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Brain tumour development in rats exposed to electromagnetic fields used in wireless cellular communication

Leif G. Salford *, Arne Brun and Bertil R.R. Persson

Lund University, S-221 85 Lund, Sweden

It has been suggested that electromagnetic fields (EMF) act as promoters late in the carcinogenesis process. To date, however, there is no convincing laboratory evidence that EMFs cause tumour promotion at non-thermal exposure levels. Therefore the effects of exposure to electromagnetic fields were investigated in a rat brain glioma model. Some of the exposures correspond to electromagnetic fields used in wireless communication. Microwaves at 915 MHz were used both as continuous waves (1 W), and pulse-modulated at 4, 8, 16 and 217 Hz in 0.57 ms pulses and 50 Hz in 6.67 ms pulses (2 W per pulse). Fischer 344 rats of both sexes were used in the experiments. By stereotaxic technique rat glioma cells (RG2 and N32) were injected into the head of the right caudate nucleus in 154 pairs of rats, exposed and matched controls. Starting on day 5 after inoculation, the animals were exposed for 7 hours a day, 5 days a week during 2–3 weeks. Exposed animals were kept unanaesthetized in well-ventilated TEM cells producing 915 MHz continuous or modulated microwaves. Their matched controls were kept in identical TEM cells without EMF exposure. All brains were examined histopathologically and the tumour size was estimated as the volume of an ellipsoid. Our study of 154 matched pairs of rats does not show any significant difference in tumour size between animals exposed to 915 MHz, and those not exposed. Thus our results do not support that even an extensive daily exposure to EMF promotes tumour growth when given from the fifth day after the start of tumour growth in the rat brain until the sacrifice of the animal after about 16 days.

1. Introduction

Radiofrequency fields, and especially microwaves (300 MHz–300 GHz) constitute a very important part of the electromagnetic spectrum in human exposure and risk assessment. In the past decade there has been much concern especially about the safety of microwave ovens (2450 MHz) and, before that, radar equipment (GHz). At present, wireless communications (for example, mobile telephones) have been particularly considered. Little, however, is still known about the health hazards of radiation from cellular telephones. While cellular communications technology has advanced rapidly, there have been few scientific studies on whether the electromagnetic radiation emitted by cellular phones poses a risk to human health. There exist only a few epidemiological studies of the cancer incidence in RF-exposed subjects comparable to the ones discussed for ELF electromagnetic fields [10,13]. In a study conducted by Robinette et al. [20] on naval personnel and radar operators it was shown that workers in this occupation had a higher than normal risk for brain cancer. Mortality from cancer was reported to be increased close to air force bases compared with other places. It was concluded that this was due to the proximity to radar installations [15]. This study was criticised [19] on the grounds of faulty data collection, absence of physical measurements and failure to investigate potential confounders. A possible association between chronic exposure to microwaves and polycythemia was mentioned in a letter to the New England Journal of Medicine [11], but this was not based on an extensive investigation. It was also reported that amateur radio operators have a higher incidence of myeloid leukaemia [18], but this observation was subject to the same criticisms as above. A short communication also reported a cluster of 6 cases of testicular cancer in a cohort of 340 American police officers over a 12-year period [9]. This is about 7 times the expected number and according to the authors, the real risk may be even higher. All six were regularly exposed to hand-held speedometers operating through radar waves. Other known risk factors for testicular cancer did not apply to these men.

According to a series of papers, low level, low frequency-modulated microwave radiation may affect intracellular activities of enzymes involved in neoplastic promotion without measurable influence on the overall DNA synthesis. Byus et al. [6] reported, for example, evidence of an effect of microwaves on intracellular levels of ornithine decarboxylase (ODC) which is an enzyme implicated in cell growth and which is increased by tumour promoters. These effects were, however, noticed only for certain modulations of the carrier wave. Balcer-Kubiczek and Harrison [4] exposed cells to 120 Hz-modulated microwaves (SAR from 0.1 to 4.4 W/kg) followed by treatment with a phorbol ester tumour promoter. They noticed higher frequency of transformed cells with increasing SAR value. Lymphocyte transformation was another observation after exposure of the cells to pulsed or continuous radiofrequency radiation [8,24]. Pulsed RF fields were found to be more efficient than continuous waves in eliciting this type of effect. These effects may be thermal in nature. Still another interesting observation was that of Adey [2] who reported that

* Corresponding author: Department of Neurosurgery, Lund University Hospital, S-221 85 Lund, Sweden.
E-mail: Leif.Salford@neurokir.lu.se

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120 Hz-modulated 450 MHz microwaves inhibit gap junction cell-to-cell communication in cultured hamster ovary cells. Adey points out that “there is evidence that intercellular communication plays an essential role in regulation of cell growth. Functional isolation of a cell from its neighbours by the separate or joint actions of EM fields and chemical cancer promoters, both acting at cell membranes, may lead to unregulated growth with tumour formation” [3]. Several other in vitro model systems have been developed that examine the effects of EM field exposures combined with a chemical tumour promoter but with no conclusive results [7,16].

A number of in vivo investigations has also been performed. Of particular interest is the study conducted by Szmigielski et al. [25] who observed faster development of benzpyrene induced skin tumours in mice exposed for some months to subthermal 2450 MHz microwaves. Of great interest is also the study of Kunz et al. [14] who exposed 100 rats (aged 2 to 27 months) to pulsed microwaves (0.4 W/kg). The exposed group had a significantly increased frequency of primary malignant lesions as compared to the control group when lesions were pooled regardless of their location in the body.

We have found no reports of previous work done in vivo in the laboratory to study brain tumour development during exposure to radiofrequency radiation. For several years we have been using a rat brain glioma model for therapeutic studies in our laboratory. In a collaboration between the Division of Experimental Neurooncology, department of Neurosurgery, the department of Neuropathology and the department of Medical Radiation Physics, we have examined the effect of 915 MHz microwaves, both as continuous waves, and modulated with 4, 8, 16 Hz and 217 Hz in 0.57 ms pulse lengths and 50 Hz in 6 ms pulse lengths. This corresponds among others, to the type of electromagnetic fields used in cellular communications. Preliminary results of smaller number of animals examined have been presented in previous reports [22]. The present paper presents the results of the completed study including in total 308 rats with inoculated tumours.

2. Material and methods

2.1. Animal model

The rat glioma cell-line RG2, originally from an ethylnitrosourea-induced rat tumour, grows very well in infinite cell culture cycles since more than two decades [27], and produces malignant astrocytoma-like tumours when inoculated in the brain of Fischer 344 rats. When at least 1000 RG2 cells are inoculated, well-delineated tumours develop in 100% of our animals within 3 weeks and the untreated tumours have by then reached a diameter of about 3–6 mm [1].

Fischer 344 rats of both sexes, weighing 150–250 g, were used in all the experiments. The animals had free access to water and pellets (SAN-bolagen, Malmö, Sweden). By stereotaxic technique 5000 RG2 cells in 5 μl nutrient solution were injected with a Hamilton syringe into the head of the right caudate nucleus in a total of 218 rats. To avoid extra-cranial tumour growth, the injection site was cleaned with 70% ethanol after injection and the bore hole sealed with wax. In collaboration with the Department of Tumour Immunology, Lund University, we have recently developed a new ethyl-nitroso-urea induced rat glioma cell line that produces gliomas of malignant astrocytoma type with a slower growth rate. For the N32 cell line the time from inoculation to neurological symptoms is 4–5 weeks compared to 3 weeks for of the RG2 cell line [23]. The N32 cell line was used in 90 rats that received 15,000 cells in the head of the right caudate nucleus. Groups of 2–8 animals were inoculated at each instance with cells harvested immediately before the procedure.

At inoculation, every animal to be exposed was matched to a control animal that was inoculated with identical tumour cells immediately before the animal to be exposed. All animals are totally inbred and for each animal to be exposed a control animal was randomly chosen from the same cage. The exposure was started on day 5 after inoculation in both RG2 and N32 cell line series. In all, 154 of the 308 animals in the series served as controls and the other 154 animals were exposed to electromagnetic fields. When the exposed animal or its matched control started to develop neurological signs of tumour growth, both animals were sacrificed by perfusion–fixation of the brains under chloral-hydrate anaesthesia. This means that RG2 rats were sacrificed about 3 weeks after inoculation and N32 rats about 4–5 weeks after inoculation.

2.2. Electromagnetic fields

The GSM (Global System for Mobile communication) phone in the 900 MHz band has a peak output of 2 W. With a duty factor of $1/8$, this leads to a time average of 0.25 W output leaving the antenna. The maximum power absorbed is thus of the order of one tenth of a watt.

In the present study we expose the whole animal in a TEM-cell with the unique characteristic of having both linear amplitude and phase response versus frequency. Thus it lends itself to extremely broad band sweep frequency testing using a variety of wave forms including CW and pulsed (or modulated) exposure fields. These fields can be accurately generated in the TEM-cell without the distortion that is typically introduced when conventional antennas are used to establish impulse test fields.

The cell is enclosed in a wooden box that supports the outer conductor and central plate. The outer conductor is made of brass-net and is attached to the inner walls of the box. The central plate, or septum, is constructed of aluminium and is held up by teflon braces which are attached at the inner side walls. To allow access to the inside of the cell, both ends can be removed. The inside of the cell is ventilated through 18 holes (diam. 18 mm) in the side walls and top of the box and the brass-net allows air to circulate.

Table 1
Exposure scheme.

Modulation frequency (Hz)	Number of days of exposure	Pulse length (ms)	Peak power in pulse (W)	Duty cycle	SAR (W/kg)
0 (CW)	10–15	–	1	1	1.67
4	10	0.57	2	0.002	0.0077
8.33	10	0.57	2	0.005	0.016
16	13	0.57	2	0.009	0.030
50	9–13	6	2	0.3	1.00
217	9	0.57	2	0.12	0.4

These holes are also used for examination of the interior during exposure. Probes for monitoring temperature inside the cell or of test objects are inserted through these holes.

The test system consists of four TEM-cells. A microwave power generator (MCL model 15 222) is used for feeding the TEM-cells. A power splitter divides the power from the RF generator into four equal parts that are fed to each of the four cells. The output from the cells is terminated in a 50 ohms dummy load. Both forward and reflected powers are measured, with a Bird model 43 power meter, at the inputs and outputs of the cells. The output from the RF generator can be pulse modulated from an external source by applying a positive pulse of 5 volts with a pulse width of 5.0 microseconds minimum. The rise and fall time of the RF pulses used in the experiments has been 0.04 ms and 0.81 ms, respectively.

Calculations of SAR-distributions in the brain of rats exposed to three-dimensional electromagnetic fields have been performed for our TEM-cell using the Finite Difference Time Domain (FDTD) method [17,26]. The SAR values were also determined experimentally by measuring the input power and the transmitted power by power-meters on each side of the TEM cell with and without rat load.

Measurements of reflected and transmitted power were performed with and without rats in the TEM-cell and at an input power of 1 W continuous wave. The result of these measurements gave an average SAR of 1.4 ± 0.3 W/kg per 1 W input power. This is in good agreement with the theoretical, calculated value of 1.67 W/kg per 1 W input power.

The exposed animals were kept unanaesthetized in TEM cells producing 915 MHz continuous or modulated microwaves (see table 1). Exposure was started on day 5 after inoculation. The animals were exposed 7 hours a day for 5 days a week. All animals were given a half hour break for feeding after 4 hours of exposure.

The controls were kept in identical TEM cells without EMF exposure. The TEM cells were well ventilated. The rat rectal temperature was recorded with an optical temperature device (LUXTRON 2000) before exposure, and after 4 hours and 8 hours exposure, respectively.

2.3. Histopathological examination

All brains were blindly examined histopathologically by two independent examiners. One of the examiners is the chief neuropathologist Arne Brun, one of the authors. Five coronal slices from each animal were paraffin embedded, sectioned at 5 μm and studied microscopically in cresyl violet staining. In this way the entire telencephalon was covered except the frontal and occipital poles.

For tumour size measurements the slice with the largest tumour extension was measured and chosen as major axis (A) of an ellipsoid and its perpendicular axis was chosen as the minor axis (B). The number of slices at 1 mm apart was chosen as the diameter (D) of the ellipsoid. The tumour volume was estimated by the ellipsoidal volume calculated from the following equation:

$$V = \frac{\pi}{6}(A \times B \times D).$$

2.4. Statistical evaluation

Student's t-test for paired samples as well as Wilcoxon's matched pairs' test were used for the statistical evaluation.

3. Results

The exposure parameters for the various groups of experiments are shown in table 1. The number of exposure periods was from 9 to 15 which in average resulted in around 80 hours of exposure in the TEM-cells. The animals showed no signs of stress from the exposure with electromagnetic fields. They returned spontaneously into the TEM cells after break for feeding. The rectal temperature of the animals recorded before exposure, at the break after 4 hours and directly after the end of the daily exposure, did not show any significant variation.

All 154 exposed animals as well as their matched controls developed polycyclic tumours, rounded with well-defined boundaries. On the histopathological examination the tumours were usually found to be solid with minor necrotic areas without correlation to treatment, tumour size or time from inoculation to death. In most animals, a sharp clean demarcation was seen between the tumour and the surrounding brain. In some animals, the tumours had a slightly blurred border and minor satellite tumour foci or nests of migrating cells were revealed in the surrounding brain. There were no signs of brain damage outside the tumour areas, neither necrosis, gliosis nor inflammatory changes that could be ascribed to the EMF exposure.

Table 2 shows the results of the tumour volume measurements for groups of rats exposed to different modulation frequencies. The frequency distribution of difference in tumour volume between exposed animals and matched controls are shown in figure 1 for the whole population of 154 pairs of rats. The results for the different types of tumours and various modulation frequencies are shown in

Table 2
Brain tumour *volume* of *all tumours* ($RG2 + N32$) in EMF exposed rats and controls.

Modul. frequency (Hz)	Number of pairs	Tumour volume exposed (mm^3)	Tumour volume controls (mm^3)	Wilcoxon's matched pairs' test p	Student's matched pairs' t-test p
CW	15	29 ± 22	18 ± 13	0.17	0.10
4	12	21 ± 15	15 ± 12	0.53	0.43
8. 33	32	22 ± 17	28 ± 21	0.18	0.17
16	24	21 ± 13	20 ± 17	0.90	0.92
50	31	21 ± 20	19 ± 19	0.97	0.61
217	40	18 ± 20	18 ± 15	0.37	0.88
All	154	21 ± 19	20 ± 18	0.97	0.73
All PW	139	20 ± 18	20 ± 18	0.56	0.79

Table 3
Brain tumour *volume* of *RG2 tumours* EMF exposed rats and controls.

Modul. frequency (Hz)	Number of pairs	Tumour volume exposed (mm^3)	Tumour volume controls (mm^3)	Wilcoxon's matched pairs' test p	Student's matched pairs' t-test p
CW	15	29 ± 22	18 ± 13	0.17	0.10
4	12	21 ± 15	15 ± 12	0.53	0.43
8.33	28	19 ± 13	24 ± 21	0.28	0.21
16	24	21 ± 13	20 ± 17	0.90	0.92
50	19	26 ± 21	20 ± 17	0.52	0.33
217	11	11 ± 8	15 ± 12	0.25	0.19
All	109	21 ± 17	20 ± 17	0.56	0.56
All PW	94	20 ± 15	20 ± 17	0.99	0.96

Table 4
Brain tumour *volume* of *N32 tumours* in EMF exposed rats and controls.

Modul. frequency (Hz)	Number of pairs	Tumour volume exposed (mm^3)	Tumour volume controls (mm^3)	Wilcoxon's matched pairs' test p	Student's matched pairs' t-test p
CW	–	–	–	–	–
4	–	–	–	–	–
8.33	4	43 ± 24	51 ± 9	0.71	0.66
16	–	–	–	–	–
50	12	15 ± 18	19 ± 23	0.39	0.34
217	29	20 ± 23	19 ± 16	0.94	0.83
All	45	21 ± 23	22 ± 20	0.31	0.68
All PW	45	21 ± 22	22 ± 20	0.31	0.68

tables 2–4 with p values based on both Wilcoxon's matched test and Student's paired t-test. There is no difference between the average tumour volume (21 mm^3) of exposed RG2 and N32 rats. In the controls, there is no significant difference between the average volume of RG2 tumours (20 mm^3) and N32 tumours (22 mm^3).

Our study does not show any significant difference in the tumour growth between animals exposed and those not exposed. The standard deviation is large, but this is the result of the large individual variation in the model where the status of the inoculated cells as well as that of the recipient animal is influential.

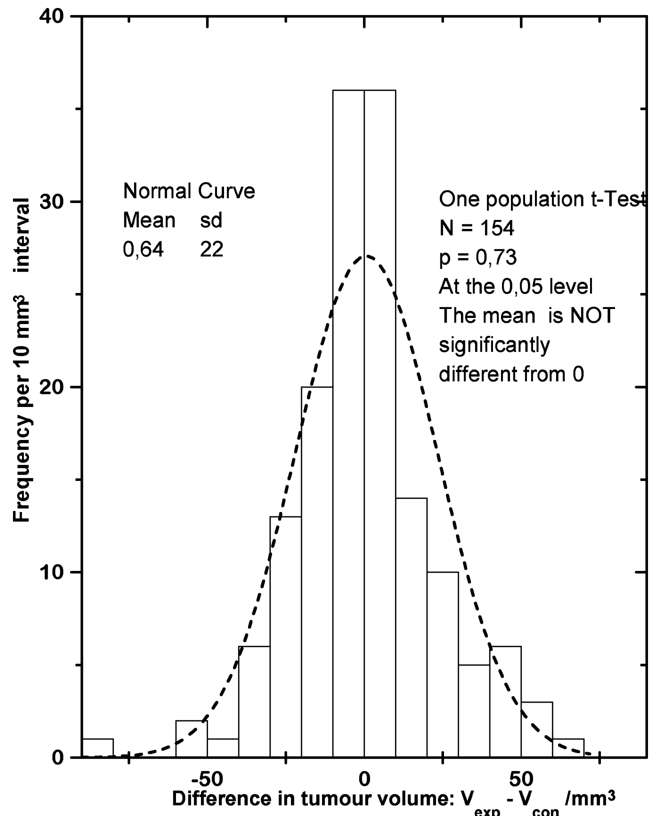

Figure 1. Frequency distribution of the matched difference between the tumour volume of exposed and control rats. The mean value 0.64 is not significantly different from zero.

From the results in tables 2–4 no obvious differences can be seen between the animals exposed to CW and the animals exposed to pulse modulated fields.

4. Discussion

During the last two decades some epidemiological studies have given weak evidence for a correlation between exposure to EMF and increased incidence of leukaemia and tumours of the central nervous system (CNS) among people living close to high power electrical lines or working in electrical occupations [13]. If such a correlation exists, it can be caused either by *initiation* of cancer development or the *promotion* of a cancer initiated by other reasons.

Our study has been conducted to evaluate the possible promoter effect of EMF in a brain tumour model. This study has no bearing on the initiation of cancer development as the tumour cells are inoculated into the animals. (In another ongoing study, we examine the possible tumour-initiating effects of long-term, 7 months, daily exposure to EMF in animals which spontaneously develop tumours in several organs including the CNS.)

In our neurooncological research, we have since long used the RG2 cell line, which causes rat gliomas. These are very similar to the human malignant astrocytoma, which grows very fast. Patients with this grim diagnosis survive

only few months on an average if no treatment is given and with all available therapy 50% are dead after one year.

The human malignant astrocytoma sends migrating tumour cells far out in the normal brain, where they hide behind an intact blood brain barrier (BBB). Also in our RG2 model, small clusters of migrating cells, separated from the main tumour, are found in the normal surrounding rat brain. The RG2 model mimics the human malignant astrocytoma also by having at least partially incompetent BBB [12]. It also produces an oedema surrounding the tumour.

We found that in the RG2 model there is no indication whatsoever of promoted tumour growth for any of the tested EMF-frequencies. However, it should be remembered that a reason for the absence of significant differences between exposed and control animals could be that the rat glioma tumour cell-line (RG2) is very aggressive. The cells may be growing at their maximal speed producing a tumour in three weeks, and the addition of external stimuli may therefore not influence upon the tumour growth rate.

This is why we were looking for a more slowly growing glioma cell line. In collaboration with the Department of Tumour immunology, Lund University, we developed the ethylnitrosourea induced rat glioma cell line N32, which also produces gliomas similar to the human malignant astrocytoma but with slower growth than the RG2 cells [23]. Thus N32 should provide a less aggressive model appropriate for continued experiments. This cell line was inoculated in 45 pairs of rats. Exposure to EMF (frequencies 8, 50 and 217 Hz studied) resulted, however, in no significant change in N32 tumour volume of the exposed animals compared to their controls.

The mean values for tumour sizes show large standard deviations. This is an expression for the large spread of tumour sizes from animal to animal. We ascribe this to small variations in the exact number of cells that are sucked into the Hamilton syringe and then reach the target point in the caudate nucleus during the stereotaxic inoculation. The condition of the cells may differ from day to day and depend upon when in their division cycle they are harvested. Even if the Fischer 344 rats used by us are inbred since several years, minor differences in their response to inoculated tumour cells may exist. This underlines the importance of matching every treated animal with its control before simultaneous inoculation of cells in identical amounts.

The control of body temperature during EMF exposures is important for the exclusion of thermal effects. We found no thermal effect of the SAR values up to 1.7 W/kg. In separate experiments we have not seen thermal effect until the power given is five times higher than that used in the present study (unpublished results).

Our results hitherto with two different tumour cell lines implanted in rat brains and exposed during around 80 hours to EMFs similar to those used in wireless communication do not indicate increased tumour growth. Even if the more slow-growing of the two tumour-models shows no promoting effect, it should be noted that both models give aggressive tumours similar to the human most aggressive malignant astrocytoma. Other forms of human brain tumours grow slower. The anaplastic astrocytoma and the even less malignant astrocytoma [5] may grow for several years up to decades in the human brain before they give symptoms leading to diagnosis.

A possibility to mimic the less aggressive growth can be the utilisation of a xenotransplantation model, based upon the implantation of a cell suspension of human glioma cells (collected during neurosurgery) into the brain of immune-competent Wistar rats. This model gives tumour takes in 50% of the animals and transplants maximally grow to diameters of 3 mm during a 3 months' period [21]. The reason for this much slower growth is the immune response of the animal that prevents the species-different tumour tissue from growing uncontrolled.

In conclusion we found no difference whatsoever between the growth of inoculated rat brain tumours in 154 EMF exposed animals and their 154 unexposed matched controls. However, more models must be used in the search for possible tumour promoting effects of EMF before they can be ruled out. Other experimental models have to be employed for evaluation of possible tumour initiating effects of electromagnetic fields.

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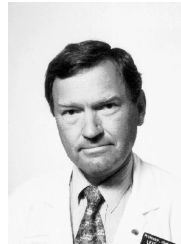
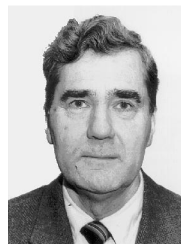


Leif G. Salford was born in Malmö, Sweden, in 1941. He received the MD degree from the University of Lund in 1969, and the Ph.D. degree from the same university in 1974 with the thesis "Influence of profound hypoxia on regional metabolism, blood flow and cell morphology in rat brain". During 1972–1973 he was Wrightsman scholar at the Dept. of Neurology, Cornell Medical Center, New York Hospital, NY. In 1977 he became associate professor of neurosurgery, Lund University, and in 1979 Consultant Neurosurgeon. He was Professor and Chairman, Department of Neurosurgery, Kuwait University, 1981–1983, and Professor of Neurosurgery and Director of the Departments of Clinical Neurosciences, Sahlgrenska University Hospital and Göteborg University, Sweden, 1993–1996. His actual positions are: Professor and Chairman, Dept. of Neurosurgery, Lund University Hospital, and Director of the Institute for Clinical Neuroscience, Lund University, Lund, Sweden. Dr. Salford is Chairman of the Neuro-oncology Committee of the World Federation of Neurosurgical Societies, and President of the European Association for Neuro-oncology. He is President of the Swedish Neurosurgical Society, Honorary President of the Scandinavian Neurooncology Society and Expert in the EU Information Society Forum.

Dr. Salfords' research is concentrated on the malignant primary brain tumours and on the search for efficient treatment against this hitherto incurable type of cancer. This has also resulted in his studies of opening the blood-brain-barrier by the use of electromagnetic fields in order to reach the tumour cells with cytotoxins, and in his interest in the biophysical effects of EMF upon brain and tumour biology. Dr. Salford is Director of the Laboratory for Experimental Neurooncology, Lund University, where animal models and tissue cultures play an important role in his research in close cooperation with technological institutions such as Medical Radiation Physics. He strives to utilize the latest progress in technology in his search for solutions to medical problems. Another result of this collaboration is the introduction of electropermeabilisation *in vivo* as a tool for treatment of intraparenchymatous tumours which has proven efficient in connection with cytotoxins in the rodent brain tumour model.



Arne Brun, MD, Ph.D., is a professor of neuropathology, University Hospital, Lund, Sweden. His publications are mainly on central nervous system diseases such as dementias, especially the Alzheimers' disease and frontal dementias, also cerebrovascular incl. BBB-studies.

Bertil R.R. Persson, Ph.D., professor of medical radiation physics, was born 1938 in Malmö, Sweden, studied at University of Lund, Sweden, and in 1970 became doctor of philosophy and associate professor in radiation physics. From July 1980 till present he is full professor in medical radiation physics at Lund University hospital where he is head of the department of radiation physics. During his scientific activity he has published more than 320 scientific publications and written 16 extensive reports and books. His scientific career began in 1963 with studies of the fall-out from atmospheric nuclear weapons tests in the food-chain lichen-reindeer-man and continued in medical use of short-lived radioisotopes. The scientific activity in environmental radiology has developed into polar research. In 1980 he participated in the Swedish Arctic Expedition Ymer-80, in January–April 1989 he participated in the Swedish Antarctic research program with a marine ecology program investigating the longitudinal profile of $^{134}Cs/^{137}Cs$ ratio from Gothenburg in Swe-

den all the way to Antarctica. In June–August 1994 he participated in the Swedish–Russian Tundra expedition with marine and terrestrial radioecology research along the cost of Siberia and in 1995 he studied the environmental radioactivity in and around the Russian uranium mines in Krasnokamensk. He just returned from the Swedish expedition Arctic Ocean-96 that visited the North Pool 1996-09-10 the latest visit ever. During the expedition he studied the oceanic transport of radioactive elements, UV-radiation and cosmic rays (myons).

In 1977 he began to study microwave induced hyperthermia for tumour-therapy and since then this project has expanded very rapidly. He developed an equipment for local hyperthermia in patients with breast carcinoma, and recently equipment for heat treatment of benign prostate hyperplasia and menorraghia. He started already in 1982 with biomedical applications of nuclear magnetic resonance (NMR) and has developed and built a special equipment for performing in vivo NMR studies in both animal and man. In close co-operation with several other scientists he is also conducting *in vitro* studies of the NMR relaxation times in tissues of thyroid and brain, also studying the influence of paramagnetic ions and super-paramagnetic particles. He has studied extensively clinical measurements of flow, microcirculation, diffusion and perfusion with NMR. During the past years he has been deeply involved in studying the effects of electromagnetic fields on healthy brain and on brain-tumours. He was co-sponsor for the New York Academy of Science conference on the biological and health effects of clinical NMR examination held in 1991. At present he is deeply involved in studies of the effect of low power pulsed microwave fields on the blood brain barrier and other biological systems. He is also engaged in using electromagnetic fields for tumour therapy and has made a lot of progress in the use of high voltage electric fields for permeabilization of tumour cell membranes in vivo. The objective of that investigation is to study the destructive effect of electropermeabilization on tumours and its ability to introduce toxic compounds into tumour cells. He is studying the therapeutic effect of electropermeabilization on tumour cells in presence of chemotherapeutic agents (for example, Bleomycin) and radioimmunotherapeutic agents.