BIOEFFECTS OF LONG-TERM EXPOSURES OF ANIMALS

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INTRODUCTION

Between 1962 and 1992 at least 10 major studies have been reported in the literature of the United States on the biological effects of long-term exposure to radiofrequency radiation (RFR) spanning a frequency range of 800 to 9270 MHz. Exposure levels in the studies ranged from 0.48 to 100 mW/cm^2 with specific absorption rates (SARs) ranging from 0.075 to 45 W/kg. The studies include the work of Prausnitz and Susskind[1] on the life span and pathology in mice; Spalding et al.[2] on the body weight, activity, hematopoiesis and life span of mice, Preskorn et al.[3] on tumor growth and greater longevity in mice after fetal exposure; Guy et al.[4] on ocular and physiologic effects in rabbits; McRee et al.[5] on hematological and immunological effects in rabbits; Chou et al.[6] on body weight, electroencephalogram (EEG), hematology, and morphology in rabbits; Szmigielski et al.[7] on development of spontaneous and benzopyrene-induced skin cancer in mice; Chou et al.[8] on body weight, food consumption, and hemotological, morphological, chemical, protein electrophoresis and lymphocyte blast transformation parameters in rabbits; Adair et al.[9] on metabolic rate, internal body temperature, blood indices, and thermoregulatory behavior in squirrel monkeys; and Chou et al,[10] on general health and longevity, serum chemistries, hematological values, protein electrophoretic patterns, thyroxine, plasma corticosterone levels and histopathological patterns in rats. An overview of each one of these studies is given below, followed by a table summarizing the results.

REVIEWS

Prausnitz and Susskind[1] conducted the first long-term exposure of animals in this country. They exposed 200 male Swiss albino mice (in groups of 10) to 9270 MHz pulsed microwaves (2 μs, 500 pps) of 100 mW/cm^2 average power density for 4.5 minutes daily over a period of 59 weeks, resulting in an average body temperature rise of 3.3° C during

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each exposure. The animals were contained in a compartmentalized circular polystyrene cage with a plastic window-screen floor. The animals were separated from each other by wedge-shaped compartments formed by small rectangular strips of plastic with the long dimensions oriented radially every 36° in the circular cage. The cage was placed above a standard gain horn (4 x 6 cm aperture) so that the animals were ventrally exposed while being rotated at 1 rpm around the vertical axis of the cylindrical cage to randomize the effect of the multiple reflections between the adjacent animals. The exposure level corresponded to one-half of a LD_{50} exposure dose. Between exposures, the colony of 300 male Swiss albino mice were housed in a cabinet where the temperature was maintained between 21° and 24° C. Histopathology was performed on all of the 200 exposed and the 100 control group mice. Based on the Radio Frequency Radiation Dosimetry Handbook (RRDH)[11] the whole-body average SAR for the animals is estimated to be 45 W/kg.

The authors reported that the rate of lethality was higher in the control group but not significantly so. Part of the colony suffered from pneumonia during the last 3 months of the exposures resulting in a number of deaths in both groups. The authors speculate that the periodically induced slight artificial fevers are of some benefit to the animal in combating disease. At the time a death of an animal was noted, it was autopsied and histological parameters of key tissues were determined. These included the liver, spleen, lymph nodes, kidneys, adrenals, gut, lungs, and testes. Other tests included random blood count, spot checks of urine for glucose, weekly weighing of all mice, and recording of body temperature. Of the total of 300 mice in the experiment, 132 were sacrificed in 3 sacrifice series to assess damage in surviving mice. Sixty eight mice that had died, because of extensive postmortem changes, were unsuitable for histological assessment. The remaining 100 animals were used for the longevity assessment. Testicular degeneration was found in 40% of the dead exposed mice and only 8% of the controls. The authors reported that cancer of the white cells was found in 35% of the exposed mice and 10% of the controls evidenced by either monocytic or lymphatic leucosis or myeloid leukemia. The authors described leucosis as a noncirculating neoplasm of the white cells and leukemia as a circulating leucosis. A three-sacrifice series was carried out at 7, 16, and 19 months from the start of the exposures. No effects in the first sacrifice series of 5% of both exposed and control groups were reported. However, in the second sacrifice involving twice as many mice, 30% of the exposed and 10% of the control animals were reported to have leucosis but no correlations were reported regarding testicular degeneration as seen in the acute death histologies. As a result of this partial contradiction, a third sacrifice series was performed on the remainder of the 67 exposed and 19 control mice. In this group 21% of the exposed and 5% of the controls were reported to show evidence of some degree of testicular degeneration, but no correlation was found with leucosis, in this sacrifice series. Elder and Cahill,[12] in examining the reported results, found that when animals in the sacrifice series 1 and 2 were ignored, the difference in survival was not significant at 14 months into the experiment but was significant at 19 months. A 2 x 2 contingency table analysis also revealed that the prevalence of leucosis in exposed animals was not significantly different from that in the control animals or any of the sacrificed groups. Elder and Cahill[12] also determined that the combined groups including all of the animals at risk in the original longevity group did not have statistically different leucosis rates.

In their analysis of the same data, Roberts and Michaelson[13] concluded that a plausible case could be made for the concept that the study of Prausnitz and Susskind[1] demonstrated that the microwaves induced beneficial rather than detrimental effects, and that the study neither confirms nor denies the correlation between the exposure to microwaves and development of neoplasia, and they asserted that the report should not be cited as suggestive of such a correlation.

Spalding et al.[2] reported the effects of 800-MHz RFR on body weight, activity, hematopoiesis and life span in mice. The authors divided forty-eight mature-strain RF virgin female mice into 2 groups of 24 each, one group exposed to 800 MHz RFR at an average incident power level of 43 mW/cm^2, 2 hours per day, 5 days a week, for 35 weeks and the other group sham-exposed for the same period of time. The mice were placed in a 12-compartment acrylic resin plastic restraining cage during sham control and RFR exposures within a standard rectangular, 9.75 x 4.875, 10 ft. long ventilated wave guide. The exposed animals were subjected to a TE_{10} mode field configuration with a spatially averaged power density of 43 mW/cm^2 over the cross-section of the waveguide. However, with the half-sinusoidal distribution of the TE_{10} mode electric field across the wave guide, the peak exposure at the center of the wave guide would be 86 mW/cm^2. The authors selected the mice at random for each exposure run. They stated that because of the large number of exposures, those for any one mouse should integrate out to be the average exposure level. Assuming that the mass of each mouse is approximately 20 grams and based on the estimated power absorbed by the mice of 0.5 Watts, the authors calculated a whole-body SAR as averaged over all exposure times of 2.1 W/kg. However, it should be noted that mice exposed at the center of the wave guide would have experienced a whole-body average SAR of 4.2 W/kg. The authors indicated that during the 33rd and the 34th RF exposures, four mice died from thermal effects. They found that the exposure field was much higher than estimated due to a standing wave. When the authors placed the exposure cages 1 inch closer to the source of rf power away from the maximum electric field, no further lethality was observed during the exposure. It should be noted that based on the RRDH, 43 mW/cm^2 and 83 mW/cm^2 free space exposures would produce whole-body average SARs of 1.29 to 12.9 W/kg for the former and 2.58 to 25.8 for the latter, depending on the orientation of the animals.

The authors concluded that no detrimental effects, detectable in the blood or the physical condition of the animals, were evoked from the rf exposure. Though the mean life span of the experimental group was 19 days longer than that of the control group, the difference was not statistically significant.

Preskorn et al.[3] reported greater longevity and retarded tumor growth in mice exposed fetally to RFR. The exposure apparatus consisted of a modified Tappan R3L multimode cavity fed by half-wave, 60 Hz sinusoidaly modulated 2450-MHz microwave energy from an overhead waveguide. Four or more maternal subjects and later, four weanling offspring were exposed for each treatment. A total of 4 in utero and 36 postnatal treatments of 20 minutes in duration were alternated across time, within days, between radiation and sham treatments. Average whole-body dose rates of 35 mW/kg were obtained calorimetrically from exposed animals. Based on the RRDH, the equivalent average whole-body plane wave exposure would be 29.2 mW/cm^2. The before and after treatment colonic temperatures averaged 37.4° and 39.8° respectively.

The authors' experiment was based on a 2 x 2 factorial design with 12 CFW mice each assigned to one of 4 exposure conditions including 2 exposure levels, 0 and 35 mW/g, and 2 treatment periods, in utero (11-14 day of gestation) and postnatal (19-54 day postpartum). During the 16-day postpartum each of the 48 weanlings was injected with a sterile homoginate of tumorous tissue containing the avian, fast reticuloendothelial T virus prepared from sarcomatous tumors of adult CFW donors. All radiation treatments were administered between 7 am and 10 am at an ambient temperature averaging 23° C, humidity ranging between 50% and 75% and a forced ambient air flow velocity of 0.1 m/sec. Group 1 mice were sham exposed during both exposure periods. Group 2 mice were sham exposed in utero and exposed later, Group 3 mice were exposed in utero and sham exposed later. Group 4 mice were exposed during both periods. One treatment was given daily with all days of treatment occurring consecutively during each of the two exposure

periods. After implantation of the homogenate each of the 48 weanlings were examined each day both visually and by palpation until the 93rd day postpartum at which time they were euthanized by an overdose of sodium pentobarbital to permit histopathological examination. The fetally and postnatally exposed animals of group 1 developed tumors at a rate normally reported for a CFW mice of the commercially available strain. Five out of 12 of the group 2 mice developed sarcomas, indicating that the postnatal treatments had little effect on the rate of tumor induction. Two of the 12 fetally but not postnatally exposed mice of group 3 and 1 of the 12 mice of group 4 exposed during both periods developed sarcomas. The reduction of incidence of tumors in the fetally exposed animals was statistically significant. One of the mice from each of the groups 3 and 4 not only had a lower incidence of sarcomas but experienced an apparent regression of tumors. The second experiment was similar to the first except larger numbers of gravid CFW mice were used to provide larger samples of experimental and control subjects and all the treatments were administered in utero. Both exposed and control animals were observed for longevity across a 3-year span of time. Eighty-four weanlings were selected from 10 exposed dams, and 60 weanlings were selected from 8 sham-exposed dams. The 44 weanlings were implanted with the T virus homogenate on the 16th postpartum and thereafter segregated by sex into groups of 5 or fewer animals and placed in cages in a mouse vivarium. As in the first experiment, observations at 2.5 months implantation indicated that the percentage of fetally exposed mice with tumors (15%) was statistically smaller than that (37%) of controls. Later, however, the difference narrowed and by the 4th month 46% of the fetally exposed mice and 40% of the control mice had tumors, an insignificant difference. Though the tumors were delayed in the fetally exposed mice, the absolute incidence was not significantly altered. Again in this group there was evidence of regression of tumors in fetally exposed mice. The authors conclude that the data of both experiments support the notion that moderate short-term elevation of fetal body temperature by microwave radiation can delay onset of an experimental neoplasms and reduce the mortality rate of tumor-bearing animals. They further believe that the data reflects an enhanced immunocompetency that has its origin in elevation of fetal, and perhaps maternal temperature.

Guy, et al.[4] report the results of exposing male New Zealand white rabbits to 10 mW/cm^2, 2450 MHz cw microwave radiation for 23 hours per day for 180 days in a miniature anechoic chamber. A total of 8 rabbits were studied simultaneously; 4 received radiation and 4 served as controls. Each rabbit was kept in an acrylic cage within a rectangular, 16 x 16 cross section, 122 cm high chamber with 20.3 cm thick microwave absorbing fiber walls and floor. A standard microwave horn was placed 1 meter above the centerline of the body of each rabbit. The sham-exposure chambers were constructed with thinner panels of fiber packing material of the same size and similar composition as the microwave absorber. In all cases the absorber and packing material panels were covered with a thin plastic film to block outgasing that might arise from the material. With the exception of the absence of microwave horns, topography of the control chambers were similar to that of the exposure chambers. The ambient temperature of within each chamber was maintained at $24^\circ \pm 2^\circ$ C by thermostatically controlled air conditioner. A watering system was designed to eliminate the problem of field intensification during drinking. As water entered the chamber through a tubing it was separated into individual, radiolucent drips by a needle valve. Each rabbit would drink by catching drops in its mouth with excess water draining immediately from the chamber, thereby breaking up any continuously conductive pathway. The floor of each acrylic box consisted of 1 cm diameter glass rods spaced at 2 cm. Urine was drained to the outside of the chamber in a collection beaker. Each animal remained in a position with its body extended along the long dimension of the cage (parallel to the electric field for exposed animals) during most of the long-term

exposure. Input power to each standard gain horn was set at 32 Watts, which provided a power density of 7 mW/cm^2 at the position of the body axis of the animal (measured with the animal absent) and 10 mW/cm^2 at the usual position of the animal's head, approximately 16 cm above the long axis of the body. Thermographic measurements, based on techniques described by Guy,[14] indicated a peak SAR of 17 W/kg in the head of the exposed animal. Maximum whole-body-average SAR is estimated to be 1.5 W/kg based on the RRDH.

The animals, weighing approximately 4 kg, received periodic examinations of the eyes with a slit lamp microscope and the following parameters were monitored throughout the exposure period: body mass, urinary output, rectal temperature, hematocrit, hemoglobin, white cell count, platelet count and basic blood-coagulation studies. No significant differences other than a decrease in a percentage of eosinophils were found between the experimental and control animals. The authors noted, however, that eosinophil percentage varies widely among animals.

McRee et al.[5] performed additional analyses on the same animals exposed by Guy et al.[4] discussed above. Immediately after termination of exposure of the rabbits, blood samples were drawn for standard hematologic and serum-chemistry analyses. Necropsies were performed on all eight animals and histopathological analyses were made on specimens of the tongue, esophagus, trachea, lung, heart, liver (including gall bladder), stomach, small and large intestine, spleen, thymus, kidney, urinary bladder, testes, skin (ear), brain, thyroid, pancreas, adrenal, pituitary, sternum (for bone marrow) and thigh muscle. A sample of splenic tissue from exposed and control animals was taken aseptically from cadavers for immunological studies. The lymphoid cells obtained from individual spleens were cultured and stimulated with the mitogens, phytohemagglutinin-P (PHA), Concanavalin A (Con A) and pokeweed mitogen (PWM).

Of the hematological and clinical chemistry parameters analyzed from the blood samples, significantly lower levels of albumin, calcium and eosinophils were found in the samples from the exposed animals. The authors stated that since only 3 of the 41 parameters were significantly different, which is close to the number expected by chance; the validity of these changes can only be determined through independent replication. No significant changes were seen in catecholamines and creatinine content in the urine. Blood specimens taken from the animals 30 days after termination of exposures showed no significant differences. Depression in eosinophil numbers seen earlier had normalized 30 days after exposure. A reliable decrease in the albumin (albumin/total globulin ratio [A/G]) was found in the exposed animals, and was due to both the decrease in the albumin and an increase in the total levels of globulin. The authors noted, while neither of these 2 parameters has changed significantly in themselves, the combination of the trends causes a significant change in the A/G ratio. They concluded that the biological importance of this finding is difficult to interpret and may be negligible since A/G ratio of the treated and controls was essentially the same at the termination of the exposure. Examination of bone marrow revealed an abnormal myeloid/erythroid ratio (ratio of leukocytes of the granulocytic series to nucleated erythrocyte precursors) in the exposed rabbits. The authors state that the importance of the finding is questionable since the hematologic (erythrocyte and leukocyte counts) parameters did not differ between treated and control rabbits. Both absolute and relative masses of organs as a percentage of body mass showed no significant differences in the exposed and control animals. At necropsy, histopathologic examination of representative tissues showed no lesions that could be attributed to microwave irradiation, which led the authors to report that the chronic irradiation at 10 mW/cm^2 either did not induce pathological changes or it induced only minimal, reversible changes. In vitro response of lymphocytes to stimulation was reduced for exposed animals as compared to control levels, but the difference was not statistically significant. The same statements are

true for splenic cells cultured in the presence of ConA, but at all three levels of PWM employed, there was a significant reduction in responsiveness of spleen cells to stimulation in microwave-exposed specimens as compared to controls. The authors state that due to the limited number of animals used in the study, the results are not meant to be definitive. Rather, they are aimed at providing insights into potential biological effects that should be further studied.

Szmigielski et al.[7] studied the effect of RFR on accelerated development of spontaneous and benzopyrene-induced skin cancer in mice. The authors exposed 2 groups of mice: C3H/HeA mice with a high incidence of spontaneous breast cancer and Balb/c mice with skin cancer, resulting from painting with 3,4-benzopyrene (BP). The animals were exposed at 5 or 15 mW/cm^2 for 2 hours daily, 6 sessions per week. The C3H/HeA mice were exposed from the 6th week to the 12th month of life. The BP-treated Balb/c were exposed either 1 or 3 months prior to or simultaneously over 4 months with BP treatment. The mice were exposed in an anechoic chamber with walls covered with styrofoam-graphite absorber and a 2450 MHz microwave beam directed vertically from a 30 x 30 cm horn antenna placed at a distance of 220 cm above the cages. The sham-exposed animals were exposed at different times in the same chamber without microwave radiation. The power source was a microwave generator produced in the USSR with a maximum power output of 150 Watts cw. Ten mice were contained in each of 4 polymethacrylate cages placed on the 30 x 50 cm floor of the anechoic chamber. Power densities were measured with the cages removed. The authors did not indicate whether the mice were free to move around or were restrained to a particular orientation with respect to the polarization of the horn antenna. The authors determined the SAR calorimetrically by exposing a mouse cadaver and a liquid phantom, with implanted liquid crystal probes. The cadaver and phantom were exposed to power densities ranging from 20 to 60 mW/cm^2 and the temperature changes after 1, 3, and 5, minutes exposure were noted. From the temperature increase and known power density, the authors calculated the SAR for 5 and 15 mW/cm^2 exposures as 2-3 W/kg and 6-8 W/kg respectively. No comparisons between the SARs were obtained for the cadaver and the phantoms nor was any indication given of the polarization of the field with respect to the long axis of the exposed animals. According to the RRDH, the estimated maximum SARs for the 2 exposure levels of 5 and 15 mW/cm^2 would be 6 W/kg and 18 W/kg respectively for E-polarization (electric field parallel to the long axis of the animal) or 2.2-6.6 W/kg respectively for H-polarization (magnetic field vector aligned parallel to the long axis of the exposed animal). The measurements appear to be consistent with exposure to H-polarized RFR. However, if the animals were free to move around so that they could be aligned with the E vector just as often they could be aligned with the H vector, the whole-body-average SAR would likely be 4.1 and 12.3 W/kg respectively for the 2 exposure power densities. The authors did not indicate whether the measurements were done on a single cadaver or phantom in the chamber or whether they were exposed in the presence of other subjects.

The authors subjected one group of male Balb/c mice (6 weeks old) to chronic stress by placing each into a 5 x 6 x 10 cm compartment with 20 contained in a 20 x 30 x 10 cm cage. Each compartment was provided with standard food and water. The animals were grown under these conditions for 8 to 10 months depending on the schedule of the experiments. These animals served as additional controls to normal sham-exposed and microwave-exposed animals. Measurement of natural antineoplastic resistance was done by injecting solid transplantable L1 sarcoma cells into recipient healthy Balb/C mice. Biologic experiments determined that development of 1-4 nodules in the lungs would result, 14 days after injection of 2 x 105 cells (in 0.1 ml of saline). In animals exposed either to microwaves or to chronic confinement stress significantly higher numbers of lung nodules were observed after 1 or 3 months of treatment. After 3 months of exposure at 15 mW/cm^2,

10.8 ± 2.1 lung nodules were found vs 6.1 ± 1.8 nodules found in mice exposed at 5 mW/cm^2; the latter was close to the 7.7 ± 2 nodules found in mice with chronic confinement stress. Nodules in sham-exposed mice (3.6 ± 2.2) was significantly less than those in mice exposed with microwaves at 5 mW/cm^2. Experiments on C3H/HeA mice showed similar results. Radiation with microwaves resulted in significant acceleration of breast tumors in mice exposed at 15 mW/cm^2. The first tumors developed in the 5th month of life with half of the animals developing tumors at 219 days. Of the animals exposed to microwaves at 5 mW/cm^2 or to chronic confinement stress, half of the animals had tumors at 261 days and 255 days respectively. Both were significantly different from controls or sham-exposed animals (297 days). Survival times in the mice also followed the same trends. The mean survival time of one-half of the animals (MST50) in controls was 358 days, MST50 for 15 mW/cm^2 exposed, 5 mW/cm^2 exposed, and confined mice was 231, 264 and 272 days, respectively while MST50 for control and sham exposed mice was 326 days. For control Balb/C mice the mean skin cancer development time (MCDT) was 285 days; the time where half the animals developed skin cancer (CDT50) was 296 days; and MST50 was 205 days. Microwave exposure and confinement stress prior to treatment with BP resulted in significant acceleration of the development of skin cancer and shorter survival time. For mice exposed to 15 mW/cm^2 microwaves for 3 months and later treated to BP, MCDT was 160 days; CDT50 was 171 days; and MST50 was 205 days. For animals sham exposed for 3 months and later treated with BP, MCDT was 256 days; CDT50 was 272 days; and MST50 was 312 days. Similar results were found in groups of mice exposed to microwaves and treated with BP at the same time. The shortened survival time was also found for these animals as a result of the earlier appearance of cancer. Also for mice exposed to 15 mW/cm^2 microwave radiation prior to treatment with BP and simultaneously with application of BP was early appearance of fully developed skin cancer; MCDT was 121 days; CDT50 was 131 days; and MST50 was 165 days. There was very little difference in the results for mice exposed to 5 mW/cm^2 and those exposed to chronic confinement stress. The authors concluded that their data demonstrated significant acceleration in the development of both spontaneous and chemically induced tumors in mice exposed with 2450 MHz microwaves for 1-6 months. The authors indicate that the results are accompanied with the lowering of the natural antineoplastic resistance both for microwaves applied before or simultaneous with benzopyrene treatment.

Chou et al.[6] reported results of an experiment on the effects of continuous and pulsed chronic 2450 MHz microwave exposure on body weight, blood, eyes, electroencephalogram, evoked potentials, and pathology of young 3-month-old adult New Zealand rabbits. Eighteen rabbits (nine males, nine females) were equally divided into 3 groups. One group was exposed to 1.5 mW/cm^2 CW for 2 hours daily for 3 months, another group was simultaneously exposed to the pulsed microwaves (10 μs, 100 pps) at the same power density, and the third group was simultaneously sham exposed. Exposures were made in 3 miniature anechoic chambers of the type described by Guy[15]. The RFR in each chamber was provided by an S band standard gain horn located 1 meter above the animal. The inside walls and floors of the chambers were lined with microwave absorber. The rabbits were contained in a plexiglas cage oriented so that their long-body axis was parallel to the electric field most of the time. Urine and feces were collected in a Plexiglass pan located under the cage. The chambers were ventilated by a fan mounted at the top that drew air up through the porous microwave absorbing floor. The exposure area was illuminated by 4 lights placed at the top of the chamber around the waveguide adapter above the horn so there would be no field perturbation. The animals had access to dry rabbit food chow but no water during each 2-hour exposure. The SAR patterns were obtained thermographically at the sagittal plane by exposing a sacrificed rabbit to high-power fields for a short time as described by Guy[14]. Peak SARs of 2.1 W/kg and 1.6 W/kg

were measured in the back and head of the animal, respectively. The estimated whole-body average was 0.24 W/kg based on the RRDH. Three radiolucent carbon-loaded Teflon electrodes were implanted in the head of each animal so that electroencephalographs and evoked-potential recordings could be made during the experiments. Exposure began 1 week after the final surgery. Exposure for each group of 3 animals was rotated to control for possible effect of circadian rhythms.

The authors permanently implanted the EEG electrodes in the skull in contact with the dura, at the sensory motor, occipital, and nasal areas of the brain. The opposite end of each carbon-loaded Teflon fiber was terminated by a metal pin embedded in dental acrylic placed over the area of the implanted electrodes. At appropriate times between exposures, EEG signals were recorded by connecting the leads of the recording instruments to the metal pins embedded in the acrylic. Measurements were made every other day of body weight and blood samples were taken monthly for hematological, chemical and morphological studies. EEG and evoked potential recordings were made every Friday after the 2-hour exposure. The EEG from sensory motor and occipital areas was recorded after a 5-10 minute adaptation period from the time the electrodes were connected over a period of 20 minutes. Subsequently 5-minute, visual-evoked responses were recorded from the occipital cortex by stimulating the eyes with a strobe light at the rate of 1 flash per second. During the last 5 minutes the auditory-evoked responses were recorded from the sensory motor cortex by presenting 70 dB, 0.1 ms clicks at the rate of 1 per second. The EEG data were analyzed by computer, off line, to obtain the frequency spectrum and averaged amplitudes. The evoked responses were recorded by means of a computer-averaged transients.

Since there was so much variability in the frequency spectrum from animal to animal and different recording sessions, individual frequencies could not be compared so integration of the power spectrum was performed. Though there were large values of standard deviations and great variability in the results, there was a trend of decreased amplitude of the EEG signals in all of the animals in the later part of the experiment, probably due to acclimation. Statistical tests showed no difference in the data obtained from CW, pulsed and control animals. Slit lamp examinations both before and after the 3-month exposure revealed no cataract development in any of the 18 animals. Unsuccessful attempts were made to determine the effect of apomorphine induced hypothermia at the end of the 3-month exposure. Not only was there a large variability in observed temperature rise and behavioral responses on the small group of animals but five animals (1 CW exposed, 3 pulsed exposed, 1 control) died during the experiment. Thirteen survivors were sacrificed and histology was performed on lungs, heart, vessels, stomach, small and large intestines, pancreas, liver, gall bladder, adrenal gland, kidneys, bladder, testes or ovaries, bone marrow, spleen, and brain. Histopathological studies showed no consistent, significant differences among the 3 groups.

Chou et al.[8] studied the effect of 2450 MHz cw microwave exposure on health profile of two groups of 16 male New Zealand rabbits. The rabbits were exposed to incident power densities of 0.5 and 5 mW/cm^2 for 7 hours daily, 5 days a week for 13 weeks. The effects of the radiation were assessed on food consumption, body mass, blood parameters including hematology, morphology, chemical, protein electrophoresis, and lymphocyte blast transformation. The eyes were examined for cataract formation and pathological examinations of 28 specimens of organs and tissues of each rabbit were performed. Sixteen miniature anechoic chambers as described by Guy[15] and in the above paper overview were used for chronically exposing the rabbits to 2450 MHz cw microwaves. During the 7-hour exposures used for this experiment, water was supplied through a special water-dripping system. A water bag was hung outside of the chamber and water was guided by plastic tubing to drip into a small cup at the rate of 1 drop per second. Excess water flowed to a

plastic bag at the bottom of the chamber. The design eliminated any field enhancement due to animal contact with a voluminous water container. A total of identical 8 exposure and 8 sham-exposure chambers were set up in a single room. In the first experiment 8 New Zealand rabbits, initially weighing approximately 2 kg were exposed to 0.5 mW/cm^2, 7 hours a day, 5 hours a week for 13 weeks. The other 8 animals of similar body mass were sham exposed at the same time. The SAR patterns in the sagittal plane, obtained thermographically as described by Guy,[14] indicated a peak SAR of 0.7 W/kg in the back and 0.55 Watts in the head of the exposed animal. Based on the RRDH, the whole-body-average SAR for this exposure level was 0.075 W/kg. The second group of animals, exposed to 5 mW/cm^2, would experience a whole-body average SAR of 0.75 W/kg and peak SARs of 7 W/kg and 5.5 W/kg in the back and head, respectively. The animals were exposed between 9 am and 4 pm at an environmental temperature of 21° ± 1.5° C and relative humidity of 50% ± 10%. Body mass and food consumption during the 7-hour exposure period was measured daily. Blood samples were taken before the initial exposures and monthly thereafter from which hematological, chemical, protein electrophoresis, and lymphocyte studies were performed. Lymphocyte studies included lymphocyte blast transformation, mitotic index and stimulation index. All animals were examined for cataracts with a slit lamp before and at the end of the 13-week exposure period. Complete gross necropsy on each animal was performed, and histological examinations and evaluations were made on tissues from all organs including femur bone marrow, pituitary, adrenals thyroid, parathyroid, trachea, esophagus, brain, heart, skeletal muscle, spleen, liver, gall bladder, pancreas, lungs, salivary glands, cervical lymph node, kidneys, urinary bladder, testes, epididymis, prostate, stomach, duodenum, ileum, colon, mesenteric, and any grossly observable lesions. The tissue samples were microscopically evaluated in the blind by a pathologist. The only statistical difference found between test results on the exposed and the control animals was a significant ($P < .01$) decrease in food consumption in the 5 mW/cm^2 exposed animals. Based on this finding and similar findings by other investigators, the authors felt that the absorbed microwave energy (0.75 W/kg is 20% of metabolic rate) was used by the animals partially for metabolism causing 23% reduced food consumption during the 7 hour 5 mW/cm^2 exposure. It is also pointed out that the failure to find changes in blood tests was not in agreement with several other similar investigations conducted in the Soviet Union and East European countries.

Adair et al.[9] investigated the effect of 2450 MHz CW RFR on changes of both behavioral (stable internal temperature controlled by voluntary behavior) and physiological (stable internal temperature controlled by involuntary autonomic mechanisms) thermoregulatory responses in squirrel monkeys exposed for 40 hours/week for 15 weeks at power densities of both 1 mW and 5 mW/cm^2 at controlled environmental temperatures of 25° , 30° , or 35° C. The monkeys were chronically exposed, a pair at a time, in a standard 2.45 x 2.45 x 3.66 m anechoic chamber to microwave radiation from a 15-dB standard gain horn. During exposure within the anechoic chamber and sham exposure outside of the chamber, the monkeys were housed individually in exposure cages consisting of 61 cm high, 30.5 cm diameter plexiglas cylinders, each contained in environmentally controlled 5 cm thick 61 x 91 x 122 cm insulating foam chambers. A drip water system as described by Guy[15] supplied water to the animals. Although the RRDH indicates an SAR in the squirrel monkey of 0.1 (W/kg)/(mW/cm^2), the authors measurements on exposed phantom models indicated a whole-body-average SAR of 0.16 (W/kg)/(mW/cm^2). The difference is probably due to the difference in heights of the models (33 cm used by the authors versus 23 cm used by the RRDH).

The authors performed standardized tests of the physiological thermoregulation on each animal prior to and at predetermined times during and after chronic exposures to establish reliable baselines. The tests were conducted in a 92 x 102 x 66 cm insulated

chamber with no microwaves present. Under different controlled environmental temperatures, four skin temperatures (abdomen, tail, leg and foot), colonic temperature, oxygen consumption, and sweating rate from the foot of a restrained monkey were measured each minute by an on-line computer. All environmental temperatures were controlled within ± 0.5° C.

The authors performed standard behavioral tests by restraining the monkey in the far field of a 15-dB standard gain horn while it was enclosed by an environmentally controlled insulated foam box within an anechoic chamber. Each monkey was trained to select between between 2 preset air (flow velocity of 0.36 m/s) temperatures of 10° and 50° by means of pulling a response cord. Each response was reinforced by a 15-second application of 50° C air followed by an application of 10° C air lasting until the animal responded again. During the behavioral tests, the monkeys were exposed to microwave power densities of 0, 4, 6, 8, 10, and 12 mW/cm^2 with corresponding SARs (obtained from phantom and confirmed by live-animal measurements) of (0.15 W/kg)/(mW/cm^2). The test protocol required each animal to equilibrate for a minimum of 1 hour to an environmental temperature of 20° ± 0.5° C. The temperature was then successively ramped to different levels (6.6° increment for the first, maintained for 45 m, and 3.3° increments maintained for approximately 37.5 m, each, for the remaining increments) up to 36.5° C. After training, each animal underwent 3 or more 4-hour baseline test sessions of behavioral regulation to establish the normally preferred environmental temperature pattern of responding and the preferred colonic and skin temperatures. Prior to chronic microwave or sham exposure, each animal was given 3 standardized behavioral tests to determine the microwave intensity for whole-body exposure that would reliably alter thermal regulatory behavior, which was found to be 10 mW/cm^2.

Animals were chronically exposed to 2 power densities, 1 and 5 mW/cm^2, and 3 environmental temperatures, 25°, 30°, and 35° C, for a total of 6 treatment groups. All groups of animals were subjected to 3 test phases including: 1) pre-exposure phase of 8 to 12 weeks duration during which a number of physiological, baseline behavioral thermal regulation without microwaves, and behavioral with microwaves (to determine the microwave threshold to alter thermoregulatory behavior) tests were given; 2) chronic microwave or sham exposure phase of 15 weeks duration during which a number of physiological and behavioral tests were given; and 3) post exposure follow-up phase of 4 to 8 weeks where the animals remained in the home cage, except when additional physiological behavioral and tests were administered.

Blood samples were taken at 1, 5, 10, 15, and 20 weeks of the treatment. Assessments were made of cell counts, hemoglobin determination, serum-thyroxine concentration, thyroxine binding capacity, blood sodium, potassium, bicarbonate, chloride concentrations, total serum protein and albumin concentrations.

Tests showed no significant differences in metabolic rate, internal body temperature, blood indexes, or thermoregulatory behavior between sham- and microwave-exposed animals. However, the authors found that the ambient temperature prevailing during chronic exposure could produce an effect, especially in the 35° C environment where there was an increase in sweating rate. Skin temperature was found to be reliably influenced by both ambient temperature and microwaves, and the most robust effect of microwave exposure was found to be a reduction in body mass as a function of power density.

Chou et al.[10] reported the biological effects in 100 male Sprague-Dawley rats exposed over a lifetime to pulsed-microwave radiation as compared to those in an equal number of sham-exposed animals. All 200 rats were maintained under specific pathogen-free (SPF) conditions while individually housed in circular waveguides, half of which were energized to produce circularly polarized 2450 MHz pulsed (10 μs duration, 800 pps) exposure and half of which were not energized. The pulsed microwaves were square-wave amplitude

modulated at 8 Hz, producing 62.5 ms wide trains of 50 pulses each separated by 62.5 ms intervals. The modulation was applied, based on evidence of modulation frequency window effects[16] which were reported to be most pronounced at dominant EEG frequencies (16 Hz) of exposed chicken and cat brains. Eight Hz is the equivalent frequency for the rat brain.

Exposed and sham-exposed animals were individually housed in single plastic cages placed in each waveguide. Exposure and sham-exposure guides were randomly placed in alcoves in the SPF rooms where air-flow rate was programmed for 22 exchanges each hour at a temperature of 21° ± 1° C and a relative humidity range of 30% to 70%. The exposure waveguides were each energized by a transducer at one end that excited circularly polarized waves with power that was partially absorbed by the exposed animal, partially absorbed by the waveguide walls, partially reflected back to the feed transducer, and partially absorbed in matched terminating loads at the other end. As described by Guy et al.,[17] the whole-body-average SAR of the rats exposed to the guided circularly polarized waves remains relatively constant regardless of the orientation of the animals. Whole-body-average SAR in the exposed rats was determined by 2 methods, 1) measuring the SAR by twin-well calorimetry in sacrificed animals ranging from 200 to 800 grams in mass, exposed to high power density and 2) measuring the waveguide reflected and terminal load absorbed powers and subtracting them from the incident power to determine the total power absorbed in the waveguide walls and exposed animal. The waveguide power loss may be quantified by subtracting the the power loss in the animal, determined by the twin-well calorimetry, from the total. During the long-term exposure period, the whole-body SAR of each exposed animal was continuously monitored for one day out of each 50 days by this method. An average power density exposure level of 480 $\mu W/cm^2$ was chosen since the dosimetry measurements showed that the whole-body-average SAR ranged from 0.4 W/kg (basis of a number of exposure standards) for a 200 gm rat to 0.15 W/kg for an 800 gm rat. The exposures began at 8 weeks of age and continued daily, 21 1/2 hours per day, for 25 months. Blood samples taken at intervals of every 6 weeks up to the 60th week and every 12 weeks thereafter, were analyzed for serum chemistries, hematological values, protein electrophoretic patterns, thyroxine, and plasma and corticosterone levels. In addition, daily measures were made of body mass, food and water consumption by all animals, and O_2 consumption and CO_2 production in a sub-sample (N = 18) of each group. Behavioral activity was assessed in an open field apparatus at regular intervals throughout the study. After 18 months, ten rats from each group were euthanized to test for immunological competence and to permit whole-body analysis, as well as gross and histopathological examinations. At the end of 25 months, the survivors, consisting of 11 sham-exposed and 12 radiation-exposed rats, were euthanized for a similar analysis. The remaining 159 animals, upon either spontaneous deaths or terminated in extremis, were examined histopathologically. A total of 155 biological parameters were examined.

Microwave exposure did not produce any significant effects on activity levels of the animals nor did it cause any significant effects seen in the serum corticosterone levels, except for a significant transient elevation at the time of the first session only for the exposed animals, and at the time of the third session only for the sham-exposed animals. Exposed and sham-exposed animals had comparable levels of corticosterone on all other regular sampling sessions. A follow up study by Chou et al.[18] of two groups of 20 animals each, exposed in the same system for 6 and 12 months revealed no statistically significant differences in corticosterone levels between exposed and sham-exposed animals. Immunological competence studies of rats sacrificed at 13 months indicated a significant increase in both splenic B cells and T cells, which was not detected in the animals sacrificed at the end of the 25 months of exposure. No significant effects were seen in the percentage of complement-receptor-positive cells in the spleen either for the interim or final euthanasia. Mitogen-stimulation studies on the animals sacrificed at 13 months

exposure revealed significant differences between groups in their responses to B- and T cells specific mitogens. Exposed animals had a nonsignificant increase in the response to PHA but a significant increase in response to lipopolysaccharide (LPS) and mitogen PWM as compared with sham-exposed animals. The exposed animals had a significantly increased response to ConA and a decreased response to purified protein derivative of tuberculin (PPD). Mitogen-response data were not available from the 25-month euthanized animals since lymphocyte cultures failed to grow. In the follow-up study by Chou et al.[18] no significant differences between 20 exposed and 20 sham exposed rats were observed in the proliferation of thymocytes to ConA, PHA, and PWM after 6 and 12 months of RF exposure, nor were there any differences found for splenocytes stimulated by LPS, PHA, PPD, ConA, and PWM. The follow-up study shows cytometry revealed no group alterations in the number and in the frequency of B and T cells. However, the follow-up study did show that after a 12-month exposure, there was a reduction in cell surface expression of Thy 1.1 (T-cell related) surface antigen and a reduction in the mean cell-surface density of s-IG (B-cell related) on small lymphocytes and spleen. Stimulatory effects observed in the original study were not confirmed. Evaluation of mytology, serum chemistry, protein electrophoretic patterns and fractions, and thyroxene levels revealed no significant differences between groups. Likewise, no effects of microwave exposure were observed on overall patterns of growth, food and water consumption, and body-mass loss and recovery. However, there was a highly significant elevation of adrenal mass (75% increase) observed for exposed rats as compared with sham-exposed animals, which became insignificant when the animals with benign tumors in the adrenal gland were separated from those without tumors. For the animals with tumors the adrenal mass was significantly higher in the exposed group than in the sham exposed group. Thus the analysis demonstrated that the increase in adrenal mass was related to tumors and was therefore independent of the metabolic processes in the rats. A significant decrease in O_2 consumption and CO_2 production was seen in the exposed young rats but not in the exposed mature animals. Though the survival curves indicated a lower mortality rate and longer mean survival time (668 days) for the exposed animals than for the sham-exposed (663 days), the differences were not significant. No association was found between specific cause of death and treatment conditions; however for cause of death due to urinary tract blockage (9 in the exposed group and 19 in the sham group), there was some indication that survival times were longer in the exposed animals. Histopathological studies indicated that chronic glomerulonephropathy was the most frequent cause of death and one of the most consistently encountered non-neoplastic lesions. Statistics indicated that the lesion was less frequently observed in the exposed than in the sham-exposed animals. There were no significant differences in other non-neoplastic lesions. The incidence of neoplastic lesions corresponds with that normally reported for the Sprague-Dawley rats. Due to the low incidence of neoplasia with no significant increase in specific organ or tissue, it was necessary to collapse the data and to make evaluations with respect to occurrence of neoplasms with no attention given to the site or organ of occurrence. There was no evidence of either the exposed or sham group having an excess of benign lesions, nor was there any significant difference between the total neoplastic incidence if both benign and malignant lesions were included. However, there was nearly a fourfold increase in primary malignancies in exposed animals (18 vs. 5) as compared to the sham-exposed animals. The authors state that though the overall difference in numbers of primary malignancies is statistically significant, biological significance is open to question since: 1) the detection of the difference required the collapsing of sparse data without regard for specific type of malignancy or tissue origin; 2) the incidence of the specific primary malignancies in exposed animals is comparable to specific tumor incidence reported in the literature; 3) no single type of primary malignancy was enhanced in the exposed animals; 4) the lack of any

TABLE 1. SUMMARY OF STUDIES CONCERNING LONG-TERM RADIATION EFFECTS ON ANIMALS

		EXPOSURE CONDITIONS					
Effects	Species	Frequency (MHz)	Intensity (mW/cm2)	Duration (daysxmin)	AVG. SAR (W/kg)	Peak SAR (W/kg)	References
Increase in longevity/suggestion of testicular degeneration/ pneumonia infection during exposure.	Swiss albino mice	9270 pulsed	100	295x4.5	45 (HB)*		Prausnitz and Susskind (1962)
Slight but nonstatistical increase in longevity	strain RF female mice	800	43 avg. 86 peak	175 x 120	12.9 (HB) 25.8 (HB)		Spalding et al (1971)
Increase in immunocompetency/slower development of implanted tumors but final number not affected/ > mean longevity in exposed mice with and without tumors.	CFW mice	2450 half wave 60 Hz modulation	29.2 (HB)	4 x 20 (in utero)	35		Preskorn et al (1978)
No effects on eyes as seen by slit lamp, body mass, urinary output, rectal temperature, hematocrit, hemoglobin, white cell count, platelet count and basic blood-coagulation	New Zealand white male rabbits	2450	7 - 10	180x1380	1.5 (HB)	17	Guy et al. (1980)
Decrease in albumin to globulin ratio/abnormal myeloid to erythrocyte ratio in bone marrow/no pathological changes/reduced response of spleen cells to stimulation by pokeweed but not other mitogens/ results not meant to be definitive due to low number of animals	New Zealand white male rabbits	2450	7 - 10	180x1380	1.5 (HB)	17	McRee et al. (1980)
Acceleration of appearance of spontaneous mammary cancer in females, skin cancer in males treated with 3,4-benzopyrene, and lung cancer in males injected with L_1 sarcoma /5 mW/cm^2 exposure produced same effect as cage confinment	Balb/c male mice C3H/HeA female mice	2450	5, 15	217x120	2, 6 6, 18 (HB)		Szmigielski et al.(1982)

TABLE 1. SUMMARY OF STUDIES CONCERNING LONG-TERM RADIATION EFFECTS ON ANIMALS (continued)

		EXPOSURE CONDITIONS					
Effects	Species	Frequency (MHz)	Intensity (mW/cm^2)	Duration (daysxmin)	AVG. SAR (W/kg)	Peak SAR (W/kg)	References
No effects on EEG, evoked potentials, eyes seen by slit lamp, body weight, blood parameters, histopathological parameters	young adult New Zealand male & female white rabbits	2450 cw and pulsed	1.5	92x120	0.24 (HB)	2.1	Chou et al. (1982)
No effects on body mass, blood parameters including hemotology, morpology, chemical, protein electrophoresis, lymphocyte blast transformation, mytotic index and eyes seen by slit lamp and histology/food intake was reduced by 23% during exposure which is equal to percentage of SAR to metabolic rate	New Zealand male white rabbits	2450	0.5, 5	420x65	0.075, 0.75 (HB)	.7, 7	Chou et al. (1983)
No effects on behavioral thermoregulatory function, metabolic rate, internal body temperature, thermoregulatory behavior and blood indices, effects on skin temperature and reduction of body weight as function of power density	squirrel monkeys	2450	1, 5	75x480	0.16, 0.8 0.1, 0.5 (HB)		Adair et al. (1985)
Transient changes in corticosterone levels and immunological parameters excess primary malignancies in exposed animals; but authors state, in light of other parameters in study, it is conjectural whether excess reflects a true biological influence	Sprague-Dawley male rats	2450 pulsed	0.5 avg 125 peak	758 x 1290	0.15-0.4		Chou, et al (1992)

* HB refers to estimates made from the Radiofrequency Radiation Dosimetry Handbook (Durney et al., 1986)

significant difference in benign neoplasia which, morphologically, generally precedes or is part of the process leading to malignant neoplasia; and 5) with the induction of a cancer by a carcinogen, tissue specific effects are usually induced, so that an agent is not usually considered carcinogenic unless it induces a significant response in any one tissue.

The authors conclude by indicating that the study showed no biologically significant effects on general health, serum chemistry, hematological profiles, longevity, cause of death, and lesions commonly associated with aging and benign neoplasia. Statistically significant effects in corticosterone levels and the immunological parameters 13 months exposure were not confirmed in the 25-month exposure nor the follow-up study. O_2 consumption and CO_2 production were lower in the exposed rats but the effects were not observed in the mature rats. The findings of excess primary malignancies in exposed animals is provocative but single findings considered in light of other parameters is conjectural and may not reflect a true biological influence. Positive findings need further independent experimental evaluation.

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