IARC's 1982 Benzene Human Carcinogen Assessment applied to Microwaves

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Abstract:

The International Agency for Research on Cancer (IARC) is a World Health Organization Department, with the responsibility to assess carcinogenic effects of environmental and occupational toxins. Epidemiology is the fundamental science and the strongest evidence for the assessment of human health effects of disease agents. In 1974 an IARC review concluded that animal carcinogenicity had not been demonstrated and human studies showed that benzene mixtures resulted in damage to the haematopoietic system with suggestion of leukaemia from several case studies and one Japanese case-control study. In 1982 the IARC re-evaluation reported that workers and the general public were exposed to Benzene from numerous sources including chemicals and the production and use of gasoline. IARCs 1982 re-evaluation concluded that benzene was a human carcinogen. Therefore it is appropriate to investigate what the level of evidence was for Benzene in 1982. If we apply the same principles and level of evidence to radar and microwave exposures up to 1982, would the evidence have reached or exceeded the IARC evaluation standard? This review shows that the evidence that microwaves enhanced the rates of cancer in people was stronger in 1982 than the evidence for Benzene.

Introduction: The IARC Assessment:

In 1974 an IARC review concluded that animal carcinogenicity had not been demonstrated and human studies showed that benzene mixtures resulted in damage to the haematopoietic system with suggestion of leukaemia from several case studies and one Japanese case-control study. In 1982 the IARC re-evaluation reported that workers and the general public were exposed to Benzene from numerous sources including chemicals and the production and use of gasoline. Chronic human exposure resulted in serious blood changes, benzene crossing the placenta can causing chromosome aberrations (CAs) in bone marrow and peripheral lymphocytes in individuals exposed to high levels of benzene (> 100 ppm), with some findings showing CAs with exposures as low as 10 ppm, but not consistently. A series of epidemiological studies of benzene exposed workers, both cohort and case-control, showed statistically significant associations between leukaemia (predominantly myelogenous) and occupational exposure to benzene and benzene-containing solvents.

Even though: "There was *limited evidence* that benzene is carcinogenic in experimental animals", "There is *sufficient evidence* that benzene is carcinogenic to man."

Hence with the evidence available in 1982 IARC classified benzene as a human carcinogen.

The acceptance is interesting because the epidemiological evidence was relatively small. Some studies found no elevated leukaemia associated with chronic benzene exposure. e.g. Fishbeck et al. (1978), Ott et al. (1978) and Townsend et al. (1978). Note the full author lists, and the papers acknowledge that the work was done on behalf of Dow Chemical Company:

- Fishbeck, W.A., Townsend, J.C., and Swank M.G. (1978)
- Ott, M.G., Townsend, J.C., Fishbeck, W.A., and Langer R.A. (1978)
- Townsend, J.C., Ott, M.G. and Fishbeck, W.A. (1978)

This could well have been the result of the failure to apply the Healthy Worker Effect.

Studies showing elevated leukaemia were:

Thorpe (1974) found elevated, SMR = 121, n=8.

Askoy et al. (1974) reported an OR = 2.2, n= 34.

Askoy et al. (1980) reported 8 additional cases, raising the significance but failing to give good personal exposure data.

Infante et al. (1977), in a retrospective cohort mortality study, found a significantly elevated leukaemia rate in benzene-exposed white male workers with SMR = 474, n= 7, $p<0.002$ for one comparison group and SMR = 506, n=7 for another. No dose-response assessment was made.

Rinsky et al. (1981) extended the Infante et al. study with a more complete case follow-up. They reported 7 leukaemia deaths giving a significantly elevated SMR of 560, $p<0.001$.

This epidemiological evidence was appropriate in 1982 for IARC to conclude that it was sufficient to classify benzene as a human carcinogen. It is based on six published epidemiologic studies showing increased leukaemia, two of these studies from one factory site with significantly increased leukaemia ($p<0.002$ and $p<0.001$) from chronic benzene exposures.

Hence there were five studies showing elevated leukaemia and three showing significantly elevated leukaemia and no dose-response relationships, when IARC decided to declare Benzene a Human Carcinogen in 1982.

Since then many other epidemiological studies have reinforced and confirmed this assessment by providing significant and dose-response elevation of leukaemia and

other cancers from chronic low exposure to benzene. The evidence continues to strengthen and confirm the 1982 assessment.

Assessment criteria for national and international bodies vary slightly but the example given here is from the department of the World health Organisation called the International Agency for Cancer research (IARC).

Table 1: IARC Classifications for Evidence of Carcinogenicity

1: Sufficient: Human Carcinogen

Sufficient evidence of carcinogenicity.

Exceptionally: less than sufficient evidence in humans, but sufficient evidence in experimental animals and strong human evidence that the agent acts through a relevant mechanism.

Examples: asbestos, benzene, dioxin, hepatitis C virus, radon, vinyl chloride. Total number of agents: 75.

2A: Limited-Probable Human Carcinogen

Limited evidence in humans and sufficient evidence in experimental animals.

Also: inadequate evidence in humans and sufficient evidence in experimental animals, and strong evidence that the mechanism also operates in humans. Exceptionally: only on the basis of limited evidence in humans.

Examples: benzo[a]pyrene, formaldehyde, PCBS, ultraviolet (A, B & C) radiation. Total number of agents: 59.

2B: Limited-Possible Human Carcinogen

Limited evidence in humans and less than sufficient evidence in animals.

Also: inadequate evidence in humans, but sufficient evidence in experimental animals. Sometimes: inadequate evidence in humans, but limited evidence in experimental animals, with supporting evidence from other relevant data.

Examples: carbon tetrachloride, chloroform, DDT, lead, PBBS, saccharin. Total number of agents: 225.

3: Inadequate evidence of carcinogeniocity

Inadequate evidence in humans and inadequate or limited evidence in experimental animals.

Exceptionally: inadequate evidence in humans, but sufficient evidence in experimental animals, with strong evidence that the mechanism in experimental animals does not operate in humans.

Examples: coal dust, fluorescent lighting, mercury, parathion, phenol, xylene. Total number of agents: 474.

4: Lack of evidence of carcinogenicity

Evidence suggesting lack of carcinogenicity in humans and in experimental animals.

Sometimes: inadequate evidence in humans, but evidence suggesting lack of carcinogenicity in experimental animals, consistently and strongly supported by a broad range of other relevant data.
Only example: caprolactam.

Evidence for RF/MW associated cancer available up to 1982:

Genotoxic Studies:

Heller and Teixeira-Pinto (1959) report that a 5 min isothermal pulsed RF exposure of garlic roots produced serious chromosome aberrations that the authors conclude "mimicked the effects of c-mitotic chemicals and ionizing radiation". Thus Pulsed RF generators were recommended as a controlled laboratory method to produce controlled chromosome aberrations.

Stodolnik-Babanska (1972) carried out microwave exposures of lymphoblastoid cells in isothermal conditions so that the effects they observed were from microwave exposures non-thermal effects. Significant chromosome damage and micronuclei formation was observed and the cells were transformed to cancer cells.

Baranski and Czerski (1976) reviewed the biological effects of RF/MW exposure. In chapter 4 they outline the studies published up until then showing genotoxic effects. They conclude that chromosome aberrations and mitotic abnormalities may be induced under certain conditions and certain cells as a well-established fact since several reports exist from at least, 5 independent laboratories in the West.

Yao (1978) cited 5 previous studies showing that microwaves significantly damage chromosomes, including Heller and Teixeira-Pinto (1959). He then reports that he exposed living Chinese Hamster's eyes to microwaves. This enhanced opacities in the eyes and significantly increased chromosome aberrations. Yao (1982) exposed rat kangaroo RH5 and RH16 cells to 2.45 GHz microwaves, maintaining the temperature at 37°C in the incubator. Chromosome aberrations became evident after multiple passages through the microwave-exposed incubator. The CAs were significantly enhanced in the RH16 cells after 10 or more passages.

Therefore by 1982 it was well established from multiple, independent studies that non-thermal pulsed RF/MW exposure was genotoxic. It is recognized and well establish that a genotoxic substance causes cancer, Dorlands' Medical Dictionary (1994).

Human Studies:

Goldsmith (1997) reported elevated mutagenesis and carcinogenesis among the employees and their dependents that were chronically exposed to very low intensity radar signals the U.S. Embassy in Moscow in the 1950's to the mid-1970's. For most of the time 1953-May 1976, the external signal strength was measured at 5 $\mu W/cm^2$ for 9 hours/day on the West Facade of the building where the radar was pointed, Lilienfeld et al. (1978). It is stated that the exposure is fairly smooth and consistent over the study period. To get the full strength of the signal a person would have to stand at an open window on the west side of the building at the 6th floor, Pollack (1979). Hence allowing for the internal signal strengths to be between 20 and 100 times lower, the occupants of the embassy were exposed to a long-term average radar signal in the range of 0.02 to 0.1$\mu W/cm^2$. Blood tests showed significantly elevated chromosome aberrations in more than half of the people sampled. Leukaemia rates were elevated for adults and children. Most of the staff, with their families, were in Moscow for only one tour of 2 years. Chromosome aberrations found in blood samples are shown in Table 2.

Table 2: Blood samples showed a high proportion of the staff had significantly altered red and white blood cell counts and well above average chromosome aberrations (CA). The CA data is set out in Goldsmith (1997), citing Jacobson (1969) i.e.

Mutagenic Level	Designator	Subjects, No.
5	Extreme	0
4	Severe	6
3.5	Intermediate	5
3	Moderate	7
2.5	Intermediate	5
2	Questionable	5
1	Normal	6

Comparing the 11 level 1 and 2 reference group with the 23 cases above gives:

RR = 2.09, 95%CI: 1.22-3.58, p=0.004

Because of the extensive employee concern about the possible health effects of the chronic radar exposure during their service in Moscow, the U.S. State Department contracted Professor Abraham Lilienfeld, Professor of Epidemiology at Johns Hopkins University, to carry out an epidemiological study of staff and dependents. The staff of other US Embassies in Eastern Europe were also surveyed. Health effect rates were compared with the general US population of the similar sex and age range. The sickness and mortality rates for the Moscow Embassy staff and families are summarized in Table 3.

U.S. State Department Embassy employees are careful selected, including physical fitness. This is one basis of the Healthy Worker effect. For these employees, including non-State Dept employees at the Embassy, their overall mortality rate was 47% of the general US population of similar ages. Their cancer mortality rate was 89%.

Table 2: Increased cancer mortality, accidents and suicide in the staff and dependents at the US Embassy in Moscow shown by data in Lilienfeld et al. (1978). This is associated with chronic extremely low intensity (distant and indoor) radar signals. "Adult" refers to adult dependant. # refers to the HWE adjustment. Note (*) p<0.05)

Cancer Site	Observed	Expected	SMR
All Cancer			
Employee	17	19.0	0.89
Employee Adjusted#	17	9.5	1.79
Adult	12	4.5	2.67*
Childhood	4	1.33	3.01*
Total Dependants	16	5.83	2.74*
Total#	33	15.33	2.15*
Leukaemia:			
Employee	2	0.8	2.5
Employee Adjusted#	2	0.4	5.0
Adult	0	0.18	-
Childhood	2	0.5	4.0*
Total Dependants	2	0.68	2.94
Total#	4	1.08	3.70
Breast Cancer:			
Employee	2	0.5	4.00*
Employee Adjusted #	2	0.25	8.00*
Adult	1	0.93	1.08
Total Breast Cancer#	3	1.18	2.54
Adult Hodgkins disease	1	0.07	14.29
Adult Lung Cancer	1	0.56	1.79
Adult Brain Cancer	2	0.15	13.33
Accidents:			
Employees	6	11.6	0.52
Employee adjusted#	6	5.8	1.03
Adults	6	1.39	4.32*
Children	5	6.8	0.74
Total Dependants	11	8.19	1.34
Total#	17	13.99	0.86
Suicide:			
Employees	0	3.9	-
Employees Adjusted#	0	1.95	-
Adults	1	0.56	1.79
Children	1	0.59	1.70
Total Dependents	2	1.15	1.74
Total#	2	3.10	0.65

The overall mortality rate suggests a HWE factor of 2. In Table 3 including the "employees" dilutes the health effects significantly. The employee expected mortality data were adjusted by a factor of 2 for the Healthy Worker Effect (HWE).

Table 3 demonstrates how a human population that was chronically exposed low level microwave, with significantly elevated chromosome aberrations, showed significant elevation and elevation of cancers in multiple sites, even though the exposure period was generally a maximum of 2 years. This is consistent with RF/MW radiation being a Universal Genotoxic Carcinogen. Suicide is included in Table 3 because it is evidence of reduced melatonin and reduced melatonin is also associated with increased cancer rates.

US Radar Repair Workers:

Zaret (1977) reports that 2 brain tumours (astrocytomas) occurred in a group of about 18 workers servicing radar equipment. In another group of 17 workers exposed to highly pulsed EMR for 7 years there were 5 cancers (2 leukaemia, 2 skin cancer, 1 genitourinary tract cancer). Another group of 3 out of 8 men working on another site developed cancer (2 pancreatic cancers).

The SEER (Surveillance, Epidemiology and End Results) cancer data for the US was used to determine the age-specific (30-34 years) cancer for the 1974 period. For the Astrocytoma brain cancer rate the overall brain/CNS cancer rate was used and multiplied by 20%, which is the proportion of all brain cancers identified as Astrocytomas.

Table 4 supports the hypothesis that RF/MW radiation is a Universal Genotoxic Carcinogen that causes cancer across many body organs when the whole body is exposed.

Table 4: Cancer rates in groups cited by Zaret (1977) compared with the cancer incidence rates for males aged 30-34 years in 1974, from the SEER data-base. [n: cases; N group size; P exposure period; Rate: Incidence Rate; Ref.Rate: SEER reference rate.]

Cancer Site	n	N	P	Rate	Ref Rate	Risk Ratio	95%CI	Fisher Exact
			Yrs	(/100,000)				p-value
Astrocytoma	2	18	10	1111.1	0.68	1634	385-6939	0.0000009
Skin Cancer	2	17	7	1680.6	0.62	2711	641-11462	0.0000003
Leukaemia	2	17	7	1680.6	3.6	467	70-3126	0.0000257
Pancreas	2	8	7	3571.4	0.348	10263	2287-61962	<0.0000001
Urinary	1	17	7	840.3	1.24	678	92-5007	0.00162
All Cancer	10	43	8	2907.0	62.62	46.4	20.4-105.7	<0.0000001

While the radar repairing staff in Table 4 were likely to have experienced close to or including thermal exposures, the data in Table 3 certainly does not involve thermal exposures, the peak measured outdoor exposures being over 2000 times below the thermal threshold, and the indoor exposures being much lower. The following table

sets out the results. Table 4 is highly supportive of the hypothesis that RF/MW exposure is a Universal Genotoxic Carcinogen, with significant at a causal level, p<<0.001.

Korean War Study:

A major study of US Naval servicemen exposed to radio and radar signals during the Korean War, Robinette, Silverman and Jablon (1980), found significant differences among occupational groups in all end-points studied, Silverman (1979). They carried out a job exposure matrix survey to assess the microwave exposures of 5% of the about 20,000 servicemen in the "high" exposure group. This resulted in significant exposure related dose-response increase of all mortality, trend p=0.03 and for respiratory cancer, trend p<0.05. This can be classically causal on its own, Hill (1965).

Table 5 and the evidence of chromosome damage adds significant support for the hypothesis of RF/MW radiation being a Universal Genotoxic Carcinogen.

Table 5: The Mortality Rate of the surveyed high group (Hazard Number: 5000+) compared with the very low exposure group (Hazard Number=0), Robinette et al. (1980). RR is the ratio of the High Group MR to the Low Group MR.

Cause of Death	Risk Ratio	95%CI	p-value
All Mortality	1.28	1.01-1.61	0.038
All Cancer	1.54	1.03-2.29	0.035
Respiratory Cancer	2.68	1.09-6.70	0.022
Leukaemia/Lymphoma	1.50	0.72-3.29	0.26
Other Cancers	1.50	0.68-3.36	0.31
Circulatory Diseases	1.26	0.90-1.72	0.18
Other Diseases	3.60	1.50-8.71	0.0013

The job exposure matrix survey of the "high" exposure groups, ET, FT and AT, showed, Robinette Table 2, that the ET group was generally low exposed and the FT group was generally high exposed. Hence a dichotomy difference of exposed can be used to see if health effects are elevated in high group compared with the low group.

It is important to recognize that the reference or control group is not a “no exposure” group but a lower exposure group. The Healthy Worker Effect is much stronger for uniformed services because of the strong physical requirements for military service. Adjusting for both of these effects suggests an adjustment factor of 2 would be quite conservative.

Grouping the job groups into three exposure ranked groups, Low RM and RD; Middle AE and ET and high FT and AT, allows for a possible dose-response relationship, Figure 1.

Table 6: Comparison of mortality rates in the high exposure AT group (3273) and lower exposure ET group (13078) from Table 5, Robinette et al. (1980).

Cause of Death	ET	AT	RR	95%CI	χ^2	p-value
All Causes of Death	441	198	1.79	1.52-2.11	46.97	$<10^{-7}$
All Causes excl[g] accidents	265	101	1.52	1.21-1.91	13.43	0.00025
All Diseases	199	77	1.55	1.19-2.01	10.89	0.0013
All cancer	65	27	1.66	1.06-2.60	5.93	0.025
Respiratory	16	7	1.75	0.72-4.35	1.56	0.21
Brain/CNS (FT/ET)	5	3	2.38	0.57-9.95	1.50	0.22
Skin	3	2	2.66	0.45-15.9	1.25	0.26
Leukaemia/Lymphoma	18	10	2.22	1.03-4.50	4.32	0.038
Other Cancer	8	4	2.00	0.60-6.63	1.33	0.25
Digestive Disease	11	9	3.27	1.36-7.88	7.81	0.0052
Circulatory Disease	100	31	1.24	0.83-1.85	1.10	0.29

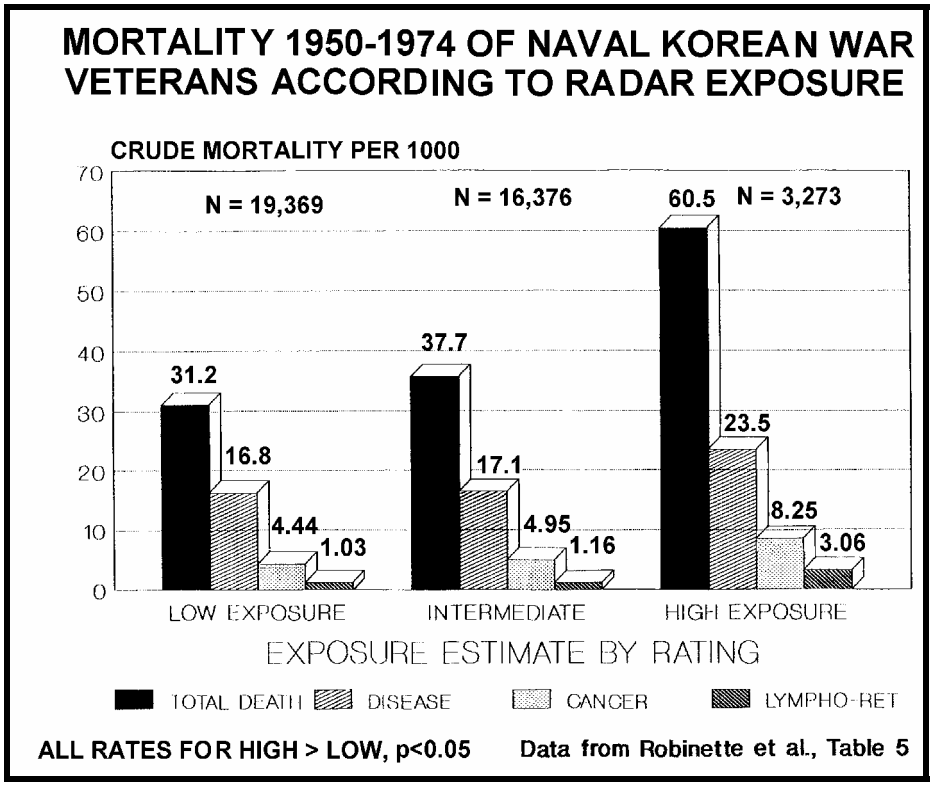

Figure 1: Naval occupations grouped by exposure category, showing dose response increases in mortality for all mortality, all disease, cancer and Lymphatic/Leukaemia. Low exposure (RM+RD), Intermediate exposure ET+FT), High exposure (AT), prepared by the late Prof John Goldsmith.

Conclusions from Occupational Studies:

Hence up to 1982 there was and established fact that RF/MW radiation is genotoxic (damaging DNA through measurements if chromosome aberrations) with exposed at

non-thermal levels. Three studies show that pulsed microwaves from radar chronic exposed people have highly significantly increased cancer in multiple body organs and a dose-response derived from a job-exposure matrix study.

Residential cancer radar-exposure studies:

In 1982 Lester and Moore published a study of radar related cancers in residential populations in Wichita, Kansas, based on a hypothesis that radar could produce cancer. This was based on the evidence of chromosome damage and the Zaret (1977) evidence of cancer rates in radar repairing workers. Because there were airport and air force base radars to the east and west of Wichita they used geographic distributions of total cancer incidence on ridges exposed to both radars, sides of hills exposed to only one radar and valleys sheltered from both radars. Mortality data was obtained from the period 1975-1977.

A significant linear trend (p=0.034) was found with incident rates (/100,000 p-yrs) of 470, 429 and 303 respectively from high to low RF/MW exposures, Figure 2. They concluded that their results established a correlation between radar exposure and cancer incidence, but that more research was necessary for causation. They were unaware of the Moscow Embassy and the Korean War study results that support and confirm their findings.

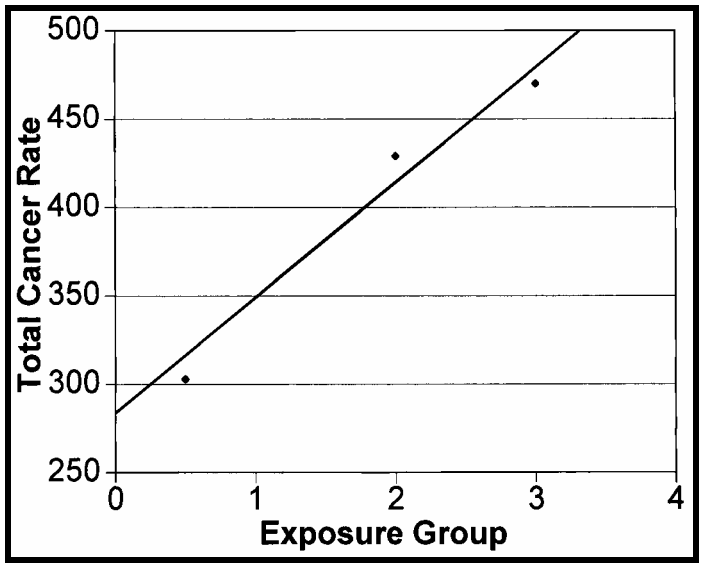

Figure 2: Cancer rates in Wichita, Kansas, for the population not exposed to a radar, exposed to one radar and exposed to two radars, at their residences, Lester and Moore (1982a), Trend p= 0.034.

They then carried out their own follow-up study to test the hypothesis that cancer mortality is associated, in part, with the possibility of chronic exposure to radar. They studied the cancer rates in 92 counties associated with US Air Force Bases (AFBs) with radars, over the period 1950-1969. They found that counties with AFBs (and radars) had significantly higher cancer rates for males (p=0.04) and females (p=0.02).

Thus the hypothesis is strongly supported by this study with significant and dose-response increases in All Cancer mortality.

Conclusions from EMR evidence up to 1982:

The evidence that RF/MW radiation is genotoxic is strong, giving a plausible mechanism for a Universal Genotoxic Cancer agent. The human evidence is very significantly higher than for Benzene in 1982, with the very high level of significance and significant dose-response relationships at occupational and at residential exposure levels. Hence, if the Universal Genotoxic Carcinogen hypothesis for RF/MW exposure was tested by IARC in 1982, using the same criteria as they did for Benzene, but with the broader view, then they should have declared RF/MW radiation a human carcinogen in 1982.

This review shows that in 1982 there was much stronger evidence for RF/MW being a Class A, human carcinogen that IARC decided for their benzene carcinogen assessment.

The most probable source of the difference is that benzene is a chemical at any seen, felt and smelled, and other chemicals are known to be carcinogens. Their full of the level of question is can this chemical also be a carcinogen. When it in multiple studies showed elevated leukaemia rates and two were large enough to show significant rates and they concluded that was a human carcinogen. On the other hand it has been strongly assumed that because radio frequencies and microwaves non-ionizing radiation therefore they can't cause cancer that can only produced heating tissues. This ignores what we have known since 1959, radiofrequency radiation mimics ionizing radiation in the way that it damages chromosomes, without any heat being involved.

Just like benzene, all their frequency and microwave radiation the evidence on since 1982 is getting stronger and stronger with many more studies showing chromosome damage and direct DNA strand breakage, and many epidemiological studies showing elevated cancer rates in occupational studies and many residential studies around radio and TV towers, several showing dose-response relationships.

While the evidence is now significantly stronger, the international ICNIRP, IARC and WHO official conclusions are get weaker and weaker. Has partly because of the long-standing tissue heating issue, the search for a specific cancer similarly to the chemical cancer approach, and the strong influence of industry which tends to leave to dismissive approach and the application of higher and higher evidence thresholds levels. So the same time the general public exposures to radiofrequency radiation and microwaves busying progressively stronger and stronger and is producing high and high cancer rates in the assumed unexposed general population. It is making the epidemiological studies results of occupational exposures weaker and weaker because of the stronger effects in the assumed non-exposed group.

The strongest and most ubiquitous exposures over the last 20 years has been the exploder of a number of radio stations and TV stations, and the use of computers. Over the last 10 years it is the usage of cordless phones and the mobile phone system and the mobile phone usage. Mobile phones have already been shown to significantly increase the rates of brain cancer specially in the very large Swedish studies, Hardell et al. (1999 a,b, 2000 a,b, 2001, 2002a,b). Hardell et al. (2002b)

shows OR = 9.0, 95%CI: 1.14-71, n=12, for Astrocytoma from analogue cellphone usage.

Mobile phone usage by pregnant mothers has already been shown to double risk rate of children developing Neuroblastoma, Adj OR =2.1 (0.4-11.0), the same rate as ionizing radiation, De Roos et al. (2001). All radio frequencies exposures resulted in Adj OR = 2.8 (0.9-8.7), the highest observed risk factor.

If in 1982 or soon after the WHO/IARC had declared that radio-frequency and microwave radiation was a human carcinogen, then the modern technology would have been required to be made much safer. How would it have been allowed for over a billion people to regularly place a genotoxic carcinogen source right next to their head for long-term usage, with seriously elevating the risk of brain cancer and many other serious health effects. The failure to do so is not only putting the users at higher risks but also their children. Many millions of people who live within about 500m of cellphone base stations and are also at serious risk of getting elevated rates of many genotoxic related health effects.

Cherry (2002) has shown that there are very robust scientific evidence the natural electromagnetic signal, the Schumann Resonance signal, with a mean strength of $0.1pW/cm^2$, that as one million times lower than the cellphone base stations exposure levels within 500m, and is over a billion times lower than the cellphone exposes the user's head, being absorbed and reacted to by the brain, modulates the melatonin output and when it has altered by Solar and Geomagnetic Activity, causes modulation of cancer, cardiac, reproductive and neurological health and mortality rates in human populations. Therefore it is very strongly scientifically plausible and the failure to declare radio-frequency and microwave radiation a human carcinogen is leading to significant elevation of all these health effects in human populations especially in urban areas.

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