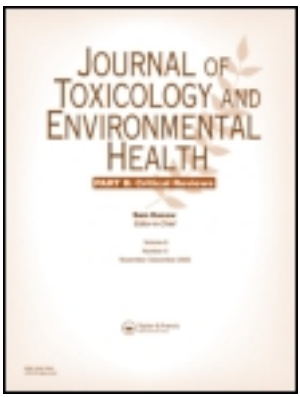

Journal of Toxicology and Environmental Health, Part B
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/uteb20

RECENT ADVANCES IN RESEARCH ON RADIOFREQUENCY FIELDS AND HEALTH

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Journal of Toxicology and Environmental Health, Part B, 4:145–159, 2001

1093-7404/01 $12.00 + .00

Update

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Since the Royal Society of Canada report on potential health risks of radiofrequency (RF) fields from wireless telecommunications in the spring of 1999, there have been several newly published reports on risks associated with the use of mobile phones. This article provides a summary of scientific research on the potential health effects of radiofrequency fields that has been reported since the original Royal Society report was published. This update also discusses several earlier results not included in the original report.

THERMAL EFFECTS

Adair et al. (1998) reported that human dorsal exposure to a supra-resonant frequency of 450 MHz at local peak specific absorption rates up to 7.68 W/kg is mildly thermogenic, and is counteracted efficiently by normal thermo-physiologic heat loss mechanisms. In a comparison of thermo-regulatory response of human dorsal exposure at frequencies of 450 and 2450 MHz, Adair et al. (1999) found no change in metabolic heat production at either frequency.

Using a thermal model developed at the National Radiation Protection Branch in the United Kingdom, Wainwright (2000) calculated temperature increases in the brain following radiation from cellular phones and similar electromagnetic devices. The maximum temperature rise found in the brain was about 0.1°C. Using another model to predict the absorbed electromagnetic power distribution and bioheat transfer, Van Leewuen (1999) estimated the maximum rise in brain temperature to be 0.11°C for an antenna with an average emitted power of 0.25 W. In another study, Khudnitskii et al. (1999) found that ultrahigh frequency radiation causes significant changes in local temperature and in physiologic parameters of the central nervous system, as well as in the cardiovascular system.

Jauchem et al. (1999) conducted an experiment involving sixteen ketamine-anaesthetized Sprague-Dawley rats individually exposed to 94 GHz radiofrequency electromagnetic radiation at a power density of 75 mW/cm^2. This exposure was found to produce extreme peripheral heating without similar core heating, sufficient to produce circulatory failure and subsequent death.

BIOLOGICAL EFFECTS

Ornithine Decarboxylase

Several studies have examined the relationship between electromagnetic field (EMF) exposure and ornithine decarboxylase (ODC) activity in both cells and tissues. These studies were performed in experimental animals and were associated with an alteration in increased tumor incidence. However, as discussed below, one laboratory has published two reports that suggest an inability to measure enhanced magnetic field ODC activity in two different systems.

As stated by Mullins et al. (1999), an alteration of ODC activity in animals or cultured cells exposed to extremely low frequency electromagnetic fields, or to modulated microwave fields, has been well documented by a number of laboratories. However, one significant limitation to these studies has been a lack of determination or evaluation of the dose-response relationship between ODC activity and the intensity of, in this case, the magnetic field employed. Mullins et al. (1999) measured

ODC activity in L929 fibroblasts exposed for 4 h to a 60 Hz magnetic field of different amplitudes. Their results revealed a clear threshold response which could be fitted to a sigmoidal function with the 50% point of increased ODC activity occurring at approximately 5 μT. These investigators further speculated that this sigmoidal response was characteristic of biological responses governed by ligand-receptor binding and discussed the implications of this study in terms of environmental exposures to EM fields.

In a series of experiments with the chemical carcinogen DMBA, Mevvisen, Haussler and Loscher (1999) observed that exposure of female rats to 50 Hz magnetic fields in the μT range facilitated the development and growth of mammary tumors. These investigators performed a series of experiments designed to measure the levels of ODC in different complexes of the rat mammary gland in order to evaluate whether differences in response to magnetic fields exist over the anterior-posterior extension of this organ following exposure to magnetic fields. It was observed that exposure of young female Sprague-Dawley rats induced marked increases in ODC activity in the mammary gland, similar to those observed following treatment of these animals with the well-known chemical tumor promoter, 12-*O*-tetradecanoylphorbol-13-acetate (TPA). These investigators measured a robust and reproducible enhancing effect on ODC activity by a 50 Hz 100 mT magnetic field following a two weeks exposure period. Furthermore, they observed that the enhanced ODC activity depended upon the location of the mammary complex examined with the cranial thoracic (or cervical) complexes being particularly sensitive to ODC alterations in response to magnetic fields. The authors concluded that this finding was in agreement with their recent DMBA experiments, in which magnetic field induced increases in tumor development and growth occurred at the same location in the mammary gland.

Kumlin et al. (1998) performed a series of experiments to determine whether magnetic fields and simulated solar radiation (SSR) could alter ODC, polyamines and tumor development in the mouse epidermis. Chronic exposure to combined magnetic field and SSR did not cause persistent effects on ODC activity or polyamines compared to the animals exposed to UV only, although the same MF treatment was previously found to accelerate skin tumor development. However, in an acute 24-h experiment, an elevation of putrescine and down regulation of ODC activity was observed in the animals exposed to a 100-μT magnetic field. The authors concluded that their results indicate that acute exposure to a 50 Hz magnetic field does exert distinctive biological effects on epidermal polyamine biosynthetic pathways.

At least one laboratory failed to observe increases in ODC activity in L929 cells (Cress et al., 1999) and in developing chick embryos (Desta et al., 1999) following exposure to a 60 Hz magnetic field. These investigators appear to have gone to a considerable lengths to replicate the effects

of 60 Hz magnetic field exposure upon ODC activity in the L929 cells. These authors concluded that in their laboratory, using the most important elements of the original investigator's exposure system, they did not demonstrate any enhancement of ODC activity. Given the rather low absolute level of ODC activity that exists in these cells, it is not unexpected that some laboratories may not be able to consistently measure a two-fold change in ODC activity.

There has also been increasing support in the literature for the idea that constitutive ODC overexpression induced by oncogenes can lead to an altered phenotype which may be relation to malignant transformation. In this regard, we have recently identified a series of genes which are regulated by putrescine, a product of ODC. At least several of these genes are known to have a direct relationship previously identified in the literature to the development of a cancer-like malignant phenotype (Pastorian et al., 2000).

As originally noted by the Panel, it remains important to determine whether the biological effects of EMF/RF exposure (for example, increased alterations in ODC activity and polyamine levels) can lead to adverse health effects. The toxicological and animal bioassays performed to date do not have sufficient statistical power to completely rule out the deleterious health effects of magnetic or RF exposure. Molecular technology capable of addressing this issue has only recently become available. For this reason, support for molecular bioeffects research in relation to magnetic and radiofrequency fields should be continued until these determinations are completed.

Ca^{2+} Efflux and Blood/Brain Barrier (BBB)

In a study on Wistar rats exposed to 112 MHz modulated to 16 Hz, at a power level of 1.0 mW/cm^2, for 35 days, Paulraj et al. (1999) found enhanced Ca^{2+} efflux and ornithine decarboxylase activity in the brain. Linz et al. (1999) evaluated a number of electro-physiological parameters (including membrane potential, action potential, L-type Ca^{2+} current, and potassium currents of isolated ventricular myocytes) in 90 guinea-pig myocytes and 20 rat myocytes which had been exposed to 180, 900, and 1800 MHz pulsed radiofrequency fields corresponding to the GSM (global system for mobile telecommunications) standard for cellular phones. The author concluded that none of the tested electro-physiological parameters was significantly altered by exposure to RF fields.

Using an in vitro model, Schirmacher et al. (2000) reported that exposure to 1.8 Ghz frequency electromagnetic led to a significant increase in the permeability of the blood/brain barrier (BBB) to [^{14}C]sucrose. Tsurita et al. (2000) found that a 1439 MHz TDMA (time division multiple access) field, as used in cellular phones, did not appear to have an effect on permeability of the blood/brain barrier (BBB), morphology of the cerebellum, or body mass in rats.

Melatonin

Several recent studies examined the relationship between exposure to electromagnetic fields and circulating melatonin. Wood et al. (1998) examined the effect of magnetic fields on human plasma melatonin levels, and reported that magnetic fields generated by square-wave currents produce more marked reductions in the maximum level of melatonin than did sinusoidal waveforms. Karasek et al. (1998) reported significant depression of nocturnal melatonin as a result of exposure to very low-frequency magnetic fields.

In a study conducted by de Seze et al. (1999) 37 young human males were exposed to cellular phones (900 MHz GSM and 1800 MHz DCS) 2 h/day, 5 days/wk for 4 wk. This investigation indicated that serum melatonin circadian profiles were not altered. However, Graham et al. (2000) found some evidence of a possible cumulative effect of magnetic field exposure on the stability of individual melatonin measurements over time.

Cell Proliferation

Kwee et al. (1998) showed that exposure of transformed human epithelial amnion cells to a modulated 960 Mhz radio-frequency field at different power levels and exposure times resulted in significant changes in cell proliferation. Velizarov et al. (1999) found that cell proliferation increased at the same order of magnitude in those cells exposed to RF as compared to sham-exposed at 39 or 35 ± 1°C, suggesting that cell proliferation is related to a factor other than RF-induced hyperthermia.

TOXICOLOGICAL EFFECTS

DNA Damage

Vijayalaxmi et al. (2000) exposed peripheral lymphocytes from from three healthy human volunteers in vitro to pulsed-wave 2450 MHz radiofrequency radiation for 2 h. This study revealed no evidence for induction of DNA single-strand breaks and alkali-labile lesions in lymphocytes either immediately following exposure or 4 h thereafter.

Goswami et al. (1999) exposed serum-deprived cells from C3H 10T 1/2 mouse embryo fibroblasts to an 835.62 MHz frequency-modulated continuous wave (FMCW) or to 847.74 MHz code division multiple-access (CDMA) microwaves at an average specific absorption rate (SAR) of 0.6 W/kg. No significant changes in the kinetics of proto-oncogene expression was observed. These exposures did not affect either the Jun and Myc mRNA levels, or the DNA-binding activity of AP1, AP2 and NF-kappaB in exponential cells during transit to plateau-phase growth. The authors concluded that exposure to radiofrequency fields is unlikely to result in a general stress response in this cell line under these exposure conditions. However, they did observe significant increases in Fos mRNA

levels in exponential cells in transit to the plateau phase and in plateau-phase cells exposed to 835.62 MHz FMCW microwaves.

The effects of microwave exposure on protein kinase C and gene expression in human mast cells (HMC-1) were studied by Harvey et al. (2000). They found that low-power microwave exposure may act on HMC-1 cells at temperatures well below those known to induce a heat shock response by altering gene expression through a mechanism involving activation of protein kinase C.

Romano-Spica et al. (2000) analyzed hematopoietic and testicular cell types for gene expression following exposure to 50 MHz radiofrequency waves modulated with a low frequency 16 Hz wave. They reported over-expression of ets1 mRNA in Jurkat T-lymphoblastoid and Leydig TM3 cell lines. This effect was observed only in the presence of 16 Hz modulation.

Carcinogenicity

Three different studies of long term exposure to frequency-modulated radiofrequency fields on central nervous system (CNS) tumors in Fischer rats have been reported recently (Adey et al., 1999, 2000; Higashikubo et al., 1999). One of these studies (Adey et al., 1999) demonstrated an inhibition in spontaneous and ENU-induced CNS tumors in animals exposed to 836.55 MHz fields. In this experiment, the investigators studied the effects of an 836.55 MHz field with North American digital cellular (NADC) modulation in a 2-yr bioassay that included fetal exposure. The authors examined both spontaneous tumorigenicity and the incidence of induced central nervous system (CNS) tumors in the offspring of pregnant Fischer 344 rats, following a single dose of ENU in utero, followed by intermittent digital-phone field exposure for 24 mo. Far-field exposures began on day 19 of gestation and continued until weaning at age 21 days. Near-field exposures began at 35 days and continued for the next 22 mo, for 4 2 h/day, 4 consecutive days weekly. Specific absorption rates (SAR) simulated localized peak brain exposures of a cellular telephone user. There was no evidence of tumorigenic effects in the CNS from exposure to the TDMA field. However, some evidence of tumor-inhibiting effects of TDMA exposure was apparent.

In the second study, Adey et al. (2000) exposed Fischer 344 rats to a frequency-modulated signal (836.55 MHz ± 12.5 KHz) simulating radiofrequency exposures in the head of users of hand-held mobile phones. They tested for effects on CNS tumors in the offspring of pregnant rats with and without in utero exposure to a single dose of ENU (4 mg/kg), selected to yield a brain tumor incidence of 10–15% over the mean life span of 26 mo. Pregnant dams were randomly assigned to one of six groups treated as follows: sham ENU/sham field, sham ENU/field exposed, ENU/sham field, ENU/field exposed, ENU/cage control, and sham ENU/cage control. Intermittent field exposures began on day 19 of gestation,

continued until weaning at 21 days, resumed at 31 days, and continued until termination of the experiment at 731–734 days. SARs in the rats' brains were similar to localized peak brain exposures of a typical cellular telephone phone user (female: 236 g, 1.0 W/kg; male: 450 g, 1.2 W/kg). Exposure to ENU resulted in a significant reduction in survival. There were no effects on survival attributable to frequency-modulated field exposure in either ENU-treated or in sham-treated animals. Spontaneous CNS tumor incidence ranged from 1.1–4.4% in unexposed controls, but increased to 14.4–22.2%; in animals exposed to ENU. No FM field-mediated changes were observed in the number, incidence, or histological type of either spontaneous or ENU-induced brain tumors, nor were gender differences detected in tumor occurrence.

In the third study, Higashikub et al. (1999) used the intracranial 9L tumor model to determine if exposure to a radiofrequency electromagnetic field similar to those used in cellular telephones CNS tumor growth. Fischer 344 rats implanted with different numbers of 9L gliosarcoma cells were exposed to a 835.62 MHz FMC to a 847.74 MHz CDMA RF field with nominal brain SARs of 0.75 ± 0.25 W/kg. The animals were exposed to the RF fields for 4 h/day, 5 days/wk starting 4 wk prior to and up to 150 days after the implantation of tumor cells. Among sham-exposed animals injected with 2 to 10 viable cells (group 1), the median survival was 70 days, with 27% of the animals surviving to 150 days. The median survival time and final survival fraction for animals injected with 11 to 36 viable cells (group 2) were 52 days and 14%, respectively. The corresponding values for those animals injected with 37 to 100 cells (group 3) were 45 days and 0%, respectively. The animals exposed to CDMA or FMCW exhibited similar survival patterns, with no statistical differences in the survival curves for groups 1, 2 and 3 as compared to sham-exposed controls.

In another recent study, Mandeville et al., (2000) investigated the possible effect of 60 Hz magnetic fields as promoters of neurogenic tumors initiated transplacentally by ENU. Female Fischer rats were divided into eight different groups (50 animals/group) and exposed in utero (on day 18 of gestation) to a single intravenous dose of either saline (group I), or 5 mg/kg of ENU (groups II to VIII). Dams in group II were given no further treatment, whereas dams in Groups III to VII were exposed to 5 different magnetic field intensities 48 h later. Animals in group III were sham exposed (<0.02 μT), while groups IV to VII were exposed to 2, 20, 200, and 2000 μT, respectively. Dams in group VIII were injected intraperitoneally with 10 μg/kg TPA from day 19 until delivery; their female offspring continued to be injected every 15 days, starting at day 14 after birth until sacrifice (positive controls). This study thus included internal controls (groups II and III) and a positive control group (group VIII). Body weight, mortality, and clinical signs were evaluated in all groups throughout in-life exposure. All exposed and control animals that died, were found

moribund, or sacrificed at termination of the study were necropsied. Histopathological evaluation was done for all brains, spinal cords, cranial nerves, and major organs (lungs, liver, spleen, kidneys, pituitary, thyroid and adrenals), and for all gross lesions observed during necropsy. Exposure to 60 Hz linear (single axis) sinusoidal, continuous wave magnetic fields had no effect on the survival of female rats or on the number of animals bearing neurogenic tumors. These results suggest that magnetic fields have have no promoting effect on neurogenic tumors in the female F344 rats exposed transplacentally to ENU.

Imaida et al. (1998) examined the possible promotional effects of pulse modulated 929.2 MHz electromagnetic near field on rat liver carcinogenesis initiated with diethylnitrosamine, using a 6 wk medium term bioassay. The maximum local SARs in this experiment were up to 7.2 W/kg on a whole body basis and 2.0 W/kg in the liver. Exposure to this modulated electromagnetic field had no apparent effect on rat liver carcinogenesis.

Testicular Function and Teratogenicity

There is some evidence that exposure to thermal levels of RF fields can cause heat damage to testes in mice (Saunders & Kowalczuk, 1981). However, other studies involving whole body heating of Sprague-Dawley rats have found no significant disruption of testicular function (Lebovitz & Johnson, 1983, 1987).

Most of the work on the teratogenic effects of RF fields have focused on thermal effects (Nelson et al., 1991; Lary et al., 1982, 1983a, 1986). A series of studies published by Lary and colleagues examined the effect of thermal RF irradiation of pregnant Sprague-Dawley rats on various fetal outcomes (Lary et al., 1982, 1983b, 1986). They found that RF-induced hyperthermia in the postimplantantion period caused a significant rise in fetal malformations, and a reduction in fetal weight and crown-rump length; hyperthermia during days 7 or 9 caused an increase in dead or resorbed fetuses; exposure pre-implantation was associated with a significant increase in pre-implantation malformations (Lary et al., 1982). Further research by Lary and colleagues found that the teratogenic and embryotoxic effects of thermal RF exposures are related to the temperature induced in the pregnant rat as well as the length of time her temperature is elevated (Lary et al., 1983). In a subsequent article, they reported a threshold temperature of 41.5°C for both birth defects and prenatal death (Lary et al., 1986).

Radiofrequency field induced hyperthermia has been found to have a synergistic effect with chemical teratogens. Nelson et al. (1991) found that combined exposure to thermal RF-levels and the industrial solvent 2-methoxyethanol (2ME) led to a significant increase in the both the frequency and the severity of fetal malformations in rats. While 2ME alone produced a malformation rate of 14% and RF hyperthermia alone pro-

duced a rate of 30%, combined exposure results in fetal malformation rate of 76% (Nelson et al., 1991). Nelson et al. (1997) found a threshold temperature of 41°C for these synergistic effects, similar to that reported by Lary et al. (1986).

There is some evidence that RF exposure may lead to teratogenic effects beyond those resulting from increased body temperatures. Nawrot et al. (1981) exposed groups of mice to thermal levels of RF, direct heating to the same temperature as the RF-exposed mice, and to non-thermal RF fields. They found significant decreases in implantation sites per litter and in fetal weight in mice exposed to 30 mW/cm^2 during days 1–6 and a significant increase in fetal malformations to RF exposed litters in days 6 to 15 over those mice exposed to ambient temperatures. However other studies which used non-thermal RF-exposures, rather than controlling for the thermal effects, found no teratogenic effects in rats or mice (Lary et al., 1983; Marcickiewicz et al., 1986). A study in which mice were placed at different distances from an RF antenna found a progressive decrease in the number of offspring with closer proximity, with irreversibly infertility at the most proximate location (Magras & Xenos, 1997). However, prenatal development of the newborns improved with proximity in this study (Magras & Xenos, 1997). Marcickiewicz et al. (1986) noted that exposing pregnant mice to non-thermal RF fields enhanced the effect of the chemical teratogen cytosine arabinoside, producing more fetal resorptions and malformations than with arbinoside alone.

EPIDEMIOLOGICAL STUDIES

Several case reports of potential adverse health effects associated with radiofrequency field exposure have been communicated recently (Hocking, 1998; Schilling, 2000), including contact dermatitis associated with nickel in mobile phones (Pazzaglia et al., 2000). While such case reports are useful indicators of potential problems, analytical epidemiologic studies are required in order to address hypotheses formulated on the basis of such reports.

Morgan et al. (2000) reported on cause-specific mortality, in particular deaths from brain cancers, lymphomas, and leukemias in a cohort of almost 196,000 workers at Motorola, a manufacturer of wireless communication products. Workers were classified into high, moderate, low, and background RF exposure groups using job titles. A total of 2.7 million person-years of observation were accumulated over the period from 1976 to 1996. Using external comparisons, the standardized mortality ratios for RF-exposed workers were 0.53 (95% confidence interval or CI: 0.21–1.09) for deaths from central nervous system/brain cancers, and 0.54 (95% CI: 0.33–0.83) for deaths due to all lymphomas/leukemias. This may be explained by the fact that workers are generally healthier than the overall population. Using Poisson regression models based on internal com-

parisons, the mortality rate ratios were near 1.0 for brain cancer, and below 1.0 for deaths from all lymphomas/leukemias. The findings were essentially the same using cumulative, peak, and usual exposure classifications. The study was limited by the use of indirect exposure assessment, use of deaths rather than incidence for outcome, and the relatively young age of the cohort.

Hardell et al. (1999) reported the results of a case-control study of incident brain tumors and cellular phone use in Sweden. Eligible cases were diagnosed in a specified time period (either 1994–1996 or 1995–1996), resident in one of two regions within Sweden, and currently alive. Each case was matched to two population-based controls. Information on exposures was collected via postal questionnaires, and in some cases supplemented by telephone interviews. A total of 209 cases and 425 controls were included in the analysis. The study found no association between the occurrence of brain tumors and the overall cellular telephone phone use (odds ratio or OR = 0.98, 95% CI: 0.69–1.41); however, a elevated although non-significant risk was reported for tumors in the temporal or occipital lobe of the brain on the same side as the cellular phone was used (OR [right side] = 2.45, 95% CI: 0.78–7.76; OR [left side] = 2.40, 95% CI: 0.52–10.9). The authors pointed out that these results were based on small numbers (13 cases) with a short observation time, and therefore need to be interpreted with caution. A weakness of this study is that self-reported information on cellular phone use might be biased for cases. Ahlbom and Feychting (1999) also pointed out that a substantial number of eligible patients may not have been identified by the investigators for inclusion in the study, thereby raising the possibility of biased results due to non-representativeness of study subjects.

An update of this study was subsequently published by Hardell et al. (2000). The authors reanalyzed the data by classifying different areas of the brain into different exposure categories, and arrived at an odds ratio of 2.42 (95% CI: 0.97–6.05), again elevated but not significantly different from unity.

Dreyer (1999) has updated the study of the cohort of cellular telephone users published earlier by Rothman et al. (1996). Even with extended follow-up, no significant differences in disease-specific mortality was found between users of non-handheld (characterized as "non-exposed") and handheld phones, including mortality from all cancers, brain cancer, leukemia, circulatory disease, and all cause mortality. A slight increase in the risk of death from motor vehicle collisions was seen with increasing minutes of use.

NEUROLOGICAL AND BEHAVIORAL EFFECTS

Some of the energy associated with the RF field emitted during the use of hand held cellular phones is absorbed by the head and brain of

cellular telephone users users (Schonborn et al., 1998). Several recent studies have been conducted to study possible effects of RF on brain function. Freude et al. (1998) and Eulitz et al. (1998) reported that exposure to 900 MHz fields can affect EEG. Krause et al. (2000), conducted an experiment on sixteen adults to study the effects of electromagnetic fields emitted by GSM cellular phones on the EEG during a memory task. They concluded that exposure to EMF influences neural oscillatory systems during cognitive tasks. The effects of 902 MHz electromagnetic fields emitted by cellular phones on response times in humans was examined by Koivisto et al. (2000a). The results of this study suggested that RF might affect performance on tasks that primarily depend on prefrontal cortical function (sustaining, concentration, and preparatory attention), but not on tasks that depend on occipitotemporal functions (visual perception and semantics). Koivisto et al. (2000b) further studied the effects of EMF emitted by cellular (GSM) phones on working memory, and found found a measurable effect on human cognitive performance. Finally, Wang and Lai (2000) reported that acute exposure of rats to pulsed 2450 MHz resulted in spatial memory deficit.

OCULAR EFFECTS

In our original report, we noted a lack of scientific data on the potential health effects of radiofrequency fields on the eye, an exposed organ with a limited ability to dissipate heat. Lu et al. (2000) studied the retinal effects of 1.25 GHz high peak power microwaves in Rhesus monkeys, using fundus photographs, retinal angiograms and electroretinograms to evaluate ocular structure and function. The authors concluded that retinal injury is unlikely at 4 W/kg, and that functional changes at higher retinal SAR values are likely reversible. Griffith et al. (1999) recently developed an in vitro model of the human cornea that may prove useful in future studies of the potential effects of RF field exposure on the eye.

CONCLUSIONS

In this article, we have summarized a number of recent research results on the potential health risks of radiofrequency fields published largely since the time of our original review. Whereas these new findings represent valuable additions to the increasing scientific database on the potential health implications of exposure to radiofrequency fields, additional research is needed to clarify this issue.

Of particular interest are the results of the new epidemiologic studies, since these studies provide direct information on potential health risks in human populations exposed to radiofrequency fields. However, these additional study results are not sufficient to alter our original conclusion that the epidemiologic evidence on potential health risks associated with

RF field exposure is inadequate for a comprehensive evaluation of risk (cf. Ellwood, 1999), and that further studies addressing some of the limitations of studies to date, including limitations in exposure assessment, need to be carried out.

Following the release of the Royal Society of Canada Expert Panel report in 1999, a similar evaluation of mobile phones and health was prepared by an expert group in the United Kingdom, chaired by Sir William Stewart (Independent Expert Group on Mobile Phones, 2000). This detailed review concurred with our initial conclusion that the balance of evidence available at this time does not suggest that human exposure to radiofrequency fields is associated with adverse health effects, and noted that the biological effects identified in some studies does not necessarily mean that health is affected. Like the Royal Society Report, the Stewart Report recommended that additional research on the potential health effects of radiofrequency fields, including epidemiologic studies of cellular telephone users, is needed. Finally, the Stewart report also recommended a precautionary approach until more definitive scientific information becomes available. This approach is discussed further by Foster et al. (2000).

The health risks of mobile phones are discussed in two review papers appearing in a recent issue of Lancet (Rothman, 2000; Hyland, 2000), along with a commentary by Dendy (2000). Rothman concludes that epidemiologic studies of neither occupational exposures to RF fields nor of cellular telephone users have demonstrated an association with brain cancer or other malignancies. Hyland comments on the subtle biological effects associated with RF field exposure, and the need for further research to clarify the potential health implications of such effects. Dendy argues that public perception of potential health risks of cellular telephones will be influenced by the perceived benefit, an issue outside the scope of the present scientific update.

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