expression of the DNA repair processes.

When the cellular processes fail to repair damaged DNA, cell-to-cell communication systems may detect a damaged cell. A damaged cell, through its own processes, or through cell-to-cell communication processes, can initiate programmed cell death, Apoptosis. One cell-to-cell communication process involves oscillating calcium ion currents flowing through gap-junctions. Electromagnetic fields close gap junctions in a dose-response manner, forming another path towards cancer, Li et al. (1999). When the cellular calcium ion concentrations are elevated they can change the cellular response from cell death to cell proliferation, causing the propagation and survival of mutated cells. Chemical cancer promoters, such as phorbol esters (e.g. TPA), act by elevating cellular calcium ion levels, Balcer-Kubiczek (1995).

Two backup cell protection systems involve the immune system. The immune system has the ability to detect a "foreign cell" and Natural Killer (NK) cells eliminate them. Alternatively, enhanced cell damage can stimulate the production of "toxic shock" proteins, such as heat shock proteins. Some carcinogens act through impairing the immune system competence or inhibiting the activity of toxic shock proteins. Hence carcinogens are agents that can cause genetic damage that enhances the production of mutated cells, can impair the cellular repair or apoptosis by changing the cellular calcium ions, for example, or can impair the immune system or any of the repair processes.

For any given level of exposure to a genotoxic and/or carcinogenic agent, only a proportion of exposed individuals will develop cancer, Spitz and Bondy (1993). One contributing factor is variation in the activity of metabolizing enzymes responsible for conversion of procarcinogens to proximate carcinogens. There is also a wide spectrum of DNA repair capability within the general population. Hence not all police officers exposed to radar radiation will necessarily develop a brain tumor. But since radar signals are genotoxic then they do cause some officer(s) to get a brain tumor.

3.4 Bioelectromagnetic exposure principles:

Extremely low frequency (ELF) fields that are used in power supplies, e.g. 50/60 Hz fields, induce electric and magnetic fields within exposed people. The electric field gradient induces currents that flow down the gradient to earth. The magnetic field component induces currents circulating around the magnetic fields lines. Both of these electromagnetic components induce ELF oscillations from the primary frequency, as well as harmonics and sub-harmonics.

Natural systems such as lightning, and invented systems such as radio, TV, radar and cellular telephones, produce radiant fields that travel at the speed of light. They tend to carry information in the form of modulations of frequency or amplitude that is encoded in the carrier signal. The radiation speed (c) is the product of the frequency (f) and the wavelength (λ) $\{c = f \lambda\}$. In a given medium c is nearly constant and therefore the frequency and wavelength are inversely proportional. High frequencies have short wavelengths and long wavelengths have low frequencies.

When the carrier wavelength is much longer than the body dimension then it passes easily through the body with little absorption, unless the modulation frequency is close to a natural oscillating frequency. In the UHF (0.3 to 3 GHz) and SHF (3 GHz to 30 GHz) microwave range the wavelengths are 1 m to 0.1 m and 0.1 to 0.01 m, respectively.

Radiofrequency/microwave signals interact with whole bodies like an aerial. Aerials have strong resonant absorption characteristics when their length is a proportion of the

wavelength. Common aeriels are called half-wave and quarter-wave dipoles because their length is equal to one half or one quarter of the wavelength of the signal they are tuned in to. Thus low frequency signals, with very long wavelengths, pass through bodies with little absorption. As the wavelength approaches the body dimension, broad peak resonant absorption takes place, Figure 2.

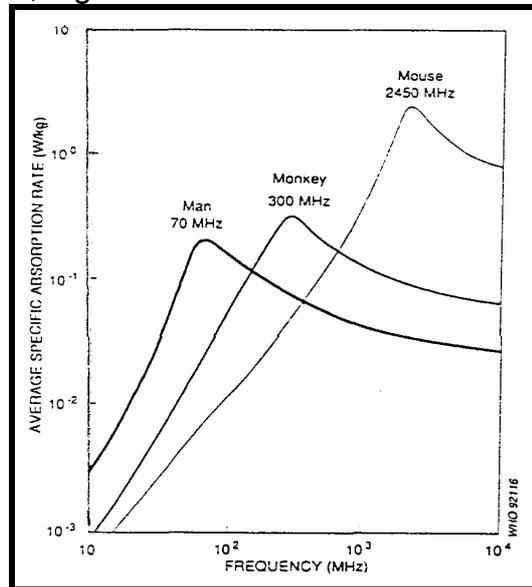


Figure 2: Average SAR for 3 species exposed to 10 W/m^2 with E parallel to the long axis of the body, WHO (1993).

Figure 2 shows a broad absorption peak for "man" at 70 MHz, Monkey at 300 MHz and mouse at 2450 MHz. The amount of energy absorbed is expressed as the Specific Absorption Rate (SAR) in W/kg. Above these frequencies resonant absorption occurs in body parts, creating what is known as "Hot Spots", WHO (1993). Individual body parts have resonant absorption peaks relative to their dimensions. The optimal absorption frequency for adult heads is close to 400 MHz, Spiegel (1984).

In the microwave range resonant absorption occurs at the cell membrane and signals are absorbed quite strongly and penetrate less and less as the frequency rises and the wavelength gets smaller and smaller.

This means that in the microwave range absorption rates in tissue are quite strong. Cellphone and radar signals in the GHz range penetrate the brain in a number of centimeters (cm), Figure 3. Penetration depths are reduced as the absorption rate increases as a function of the frequency as indicated by the dielectric constant in Figure 4.

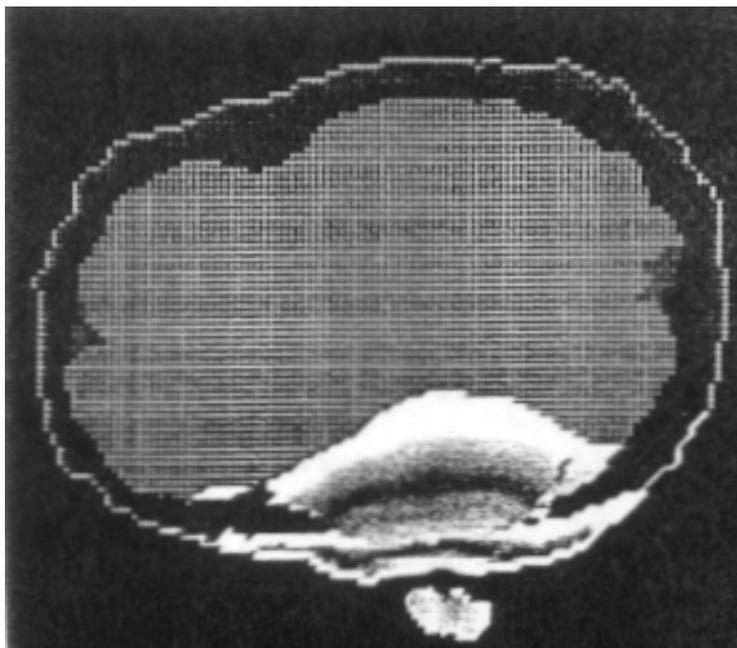


Figure 3: SAR distribution for a model of an adult male with exposure to a cellphone, frequency = 835 MHz and radiated power = 600 mW.

The edge of the inner white band in Figure 3 is 0.11 W/kg. The level of DNA breakage shown by Phillips et al. (1998) was extremely significant ($p < 0.0001$) at 0.0024 W/kg, 45.8 times lower than this. Such an exposure level would be exceeded in more than half of the brain. With the extremely significant effect it is most likely to damage DNA over the whole brain. This would kick-in the DNA repair processes but leave some mutations and enhance apoptosis.

The basic equations relations to SAR and the exposure intensity (S , $\mu\text{W}/\text{cm}^2$) for about 1 GHz are:

$$\text{SAR} = \sigma E^2 / 2\rho, \text{ and } S = E^2 / 3.77 \mu\text{W}/\text{cm}^2 \therefore S = 504 \text{ SAR}$$

Where E is in V/m, $\sigma = 1 \text{ S/m}$ and the density $\rho = 950 \text{ kg}/\text{m}^3$.

Hence SARs of 0.0024 and 0.026 W/kg are 1.2 and 13.1 $\mu\text{W}/\text{cm}^2$ (0.0012 and 0.0131 mW/cm^2).

3.5 EMR Spectrum Principle:

It is observed that both biological effects and epidemiological effects are the same or very similar from ELF exposure and from RF/MW exposures, including calcium ion efflux, melatonin reduction, DNA strand breakage, chromosome aberrations, leukaemia, brain cancer, breast cancer, miscarriage and neurological effects. The biophysical principle behind this is shown by the way in which the dielectric constant, the alternating current equivalent of resistance, varies in tissue with the frequency of the signal, Figure 4.

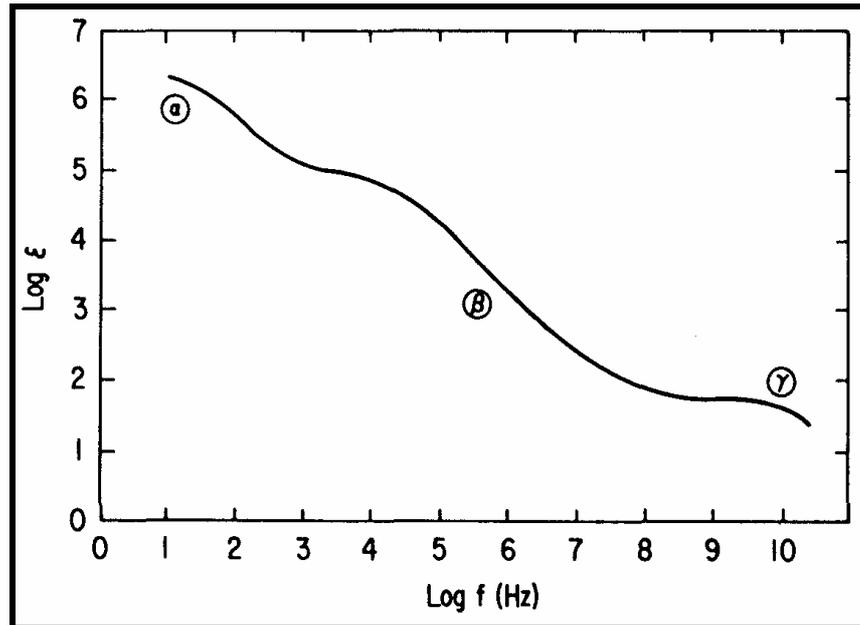


Figure 4: The dielectric constant of muscular tissue as a function of frequency decreases in 3 major phases from about 3×10^6 to about 30 as the frequency increases from 10 Hz to 20 GHz, Schwan (1985).

With the decrease in the dielectric constant with increasing frequency, the induction of electric fields and induced currents for a constant external field strength, should increase with increasing frequency. This was confirmed by Vignati and Giuliani (1997). They show that a unit field exposure induced the current increases significantly as a function of frequency, Figure 5.

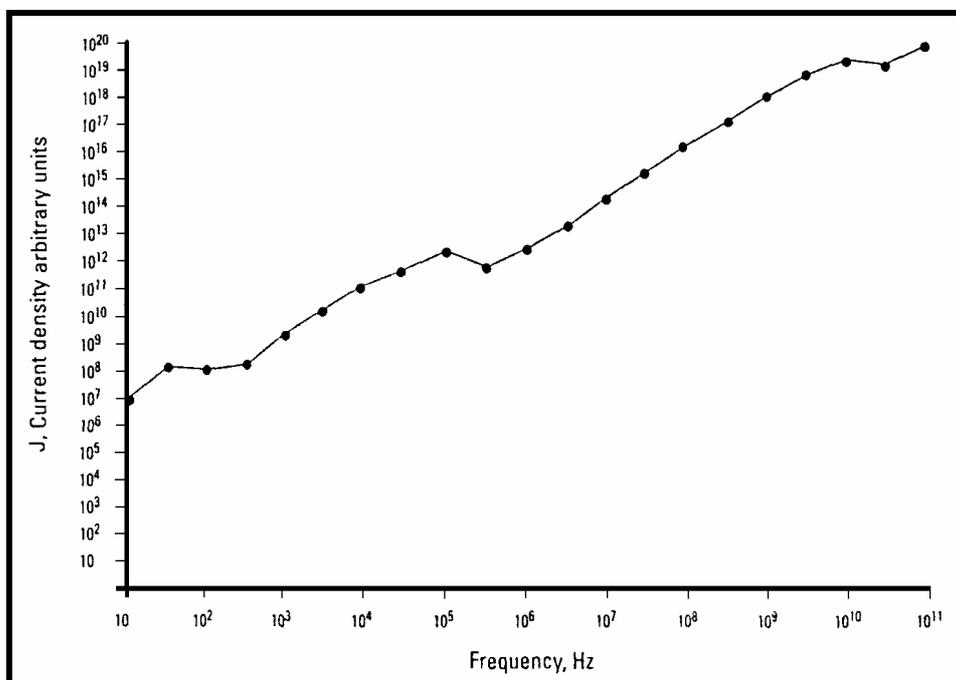


Figure 5: Capacitive current density in a toroid of human muscle tissue of unitary radius, to a unitary magnetic induction, Vignati and Giuliani (1997).

Bawin and Adey (1976) show that a 56 V/m ELF field induces a tissue gradient of 10^{-7} V/cm, whereas a 56 V/m 147 MHz signal induces a tissue gradient of 10^{-1} V/cm, a million times higher. This is a very large factor but it is slightly smaller than that given by Figure 5.

The KR-10SP radar operates at 24.15 GHz. Compared to a 1 GHz cellphone signal the 24.15 GHz signal induces about 10 times higher current, Figure 4. The cellphone radiation at 813-837 MHz highly significantly ($p < 0.0001$) damages DNA at very low exposure levels (SAR = 0.0024 W/kg), Phillips et al. (1998). Hence the police traffic radar microwave radiation will strongly damage DNA much higher rates but penetrate the brain less.

This biophysics phenomenon shows that it is inappropriate to separate considerations of effects of EMR into small frequency and intensity bands. The EMR interaction in tissues is primarily at the cell membrane, Adey (1988, 1990a). At the membrane the EMR interaction is resonantly non-linearly absorbed. This creates exposure "windows", Blackman et al. (1988, 1989, 1991, 1992). The "windows" involve the field intensity, carrier and modulation frequencies, ambient temperature and the local earth's magnetic field. Exposure window responses have been documented for EMR induced calcium ion efflux and EMR induced oncogene activity. This explains why some laboratory replication experiments show different results, probably because the local geomagnetic field or temperature is different.

People are constantly moving through changing exposures, temperature and geomagnetic field changes. By passing through exposure windows of effect and no effect, the average is biological and health effects. On average the higher the carrier frequency the higher the biophysical impact. The smoothing effect produces a near continuous variation across the spectrum.

The EMR Spectrum Principle predicts that effects that are associated with ELF exposure are reasonably expected to be found, but with much lower intensities, from RF/MW exposure. This is demonstrated for calcium ion efflux, Bawin and Adey (1976). The studies of genotoxicity and epidemiological studies of brain tumours show that they occur across the spectrum over a wide range of frequencies. The EMR Spectrum Principle counters the approach that tries to dismiss evidence by dividing exposures into small specific regions of exposure, frequency and modulation. The effects are similar across the spectrum and integration of results is much more scientifically appropriate.

3.6 Our brains are exquisitely sensitive:

Our brains are exquisitely sensitive bioelectrochemical organs that are the seat of human creativity, memory, emotions and intelligence. We use electrical signals, including charged ions, to think, remember and see, to regulate the beating of our heart, and for communication in our central nervous system and between and within our cells. Human brains were proven to be very sensitive to and reactive to extremely small, naturally occurring Schumann Resonances in the 1950's and 1960's, König (1974) and Wever (1974). The Schumann Resonance signal is produced by tropical thunderstorms, propagates around the world in the resonant cavity formed between the ionosphere and the earth, and has a mean intensity of around 0.1 pW/cm^2 , $0.000000001 \text{ mW/cm}^2$. That our brains detect and respond to the Schumann Resonance signal it is biophysically reasonable because it shares the same frequency spectrum range as the human EEG.

Brain functions are monitored using the electroencephalogram (EEG). At the individual neuron level the oscillating electrical currents are dominated by calcium ions within the neurons. The calcium ions regulate the release of neurotransmitters that communicate between neurons. Electromagnetic signals whose frequencies, their harmonics and sub-harmonics, occur within the natural frequency range of the brain cells, the EEG frequency

range from 0 to 50 Hz, primarily from 2 to 40 Hz, resonantly interact with neurons at extremely low intensities.

Biological Systems detect and respond to external ULF/ELF signals using their built-in receiving and decoding systems (cell-to-cell communication, EEG, ECG). Table 1 summarizes observations of actual field levels involved in biological processes, Adey 1990.

Early claims that living cells could not detect fields less than the membrane potential, 10^5 V/cm, are demonstrably wrong. Not only does the brain's EEG signals operate using ELF oscillating signals whose field strength is a million times lower, 10^{-1} V/cm, but fish, birds, primates and humans detect and respond to ELF oscillating signals over a million times smaller than the EEG signal, 10^{-7} to 10^{-8} V/cm.

Table 1: Bioelectric sensitivities to ELF fields, Adey (1990).

	Function	Tissue Gradient	Imposed field
Sharks and rays	Navigation and predation	10^{-8} V/cm	DC to 10 Hz
Birds	Navigation	10^{-7} V/cm	0.3 gauss
Monkeys	Circadian rhythms	10^{-7} V/cm	10 Hz, 2.5 V/m
	Subjective time estimations	10^{-7} V/cm	7 Hz, 10 V/m
Humans	Circadian rhythms	10^{-7} V/cm	10 Hz, 2.5 V/m
Comparison With Intrinsic Cell and Tissue Neuroelectric Gradients			
Membrane potential	10^5 V/cm		
Synaptic potential	10^3 V/cm		
Electroencephalogram	10^{-1} V/cm		

These processes involve non-linear, non-equilibrium reactions to resonant absorption of oscillating signals, Adey (1990). The oscillating signal oscillates across the cell membrane, triggering ion channels to increase or decrease the natural ion flows. In the brain this alters the neurotransmitters signals, changing the EEG pattern.

The relevance of this evidence to brain cancer is two-fold. The observations confirm biological interactions at extremely low induced electric field intensities. Radar signals induced very high proportional intensities in tissues. The alteration of cellular calcium ions, a well established EMR interaction phenomenon, Blackman (1990), is a determinant of the survival or programmed cell death of genetically damaged cells, Fanelli et al. (1999).

4. EMR Reduces Melatonin in People:

Melatonin is the most potent natural antioxidant agent in our bodies. Many processes produce free radicals that can damage DNA. Melatonin at night strong scavenges the free radicals as part of the cells genotoxic protection. Agents that reduce melatonin produce many diseases, Reiter and Robinson (1995), including neurological damage and cancer.

Many animal studies find that ELF fields reduce melatonin, Rosen, Barber and Lyle (1998). More than 15 studies from show that ELF and RF/MW exposure reduces melatonin in people and/or increases serotonin. Evidence that EMR altered the melatonin/serotonin balance in human beings commenced with Wang (1989) who found that workers who were more highly exposed to RF/MW had a dose-response increase in serotonin, and hence indicates a reduction in melatonin.

EMR melatonin reduction studies involve a wide range of exposure situations, including:

- 50/60 Hz fields, Wilson et al. (1990), Graham et al. (1994), Davis (1997) [in a dose response manner], Wood et al. (1998), Karasek et al. (1998), and Burch et al. (1997, 1998, 1999a, 2000), Juutilainen et al. (2000) and Graham et al. (2000);
- 16.7 Hz fields, Pfluger and Minder (1996);
- VDTs, with a wide range of ELF to RF exposures, Arnetz et al. (1996);
- a combination of 60 Hz fields and cell phone use, Burch et al. (1997), and
- increased geomagnetic activity around 30nT, Burch et al. (1999b)

Burch et al. (1999b) found an extremely significant dose-response, Figure 6.

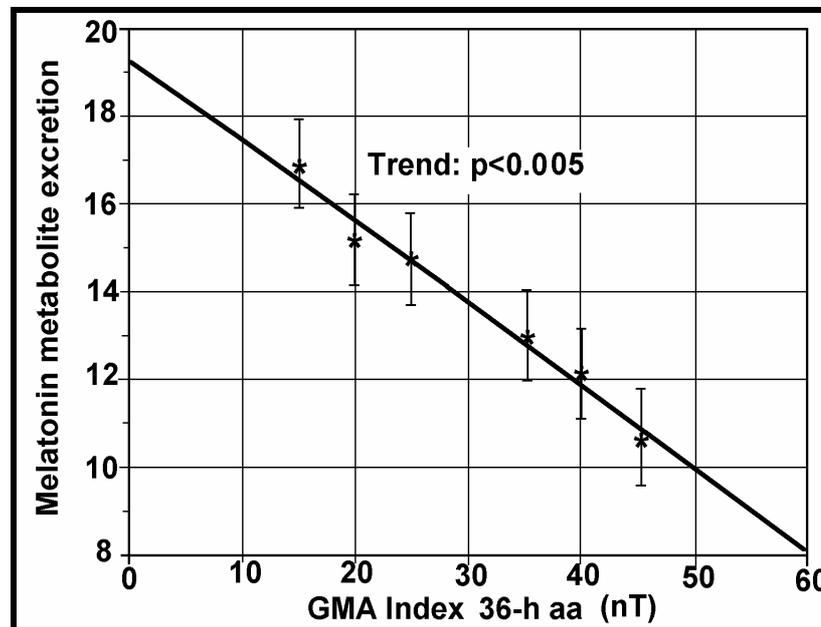


Figure 6: Reduction in the melatonin metabolite 6-OHMS in μg in urine from U.S. electric utility workers, as a function of the 36 hr global GMA aa-index, Burch et al. (1999b).

A similar effect is found in workers exposed to 60 Hz fields, Figure 7.

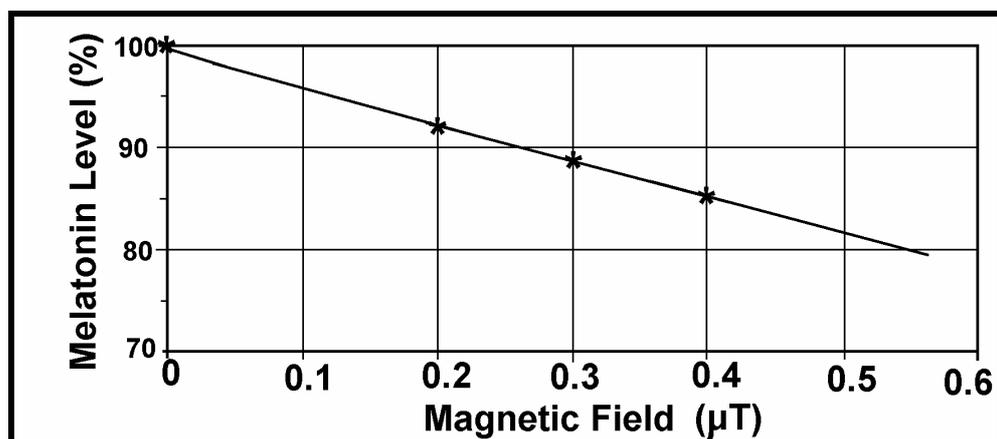


Figure 7: Reduction in melatonin in electrical workers exposed to 60 Hz magnetic fields, Davis (1997). Note $0.1 \mu\text{T} = 1 \text{ mG.}$, Trend $p < 0.005$.

Because of the EMR spectrum principle effect, if ELF signals reduce melatonin, then it is much more probable the RF/MW will reduce melatonin. This is confirmed by VDT and cellphone exposures.

A further study showed that RF exposure reduced human melatonin. Urinary melatonin was measure in humans prior to and after the Schwarzenburg shortwave radio tower was turned off, Theo Abelin (seminar and pers.comm.). This showed a significant rise in melatonin after the RF SW signal was turned off. The primary health effect of this study, Altpeter et al. (1995), was sleep disturbance which is a direct consequence of melatonin reduction. The RF exposure at down to extremely low levels ($0.0004 \mu\text{W}/\text{cm}^2$) caused significant sleep disturbance.

Melatonin, because of its vital diurnal functions in the daily cycle, has receptors in all of the body's vital organs, including the central nervous system, the brain, heart, lungs, liver, uterus, testes and fetus.

Hence it is established from multiple, independent studies, that EMR from ELF to RF/MW reduces melatonin in animals and human beings in a causal manner. Since melatonin is a potent antioxidant this establishes an independent mechanism showing the EMR is neurologically, reproductively and cardiologically active and genotoxic.

5. Direct evidence of EMR genotoxicity:

5.1 Background:

Because EMR alters calcium ion signalling and reduces melatonin it is predicted to be genotoxic and to cause enhanced cell death through apoptosis. This is also a process of premature aging. Substances that damage cellular genetic material, such as DNA-strands, chromosomes, and genes are called "genotoxic". Genotoxic substances cause cancer, cardiac and reproductive health effects and neurological damage. Chromosome aberrations are visible through powerful microscopes. Chromosomes are formed from folded segments of DNA. Damage to chromosomes is therefore evidence of damage to DNA. The genes are a section of the DNA in the chromosomes. Hence DNA damage damages chromosomes and alters genes.

DNA-strands are frequently damaged by natural substances, such as oxygen free radicals. Gey (1993) observes that free radicals may be involved in the etiology of cancer and cardiovascular diseases. In epidemiological studies poor plasma levels of antioxidants (free radical scavengers) are associated with increased relative risks of cancer and ischemic heart disease. Cells have elaborate DNA repair mechanisms because DNA stability is vital for species survival. Uncorrected DNA damage is mutation, Alberts et al. (1994). Alberts et al. outline many DNA repair mechanisms, including Repair Enzymes. They also outline the way apoptosis can digest and destroy damaged cells by internal "programming" of the process. The Immune System has B lymphocytes that produce antibody proteins to protect against 'foreign' cells, such as mutated cells. Natural Killer (NK) cells kill some types of tumours and some virus-infected cells, Alberts et al. (1994).

Enhanced DNA strand breakage leads to enhanced DNA repair. Hence enhanced DNA repair rates are also used as evidence of DNA damage, Meltz (1995).

Many studies have shown that radiofrequency/microwave (RF/MW) radiation and extremely low frequency (ELF) fields cause increased DNA strand breakage and chromosome aberrations. This has been shown in cell lines, human blood, animals and living human beings. This means that epidemiological studies of people exposed to electromagnetic radiation (EMR) are likely to show increased cancer, miscarriage and reproductive adverse effects. In fact many epidemiological studies have shown these effects, Goldsmith (1995, 1996, 1997a,b,c), Szmigielski (1996).

Two plausible biological mechanisms involving free radicals are involved in this effect. The first involves increased free radical activity and genetic damage as a response to exposure. The second involves increased free radical activity and genetic damage because of an induced reduction of a free radical scavenger, e.g. reduced melatonin, Reiter (1994). It is clear however, that both mechanisms have the same effect of damaging the DNA and chromosomes. Another established biological mechanism, EMR-induced alteration of cellular calcium ion homeostasis, Blackman (1990), is also involved in cell regulation, cell survival and apoptosis, DNA synthesis and melatonin regulation.

5.2 Direct measurements of Chromosome aberrations:

Direct evidence that EMR induces significant increases in chromosome damage, with significant dose response relationships, is evidence of a causal effect when replicated or extended by independent laboratories.

5.2.1 Chromosome damage from RF/MW exposure:

The first identified study that showed that pulsed RF radiation cause significant chromosome aberrations was Heller and Teixeira-Pinto (1959). Garlic roots were exposed to 27 MHz pulsed at 80 to 180 Hz. for 5 min and then they were examined 24 hrs later. They concluded that this RF signal mimicked the chromosomal aberration produced by ionizing radiation and c-mitotic substances. No increased temperature was observed.

Blood samples were taken from the staff of the U.S. Embassy in Moscow. They had been chronically exposed to a low intensity radar signal. Significant increases in chromosome damage was reported, Tonascia and Tonascia (1966) cited in Goldsmith (1997a).

Yao (1978) found a dose-response elevation chromosome damage in Chinese Hamster cells with exposure time in minutes, Figure 8.

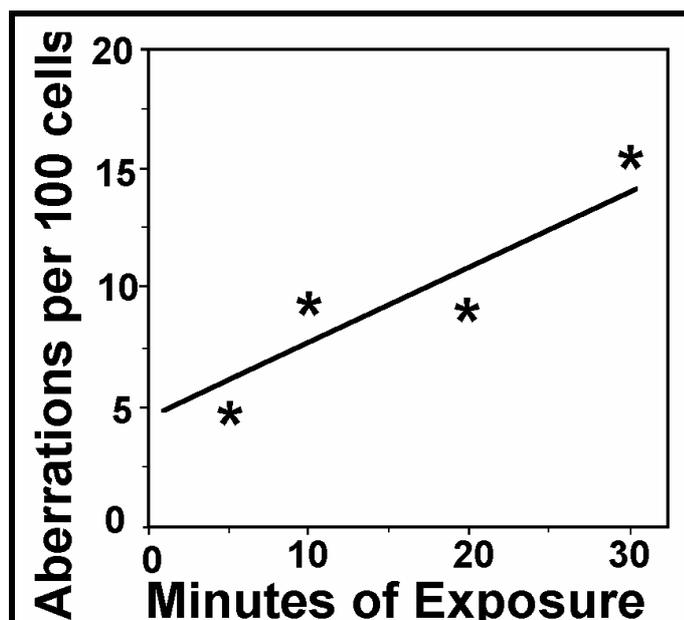


Figure 8: Chromosome aberrations, per 100 cells, in live female Chinese Hamsters eyes, exposed to 2.45 GHz microwaves at 75 mW/cm² for 10 minutes and then 1 month later to 100 mw/cm² for 5, 10, 20 and 30 minutes, Yao (1978). Trend, $p < 0.05$.

Yao (1982) exposed rat kangaroo RH5 and RH16 cells to 2.45 GHz microwaves, maintaining the temperature at 37°C in the incubator. After 50 subculture passages with microwave exposure there were 30 passages without. Significant chromosome aberrations were measured after 20 microwave exposed subculture passages.

Elevated and significantly elevated (*) chromosome damage with RF/MW exposure has been observed by Chen, Samuel and Hoopingarner (1974), Manikowska et al. (1979), Nawrot, McRee and Staples (1981*), Banerjee, Goldfeder and Mitra (1983a,b*), Antipenko, Koveshnikova and Timchenko (1984), Manikowska-Czerska, Czerska and Leach (1985), and Beechey et al. (1986). Thus before the end of the 1980's at least 12 studies had reported increased or significantly increased chromosome aberrations (CAs) from RF/MW exposure. Since then many more studies have found that RF/MW is genotoxic by damaging chromosomes and DNA and increases cell death.

Garaj-Vrhovac et al. (1990) noted the differences and similarities between the mutagenicity of microwaves and VCM (vinyl chloride monomer). They studied a group of workers who were exposed to 10 to 50 $\mu\text{W}/\text{cm}^2$ of radar produced microwaves. Some were also exposed to about 5 ppm of VCM, a known carcinogen. Exposure to each of these substances (microwaves and VCM) produced highly significant ($p < 0.01$ to $p < 0.001$) increases in Chromatid breaks, Chromosome breaks, acentric and dicentric breaks in human lymphocytes from blood taken from exposed workers. The results were consistent across two assays, a micronuclei test and chromosome aberration assay.

Chromosome aberrations and micronuclei are significantly higher than the controls, ($p < 0.05$, $p < 0.001$, $p < 0.0001$), for each of the exposure intensity. All people are exposed to toxins in the air and in water and so evidence of synergistic effects is relevant. The exposure levels showing highly significant chromosome aberrations and micronuclei in this study are within the range of exposures to police radar. The micronuclei test is strongly indicative of genotoxicity, Berwick and Vineis (2000).

To extend the result found in microwave exposed workers, Garaj-Vrhovac, Horvat and Koren (1991) exposed Chinese hamster cells to 7.7 GHz microwave radiation in a constant temperature system, to determine cell survival (cell death) and chromosome damage. They assayed chromosome aberrations and micronuclei and found that microwaves increased these in a dose-response manner, Figure 9, to levels that were highly significantly elevated ($p < 0.02$ to $p < 0.01$).

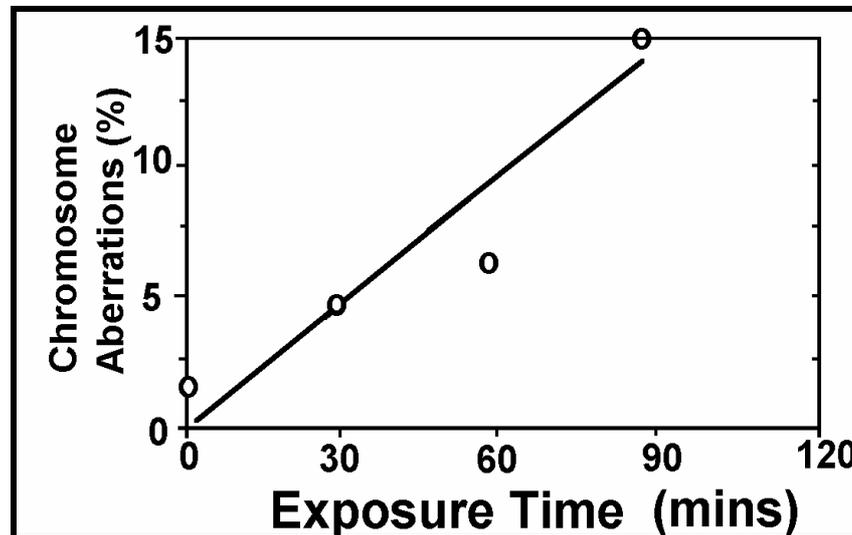


Figure 9: Chromosome aberrations in V79 Chinese hamster cells exposed to 7.7 GHz microwaves at 30 mW/cm^2 , Garaj-Vrhovac, Horvat and Koren (1991). Trend $p < 0.05$.

An exposure level of 30 mW/cm^2 is usually able to slightly raise the temperature over an hour. This experiment was undertaken under isothermal conditions, with samples being kept within 0.4°C of 22°C . The consistency of the time exposure and the survival assay at non-thermal exposure levels, confirms that this is a non-thermal effect.

This is very strong evidence of genotoxic effects from RF/MW exposures. When chromosomes are damaged one of the primary protective measures is for the immune system natural killer cells to eliminate the damaged cells. Alternatively the cells can enter programmed cell suicide, apoptosis. Garaj-Vrhovac, Horvat and Koren (1991) measured the cell survival rates. They found that cell survival reduced and the cell death increased in a time dependent and exposure dose response manner, Figure 10.

Figure 10 shows that cell death varies with time and intensity of exposure, down to very low exposure levels. An apparent 'saturation' at high levels also become apparent. This is probably because of the lethal effect of high intensity microwaves. Since this is an isothermal experiment it raised important questions about the reasons for the cell death as acute genetic damage which is continuously related to microwave exposure down to non-thermal levels.

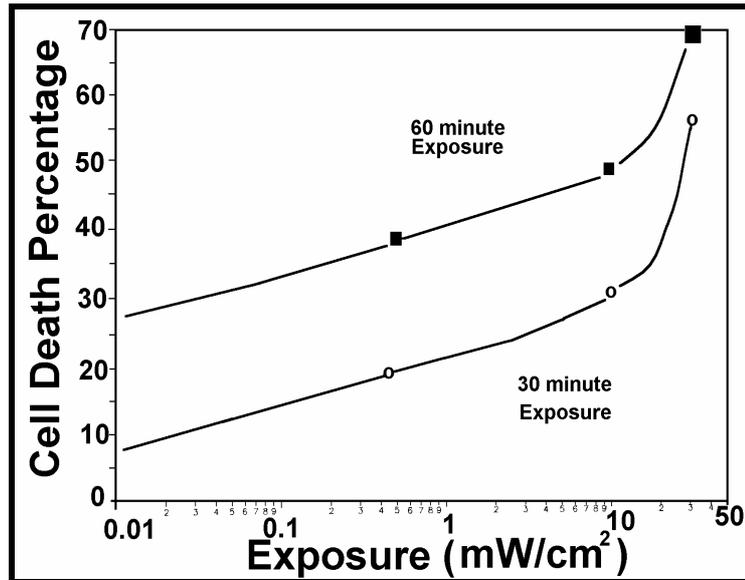


Figure 10: Cell death percentage of Chinese hamster cells exposed to 7.7 GHz microwaves (CW) for 30 minutes and 60 minutes in an isothermal exposure system, Garaj-Vrhovac, Horvat and Koren (1991).

Note that the US standard for microwaves above 2 GHz is 1 mW/cm², and for workers is 10 mW/cm². Even at 100 times below the public exposure guideline a 60 minute exposure kills 28% of the cells and 30 minutes kills 8 % of the cells. Garaj-Vrhovac et al. (1992) exposed human lymphocytes and showed that microwave radiation produced a dose response increase in various types of chromosome aberrations, Figure 11.

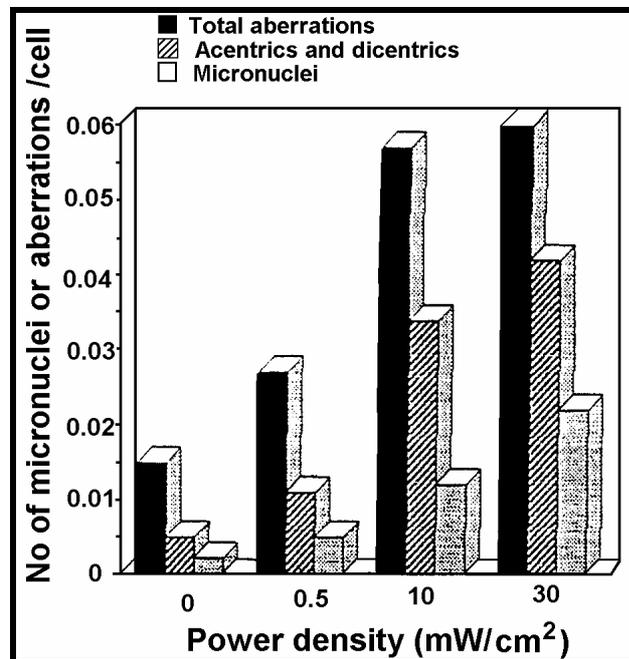


Figure 11: The relation of total chromosome aberrations. Micronuclei and specific chromosome aberrations for each cell in human lymphocyte cultures in the dose of microwave radiation in vitro, Garaj-Vrhovac et al. (1992).

For the micronuclei the dose-response trend is significant, $p=0.03$.

Having established that microwave exposure damaged chromosomes, this research group were asked to analyze blood samples from workers who had been exposed to pulsed

microwaves generated by air traffic control radars while they were repairing them. Garaj-Vrhovac, Fucic and Pevalek-Kozlina (1993) analysed the chromosome aberration (CA) in 6 technical staff who had experienced accidental exposure to the radar. The initial CA percentage ranged from 3% to 33%, all being significantly higher than unexposed people. The repair rate over time was monitored.

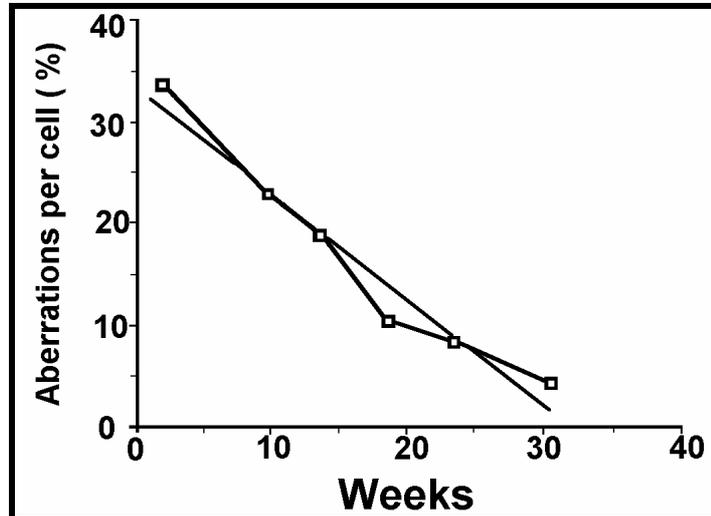


Figure 12: The time-dependent decrease in the number of chromosome aberrations for subjects with high numbers of chromosomal impairments, $y = 0.318 - 0.010x$, $r=0.98$. Garaj-Vrhovac, Fucic and Pevalek-Kozlina (1993).

Figure 12 shows the man who had 33 % CA which was followed over 30 weeks following this exposure. The repair rate follows a significant linear rate ($r=0.98$), dropping from 33% to 3% over 30 weeks, 1 %/week.

CA Repair rates in other workers are shown in Figure 13.

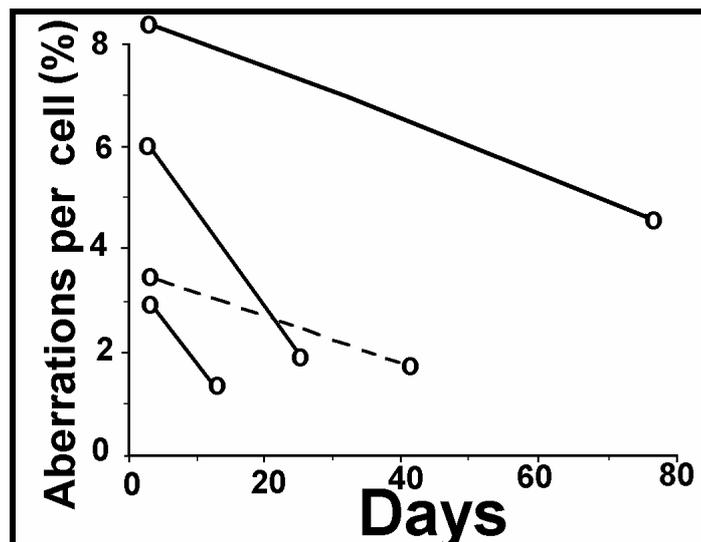


Figure 13: Decreases in human blood Chromosome Aberrations over time from microwave exposed radar repair workers, Garaj-Vrhovac and Fucic (1993).

Two different repair rates are evident. Two at 0.6 to 1.1 %/week and two at 0.25 to 0.35 %/week. The authors note that Sagripanti and Swicord (1986) showed that microwave radiation damaged single-strand DNA and the Szmigielski (1991) showed that out of 29

epidemiological studies in the previous decade, 22 suggested a relationship between various neoplasms and exposure to electromagnetic fields.

Garaj-Vrhovac (1999) found that 12 workers occupationally exposed to microwave had significantly increased chromosome damage as well as disturbances in the distribution of cells over the first, second and third mitotic divisions.

Quite independently, Maes et al. (1993) found highly significant ($p < 0.001$) increases in the frequency of chromosome aberrations (including dicentric and acentric fragments) and micronuclei in human blood exposed to 2.45 GHz microwaves for 30 to 120 minutes in vitro. The micronuclei assay showed a dose response with time, Figure 14.

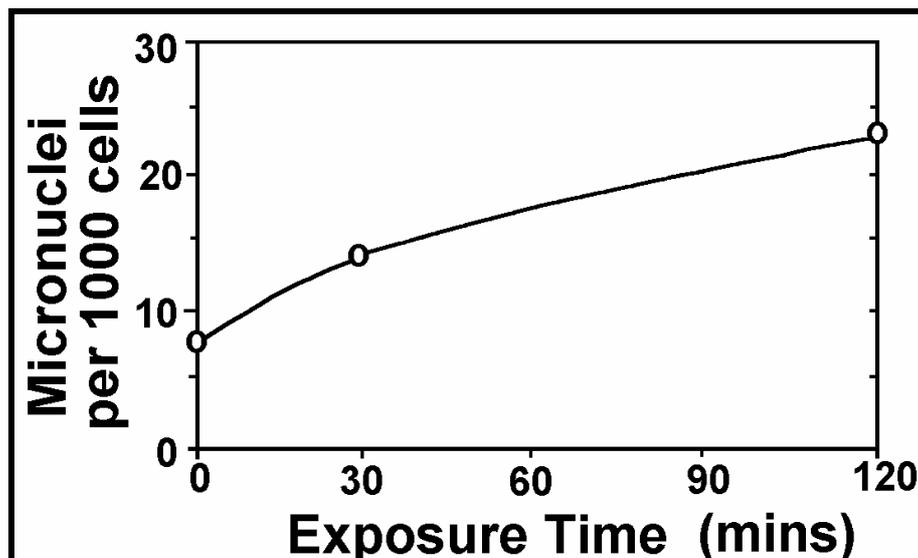


Figure 14: Micronuclei in microwave exposed human lymphocytes, the average of 4 donors, Maes et al. (1993). Exposure was to 75 W/kg, 2.45 GHz microwaves pulsed at 50 Hz, under controlled isothermal conditions

Maes et al. (1997) observed elevated CAs from microwave exposure. Koveshnikova and Antipenko (1991a,b), Haider et al. (1994) Timchenko and Ianchevskaia (1995), Balode (1996), Mailhas et al. (1997) and Vijayalaxmi et al. (1997), and Pavel et al. (1998) have reported significant chromosome aberrations from RF/MW exposures.

Vijayalaxmi et al. (1997) chronically exposed cancer prone mice to 2.45 GHz CW microwaves at an SAR of 1 W/kg for 20 hr/day, 7 days/week for 18 months. Their aim was to determine whether microwaves were genotoxic through determining if there was significant chromosome damage. They found highly significant increases in micronuclei in peripheral blood, from 8 per 2000 cells in sham exposed mice to 9 per 2000 cells microwave exposed mice, and increase of 12.5 %, $p < 0.001$. There was a significant increase of 6.6%, $p < 0.025$, of micronuclei in the bone marrow. They also observed a significant 41 % increase in tumours in the exposed mice compared to the sham exposed mice.

With the mobile phone introduction research into the signals used has shown a dose-response increase in chromosome aberrations. Tice, Hook and McRee (1999) showed micronuclei formation from all cell phones tested. All were statistically significant and all but one highly significant with dose-response relationships up to a factor of three increase in micronuclei formation. They repeated the experiment and confirmed that the results were robust and not an artifact.

Roti Roti (2000) found that cell phone radiation caused significant micronuclei formation, cited in Carlo and Schram (2001).

Micronuclei formation is very strong evidence of genotoxicity and the ability of an agent to produce cancer, Berwick and Vineis (2000). The published data shows multiple studies in which RF/MW radiation significantly damages the chromosomes and forms micronuclei in dose-response manner, showing a causal relationship.

Maes et al. (1996) also showed that 954 MHz cell phone radiation significantly increased chromosome aberrations, Figure 15.

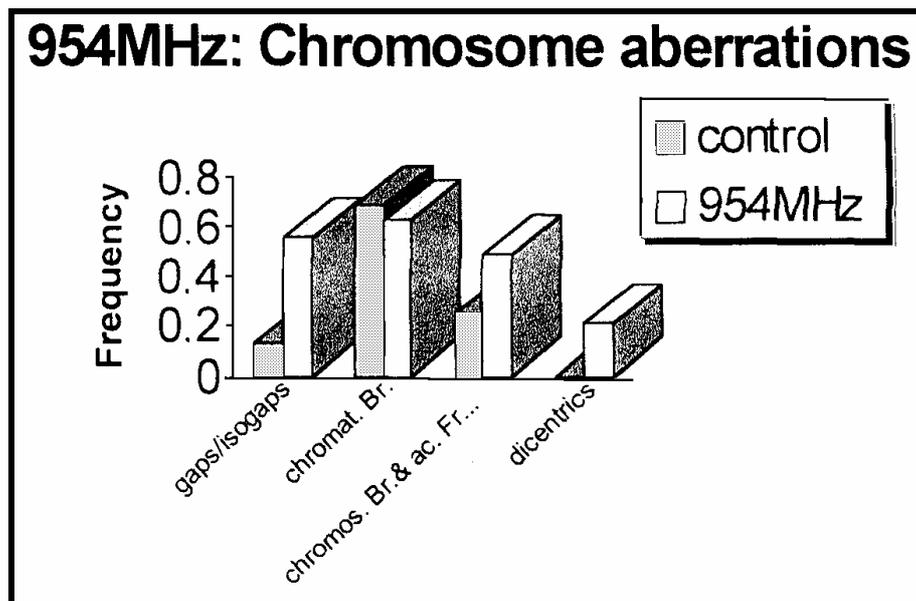


Figure 15: Cellphone radiation of 954 MHz significantly enhances several types of chromosome damage including gaps, acentric and dicentric aberrations, Maes et al. (1996)

Concerns about the health effects of cellphones led to three studies (Maes et al. (1996), Tice, Hook and McRee (1999) and Roti Roti (2000), found that cellphone-type radiation significantly damages chromosomes. This is not surprising given the very large evidence already published showing that RF/MW damages chromosomes in similar manners to c-mitotic chemicals and ionizing radiation. This description was first given in 1959. Between 1990 and 2000 another 18 studies have been added to bring the total to at least 30.

Multiple independent studies, in over 30 papers, show increases and most show significant increases in chromosome aberrations from RF/MW exposure. Four studies show dose-response relationships. This is more than adequate to classify RF/MW radiation as causal genotoxic.

5.2.2 Chromosome damage from ELF exposure:

The results for RF/MW are reinforced from studies involving ELF exposures. El Nahas and Oraby (1989) observed significant dose-response dependent micronuclei increase in 50 Hz exposed mice somatic cells. Elevated CAs have been recorded in a number of workers

in electrical occupations. In Sweden Nordenson et al. (1988) found significant CA in 400 kV-substation workers and with 50 Hz exposures to peripheral human lymphocytes, Nordenson et al. (1984) and human amniotic cells, Nordenson et al. (1994).

Significant CA in human lymphocytes exposed to 50 Hz fields are also reported by Rosenthal and Obe (1989), Khalil and Qassem (1991), Garcia-Sagredo and Monteagudo (1991), Valjus et al. (1993) and Skyberg et al. (1993). Skyberg et al. collected their samples from high-voltage laboratory cable splicers and Valjus et al. from power linesmen. Other studies showing ELF associated CAs include Cook and Morris (1981), Cohen et al. (1986 a,b), Lisiewicz (1993), Hintenlang (1993) and Timchenko and Ianchevskaia (1995). This currently involves 15 studies.

Hence chromosome damage has been recorded from exposures across the EMR spectrum from ELF to RF/MW exposures, in plants, mammal and human cells, animals and human beings, and from many independent laboratories. This confirms that EMR does damage chromosomes and establishes EMR induced chromosome aberrations as a biological effect. For a neoplastic cell to survive it must have an altered genetic structure to store the damage and to hide this from the immune system so that NK cells do not kill the neoplasm transformed cells.

5.2.3 Chromosome Aberrations Conclusions:

Many studies, from independent laboratories, have shown that ELF, RF/MW and cell phone radiation, significantly increases chromosome aberrations in exposed cells and animals, and including cells taken from human beings who have been exposed to EMR in occupational situations. Even at very low intensity radar exposures that were experienced at the U.S. Embassy in Moscow, significant increases in chromosome damage was measured from human blood samples. This evidence shows conclusively that across the EMR spectrum, EMR is genotoxic. Hence it is carcinogenic and teratogenic. There is some support that pulsed microwaves are more genetically hazardous than CW microwaves though both are shown to be genotoxic. Hence radars, including police radars, that used pulsed and/or modulated microwaves, are strongly genotoxic and carcinogenic hazards.

5.3 Direct evidence of neoplasm in microwave exposed cells:

Balcer-Kubiczek and Harrison (1991) observed a significant dose response increase of neoplastic transformation in a standard cell set (C3H/10T1/2) from a 24 hr exposure to 2.45 GHz microwaves. The transformation was assayed after 8 weeks of exposure to a known cancer promoter chemical TPA, Figure 16. The method was confirmed with a positive control using X-rays. This also showed that 60Hz magnetic fields also significantly increased neoplastic transformation.

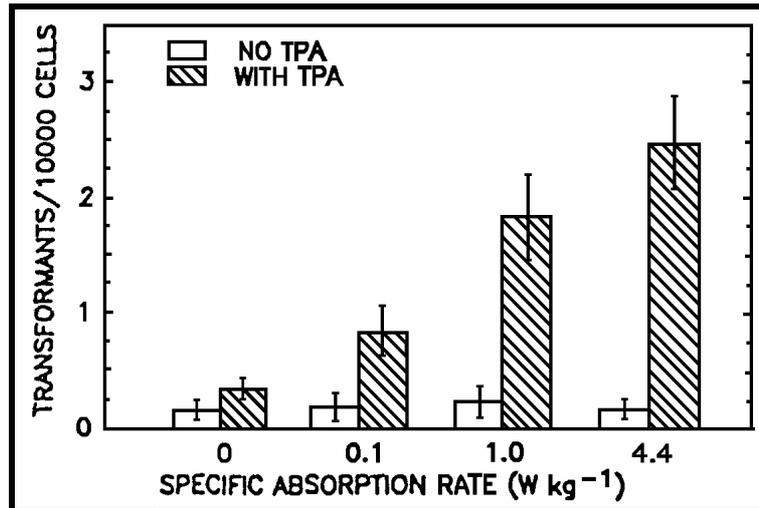


Figure 16: Dose-response relationship for induction of neoplastic transformation in C3H/10T1/2 cells by a 24h exposure to 2.45 GHz microwaves at the specific absorption rate (SAR) with and without TPA post-treatment for 8 weeks, Balcer-Kubiczek and Harrison (1991).

This research result confirms the observations of Stodolnik-Baranska (1967) that found a significant, non-thermal microwave induced transformation of human lymphocyte cells into lymphoblastoid and macrophages in a similar reaction to specific toxins.

5.4 Toxic Shock Proteins

A class of proteins, called Heat Shock Proteins (HSPs) are produced when cells are over heated to the extent that DNA is damaged. They assist with the repair of the damaged DNA. These proteins are also produced when toxic chemicals damage the DNA. Hence they can also be called Toxic Shock Proteins. Toxic Shock Proteins are significantly enhanced by 0.001 W/kg 750 MHz microwaves, Figure 17.

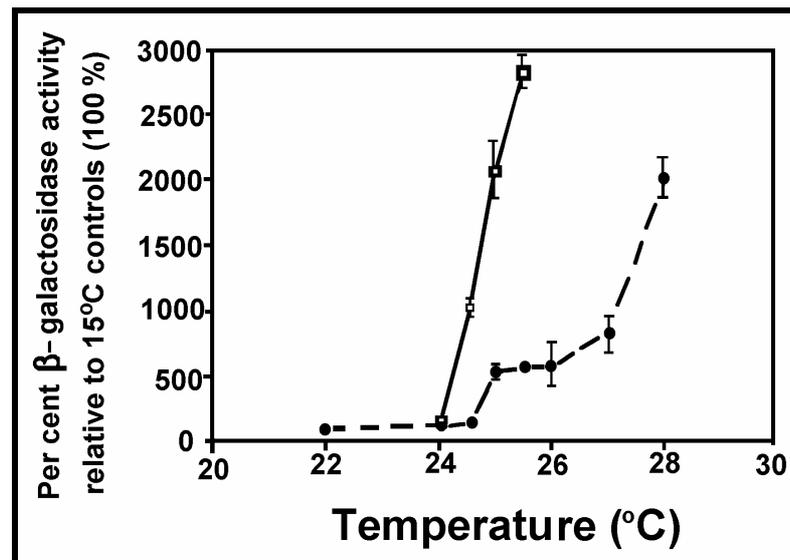


Figure 17: Toxic shock proteins (heat shock proteins) in young adult PC72 soil nematode worms (*Caenorhabditis*) exposed to 750 MHz microwaves compared with at 15 °C control, at 24.0, 24.5, 25 and 25.5 °C, with 12 replicates for each condition (solid line). It was also run at 22, 26, 27 and 28 °C (dashed line). De Pomerai et al. (2000).

The expression of HSP16 at 25°C under the low intensity microwave exposure produces the same level of HSP16 as heat alone at 28°C. This is extremely significantly different, $p < 0.001$. The trend is also extremely significant, $p < 0.001$. The exposure condition used is SAR = 0.001 W/kg. This is about $0.5 \mu\text{W}/\text{cm}^2$. This is further direct evidence that microwaves are toxic by significantly stimulating the production of toxic (heat) shock proteins.

5.5 Direct evidence of RF/MW caused DNA strand breakage:

5.5.1 Altered genome by microwave exposure:

Sarkar, Ali and Behari (1994) investigated the effect on DNA of exposures accepted a safe by the Non-ionizing Radiation Committee of IRPA (International Radiation Protection Association - the predecessor of ICNIRP). The exposure regime was a 2 hr exposure to 2.45 GHz CW microwaves at $1 \text{ mW}/\text{cm}^2$, SAR = 1.18 W/kg. They observed significant alterations in the DNA from rat brains and testis in the 7 to 8 kb region of the DNA in the hybridization profile and in a densitometric analysis, Figure 18.

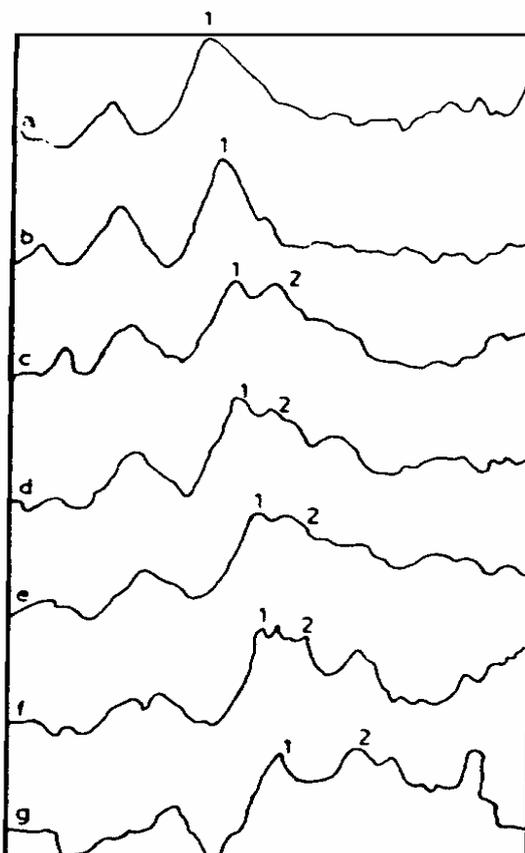


Figure 18: Densitometric analysis of the brain DNA, a and b are control DNA, c to g are DNA from microwave exposed animals. Peak 1 is present in both control and exposed animals while peak 2 appears only in all of the exposed animals.

5.5.2 The Comet Assay Method:

A very advanced assay of DNA strand breakage has been developed by Dr N.P. Singh at the University of Washington. This is called the microgel electrophoresis or Comet Assay, Singh et al. (1994). The Comet Assay involves migration of segments of DNA down an electric field gradient, Figure 19.

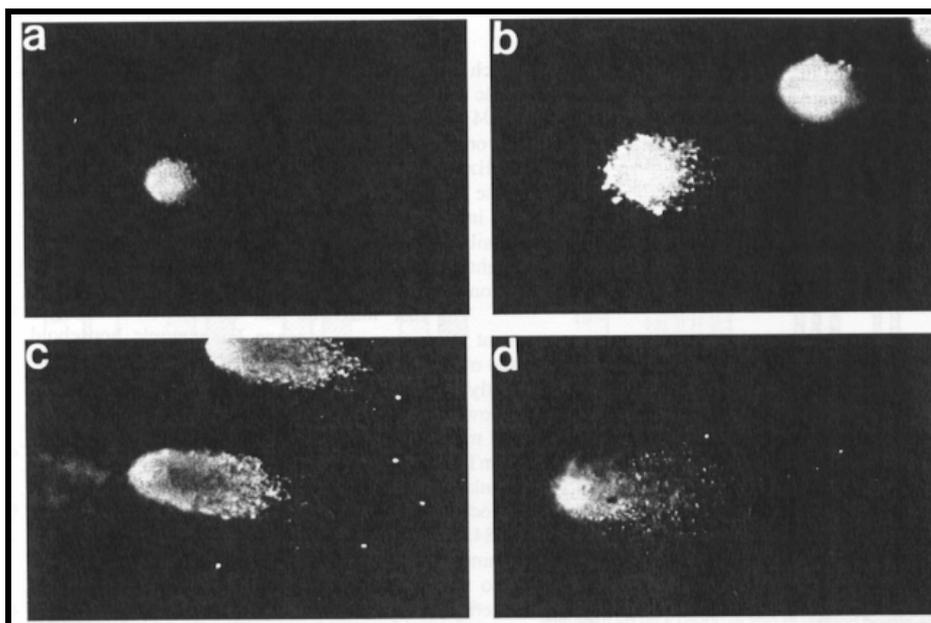


Figure 19: Photographs of double-strand break DNA migration pattern of individual brain cells from rats exposed to (a) bucking condition (0.1 mT), (b) magnetic fields of 0.1 mT, (c) 0.25 mT and (d) 0.5 mT, Lai and Singh (1997a). The “bucking mode” is the condition to reverse the field to cancel the magnetic fields with all else remaining constant.

The modified microgel electrophoresis assay or Comet Assay for single DNA-strand breaks, involves extraction of a sample of tissue, washing it several times to remove blood, snipping the tissue with sharp scissors to reduce the sample sizes and further washing to remove blood. Single cell suspensions are mixed with agarose to make a microgel on a slide that is cooled to form a gel. Slides are immersed in an ice-cold lysing solution and then stored in the dark at 4 °C.

DNA is closely associated with protein and RNA. They help to fold and bind the DNA. DNA is negatively charged and the bound protein is positively charged. To release DNA from these bonds, and to separate the charges, Proteinase K must be used to digest proteins and RNAase A to digest RNA. Hence in the morning the slides were treated with DNAase-free proteinase K for 2 hr at 37 °C to remove the bound protein from the DNA. They were then placed on the horizontal slab of an electrophoretic assembly. An electrophoresis buffer is added and the sample is left for 20 min to allow the DNA to unwind. The buffer includes antioxidants to counter the free radicals produced by electrophoresis.

The electrophoresis was then carried out for 60 minutes with 0.4 V/m, 250 mA. During this process the fluid in the assembly is re-circulated at the rate of about 100 ml/min. The negatively charged segments of DNA migrate down the electric field gradient, forming a comet-like tail, the mass of which is proportional to the amount of damaged DNA material and the electric field gradient and time of exposure.

For DNA double-strand breaks the microgel preparation is the same as above. Slides are then treated with ribonuclease A for 2 hr and then proteinase K for 2 hr. They are then placed in the neutral electrophoresis buffer (pH 9) for 20 mins and then electrophoresed for 1 hr at 0.4 V/cm. For both single- and double- strand assays the sample are stained with an intense fluorescent dye solution of YOYO-1 and then examined in a vertical fluorescent microscope.

The proteinase K treatment is vital. It removes the positive charged bound protein from the negative charged DNA strands. Because the DNA and bound protein have the opposite charged the protein must be removed so that the electric field gradient produces the migration of the broken DNA strands. Four slides were prepared for each animal, two for single and two for double-strand assays. Fifty representative cells were scored off each slide, giving 100 cells scored for each of the single and double-strand DNA breaks. Frequency distributions for the 100 assayed cells are presented, Figure 20, and the comet tail moment calculated.

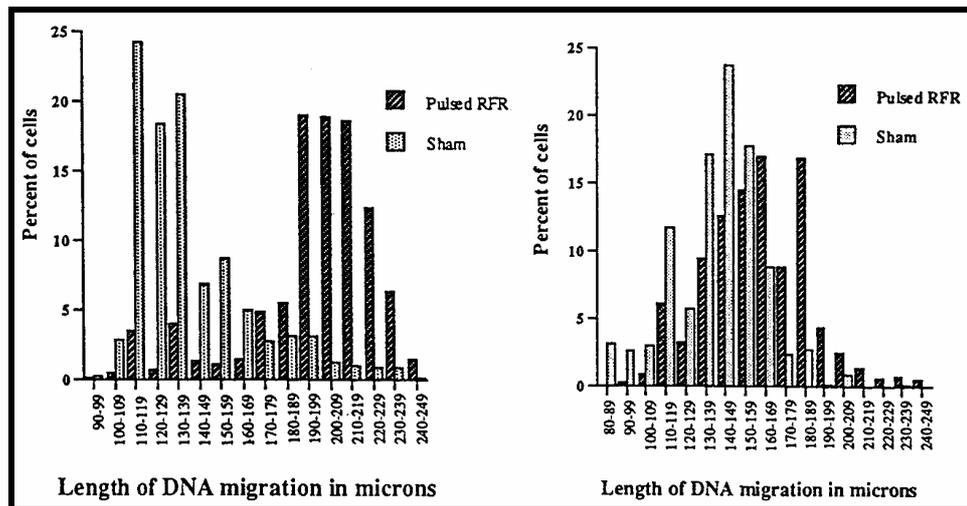


Figure 20: Single-strand (left) and double-strand (right) DNA breaks frequency distribution for percentage of cells of a given tail length from pulsed RFR and sham exposed brain cells, from 8 animals and 100 cells per animal, Lai and Singh (1996).

Figure 20 clearly shows significant increases in single- and double-strand DNA breaks from the pulsed microwave exposed animal brains compared with the sham exposed animals. The tail DNA fragments extend out to 250 microns. The Comet tails in the Malyapa et al. assay extend to less than 40 microns, Figure 21. This clearly documents how less sensitive their method is.

5.5.3 Motorola Funded Counter Research on DNA breakage:

Motorola funded Dr Joseph Roti Roti's group at Washington University, St Louis, to replicate the Lai/Singh DNA damage research and to extend it to cell phone frequencies, Malyapa et al. (1997a,b). "Replication" requires the work to very closely follow the method and conditions of the earlier study. It is usual to engage independent researchers who are well qualified. While both Lai and Singh and Dr Joseph Roti Roti's group used 2.45 GHz microwaves for exposure, the follow up study used a cell line (C3H/10T1/2) compared to Lai/Singh use of living rats. The St Louis group also used a very different DNA damage assay based on Olive et al. (1992) not Singh et al. (1988, 1994). This follow up study used a much weaker fluorescent stain, an overall weaker electrophoresis field (0.6 V/cm for 25 mins c.f. 0.4 V/cm for 60 mins) and most importantly, did not use proteinase K to separate the positively charged bound protein from the negatively charged DNA strands.

It is therefore not a "replication" study and could be seen as an attempt to deliberately avoid the results by using a much less sensitive assay. This less sensitive effect produces comet tails up to 38 microns, Figure 21, compared with the Lai and Singh assay having 250 microns tails, Figure 20.

A close consideration of the data presented in Malyapa et al. shows frequency distributions of comet tail lengths and moments that are clearly different after exposure to the microwave radiation. It was decided to analyse some of the data that was visually different to determine the time sequence of the DNA damage and repair process and to determine the significance of the changes. A 2x2 analysis was carried out with a cut-off point typically about 2/3rds of the way up the x-axis.

The first example was from Malyapa et al. (1997a), Figure 5, shown in Figure 21. The sham exposure distribution is very narrow with a maximum at 32 microns. The 2hr distribution has much less at 25 microns and more above 28 microns. The 2x2 analysis is presented in Table 1.

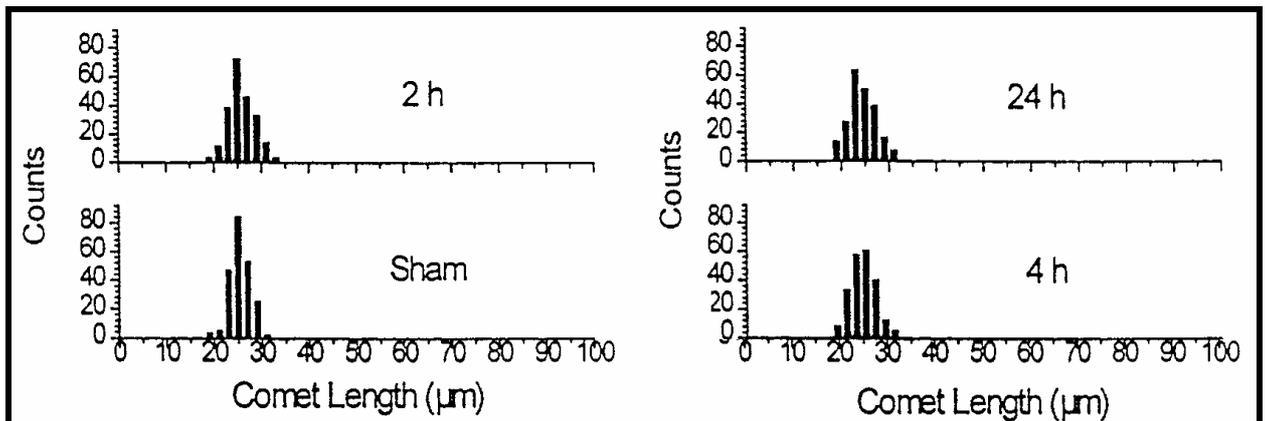


Figure 21: Frequency Distribution of Comet tail lengths for 2.45GHz exposed C3H10T1/2 cells, Malyapa et al. (1997a).

Table 1: The 2x2 table of results for DNA strand breakage after exposure of U87MG cells to 2.45 GHz microwaves, Figure 11:

Time	Comet Length Class		RR	95%CI	χ^2	p-value
	$\leq 28\mu\text{m}$	$> 28\mu\text{m}$				
Sham	196	29	1.00			
2hr	174	51	1.75	1.16 -2.76	7.34	0.0067
4 hr	206	20	0.06	0.40 -1.18	1.90	0.169
24 hr	197	25	0.87	0.53 -1.44	0.28	0.60

The time sequence of variations reveals a significant increase in DNA strand breakage after 2 hours and then the repair process kicks in and over compensates, Figure 22.

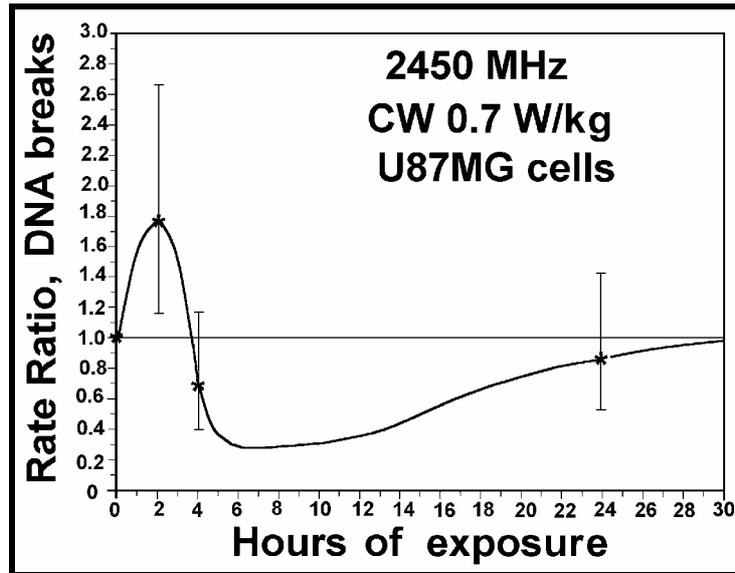


Figure 22: The time sequence of DNA damage and enhanced repair for Figure 5 in Malyapa et al. (1997a).

This confirms the Lai and Singh results rather than contradicting them. This shows significant DNA strand breakage after 2 hours.

Two other figures of frequency distributions in Malyapa et al. (1997a and b) were digitized and analysed using a 2x2 analysis of the Risk Ratio, Chi Squared and p-values, using a cut-level in the middle of the distribution. The following time courses of DNA breakage and repairs resulted. Figure 23 shows the frequency distribution of normalized comet moment for CW exposure of 2450 MHz at 0.7 W/kg of C3H 10T1/2 cells, Malyapa et al. (1997a), Figure 6.

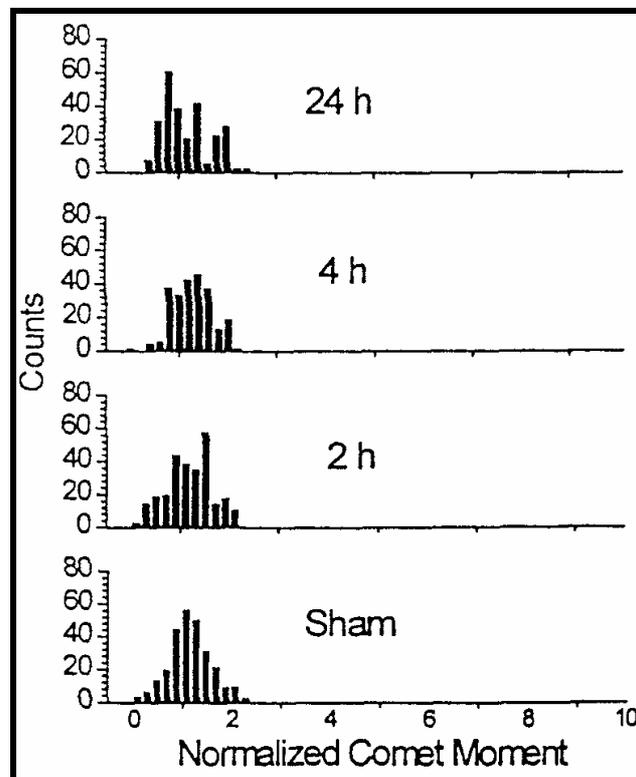


Figure 23: The frequency distribution of normalized comet moment for CW exposure of 2450 MHz at 0.7 W/kg of C3H 10T1/2 cells, Malyapa et al. (1997a) Figure 6.

Table 2: The 2x2 table of results for DNA strand breakage after exposure of C3H 10T1/2 cells to 2.45 GHz microwaves, Figure 13:

Time	Comet Moment Class		RR	95%CI	χ^2	p-value
	≤6	>6				
Sham	194	75	1.00			
2hr	176	101	1.31	1.02 -1.67	4.59	0.0321
4 hr	126	119	1.74	1.38 -2.20	23.31	0.0000014
24 hr	159	132	1.63	1.29 -2.05	18.30	0.0000189

The time sequence from Table 2 is plotted in Figure 24.

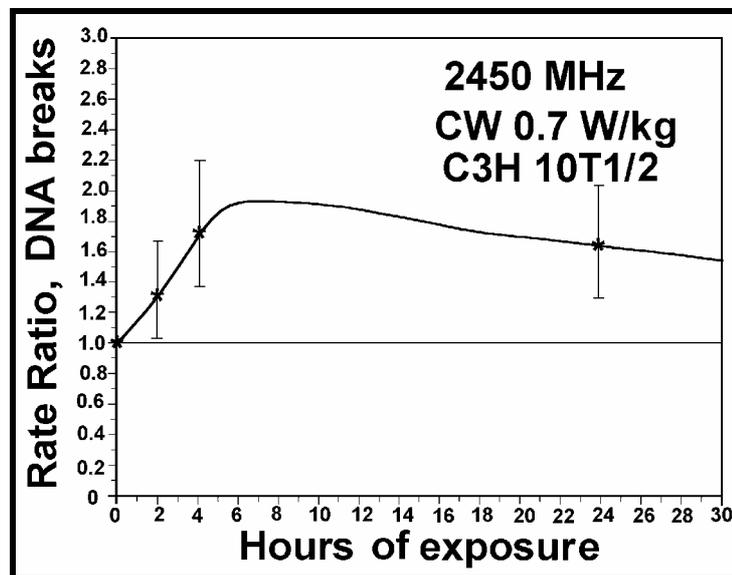


Figure 24: DNA strand breakage Risk Ratio and 95% confidence intervals for the frequency distribution of the Normalized Comet Moment of Malyapa et al. (1997a), Figure 6

Table 2 and Figure 24 shows significantly increased DNA strand breakage for more than 24 hours after a non-thermal microwave exposure of 0.7 W/kg of 2450MHz CW microwaves of C3H 10T1/2 cells.

The third example is derived from Figure 2 in Malyapa et al. (1997b) in which a cell phone signal, CDMA, at an exposure SAR of 0.6 W/kg of U87MG cells, Figure 25.

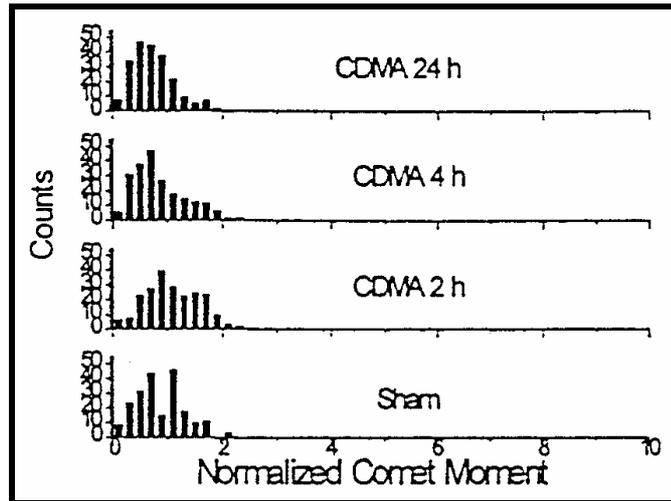


Figure 25: The frequency distribution of normalized comment moment for CW exposure of 847.72 MHz at 0.6 W/kg of U87MG cells, Malyapa et al. (1997b) Figure 2.

Table 3: The 2x2 table of results for DNA strand breakage after exposure of U87MG cells to 847.74 MHz microwaves, Figure 13:

Time	Comet Moment Class		RR	95%CI	χ^2	p-value
	≤ 6	> 6				
Sham	168	42	1.00			
2hr	138	92	2.00	1.46 -2.74	20.68	0.0000052
4 hr	158	50	1.20	0.84 -1.73	0.99	0.3196
24 hr	195	24	0.55	0.34 -0.87	6.72	0.00956

Table 3 shows an extremely significant increase in DNA strand breakage 2 hours after the cellphone radiation exposure, $p < 0.00001$. The time sequence, Figure 26, shows the same general pattern as also seen for U87MG cells exposed to 2.45 MHz radiation in Figure 22 above.

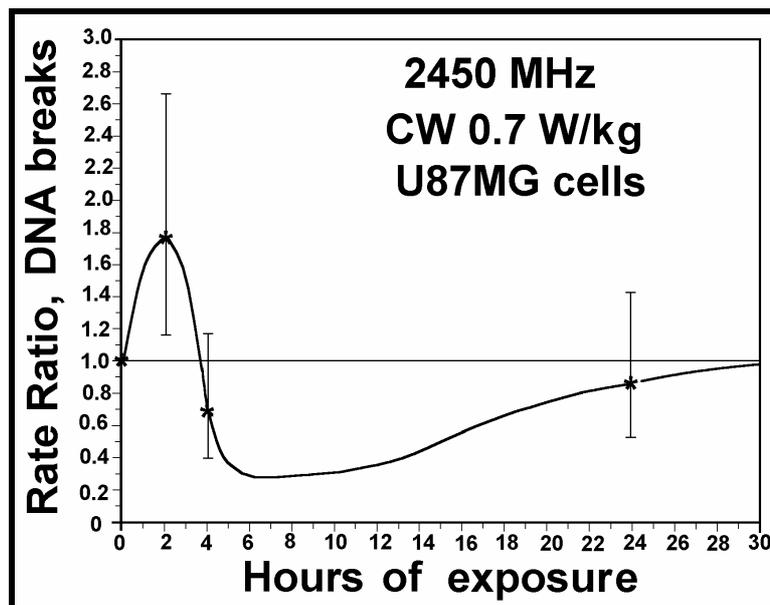


Figure 26: DNA strand breakage Risk Ratio and 95% confidence intervals for the frequency distribution in Figure 2 Normalized Comet Moment of Malyapa et al. (1997b).

These results confirm the Lai and Singh results and confirm that microwave radiation and cell phone radiation significantly damages DNA strands and induces repair and significant repair after 4 hours in some cases. The C3H 10T1/2 cells show much slower DNA repair rates than the U87MG cells, indicating a cell-specific characteristic. It is also well known that the damage and repair rates are strongly dependent on the position in the cell cycle, Durante et al. (1994).

This also puts the results of Phillips et al. (1998) into context. They found highly significant ($p < 0.0001$) DNA strand breakage at 0.0024 W/kg exposure to cell phone radiation. They also found significant DNA strand repair ($p < 0.0001$) with other exposure regimes at a similar SAR level. Significant DNA strand repair is initiated by DNA strand breakage. This is why earlier assays were based on looking for induced DNA repair as an indicator of DNA damage, Meltz (1995).

5.5.4 Lai and Singh extended results:

Lai and Singh used a considerably more sensitive DNA comet assay. In Lai and Singh (1995) a significant level ($p < 0.001$) of DNA single-strand breakage was measured 4 hours after the exposure of living rat brains. Using sham exposure, 0.6 W/kg and 1.2 W/kg they observed a dose-response increase in DNA single-strand breakage for the hippocampus and for the Rest of the Brain, Figure 27.

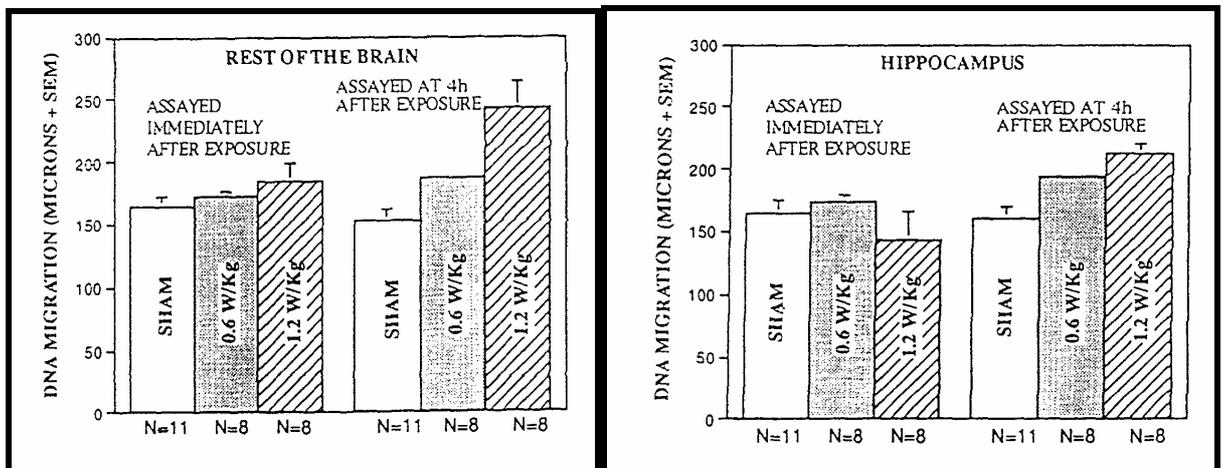


Figure 27: DNA single-strand breakage in cells from the rat brain and hippocampus, immediately after a 2 hr exposure to a whole body SAR of 0.6 and 1.2 W/kg to 2.45 GHz microwave radiation, pulsed at 500 pps. N is the number of rats studied. Lai and Singh (1995).

The dose-response increase in the Rest of the Brain immediately after exposure was not found in the Hippocampus showing the complex different rates of expressing the DNA damage that also depends on the position in the cell cycle, Durante et al. (1994).

The comet assay method was extended to measure DNA double-strand breakage by using ribonuclease A to remove the RNA before using the proteinase K to remove the bound protein. Lai and Singh (1996) reported that both continuous wave (CW) and pulsed microwaves caused significant ($p < 0.01$) increased single-strand DNA breakage, and double-strand breakage, CW, $p < 0.05$ and pulsed, $p < 0.01$), Figure 28.

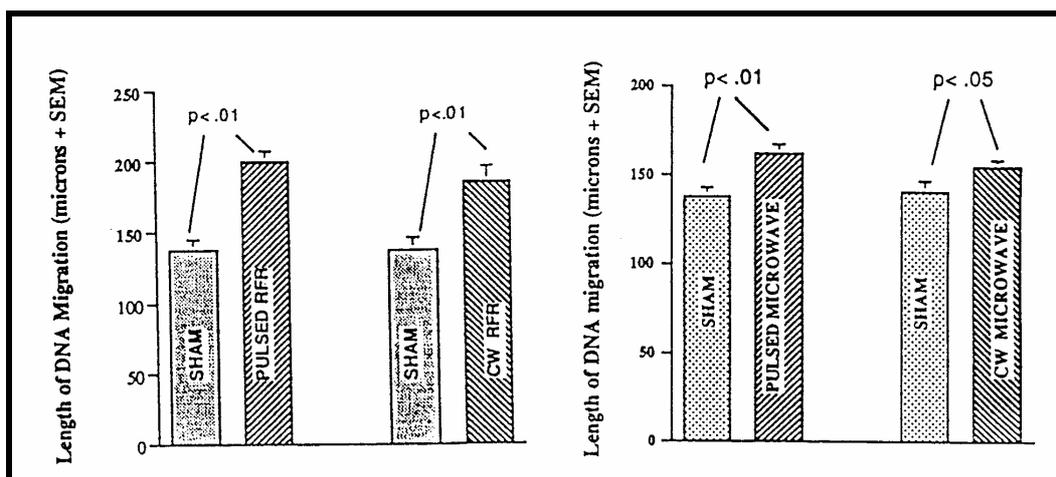


Figure 28: Single-strand (left) and double-strand (right) breaks in brain cells of rat after exposure to pulsed or continuous-wave RFR. Each bar represents data from 8 rats, Lai and Singh (1996).

This shows that both continuous and pulsed microwaves cause single and double DNA strand breakage, but pulsed microwaves cause more than continuous waves. Hence pulsed cell phone signals and radar signals cause significant DNA damage. This has been confirmed for radar causing significantly increased chromosome aberrations and for cell phones significantly damaging DNA, Phillips et al. (1998).

In the mean time Lai and Singh (1997) investigated the mechanism which is involved with this genotoxic effect of RF/MW radiation. They treated the microwave exposed rats with melatonin and a spin-trap compound (PBN) to determine the role of free radicals. They showed that both melatonin and PBN eliminated the microwave induced DNA damage. Figure 29 shows the effect of melatonin for single- and double-strand DNA breaks and Figure 30 the same for PBN.

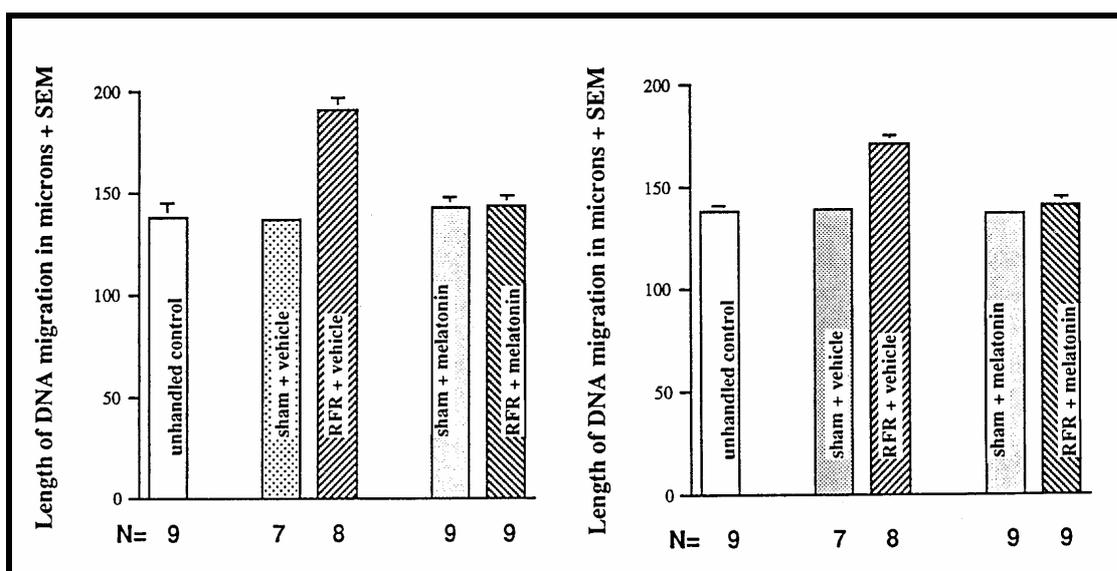


Figure 29: Effect of treatment with melatonin for RFR-induced increase in DNA single-strand (left) and double-strand (right) breaks in rats brain cells. Data was analysed using the one-way ANOVA, which showed a significant treatment effect ($p < 0.001$) for both cases. "vehicle" involves injecting with the physiological saline without the active substance. Lai and Singh (1997)

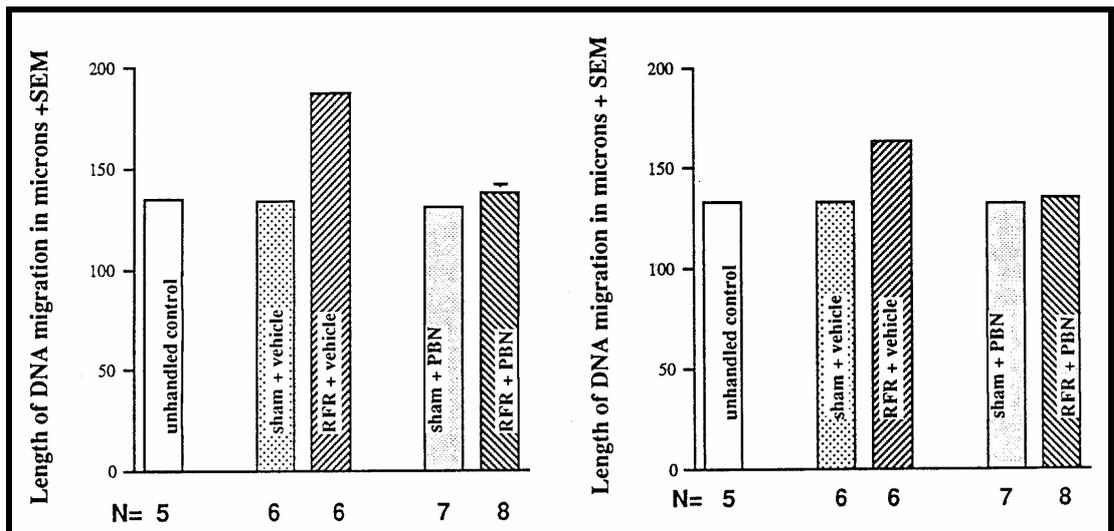


Figure 30: Effect of treatment with PBN for RFR-induced increase in DNA single-strand (left) and double-strand (right) breaks in rats brain cells. Data was analysed using the one-way ANOVA, which showed a significant treatment effect ($p < 0.001$) for both cases. "vehicle" involves injecting with the physiological saline without the active substance. Lai and Singh (1997).

Lai and Singh (1997) conclude that if free radicals are involved in the RFR-induced DNA strand breaks in brain cells, the results of their study could have an important implication of the health effects of RFR exposure. Involvement of free radicals in human diseases, such as cancer and atherosclerosis has been associated. Free radicals also play an important role in aging processes, Reiter, (1995). They also point out that both melatonin and PBN can have other actions on cells in the brain that can decrease DNA damage. Therefore further support is necessary to interpret these results.

Phelan et al. (1992) exposed B-16 melanoma cell line to pulsed 2.45 GHz, 100 pps, 1hr exposure SAR = 0.2 W/kg. This resulted in changes of membrane ordering. Their data indicated that a significant, specific alteration of the cell-membrane ordering followed microwave exposure and that the alteration was due at least part, to the generation of oxygen radicals. Hence there is independent support for the generation of free radicals by microwaves, as well as the Lai/Singh evidence that PBN and Melatonin reduce the RFR induced DNA damage.

Two other laboratories have recorded RF/MW produced significant DNA strand breaks. Verschave et al. (1994), who used a GSM cell phone signal to expose human and rat peripheral blood lymphocytes, found significantly increased strand breaks at high, but non-thermal exposure levels. Phillips et al. (1998) exposed Molt-4 T-lymphoblastoid cells to cell phone radiation in the SAR range 0.0024 W/kg to 0.026 W/kg for both iDEN and TDMA signals.

A 2 hr and 21 hr exposure to these low levels of cell phone radiation significantly increased ($p < 0.0001$) or decreased ($p < 0.0001$) the DNA damage. Decreased DNA damage is evidence of increased repair that is evidence of damage, Meltz (1995). This is shown in the DNA damage time sequences above. Significance at these levels is usually taken as causal because there are other independent laboratories showing the RF/MW radiation and cellphone radiation damages chromosomes, forms micronuclei and damages DNA, and the change of the result being random is less than 1 in 10,000.

Hence RF/MW radiation has been confirmed to enhance DNA damage under RF/MW exposure from radar-like and cell phone exposures, including an exposure level which is 0.22% of the ICNIRP guideline, and 0.022% of the US guideline. And since this is causally genotoxic the damage occurs cell-by-cell and the safe level of exposure is zero.

5.5.5 ELF Exposure and DNA strand breakage:

Because of the much higher coupling of fields to tissue at the higher frequencies, if chromosomes and DNA is damaged from 50/60 Hz exposures then it is much more likely to occur at RF/MW frequencies. Hence evidence that 50/60 Hz exposures damage chromosomes and DNA supports and confirms that EMR across the spectrum is genotoxic.

Four independent laboratories have also published data on ELF induced DNA strand breaks confirming that ELF EMR damages DNA strands; Lai and Singh (1997a), Svedenstal et al. (1998), Phillips et al. (1998a), and Ahuja et al. (1997). Lai and Singh (1997a) also demonstrate the involvement of free radicals and the protective effect of melatonin. With the evidence above that EMR reduces melatonin this confirms that reduced melatonin causes higher concentrations of free radicals which produce more DNA strand breaks from EMR exposure from ELF to RF/MW frequencies. Increased DNA strand breaks will result in increased chromosome aberrations.

Multiple evidence from independent laboratories established that EMR from ELF to RF/MW causes DNA single- and double-strand breaks at very low, non-thermal exposure levels. This extends and confirms the genotoxic evidence from chromosome aberration studies.

5.5.6 EMR Altered Gene Activity

There is also evidence that EMR not only can damage chromosomes and DNA strands, but it is observed to alter cellular calcium ions and the activity levels of proto oncogenes (cancer genes).

Blackman (1990) concluded that there was overwhelming evidence that EMR can alter normal calcium ion homeostasis and lead to changes in the response of biological systems to their environment. One of these changes is altered gene transcription and expression. The lowest published exposure level associated with significant EMR-induced alteration of cellular calcium ions occur is reported by Schwartz et al. (1990). It was 0.00015 W/kg in a 30 min exposure to a 240 MHz signal modulated at 16 Hz. The medium was frog hearts. This is equivalent to an exposure level of about 0.08 $\mu\text{W}/\text{cm}^2$.

Calcium ion fluxes occur in “windows” of exposure parameter combinations. Two studies associate EMR exposure alteration of gene transcription with exposure windows. Litovitz et al. (1990) identified amplitude (intensity) windows, and Wei et al. (1990) frequency windows in the range 15 to 150 Hz. They observed a peak effect in c-myc gene transcription at 45 Hz. Liburdy et al. (1993) show that c-myc induction occurs in a direct sequence from calcium ion influx. Increased c-myc gene transcripts by 50/60 Hz fields has also been observed, Goodman et al. (1989, 1992) and Lin et al. (1994). Phillips et al. (1992, 1993) observed time-dependent changes in the transcription of c-fos, c-jun, c-myc and protein kinase C, from 60 Hz exposure and a linear reduction in ras p21 expression by a 72 Hz signal. 50/60 Hz signals altered c-jun and c-fos gene expression as observed by and Lagroye and Poncy (1998) and c-fos expression by Rao and Henderson (1996) and

Campbell-Beachler et al. (1998). The ppSom gene is very important in human neurological disorders, and is regulated by calcium ions Capone, Choi and Vertifuille (1998).

Cell phone radiation (836.55 MHz) significantly altered c-jun transcript levels, Ivaschuk et al. (1997). Cell phone radiation significantly enhances the proto oncogene c-fos activity in C3H 10T 1/2 cells, from a 40 % (p=0.04) increase from a digital cell phone and a 2-fold increase (p=0.001) from an analogue cell phone, Goswami et al. (1999).

Hence proto-oncogene activity is altered and enhanced in multiple independent experiments from ELF and RF/MW exposure, including cell phone radiation.

5.5.7 Immune system impairment by EMR

Immune system impairment by an agent is evidence of carcinogenicity because one of the primary repair and body protection systems is the immune system. Hence any evidence that EMR impairs the immune system is highly relevant to cancer risk.

Impairment of the immune system is related to calcium ion efflux, Walleczek (1992) and to reduced melatonin, Reiter and Robinson (1995). Cossarizza et al. (1993) showed that ELF fields increased both the spontaneous and PHA and TPA- induced production of interleukin-1 and IL-6 in human peripheral blood. Rats exposed to microwaves showed a significant reduction in splenic activity of natural killer (NK) cells, Nakamura et al. (1997).

Dmoch and Moszczynski (1998) found that microwave exposed workers had decreased NK cells and a lower value of the T-helper/T-suppressor ratio was found. Moszczynski et al. (1999) observed increased IgG and IgA and decreased lymphocytes and T8 cells in TV signal exposed workers. Quan et al. (1992) showed that microwave heating of human breast milk highly significantly suppressed the specific immune system factors for E.Coli bacteria compared with conventional heating. Chronic, 25 year, exposure to an extremely low intensity ($<0.1\mu\text{W}/\text{cm}^2$) 156-162 MHz, 24.4 Hz pulse frequency, radar signal in Latvia produced significant alterations in the immune system factors of chronic exposed villagers, Bruvere et al. (1998).

5.6 Genotoxicity Conclusions:

There is strong and consistent evidence from multiple independent laboratories, and at very low non-thermal exposure levels, the electromagnetic radiation across the spectrum is genotoxic.

This is shown by significant and dose-response increases in chromosome aberrations, micronuclei and DNA strand breakage. It is confirmed by significantly altered oncogene activity, cell malignant transformation, stimulating Toxic Shock Proteins, reduced melatonin and impaired immune system competence resulting from exposure to electromagnetic radiation.

This is independently confirmed by hundreds of groups experiencing elevated and significantly elevated rates of cancer from EMR exposure across the spectrum, including many dose-response relationships that indicate a near zero threshold, fully consistent with EMR being genotoxic. Two of the strongest and most consistent epidemiological associations with EMR are Leukaemia/Lymphoma and Brain Tumor, including Astrocytoma.

6. Neurological evidence of EMR effects:

6.1 Early Evidence of Neurological Symptoms from chronic radar exposure:

Early and acute symptoms of neurological interactions between external EMR and brain cells is evidence of headaches, dizziness, loss of concentration, memory loss, lethargy and fatigue. These symptoms were frequently recorded from radar exposed workers and became known as the Radiofrequency Syndrome or the Microwave Syndrome.

Table 4: Neurological Symptoms per 1000 p-y, Male employees: (Lilienfeld et al. (1978) Table 6.31).

	Moscow	Comparison	RR	p-value
Depression	1.3	0.73	1.78	0.004
Migraine	1.8	0.97	1.86	
Lassitude	1.2	0.78	1.54	
Irritability	1.3	0.66	1.97	0.009
Nervous Disorders	1.5	0.64	2.34	
Difficulty in Concentrating	1.4	0.52	2.96	0.001
Memory Loss	1.6	0.50	3.20	0.008
Dizziness	1.2	0.85	1.41	
Finger Tremor	1.3	0.71	1.83	
Insomnia	1.1	0.90	1.22	
Neurosis	1.3	0.76	1.71	

These symptoms were observed in the staff of the US Embassy in Moscow, Lilienfeld et al. (1978), Table 6.31, here in Table 4. The radar intensity was measured outside the Embassy and over most of the time the maximum was $5 \mu\text{W}/\text{cm}^2$. Inside the signal strength was typically less than $0.1 \mu\text{W}/\text{cm}^2$. This is consistent with the sleep disturbance in the Schwarzenburg Swiss study, Altpeter et al. (1995). This found that significant resident sleep disturbance occurred down to RF exposure of $0.0004 \mu\text{W}/\text{cm}^2$.

Lilienfeld et al. (1978) also shows a number of increased cancers in various groups related to the U.S. Embassy in Moscow. One of these was adult dependents who had a significant increase in brain tumor, SMR = 20.0, 95%CI: 2.4-72.2, n=2.

A very large Scandanavian study of cellphone users found highly significant dose-response increases in these symptoms with both analogue and digital phone users, Mild et al. (1998), Figure 31.

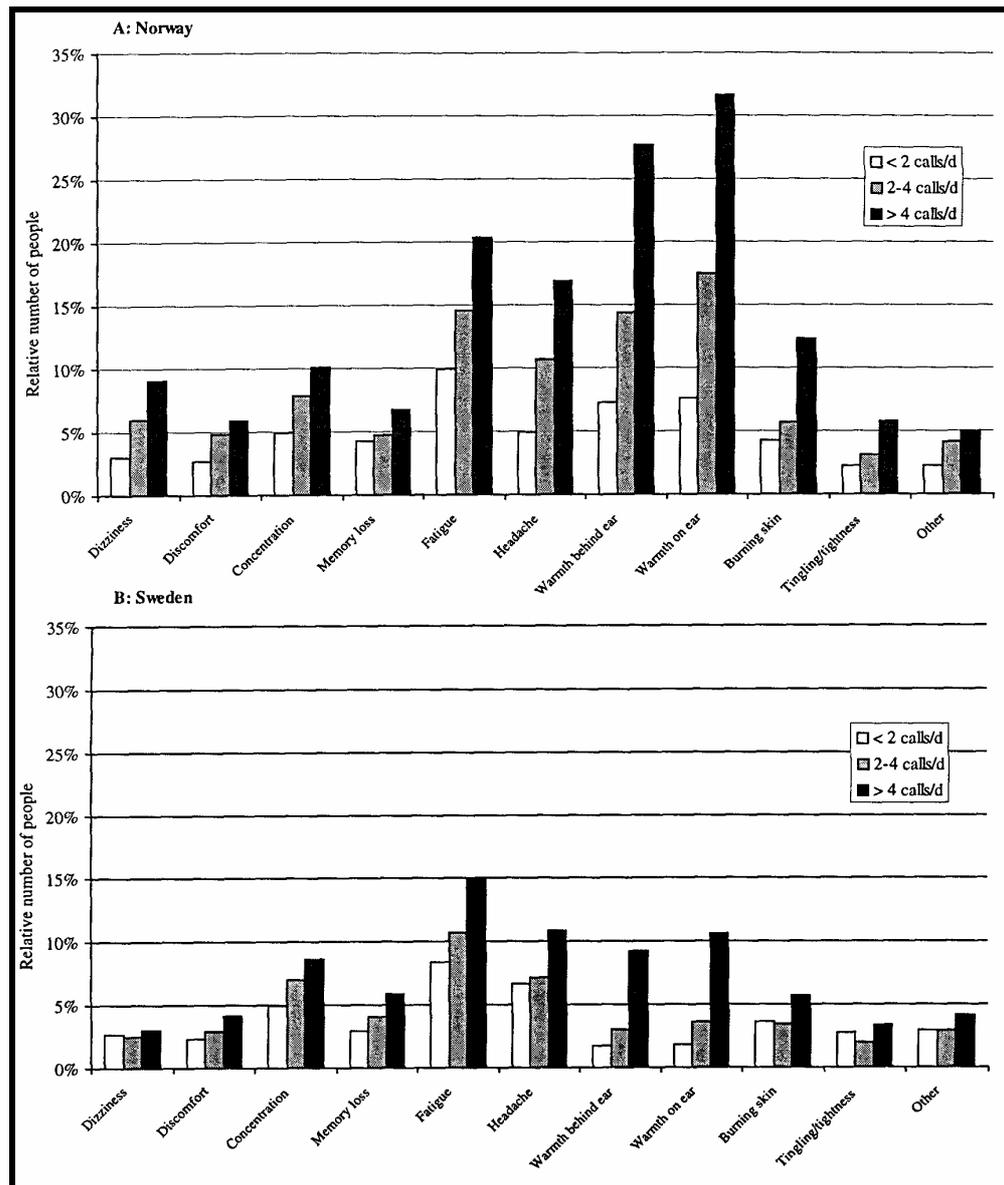


Figure 31: The prevalence of symptoms with various categories of calling times/day, A. Norway, B. Sweden, Mild et al. (1998).

With the concentration of research over recent years to the effects of cellphone radiation, we now have multiple, independent confirmation that the radar-like radiation from cellphones affects the brain electrical activity:

- Alters brain activity including EEG, Von Klitzing (1995), Mann and Roschkle (1996), Krause et al. (2000), Huber et al. (2000) and Lebedeva et al. (2000).
- Disturbs sleep, Mann and Roschkle (1996), Bordely et al. (1999).
- Alters human reaction times, Preece et al. (1999), Induced potentials, Eulitz et al. (1998), slow brain potentials, Freude et al. (1998), Response and speed of switching attention (need for car driving) significantly worse, Hladky et al. (1999). Altered reaction times and working memory function, Koivisto et al. (2000), Krause et al. (2000).
- Weakens the blood brain barrier ($p < 0.0001$): Persson, B.R.R., Salford, L.G. and Brun, A., 1997. Opening the BBB allows toxic chemicals to move into the brain.

The evidence was strong enough in 1982 for the Supreme Court of New York to award workers compensation for “Radiofrequency Sickness Syndrome” for chronic occupational microwave exposure to a technician servicing TV transmitters in the 87th floor of the Empire State Building, Yannon vs New York Telephone Co. The compensation also recognized that the chronic microwave exposure caused his death. A request to get leave to appeal was declined by the court. A primary expert witness in this case was Dr Milton Zaret. Dr Zaret's studies also show that radar exposed workers had highly significant increased Astrocytoma brain tumors, Zaret (1977).

The evidence is now very higher significantly stronger with the genotoxic and epidemiological studies.

6.2 Biological Mechanisms for Neurological Effects:

Biological mechanism for these neurological effects have been well identified. Pulsed and modulated RF/MW radiation is shown to induce efflux/influx of calcium ions and GABA from brain cells. ELF induced altered calcium ions efflux and GABA efflux was observed, Kaczmarek and Adey (1973).

GABA related neurotransmitters are changed in a dose response manner by 915 MHz microwaves, Figure 32. Altered GAMA is shown to cause all of the neurological symptoms identified above. GABA (gamma-amino butyric acid) and glutamatergic synapses make up to 60 % of the synapses in the CNS and 40 % in the brain, Kolomytkin et al. (1994).

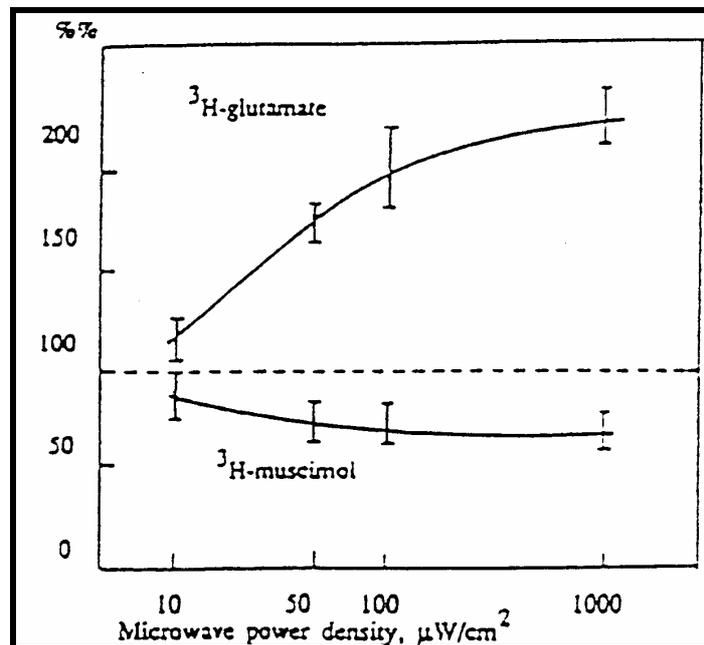


Figure 32: Exposure related alteration of GABA related molecules in rat brains exposed for 5 minutes to 915 MHz microwaves, pulsed at 16 pps. Differences from controls are still significant at $10\mu\text{W}/\text{cm}^2$. Kolomytkin et al. (1994)

Hence induced alteration of GABA in the brain can have serious consequences. Figure 30 shows that a 5 minute exposure to pulsed microwaves have a dose-response effect on GABA related receptors. This biological reaction in the brain is occurring below the exposure level of the police traffic radar of the officer in the car. Frey (1995) concludes that EMR affects the dopamine systems of the brain through its effects on GABA. He also

notes that the dopamine-opiate systems interact with the pineal melatonin/serotonin system. Hence there is a biochemical pathway to cancer.

6.3 Biophysical Mechanism:

The primary biophysical mechanism for EMR to interact with brain cells at very low levels of exposure intensity is the resonant interaction with the calcium ion currents that regulate the neurotransmitters and form part of the EEG system. Dr Carl Blackman reviewed the extensive research literature on calcium ion efflux. He was well qualified to do this since he and his group at the U.S. E.P.A. had been responsible to replicating and extending all of the research shown in other laboratories. Blackman (1990) concludes:

"Taken together, the evidence overwhelmingly indicates that electric and magnetic fields can alter normal calcium ion homeostasis and lead to changes in the response of biological systems to their environment".

Veteran EMR researcher, Dr Ross Adey considered and rejected the conclusions of a WHO review panel. He states, Adey (1975):

"Even a recent review body of the World Health Organization decided after discussion to dismiss from its concerns possible biological effects that might occur in the absence of significant heating. It has become clear, however, that interactions with the mammalian central nervous system can be reliably produced by oscillating electric and electromagnetic fields without significant heating of tissues."

Now that we are 27 years later, the evidence of significant effects at extremely low field intensities, especially in brain tissue, is overwhelmingly strong and conclusive.

6.4 Neurological human studies:

This is illustrated by the Schwarzenburg Study in Switzerland where a causal relationship between extremely low mean intensity of a shortwave radio signal and human sleep disturbance. This was causal through a measured mechanism, reduced melatonin, significant dose response relationships, and experimentation by turning the transmitter off and altering the beam angles which both resulted in altered sleep disturbance.

Figure 33 shows the significant dose-response relationship with measured mean RF exposure and the level of sleep disturbance in the rural residential population.

The lowest exposure level for Group C was 0.4 nW/cm^2 . The impact of the radio signal was firmly confirmed when the transmitter was secretly turned off for 3 days. Group C showed a significant, $p < 0.001$, improvement in sleep quality, revealing that 25 % sleep disturbance is elevated by the RF exposure even at 0.4 nW/cm^2 ($0.0000004 \text{ mW/cm}^2$). This is a proven RF/MW exposures cause a significant neurological effect at millions of times lower than the radar exposure of the policeman in this case.

Sleep disturbance led to many other significant increased health effects including chronic fatigue, anxiety, aches and pains, head aches, vertigo, altered heart beat, cough and sputum, Altpeter et al. (1995). These effects are related to reduced melatonin, which is also related to cancer.

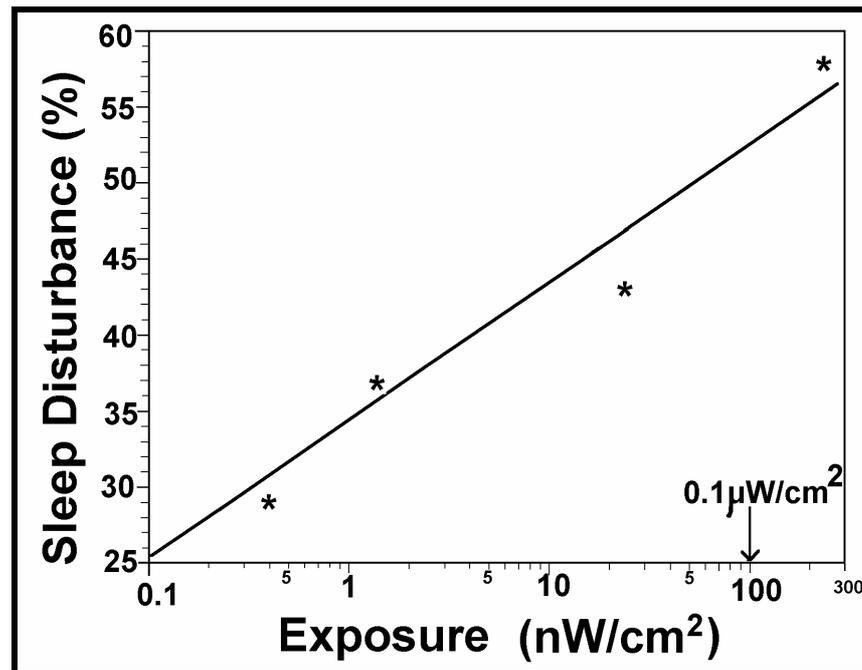


Figure 33: Dose-response relationship for Sleep Disturbance at Schwarzenburg with exposure in nW/cm^2 . Note: $1\text{nW}/\text{cm}^2 = 0.001\mu\text{W}/\text{cm}^2$.

Deapen and Henderson (1986) found that electrically related occupation had significantly increased amyotrophic lateral sclerosis disease, OR = 3.8, 95%CI: 1.4-13.0. Davanipour et al. also found increased amyotrophic lateral sclerosis in electrical occupations for the 75th percentile and total exposure OR = 7.5, 95%CI: 1.4-38.1, and average exposure OR = 5.5, 95%CI: 1.3-22.5.

Savitz, Checkoway and Loomis (1998) also found that long-term electric utility workers in the United States had significantly higher amyotrophic lateral sclerosis, OR = 3.0, 95%CI: 1.0-9.2. In a large study of Danish utility workers, Johansen (2000) found dose-response increases in Senile dementia, Pre-senility, and Epilepsy. These studies confirm that electromagnetic fields and radiation cause significant increases of neurological diseases.

A reduced melatonin related neurological health effect is depression and suicide. Perry et al. (1981) found a highly significant association between suicide and the exposure to magnetic fields from High Voltage Powerlines. Baris and Armstrong (1990) also found RF exposure shows a significant 53% increase in suicide or British Radio and Radar Mechanics, and 156 % increase for Telegraph radio operators.

A very large study in the United States electric utility workers, with many having their electromagnetic fields measured, identified a significant dose-response increase in suicide in relation to recent exposure to EMF, Van Wijngaarden et al. (2000), Figure 34.

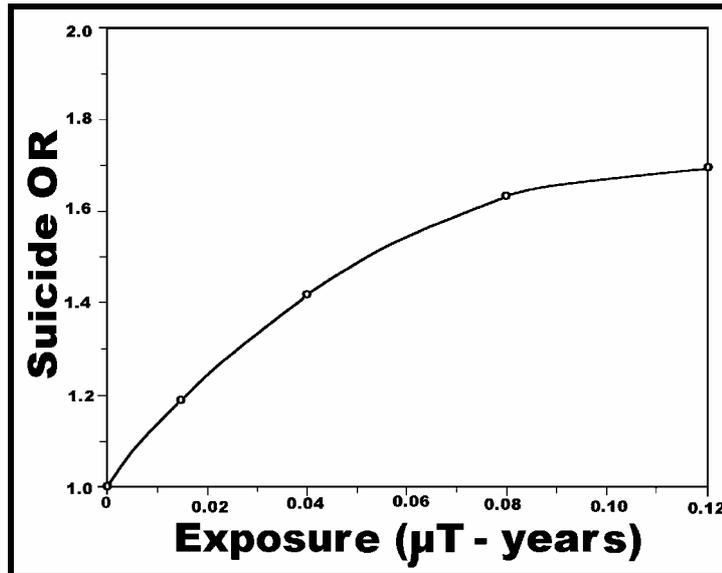


Figure 34: Dose response relationship of Suicide after recent monitored exposure to cumulative 50 Hz magnetic fields for men <50 years, adjusted for work, class, location and exposure to sunlight and solvents, Van Wijngaarden et al. (2000).

Hence, there is strong evidence that ELF and RF/MW, including radar and cellphone exposure, produces significant increases of neurological effects, diseases and death.

7. Epidemiology of RF cancer:

Residents, workers and military personnel who are exposed to an RF signal, usually experience whole body exposure. Their body acts as an aerial and induced fields produce currents that flow to earth through people's feet. Hence there is no single organ that is more exposed than any other and being genotoxic, RF radiation is predicted to cause cancers across many body organs and sites. This is confirmed by multiple studies, Table 5.

Because the whole body is exposed to a damaging toxic substance the whole body organs, the blood, bone marrow and lymph system are particularly vulnerable and leukaemia and lymphoma are predicted to result. This is confirmed by multiple studies, Table 6. The studies have been ranked by mean exposure levels and Relative Risk shows a global dose-response relationship.

Since brains are electromagnetically active they are especially sensitive to interference and genetic damage from external electromagnetic signals. The very slow rate of cell replacement means that compared to other body organs, there is limited repair mechanisms. The extensive number of brain tumor epidemiological studies associated with ELF/RF/MW exposure is set out in tabular form in section 8.

Table 5: Summary of all site cancers from Robinette et al. (1980), using AT/ET except for Brain cancer (FT/ET), Milham (1985), Szmigielski (1996) and for Dolk (1997a,b) using the maximum and/or significant result in the radial patterns.

Exposure Regime	Robinette RF/MW High	Milham Mixed Mod. PMR	Szmigielski RF/MW High	Dolk(a) RF/MW Low	Dolk(b) RF/MW Low
Relationship	RR		RR	O/E	O/E
Sample Size(N)	202	2649	55,500	17409	13372
Symptoms					
All Malignant Neoplasms	1.66*	106**	2.07*	1.20*	
Esophageal and Stomach			3.24**		
Respiratory Tract, Lung	1.75	114**	1.06		
Colorectal/ bladder (1)			3.19**	1.36/1.76	1.10
Liver, pancreas		117*	1.47		
Skin, Melanoma	2.66		1.67*	2.39*	1.11
Thyroid			1.54		
Brain, CNS (2)	2.39	143**	1.91*	1.31	1.06
Leukaemia	2.22*	136*	6.31***	1.74*	1.15
Non-Hodgkins Lymphoma		164**	5.82***	1.30*	
Acute Leukaemia (Lympho)		162**	5.75*	3.57	1.04
Acute Myeloblastic Leuk.			8.62***	1.02	1.17
Chronic Myelocytic Leuk.			13.90***	1.23	
Chronic Lymphoblastic Leuk			3.68**	2.56*	1.20

p-values: * <0.05; ** <0.01; *** <0.001

Note (1): Colorectal for Szmigielski and the left Dolk(a) and bladder for the right Dolk(a) and Dolk(b).

Note (2): In Milham 16 of the unspecified neoplasms were brain tumors that have been added to this group.

Table 6: A summary of epidemiological studies involving adult leukaemia mortality or incidence, ranked by probable RF/MW exposure category.

Study	Reference	Exposure Category	Leukaemia Type	Risk Ratio	95% Confidence Interval
Polish Military (Mortality)	Szmigielski (1996)	High	ALL	5.75	1.22-18.16
			CML	13.90	6.72-22.12
			CLL	3.68	1.45-5.18
			AML	8.62	3.54-13.67
			All Leuk.	6.31	3.12-14.32
Radio and TV Repairmen	Milham (1985)	Moderate	Acute Leuk. Leuk.	3.44 1.76	
Amateur Radio (Mortality)	Milham (1988)	Moderate	AML	1.79	1.03-2.85
North Sydney TV/FM towers (Mortality)	Hocking et al.(1996)	Low	All Leuk.	1.17	0.96-1.43
			ALL+CLL	1.39	1.00-1.92
			AML+CML	1.01	0.82-1.24
			Other Leuk	1.57	1.01-2.46
UK TV/FM (Incidence)	Dolk et al. (1997a)	Low	Adult Leuk.	1.03	1.00-1.07

Note: ALL : Acute Lymphatic Leukemia; CLL: Chronic Lymphatic Leukaemia; AML Acute Myeloid Leukaemia; CML: Chronic Myeloid Leukaemia; and All Leuk.: All Adult Leukaemia.

8. Epidemiology of Brain Tumour:

8.1 Context:

WHO staff and ICNIRP members, such as Dr Michael Repacholi, along with most western EMR authorities, including the FCC, ANSI and IEEE, make the assumption that the only established effect of EMR exposure is tissue heating. Hence standards are based on avoiding measurable body temperature rises. This report shows conclusive evidence that EMR is genotoxic with a safe exposure level of zero. This is confirmed by many studies showing increases, significant increases and dose-response increases of cancer for people exposed to EMR/EMF. Other scientists, including epidemiologists, have come to similar conclusions with even less evidence than is presented here.

Eminent academic epidemiologist late Dr John Goldsmith, reviewed the research of RF/MW health effects. He concludes, Goldsmith (1995):

“There are strong political and economic reasons for wanting there to be no health effect of RF/MW exposure, just as there are strong public health reasons for more accurately portraying the risks. Those of us who intend to speak for public health must be ready for opposition that is nominally but not truly, scientific. At present there seems to be little interest in or understanding of epidemiologic information among regulatory bodies that should provide protection.”

Goldsmith (1997):

“Available data suggest that RF radiation be considered a carcinogenic risk, a position already taken in an internal U.S. E.P.A. document in 1990 when there was much less evidence of the potential harmfulness of RF radiation.”

The evidence in this report confirms and significantly strengthens Dr Goldsmith's conclusions.

Dr Goldsmith had not seen the evidence of genotoxicity except the blood tests from the US Embassy in Moscow and the early Lai and Singh paper. He had seen some of the cancer studies. They are cited in Goldsmith (1997). However there is much more now.

8.2 Brain Tumor Epidemiological studies:

For brain tumor alone the epidemiological studies showing elevated, Table 7, significantly elevated, Table 8 and dose-response elevated, Table 9, brain tumors in human populations exposed to EMR across the spectrum, including microwaves and radar.

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|---|----------------------------------|-------------------------------|
| ELF fields | OR = 1.28 (0.95-1.74) | |
| • Germany, Meta Analysis | | Meinert and Michaelis (1996) |
| | OR = 1.5 (0.69-3.26) | |
| • North Sydney, FM/TV, residential
Childhood incidence | RR = 1.10 (0.59-2.06) | Hocking et al. (1996)
n=64 |
| • Sweden Powerlines | | Feychting et al. (1997) |
| Residents CNS tumor | RR = 1.3 (0.3-4.8) | n=3 |
| Astrocytoma III - IV | RR = 2.2 (0.6-8.5) | n=3 |
| • United Kingdom, Sutton Coldfield FM/TV tower
Residents 0-2 km from the tower | O/E = 1.31 (0.75-2.29) | Dolk et al. (1997a) |
| • United Kingdom, 20 Regional FM/TV towers
Residents 0-10km from the towers | O/E = 1.03 (0.93-1.20) | Dolk et al. (1997b) |
| • United States, Commercial aircrew
ELF/RF | Increased brain tumour mortality | Nicholas et al. (1998) |
| • New Zealand, Residential Fields
Childhood CNS Tumors.>0.2μT | OR = 1.6 (0.4-7.1) | Dockerty et al. (1998) |
| • Sweden | | Rodvall et al. (1998) |
| Electrical Occupations | meningioma RR = 1.8 (0.3-3.6) | n=20 |
| Measured > 0.4 μT | glioma RR = 1.9 (0.8-5.0) | |
| | meningioma RR = 1.6 (0.3-10.2) | |
| • Sweden, Cell phone users | | Hardell et al. (1999) |
| | left side, OR = 2.40 (0.52-10.9) | |
| | right side OR = 2.45 (0.78-7.76) | n = 206 |
| • United States Utility Workers
5 studies combined
>10 μT-years | RR = 1.12 (0.98-1.28) | Kheifets et al. (1999) |
| • United States, Motorola Study | | Morgan et al. (2000) |
| High Exposure | RR = 1.07 (0.32-2.66) n = 3 | |
| Moderate Exposure | RR = 1.18 (0.36-2.92) n = 3 | |
| High/Mod vs Low | RR = 1.13 (0.49-2.31) n = 6 | |

Definitions: RR is the Risk Ratio, OR the Odds Ratio, SMR the Standardized Mortality Ratio, PMR the Proportionate Mortality Ratio, SIR the Standardized Incidence Ratio, MOR the Mortality Odds Ratio, O/E the observed vs expected ratio, PPR the Proportionate Probable Ratio, N is the total population sample size and n is the exposed sample size.

Table 8: Significant Increases in Brain Tumor from EMR exposure:

- United States: Microwave repair workers
Astrocytoma brain tumors RR = 74.1 (15.0-367), p<0.0001 Zarek (1977)
n=2
- United States Embassy in Moscow:
Radar RF/MW exposure Lilienfeld et al. (1978)
Males Working in the Embassy SMR = 20 (2.4-72.2) , p<0.01 n=2
- Navy, Korean War, FT/ET groups, RF/MW
For brain, eye and CNS RR = 1.66 (1.06-2.60) Robinette et al. (1980)
n=8
- Washington State, Electrical Workers Milham (1985)
 - All Groups PMR = 123, p<0.05) n=101
 - Electrical and electronic technicians PMR = 134 n=7
 - Power Station Operators PMR = 130 n=3
 - Electricians PMR = 155 p<0.01) n=46
- Maryland, U.S. electrical industries
Brain tumours Died significantly earlier Lin et al. (1985)
n=951
- Sweden, Power lines, Children
CNS tumour RR = 3.9, p<0.05 Tomenius (1986)
- Denver, Residential, children
(1988) Savitz et al.
2-level wire code OR = 2.04 (1.11-3.76)
- New Zealand, electrical workers Pearce, Reif and Fraser (1989)
 - Radio and TV repair OR = 7.86 (2.2-28.1) n=2
 - Electricians OR = 1.68 (0.75-3.79) n=6
 - Total electrical workers OR = 1.62 (1.04-2.52) n=21
- U.S., prenatal electric blanket
Brain cancer in children Savitz, John and Kleckner (1990)
OR = 2.5 (1.1-5.5)
- United States, 16 States (Mortality) Loomis and Savitz (1990)
 - Electrical engineers and technicians OR = 2.7 (2.1-3.4)
 - Telephone workers, OR = 1.6 (1.1-2.4)
 - Electric power workers OR = 1.7 (1.1-2.7)
 - Electrical workers in
manufacturing industries OR = 2.1 (1.3-3.4)
- Finland, Occupational exposures Juutilainen, Laara and Pukkala (1990)
 - CNS tumour, 25-64 year old male
 - Probable exposure RR = 1.3 (0.7-2.3)
 - Possible exposure RR = 1.3 (1.0-1.6)
- Aerospace electromechanical workers Park et al. (1990)
 - All workers PMR = 4.2 (p<0.0001) n=583
 - For hourly workers with up to

20 years of work

PMR = 8.7 (p=0.000003).

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| <ul style="list-style-type: none"> • Germany, electrical workers <li style="padding-left: 20px;">Women <li style="padding-left: 20px;">Men | <ul style="list-style-type: none"> RR = 5.2 (1.4-20.1) RR = 0.9 (0.2-2.3) | Schlehofer et al. (1990) |
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| <ul style="list-style-type: none"> • Canada, Commercial pilots <li style="padding-left: 20px;">ELF/RF | <ul style="list-style-type: none"> SMR = 4.17 (1.4-9.5), p=0.017 SIR = 3.45 (1.2-7.9), p=0.03 | Band et al. (1990) |
| | | n=4
n=4 |

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| <ul style="list-style-type: none"> • Canada, British Colombia pilots <li style="padding-left: 20px;">Elevated PMRs for brain cancer and nervous system cancer. | Salisbury et al. (1991) |
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| <ul style="list-style-type: none"> • Sweden, Occupational exposure <li style="padding-left: 20px;">Assemblers and repairmen in radio and TV industry <li style="padding-left: 40px;">All brain tumours <li style="padding-left: 40px;">Glioblastomas <li style="padding-left: 20px;">All welders <li style="padding-left: 40px;">Iron/steel industry <li style="padding-left: 40px;">For glioblastomas | <ul style="list-style-type: none"> SMR=2.9, (1.2-5.9) SMR=3.4, (1.1-8.0) SMR=1.3, (1.0-1.7) SMR=3.2, (1.0-7.4) SMR=1.5 (1.1-2.1). | Tornqvist et al. (1991) |
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| <ul style="list-style-type: none"> • England, British Airways pilots <li style="padding-left: 20px;">ELF/RF | <ul style="list-style-type: none"> PMR = 2.68, p<0.05 | Irvine and Davies (1992) |
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| <ul style="list-style-type: none"> • Norway, Occupational electric and magnetic exposures. | Tynes, Andersen and Langmark (1992) |
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| <ul style="list-style-type: none"> • Finland, power lines <li style="padding-left: 20px;">CNS tumours in boys <li style="padding-left: 40px;">>0.2µT or > 0.4µT-years | <ul style="list-style-type: none"> SIR = 4.2, (1.4-9.9) | Verkasalo et al. (1993) |
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| <ul style="list-style-type: none"> • Denmark, High voltage installations <li style="padding-left: 20px;">Children, >0.4µT | <ul style="list-style-type: none"> Olsen et al. (1993) Significantly increased cancer, including CNS tumours. |
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| <ul style="list-style-type: none"> • Denver, Residence <li style="padding-left: 20px;">Children, Wire Code | <ul style="list-style-type: none"> OR = 2.5 (1.1-5.5) | Savitz and Kaune (1993) |
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| <ul style="list-style-type: none"> • New Zealand, electrical workers <li style="padding-left: 20px;">Electricians <li style="padding-left: 20px;">Electrical engineers | <ul style="list-style-type: none"> OR = 4.6 (1.7-12.2) OR = 8.2 (2.0 - 34) | Preston-Martin et al. (1993) |
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| <ul style="list-style-type: none"> • Sweden, Occupational exposure <li style="padding-left: 20px;">Low exposure <li style="padding-left: 20px;">Moderate Exposure <li style="padding-left: 20px;">High Exposure | <ul style="list-style-type: none"> RR = 1.0 (0.7-1.6) RR = 1.5 (1.0-2.2) RR = 1.4 (0.9-2.1) | <ul style="list-style-type: none"> Floderus et al. (1993) n=261 |
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| <ul style="list-style-type: none"> • U.S. Meta Analysis of 13 studies | Washburn et al. (1994) |
|--|------------------------|

Residential Children, CNS	RR = 1.89 (1.34-2.67)	
Overall brain tumour	SIR = 1.09 (0.9-1.41)	n=77
ISCO code weak magnetic/ electric exposure	SIR = 2.20 (1.01-4.18)	
Tram drivers	SIR = 2.04 (0.42-5.98)	
Radio/telegraph operators	SIR = 1.20 (0.25-3.49)	
Electricians, installation	SIR = 1.23 (0.67-2.07)	
Electricians, power supply	SIR = 1.16 (0.43-2.53)	
Power plant operators	SIR = 1.15 (0.24-3.36)	
Power line workers	SIR = 1.51 (0.78-2.64)	
Telephone installers	SIR = 1.22 (0.49-2.51)	
Railway track walkers	SIR = 2.20 (1.1-4.18)	
Total for these groups	SIR = 1.14, (0.9-1.42)	
• United States, Telephone industry Women from 24 states	OR = 2.1 (1.2-3.7)	Dosemeci and Blair (1994)
• England, Electrical workers	PPR = 118 (103-136)	Fear et al. (1996)
• France electrical utility workers Brain tumour 5 year latency	OR = 3.06 (1.08-8.74) OR = 3.69 (1.10-12.43)	Guenel et al. (1996) n=69
• Polish Military Mortality	RR=1.9 (1.08-3.47), p<0.05	Szmigielski (1996) n=55,500
• U.S. Air Force Aircrew compared with non-flying crew Adjusted for rank, socioecom.	OR = 1.77(1.17-2.68) OR = 1.22 (0.76-1.95)	Grayson and Lyons (1996) n=37
• U.S. Air Force personnel Age-race-adjusted odds ratios Senior Rank Ionizing radiation ELF fields RF/MW fields	OR = 2.11 (1.48-3.01) OR = 0.58 (0.22-1.52) OR = 1.28 (0.95-1.74) OR = 1.39 (1.01-1.90)	Grayson (1996)
• Brazil, San Paulo, Utility workers	PCMR = 7.7 (1.02-9.65)	Mattos and Koifman (1996)
• Danish Utility Workers Women Women >10yrs < 0.09 μ T 0.1-0.29 μ T	SIR = 1.3 (0.7-2.2) SIR = 1.9 SIR = 9.2 p<0.05	Johansen and Olsen (1998) n=15 n=4 n=2

- United States, Occupational CNS cancer Cocco, Heineman and Dosemeci (1999)
RR=1.2 to 1.3 p<0.05 n =12980
- Brazilian Naval Personnel Santana, Silva and Loomis (1999)
Aged <56 yrs OR = 4.63 (2.54-8.45) n=40
Unmarried men OR = 3.18 (1.69-5.99)
- United States Utility Workers Savitz et al. (2000)
Highest exposure categories:
Career exposure (2yr lag) Cumulative RR = 1.8 (0.7-4.7)
Career exposure (2yr lag) Average RR = 2.5 (1.0-6.3)*
Cumulative exposure (2-10 years) RR = 2.62 (1.2-6.0)*
Average exposure (2-10 years) RR = 2.38 (1.1-5.0)*
Cumulative exposure (10-20 years) RR = 1.58 (0.7-3.7)
Average exposure (10-20 years) RR = 2.25 (0.9-5.4)
- Multivariate Analysis of therapeutic Exposures and cellphone use, Sweden Hardell et al. (2000)

For cellphone use and head exposure position OR = 2.62 (1.09-6.71)

Table 9: Studies showing dose-response relationships for EMR exposure and brain tumor:

- Denver, United States, power lines Wertheimer and Leeper (1979)
Childhood) Birth Address RR = 1.83, p=0.04 n=22
CNS tumors) Death Address RR = 1.76, p=0.017 n=30
Dose related for children living at same address.
- Electrical Occupations in Maryland, U.S. Lin et al. (1987)
Glioma/Astrocytoma Other Brain Tumors
Definite Exposure 2.15 (1.10-4.06) n = 27 1.54 (0.68-3.38) n = 15
Probable exposure 1.95 (0.94-3.91) n = 21 1.30 (0.60-2.78) n = 19
Possible exposure 1.44 (1.00-1.95) n =128 0.94 (0.68-1.31) n = 87
No exposure 1.0 n =323 1.0 n =286
Trend p<0.01 Trend p<0.05
- Eastern U.S. Electronic Industries Thomas et al. (1987)
Astrocytic brain tumours RR=4.9 (1.9-13.2)
Duration Employed (yr)
Unexposed <5 5-19 ≥ 20
RR 1.0 3.3 7.6 10.4
Solder fume adjusted RR 1.0 1.65 3.8 5.2
(trend p<0.05)
- East Texas, Males Glioma Speers, Dobbins and Miller (1988)
n=202
Transportation, communication and utilities industries OR = 2.26 (1.18-4.32)
Electricity or electromagnetic fields OR = 3.94 (1.52-10.20) Trend: p<0.01

- | | | |
|---|--------------------------------------|-------------------|
| <ul style="list-style-type: none"> • Los Angeles County, Occupational exposure
High exposure to electric and magnetic fields | Preston-Martin et al.(1989)
n=272 | |
| Glioma | OR=1.8 (0.8-4.3) | p for trend =0.05 |
| Astrocytoma, >5 years empl. | OR=4.4 (1.2-15.6) | |

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|--|-----------------------------|---------------|
| <ul style="list-style-type: none"> • Los Angeles Country, electrical industry | Mack et al. (1991)
n=272 | |
| Astrocytomas | RR = 10.3 (1.3-80.8) | trend, p=0.01 |

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| <ul style="list-style-type: none"> • San Francisco, Sutra Tower (FM/TV)
Children < 21 yrs
Comparing <4.5km and >4.5 km | Selvin et al. (1992)
n=35
Trend p <0.001 | |
| | RR = 2.87, (1.30-6.32), p<0.01 | |

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| <ul style="list-style-type: none"> • Canada, Provincial Residential Electric Consumption (REC)
Childhood brain cancer significantly increases with REC in a
dose-response manner. | Kraut et al. (1994) |
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| <ul style="list-style-type: none"> • U.S. Electrical Workers
Mortality Dose-response | Savitiz and Loomis (1995) |
| | OR = 1.94 per μ T-yr |

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| <ul style="list-style-type: none"> • U.S. Computer exposures
Computer Programmers (>10 yrs)
Engineering/Technical (>10 yrs)
Glioma, All subjects, 5yr progrm. | Beall et al. (1996)
Trend p = 0.04
Trend p = 0.07
Trend p = 0.08 |
| | OR = 2.8 (1.1-7.0) |
| | OR = 1.7 (1.0-3.0) |
| | OR = 3.9 (1.2-12.4) |

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|--|---|
| <ul style="list-style-type: none"> • United States, office workers
Transformer fields | Milham (1996)
N=410
Exposure trend p=0.0034
Employment period trend p<0.05 |
| | SIR = 389 (156-801) |

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| <ul style="list-style-type: none"> • Ontario Hydro male employees (Adjusted ORs)
Brain Tumour Mod. Field
 High Field
Benign Brain Mod. Field
Tumour High Field | Miller at al. (1996)

Both show trends. |
| | OR = 1.27 (0.32-5.41) |
| | OR = 1.33 (0.52 -10.8) |
| | OR = 5.38 (0.42-69.3) |
| | OR = 5.64 (0.3-105) |

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| <ul style="list-style-type: none"> • French electric utility workers
Allowing for a 10 year latency, V/m-yrs | Guenel et al. (1996) |
| | <166 OR = 1.0 n = 22 |
| | 166-229 OR = 1.67 (0.67-4.19) n = 14 |
| | 230-294 OR = 1.79 (0.60-5.36) n = 9 |
| | >294 OR = 2.15 (0.63-7.26) n = 7 |

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|---|--|
| <ul style="list-style-type: none"> • Norway
Children | Tynes and Haldorsen (1997)
n=10
p=0.07 |
| RR = | <0.05 μ T 0.05-<0.14 μ T >0.14 μ T |
| | 1.0 2.6 (0.5-12.0) 2.3 (0.8-6.6) |

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|--|-----------------------|
| <ul style="list-style-type: none"> • United States Electric Utility Workers
Reference | Savitiz et al. (2000) |
| | OR = 1.0 |

Middle exposure OR = 1.8 (0.7-4.7)
 High exposure OR = 2.5 (1.0-6.3) Trend p<0.05

- Cell phone users in Denmark Johansen et al. (2001)

Duration of digital subscription	<1 yr	1-2yrs	≥3 yrs
Relative to reference group SIR	0.7	0.9	1.2
Relative to <1 yr group RR	1.0	1.29	1.71

8.3 Brain Tumor Summary:

The US electrical utility workers who were studied by Professor David Savitz's group had a dose-response increase in cardiac death, suicide and brain tumor. Figure 35 shows the brain tumor dose-response with a threshold for RR=1.0 of zero exposure.

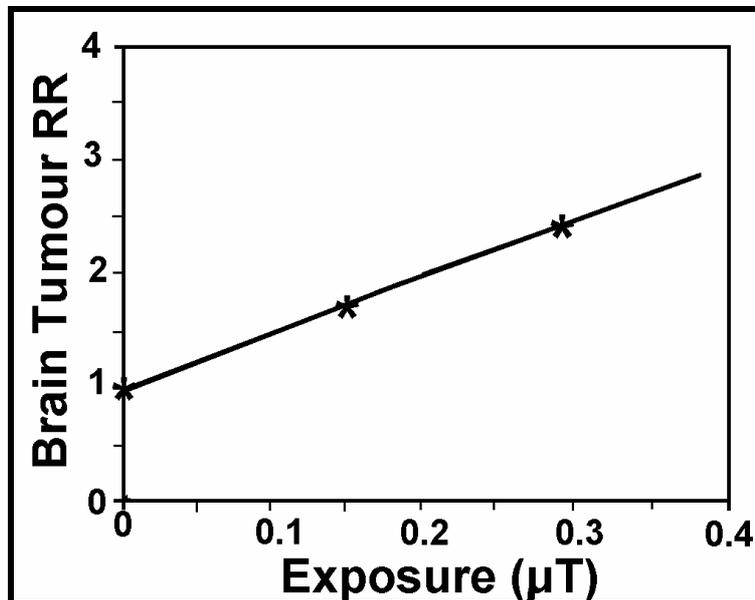


Figure 35: U.S. electric utility workers dose-response increase in brain tumours, Savitz et al. (2000).

The epidemiological studies on EMR associated brain cancer include over 60 studies of over 110 separate groups with elevated brain cancer from EMR exposure from across the spectrum. Of the 119 groups, 85 show significant increases, 16 show dose-response relationships and 10 of these are statistically significant. This evidence is more than sufficient, using the Bradford-Hill guidance, Hill (1965), to conclude that there is a causal relationship between EMR exposure across the spectrum and brain tumour incidence and mortality. Six studies specifically identify Astrocytoma from EMR exposure.

9. Cellphone radiation evidence:

Because cellphone radiation is similar to radar signals it is appropriate to consider cellphone genotoxic and brain tumor evidence.

The cellphone brain tumor study provided, Muscat et al. (2000) reports an elevated neuroepitheliomatous cancer, OR = 2.1, 95%CI: 0.9-4.7, n=9. This is reported by the director of the WTR program that funded this study, Dr George Carlo (Carlo and Schram (2001)) to originally have been significantly elevated at OR = 2.8.

Johansen et al. (2001) showed a dose-response increase in brain cancer associated with GSM cellphone use. Repacholi et al. (1997) showed that GSM phone microwave radiation double the lymphomas in mice. Hardell et al. (2001) showed that there was a significant increase in brain tumors for cellphone users between where the aerial was and the brain tumor site, OR = 2.62, 95%CI: 1.09-6.71. Significantly increases the incidence of eye cancer (Uveal Melanoma) was caused by cellphone use by between OR = 4.2, 95%CI: 1.2-14.5, and OR = 10.1, 95%CI: 1.1-484.4, Stang et al. (2001).

Despite the fact that the vast majority of cell phone users have been activity using cellphones from less than 5 years and that laboratory research into cellphone radiation effects is limited, significant adverse effects have already emerged. Cellphone analogue systems are similar to FM radio and digital systems are very similar to radar. Laboratory studies have shown that cellphone radiation is genotoxic though chromosome damage, micronuclei formation, DNA strand breakage, enhancement of proto oncogene activity and increases in brain tumor, eye cancer, testicular cancer cervical cancer and other cancers, especially around the head and neck. This is completely consistent with ELF and RF/MW effects shown over more than 40 years of research. It confirms that RF/MW radiation is genotoxic with a safe exposure level of zero.

The genetic and environmental diversity of the general population means that most people will not get cancer but a significant number do and will get cancer and die earlier because of their use of cellphones. This was evident as early as 1994, Rothman et al. (1996), Table 10.

Table 10: Comparative mortality rates between mobile phone users and portable phone users in the United States for 1994, Rothman et al. (1996).

Group	RR	95%CI	p-value
For Men	1.40	1.06 - 1.86	0.017
For Women	1.52	0.78 - 2.95	0.31
All People	1.38	1.07 - 1.79	0.013

Rothman et al. (1996) show that those who used the early larger mobile phone with the transmitter in a box with an aerial that exposed the whole body, had higher mortality rates that those using the small cellphone held against the head. Since this time the smaller head exposing phones have been shown in four studies to increase the incidence of brain tumors and other cancers.

10. Police radar Genotoxicity:

Some studies are not relevant for this case and they are general and do not investigate the specific use of radio or radar. Demers et al. (1992) found that a number of cancers were elevated in Tacoma fire fighters and police officers but not brain cancer. The study data set covered the period 1944-1979 before the type of radar in use in this case has been introduced. Because of the cancer registry and the growth of the services, over 90% of the mortality occurred in the 1974-89 period. Both Fire and Police officers had widespread use of radio with aerials on their vehicles not aimed at their heads. EMR was not a subject of the investigation. One of the objects of the study was to compare the use of cancer registries versus death certificates. The reference group was white males in Washington State, bringing in an element of the healthy worker effect.

The papers reviewing the overall mortality and cancer in police officers show varied results.

Vena et al. (1986) found that police officers had significantly higher malignancies, O/E= 1.27, 95CI: 1.08-1.49 and elevated brain and CNS cancer, O/E = 1.63, 95%CI: 0.52-3.79. For the 20-29 year group it is significantly elevated, O/E = 4.4. They note that brain cancer is related to some chemical exposures but it had been significantly related to exposure to electromagnetic radiation and that police officers used radios and radars.

Violanti, Vena and Petralia (1988) studied New York police officers and compared them with the average white male population. They found significantly higher than expected mortality for all cause mortality, for malignant neoplasms, SMR = 1.25, 95%CI: 1.10-1.41 and for suicide, SMR = 1.53, 95%CI: 1.0-7.29. Brain and CNS cancers were elevated, SMR = 1.40, 95%CI: 0.56-2.88. If an internal cohort has been used these rates would have been higher.

Finkelstein (1998) studied cancer in Ontario police officers and found elevated testicular cancer, SIR = 1.3, 95%CI: 0.89-1.84, n=23. This could have been the result of the use of hand-held radar. He found no elevation of brain cancer and overall cancer rates were lower than average, All cancer SIR = 0.9, 95%CI: 0.83-0.96. This is probably an example of the Healthy Worker effect and the reference group was the general Ontario population. When the overall mortality or cancer rate are less than 1.0 then this confirms that it is not a reliable comparison.

Davis and Mostofi (1993) specifically investigated testicular cancer and the use of hand-held radars by police officers. They found a highly significant ($p < 0.001$) 6.9-fold increase in testicular cancer.

The observed elevated rates in this study could partly be the response to stress. However, but it is consistent with a recently published set of studies from a large group of electric utility workers, over 2800 of whom had their personal EMR exposures measured, Van Wijngaarden et al. (2000) and Savitz et al. (1999, 2000). These studies showed significant dose-response increase in suicide, heart disease and brain tumor mortality. These are all related to melatonin reduction and show the strong link between melatonin reduction and neurological diseases, suicide and brain cancer. Hence since police officers are regular users of radios, a proportion of the elevated risks can be associated with EMR exposure.

This small and rather limited set of studies confirms that police radar radiation is genotoxic and carcinogenic. When it exposes the testes and brains it increases the incidence of testicular cancer and brain tumors.

11. Conclusions and Recommendations:

A somewhat more comprehensive review of the scientific evidence of genotoxicity, cancer, brain tumor and astrocytoma from chronic pulsed microwave exposure shows that the risk is real and significant. The safe level of exposure of this toxic agent is zero for human populations. The police officer concerned had his head regularly exposed to a pulsed radar signal from a traffic radar over an 8 year period, at intensities 23 to 167 times higher than the level that significant DNA damage has been observed. Hence he had a real and significant risk of developing a brain tumor. His astrocytoma shows that he did.

This officer's fellow police officers were at the same risk and some may still develop cancer or neurological diseases in their brains, but their genetics and broader exposure history and DNA repair capability has thus far helped to protect them.

Hence it is my expert scientific opinion that State Trouper, Wayne Dixon, received his astrocytoma from his police service that involved regularly and chronically exposing his head to a pulsed microwave signal from a traffic radar.

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