

# **Evidence in support of the *a priori* hypothesis that Electromagnetic Radiation across the spectrum is a Ubiquitous Universal Genotoxic Carcinogen.**

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## Abstract:

By October 2001 Dr Cherry had concluded that there was very strong evidence that electromagnetic radiation across the spectrum is a Ubiquitous Universal Genotoxic Carcinogen. The evidence is strong from multiple independent studies showing that from extremely low-frequency to microwave radiation signals and fields damage DNA. Epidemiological studies confirm this by showing that increased cancer in many body organs because the whole body is exposed, from residential studies from power ELF frequencies and radio frequencies causing increased cancer rates and from a large body of studies showing cardiac, reproductive and neurological health effects.

## Introduction:

Cancer rates have risen progressively and significantly over last century in developed countries, Figure 1 and 2. It is important to identify if there are any avoidable environmental factors that could significantly reduce the cancer rate if exposure can be reduced.

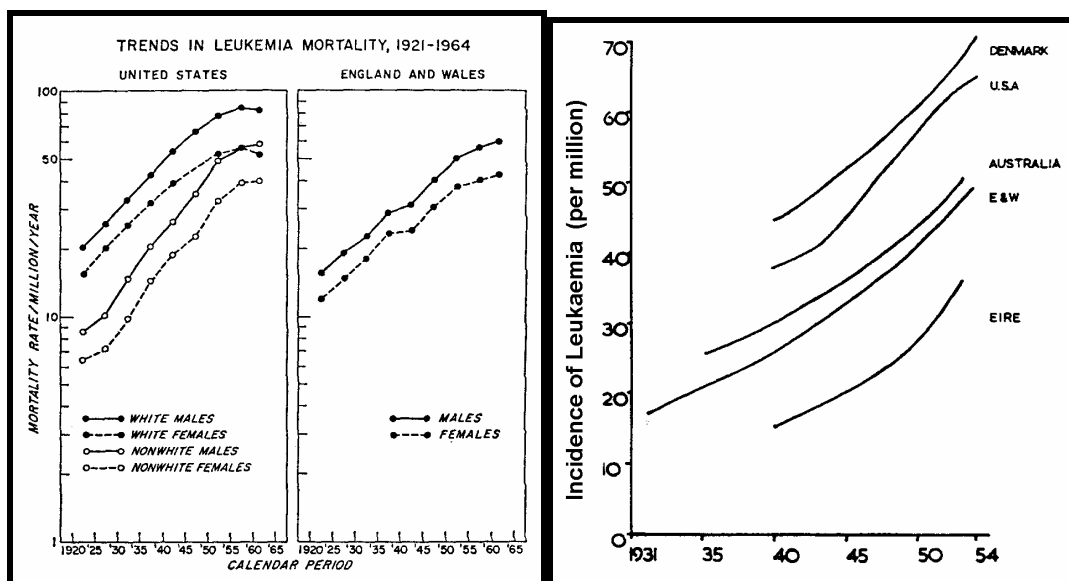


Figure 1: Historical trends in leukaemia mortality for several groups and countries, Fraumeni, and Miller (1966), left, and Burnet (1958), right.

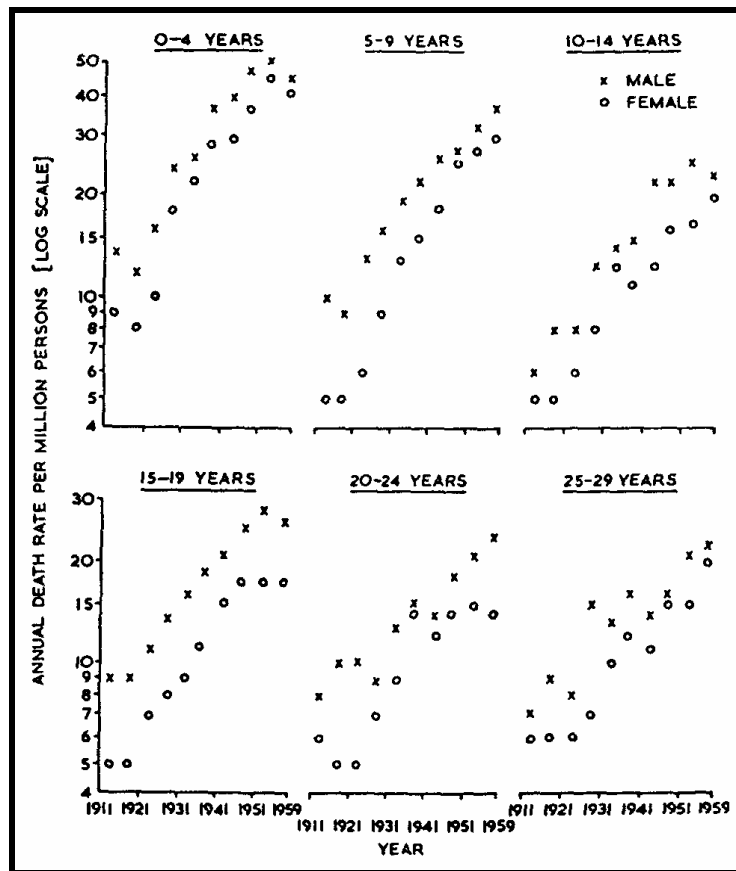


Figure 2: Trend in leukaemia mortality with time for England and Wales for 5-year age-groups by sex, from 1911-1959, Court-Brown and Hill (1961).

We now know that the dominant and primary cause of this cancer increase is the residential electromagnetic fields when houses are connected to residential electricity supplies, Kraut et al. (1994) and Milham and Ossiander (2001). They point out that the fields initiating this cancer were very low because the main early usage of electrical energy was for radio, lights and irons. Heating and cooking were still using wood and coal burning stoves for several decades. Figure 3 shows the dose-response increase in the indicative early childhood cancer, Acute Lymphoblastic Leukaemia (ALL) which was not occurring in young children with the 2-4 year age peak prior to 1900, Figure 4.

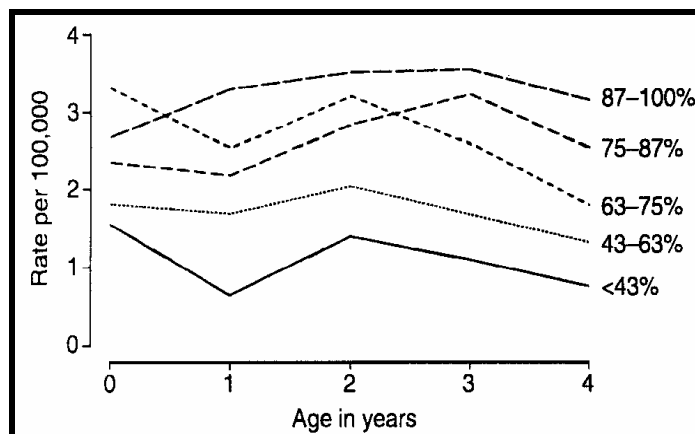


Figure 3: Childhood leukaemia mortality rates for all races 1928-32, by percent of residential electrification and age of death, Milham and Ossiander (2001).

The cancer increase of very young children from the exposure to residential electricity generated fields was independently confirmed by Kraut et al. (1994).

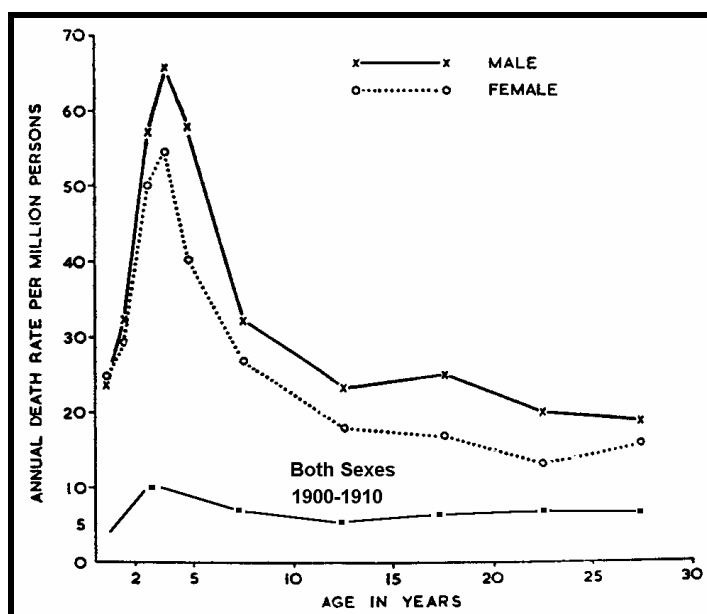


Figure 4: Age-specific death rates from leukaemia under the age of 30 years, by sex in England and Wales, 1945-1959, Court-Brown and Hill (1961).

#### Chromosome damage from ELF exposure:

The well established biological mechanism of ELF cancer causation is the genotoxicity of the ELF fields. That is, they directly damage the DNA as is shown by chromosome aberrations, micronuclei formation and DNA strand breakage.

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), and Timchenko and Ianchevskaia (1995). This currently involves 14 studies.

#### ELF Exposure and DNA strand breakage:

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. (1999a,b), Phillips et al. (1998a), and Ahuja et al. (1997, 1999). 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.

Svedenstal et al. (1999a) installed a cage of mice under some high voltage powerlines at a field strength of  $8\mu\text{T}$ . Their DNA-strand breakage was compared with a group of sham exposed mice, Figure 5.

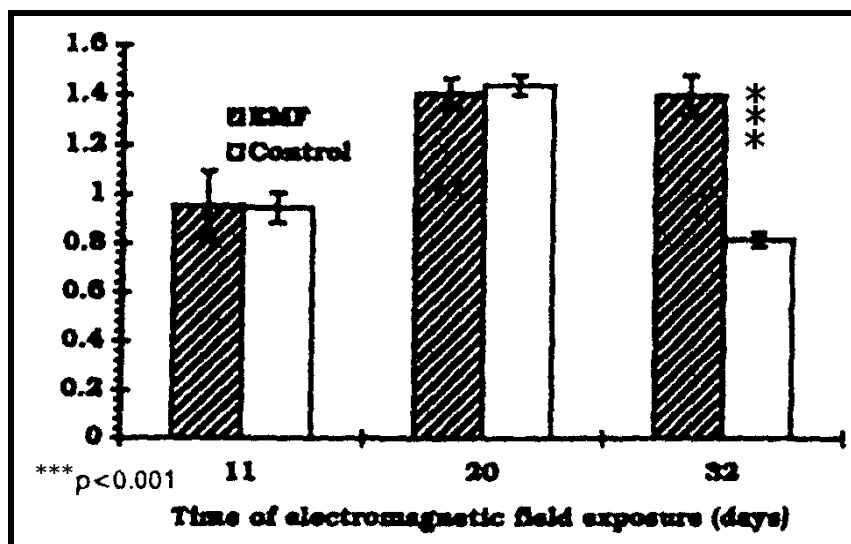


Figure 5: Comet assay assessed DNA strand breakage from the brains of mice chronically exposed to the magnetic field under a high voltage powerline, compared to a sham exposed group, Svedenstal et al. (1999a).

Figure 5 shows that after 32 days of exposure to a very low intensity,  $8\mu\text{T}$ , magnetic field there was significant ( $p < 0.001$ ) DNA-strand breakage in brain cells.

An example of a dose-response is given by Ahuja et al. (1999).

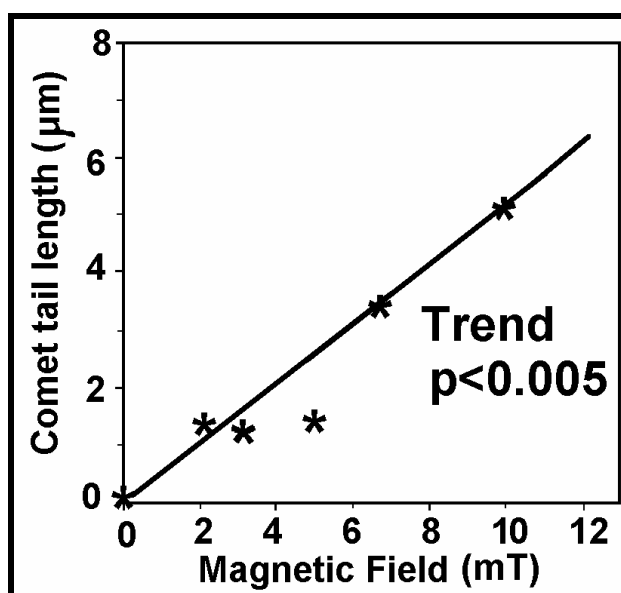


Figure 6: Comet tail length difference in before and after blood samples of peripheral blood leukocytes from human male subjects exposed to ELF magnetic fields of varying intensities for 1 hr, Ahuja et al. (1999).

A dose-response is supportive of a causal effect, Hill (1965). When backed by other studies it is confirmed to be causal.

Li and Chow (2001) showed that 50 Hz, 1.2 mT magnetic fields found that E Coli. cells:

**"that without protection of heat shock response, magnetic field exposure indeed induced DNA degradation and this deleterious effect could be diminished by the presence of an antioxidant, Trolox C. In our in vitro test, we also showed that the magnetic field could potentiate the activity of oxidant radicals."**

Thus acute high exposure and chronic low exposure to 50/60 Hz magnetic fields causes significant Chromosome Aberrations, Micronuclei Formation and DNA-strand breakage in multiple, independent laboratories. This is classically sufficient to conclude that there is a causal effect. This robustly supports the hypothesis that ELF electromagnetic fields are a Universal Genotoxic Carcinogen.

### **Other Biological EMF/EMR Mechanisms:**

Carcinogens are important toxic substances that act in at least one of several ways to enhance genetic damage of cells or to reduce the repair of cells by altering the cell-to-cell communication, Li et al. (1999), altering the cellular calcium ion homeostasis, Fanelli et al. (1999), reducing melatonin, Reiter (1994).

The cell-to-cell communication, for example through gap-junctions, is a vital part of the checking process to detect genetically damaged cells and to initiate apoptosis (programmed cell death). Enhanced cell death is associated with a total process called premature aging. Increased cancer rates also occur with age, particularly over 65 years.

The cellular ion concentration plays a central role in the cell's decision as to whether to survive to become a neoplastic cancer cell or to initiate apoptosis. Elevated cellular calcium ions favour cellular survival and cancer formation.

Melatonin is a highly potent free radical scavenger. Hence any substance that reduces the melatonin allows naturally occurring free radicals to produce more DNA damage, leading to greater cell death and cancer cell formation.

EMR induced alteration of cellular calcium ion homeostasis is a well-established effect, Blackman (1990). Melatonin reduction from EMR/EMF exposure has been observed in multiple independent studies, for example Arnetz and Berg (1996), Burch et al. (1997,1998,1999a,b, 2000), Davis (1997), Graham et al. (1994, 2000), Karasek et al. (1998), Juutilainen et al. (2000), Pfluger and Minder (1996) and Wilson et al. (1990).

### **Genotoxic Carcinogens:**

A genotoxic agent is a toxic substance that causes direct damage to the cellular DNA molecule, causes chromosome aberrations, micronuclei formation, DNA strand breakage and altered genetic activity. Because genotoxic agents damage the DNA in individual cells, producing damage cell-by-cell, there is no safe threshold. The only truly safe exposure level is zero exposure.

**Health Implications Corollary:**

A universal genotoxic substance damages cells and advances the aging process across most organs of the body, including neurological, cardiac and reproductive effects.

**Specific vs Universal Carcinogen:**

Some substances target particular body organs. For example radiative iodine is a carcinogen that becomes concentrated in the thyroid gland through natural processes, producing higher rates of thyroid cancer. Air pollution fine particles and smoking produce lung damage and lung cancer.

Other toxic substances can expose the whole body and if they are genotoxic they will damage the cellular genetic material in any cell in the body. This can potentially produce cancer and premature aging across the whole body. Some organs might have greater susceptibility because of their natural activity, lack of repair or frequency of damage.

For example ELF electromagnetic fields induce electric currents to flow through the body to earth, particularly through electrical circuits, such as the brain and central nervous system or water dominated conductors such as the brain, blood and bone marrow.

Electromagnetic Radiation propagates through space at the speed of light. It is attracted to an aerial and induces a current in a receiver that flows to earth. The induced currents are much stronger than for ELF fields, their strength being inversely proportional to the dielectric constant. The dielectric constant declines significantly with increasing RF/MW carrier frequency, Vignati and Giuliani (1997). The increase in induced current from a unit field from 50Hz to 50 MHz is about 1 million times higher.

Hence both ELF fields and RF/MW radiation expose the whole body to induced currents flowing to earth. Sensitive organs include the brain and Central Nervous System, producing Brain/CNS cancer and lymphoma and leukaemia from cancers in the lymphoid system, blood and bone marrow. All other organs are susceptible to cancer. If melatonin reduction is also involved then the breast, fetus and testes would be particularly vulnerable. The latency time from the initiation to the detection of cancer ranges typically from 5 to 40 years. For early childhood cancer to occur, damage is likely to start in the fetal stage within the womb. Cancer can develop faster in young children because of their reduced melatonin and lack of immune system competence. Sperm and ovary damage can pass cancer on to the newborn children.

**Genotoxic Implications for a Ubiquitous Agent:**

A ubiquitous agent is one that exposes every person in a society where the agent is generated. A ubiquitous genotoxic agent will enhance cancer and other associated health effects in the general population, raising the background incidence rates to mask the effects of occupational exposure. This results in significant under reporting of occupational effects and inappropriate dismissal of evidence because of misunderstood scientific integration of all knowledge and the ignorance of the agent's genotoxicity.

**History of ELF and RF/MW Exposures of the Population:**

If electromagnetic radiation across the spectrum is a Universal Genotoxic Carcinogen, then a high proportion of the rising cancer rates of the 20<sup>th</sup> century are attributable to the electrical wiring of homes, electrical, electronic, communication, computing technologies and telecommunication technologies. The electric wiring of homes provided the majority of the ELF exposure rise of the population prior to 1950. Prior to the second world war and in the period following the war radio and TV systems have exposed the general population to rising RF intensities. Computers have enhanced this exposure and the mobile telephone system has significantly raised the general population exposure to microwaves from base stations and the mobile phone users' heads and bodies to levels previously received by radar exposed workers, primarily because of the close proximity of the phone's antenna.

**Epidemiological Studies:**

Epidemiological studies are the primary human tool for identifying the effect of disease agents in human populations. They are strong and direct evidence of human health effects. Consistency, strength of association, dose-response relationship and dealing with confounders are important aspects of the science of epidemiology.

The strongest direct proof of cause and effect comes from dose-response relationships that show that a higher exposure to an agent produces a higher disease rate, for an agent that has a known plausible mechanism, in the context of other multiple independent studies showing elevated and significantly elevated disease rates with residential and/or occupational exposures to the agent. Strength of association, through statistical significance and the size of the Odds or Risk Ratio, also adds strong support for a cause and effect relationship.

**Healthy Worker Effect:**

Because employed workers are on average much younger than the average whole population, and because many employment situations require a level of health and fitness, especially in uniformed public service groups such as the police, fire fighters and the military, there is a well understood and accepted Healthy Worker Effect. Beaglehole, Bonita and Kjellstrom (1993) describe this as "an important selection bias", because the "working population has a lower total morbidity and mortality than the population as a whole". They also state that "rates among health workers are 70-90% of those in the general population." In fact in some circumstances that involve younger than average workers the rates can be lower than 40-50% of the general population.

**Summary of the Epidemiological Evidence:**

The first published residential epidemiological study of childhood cancer was Wertheimer and Leeper (1979). The first RF/MW residential study showing increased cancer was Lester and Moore (1982). Both of these studies reported dose-response relationships in residential cancer rates. The first published chromosome damage study was in the journal Nature in 1959, Heller and Teixeira-Pinto (1959).

An early occupational observation was in a letter to a journal by Dr Milton Zaret who reported that in a group of 18 radar exposed workers, 2 had astrocytomas, Zaret



(1977). Compared with the astrocytoma rate in the average young male population of the US in the 1960-70 period:

**RR = 482.6 (45.8-5085), p<0.00005**

Theriault et al. (1994) studied electric utility workers and found an astrocytoma rate for the whole working group of :

**OR = 28.48 (1.76-461.3)**

Together these 5 studies form a strong and consistent pattern for a universal genotoxic carcinogen with residential studies showing dose-response relationships at very low mean exposure levels and occupational studies showing highly significant much higher specific rates for brain cancer, with RF/MW producing much higher rates than ELF exposure.

Brain/CNS Cancer Studies:

Across the spectrum there are at least 76 studies showing elevated brain/CNS cancer in EMR/EMF exposed populations, with elevated brain cancer in over 202 individual groups. Dose-response relationships so far have been shown in 21 studies, Cherry (2001a). This is my evidence in the Dr Christopher Newman cellphone brain cancer case presented in August this year in Baltimore.

**Leukaemia Studies:**

A totally independent team of Swedish medical scientists, reviewed almost 100 epidemiological papers published up to July 1994, Hardell et al. (1995). They state:

***We concluded that there are possible associations between:***

- (i) an increased risk of leukaemia in children and the existence of, or distance to, power lines in the vicinity of their residence,***
- (ii) an increased risk of chronic lymphatic leukaemia and occupational exposure to low frequency electromagnetic fields and,***
- (iii) an increased risk of breast cancer, malignant melanoma of the skin, nervous system tumours, non-Hodgkin lymphoma, acute lymphatic leukaemia or acute myeloid leukaemia and certain occupations.***

Many more studies have now been published to strengthen these conclusions.

Milham and Ossiander (2001) investigated a bio-indicator cancer in very young children, Acute Lymphoblastic Leukaemia (ALL). They showed that ALL is time sequence, geographically and dose-response related to the residential electric wiring. The ALL was initiated by very low exposure of mothers and children to extremely low frequency fields less than 1 mG. The early childhood leukaemia (ALL) is increased by a factor of 7-8 in the period 1900-1959, in parallel with all other age groups <40 years as reported by Brown and Doll (1960).

Milham and Ossiander reviewed about 100 occupational studies and 40 residential studies of the ELF-cancer association. They found that of the approximately 500 separate risk ratios, for every one that showed a reduction in cancer there were 6 that showed an increase in cancer, i.e. about 430 groups have elevated cancer from ELF exposure. The majority of these studies involve leukaemia. None of these studies make adjustments for the Healthy Worker Effect. An adjustment for a 70% effect would make most of the reduced rates increased rates. If the Ubiquitous Genotoxic Effect was taken into account it would make all of the rates much greater than 1.

For childhood cancer there have been published over 10 studies showing dose-response relationships.

### **Breast Cancer:**

The first published occupational study of breast cancer in electrical workers was Demers et al. (1991), for male breast cancer. To date there have been 3 studies, with 5 groups showing elevated male breast cancer and 3 with significant elevation.

There have now been at least 10 papers published on female breast cancer from EMR/EMF exposure. They show 35 groups with elevated female breast cancer and 18 with significantly elevated breast cancer.

There have been a relatively small number of published studies on RF/MW exposure in large groups that are able to investigate cancer across many body organs. One published review, Elwood (1999) is shown in summary form in table 1 below.

This table was used by Elwood to claim that the data was "weak and inconsistent" for the association between RF/MW exposure and cancer. Under cross-examination in a cellphone base station appeal case in Adelaide, Australia, I asked the Commissioner hearing the case, if he would look at Table 1 and ask the question "Does it show that RF/MW exposure enhanced the cancer rate across many organs in the body in multiple independently published studies?"

The Commissioner's response after considering the table was: "I thought this paper was peer reviewed". This clearly expressed his doubts about the validity of Professor Elwood's conclusions. The cellphone company's response was to withdraw their appeal and seek an alternative site.

Table 2 summarizes a set of studies on leukaemia from RF/MW exposure. This shows an ecological dose-response relationship supporting the a priori hypothesis that EMR is a Universal Genotoxic Carcinogen.

Table 3 summarizes similar and other studies cited by Elwood (1999). This shows that there are other studies supporting the Universal aspect of the Hypothesis with increased cancer across many body organs.

The Danish study on cellphone use and cancer, Johansen et al. (2001), was a cohort study comparing the cancer rates for cellphone users with the general population. This results in many rates in the range 60-70%, consistent with the Health Worker Effect applying to cellphone users. The study group included 67% of cellphone users who had used their phones for 2 years or less. The a priori hypothesis was that cellphone

produced cancer. A single direction effect. However, a two-tailed significance test was applied (inappropriately). This doubles the p-value. Adjusting for this would place many of the ratios above the significance threshold. Johansen et al's table 2 (Table 4 below) shows that the male rates are much lower than the female rates, largely because of the Healthy Worker Effect (HWE). Dividing by 0.7 to adjust for a 70% HWE, elevates most of the cancer rates.

**Table 1.** Radiofrequency emissions and cancers: occupational studies and study of amateur radio operators. (Elwood (1999))

	Occupational/amateur radio operator studies				
	Polish military	U.S. Navy	U.S. amateur radio operators	Norwegian female ship radio operators	Canada/France electric utility workers
<b>Study characteristics</b>					
Exposure	RF/microwave	Microwave (radar)	Radio operation	Radio operation	PEMF
Ascertainment	Service records	Service records	Radio operators license records	Employment records field measurements	Employment records
Exposed group	Military, male	Military, male	Amateur radio operators, male 0.4-25	Radio operators on ships, female 5-20, possibly up to 300	Electric utility workers
Frequency, MHz	150-3500				
Outcome data	Incidence	Mortality	Mortality	Incidence	Incidence
Exposed group for RR values			Hazard no, 5001 + -		≥90th percentile
<b>Outcome</b>					
Total deaths, all causes		7.23 (0.98-1.52)	0.71 (0.69-0.74)	- -	
All cancer	2.07 (1.12-3.58)	1.44 (0.96-2.07)	0.89 (0.82-0.95)	1.2 (1.0-1.41)	1.39 (1.05-1.85)
Lymphatic and hematopoetic	6.31 (3.12-14.32)	1.64 (0.70-3.25)	1.23 (0.99-1.52)	- 0.96 (0.48-1.90)	
All leukemia	-	-	1.24 (0.87-1.72)	1.1 (0.1-4.1)	0.80 (0.19-3.36)
Acute myeloid leukemia	8.62 (3.54-13.67)	-	1.76 (1.03-2.85)	- 1.02 (0.08-13.04)	
Acute lymphatic leukemia	5.75 (1.22-18.16)	-	1.20 (0.26-3.81)	- -	
Acute nonlymphoid	-	-	- -	1.91 (0.19-19.39)	
Chronic myeloid leukemia	13.90 (6.72-22.12)	-	0.86 (0.17-2.50)	- -	
Chronic lymphatic leukemia	3.68 (1.45-5.18)	-	1.09 (0.40-2.38)	- 2.98 (0.21-41.57)	
Hodgkin disease	2.96 (1.32-4.37)	-	1.23 (0.40-2.88)	- -	1.33 (0.23-7.68)
Non-Hodgkin lymphoma	5.82 (2.11-9.74)	-	- -	1.80 (0.62-5.25)	
Lymphoma/lymphosarcoma	5.82 (2.11-9.74)	-	0.47 (0.15-1.1)	1.3 (0.4-2.9)	-
Other lymphatic	-	-	1.62 (1.17-2.161)	-	-
Lung	-	-	- 1.2 (0.4-2.7)	3.11 (1.60-6.04)	
Larynx/Lung	1.06 (0.72-1.56)	-	- -	-	
Respiratory tract	-	2.20 (1.05-4.06)	0.66 (0.58-0.76)	-	-
Other respiratory	-	-	- -	1.20 (0.13-11.28)	
Oral cavity	0.71 (0.42-1.32)	-	- -	-	
Pharynx	1.08 (0.82-1.24)	-	- -	-	
Lip oral cavity, pharynx	-	-	- -	4.98 (0.73-34.0)	
Esophagus	-	-	1.13 (0.71-1.72)	-	-
Stomach	-	-	1.02 (0.68-1.45)	0.4 (0.1-2.0)	2.16 (0.22-20.88)
Esophagus, stomach	3.24	-	- -	-	
Colon	-	-	1.11 (0.89-1.37)	1.3 (0.6-2.6)	1.35 (0.43-4.19)
Rectum	-	-	0.77 (0.42-1.29)	1.8 (0.7-3.9)	1.54 (0.42-5.61)
Colorectal	3.19 (1.54-6.18)	-	-	-	-
Digestive organs	-	0.78 (0.15-2.31)	-	-	-
Liver	-	-	0.65 (0.33-1.17)	-	-
Pancreas	-	-	0.64 (0.42-0.94)	0.6 (0.0-3.5)	-
liver, pancreas	1.47 (0.76-1.56)	-	-	-	-
Other gastrointestinal	-	-	-	-	1.47 (0.47-4.54)
Prostate	-	-	1.14 (0.90-1.42)	-	0.78 (0.23-2.56)
Kidney	-	-	0.94 (0.57-1.48)	1.6 (0.3-4.8)	-
Kidney, prostate	0.86 (0.54-1.67)	-	-	-	-
Bladder	-	-	0.66 (0.38-1.08)	0.6 (0.0-3.6)	-
Urinary tract	-	-	-	-	1.02 (0.34-3.07)
Female breast	-	-	-	1.5 (1.1-2.0)	-
Cervix	-	-	-	1.0 (0.6-1.7)	-
Uterus (endometrium)	-	-	-	1.9 (1.0-3.2)	-
Ovary	-	-	-	0.8 (0.3-1.6)	-
Bone	0.67 (0.36-1.42)	-	-	-	-
Skin	1.67 (0.92-4.13)	-	-	-	-
Melanoma	-	-	-	0.9 (0.4-1.7)	0.31 (0.03-2.82)
Thyroid	1.54 (0.82-2.59)	-	-	-	-
Multiple myeloma	0	-	-	-	0.20 (0.03-1.39)
Brain/nervous system	1.91 (1.08-3.47)	-	1.39 (0.93-2.00)	1.0 (0.3-2.3)	1.90 (0.48-7.58)
Other astrocytoma	-	-	-	-	6.26 (0.30-132.2)
Brain, glioblastoma	-	-	-	-	0.57 (0.08-3.91)
Other cancers	-	1.17 (0.50-2.32)	-	0.7 (0.3-1.3)	-

Polish Military, Szmigielski (1996), U.S. Navy, Robinette et al. (1980), US Amateur radio operators, Milham (1988), Norwegian female radio operators, Tynes et al. (1996) and Canada/France electric utility workers, Armstrong et al. (1994).

**Table 2: 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
Korean War Radar Exposure (Mortality)	Robinette et al. (1980)	High AT/ET	Leuk/Lymp	2.22	1.02-4.81
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
UK Sutton Coldfield <=2km	Dolk et al. (1997a)	Moderate	Leuk	1.83	1.22-2.74
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. (1997b)	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.

**Table 3: Summary of all site cancers from Robinette et al. (1980), using AT/ET except for Brain cancer (FT/ET), Milham (1988), 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.	Szmigielski RF/MW High	Dolk(a) RF/MW Low	Dolk(b) RF/MW Low
Relationship	RR	PMR	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 4: Johansen et al. (2001) table 2 for a cohort study of cellphone users in Denmark.**

Site of cancer (ICD-7)‡	Men				Women			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
All cancers (140-205)	2876	3327.6	0.86	0.83 to 0.90	515	497.6	1.03	0.95 to 1.13
Brain, nervous system (193)	135	142.8	0.95	0.79 to 1.12	19	18.5	1.03	0.62 to 1.61
Salivary glands (142)	7	9.0	0.78	0.31 to 1.60	0	0.7	---	---
Leukemia (204)	77	79.6	0.97	0.76 to 1.21	7	6.6	1.07	0.43 to 2.20
Other cancers								
Pharynx (145-148)	32	51.5	0.62	0.42 to 0.88	4	1.7	2.43	0.65 to 6.22
Esophagus (150)	42	57.1	0.74	0.53 to 0.99	3	2.0	1.53	0.31 to 4.46
Stomach (151)	63	81.2	0.78	0.60 to 0.99	2	4.5	0.45	0.05 to 1.61
Colon (153)	190	199.4	0.95	0.82 to 1.10	22	22.7	0.97	0.61 to 1.47
Rectum (154)	133	133.1	1.00	0.84 to 1.18	12	10.6	1.13	0.58 to 1.98
Liver (155)	18	29.8	0.60	0.36 to 0.96	2	2.0	1.00	0.11 to 3.61
Pancreas (157)	57	69.1	0.82	0.62 to 1.07	5	6.9	0.73	0.23 to 1.70
Larynx (161)	53	65.3	0.81	0.61 to 1.06	2	1.6	1.24	0.14 to 4.48
Lung (162)	301	460.7	0.65	0.58 to 0.73	34	39.1	0.87	0.60 to 1.22
Breast (170)	5	5.0	0.99	0.32 to 2.32	152	141.3	1.08	0.91 to 1.26
Cervix uteri (171)	---	---	---	---	37	27.5	1.34	0.95 to 1.85
Corpus uteri (172)	---	---	---	---	18	17.6	1.02	0.60 to 1.61
Ovary (175)	---	---	---	---	24	22.0	1.09	0.70 to 1.62
Prostate (177)	159	175.6	0.91	0.77 to 1.06	---	---	---	---
Testis (178)	187	166.6	1.12	0.97 to 1.30	---	---	---	---
Kidney (180)	104	101.3	1.03	0.84 to 1.24	7	6.7	1.04	0.42 to 2.15
Bladder (181)	233	239.3	0.97	0.85 to 1.11	12	9.0	1.34	0.69 to 2.33
Melanoma (190)	123	142.7	0.86	0.72 to 1.03	21	26.3	0.80	0.49 to 1.22
Other skin (191)	567	614.8	0.92	0.85 to 1.00	79	79.1	1.00	0.79 to 1.24
Eye (192)	8	12.4	0.65	0.28 to 1.27	0	1.1	---	---
Thyroid (194)	13	12.9	1.01	0.54 to 1.72	4	4.4	0.92	0.25 to 2.35
Non-Hodgkin's lymphoma (200, 202)	109	116.7	0.93	0.77 to 1.13	11	10.6	1.04	0.52 to 1.86
Hodgkin's lymphoma (201)	27	30.6	0.88	0.58 to 1.29	3	2.6	1.18	0.24 to 3.43
Other and unspecified cancers	233	331.1	0.70	0.62 to 0.80	35	32.7	1.07	0.75 to 1.50

\*Ever use of a cellular telephone (NMT 450, NMT 900, or GSM).  
†Obs = observed; Exp = expected.  
‡ICD-7 = International Classification of Diseases, 7th revision. NMT = Nordic Mobile Telephone System; GSM = General System for Global Telecommunications.

Johansen et al's Table 2 shows that with a very little cellphone use, with the use of a high cancer reference group and a 2-tailed test, there are several elevated cancers approaching significance. For example: Testicular cancer SIR = 1.12, 95%CI: 0.97-1.30, Cervical cancer, SIR = 1.34, 95%CI: 0.95-1.85, Female Pharynx cancer, SIR 2.43, 95%CI: 0.65-6.22, Esophagus cancer, SIR = 1.53, 95%CI: 0.31-4.46 and female breast cancer, SIR = 1.08, 95%CI: 0.91-1.26.

Johansen et al. also claims that they found no dose-response relationships. When they compared the brain cancer rate in digital phone users they found that the rate was 0.7 for those using their phones for a year or less. This documents the level of the Health Worker Effect. The following table shows the original and the adjusted rates.

Cell phone users in Denmark, incidence of gliomas associated with digital phone use duration, 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

**This strongly supports the hypothesis and included cell phone usage, as would be expected.**

## **Evidence of EMR Genotoxicity:**

### **Introduction:**

There is no doubt that chromosome aberrations cause cancer.

Smerhovsky et al. (2001) studied a large group of miners who had been exposed to radon. They found that a 1% increase in chromosome aberrations resulted in a 64 % increase in the incidence of cancer,  $p < 0.0001$ .

### **Earlier published statement:**

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.

Baranski and Czerski (1976) wrote a book on the biological effects of microwaves based on studies published up to that time. In their section on Chromosome and possible genetic effects, they open with the statement:

**"Chromosome aberrations and mitotic abnormalities may be induced, at least under certain conditions and in certain cell types, by exposure to microwave or radiofrequency fields. This is a well-established fact, as several reports from at least five independent laboratories exist".**

Baranski and Czerski note some uncertainties about exposure conditions. Also that in some experiments no temperature increases were observed. The evidence available in 2001 is much stronger and confirms that the genetic damage effects occur at non-thermal levels of RF/MW exposure and they do not require heat to be involved for DNA damage to occur.

The following evidence on RF/MW and ELF genotoxicity was first presented in my paper that I was invited to present to the conference at the European Parliament on June 29<sup>th</sup>, 2000.

### **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 (1997), Table 8-4.

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 passages with microwave exposure there were 30 passages without. Significant chromosome aberrations were measured after 20 MW passages. Yao (1987) also found elevated chromosome damage in Chinese Hamsters.

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 micronucleus 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.

Garaj-Vrhovac, Horvat and Koren (1991) exposed Chinese hamster cells to 7.7 GHz microwave radiation to determine cell survival and chromosome damage. They assayed chromosome aberrations and micronuclei and found that microwaves increased these in a dose response manner, Figure 7, to levels that were highly significantly elevated ( $p < 0.02$  to  $p < 0.01$ ).

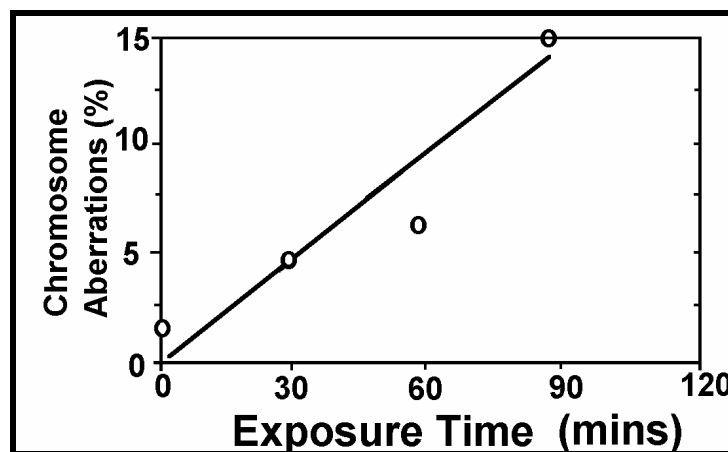


Figure 7: Chromosome aberrations in V79 Chinese hamster cells exposed to 7.7 GHz microwaves at 30  $\text{mW}/\text{cm}^2$ , Garaj-Vrhovac, Horvat and Koren (1991).

An exposure level of 30  $\text{mW}/\text{cm}^2$  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 8.

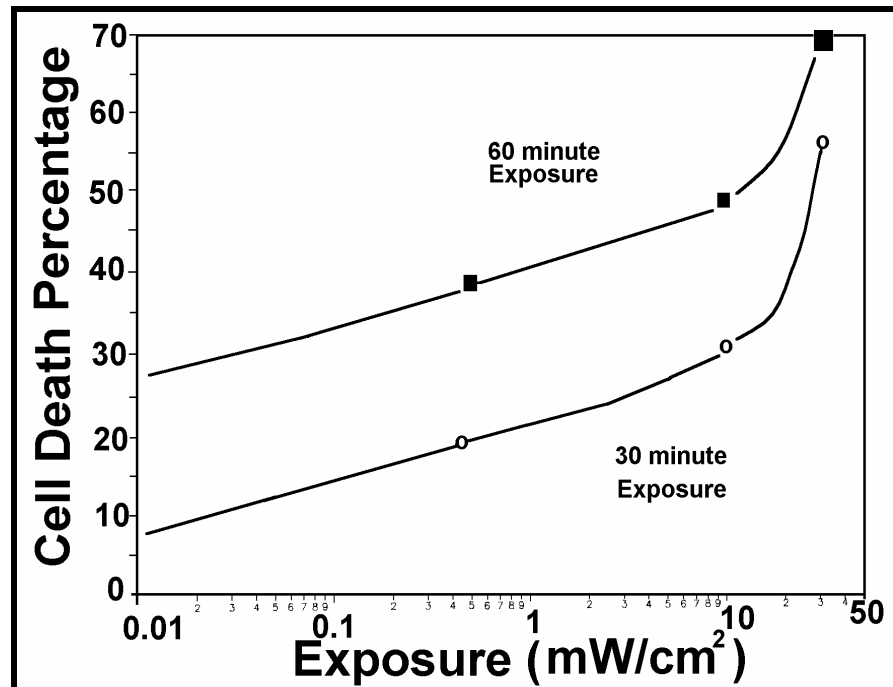


Figure 8: Cell death percentage of Chinese hamster cells exposed to 7.7 GHz microwaves (CW) for 30 minutes and 60 minutes in an isothermal exposure system, Garaj-Vrhovac, Horvat and Koren (1991).

Figure 8 shows that cell death varies with time and intensity of exposure, down to very low exposure levels. An apparent 'saturation' at high levels also evident. 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.

Note that the general public ICNIRP guideline for microwaves above 2 GHz is 1 mW/cm<sup>2</sup>, and for workers is 5 mW/cm<sup>2</sup>. 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 chromosome aberrations, Figure 9.

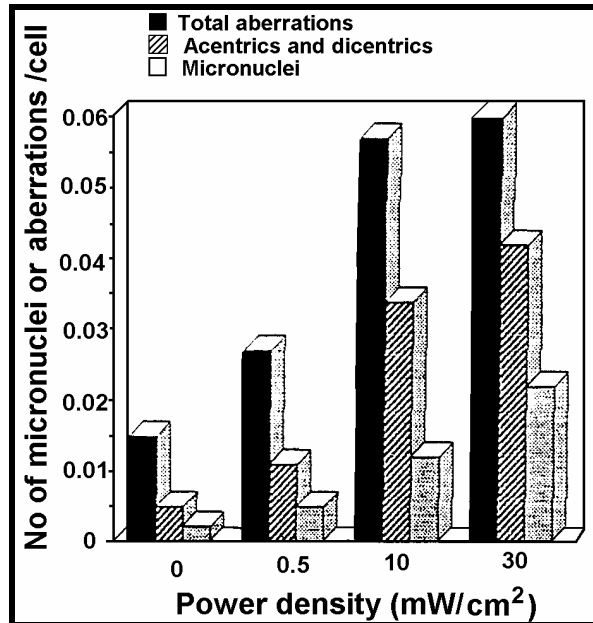


Figure 9: The relation of total chromosome aberrations, micronuclei and specific chromosome aberrations for each cell in human lymphocyte cultures in the dose of microwave radiation in vitro, Garaj-Vrhovac et al. (1992).

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 and Fucic (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.

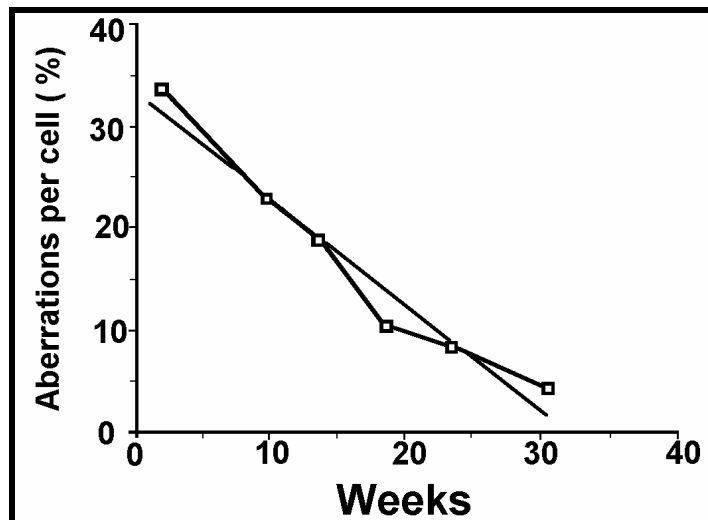


Figure 10: The time-dependent decrease in the number of chromosome aberrations for subjects with high numbers of chromosomal impairments,  $y = 0.318 - 0.010x$ ,  $r=0.98$ . Garaj-Vrhovac and Fucic (1993).

Figure 10 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 four other workers are shown in Figure 11.

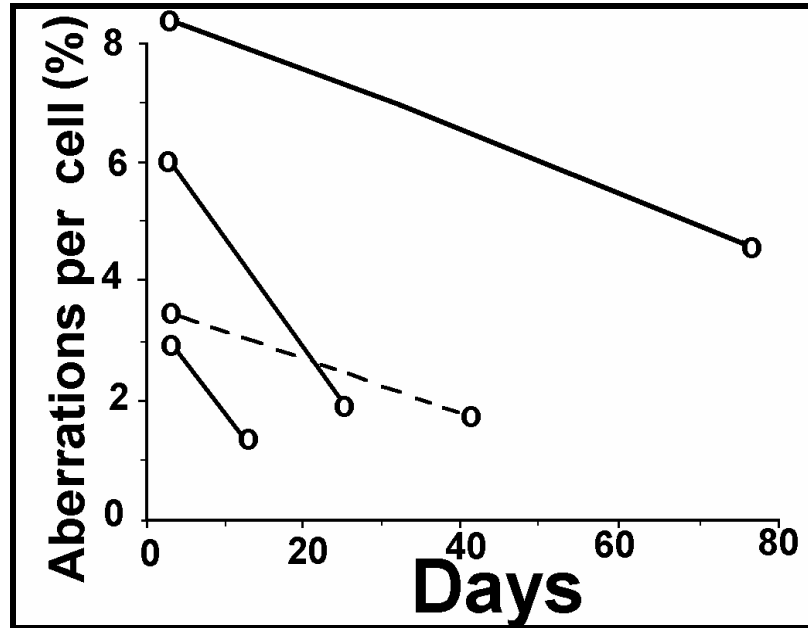


Figure 11: Decreases in human blood Chromosome Aberrations over time from microwave exposed radar repair workers, Garag-Vrhovac and Fucic (1993).

Two different rates are evident. Two people had repair rates 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.

Figure 12 shows the actual microscopic images of chromosomes in human blood taken from a man exposed to radar.

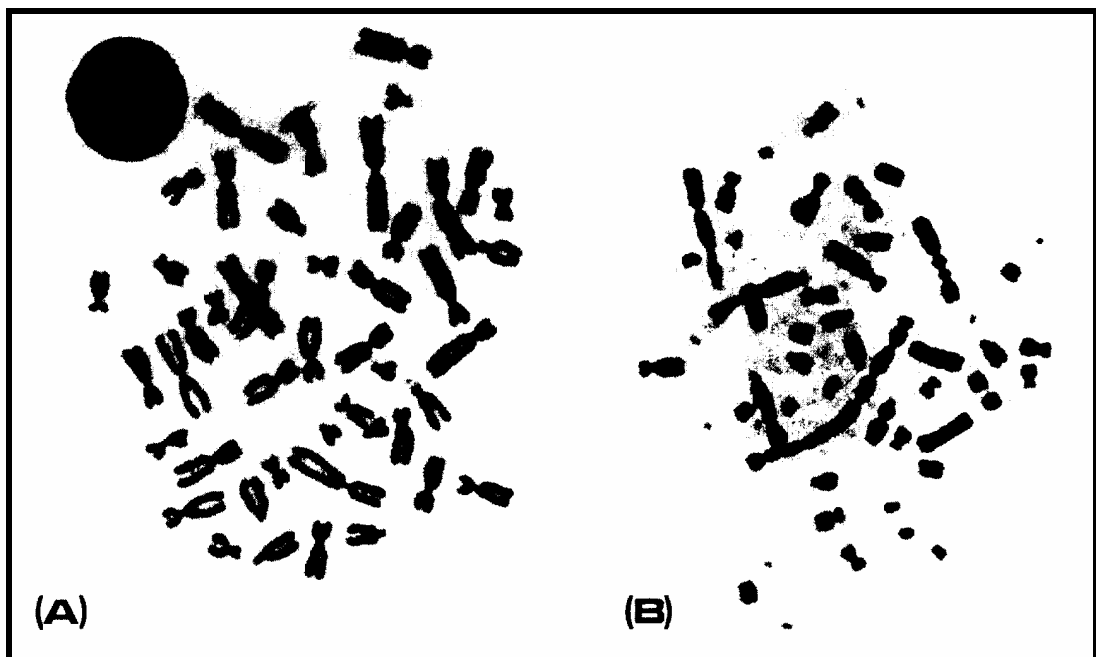


Figure 12: Chromosomes from the highly exposed subject, (a) before exposure and (B) after accidental exposure to a microwave radar signal, Garaj-Vrhovac et al. (1993).

There is no doubt that the radar exposure damaged the chromosomes. The damage is highly visible. Figure 12 shows that after microwave exposure there were many acentric, dicentric, polycentric, fragments, chromatid, ring chromosomes, chromosome breaks and chromatid interchange.

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 13.

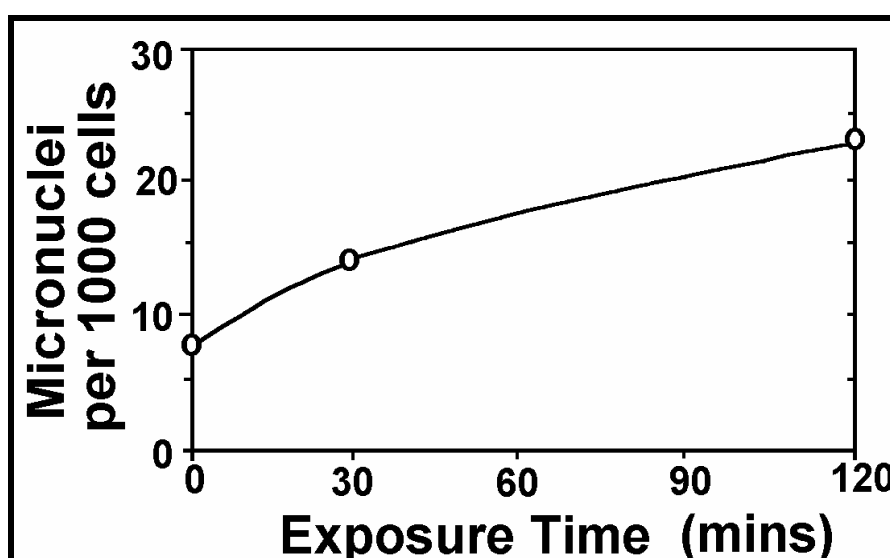


Figure 13: Micronuclei in microwave exposed human lymphocytes, the average of 4 donors, Maes et al. (1993). Exposure was to 75 W/kg, 2.45 GHz microwaves pulsed at 50 Hz, under controlled isothermal conditions.

Maes et al. (1997) observed elevated CAs from microwave exposure. Kovesnikova 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 %,  $p < 0.01$ . 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.

This was a totally unexpected result from this group. A great deal of effort was put into playing down the implications. They describe the increase in peripheral blood as a 0.05%,

by dividing the increase of 1 by 2000. This is not a significant increase and this is not the right comparison. It is a deliberate attempt to disguise their true result that shows that microwaves are genotoxic.

Garaj-Vrhovac (1999) used a micronuclei assay and lymphocyte mitotic activity to assess genetic damage of a group of 12 men occupationally exposed to microwave radiation. Exposures ranged from  $10\mu\text{W}/\text{cm}^2$  to  $20\text{mW}/\text{cm}^2$ , in the frequency range 1250-1350 MHz. It was found that there was significantly enhanced micronuclei formation and significantly altered mitotic activity. Figure 8 shows the table of the micronuclei assay of 12 control subjects and 12 MW exposed subjects with MW exposure producing. The exposed group had more than 3 times more micronuclei, 174 vs 50,  $p < 0.0001$ .

SUBJECT No	CONTROL GROUP				EXPOSED GROUP			
	No. of binucleated cells with			MN/500 cells	No. of binucleated cells with			MN/500 cells
	1 MN	2 MN	3 MN		1 MN	2 MN	3 MN	
1	5	0	0	5	17	3	0	23
2	4	1	0	6	13	2	0	17
3	2	0	0	2	10	1	0	12
4	5	0	0	5	10	2	0	14
5	7	0	0	7	14	1	0	16
6	3	0	0	3	11	2	0	15
7	5	0	0	5	8	0	0	8
8	2	1	0	4	12	0	0	12
9	2	0	0	2	22	0	0	22
10	2	0	0	2	7	1	0	9
11	3	0	0	3	2	0	2	8
12	6	0	0	6	8	0	0	8

Figure 14: The number of cells/500 with one, two or three micronuclei per cell, comparing the control group with the microwave exposed group, Garaj-Vrhovac (1999).

A relatively high exposure from a cell phone antenna resulted in significant chromosome aberrations, Maes et al. (1996), Figure 15.

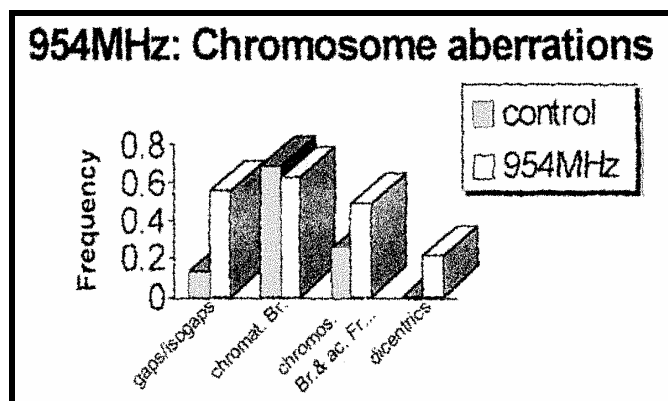


Figure 15: Cellphone radiation of 954 MHz significantly enhances several types of chromosome damage including gaps, acentric and dicentric aberrations, in human lymphocytes, Maes et al. (1996)

Tice, Hook and McRee (1999) showed chromosome damage from all cell phones tested, all being statistically significant and all but one highly significant with dose-response relationships up to a factor of three increase in chromosome aberrations. They repeated the experiment and confirmed that the results were robust and not an artifact.

Multiple independent studies, over 25 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 genotoxic.

### 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 electromagnetic radiation is carcinogenic.

### 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.

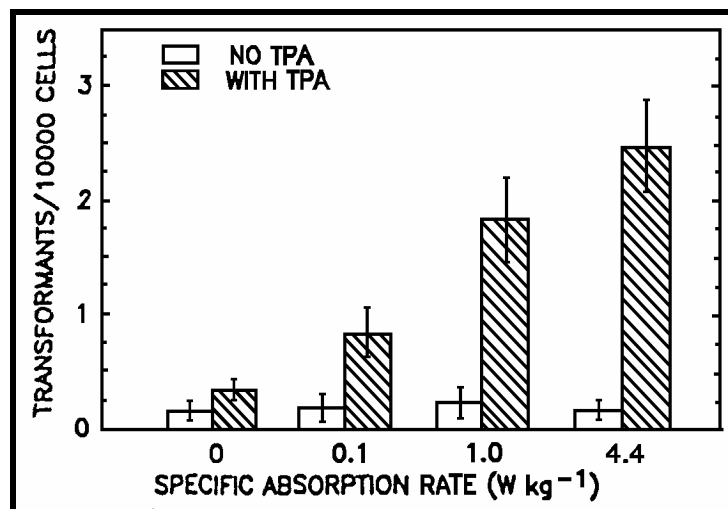


Figure 16: Dose-response relationship for induction of neoplastic transformation in C3H/10T1/2 cells by a 24h exposure to 2.45 GHz microwaves at the specific absorption rate (SAR) with and without TPA post-treatment for 8 weeks, Balcer-Kubiczek and Harrison (1991).

### Direct evidence of DNA-strand alteration and breakage:

Sarkar, Ali and Behari (1994) used Southern Blots of mouse DNA to study sections of the genome in cells with and without microwave exposure. The intensity used was that

accepted as safe by the Non-ionizing Radiation Committee of IRPA (International Radiation Protection Association - the predecessor of ICNIRP),  $1\text{mW}/\text{cm}^2$ .

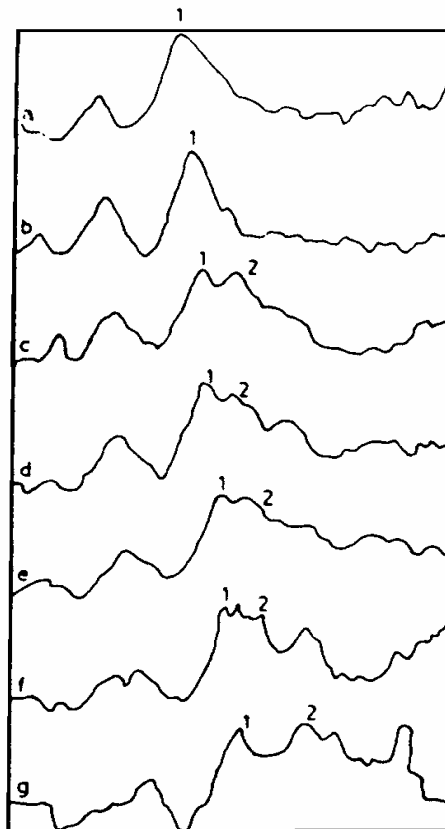


Figure 17: Densitometric analysis of the brain DNA, a and b are control DNA, c to g are DNA from exposed animals. Peak 1 is present in both control and exposed animals while peak 2 appears only in all of the exposed animals.

The exposure regime was a 2 hr exposure to 2.45 GHz CW microwaves at  $1\text{mW}/\text{cm}^2$ , SAR =  $1.18\text{W}/\text{kg}$ . 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 11.

### **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 18.



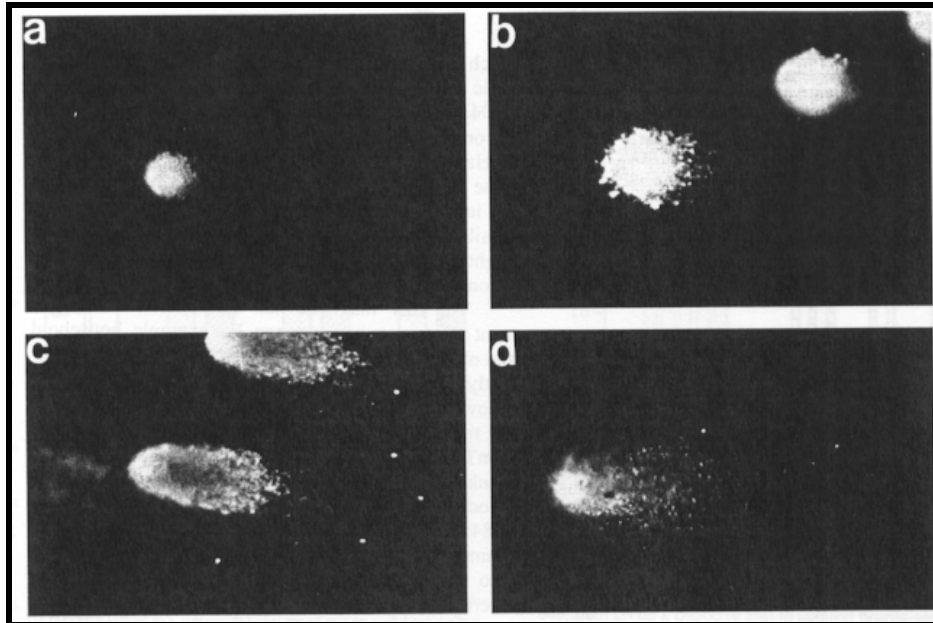


Figure 18: Photographs of double-strand break DNA migration pattern of individual brain cells from rats exposed to (a) bucking condition (0.1 mT), (b) magnetic fields of 0.1 mT, (c) 0.25 mT and (d) 0.5 mT, Lai and Singh (1997a). The “bucking mode” is the condition to reverse the field to cancel the magnetic 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 bound protein from the DNA strands. DNA and protein have the opposite charge and so for the electric field to cause migration, the protein must be removed. 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 19, and the comet tail moment calculated.

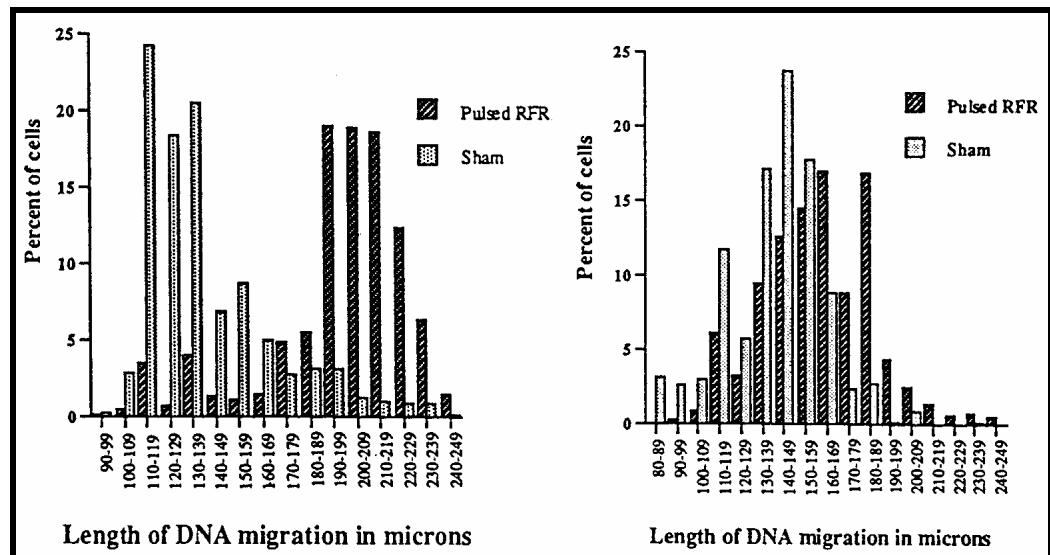


Figure 19: Single- and double-strand DNA breaks frequency distribution for percentage of cells of a given tail length from pulsed RFR and sham exposed brain cells, from 8 animals and 100 cells per animal, Lai and Singh (1996).

Figure 19 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. This clearly documents how less sensitive their method is.

### The Comet Assay and EMR effects:

Drs Lai and Singh have now shown that ELF and RF/MW radiation both cause single and double strand DNA breakage and are associated with free radical and reduced melatonin in living exposed rats. Lai and Singh (1995) observed a dose response increase in Single-strand DNA breakage in the rat's brain and hippocampus that increased significantly after 4 hours, Figure 20. The increases in DNA single-strand breakage after 4 hrs is highly significant,  $p < 0.001$  and they show a dose-response relationship.

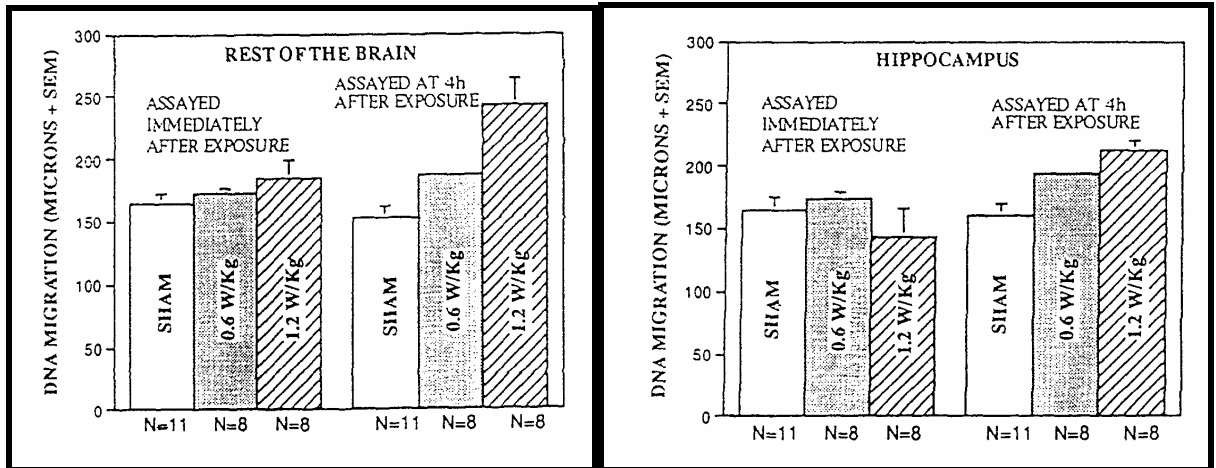


Figure 20: DNA single-strand breakage in cells from the rat brain and hippocampus, immediately after a 2 hr exposure to a whole body SAR of 0.6 and 1.2 W/kg to 2.45 GHz microwave radiation, pulsed at 500 pps. N is the number of rats studied. Lai and Singh (1995).

The assay method was extended to measure DNA double-strand breakage. 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 21.

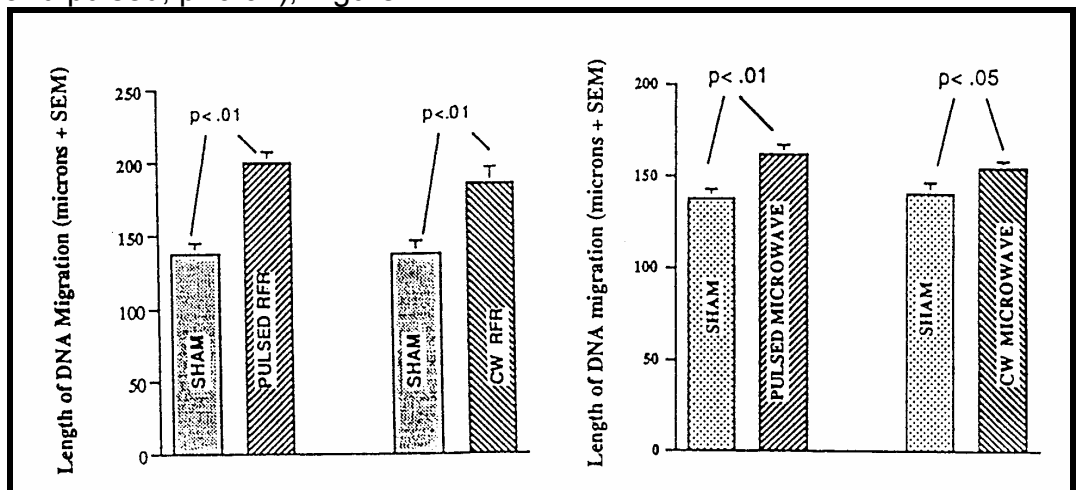


Figure 21: Single-strand (left) and double-strand (right) breaks in brain cells of rat after exposure to pulsed or continuous-wave RFR. Each bar represents data from 8 rats, Lai and Singh (1996).

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 are highly likely to cause DNA damage.

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 22 shows the effect of melatonin for single- and double-strand DNA breaks and Figure 23 the same for PBN.

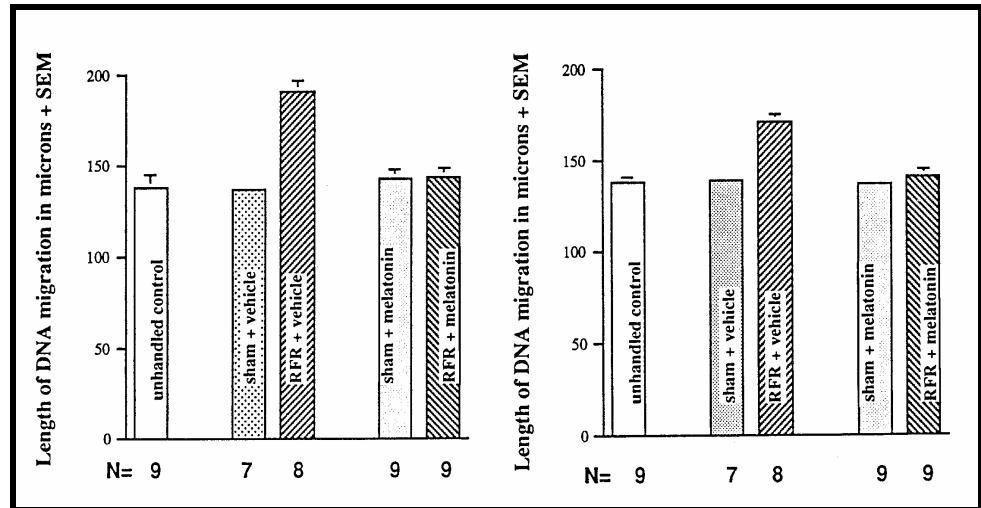


Figure 22: Effect of treatment with melatonin for RFR-induced increase in DNA single-strand (left) and double-strand (right) breaks in rats brain cells. Data was analysed using the one-way ANOVA, which showed a significant treatment effect ( $p < 0.001$ ) for both cases. "vehicle" involves injecting with the physiological saline without the active substance. Lai and Singh (1997)

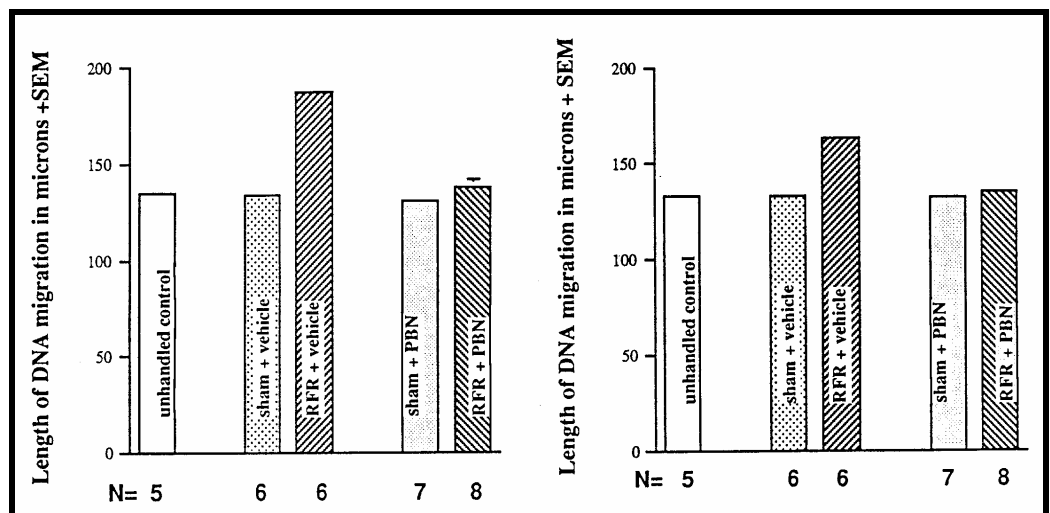


Figure 23: Effect of treatment with PBN for RFR-induced increase in DNA single-strand (left) and double-strand (right) breaks in rats brain cells. Data was analysed using the one-way ANOVA, which showed a significant treatment effect ( $p < 0.001$ ) for both cases. "vehicle" involves injecting with the 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, have been suggested. Free radicals also play an important role in aging processes, Reiter (1994). 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. (1998), 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, Figure 24.

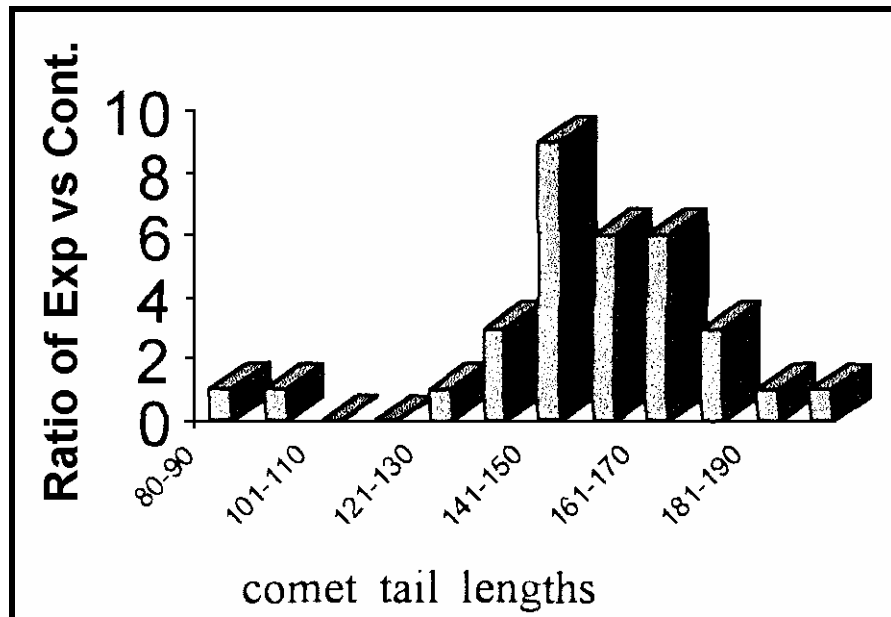


Figure 24: The ratio of comet tail lengths in 954 MHz exposed human lymphocyte cells vs corresponding unexposed samples, Verschaeve and Maes (1998).

Phillips et al. (1998) exposed Molt-4 T-lymphoblastoid cells to a range of cell phone radiation in the SAR range 0.0024 W/kg to 0.026 W/kg for both iDEN and TDMA signals. Using the basic equations, these SARs at the 813-836 MHz range [ $SAR = \sigma E^2 / 2\rho$ ,  $\sigma = 1$  S/m,  $\rho = 800$  kg/m<sup>3</sup>, and  $S = E^2 / 3.77 \mu\text{W}/\text{cm}^2$ , E: the electric field gradient in V/m and S the exposure in  $\mu\text{W}/\text{cm}^2$ ] result in 1.0 to 11.0  $\mu\text{W}/\text{cm}^2$ . A 2 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). Significance at these levels is often taken as causal.

Hence RF/MW radiation has been confirmed to enhance DNA damage under RF/MW exposure from radar-like and cell phone exposures in four independent laboratories. One study included an exposure level which is 0.22% of the ICNIRP guideline.

#### **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. "Replication" requires the work to be very closely following the method and conditions of the earlier study, usually carried out by independent researchers who are well qualified. Both groups used 2.45 GHz microwaves for exposure. However, the follow-up study used a cell-line (C3H/10T1/2) compared to Lai/Singh's living rats. The St Louis group also used

a very different DNA damage assay based on Olive et al. (1992) not Singh et al. (1994). The follow-up study also 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). Most importantly, they did not use proteinase K to separate the bound protein from the DNA strands. It is therefore understandable evident that they used a much less sensitive method. However, despite this and their claims to find no DNA breakage from 2.45 GHz nor cellphone radiation, the data shows that they actually did.

The first example was from Malyapa et al. (1997a), Figure 5, shown in Figure 25. 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.

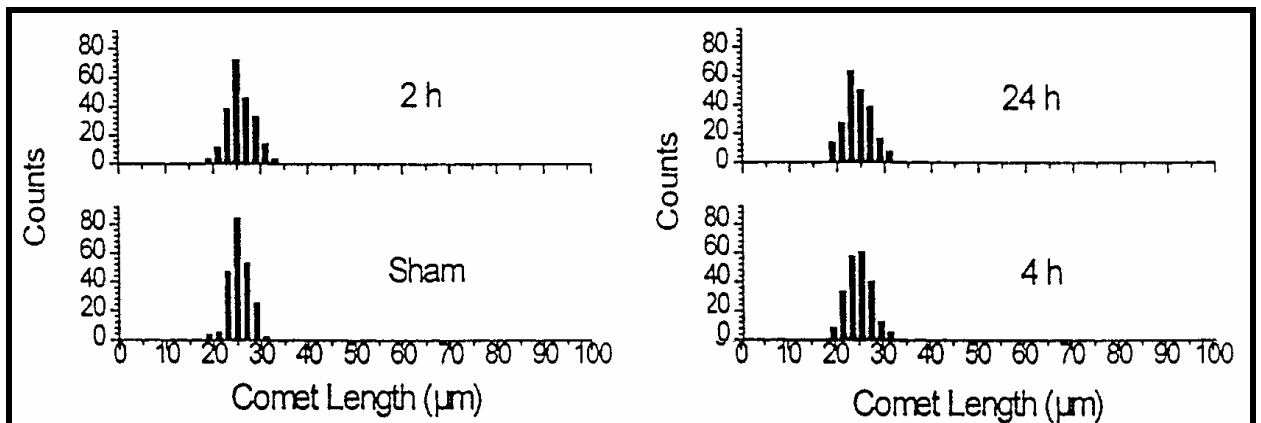


Figure 25: Frequency Distribution of Comet tail lengths for 2.45GHz exposed U87MG cells, Malyapa et al. (1997a).

**Table 1: The 2x2 table of results for DNA strand breakage after exposure of U87MG cells to 2.45 GHz microwaves, from Figure 14:**

Time	Comet Length Class		RR	95%CI	$\chi^2$	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 26.

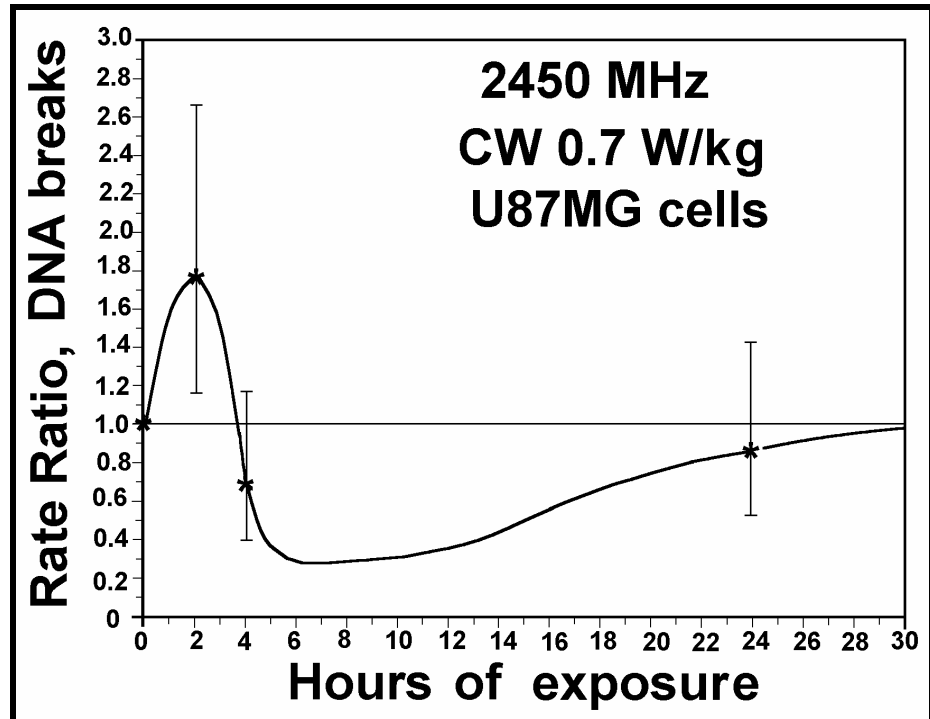


Figure 26: The time sequence of DNA damage and enhanced repair for Figure 5 in Malyapa et al. (1997).

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 21 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.

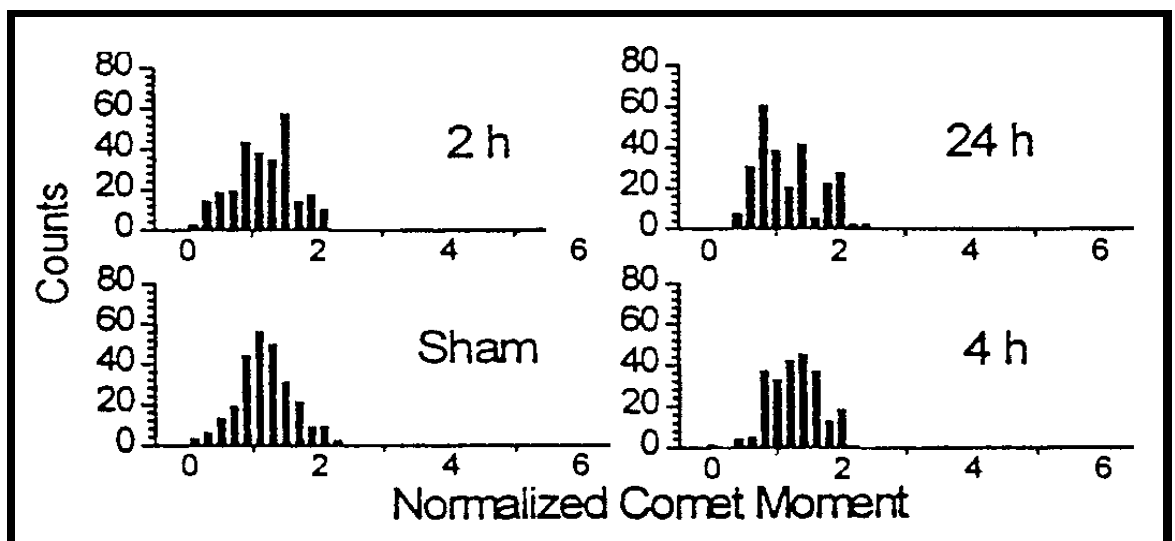


Figure 27: The frequency distribution of normalized comet moment for CW exposure of 2450 MHz at 0.7 W/kg of C3H 10T1/2 cells, Malyapa et al. (1997a) Figure 6.

**Table 2: The 2x2 table of results for DNA strand breakage after exposure of C3H 10T1/2 cells to 2.45 GHz microwaves, from Figure 16:**

Time	Comet Moment Class		RR	95%CI	$\chi^2$	p-value
	$\leq 6$	$> 6$				
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 28.

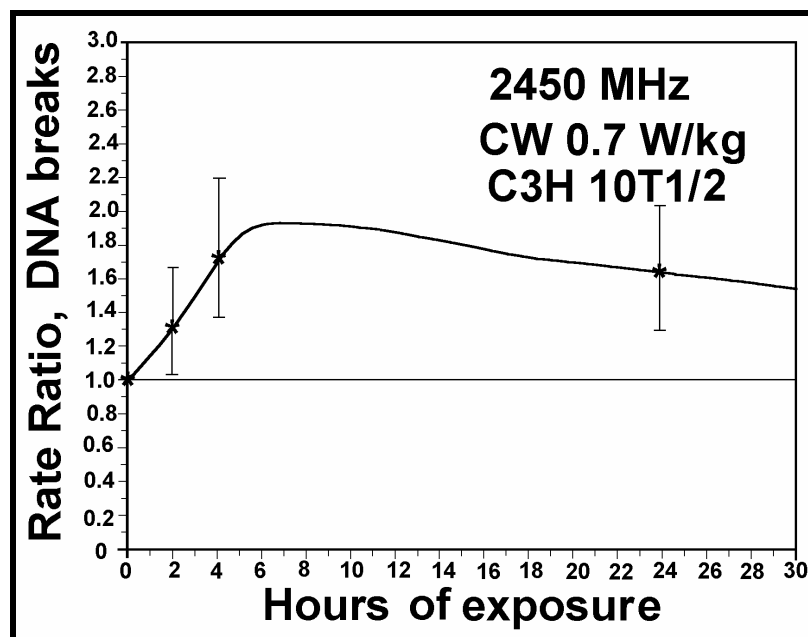


Figure 28: DNA strand breakage Risk Ratio and 95% confidence intervals for the frequency distribution of the Normalized Comet Moment of Malyapa et al. (1997a), Figure 6. The time line is a fitted estimate.

Table 2 and Figure 28 show 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 29.



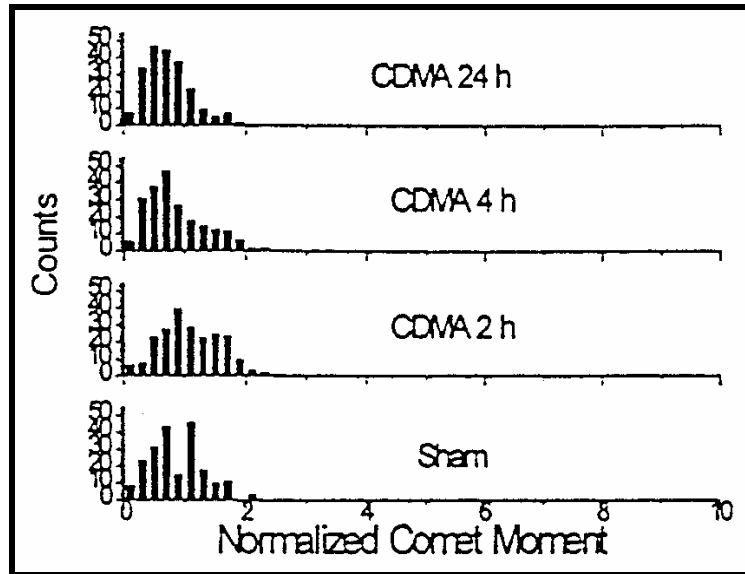


Figure 29: The frequency distribution of normalized comment moment for CW exposure of 847.72 MHz at 0.6 W/kg of U87MG cells, Malyapa et al. (1997b) Figure 2.

**Table 3: The 2x2 table of results for DNA strand breakage after exposure of U87MG cells to 847.74 MHz microwaves, from Figure 18:**

Time	Comet Moment Class		RR	95%CI	$\chi^2$	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

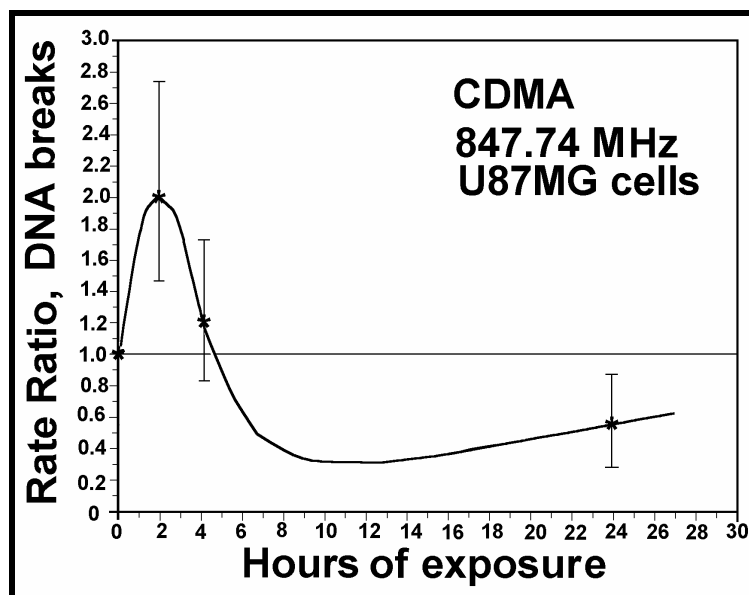


Figure 30: DNA strand breakage Risk Ratio and 95% confidence intervals for the frequency distribution in Figure 2 Normalized Comet Moment of Malyapa et al. (1997b).

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 24, shows the same general pattern as also seen for U87MG cells exposed to 2.45 MHz radiation in Figure 20 above.

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).

The results of Malyapa et al. (197a,b) put the results of Phillips et al. (1998) into context. Phillips et al. (1998) 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).

The lower sensitivity of the assay used by Malyapa et al. is directly demonstrated by the comet tail lengths. The longest comet tail lengths were 32 microns for Malyapa et al. and 250 microns for Lai/Singh. Despite the lower sensitivity of the Malyapa et al. assay, the results confirm the initial results of Lai and Singh (1995), that pulsed microwaves, including cell phone radiation, significantly enhances DNA-strand breakage.

### **Genotoxicity Conclusions:**

A large body of scientific evidence is cited above that shows that electromagnetic radiation, across the EMR spectrum, causes comprehensive damage to the genetic material in cells. It also impairs the body's ability to repair and eliminate damaged cells through reducing melatonin, altering calcium ion signalling and impairing the performance of the immune system.

There is strong and robust evidence of chromosome aberrations, micronuclei formation, DNA-strand breakage altered oncogene activity and neoplastic transformation of cells, to conclude that EMR across the spectrum from ELF to RF/MW is genotoxic. This is independently confirmed by the established biological mechanisms of calcium ion efflux and melatonin reduction.

This is also totally independently confirmed by over a hundred occupational groups showing elevated cancer from EMR exposure, scores showing significantly to very highly significantly elevated cancer incidence and mortality, and several with dose-response relationships.

## **CONCLUSIONS:**

The genotoxic and epidemiological evidence strongly support and reinforce each other as confirmation of the hypothesis that:

**Electromagnetic Radiation across the spectrum is  
a Universal Genotoxic Carcinogen.**

**This means that the safe level of chronic exposure  
is zero.**

## **Recommendations:**

- **Based on this robust and extensive evidence, or, if there is any doubts, the Precautionary Principle, then public health protection standards should have residential and occupational levels lower than:**

**$0.1\mu\text{W}/\text{cm}^2$  (0.6 V/m)**

- **Cellular telephones can be made more than 99% safer. Exposing the user's head and body to less than 1% of the present SAR.**
- **Cellphone base stations can generally be sited away from residences and workplaces, with side-lobe protection, to significantly reduce the near vicinity exposure levels.**
- **Legislative measures to enforce these provisions are vital for protecting public health.**
- **Italy is a World Leader with its 6 V/m standard. It can be now reinforce and strengthen its lead by adopting these recommendations of an even stronger and lower standard.**

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