Eur. J. Oncol., vol. 16, n. 1, pp. 21-54, 2011

A sentinel case series of cancer patients with occupational exposures to electromagnetic non-ionizing radiation and other agents

Una serie di casi sentinella di pazienti affetti da cancro con esposizione lavorativa a radiazioni elettromagnetiche non ionizzanti e altri agenti

Yael Stein, Or Levy-Nativ, Elihu D. Richter

Unit of Occupational and Environmental Medicine, Hebrew University-Hadassah School of Medicine, Jerusalem, Israel

Summary

***Background*. There are reports that intense prolonged occupational exposure to non-ionizing radiation may increase risks for cancer. We previously have reported a sentinel cluster, of 7 workers with high exposures and short latent periods, and individual patients with brain cancer high occupational exposures and short latent periods. We present a sentinel case series (n=47, 40M, 7F) of cancer patients, referred to our medical unit with occupational exposures to non-ionizing radiation of all types. *Objectives*. Our aims were to report the findings on tumour types, age of first diagnosis, and latency, to describe their exposures and to examine the hypothesis that latencies for all tumour types (solid tissue, hematolymphatic, testicular) were coherently related to high occupational exposures starting at young ages. *Methods*. We divided the patients into groups by latency. We categorized each patient's exposures in regard to types of radiation, far or near field exposure and direct body contact. For some we had data on frequencies, for others we provided assessments. We also present the patient data categorized by age of diagnosis. We used a case-case type comparison to examine laten-**

Riassunto

***Premessa*. Esistono prove che un'esposizione lavorativa intensa e prolungata a radiazioni non ionizzanti può aumentare il rischio di cancro. Abbiamo precedentemente riportato un gruppo di casi sentinella, comprendente 7 lavoratori con alta esposizione e breve periodo di latenza, singoli pazienti con tumori del cervello ed alta esposizione lavorativa e breve periodo di latenza. Presentiamo ora una serie di casi sentinella (n=47, 40M, 7F) di pazienti affetti da cancro, che si sono presentati alla nostra unità medica, con esposizione lavorativa a radiazioni non ionizzanti di vari tipi. *Obiettivi*. Il nostro intento era di riportare i dati riguardanti i tipi di tumore, l'età alla prima diagnosi e la latenza, di descrivere le loro esposizioni ed esaminare l'ipotesi che i tempi di latenza dei vari tipi di tumore (dei tessuti solidi, ematolinfatici, dei testicoli) fossero correlati coerentemente ad alte esposizioni lavorative a partire dalla giovane età. *Metodi*. Abbiamo suddiviso i pazienti in gruppi, per latenza. Abbiamo poi classificato l'esposizione dei singoli pazienti a seconda del tipo di radiazione, vicinanza o lontananza dal campo di esposizione e contatto diretto. Per alcuni avevamo dati sulle frequenze, mentre per altri abbiamo do-**

Received/Pervenuto 30.12.2010 - Accepted/Accettato 17.1.2011
Address/Indirizzo: Dr. Yael Stein, Unit of Occupational and Environmental Medicine, Hebrew University-Hadassah School of Medicine, Jerusalem, Israel - E- mail: stein444@g.mail.com

cies for tumour types [solid, hematolymphatic (HL), testicular]. *Results*. 15 patients developed cancer with latent periods of less than 5 years and 12 patients with latent periods between 5 and 10 years. The remaining 20 patients had longer latent periods between first occupational exposure to EMF and diagnosis of cancer. 6 patients (12.7%) had multiple tumours. 12 patients (25.5%) reported cancer cases in co-workers. In the <5 years latency group there were 8 hematolymphatic cancers, 3 testicular cancers and 6 solid tumours [head & neck (including brain) and GI tract]. In all latency groups there were patients who were exposed to intense levels of electromagnetic fields (EMF), to several types of EMF, or to EMF in combination with ionizing radiation (IR) or other exposures, and patients who had direct body contact with the equipment, were in direct focus of high radiation, or worked in small, electronically dense environments. Case classification by age showed shorter latencies with younger ages, but this association is complicated by the fact that shorter latencies co-vary with younger ages especially for testicular tumours. But patients with testicular and hematolymphatic tumours had shorter latencies than those with solid tumours. *Conclusion*. Many of the patients were young and had extremely short latent periods, especially for HL and testicular cancers. The fact that latent periods for testes were very short, HL longer and solid still longer suggests a coherent and biologically plausible pattern of latency in relation to the onset of exposure to EMF and other agents. The findings strengthen the hypothesis that these exposures may possibly be the major cause of many of these tumours. The findings state the case for (1) better modelling of exposure sources and penetration into the body and (2) preventive and protective measures based on control of exposure at source, barriers, and personal protection. Eur. J. Oncol., 16 (1), 21-54, 2011

Key words: **non ionizing radiation (NIR), electromagnetic fields (EMF), occupational exposures, cancer, short latencies**

vuto fornire delle stime. Abbiamo anche presentato i dati dei pazienti classificandoli per età alla diagnosi. Abbiamo utilizzato un metodo di confronto caso per caso al fine di esaminare le latenze per tipi di tumore [solido, ematolinfatico (EL), testicolo]. *Risultati*. 15 pazienti hanno sviluppato cancro con un periodo di latenza inferiore a 5 anni e 12 pazienti con un periodo di latenza tra 5 e 10 anni. I restanti 20 pazienti presentavano periodi di latenza più lunghi tra la prima esposizione lavorativa ai campi elettromagnetici (CEM) e la diagnosi di cancro. 6 pazienti (12,7%) avevano tumori multipli. 12 pazienti (25,5%) denunciavano casi di cancro fra loro colleghi. Nel gruppo in cui la latenza era inferiore a 5 anni, ci sono stati 8 tumori EL, 3 tumori del testicolo e 6 tumori solidi [testa e collo (incluso il cervello) e tratto gastroenterico]. In tutti i gruppi di latenza erano presenti pazienti esposti a livelli intensi di CEM, a diversi tipi di CEM, o a CEM in combinazione con Radiazioni Ionizzanti (RI) o altre esposizioni, e pazienti entrati in contatto corporeo diretto con apparecchiature, che avevano lavorato in piccoli ambienti ad alta densità elettromagnetica. La classificazione dei casi per età ha mostrato periodi di latenza più brevi nelle età più giovanili, ma questa associazione è complicata dal fatto che latenze più brevi co-variano con età più giovani. Tuttavia, pazienti con tumori EL e del testicolo presentavano periodi di latenza più brevi rispetto ai pazienti con tumori solidi. *Conclusioni*. Molti dei pazienti erano giovani e presentavano periodi di latenza estremamente brevi, soprattutto per tumori EL e del testicolo. Il fatto che il periodo di latenza per i testicoli sia molto breve, per l'EL più lungo e per i tumori solidi ancora più lungo, suggerisce un andamento della latenza coerente e biologicamente plausibile in relazione all'inizio di esposizione a CEM e altri agenti. Queste conclusioni rafforzano l'ipotesi che queste esposizioni potrebbero forse essere la causa principale di molti di questi tumori. Questi dati pongono le basi per cercare (1) di definire meglio l'origine dell'esposizione e la penetrazione nel corpo e (2) misure preventive e protettive basate su un controllo dell'esposizione alla fonte, barriere, e protezione personale. Eur. J. Oncol., 16 (1), 21-54, 2011

Parole chiave: **radiazioni non ionizzanti (RNI), campi elettromagnetici (CEM), esposizione lavorativa, cancro, breve periodo di latenza**

Background

Many workers in the electronics industries, in defence-related electronic industries and in the defence forces have direct and bystander exposures to electromagnetic non-ionizing radiation: ELF - magnetic fields, RF/MW - radiofrequency and microwave radiation and other frequencies, such as high frequency voltage transients. Over the years, patients with cancer and past occupational exposures to RF/MW have come to the Hebrew University-Hadassah Unit of Occupational and Environmental Medicine for evaluation of the case for a cause-effect relationship between their diseases and their occupational exposures. In many cases, the patients were exposed to ELF or RF/MW and sometimes also to Ionizing Radiation at young ages as workers in one of these electronically dense occupational settings. In many, the latent periods have been brief.

Richter *et al.* have reported exposures and cancers in several sentinel patients (1) in a cluster of such workers (2), and in patients with brain cancer and latent periods <10 years (3). In addition, we have previously attempted to assess findings on exposure in terms of intensity and frequency and risk in the context of a model of exposure-effect relationships (4) based on data from Goldsmith's study of the Moscow Embassy (5, 6), our findings, and Szmigielski's data (7).

There are thermal and non-thermal effects of ELF and RF/MW radiation on humans. Experimental studies in human lymphocytes and endothelial cells and in rats have shown that electromagnetic irradiation can increase DNA breakage, anaploidy and chromosomal aberration, including exposures below those producing thermal effects (8-20). Experimental studies in rats have shown effects on permeability of the blood-brain barrier and neuronal damage following exposure to low levels of MW radiation from mobile phones (21, 22). Goldsmith communicated reports on chromosomal effects in workers of US Embassies in former USSR exposed to levels in the range of 7-18 uw/cm^2 (5, 6). Nordenson *et al.* reported significantly increased rates of chromatid and chromosome breaks in switchyard workers (9).

Several biologically plausible mechanisms have been suggested to explain these effects in cells and DNA. Han *et al.* summarize the known mechanisms of so-called thermal effects and describe some suggested mechanisms of non-thermal effects, which are still being researched (23). For example, it has been suggested that RF/MW may interact directly with molecules or with tissue components, changing electron conformation, altering stress proteins (heat shock proteins), or effecting function of the immune-system. Biochemical and electrophysiological effects can result in changes in the nervous, immune and cardiovascular systems, in metabolism and on hereditary factors (24-31). A recently published study by Volkow *et al.* found increased brain metabolism in regions closest to the antenna during acute cell phone exposure, which suggest that brain absorption of RF-EMFs may enhance the excitability of brain tissue (32).

Much of this information is not new, but is being rediscovered. Glaser published a 106 page report for the US Naval Medical Research Institute in 1972, in which he reviewed over 2,300 articles which assess biological responses and effects of non-ionizing radiation on humans, many of which have been called in general "microwave sickness" (33). The author classified the biological effects into 17 categories including both thermal and non-thermal effects. These include: changes in physiologic function such as blood and vascular disorders, biochemical changes (enzymes and others), metabolic, gastro-intestinal, and hormonal disorders, alterations in the nervous system, histological changes, genetic and chromosomal effects, psychological disorders, behavioural changes in animal studies, and others. Steneck, Cook *et al.* reported in 1980 about early research on the biological effects of microwave radiation, from 1940-1960 (34, 35). Much of these data was translated from research conducted in Russia or in other East European countries.

Epidemiological studies on potential effects of exposure to ELF have suggested a threshold for increased risks for leukemia from exposure to ELF magnetic fields from powerlines, at levels of 2mG (36-41).

Occupational exposure to RF/MW

As far back as 1953, McLaughin suggested a connection between leukemia and exposure to radar (42). In 1985, Milham reported a two-fold ratio of

leukemias in operators of amateur short-wave radio stations, in a case-control study based on 1961 death certificates of the male members of the American Radio Relay League (43). In a larger study in 1988 on amateur radio operators, he found a slightly elevated ratio of all-leukemia standardized mortality, with elevation of acute myeloid leukemia (44). Goldsmith presented epidemiological evidence relevant to radar (microwave) effects (6). In 1996, Szmigielski collected data on all Polish military career personnel exposed to RF/MW during a 15 year period. He compared the cancer rates by age groups to the expected ratio, and found higher morbidity rates in the alimentary tract, [Observed/Expected Ratio (OER) 3.19-3.24], brain tumours (OER=1.91) and malignancies of the haematopoietic system and lymphatic organs (OER=6.31). Of the last, the largest difference was found for chronic myelocytic leukemia (OER=13.9), acute myeloblastic leukemia (OER=8.62) and non-Hodgkin lymphomas (OER=5.82) (45). In 2001, Smigielski presented additional data on military personnel occupationally exposed to radar for a period of 20 years (7). These reports indicated that risks for cancer, notably hematolymphatic and brain were associated with exposures to radiofrequency-microwave from radar microwave in electronics, radar and communications workers.

Grayson found slight excess risk for brain tumour after exposure to electromagnetic radiation (ELF and RF/MW) in the US air force. Exposure was assessed using a job exposure matrix. Exposure to ionizing radiation was assessed using dosimetry – an association with IR was not found in this study. Military rank was found to be associated with brain tumour risk (46).

Robinette *et al.* in 1980 did not find excess mortality in 20,000 US Korean War Naval Veterans 1954-58 exposed to radar. The authors compared two cohorts exposed to radar work, electronic equipment repair technicians (defined as high exposure) and equipment operators (defined as low exposure). The study was limited by the fact that it did not have real time data on individual exposure. The authors attempted to address this limitation by assessing exposure by generating what they called a Hazard Number. This ranking scale was based on the name of occupation, length of time in the occupation (months) multiplied by power of equipment on the ship or aircraft at the time of exposure. The "Hazard Number", which describes *potential exposure*, was calculated for a percentage of the men (47).

Despite problems of exposure misclassification, the investigators identified a subgroup with higher exposures and greater risks for HL outcomes.

Groves *et al.* conducted a follow-up study of Robinette's cohort, 40 years later. Their data did again not find excess cancer, except in one high-exposure occupation group out of three, in which non-lymphocytic leukemia was significantly elevated (48). One question not examined in the follow-up study was whether there were beneficial effects over the long-term from termination of military exposure – a possibility suggested by the fact that latencies are often short. Partial reversal of risks for lung cancer following termination of exposure has been seen in asbestos workers who cease smoking and in smokers themselves, and in Israel, in divers with past exposures to pollutants in the Kishon River, above the age of 40 (49).

Degrave *et al.* in a retrospective cohort study in Belgian male military personnel exposed to anti-aircraft radars in Western Europe between 1960-1990s, found excess incidence of hematolymphatic cancers (50, 51).

Occupational exposure to high frequency voltage transients

An occupational study on exposure to high frequency voltage transients, by Milham & Morgan 2008 (52), in teachers in a California school, shows unusually high cancer incidence in the teachers at this school, strongly associated with high frequency voltage transients. The authors suggest that high frequency voltage transients may be a universal carcinogen, similar to ionizing radiation.

Exposure assessment

Szmigielski, in his classic paper from 2000 on dosimetry, outlines a system of categorization of workers into exposure groups and presents methods of calculation (53).

There is controversy concerning the validity of various methods of exposure assessment (54-56). A new job-exposure matrix defined for professions of

women in Stockholm (57), has been criticized because several specific job exposure measurements did not match measurement from these same jobs in other studies (58).

Penetration into tissues

Researchers have developed computer-derived or MRI-derived models showing the depth of penetration of RF/MW radiation into the brain. Gandhi and others have developed models for estimating and mapping diffusion and intensity of penetration into the brain from cell phones in adults and children, taking into account the size of the skull and width of the bones (23, 59, 60). Most of the studies examining the penetration of radiation into tissues have focused on the brain, in regard to cellphone exposure. Christ *et al.* have extended these models to MRI-based whole body "virtual family" models which predict intensity of penetration into more than 80 different tissue types (61, 62).

Weinberger and Richter have suggested that the frequencies for transmission and reception by cellular telephones (900 Hz and 18 Hz) exploit the head as a lossy resonator (63). It is not known whether similar interactions occur between radiation and other body tissues.

Cellphone epidemiologic research

Two major groups have been conducting epidemiological research to assess whether cellphones pose a risk of cancer. Although the latent period since beginning of use of cellphones has not been long enough to collect much data on long-term users, Hardell's groups' case control studies have consistently found associations between brain cancer of all kinds and prior prolonged use, of cell phones and cordless phones on the side of the head with the tumour, with risks increasing with for over 10 years in different age groups (64-72).

The Interphone multinational group study did not find excess risk of gliomas to the entire population from exposure to cellphones, but they too have found excess risk in a small subgroup of more heavily exposed users associated with latency and laterality. Some subgroups of the Interphone study found similar data, but some chose to interpret these findings in a subgroup of their population as inconsequential (73-79). Morgan has called attention to many methodologic problems with the Interphone study pertaining to selection biases and exposure misclassification (80, 81). A large cohort study from Denmark which used cellphone registries linked to cancer registry data, without recall bias, did not find excess cancer risk to the population, and concluded that such risk was unlikely (82). But a major setback of this study was that the researchers excluded 200,507 corporate subscriptions out of the total records of all 723,421 cellular telephone subscriptions in Denmark during the period 1982-1995, because the individual users could not be identified. In the early years of cellphone use, it is highly possible that these corporate users may have been the most intensely exposed group, and their exclusion is a potential underestimation or dilution of the risk. Another concern raised by Ahlbom *et al.* in regard to this cohort study was the fact that only 61% of a small sample of the subscribers reported use of mobile phones when responding to a questionnaire (83).

Hardell *et al.* re-evaluated their own data for risk of glioma using the Interphone protocol (84), i.e. regarding cordless phone use as a non-exposure, and concentrating on the same age-group as the Interphone study – and found that the results of this analysis were very similar to the Interphone study results. This exercise demonstrates the exposure and age range bias factors which caused underestimation of risk in the Interphone study.

Recently, the primary researcher of the Interphone study, Cardis, and the Israeli researcher, Sadetzki, published a paper in which they endorse precautionary measures and call attention to potential effects on Public Health from even a small risk at the individual level in over 4 billion people, including children, using cellphones today (85). They also acknowledge that the Interphone study, like other studies which did not find excess risk, was conducted at a time when mobile communication was still a relatively new phenomenon with low levels of use compared with today. They discuss additional possible biases. For example, the fact that most of the risk estimates were below 1 in the Interphone study, indicates a potential selection bias, and is confirmed by the observation of high refusal rates among controls. Another example, is that the median cumu-

lative call time over life study participants was very low, around 100 hours, as was the median call time, 2-2.5 hours per month, which can cause dilution of risk in analyses of 'ever use' or 'ever regular use' of mobile phones. The significant protection among light users may most likely be an artifact of the multiple biases reported by Morgan and others (80, 81).

Objectives

Our aims were (1) to report the findings on tumour types, age of first diagnosis, and latencies, in a case series of workers with occupational exposures to EMF and other agents, (2) to describe their exposures, and (3) to examine the hypothesis that latencies for all tumour types were coherently related to high occupational exposures to EMF starting at young ages in a case series of workers.

Methods

We divided the patients into groups by latency. We categorized each patient's exposures in regard to types of radiation, far or near field exposure and direct body contact. For some we had data on frequencies, for some others we provided assessments. We also present the patient data categorized by age of diagnosis. We used a case-case type comparison to examine latencies for tumour types (solid tissue, testicular, hematolymphatic).

Our initial database was a list of 106 patients who had been exposed to non-ionizing radiation of some sort in the past and had been diagnosed with various diseases.

All of these patients were referred to the Unit of Occupational and Environmental medicine over a period of 18 years, for assessment of eligibility for compensation. Some came on their own initiative. These were either occupational or environmental exposures. For this case series we included only the 53 patients with occupational exposures, and of these, we included only the 49 cancer patients. The other 4 patients: (age range 20-39) 2 had epileptic attacks, 2 had headaches, ear pain, and dizziness – one of these reported 7 co-workers with similar symptoms.

Exposures

In our past assessment of these patients for a medical opinion, we had interviewed them to assess the extent and type of their exposures to radiation. In several cases we had consulted with experts about the kind of equipment the patient had used or was indirectly exposed to in the work environment, the type of radiation emitted by it and the known short-term and long-term physical effects. The medical assessment often did not include precise measurements of duration and intensity of exposure. For this paper, an electronics engineer assessed the frequency and intensity of exposure wherever it was possible to infer these from circumstances of the work environment, in patients concerning whom we did not have measurement data. These assessments were the best available estimates, to give a general idea of the exposures, but these have to be regarded as preliminary.

Outcomes

We included only patients for whom we had clear diagnoses from their medical file– either confirmed by pathology or a clinical diagnosis signed by a physician. 2 patients were excluded because there was insufficient medical data on their diagnoses. All diagnoses were written out in ICD10 classification numbers.

In Table 1 we grouped the patients into 3 groups by the length of the latent period of their disease, <5 years latency, 5-9 years latency and latency 10+ years.

We grouped their occupational exposure sources into two categories: immediate work environment (e.g. indoor work stations, rooms) and far field, (e.g. outdoor antenna). Within the first category, we noted if the patient was exposed to equipment carried directly on his/her body or very close to the body (e.g. cell phones, radio packs attached to the body), or in a confined small environment (e.g. inside a car or aircraft). We also listed other reported exposures, e.g. solvents, pesticides, shift work, or prior illnesses such as sarcoidosis or EBV.

In Figure 1, Table 2, and Table 3 we grouped the patients by age of diagnosis. Figure 1 displays information from tables 2 and 3.

All median and average latencies were calculated by n=patients.

Table 1 - Occupational exposure of cancer patients in sentinel case series

Latency < 5 years		**Exposure: Immediate environment**	**Exposure: Background/ external - outdoors**	**Age at initial exposure & Sex**	**Duration (years)**	**Latency (years)**	**Diagnosis**
Solid							
Brain Glioma/ Astro	ELF	High-power electronic equipment & powerline >3-4 mG		25 M	2	2	C71.1 – Malignant neoplasm of brain stem. M9440/3 – Glioblastoma NOS Died
1	RF/MW	Maintenance of receiving and transmitting RF/MW equipment	Antennas				
	Other exposures	**Ionizing Radiation:** Outdoors - Direct contact with wave guides, emitting X, α, β radiation					
	Comments:	Intense exposure during 6 wks. Previously suffered head trauma. **Co-worker with glioblastoma**					
Other CNS	ELF	Generators. ELF equipment, fuse boxes, 3 years exposure		19 M	4	4	C71.6 - Malignant neoplasm of cerebellum M9470/3 – Medulloblastoma NOS
2	RF/MW	Wave conductors. Performed exposure assessments of all RF equipment	1.5 years exposure to powerful Radar 10-25 GHz, high or extreme intensity (maintenance work during transmission)				
	Comments:	Worked inside bunker					
Other Head & Neck	ELF	2 power generators near room. Fixed electrical devices 10 hrs/day		18 M	4	4	C11.9 – Malignant neoplasm of nasopharynx, unspecified
3	RF/MW		RF/MW - antennas & aircrat. Transmitting antenna opposite office.				

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

			Climbed antenna to roof. Probable high intensity exposure				
	Other exposures	**Ionizing Radiation:** Immediate Environment – handled high power Klystrons and TWT (travelling wave tubes). Other: Celluloid film (& fumes from film), welding chemicals. EBV virus					
	Comments:	Windowless room, bad ventilation					
GI – Stomach, Bowel upper/ lower, liver & gall blad.	ELF			22 M	1.5	1.5	C20 - Malignant neoplasm of rectum
							C18.2 - Malignant neoplasm of ascending colon M8140/6 - Adenocarcinoma, metastatic NOS Died
4	RF/MW	Strong transmitting RF radios RF 700-1,000 MHz with 2 antennas concealed inside car- 9-16 hrs/day. Transmitting RF equipment & screens. **Direct body contact:** Carried RF radio on waist & earphone	Many powerful transmitting antennas in close proximity				
Renal/ genito-urinary Bone chondrosar-coma	ELF			22 M	6	3	C62 - Malignant neoplasm of testis – Rt. M9085/3 – Mixed germ cell tumour (Seminoma+ embryonal carcinoma)
5	RF/MW	RF equipment. One extreme intensity accident	Transmission antennas – participated in transmission experiments <1 m from antennas			6	C41.1 - Malignant neoplasm of bone and articular cartilage – Mandible – K10.1 - Giant cell granuloma
	Comments:	3 years constant intense exposure. 3 years intermittent exposure for several wks/year					

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

Renal/ genito-urinary	ELF	Electronic equipment		18 M	3	4	C62 – Malignant neoplasm of testis M9070/3 – Embryonal Carcinoma NOS
6	RF/MW	Communications RF/MW equipment in close vicinity. Radars. 1 MHz to probably 30 GHz.	Several powerful antennas 5-8 m away				
	Other exposures	**Ionizing Radiation:** Outdoors - X-ray tubes Other: Engine smoke, grease, paints, sun exposure					
	Comments:	High to extreme exposures					
7	ELF			18 M	2.5	2.5	C62 – Malignant neoplasm of testis M9061/3 - Seminoma NOS
	RF/MW	Transmitting communications equipment. Extreme exposure radiation accident over many hours	High-power transmitters, MW 11 GHz (possibly other frequencies), high to extreme exposures 24 hrs/day				
Non-Solid							
Leukemia	ELF	Computers & screens		18 M	9 mths	9 mths	C92.0 - Acute myeloid leukaemia Died
8	RF/MW		Powerful antennas				
	Other exposures	Stress. Poor nutrition. Abscess in ankle 4 mths before diagnosis of cancer					
9	ELF			18 M	5 mths	5 mths	C91.0 - Acute lymphoblastic leukaemia
	RF/MW	Possible short exposure to RF equipment	Antennas 20m away – 24 hrs/day exposure for 2 weeks. Radar – 3 mths exposure.				

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

	Other exposures	**Ionizing radiation:** Outdoors - IR-emitting equipment – 1 mth exposure Other: Phosphated particulates					
10	ELF		Powerlines, several weeks exposure	18 M	8 mths	8 mths	C91.5 - Adult T-cell leukaemia M9827/3 - Adult T-cell leukaemia/ lymphoma
	RF/MW	Much transmitting/ receiving RF/MW equipment, including inside van. **Direct body contact:** RF equipment carried constantly on his back & earphone.	Extreme accidental exposure - directly under high-power transmitting Radar – 7 hrs.				
11	ELF			18 M	1	1	C91.0 - Acute lymphoblastic leukaemia
	RF/MW		Radars & antennas, 1-2 m away. 10 MHz -30 GHz, high to extreme intensity. 50 cm length antenna on vehicle				
	Other exposures	Diesel. Greasing chemicals. Phosphated particulates					
12	ELF	Generator for Radar		18 F	1years 5 mths	3	C91.0 - Acute lymphoblastic leukaemia Died
	RF/MW	Much RF equipment in small vicinity, 18 days/mth. Often Radar transmitting during maintenance	Radars & antennas				
	Other exposures	**Ionizing Radiation:** Direct contact with CRT displays, 3 hrs/day Other: Diesel - activation of generator for Radar					
Lymphoma	ELF	Computers & screens		18 F	1 year 4 mths	1year 4 mths	C81 – Hodgkin's disease (Chest & lungs & stomach) stage 4B
13	RF/MW	RF/MW equipment 6-12 hrs/day, 3 night shifts/week	Transmitters, high intensity Radar – sat in direct focus				
		Possibly 1 MHz - 30 GHz					

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

14	ELF			18 M	6 mths	6mth	C84.4 – Peripheral T-cell lymphoma
	RF/MW	RF/MW equipment maintenance Probably between 2 MHz - 20 GHz	Powerful transmitters, drum & dish antennas 2-5 m wide				
	Comments:	Asthma, overweight					
15	ELF	Electronic equipment in aircraft		18 M	1 year 2 mths	1y 2mth	C85.9 - Non-Hodgkin's lymphoma, unspecified type M9714/3 - Large cell (Ki-1+) lymphoma
	RF/MW	Radar operator. **Direct contact** with RF/MW equipment in aircraft	RF/MW emissions while waiting outside aircraft 3 times/week				
Total	15 cancer patients, 13 males and 2 females. 6 solid tumours, 3 testicular cancers and 8 hematolymphatic non-solid tumours. 2 patients had 2 tumours each. 4 patients died				Age (m, r) initial exposure: 18 years (18-25)		Latency (m, r)*: 1.1667 years (0.42-4)

Latency 5-9 years		**Exposure: Immediate environment**	**Exposure: Background/ external - outdoors**	**Age at initial exposure & Sex**	**Duration (years)**	**Latency (years)**	**Diagnosis**
Solid							
Other Head & Neck	ELF	Powerline ELF, up to 7.7 mG, from ceiling above desk		48 F	6	6	C41.0 - Malignant neoplasm of bones of skull and face M8200/3 – Adenoid cystic carcinoma
16	RF/MW						
	Other exposures	Chemicals lab in same corridor					
	Comments:	ELF exposure found on several floors in building. **10 workers with Ca**. in same building					

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

17	ELF			23 M	7	5	C72.4 - Malignant neoplasm of acoustic nerve M9560/0 - Neurilemmoma NOS
	RF/MW	Worked in office directly close to active Radar	**Direct contact** with high power Radar. 3y constant exposure, 4y intermittent exposures, several wks/year.			12	C07 - Malignant neoplasm of parotid gland
Breast	ELF	Main electric fuse box directly across drywall – at chest height. ELF 9-25 mG, 10 hrs/day 5-6 days/wk, 6 years.		43 F	8	6	C50 - Malignant neoplasm of breast
18	RF/MW						
	Other exposures	Possibly – **Ionizing Radiation:** CRT display tubes Other: Windowless small room, fluorescent lights					
19	ELF	7 computers, screens & much electronic equipment in small vicinity. 40 cm-1 m from body. 12-36 hrs shifts 5 days/wk.		18 F	5	5	C50 - Malignant neoplasm of breast
	RF/MW		Powerful Radars, intermittent exposures – when working in room on roof, 7 m distance, probably 1-30 GHz, high to extreme intensity				
	Other exposures	Underground bunker					
	Comments:	**Co-worker with brain cancer** (out of 6 workers)					

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

Renal/ genito-urinary	ELF	Computers & electronic equipment in small vicinity. 12-36 hrs shifts		18 M	6	6	C62 – Malignant neoplasm of testis M9061 – Seminoma NOS
20	RF/MW	RF equipment, screens	Radar 40-80 m away, including sleeping quarters				
		Probably between 1 MHz - 30 GHz, probably high exposure					
	Comments:	Underground bunker beneath Radar					
Bone chondrosar-coma	ELF	ELF emission from electric board. 2,000 mG from each modem		54 M	5	5	C41.2 - Malignant neoplasm of vertebral column M9260/3 - Ewing's sarcoma
21	RF/MW	Sat 50 cm from board containing 20 modems. Small room with servers and other RF/MW & electronic equipment, without windows					
Skin	ELF			18 M	3	9	C43 – Malignant melanoma of skin Died
22	RF/MW	RF/MW equipment in room, and inside vehicle. RF 2-160 MHz, extreme intensity					
	Comments:	3 years constant exposure & 6 years intermittent exposure					
Non-Solid							
Leukemia	ELF			14 M	7	7	C92.0 - Acute myeloid leukaemia Died
23	RF/MW	Shoulder-held RF equipment. RF & Doppler equipment 13 GHz, wave conductors.	Antennas, Radar possibly 10 MHz - 30 GHz.				

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

		Last 2 years extreme exposure intensities					
	Other exposures	Applied greasing chemicals (carcinogenic) – on antennas. Aspergillus					
	Comments:	5 years vocational school. Workers complained of alopecia					
Lymphoma	ELF			18 M	5	8	C81.1 – Hodgkin's disease, nodular sclerosis
24	RF/MW	RF equipment 14 hrs/day in small vicinity. Earphones Possibly 1 MHz - 30 GHz,	Radar, antennas, wave conductors 10m away, 8 hrs/day high intensity, 5 years				
	Other exposures	**Ionizing Radiation:** Immediate Environment - CRT screens					
	Comments:	Windowless room.					
	ELF			18 M	9	9	C81.1 – Hodgkin's disease, nodular sclerosis
25	RF/MW	Maintenance of antennas - covered. MW high and sometimes extreme exposure. Accident – extreme MW radiation					
	Other exposures	**Ionizing Radiation:** Outdoors - X-ray TWT tubes Other: oils - cooling & cleaning (111trichloroethylene). Previously – malaria					
	ELF			19 M	7	7	C83.3 Diffuse non-Hodgkin's lymphoma, Large cell
26	RF/MW	Monitors Portable high-output Radar equipment 16 GHz used 11-12 hrs/day for 4 mths, later less hrs/day. **Direct body contact**:	Antennas – climbed antennas to calibrate (10 cm distance)				

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

		with portable high-output Radar- 6 wks					
	Comments:	**5 co-workers with Ca.:** 2 lymphoma, 1 lymphoma & melanoma, 1 leukemia, 1 brain Ca.					
HL others	ELF			28 M	8	8	C90.2 - Plasmacytoma, extramedullary (Plasma B cells)
27	RF/MW	RF/MW equipment. Electronically dense work environment, including many wave guides 1 MHz - 30 GHz, high intensity	Radar - transmission source 3-20 m distance				
	Other exposure	Past – Fiberglass exposure **4 co-workers with Ca.:** breast Ca., leukemia, lymphoma, Ca. of larynx. 2 died					
Total	12 cancer patients, 9 males and 3 females. 7 solid tumours, one testicular cancer and 5 hematolymphatic non-solid tumours. One patient had 2 tumours. 2 patients died				Age (m, r) initial exposure: 18.5 years (14-54)		Latency (m, r)*: 6.5 years (5-9)

Latency 10+ y		**Exposure: Immediate environment**	**Exposure: Background/ external - outdoors**	**Age at initial exposure & Sex**	**Duration (years)**	**Latency (years)**	**Diagnosis**
Solid							
Brain Glioma/ Astro	ELF	ELF exposure above Lt. side of head (in aircraft)		18 M	23	23	C71.6-7 Malignant neoplasm of brain – parietal lobe, occipital lobe M9400/3 Astrocytoma NOS
28	RF/MW	RF/MW exposure - high intensity Probably 30 MHz to 30 GHz. 5 years continuous exposure and 18 years intermittent exposures, several wks/year	Radar with high intensity exposure				
	Other exposures	**Ionizing Radiation:** Outdoor - Cosmic rays (flight)					
	Comments:	**3 co-workers with Ca.:** Brain Ca., Kidney Ca., Melanoma. 1 died					

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

29	ELF			23 M	14	10	C71.1/3 Malignant neoplasm of brain, fronto-parietal lobe. M9382/3-Mixed glioma Recurrence as: M9401/3 – Astrocytoma, anaplastic
	RF/MW	**Direct body contact:** RF radio 700-1,000 MHz >2,000 μW/cm^2 on waist. Earphone 0.1–0.2 μW/cm^2. Cellphone exposure 5-10 hrs/day					
	Other exposures	Grease, diesel, for 5 years before EMF exposures					
	Comments:	**3 co-workers with Ca.:** kidney & inf vena cava Ca., sarcoma of calf, melanoma					
30	ELF			40 M	20	20	C71.9 - Malignant neoplasm of brain, unspecified M9440/3 - Glioblastoma NOS Died
	RF/MW	Performed calibration of Radars and RF/MW devices during transmission	Powerful Radars and transmission antennas. Often climbed antenna for maintenance, including during transmission. Extreme exposure to 500 MHz - 2,000 MHz during last 6 years				
	Comments:	**3 co-workers with Ca.:** 2 brain cancer, 1 no details. 2 died					
Meninges	ELF	Electronically dense work environment		18 F	8	27	C70.0 – Malignant neoplasm of cerebral meninges H06.2* - Dysthyroid
31	RF/MW	RF equipment 1-100 MHz & possibly other frequencies	Radar & antennas on roof				

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

		3 years constant exposure, 5 years intermittent exposure. High intensities					exophthalmos
	Other exposures	**Ionizing Radiation: Direct body contact** with many CRT display tubes, for many hrs/day. Often fell asleep leaning on screen					
Head & Neck Endocrine	ELF	Powerful computers		27 M	13	14	C75.1 - Malignant neoplasm of pituitary gland
32	RF/MW	High-power high-frequency RF/MW equipment. **Direct body contact**: handled/ developed equipment	High-frequency signal generators, high exposure 2-3 days/mth during last 3 years				
		10 MHz-18 GHz					
	Comments:	**Co-worker with Ca.:** Lt. ear neurocytoma					
33	ELF			18 M	11	11	C75.3 – Malignant neoplasm of pineal gland
	RF/MW	RF equipment. **Direct body contact**: RF telephone equipment 30-800 $\mu W/cm^2$	8-dipole antenna on vehicle				
		20 MHz to 2 GHz. High intensity					
	Comments:	3 ears constant exposure. 8 years intermittent – several wks/year. Large tumour 3 X 3 X 4 cm					
Other Head & Neck	ELF			18 M	3	21	C06.7 - Malignant neoplasm of cheek mucosa M8070/3 - Squamous cell carcinoma NOS
34	RF/MW	Worked in storehouse beneath Radar	Radar – intensity probably high				
	Other exposures	Possibly: dust, greasing materials					
35	ELF			15 M	8	25	C11.9 – Malignant neoplasm of
	RF/MW	Waveguides, power	Radars - long range,				

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

		amplifiers. Maintenance of Radar equipment.	1,200 – 2,900 MHz, high and probably occasionally extreme exposure during last 3 years. Antennas				nasopharynx, unspecified M8070/3 - Squamous cell carcinoma NOS
	Other exposures	**Ionizing Radiation:** Immediate environment and outdoors – High voltage linear beam tubes (klystrons) and oscillators (magnetrons). Other: Oil refineries close to work environment					
36	ELF			27 M	15	13	C69.3 – Malignant neoplasm of choroid C43.3 - Malignant melanoma of other and unspecified parts of face H33.2 – Serous retinal detachment
	RF/MW	Transmitting communications equipment	CM3 short-wave transmitter 20 m distance				
		RF 0.2-18 MHz, high to extreme exposures					
Lung, chest, peritoneum	ELF			18 M	33	33	C34.9 - Malignant neoplasm of bronchus or lung, unspecified M8012/3 - Large cell carcinoma NOS (metastases to spine & skull)
37	RF/MW	Mobile Radars and other RF/MW equipment. **Direct body contact**: with RF equipment inside aircrafts	Powerful Radars				
		Possibly 1 MHz - 30 GHz, high and possibly extreme intensities. 24 hrs shifts. 12 years constant exposure + 21 years intermittent exposures					
	Other exposures	**Ionizing Radiation:** Outdoors – Cosmic rays Other: 3 years photography lab materials. Aircraft accident – smoke exposure					
	Comments:	Sarcoidosis					

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

GI – Stomach, Bowel upper/low, liver & gall blad	ELF	Office in basement next to power generator >10 years		19 M	19	16	C22.3 - Angiosarcoma of liver M9133/1 - Epithelioid haemangio-endothelioma NOS
38	RF/MW	Strong transmitting RF radios – inside office and car. 0.5 MHz - 300 MHz, high to extreme exposures. **Direct body contact:** Carried RF radio	Many transmitting and receiving antennas. Radio station antennas				C34.9 - Malignant neoplasm of bronchus or lung, unspecified
Lung, chest, peritoneum 39	ELF	ELF from point welding machine. Welded 1,500 points per shift. Sat 10 cm distance from machine		36 M	17	27	C64 - Malignant neoplasm of kidney M8312/3 – Renal cell carcinoma
Renal/ /genito-urinary	RF/MW					28	C75.1 - Malignant neoplasm of pituitary gland M8140/0 - Adenoma NOS
Head & Neck Endocrine						29	C61 - Malignant neoplasm of prostate
GI-Stomach/ Bowel						30	K63.5 - Polyp of colon
	Other exposures	Many organic solvents, including perchloroethylene					
	Comments:	4 primary cancers, in 4 consecutive years					

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

Renal/ genito-urinary	ELF			20 M	10	18	C62 – Malignant neoplasm of testis M9061 – Seminoma NOS M9070/3 – Embryonal carcinoma NOS
40	RF/MW	**Direct body contact:** Handheld Radar 8-35 GHz. Probably high exposure					
	Comments:	2 years intermittent, 8 years constant exposure					
41	ELF	Electronically dense work environment		19 M	7.5	11	C62 – Malignant neoplasm of testis M9061 – Seminoma NOS
	RF/MW	RF/MW equipment	2 years - Radar. 2 powerful transmitting antennas. Intermittent exposure at office directly close to transmitting antennas 5.5 years- mostly receiving antennas, some transmitting. Intermittent extreme exposures for several days				
		Probably 1 MHz - 30 GHz. Exposure probably high with intermittent extreme exposure. Worked 12 hrs/day for 18 days/mth					
Non-Solid							
Leukemia	ELF	Technician of electronic equipment		23 M	19	19	C91.1 - Chronic lymphocytic leukaemia Died
42	RF/MW		Radar. Powerful antennas – climbed antennas to perform maintenance				
	Other exposures	Welding chemicals					
	Comments:	Long shifts					

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

43	ELF			26 M	20	13	C91.4 – Hairy-cell leukaemia (B lymphocytes)
	RF/MW		Radar 1.25-1.35 GHz, high exposure, 5-10 m distance				
	Comments:	Intermittent exposures. **3 co-workers with Ca.:** Hodgkins' lymphoma, malignant epithelioid hemangioendothelioma – Liver, Rt Testicular cancer- seminoma					
Lymphoma	ELF	**Direct contact:** Occasionally sat on the generators	Large generators in electric power stations. Intermittent exposures, several days/mth	23 F	15	15	C83.5 – Diffuse non-Hodgkin's lymphoma**,** lymphoblastic
44	RF/MW	RF equipment					
	Other exposures	Asbestos, short exposure					
	Comments:	Miscarriages, bleeding during pregnancies and fetal brain abscesses					
45	ELF	Electric technician at radio station	High power board – building's central power box	31 M	33	30	C85 - B-cell lymphoma, unspecified
	RF/MW	Fixed RF/MW equipment	31 receiving antennas. 3 powerful transmitting antennas				
	Other exposures	**Ionizing Radiation:** Outdoors - Transmitting antennas with TWT & X-ray tubes Other: Aromatic amines, acids and bases (sulfuric acid), CO, CO2. Earlier exposure to hydraulic oil					
	Comments:	**15 co-workers with Ca.:** 7M/8F, brain Ca., pancreatic Ca., prostate Ca., breast Ca. and others (12 died)					
46	ELF			19 M	25	21	C81.9 - Hodgkin's disease, unspecified
	RF/MW	Radars and RF equipment in small vicinity, high to extreme exposures	Transmitting Radars, extreme exposures			25	C83.7 - Diffuse non-Hodgkin's lymphoma. Burkitt's tumour M9687/3 - Burkitt's lymphoma NOS
	Other exposures	**Ionizing Radiation:** Outdoors - X-ray waveguide Other: 111trichloroethylene, engine fumes					

(continued)

Table 1 (continued) - Occupational exposure of cancer patients in sentinel case series

Myeloid	ELF	Many computers & screens		24 M	20	17	C92.9 - Myeloid leukaemia, unspecified
47	RF/MW	RF equipment RF 30-300 MHz. **Direct body contact:** Handheld Radar 24-36 GHz (speed detector)- often transmitting. Antenna concealed inside car	Antennas on roof				
	Other exposures	Pesticides, during 3 years pause from work. Chemical factories close to work environment					
	Comments:	**11 co-workers with Ca.**: 9 leukemias, 1 colon Ca., 1 prostate Ca. (9 died)					
Total	20 cancer patients, 18 males and 2 females. 16 solid tumours, 2 testicular cancers and 7 hematolymphatic non-solid tumours. 2 patients had 2 solid tumours. One patient had 4 solid tumours. One patient had 2 hematolymphatic tumours. 2 patients died				Age (m, r) initial exposure: 21.5 years (15-40)		Latency (m, r)*: 18.5 years (11-33)
Grand total:	**47 cancer patients, 40 males and 7 females. 55 tumours: 29 solid, 6 testicular and 20 non-solid. 5 patients had 2 primary tumours. 1 patient had 4 primary tumours. 8 patients died**				**Age (m, r) initial exposure: 19 years (14-54)**		**Latency (m, r)*: 8 years (0.417-33)**

* All latencies are calculated for the first cancer diagnosis in each patient.

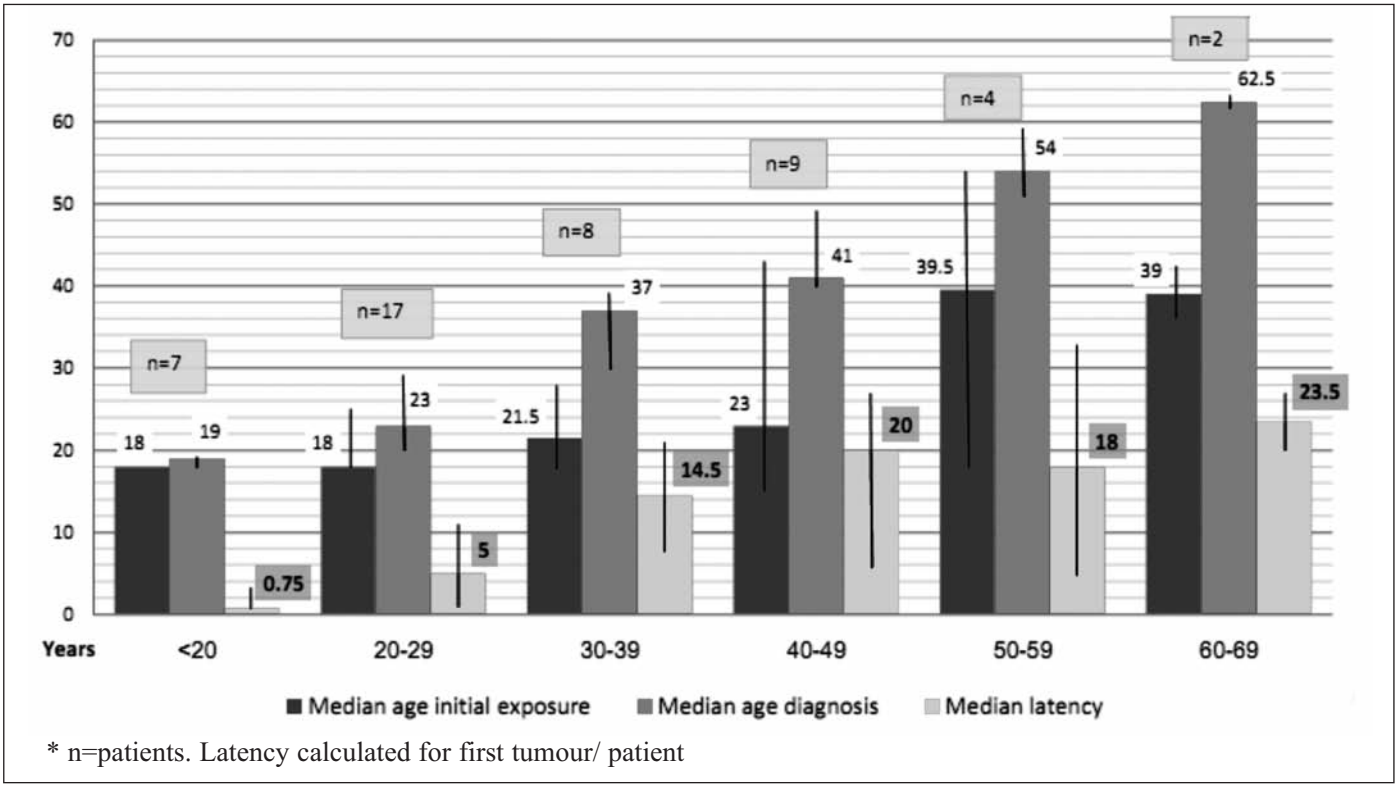

Fig. 1. 47 patients with cancer and RF/MW and other exposures: median ages of initial exposure and diagnosis, and latency*

Table 2 - Description of population

Age group (years)	n patients	M/F	Died	N tumours	Age Dx (m,r) (years)	Age initial exposure (m,r) (years)	Diagnoses
<20	7	6M/1F	1	7	19 years (18-19)	18 years	Hematolymphatic
20-29	17	15M/2F	5	20	23 years (20-29)	18 years (18-25)	5 hematolymphatic , 4 testis, 7 head & neck, 2 GI tract, 1 breast cancer, 1 skin
30-39	8	7M/1F	0	9	37 years (30-39)	21.5 years (18-28)	3 hematolymphatic , 2 testis, 2 head & neck, 1 liver, 1 lung
40-49	9	7M/2F	1	10	41 years (40-49)	23 years (15-43)	3 hematolymphatic, 5 head & neck, 1 breast
50-59	4	3M/1F	0	4	54 years (51-59)	39.5 years (18-54)	1 hematolymphatic, 1 lung, 1 head & neck (bone), 1 bone
60-69	2	2M	1	5	62.5 years (62-63)	39 years (36-42)	2 head & neck, 1 kidney, 1 prostate, 1 GI tract
Total	**47**	**40M/7F**	**8**	**55**	**29 years (18-63)**	**19 years (14-54)**	20 hematolymphatic, 6 yestis, 17 head & neck (6 brain, 4 bone, 3 endocrine), 4 GI tract, 2 breast, 2 kidney& prostate, 1 skin, 1 bone

Table 3 - Median and average latencies for different tumour types within each age group (n=patients)

<20 years Age at Diagnosis				
Totals, patients	**7**	Average **Median**	0.83 **0.75**	(0.417-1.67) **(0.417-1.67)**
Totals, for Solid tumours	**0**	Average **Median**	NA **NA**	NA **NA**
Totals, Non-solid tumours	**7**	Average **Median**	0.83 **0.75**	(0.417-1.67) **(0.417-1.67)**
20-29 years Age at Diagnosis				
Totals, patients	**17**	Average **Median**	5.35 **5**	(1.5-11) **(1.5-11)**
Totals, for Solid tumours w/o testis (head & neck (including brain), breast, GI tract)	**8**	Average **Median**	5.187 **4.5**	(1.5-11) **(1.5-11)**
Totals, Testicular tumours	**4**	Average **Median**	3.875 **3.5**	(2.5-6) **(2.5-6)**
Totals, Non-solid tumours	**5**	Average **Median**	6.8 **7**	(3-9) **(3-9)**
30-39 years Age at Diagnosis				
Totals, patients	**8**	Average **Median**	14.63 **14.5**	(8-21) **(8-21)**
Totals, for Solid tumours w/o testis (head & neck (including brain), liver)	**3**	Average **Median**	15.667 **16**	(10-21) **(10-21)**
Totals, Testicular tumours	**2**	Average **Median**	3.875 **3.5**	(2.5-6) **(2.5-6)**
Totals, Non-solid tumours	**3**	Average **Median**	13.667 **13**	(8-20) **(8-20)**
40-49 years Age at Diagnosis				
Totals, patients	**10**	Average **Median**	19 **20**	(6-27) **(6-27)**
Totals, for Solid tumours (head & neck (including brain), breast)	**6**	Average **Median**	18 **18.5**	(6-27) **(6-27)**
Totals, Non-solid tumours	**4**	Average **Median**	20.5 **20**	(17-25) **(17-25)**
50-59 years Age at Diagnosis				
Totals, patients	**4**	Average **Median**	14.7 **18**	(5-33) **(5-33)**
Totals, for Solid tumours (bones (head & neck, vertebral column), lung)	**3**	Average **Median**	14.7 **6**	(5-33) **(5-33)**
Totals, Non-solid tumours	**1**	Average **Median**	30 **30**	NA **NA**
60-69 years Age at Diagnosis				
Totals, patients	**2**	Average **Median**	23.5 **23.5**	(20-27) **(20-27)**
Totals, for Solid tumours (brain, kidney)	**2**	Average **Median**	23.5 **23.5**	(20-27) **(20-27)**
Totals, Non-solid tumours	**0**	Average **Median**	NA **NA**	NA **NA**

Table 4 - Mean latency periods by type of organ

Tumour type	n = patients	Median Latency (y, r)	N = tumours	Median Latency (y, r)
Hematolymphatic	19	7 (0.417-30)	20	7.5 (0.417-30)
Testis	6	5 (2.5-18)	6	5 (2.5-18)
Head & Neck (total)	14	13 (2-27)	17	12 (2-28)
Brain (& meninges) only	9	11 (2-27)	10	12.5 (2-28)
Head & Neck Other	5	13 (4-25)	7	12 (4-25)
Other Organs (GI tract, lung, breast, liver, kidney, prostate, skin, bone)	8	7 (1.5-33)	12	12.5 (1.5-33)
Total	**47 patients**	**8 years (0.4167-33)**	**55 tumours**	**9 years (0.4167-33)**

In Table 4 we grouped the tumours by body organ, and calculated median latencies by n=patients and by N=tumours.

Results

We present 47 cancer patients, 40 men and 7 women, with a total of 55 tumours: 29 solid, 6 testicular and 20 non-solid. The median age was 19 years (14-54) and the median latency was 8 years (0.417-33). 8 patients died (17%).

6 patients (12.7%) had multiple tumours. 5 of the 47 patients had 2 primary tumours, one had 4 tumours. In two patients, both primary tumours were diagnosed at the same time, in others, the latent period for the second tumours were longer. In only one patient the second cancer was hematolymphatic (Burkitt's lymphoma), but so was his initial diagnosis. In all the other cases, the second malignancy was a second primary solid tumour in another location. We did not count metastases in this assessment. 12 workers (25.5%) reported cases of cancer in co-workers.

Table 1: We grouped the patients by latency. The internal order in each group is by target organ of cancer & by diagnosis.

In the group with <5 years latency: there were 15 cancer patients, 13 male and 2 female. 4 patients died (3 male, 1 female). The median age of initial exposure was 18 years (18-25). The median latency was 1.667 years (0.42-4). Tumours: 6 solid tumours, 3 testicular cancers and 8 hematolymphatic non-solid cancers. 2 patients had 2 tumours each.

Distribution of tumours:

Solid tumours: 4 head and neck tumours: 2 brain-glioblastoma (patient died) and meduloblastoma of cerebellum, 1 nasopharyngeal carcinoma and one giant cell granuloma of mandible.

One patient had 2 primary gastrointestinal tumours - rectum and ascending colon (patient died).

Testicular cancers: 3 tumours of the testis (2 seminomas and one embryonal carcinoma)**.** One of these patients later developed a second primary solid tumour (mandible).

Hematolymphatic cancers: 5 leukemias: 1 acute myeloid leukemia (patient died), 3 acute lymphoblastic leukemia (1 patient died), 1 adult T-cell leukemia. 3 lymphomas: 1 Hodgkin's lymphoma, 1 non-Hodgkin's NK cell lymphoma and 1 peripheral T-cell lymphoma.

Occupational exposures:

The exposures were usually high and fairly constant, some for relatively short periods (weeks to months), others longer. 5 out of these 15 patients were exposed to ionizing radiation in addition to their exposures to EMF non-ionizing radiation. 3 other patients were exposed to constant direct body contact with radiation-emitting sources. 6 additional patients were exposed to high to extreme intensities of radiation, either in close proximity to the source,

in electronically dense or small closed environments, or in direct focus of the source. Of these, 2 were exposed to extreme levels in a radiation incident. One patient was probably exposed to less intense levels of EMF, but he reportedly suffered from stress and poor nutrition and as a result may have been immunologically compromised or susceptible. 5 patients were also exposed to other occupational hazards, e.g. engine smoke, phosphated particulates, welding chemicals. One patient reported cancer in a co-worker.

In the group with 5-9 years latency: there were 12 cancer patients, 9 males and 3 females.

2 male patients died. The median age of initial exposure was 18.5 years (14-54). The median latency was 6.5 years (5-9). Tumours: 7 solid tumours, 1 testicular cancer and 5 hematolymphatic non-solid cancers. One patient had 2 tumours.

Distribution of tumours:

Solid tumours: 3 head and neck tumours (2 in one patient): adenoid cystic carcinoma of mandible, acoustic neuroma and parotid gland tumour, 2 breast tumours, 1 Ewing's sarcoma of vertebral column, 1 melanoma (patient died).

Testicular cancers: 1 seminoma of testis.

Hematolymphatic cancers: 1 acute myeloid leukemia (patient died), 3 lymphomas: 2 Hodgkin's and 1 non-Hodgkin's, and 1 extramedullary plasmacytoma (B cell).

Occupational exposures:

3 of these 12 patients were exposed to ionizing radiation in addition to EMF. 2 other patients were in constant direct body contact with the EMF radiation sources. 5 patients worked in small windowless rooms, or underground rooms, or were exposed inside a small space such as a vehicle. 4 workers were also exposed to other occupational hazards, e.g. greasing chemicals, oils. 4 patients reported cases of cancer in co-workers.

In the group with 10+years latency: there were 20 cancer patients, 18 males and 2 females. 2 male patients died. The average age of initial exposure was 23.1 years (15-40), and the average latency was 19.35 years (11-33). Tumours: 16 solid tumours, 2 testicular cancers and 7 hematolymphatic non-solid cancers. One patient had 4 solid tumours. One patient had 2 hematolymphatic tumours.

Distribution of tumours:

Solid tumours: 10 head and neck tumours: of these 3 brain neoplasms – 2 astrocytomas and 1 glioblastoma multiforme (patient died), 1 meningioma, 1 melanoma of choroid (eye), 2 adenomas: pituitary and pineal, 1 carcinoma of cheek, and 1 nasopharyngeal carcinoma. Other cancers: 2 neoplasms of testis: 1 seminoma and 1 mixed seminoma/ embryonal carcinoma, 1 non-small cell lung cancer, 1 patient with epithelioid haemangioendothelioma of both liver and lung, and one patient with 4 tumours: renal cell carcinoma, pituitary adenoma, prostate cancer and polyp of colon.

Hematolymphatic cancers: 2 leukemias – 1 chronic lymphocytic leukemia (patient died) and 1 hairy cell leukemia (B lymphocytes). 3 lymphomas – 1 diffuse non-Hodgkin's lymphoma, 1 Hodgkin's and 1 B-cell lymphoma. 1 myeloid leukemia.

Occupational exposures:

6 of these 20 patients were exposed to ionizing radiation in addition to EMF. 8 patients had direct body contact with EMF radiation sources, and at least 9 patients worked in dense, small environments. 9 workers were also exposed to other occupational hazards, e.g. solvents, oils, engine smoke. 7 patients (35% of 10+years latency group) reported cases of cancer in several co-workers.

Figure 1 and Tables 2 and 3 present data grouped by the patients' age at diagnosis.

Figure 1 presents, for each age group (<20 years, 20-29 years, 30-39 years, 40-49 years, 50-59 years, and 60-69 years), the median age of initial exposure to EMF, median age of diagnosis, and median latencies. The data on latency by age group is also shown in Table 2.

Table 2 presents basic information on the population.

Table 3 presents the median and average latencies for different tumour types within each age group (n=patients).

Age group <20 years: 7 patients, 6M/1F. 1 patient died. The median age at diagnosis was 19 years (18-19). The median age at initial exposure was 18. All 7 patients had hematolymphatic non-solid tumours.

Latencies for all tumour types ranged between 5-16 months, with an average latency of 0.83 years, (10 months) and a median of 0.75 years – notably less than one year.

Age group 20-29: 17 patients. 15M/2F. 5 patients died. Median age of diagnosis was 23 years (20-29). Median age of initial exposure was 18 years (18-25).

N tumours: 20-5 hematolymphatic, 4 testis, 7 head & neck, 2 GI tract, 1 breast cancer, 1 skin. 12 patients had solid tumours, of these 4 testicular tumours, which were grouped separately. 5 patients had hematolymphatic tumours.

The average latency for all tumour types was 5.35 years, (1.5-11). The median latency was 5 years.

The average and median latencies for the solid tumours were 4.643 years and 4 years (1.5-11). The average and median latencies for the testicular tumours were 3.875 years and 3.5 years (2.5-6). The average and median latencies for the non-solid tumours were 7.167 years and 7.5 years (3-9).

Age group 30-39: 8 patients. 7M/1F. No patient died. Median age at diagnosis was 37 years (30-39). Median age of initial exposure was 21.5 years (18-28).

N tumours: 9-3 hematolymphatic, 2 testis, 2 head & neck, 1 liver, 1 lung.

5 patients had solid tumours, of these 2 had testicular cancer. 3 patients had hematolymphatic cancers.

The average and median latencies for all tumour types were 14.63 years and 14.5 years (8-21). The average and median latencies for solid tumours (excluding testis) were 15.667 years and 16 years (10-21). The average and median latencies for testicular cancers were 3.875 years and 8.5 years (2.5-6) – notably very short. The average and median latencies for non-solid tumours were 13.667 years and 13 years (8-20).

Age group 40-49: 9 patients, 7M/2F. 1 patient died. Median age at diagnosis was 41 years (40-49). Median age of initial exposure was 23 years (15-43).

N tumours: 10-3 hematolymphatic, 5 head & neck, 1 breast.

6 patients had solid tumours. 4 patients had hematolymphatic tumours.

The average and median latencies for all tumour types were 19 years and 20 years (6-27). The average and median latencies for solid tumours were 18 years and 18.5 years (6-27). The average and median latencies for non-solid tumours were 20.5 years and 20 years (17-25).

Age group 50-59: 4 patients, 3M/1F. No patient died. Median age at diagnosis was 54 years (51-59). Median age of initial exposure was 39.5 years (18-54).

N tumours: 4-1 hematolymphatic, 1 lung, 1 head & neck (bone), 1 bone.

3 patients had solid tumours and 1 patient had hematolymphatic cancer.

The average and median latencies for all tumour types were 14.667 years and 18 years (5-33). The average and median latencies for solid tumours were 14.667 years and 6 years (5-33). The average and median latencies for non-solid tumours were 30 years (1 patient).

Age group 60-69: 2 patients, M, 1 patient died. Median age of diagnosis was 62.5 years (62-63). Median age of initial exposure was 39 years (36-42). The average latency was 23.5 years, (20-27).

N tumours: 5-2 head & neck, 1 kidney, 1 prostate, 1 GI tract.

Both patients had solid tumours. The average and median latencies for all tumour types were 23.5 years (20-27). The average and median latencies for solid tumours were the same.

Table 4 presents median latency periods for patients and for tumours, categorized by body organ.

19 patients had hematolymphatic non-solid cancers. The median latency for this group of patients was 7 years (0.417-30). Counting by number of tumours, there were 20 hematolymphatic cancers in this case series. The median latency for this type of cancer was 7.5 years (0.417-30).

6 patients had testicular cancer – which we categorize as semi-solid. The median latency for this group of patients was 5 years (2.5-18). The number of tumours and latency per N were the same. These were the shortest latencies in this table.

14 patients had cancer in the head and neck area of the body. The median latency for this group of

patients was 13 years (2-27). Counting by number of tumours, there were 17 head and neck cancers in this case series. The median latency for cancer of the head & neck was 12 years (2-28).

In this group, 9 patients had brain cancer (including meninges). The median latency for these patients was 11 years (2-27). There were 11 tumours in this group. The median latency for brain cancers was 12.5 (2-28).

5 patients had cancer of the head & neck area but not in the brain. The median latency for this group of patients was 13 years (4-25). The median latency for the 7 tumours in this group was 12 (4-25).

8 patients had cancer in other solid organs of the body (GI tract, lung, breast, liver, kidney, prostate, skin, and bone). The median latency for these patients was 7 years (1.5-33). Counting by tumours, there were 12 cancers in other solid organs in this case series. The median latency was 12.5 (1.5-33).

Discussion

The above findings report high exposures, many young patients, many with short latent periods and some with tumours in "hot parts" of body (more exposed or more vulnerable or both) – hematolymphatic, testes, head & neck – some patients with multiple primaries.

Their exposures were to sources which were usually in the immediate work environment – i.e. in the same room, or from point sources such as antennas just beyond the immediate work environment. The exposure was to RF/MW as well as ELF frequencies, and occasionally possibly to IR. In some of the patients there were additional exposures to toxic agents, such as solvents.

Short latencies are a recognized indicator of intense exposures (86-90). In the absence of a common genetic predisposition, multiple primaries are also a sentinel indicator of high environmental exposures (91).

Figure 1 suggests a dose-response-age association. Patients exposed at an earlier age had shorter latencies. These young patients developed mostly hematolymphatic cancers, which are more prevalent in general in younger ages, but some of these cancers are extremely rare types (for example, NK-cell lymphoma in patient no.10). Other young patients developed testicular cancer. Case classification by age showed shorter latencies with younger ages, but this association, is complicated by the fact that shorter latencies co-vary with younger ages especially for testicular tumours. We will report the breakdown of latency by age at first exposure in a subsequent communication. In the past, an 18 year old diagnosed with cancer could not have had a latency of over a year or two, since he or she would have been in school and not working with electronic equipment. But with the recent introduction of Wi-Fi into schools, personal computers for each pupil in many schools, high frequency voltage transients measured in schools – as well as the population-wide use of cellphones, cordless phones, exposure to cellphone towers, residential exposures to RF/MW from Smart Meters and other "smart" electronic equipment at the home and possibly also ELF exposures to high power generators and transformers – young people are no longer free from exposure to EMF. This raised background exposure, even if at lower intensities, may possibly raise their susceptibility to high intensity occupational exposures encountered later, at work.

The diverse range of cancers presented in this sentinel case series suggests that the classic association in the scientific literature between specific carcinogenic agents and specific target organs may be too narrow. We question whether this model is adequate to capture the possibility of a more generalized effect, either by initiation or promotion or progression regarding the potential effects of EMF radiation on body tissue, or in other words, EMF may be a universal carcinogen, similar to ionizing radiation.

The most striking finding was the short latency period of the testicular tumours and HL tumours relative to other solid tumours. In younger patients, the rapidly dividing sperm cells may be uniquely susceptible to carcinogens, including all kinds of radiation. Young men between the ages of 20-34 are the peak age group of risk for testicular cancer, but many studies show a worldwide increase in incidence in recent years. One review reports data from various studies suggesting increased risks from occupational exposures to EMF - although the reviewers interpret these data as not supportive of a cause-effect association (92, 93).

There is a coherent pattern between the onset of occupational exposure and latent periods for the three

classes of tumours we have defined in this case series (hematolymphatic, testicular, and solid). Testicular cancer latencies were very short, hematolymphatic latencies were longer and solid tumour latencies still longer. When the group of solid tumours was divided into head & neck versus other tumours, the latencies for the other organs were not much longer than for the hematolymphatic cancers, but notably longer than the latencies for the testicular cancers. The latencies for head and neck tumours were the longest. The most plausible explanation for this pattern could be that EMF is the common source and possibly a major cause of many of these tumours.

We suggest that the intensity, direction and depth of penetration of electromagnetic non-ionizing radiation are physical variables which should be investigated regarding the possibility of development of cancer in body tissues.

In the short latency-high exposure group, fewer patients reported co-workers with cancer, compared with the >10 years latency group (35%). This finding could be attributed to the older age of the latter group, but it could also suggest the possibility that the patients in the short-latency group were more intensely exposed than their colleagues. However, the young workers may not have worked for a long enough time for co-workers to have cancers ascertained. We did not have information on the total number of co-workers in each workplace.

Limitations and implications for further work

This case series is based on relatively small numbers and it does not represent the general population nor the population of cancer patients in Israel's cancer registry. It represents a small sub-group of workers with cancer, many young, nearly all males and many with short latent periods, who were occupationally exposed to intense levels of radiation, and included a non-trivial proportion with multiple primaries. It does not include children or a representative number of older adults. It does not include people with chronic disease or other known susceptibilities.

We lack epidemiologic evidence of an analytic nature regarding causation for workers with dense exposure, or for the general population. But the case-case comparisons showing a coherent pattern of latency in relation to the onset of exposure do strengthen the hypothesis we have raised that the exposures were the common factor. The findings are disturbing and cannot be ignored and state the case for further investigation regarding a possible connection between these exposures and initiation or promotion of cancer, for individual workers exposed to high levels of EMF radiation.

We did not present full information on the exposures, i.e. frequencies, intensities, direction. There is a need to re-interview all of our patients in the future, wherever possible to obtain data from measurements and in other cases use dosimetry assessments by a qualified and experienced professional.

Another limitation in our assessment of exposures to EMF is that in most cases, we did not have and therefore did not present data on the concurrent use and exposure to cellphones and cordless phones by these patients.

We did not have data on total numbers of co-workers in each workplace.

We did not have full data and therefore did not present information on the patients' smoking habits. But, as shown by Blair (94), these do not reduce or account for the severity of risks for cancer associated with intense occupational exposures to carcinogens. This statement holds especially true for younger patients, given the relatively long latencies for cigarette smoking.

There is a need for an exposure assessment matrix which takes into consideration hypothesized situations of exposures and their effect. For example, what is the effect of direct body contact with a radiation source (such as an RF transmitter carried on the body) on body organs right next to the source.

In 1967, one year after the United States adopted the standard for military and occupational exposure, Pollack and Healer reviewed literature from conferences and English translations of research from East Europe and former Soviet Union. They concluded that the U.S. Military guidelines were too high (1 mW/cm^2 averaged over 0.1 hour) and recommended that these should be lowered to approach those of the former USSR (0.01 mW/cm^2) in order to protect public health, and particularly where hazards to non-controlled personnel may be involved (95). The authors were concerned that protection of the general public was barely discussed and no public standards were set because microwaves were viewed as radar and radar

was viewed as a military and industrial issue. At the time these standards were set, most people were unlikely to be exposed to microwave radiation. Today, microwave radiation exposures are ubiquitous.

During the 1970s and the 1980s, Glaser, Steneck and Cook *et al.* raised these issues again, with no results (33, 34).

The newly published Seletun Statement (96) updates the conclusions of the previously published Bioinitiative Report (97) and Benevento Resolution (98) and again calls for lowering required thresholds of EMF exposure to the public to below levels in which risk has been shown, even if a cause effect relationship has not been fully proven.

The authors state the case for action for preventive measures - the current exposures are already too high to protect people from health harm. The combined effect of cell phones, cordless phones, cell towers, Wi-Fi and wireless internet, has already been shown to raise the risk for billions of people around the world for cancer and for neurobehavioural, reproductive and developmental impairments. Evidence suggests there are special risks for persons with occupational exposures to RF/MW as well as ELF, and recent evidence also suggests risks from exposure to high frequency voltage transients (52).

The Seletun Statement recommends precautionary action given the health effects we are seeing already – avoidance of use of cell and cordless phones by pregnant women and children of all ages, halting the population-wide use of Wi-Fi routers, DECT phones and other wireless devices like baby monitors and investigation of less harmful options and setting of new, biologically-based exposure limits to guide new technology development toward solutions that are not harmful to health.

A report just published by the Committee on the Environment, Agriculture and Local and Regional Affairs of the Council of Europe, acknowledges the need to revise the current threshold values: “One must respect the precautionary principle and revise the current threshold values; waiting for high levels of scientific and clinical proof can lead to very high health and economic costs, as was the case in the past with asbestos, leaded petrol and tobacco” (99).

The Russian National Committee On Non-ionizing Radiation Protection (RNCNIRP) has recently published a resolution regarding the use of mobile phones by children: “Urgent measures must be taken because of the inability of children to recognize the harm from the mobile phone use and that a mobile phone itself can be considered as an uncontrolled source of harmful exposure” (100).Implications for further work: the risks in the sentinel case series we have reported represent the risks in a subpopulation of workers with intense and sometimes prolonged exposures to EMF, sometimes with other exposures as well (IR, solvents). The findings state the case for (1) better modelling of exposure sources and penetration into the body, and (2) preventive and protective measures based on control of exposure at source, barriers, and personal protection.

A model for studying the penetration of radiation into the body is the “Virtual Family” (62). The Virtual Family consists of eight highly detailed anatomical whole-body models of adults and children of various ages, and includes pregnant women at 1, 3 and 9 months of gestation. The models or numerical phantoms are based on high-resolution MR images of healthy volunteers. All of their approximately 80 organs and tissues are represented by three-dimensional CAD objects yielding a high level of detail. The models have a body mass and height which is typical for their age range. The Family is available for research and is used by over 200 research facilities.

The widespread and increasing background exposures to both voluntary (cell phones) and non-voluntary sources of non-ionizing radiation as listed above - residential, occupational or school exposures, may soon preclude the possibility of so called controlled studies, and the situation can be expected to become more complex in coming years (101). Biologically based studies on sentinel occupational groups with high exposures offer the potential for providing risk assessments for large populations with “low” exposures.

A general comment: from occupation to community: population-wide exposure to EMF recalls the story of population-wide exposure to lead in gasoline. In the 1970s, a mere 35 years ago, we were arguing as to whether or not an everyday blood lead level of 30 μg/dl was a health risk. By 1979, that threshold dropped to 20 μg/dl and thereafter through the 1980s to 10 μg/dl for children, and now we are not certain whether there is a threshold below which there are no discernable adverse neurobehavioral

effects, especially for *in utero* and newborn exposures. We now know, in retrospect, that the entire urban population, notably children, were receiving exposures which were impairing their IQ, emotional well being, and long-term growth and development. These findings led to the elimination of lead from gasoline. In retrospect, the world was not heeding the early warnings regarding an impending population-wide hazard with disastrous effects. Epidemiologic evidence for hard-core proof of excess risk may not be available yet, but we suggest that in the case of population-wide exposure to RF/MW, the situation may be similar, with one exception: the warnings may no longer be early.

Acknowledgements

We thank our patients for allowing us to publish their data. We are indebted to Engineer Michael Peleg for his assistance in assessing frequencies and intensities of the patients' exposures. We thank Dr. Devra Davis of www.ehtrust.org and Prof. Richard Laster for their advice and encouragement. We thank our peer reviewers for their useful comments and suggestions.

Many forgotten scientific papers have recently been retrieved and have been made accessible to the public on the website of Dr. Magda Havas, from the archives of Dr. Zorach ("Zory") R. Glaser, a researcher who performed research of RF/microwave bioeffects for many years at the U.S. Navy and at the U.S. Public Health Service. By 1971, Dr. Glaser had collected over 2,300 references to documents that detailed the biological effects of radiofrequency and microwave radiation from various technologies. For additional information see: http://www.zoryglaser.com/the-history-of-microwave-radiation/ and http://www.magdahavas.com/2010/02/25/introduction-to-from-zorys-archive/

Comment

One of the authors (EDR) provided expert opinions for many of the patients.

References

1. Richter ED, Barach P. Occupation and Environment in Internal Medicine: Sentinel events and trigger questions. Mt Sinai J Med 1995; 62 (5): 390-400.
2. Richter ED, Ben-Michael E, Berman T, *et al.* Cancer in radar technicians exposed to RF/MW: sentinel episodes. Int J Occup Environ Health 2000; 75: 187-93.
3. Richter ED, Berman T, Levy O, *et al*. Brain cancer with induction periods of less than 10 years in young military radar workers. Arch Env Health 2002; 57(4): 270-2.
4. Richter ED. Cancer risks associated with intensity of RF/MW exposures dose-response: data for 3 studies. Presented at an ISEE conference in Vancouver, 2003.
5. Goldsmith JR. Epidemiologic Evidence of Radiofrequency Radiation (Microwave) Effects on Health in Military, Broadcasting, and Occupational Studies. Int J Occup Environ Health 1995; 1 (1): 47-57.
6. Goldsmith JR. Epidemiologic Evidence Relevant to Radar (Microwave) Effects. Env Health Perspectives 1997; 105: suppl 6: 1579-87.
7. Szmigielski S, Sobiczewska E, Kubacki R. Carcinogenic potency of microwave radiation: overview of the problem and results of epidemiological studies on Polish military personnel. Eur J Oncol 2001; 6 (2): 193-9.
8. D'Andrea JA, Gandhi OP, Lords JL. Behavioral and thermal effects of microwave radiation at resonant and nonresonant wavelengths. Radio Sci 1977; 12: 251-6.
9. Nordenson I, Mild KH, Nordström S, *et al*. Clastogenic effects in human lymphocytes of power frequency electric fields: in vivo and in vitro studies. Radiat Environ Biophys 1984; 23 (3): 191-201.
10. Cohen MM, Kunska A, Astemborski JA, *et al*. The effect of low-level 60-Hz electromagnetic fields on human lymphoid cells. II. Sister-chromatid exchanges in peripheral lymphocytes and lymphoblastoid cell lines. Mutat Res 1986; 172 (2): 177-84.
11. Khalil AM, Qassem W. Cytogenetic effects of pulsing electromagnetic field on human lymphocytes in vitro: chromosome aberrations, sister-chromatid exchanges and cell kinetics. Mutat Res 1991; 247 (1): 141-6.
12. Garaj-Vrhovac V, Fucić A, Horvat D. The correlation between the frequency of micronuclei and specific chromosome aberrations in human lymphocytes exposed to microwave radiation in vitro. Mutat Res 1992; 281 (3): 181-6.
13. Ahuja YR, Vijayashree B, Saran R, *et al*. In vitro effects of low-level, low-frequency electromagnetic fields on DNA damage in human leucocytes by comet assay. Indian J Biochem Biophys 1999; 36 (5): 318-22.
14. Mashevich M, Folkman D, Kesar A, *et al*. Exposure of human peripheral blood lymphocytes to electromagnetic fields associated with cellular phones leads to chromosomal instability. Bioelectromagnetics 2003; 24 (2): 82-90.
15. Nylund R, Leszczynski D. Mobile phone radiation causes changes in gene and protein expression in human endothelial cell lines and the response seems to be genome- and proteome-dependent. Proteomics 2006; 6 (17): 4769-80.
16. Mazor R, Korenstein-Ilan A, Barbul A, *et al*. Increased levels of numerical chromosome aberrations after in vitro exposure of human peripheral blood lymphocytes

to radiofrequency electromagnetic fields for 72 hours. Radiat Res 2008; 169 (1): 28-37.
17. REFLEX. Final report. Risk evaluation of potential environmental hazards from low frequency electromagnetic field exposure using sensitive in vitro methods. 31 May 2004. Key Action 4 "Environment and Health". Quality of Life and Management of Living Resources. European Union.
18. Cherry N. Evidence that Electromagnetic Radiation is Genotoxic: The implications for the epidemiology of cancer and cardiac, neurological and reproductive effects. 2002 Aug. Extended from a paper presented to the conference on possible health effects on health of radiofrequency electromagnetic fields, 29th June 2000. European Parliament, Brussels.
19. Lai H, Singh NP. Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. Bioelectromagnetics 1995; 16 (3): 207-10.
20. Lai H, Singh NP. Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. Int J Radiat Biol 1996; 69 (4): 513-21.
21. Nittby H, Brun AE, Eberhardt J, *et al*. Increased blood-brain barrier permeability in mammalian brain 7 days after exposure to the radiation from a GSM-900 mobile phone. Pathophysiology 2009; 16 (2-3): 103-12.
22. Salford LG, Brun AE, Eberhardt JL, *et al*. Nerve cell damage in mammalian brain after exposure to microwaves from GSM mobile phones. Environ Health Perspect 2003; 111 (7): 881-3; discussion A408.
23. Han YY, Gandhi OP, DeSalles A, *et al*. Comparative assessment of models of electromagnetic absorption of the head for children and adults indicates the need for policy changes. ICEMS Monograph. Eur J Oncol - Library Vol. 5. National Institute for the Study and Control of Cancer and Environmental Diseases "Bernardino Ramazzini". Bologna, Italy, Mattioli 1885 spa, 2010.
24. Bernhart JH. Non-Ionizing radiation safety: radiofrequency radiation, electric and magnetic fields physics on medicine and biology. Phys Med Biol 1992; 37: 807-44.
25. Tan DX, Manchester LC, Terron MP, *et al*. Melatonin protects human blood lymphocytes from radiation-induced chromosome damage. Mutat Res 1995; 346: 23-31.
26. Burch JB, Reif JS, Yost MG, *et al*. Reduced excretion of a melatonin metabolite among workers exposed to 60 Hz magnetic fields. Am J Epidemiol 1999; 150 (1): 27-36.
27. Altpeter ES, Röösli M, Battaglia M, *et al*. Effect of short-wave (6-22 MHz) magnetic fields on sleep quality and melatonin cycle in humans: the Schwarzenburg shut-down study. Bioelectromagnetics 2006; 27 (2): 142-50.
28. Leszczynski D, Joenväärä S, Reivinen J, *et al*. Non-thermal activation of the hsp27/p38MAPK stress pathway by mobile phone radiation in human endothelial cells: Molecular mechanism for cancer- and blood-brain barrier-related effects. Differentiation 2002; 70: 120-9.
29. Lai H, Singh NP. Melatonin and N-tert-butyl-alpha-phenylnitrone block 60-Hz magnetic field-induced DNA single and double strand breaks in rat brain cells. J Pineal Res 1997; 22 (3): 152-62.
30. Dmoch A, Moszczynski P. Levels of immunoglobulin and subpopulations of T lymphocytes and NK cells in men occupationally exposed to microwave radiation in frequencies of 6-12GHz. Med Pr 1998; 49 (1): 45-9.
31. Cossarizza A, Angioni S, Petraglia F, *et al*. Exposure to low frequency pulsed electromagnetic fields increases interleukin-1 and interleukin-6 production by human peripheral blood mononuclear cells. Exp Cell Res 1993; 204 (2): 385-7.
32. Volkow ND, Tomasi D, Wang GJ, *et al*. Effects of Cell Phone Radiofrequency Signal Exposure on Brain Glucose Metabolism. JAMA 2011; 305 (8) Reprinted.
33. Glaser ZR. Bibliography of reported biological phenomena ('effects') and clinical manifestations attributed to microwave and radio-frequency radiation. Naval Medical Research Institute. 1972. MF12.54.015-004B, Report No. 2, revised. 106 pp.
34. Steneck NH, Cook HJ, Vander AJ, *et al*. The Origins of U.S. Safety Standards for Microwave Radiation. Science 1980; 208.
35. Cook HJ, Steneck AJ, Vander AJ, *et al*. Early Research on the Biological Effects of Microwave Radiation: 1940-1960. Annals of Science 1980; 37: 323-51.
36. Wertheimer N, Leeper E. Electrical wiring configurations and childhood cancer. Am J Epidemiol 1979; 109: 273-84.
37. Savitz DA, Wachtel H, Barnes FA, *et al*. Case-control study of childhood cancer and exposure to 60Hz magnetic fields. Am J Epidemiol 1988; 128: 21-8.
38. London SJ, Thomas DC, Bowman JD, *et al*. Exposure to residential electric and magnetic fields and risk of childhood leukemia. Am J Epidemiol 1991; 134 (9): 923-37.
39. Hardell L, Holmberg B, Malker H, *et al*. Exposure to extremely low frequency electromagnetic fields and the risk of malignant diseases - an evaluation of epidemiological and experimental findings. Eur J Cancer Prevention 1995; 4 Suppl 1: 3-107.
40. Feychting M, Schulgen G, Olsen JH, *et al*. Magnetic fields and childhood cancer - a pooled analysis of two Scandinavian studies. Eur J Cancer 1995; 31A (12): 2035-9.
41. Green LM, Miller AB, Villeneuve PJ, *et al*. A case-control study of childhood leukemia in southern Ontario Canada and exposure to magnetic fields in residences. Int J Cancer 1999; 82: 161-70.
42. McLaughin JR. A survey of possible health hazards

from exposure to microwave radiation. Hughes Aircraft Corp. Culver City, CA. 1953; 84.
43. Milham S Jr. Silent keys; leukemia mortality in amateur radio operators. Lancet 1985; 1: 812.
44. Milham S. Increased Mortality in Amateur Radio Operators Due to Lymphatic and Hematopoietic Malignancies. Am J Epidemiol 1988; 127 (1).
45. Szmigielski S. Cancer morbidity in subjects occupationally exposed to high frequency (radiofrequency and microwave) electromagnetic radiation. Sci Total Environ 1996; 180: 9-17.
46. Grayson JK, Lyons TJ. Brain cancer, flying and socioeconomic status: a nested case-control study of USAF aircrew. Aviat Space Environ Med 1996b; 67 (12): 1152-4.
47. Robinette CD, Silverman C, Jablon S. Effects upon health of occupational exposure to microwave radiation (radar). Am J Epidemiol 1980; 112 (1): 39-53.
48. Groves FD, Page WF, Gridley G, *et al*. Groves et al: Cancer in Korean war navy technicians: mortality survey after 40 years. Am J Epidemio 2002; 1355: 810-8.
49. Richter ED, Friedman LS, Tamir Y. Rebuttal to Amitai: Cancer Risk to Naval Divers in Kishon Questioned. Letter. Environ Health Perspect 2003; 111 (12): 630-6.
50. Degrave E, Autier P, Grivegnée AR, *et al*. All-cause mortality among Belgian military radar operators: A 40-year controlled longitudinal study. Eur J Epidemiol 2005; 20: 677-81.
51. Degrave E, Meeusen B, Grivegnée AR, *et al*. Causes of death among Belgian professional military radar operators: a 37-year retrospective cohort study. Int J Cancer 2009; 124: 945-51.
52. Milham S, Morgan LL. A new electromagnetic exposure metric: high frequency voltage transients associated with increased cancer incidence in teachers in a California school. Am J Ind Med 2008; 51 (8): 579-86.
53. Szmigielski S, Kubacki R, Ciolek Z. Application of dosimetry in Military epidemiological studies. Rad Freq Radiat Dosimetry 2000; 459-71.
54. Grayson JK. Radiation exposure socioeconomic status and brain tumor risk in the US Air Force: a nested case-control study. Am J Epidemiol 1996; 143: 480-6.
55. Cooper TG, Allen SG, Blackwell RP, *et al*. Assessment of occupational exposure to radiofrequency fields and radiation. Radiat Prot Dosimetry 2004; 111 (2): 191-203.
56. Yost M. Alternative magnetic field exposure metrics: occupational measurements in trolley workers. Radiat Prot Dosimetry 1999; 83 (1-2): 99-106.
57. Forssen UM, Mezei G, Nise G, *et al*. Occupational magnetic field exposure among women in Stockholm County, Sweden. Occup Environ Med 2004; 61: 594-602.
58. Szabó J, Mezei K, Thuróczy G, *et al*. Occupational 50 Hz magnetic field exposure measurements among female sewing machine operators in Hungary. Bioelectromagnetics 2006; 27 (6): 451-7.
59. Gandhi OP, Kang G. Some present problems and a proposed experimental phantom for SAR compliance testing of cellular telephones at 835 and 1900 MHz. Phys Med Biol 2002; 47 (9): 1501-18.
60. Fernández CR, Bulla G, Pedra AC, *et al*. Comparison of electromagnetic absorption characteristics in the head of adult and children for 1800 MHz mobile phones. Paper presented at: International Microwave and Optoelectronics Conference (IMOC 2005); Brasilia, Brazil, 2005.
61. Christ A, Kainz W, Hahn EG, *et al*. The Virtual Family - development of surface-based anatomical models of two adults and two children for dosimetric simulations. Phys Med Biol 2010; 55 (2): N23-38.
62. High-Resolution Whole-Body Human Models of the Virtual Population. Human & animal models. The Foundation for Research on Information Technologies in Society (IT'IS Foundation). http://www.itis.ethz.ch/research/virtual-population/virtual-population-project/
63. Weinberger Z, Richter ED. Cellular telephones and effects on the brain. The head as an antenna and brain tissue as a radio receiver. Med Hypotheses 2005; 64 (5): 1064.
64. Hardell L, Carlberg M. Mobile phones, cordless phones and the risk for brain tumours. Int J Oncol 2009; 5-17.
65. Hardell L, Carlberg M, Mild KH. Epidemiological evidence for an association between use of wireless phones and tumor diseases. Pathophysiology 2009; 16 (2-3): 113-22.
66. Hardell L, Carlberg M, Söderqvist F, *et al*. Meta-analysis of long-term mobile phone use and the association with brain tumours. Int J Oncol 2008; 32 (5): 1097-103.
67. Hardell L, Carlberg M, Söderqvist F, *et al*. Long-term use of cellular phones and brain tumours: increased risk associated with use for > or =10 years. Occup Environ Med 2007; 64 (9): 626-32.
68. Hardell L, Carlberg M, Mild KH. Pooled analysis of two case-control studies on use of cellular and cordless telephones and the risk for malignant brain tumours diagnosed in 1997-2003. Int Arch Occup Environ Health 2006; 79 (8): 630-6.
69. Hardell L, Carlberg M, Mild KH, *et al*. Case-control study of the association between the use of cellular and cordless telephones and malignant brain tumors diagnosed during 2000-2003. Environ Res 2006; 100 (2): 232-41.
70. Hardell L, Mild KH, Carlberg M, *et al*. Cellular and cordless telephone use and the association with brain tumors in different age groups. Arch Environ Health 2004; 59 (3): 132-7.
71. Hardell L, Mild KH, Carlberg M. Further aspects on cellular and cordless telephones and brain tumours. Int J Oncol 2003; 22 (2): 399-407.
72. Hardell L, Hallquist A, Mild KH, *et al*. Cellular and cordless telephones and the risk for brain tumours. Eur J Cancer Prev 2002; 11 (4): 377-86.

73. INTERPHONE Study Group, Cardis E, Deltour I, *et al*. Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. Int J Epidemiol 2010; 675-94.
74. Takebayashi T, Varsier N, Kikuchi Y, *et al*. Mobile phone use, exposure to radiofrequency electromagnetic field, and brain tumour: a case-control study. Br J Cancer 2008; 98 (3): 652-9.
75. Sadetzki S, Chetrit A, Jarus-Hakak A, *et al.* Cellular phone use and risk of benign and malignant parotid gland tumors - a nationwide case-control study. Am J Epidemiol 2008; 167 (4): 457-67.
76. Lahkola A, Auvinen A, Raitanen J, *et al.* Mobile phone use and risk of glioma in 5 North European countries. Int J Cancer 2007; 120 (8): 1769-75.
77. Hepworth SJ, Schoemaker MJ, Muir KR, *et al*. Mobile phone use and risk of glioma in adults: case-control study. BMJ 2006; 332 (7546): 883-7.
78. Schüz J, Bohler E, Berg G, *et al*. Cellular phones, cordless phones, and the risks of glioma and meningioma (Interphone Study Group, Germany). Am J Epidemiol 2006; 163 (6): 512-20.
79. Berg G, Spallek J, Schüz J, *et al*. Occupational exposure to radio frequency/microwave radiation and the risk of brain tumors: Interphone Study Group, Germany. Am J Epidemiol 2006; 164 (6): 538-48.
80. Morgan LL. Estimating the risk of brain tumors from cellphone use: Published case–control studies. Pathophysiology 2009; 16 (2-3): 137-47.
81. Morgan LL. Cellphones and Brain Tumors. 15 Reasons for Concern. Science, Spin and the Truth Behind Interphone. August 25, 2009. www.radiationresearch.org/pdfs/15reasons.asp
82. Schüz J, Jacobsen R, Olsen JH, *et al*. Cellular telephone use and cancer risk: update of a nationwide Danish cohort. J Natl Cancer Inst 2006; 98 (23): 1707-13.
83. Ahlbom A, Feychting M, Cardis E, *et al.* Cellular telephone use and cancer risk: update of a nationwide Danish cohort study. J Natl Cancer Inst 2007; 99 (8): 655; author reply 655-6.
84. Hardell L, Carlberg M, Hansson Mild K. Re-analysis of risk for glioma in relation to mobile telephone use: comparison with the results of the Interphone international case-control study. Int J Epidemiol: *in press*
85. Cardis E, Sadetzki S. Indications of possible brain-tumour risk in mobile-phone studies: should we be concerned? Occup Environ Med published online January 24, 2011.
86. Stark AA. Cancer hazards in the recombinant laboratory. Occup Med 1991; 6 (2): 311-21.
87. Armenian HK, Lilienfeld AM. Distribution of incubation periods of neoplastic diseases. Am J Epidemiol 1974; 99: 92-100.
88. Whittemore AS. Age distribution of human cancer from carcinogenic exposure of various intensities. Am J Epidemiol 1977; 106: 418-32.
89. Smith PG, Doll R. Mortality among patients with ankylosing spondylitis after a single treatment course with X-rays. Br Med J 1982; 284: 449-60.
90. Weinberg JA. Current approaches to the experimental investigation of chemicals in relation to cancer of the brain. Ann NY Acad Sci 1982; 381: 43-5.
91. Spratt JS, Hoag MG. Incidence of multiple primary cancers per man-year of follow up: 20-year review from the Ellis Fischel State Cancer Hospital. Ann Surg 1966; 164: 775-84.
92. Huyghe E, Matsuda T, Thonneau P. Increasing incidence of testicular cancer worldwide: a review. J Urol 2003; 170 (1): 5-11.
93. Yousif L, Blettner M, Hammer GP, *et al*. Testicular cancer risk associated with occupational radiation exposure: a systematic literature review. J Radiol Prot 2010; 30: 389–406.
94. Blair A, Steenland K, O'Berg M, *et al*. Control of smoking in occupational epidemiologic studies: methods and needs. Am J Ind Med 1988; 13: 3-4.
95. Pollack H, Healer J. Review of the Information on Hazards to Personnel from High-Frequency Electromagnetic Radiation. Institute for Defense Analyses, Research and Engineering Support Division. Internal Note. 1967. N-451, IDA/HQ 67-6211, Series B, copy 5 of 15, 15 pages.
96. Fragopoulou A, Grigoriev Y, Johansson O, *et al*. Scientific panel on electromagnetic field health risks: Consensus points, recommendations, and rationales. Scientific Meeting: Seletun, Norway, November 17-21, 2009. Rev Environ Health 2010; 25: 307-17.
97. The BioInitiative Working Group. BioInitiative Report: a rationale for a biologically-based public exposure standard for electromagnetic fields. www.bioinitiative.org
98. Belpoggi F, Blackmann CF, Blank M, *et al*. Benevento resolution 2006. Electromagn Biol Med 2006; 197-200.
99. Committee on the Environment, Agriculture and Local and Regional Affairs. The potential dangers of electromagnetic fields and their effect on the environment. Parliamentary assembly. Council of Europe. 6 May 2011. http://www.scribd.com/doc/55253905/The-Potential-Dangers-of-Electromagnetic-Fields-and-their-Effect-on-the-Environment
100. Russian National Committee On Non-Ionizing Radiation Protection. Resolution: Electromagnetic fields from mobile phones: health effect on children and teenagers. April 2011. http://www.powerwatch.org.uk/pdfs/20110514-rncnirp_resolution.pdf
101. Sage C. Tragedy of the common revisited: the high tech-high risk wireless world. Reviews on Environmental Health 2010; 25(4)