OVERVIEW OF THE RADIOFREQUENCY RADIATION (RFR) BIOEFFECTS DATABASE

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INTRODUCTION

For simplicity, we have used the acronym "RFR" in our database to span the nominal frequency range from 3 kHz to 300 GHz even though that range covers both the radiofrequency and microwave regions. So far, we have not included analyses of possible bioeffects of electric and magnetic fields at powerline frequencies (50-60 Hz).

Our database contains references to analyses we have done on a selection of research papers from the many thousands of accounts in the scientific literature published through about the end of 1992, plus some additional 1993 papers as they have appeared. Most of the detailed analyses are contained in Polson and Heynick,[1] a Technical Report on the proposed Office of Naval Research (ONR) and Air Force High-Frequency Active Auroral Research Program Ionospheric Research Instrument (HAARP IRI).

The papers discussed are grouped under a set of RFR-bioeffects topics. The papers were selected to provide representative coverage of each topic. Endeavors were made to describe differences in findings of the papers within each topic, and where possible, to assess the quality of the research. Most of the papers were presumed to have undergone peer review before publication.

Some of the analyses and critiques were derived from a general review of the RFR-bioeffects literature by Heynick[2] prepared by SRI International for the Human Systems Division (AFSC), U.S. Air Force School of Aerospace Medicine, San Antonio, Texas (now the Armstrong Laboratory).

We studied other general reviews of the literature on RFR bioeffects, including the report by Elder and Cahill[3] for the U. S. Environmental Protection Agency (EPA). However, the conclusions of our analyses regarding possible effects of exposure of people to RFR were reached independently.

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OVERVIEW OF THE BIOLOGICAL EFFECTS OF RFR

Much of the data on the bioeffects of RFR was derived from experiments in which various mammals and nonmammals (e.g., birds, insects, bacteria, other microorganisms) were exposed to RFR. Also investigated were tissues such as excised organs and neurons artificially kept alive (*in vitro*), blood, single cells, cultures of cells, and subcellular components. However, such results are regarded as inferential because the life processes of such animals and preparations can differ importantly from those for humans. Evidence was also obtained from various epidemiologic and occupational studies. Such studies appear to provide more direct evidence relative to possible effects on human health, but the exposure frequencies, levels, and durations are rarely known with any degree of accuracy, rendering the findings questionable. The following sections summarize the various bioeffects topics treated in more detail in the documents cited above.

Summaries of Human Studies and Related Animal Studies

Epidemiologic/Occupational Studies. Among the early epidemiologic studies done in Eastern European countries on possible detrimental effects associated with exposure to RFR were those of Pazderova,[4] Pazderova et al.,[5] Klimkova-Deutschova,[6] Kalyada et al.,[7] Sadchikova,[8] and Siekierzynski.[9] In general, mixed findings were reported, including symptomatology such as "asthenic syndrome" and "microwave sickness" that were not generally recognized or supported by subsequent epidemiologic studies in Western countries.

Among the examples of well conducted epidemiologic studies was a study by Robinette and Silverman,[10] in which the authors examined the decedence records of 19,965 Navy veterans of the Korean War classified as having had significant occupational exposure to RFR and compared them with the decedence records of 20,726 Naval men who had little occupational exposure to RFR. The data showed no statistically significant differences between exposed and control groups in the deaths from all disease, respectively 1.6% and 1.5%, both of which were significantly lower than for the age-specific general population. As another example, Lilienfeld et al.[11] had searched for possible effects of the irradiation of the U.S. Embassy in Moscow on its then resident personnel and their dependents; that study, performed by preeminent epidemiologists, who were careful to acknowledge its limitations, also yielded negative results.

Other epidemiologic studies were flawed for various reasons, such as by analysis of too-small population samples, use of mailed self-administered questionnaires to acquire the data, inappropriate statistical treatment of the data, or incorrect assembly of population data bases. This category includes studies by Hamburger et al.,[12] Lester and Moore,[13,14] Milham,[15,16] and Burr and Hoiberg.[17] The findings of such studies, whether positive or negative, is not strong evidence in either direction.

Epidemiologic studies were done that primarily sought ocular effects of RFR. In one of two studies, Cleary et al.[18] examined the records in Veterans Administration hospitals for patients with cataracts, and found only 19 of 2,644 veterans they classified by military occupation specialties (MOSs) as radar workers and 21 of 1,956 veterans classified as nonradar veterans. Thus, independent of how accurately they classified the veterans, the data yielded no evidence for RFR-cataractogenesis. Cleary and Pasternack[19] selected 736 workers employed at 16 microwave installations as occupationally exposed to RFR and 559 unexposed workers from the same locations as controls. By contrast with Cleary et al.,[18] the findings were unclear, in part because physiological aging of the lenses had occurred in both the exposed and the control groups, and the groups were not well matched in age

distribution. In addition, the authors used an arbitrary, subjective scale for grading lens changes, and that scale did not represent reductions in visual acuity.

In the third of three ophthalmologic studies, Appleton et al.[20] examined the eyes of the personnel at various Army posts where various types of electronic equipment were under development, test, or use. The authors tabulated data on three possible indicators of eye damage. On the basis of histories of microwave exposure, the pooled totals were 605 experimental and 493 control personnel. For comparisons, both groups were divided into 5 subgroups for the 10-year age spans 20-29 through 60-69 years. The findings were negative regarding RFR involvement, but were open to question because the authors used a yes or no scale to score lens damage, and they did not provide any statistical treatment of the data.

Hollows and Douglas[21] examined the lenses of 53 radiolinemen who were occupationally exposed to RFR by erecting and/or maintaining radio, television, and repeater towers throughout Australia. The RFR frequencies ranged from 558 kHz to 527 MHz. Measurements of power density in and around work areas yielded values in the range 0.08 mW/cm^2 to 4,000 mW/cm^2. The examination results were compared with those for 39 age-matched controls from the same Australian states who had never been radiolinemen. Statistically significant differences in eye changes were found between exposed and control groups, but non-RFR factors could not be ruled out, notably the reported presence in both groups of nuclear sclerosis, a type of lens opacity possibly attributable to exposure to solar irradiation.

Distinct from epidemiologic studies, there have been cases of eye damage ascribed to chronic exposure at levels well below exposure guidelines, and cases of accidental exposure to relatively high RFR levels. As an example of the former, Zaret[22] described a case of a housewife who reported in 1961 that her near vision was becoming blurred. In 1972, her ophthalmologist found extensive opacities, which were more advanced in the right eye, after which cataract extraction was done on that eye. Use of a microwave oven acquired in 1966 was the suspected cause, but measurements in 1971 of that oven by the Los Angeles County Radiological Health Division indicated a maximum leakage of 2 mW/cm^2 during operation. In 1973, Zaret,[22] an ophthalmologist, examined the patient and found that the left lens showed an advanced stage of capsular cataract. He indicated that microwave-induced cataracts are quite distinctive in appearance relative to cataracts from other causes. However, his findings regarding RFR etiology of cataracts were disputed in several letters printed with the paper.

As an example of accidental RFR exposure, Hocking et al.[23] reported on the exposure of 9 radio linemen to the RFR from an inadvertently activated open waveguide. Two of the men had been exposed to 4.6 mW/cm^2 for up to 90 minutes and comprised the "high-exposure" group; the other 7 men had been exposed to less than 0.15 mW/cm^2 and comprised the "low-exposure" group. In subsequent ophthalmic examinations, various eye abnormalities were seen in both groups, but vision in none of the subjects was affected.

Collectively, the epidemiologic studies to date have not provided any reliable evidence that chronic exposure to RFR at levels within current U.S. exposure guidelines (such as ANSI/IEEE C95.1)[24] is hazardous.

Congenital Anomalies. Sigler et al.[25] had obtained results suggestive of an association between the occurrence of Down's syndrome in children with radar exposure of the fathers during military service. However, a study by Cohen et al.[26] with a larger data base yielded negative findings, superseding those of the earlier study. Similarly, the negative findings of a study by Burdeshaw and Schaffer[27] on the incidence of birth defects from proximity to military bases superseded those of two studies by Peacock et al.[28,29]

A cohort and case-control study by Källén et al.[30] on infants born to physiotherapists presumed to have been occupationally exposed to various agents such as chemicals, drugs, X-rays, RFR yielded fewer dead or malformed infants than in the general population. The

data base for the cohort part of the study was large, thus yielding statistically credible negative findings. However, use of a questionnaire in the case-control part of the study renders questionable a finding of a weak association of malformed or perinatally dead infants with the use of shortwave equipment.

In conclusion, the studies on congenital anomalies or perinatal infant deaths have not yielded any scientifically valid evidence that such effects are caused by chronic exposure to RFR at levels below current U.S. exposure guidelines.

Ocular Effects in Animals. With the possible exception of the Kues et al.[31] study described below, all of the experiments with animals indicate that ocular damage by exposure to RFR is a gross thermal effect. Especially noteworthy are the findings of Guy and coworkers that exposure to RFR at levels that yield a temperature rise within the eye of about 5°C or more are necessary for thermal eye damage, and that no damage occurs from such RFR levels if the eye is cooled during exposure. Guy et al.[32] reported an average-power-density threshold for eye damage of roughly 150 mW/cm^2 for exposure durations of 100 minutes (or longer).

Stewart-DeHaan et al.[33] exposed excised lenses to pulsed 918-MHz RFR at specific absorption rates (SARs) in the range 10 to 1,300 W/kg. The results also comprise evidence for the thermal basis of RFR eye damage. Noteworthy is a similar study by Creighton et al.[34] with CW as well as pulsed 918-MHz RFR, because the pulsed RFR yielded almost five times greater depth of lens damage than the CW RFR under corresponding exposure conditions.

Kues et al.[31] reported increases in numbers of corneal lesions they observed by specular microscopy in the eyes of monkeys exposed to 2.45-GHz CW RFR at a SAR of 7.8 W/kg within the eye and not at lower SARs. The adequacy of the exposure technique and the use of the same monkeys in more than one aspect of the study have been questioned, as has the apparent reversibility of the corneal effect even though the primate corneal endothelium is not known to repair itself through cell division. Resolution of such points awaits further studies or replication in other laboratories.

Foster et al.[35] used 50% incidence of opacities as a threshold criterion in rabbits whose heads were exposed for 30 minutes to 2.45-GHz RFR at various input powers in a waveguide. The result was a whole-head SAR of 15.3 W/kg (for a 375-gram head).

Thus, taken collectively, the animal studies on eye damage from RFR did not yield scientific evidence that prolonged exposure to RFR at levels below the current U.S. exposure standard is likely to prove hazardous. The work of Kues et al. should be subjected to independent verification.

Auditory Effect. The RFR-auditory effect is the perception of RFR pulses by persons as apparent sound without any electronic aids. Frey and coworkers were first to study the effect in the U.S., but their hypothesis that the effect was caused by direct brain stimulation by the RFR pulses was disproved by later studies. Instead, much experimental evidence exists that RFR pulses of appropriate characteristics are transduced within the head into thermoelastic acoustic waves that propagate to the inner ear, where they are perceived as sound. Among such evidence is a study by White,[36] who demonstrated that RFR pulses can be used to generate thermoelastic acoustic waves in various media. Foster and Finch[37] confirmed White's findings in water, and proved that such waves are not generated in water at 4°C, at which its thermal expansion coefficient is zero. Olsen and Hammer[38] and Olsen and Lin[39] studied RFR-pulse transduction in spherical brain-equivalent models of the head and obtained results that support the thermoelastic theory for a homogeneous brain sphere with stress-free boundaries.

Taylor and Ashleman[40] demonstrated that the effect does not occur in cats whose cochleas are destroyed. Guy et al.[41] confirmed the latter results, as did Chou and Galambos.[42] Cain and Rissman,[43] using 3.0-GHz pulsed RFR, determined peak-power-

density thresholds in human volunteers, and obtained a peak-power-density threshold of about 300 mW/cm^2 for pulse perception. Tyazhelov et al.,[44] studied the qualities of apparent sounds perceived by humans from exposure to 800-MHz pulsed RFR, and showed that pulse perception as sound could be modulated by the concurrent reception of acoustic tones.

In summary, the preponderance of experimental results indicates that perception of RFR pulses as sound results from induction of thermoelastic waves in the head, rather than by direct brain stimulation by the RFR. Also to be noted is that because individual pulses of specific characteristics can be perceived, it is not meaningful to calculate time-averaged power densities for two or more widely spaced pulses and thereby cite such values as evidence that the effect is nonthermal in nature.

RFR Shock and Burn. It is known that RFR can cause electric shock in the body or burns in tissue under certain circumstances, and specific exposure limits have been included in the ANSI/IEEE[24] guidelines, applicable in the frequency range 0.003 to 100 MHz. Those guidelines are based on the studies by Dalziel and Mansfield,[45] Dalziel and Lee,[46] Deno,[47] Bracken,[48] Rogers,[49] Gandhi and Chatterjee,[50] Guy and Chou,[51] and Chatterjee et al.[52]

Among the considerations in deriving such limits were measurements of the currents that yielded barely perceptible sensations (perception-threshold currents), and currents that caused discomfort (let-go currents) from touching or grasping metallic structures in the vicinity of antennas radiating in the frequency range above. Also considered were the currents induced in metallic objects of various sizes and configurations by nearby antennas radiating at such frequencies, and the potential hazards to humans should they touch or otherwise come in contact with such objects.

Basically, the shock and burn sections of the ANSI/IEEE[24] guidelines for "controlled (primarily occupational) environments" specify a maximum induced current of 200 mA through both feet or 100 mA through each foot, and a maximum contact current of 100 mA, both in the frequency range 0.1-100 MHz. In the range 0.003-0.1 MHz, the induced-current limits are given by 2000 f through both feet or 1000 f through each foot, and the contact-current limit is given by 1000 f, where f is the frequency in MHz. For "uncontrolled environments" [accessible by the general public], the limits in the frequency range 0.1-100 MHz are induced currents of 90 and 45 mA respectively in both feet or each foot and a contact of current 45 mA; in the frequency range 0.003-0.1 MHz, the induced current limits are given by 900 f and 450 f, respectively, and the contact current is given by 450 f.

Studies of Nonhuman Species

Mutagenesis, Cytogenetic Effects, and Carcinogenesis in Microorganisms and Fruit Flies. Mutagenesis and carcinogenesis are considered to be related. In fact, many chemicals have been screened for carcinogenicity by testing whether they produce mutations in special mutant strains of bacteria. Similarly, mutagenic effects were sought in various plants and animals from exposure to RFR as an indication that RFR can cause or promote cancer. Various strains of yeast as well as the fruit fly are also commonly used for mutagenesis investigations.

As examples, when Blackman et al.[53] exposed cultures of *E. coli* (of a strain in which mutations can be detected readily) to either 1.7 GHz at 2 mW/cm^2 (3 W/kg) or 2.45-GHz RFR at 10 or 50 mW/cm^2 (15 or 70 W/kg) for 3 to 4 hours, they found no significant differences in genetic activity when culture temperature was held constant. Dutta et al.[54] got similar results with Salmonella cultures exposed to 2.45-GHz RFR at 20 mW/cm^2 (40 W/kg), as did Anderstam et al.[55] at 27.12 MHz and 2.45 GHz in *E. coli* or Salmonella.

Pay et al.[56] exposed male fruit flies for 45 minutes to 2.45-GHz RFR at 6 mW/cm^2 and found that subsequent matings with female fruit flies showed no significant differences between exposed and control groups in mean generation times or brood sizes. Hamnerius et al.[57] exposed fruit-fly embryos from a sex-linked, genetically unstable stock having light-yellow eyes instead of red eyes to 2.45-GHz RFR at an SAR of 100 W/kg (about 200 mW/cm^2) for 6 hours. Only 4 mutations in 7,512 exposed males (0.05%) and 2 mutations in 3,344 control males (0.06%) were seen, a nonsignificant difference. The authors also exposed fly embryos to X-rays as a positive control, and found that 1,000 rad yielded 29 mutations in 1,053 males (2.75%). They also noted that the chemical mutagen EMS yielded 444 mutations in 4,859 males (9.14%).

Carcinogenesis in Mammals and Mammalian Tissues. In a study by Prausnitz and Susskind,[58] 200 mice were exposed to 9.3-GHz pulsed RFR at an average power density of 100 mW/cm^2 [SAR about 45 W/kg] for 4.5 minutes per day, 5 days per week, for 59 weeks. The authors reported that some mice had developed leukosis, which they described as a "cancer of the white blood cells," and that leukosis incidence was higher in the exposed mice than the control mice. The effect was undoubtedly real, but its interpretation by the authors was probably faulty. In dictionaries of medicine and pathology, leukosis (also spelled leucosis) is defined as an abnormal rise in the number of circulating white blood cells, and is not regarded as a form of cancer. Various factors can give rise to leukosis, including stress, disturbances of the endocrine system, and infection. The observed liver abscesses may have been due to pneumonia in the mouse colony.

Roberts and Michaelson,[59] in reanalyzing the data of Prausnitz and Susskind,[58] with appropriate statistical treatment, found that the results do not support a link between exposure to RFR and cancer development. They also remarked that the greater longevity of the RFR-exposed mice could be taken equally plausibly as indicating that the RFR was beneficial.

Skidmore and Baum[60] exposed five pregnant rats during 17 days of gestation in a simulator of "EMP" (electromagnetic pulses resembling the RFR from a nuclear blast), with five unexposed pregnant rats as controls. The peak electric field was 447 kV/m. Following exposure, no gross abnormalities were found in the fetuses. Twenty female rats were exposed to the EMP for 38 weeks and were observed for possible development of mammary tumors, together with 20 controls. At age one year, no mammary tumors were found.

They also exposed 50 male mice of a strain known to be susceptible to spontaneous leukemia development between 6 and 12 months of age. After exposure for 33 weeks, 42 (84%) of the EMP-exposed mice and 24 (48%) of the unexposed control mice survived. Not clear is why a much higher percentage of the exposed mice survived than the control mice, a possible indication that uncontrolled non-RFR factors may have been present. Histologic examinations showed that 9 of the 42 exposed survivors (21%) and 11 of the 24 control survivors (46%) had developed leukemia. However, the sample sizes were too small to ascribe any statistical validity to that difference in percentages.

Szmigielski et al.[61] investigated whether RFR exposure: decreases the natural resistance of one mouse strain to lung cancer cells intravenously injected before exposure; increases the incidence of breast tumors in female mice of a strain known to have high spontaneous incidence of such tumors; and increases the incidence of skin cancer in mice of the first strain that were locally depilated and painted with the chemical carcinogen 3,4-benzopyrene (BP). The exposures were for 2 hours a day, 6 days a week, for up to 6 months to 2.45-GHz RFR at 5 mW/cm^2 (SAR 2-3 W/kg) or 15 mW/cm^2 (6-8 W/kg). Other mice were similarly sham exposed, and still others were raised under stress-inducing confinement.

In the lung cancer study, RFR-exposure at 15 mW/cm^2 for 3 months yielded a significantly larger mean number of neoplastic nodules (colonies originating from single cells) than exposure at 5 mW/cm^2. The mean number of nodules in those raised under confinement for 3 months was comparable to that for those exposed at 5 mW/cm^2, a possible indication that confinement stress alone may be carcinogenic and that the larger mean number of nodules in those exposed at 15 mW/cm^2 may have been due to the heat stress from the higher RFR level.

In the breast-cancer investigation, the cumulative numbers of mice with discernible tumors and their survival times were tabulated. By regression analysis, the results were summarized in terms of the mean cancer development time in 50% of the mice and the mean survival time of 50% of the mice. The mean cancer-development and survival times of those exposed at 5 mW/cm^2 were comparable to the respective means for confinement-stressed mice, and both were shorter than the corresponding values for sham exposure and longer than the corresponding times for 15 mW/cm^2.

In the skin-cancer experiments, cancer development from use of BP was determined by histopathologic examination and scored on a subjective 7-grade scale from 0 to 6. At score 4, small papillomas were found microscopically to contain cancer cells, so mice with scores of 4-6 were regarded as having skin cancer. Skin cancer occurred within 7-10 months in more than 85% of those treated with BP. As in the breast-tumor study, the numbers of mice affected by exposure at 5 mW/cm^2 or confinement stress were comparable. The mean cancer development times in 50% of those sham-exposed, confinement-stressed, exposed at 5 mW/cm^2, or exposed at 15 mW/cm^2 for 3 months before BP treatment were respectively 272, 201, 171, and 171 days. Not understandable is the lack of difference between the values for 5 and 15 mW/cm^2.

McRee et al.[62] found no significant effects of exposure of mice to 2.45-GHz RFR 8 hours per day for 28 days at 20 mW/cm^2 (about 27 W/kg) on the induction of sister chromatid exchanges, a sensitive technique for assaying genetic damage from mutagens and carcinogens, or on the rate of proliferation of bone-marrow cells.

Meltz et al.[63] investigated whether pulsed 2.45-GHz RFR alone can induce mutagenesis, chromosal aberrations, and sister chromatid exchanges in mammalian cells, and whether the RFR can alter the genotoxic damage induced by the chemical mutagen proflavin when the RFR is administered simultaneously with the mutagen. The authors found that exposure of cell cultures derived from a mouse leukemic cell line to the pulsed RFR at about 40 W/kg, either alone or in combination with the mutagen yielded negative findings: The RFR-mutagen combination produced no statistically significant increase in induced mutant frequency relative to the results for treatment with the mutagen alone. Moreover, RFR exposure alone yielded no evidence of mutagenic action.

In a comprehensive University of Washington study of chronic exposure,[64-72] 100 male rats were exposed within individual cylindrical waveguides to 2.45-GHz RFR at an average power density of about 0.5 mW/cm^2 under controlled-environmental and specific-pathogen-free conditions, a level selected to simulate, by scaling considerations, chronic exposure of humans to 450-MHz RFR at an SAR of about 0.4 W/kg (the basis of the 1982 ANSI guidelines[73]). The controls were 100 sham-exposed male rats. Both groups were concurrently treated in the same facility for 25 months (virtually their entire lifetimes), except for those withdrawn for interim tests and those that expired before the end of the exposure regimen. After 13 months, 10 each of the RFR-exposed and sham-exposed rats were euthanized (the interim kill), as were 10 of the 12 RFR-exposed and 10 of the 11 sham-exposed rats that survived to the end of the exposure regimen.

No primary malignancies were found at the interim kill. However, the most controversial finding was that among those older than one year, primary malignant lesions (of various kinds) were found in a total of 18 of the RFR-exposed rats but only 5 of the

sham-exposed rats. Among the arguments given to discount the importance of this finding were that the numbers of rats that exhibited each specific type of malignancy were similar to those reported in the literature for untreated rats of the same strain, and that the differences in the numbers for each malignancy were statistically nonsignificant. Thus, apparent statistical significance could be attained only by combining those numbers, an oncologically dubious procedure.

Santini et al.[74] investigated whether B16 melanoma would develop in black mice and whether their survival times would be affected by exposure to RFR. They exposed one group of 15 mice to 2.45-GHz CW RFR at 1 mW/cm^2 (SAR 1.2 W/kg) for 6 daily sessions per week, each 2.5 hours a day, until death (up to 690 hours total). They similarly exposed another group to 2.45-GHz pulsed RFR at the same average power density. A third group was sham-exposed. No statistically significant differences were found among the three groups in either tumor development or survival.

Balcer-Kubiczek and Harrison[75] investigated whether exposure to RFR of mouse-embryo-fibroblast-cell cultures induces malignant transformation in such cells. They exposed cultures for 24 hours to 2.45-GHz RFR alone at 0.1, 1, or 4.4 W/kg or to the RFR at 4.4 W/kg before or after exposure to X-rays (as a tumor initiator) at 0.5, 1, or 1.5 Gy. Control cultures were sham-exposed. They then incubated the cultures with or without a known tumor promoter (TPA), and assayed them for incidence of neoplastic transformations by counting the transformed foci in each culture dish.

The results for RFR alone or TPA alone (at the dose used) showed no evidence of tumor promotion. However, the mean counts of transformed foci rose with SAR for the RFR-exposed cultures incubated with TPA. Those results were interpreted by the authors as indicating that RFR acts synergistically in a dose-dependent manner with TPA to promote (rather than initiate) neoplastic transformation.

Several points in the paper are open to question. First, a plot of mean neoplastic transformation incidence versus SAR indicated an apparently linear incidence rise with SAR (0.1, 1.0, 4.4 W/kg). That result can be misleading because, unlike what was done for the plots of incidence versus X-ray dose, in which linear scales were used for both variables, the authors used a linear scale for incidence and an exponential scale for SAR. If the three SAR points had been plotted on a linear scale also, the graph would have displayed a much sharper rise with SAR between 0.1 and 1.0 W/kg than between 1.0 and 4.4 W/kg. Specifically in such a plot, the slope of the line connecting the points for 0.1 and 1.0 W/kg is more than five times larger than for the line connecting the points for 1.0 to 4.4 W/kg. Thus, it is difficult to interpret the RFR dose-response relationship.

Second, use of a well characterized carcinogen that causes chromosomal damage as the tumor initiator such as benzopyrene, rather than X-rays, might have yielded more definitive results. Third, the numbers of foci relative to the numbers of dishes treated were small and the numbers of dishes used for each treatment differed considerably. This point raises the question as to whether the authors had augmented the number of dishes for each treatment until they obtained adequate percentages of foci for statistical significance. Fourth, not mentioned in the paper was whether those who counted the foci had prior knowledge of the treatment of each dish. Foreknowledge of the treatment can yield false positives and should be controlled for by use of "blind" procedures.

Nevertheless, in view of the importance of the reported findings, the study should be replicated, perhaps with a more standard chemical initiator than X-rays, and with a "blind" foci counting procedure to avoid possible subconscious bias.

The EPA issued a draft report[76] in which a potential link between cancer incidence and exposure to electromagnetic fields was indicated. Although the emphasis was on powerline fields, exposure to microwave fields was also included. However, EPA's Radiation Advisory Committee of the Science Advisory Board (SAB) had set up a subcommittee on

Nonionizing Electric and Magnetic Fields to review the draft report, and that subcommittee has issued a report, SAB,[77] in which the subcommittee suggested numerous changes in emphasis, coverage, and wording. It concluded that the draft report[76] will have to be rewritten to accommodate all of the suggestions and comments.

In summary, although questions have been raised regarding interpretation of the findings of the University of Washington[64-72] and the Balcer-Kubiczek and Harrison[75] studies, taken collectively the studies conducted to date provide no scientifically credible evidence that exposure to low RFR within ANSI/IEEE guidelines levels causes mutations or cytogenetic effects, or that such RFR induces or promotes any form of cancer in mammals or nonmammals. In addition, little credence can be given to the EPA[76] draft report, pending issuance of a revision thereof.

Teratogenesis. Rugh et al.[78,79] exposed pregnant mice to 2.45-GHz RFR at 123 mW/cm^2 (SAR 110 W/kg) for 2 to 5 minutes on what they describe as the gestation day of highest sensitivity to ionizing radiation. They stated that they could not find any teratogenesis threshold. However, a reanalysis of their data indicated the existence of a threshold: At mean total doses less than about 3 cal/g or power densities less than about 1 mW/cm^2, 100% of the fetuses examined were normal and significant numbers of abnormal fetuses were obtained at RFR levels above that threshold, but the dependence on dose was obscure.

Chernovetz et al.[80] found that absorption of about 5 cal/g of 2.45-GHz RFR is not teratogenic to mice, a threshold considerably higher than the 3-cal/g value above. They also found that the mean total lethality dose of the dams was about 5.7 cal/g, indicating that teratogenesis would occur in pregnant mice only at levels that are close to lethality for the dams.

Stavinoha et al.[81] exposed 4-day-old mice for 20 minutes to 10.5-MHz, 19.27-MHz, or 26.6-MHz RFR pulses at an electric field strength of 5.8 kV/m and weighed them daily up to age 21 days, with sham-exposed mice as controls. There were essentially no differences in post-exposure weights at corresponding ages. Berman et al.[82,83] did find consistently smaller mean body weights of live mouse fetuses from dams exposed to 2.45-GHz RFR at 28.0 mW/cm^2 (22.2 W/kg), but other researchers either could not confirm such findings or found that growth retardation was thermally induced. However, it is difficult to reconcile those growth retardation results of Berman et al.[82,83] in the mouse with the findings by Berman et al.[84] and Smialowicz et al.[85] of no retardation in rats at RFR levels capable of heating pregnant females to temperatures over 40°C. Clearly, studies of nonhuman primates would be much more definitive.

Lary et al.[86] observed teratogenic effects in rats exposed to 27.12-MHz fields at 55 A/m and 300 V/m (SAR about 11 W/kg), but of severity that increased with colonic temperature. The largest changes were seen for prolonged exposure to maintain colonic temperature at 42.0°C. The authors ascribed those effects to the hyperthermia induced by the RFR. A subsequent study by Lary et al.[87] with the same RFR indicated the existence of a colonic temperature threshold of 41.5°C for birth defects and prenatal death.

Tofani et al.[88] reported teratogenic effects in rats exposed to 27.12-MHz RFR at field strengths of 20 V/m and 0.05 A/m (equivalent power density 0.1 mW/cm^2; author-estimated SAR about 0.00011 W/kg). They had characterized the effects as nonthermal and due to long-term exposure.

Lu and Michaelson[89] took issue with the exposure methodology used. They questioned the absence of technical details, such as a description of the means for providing food and water and for removing wastes during continuous exposures of pregnant rats. They also questioned the reality of the positive findings at an average SAR far lower than the threshold for RFR bioeffects (3-4 W/kg) that was the basis for the ANSI[73] exposure guidelines. They also noted that the use, by Tofani et al.,[88] of Durney et al.[90] for their SAR

estimation was inappropriate because that reference applied to spheroidal models in the far field, whereas the exposures in this study were in the near field. Last, as noted by Lu and Michaelson,[89] not discussed in the paper was whether RFR-absorbent materials were used in the exposure chamber to avoid multipath exposure, and the likelihood that the proximity of the rats to one another in the exposure boxes (spacings less than 1 wavelength) resulted in large dosimetric variations and uncertainties.

In their response, Tofani et al.[91] clarified several of the points raised by Lu and Michaelson,[89] but remarked that they chose to do the exposures in a room without any shielding or RFR-absorbing materials rather than in a transverse electromagnetic (TEM) cell within a shielded anechoic chamber available at their laboratory. As justification, they stated: "Our aim in this work is the evaluation of the biological effects due to a low-level, long-term exposure to a 27.12-MHz electromagnetic field in conditions as similar as possible to those people who usually are exposed (i.e., to near-field, multi-path radiation with distances between individuals shorter than a wavelength)." That response appears to beg the question, because the dose rates from such exposures could have varied considerably from rat to rat and with time for each rat.

Tofani et al.[91] also remarked: "Effects due to overcrowding ought to result in the sham-exposed group too, since that group was managed in the same way." That remark appears to miss the point that overcrowding probably introduced large spatial and temporal variations in RFR-exposure levels rather than directly causing the reported effects, so overcrowding *per se* in the sham-exposed rats would not be expected yield those effects. In summary of the foregoing, little credence can be given to the conclusion of Tofani et al.[91] that the effects they observed were nonthermal.

In a behavioral study of squirrel monkeys by Kaplan et al.,[92] an excess of unexpected infant deaths was found. However, that finding was not confirmed in a subsequent study involving enough monkeys for an adequate statistical treatment of the results.

Taken collectively, the studies above indicated that teratogenic effects can occur in both nonmammalian and mammalian subjects from RFR exposure, but only at levels that produce significant internal temperature rises. The results for mammals show that increases in maternal body temperature that exceed specific thresholds (41.5°C in rats) are necessary for causing teratogenic effects.

Nervous System. *Blood-Brain-Barrier Effects.* Early studies by Frey et al.[93] and Oscar and Hawkins[94] of RFR effects on the blood-brain barrier (BBB) were subsequently shown likely to have been caused by artifact in the methods used in their experiments. Also, Merritt et al.[95] and Ward and Ali[96] were unable to reproduce those findings, and Preston et al.[97] showed that certain specific RFR-induced changes in the brain could be interpreted wrongly as BBB alterations. Using a newer method by Rapoport et al.,[98] Preston and Préfontaine[99] and Gruenau et al.[100] found no evidence for RFR-induced alterations of the BBB. Four comprehensive studies by Williams et al.[101,102,103,104] with conscious unrestrained rats, in which several different tracers and methods were used for detecting BBB penetration, also yielded negative findings. Neilly and Lin[105] demonstrated that disruption of the rat BBB at high RFR levels is due to elevation of brain temperature. In addition, they found that high ethanol doses inhibit BBB disruption by moderating the increases in brain temperature produced by the RFR. In summary, the preponderance of negative experimental findings indicates that exposure to RFR at low levels has no effect on the permeability of the human BBB.

Histopathology and Histochemistry of the Central Nervous System. Albert et al.[106] had reported lower mean counts of Purkinje cells in 40-day-old rat pups exposed for 5 days *in utero* to 2.45-GHz RFR at 10 mW/cm^2 relative to counts for sham-exposed pups. The results are difficult to interpret in view of the large SAR variations (0.8 to 6 W/kg, 2 W/kg estimated mean) due to movement of the dams during exposure. Also, the findings of an

experiment in which rat pups were exposed to the RFR and euthanized 40 days later did not show such differences. In addition, little credence can be given to results for pups euthanized right after exposure because Purkinje cells are immature in neonates. Albert et al.[107] did a similar study on squirrel monkeys previously exposed perinatally; no significant differences in Purkinje cell counts were seen between RFR-exposed and sham-exposed groups.

Merritt et al.[108] exposed 10 pregnant unrestrained rats to pulsed 2.45-GHz RFR for 24 hours per day during gestation days 2-18. The RFR level was held constant at 0.4 W/kg for each dam as its mass increased during the exposure period. Concurrently, 10 rats were sham-exposed. All 20 rats were weighed on day 2, and were reweighed every fourth day during treatment. On day 18, they were euthanized and the fetuses were removed and examined. After each fetus was weighed, its brain was excised, weighed, and assayed for RNA, DNA, and protein. The last three endpoints were expressed in terms of both mg/brain and μg/mg of brain tissue (thus comprising a total of 8 endpoints). The difference between groups for each endpoint was nonsignificant ($p>0.05$). Also, no RFR-exposed litter was microencephalous.

Sanders et al.[109] used continuous fluorescence measurements *in vivo* to investigate whether RFR exposure of rat brain tissue stresses cells that inhibit respiratory chain function, thereby decreasing the concentrations of adenosine triphosphate (ATP) and creatine phosphate (CP). Such measurements were made through an aperture in the skull during exposure to 591-MHz RFR at 5 or 13.8 mW/cm^2 for durations of 0.5 to 5 minutes. The results indicated that such reductions in concentration by RFR occurred at levels characterized as not producing measurable brain hyperthermia (but with local hyperthermia not ruled out). Although some points are open to question, the positive findings appear to be valid, and are worthy of further study. Also, the experiments were performed on anesthetized rats, with consequent lowering of their brain temperatures; whether similar results would occur without anesthesia has not been determined.

Sanders et al.[110] performed similar experiments, but at 200 MHz and 2,450 MHz as well as 591 MHz. Noteworthy is that the effects were higher at 591 MHz than at 200 MHz but were not observed at 2.45 GHz, suggesting that the effect occurs only within a specific frequency range below 2.45 GHz.

Lai et al.[111] studied the effects of single, 45-minute exposures of rats to 2.45-GHz CW or pulsed RFR on choline uptake in several regions of the brain, the uptake being a measure of cholinergic activity. The exposures were done in two types of chambers (cylindrical waveguides and a miniature anechoic exposure chamber), and both were adjusted to obtain a whole-body SAR of 0.6 W/kg. Control rats were sham-exposed in the same chambers. Both positive and negative results were observed, but not clear were apparent inconsistencies in results for the two kinds of exposure chamber and between pulsed and CW RFR.

In summary, histopathologic and histochemical changes in the central nervous system were seen at relatively low SARs by Sanders et al.[109,110] and Lai et al.,[111] but their significance with regard to possible human health is not clear, and questions about those studies remain open. Overall, considering other experimental results in which effects were ascribed to brain temperature increases, it seems unlikely that subthermal levels would cause such changes in the human central nervous system.

Changes in Electroencephalograms (EEGS) and Evoked Responses (ERS). Various investigations have been done to ascertain the effects of RFR on the EEG or on the responses evoked by visual or auditory stimuli (ERs). As proved by Johnson and Guy,[112] the use of indwelling metallic electrodes, wires, or screws may be questioned as a procedure likely to induce artifactual effects in the animals under study as well as in the recordings themselves.

Thus, findings of early studies may be discounted because of such use. Also, EEG measurements done after and/or before RFR-exposure may present problems stemming from the time consumed in attaching the electrodes and variability in their placement. With such usage, moreover, any transient effects that may occur during exposure would not be detected.

Takashima et al.[113] sought changes in EEGs of rabbits exposed once (acute exposure) to frequencies in the range 1-30 MHz at fields of 0.5 to 1 kV/m, done between two spaced aluminum plates. They endeavored to minimize recording artifacts by using a preamplifier having a pass band of 3-100 Hz and a 60-Hz notch filter. In addition, they sampled, digitized, and converted time-domain EEG recordings taken before and after RFR-exposure into complex frequency spectra by fast-fourier-transform (FFT) and thence into power spectra. Typical pre-exposure power spectra from anesthetized rabbits showed frequency components between 5 and 15 Hz that varied during each sequence, indicating the absence of a dominant component, EEGs denoted "normal."

In one set of experiments, anesthetized rabbits in which stainless-steel electrodes had been implanted within the brain were exposed once to fields modulated at 60 Hz. Specific changes were seen in the EEGs recorded during exposure. In another set of experiments however, in which the EEG electrodes were removed before exposure and reinserted after exposure, the power spectra observed resembled normal EEGs as defined above. The authors attributed the previous EEG changes to the local field created by those metal electrodes.

Those authors also sought possible effects of chronic exposure: They exposed unanesthetized rabbits 2 hours/day for 6 weeks to 1.2-MHz fields modulated at 15 Hz, and recorded EEGs every 2 weeks with silver electrodes placed directly on the skull before and after exposure. Abnormal EEG patterns began to appear after 2 to 3 weeks of exposure. The authors constructed histograms from power-spectrum sequences derived from 4-week exposures and normal EEGs. The histogram for the exposed animals showed major peaks at 2 and 10 Hz, but the major peaks for "normal" (pre-exposure) EEGs were at 4.5, 8, and 11.5 Hz.

In this study, the rabbits were used as their own control group, in that the data taken during and after exposure were compared with the "normal" (pre-exposure) data from the same group. However, the lack of a similarly treated sham-exposed group renders it difficult to assess whether the reported EEG changes were the result of exposure *per se,* or of adaptation to the repetitive aspects of the experimental procedures, such as handling and recording. Also, techniques other than the FFT may be more appropriate for analysis of power spectra of EEGs recorded at short time intervals (3 minutes). Qualitatively, nevertheless, the chronic-exposure data appear to show an enhancement of low-frequency power-spectral components and reduction of high-frequency activity. On the other hand, comparison of data from the anesthetized animals used in the acute experiments with data from the unanesthetized animals used in the chronic experiments lacks analysis of the effects of anesthesia as a possible confounding factor.

In several studies, such as by Tyazhelov et al.[114] and Chou and Guy,[115] endeavors were made to minimize artifacts by designing electrodes and leads from materials having high resistivities comparable to those for tissue. When such electrodes were implanted before exposure and were present during exposure, no significant differences in EEGs or ERs between control and RFR-exposed animals were seen, as exemplified by the study of Chou et al.[116] In summary, with appropriate measures taken to avoid artifacts, there is no credible scientific evidence that low-level RFR affects the EEG.

Calcium Efflux. In studies by Bawin et al.,[117] calcium efflux was reported to occur in brain hemispheres of newly-hatched chicks that were excised and exposed to 147-MHz RFR amplitude-modulated at frequencies below about 100 Hz. One of the chick

hemispheres was exposed between a pair of plane-parallel plates, with the other hemispheres serving as the controls. The effect was absent with unmodulated RFR at the same frequencies; it was highest for modulation with 16 Hz. Sheppard et al.[118] obtained similar results with amplitude-modulated 450-MHz RFR. Other results by Bawin and coworkers for chick brains poisoned with cyanide indicated that calcium efflux is not an effect that involves calcium transport across cell membranes.

Bawin and Adey[119,120] also performed experiments indicating that the effect was ascribable to the modulation itself rather than to the RFR carrier frequency. In those experiments, they exposed chick-brain preparations to sinusoidal fields at discrete frequencies of 1, 6, 16, or 32 Hz instead of to RFR fields amplitude-modulated at those frequencies. However, the effects at the sub-ELF and ELF frequencies were opposite in direction to those for the modulated RFR.

Blackman et al.[121] performed experiments toward reproducing the 147-MHz chick-brain results of Bawin et al.,[117] but used a TEM line for exposure instead of parallel plates. Two series of exposures were done. In one, power density was held constant at 0.75 mW/cm^2 and the modulation frequencies were 0, 3, 9, 16, and 30 Hz. In the other, the modulation frequency was held constant at 16 Hz and the power densities were 0, 0.5, 0.75, 1.0, 1.5, and 2.0 mW/cm^2. The results for the constant power density series were similar to those of Bawin et al.,[117] with maximum effect at 16 Hz. Regarding the 16-Hz modulation series, the authors stated:

"Our results indicate that the modulation-frequency window in which calcium-ion flux is enhanced only occurs within a restricted range of power densities...These results indicate a maximal power-density effect at 0.75 mW/cm^2 and no enhancement at levels plus or minus 0.25 mW/cm^2 of this value."

Shelton and Merritt[122] investigated whether RFR pulses at repetition rates comparable to the modulation frequencies used in the chick-brain studies would elicit alterations in calcium efflux from the rat brain. They excised brain hemispheres from rats and exposed the hemispheres for 20 minutes to 20-ms pulses of 1-GHz RFR modulated at 16 pps (pulse per second), at average power densities of 0.5, 1.0, 2.0, or 15 mW/cm^2 selected to search for the reported power-density window. In other experiments, the exposures were to 10-ms pulses, 32 pps, at 1.0 or 2.0 mW/cm^2. The brain hemispheres were then assayed for calcium efflux. Their findings were negative, but as they pointed out, no direct comparisons can be made between their results and those of investigators on chick brains because besides the differences in species and exposure durations, there were important differences between pulse modulation at 16 pps and sinusoidal modulation at 16 Hz.

In a subsequent study, Merritt et al.[123] performed experiments *in vivo* as well as *in vitro* on possible pulsed-RFR-induced alterations of calcium efflux from the rat brain. For both types of experiments, brain tissue was loaded with $^{45}Ca^{++}$ by injection directly into the right lateral ventricle of rats anesthetized with ether. In the *in vitro* experiments, samples of excised brain tissue were exposed for 20 minutes to 20-ms pulses, 16 pps, of 1-GHz RFR at 1 or 10 mW/cm^2 (0.29 or 2.9 W/kg) or 2.45-GHz RFR at 1 mW/cm^2 (0.3 W/kg). Radioactivities of the incubation media and the tissue samples were assayed in terms of mean disintegrations per minute per gram of tissue (DPM/g). The difference in mean DPM/g values between RFR-exposed and sham-exposed samples was not significant for any of the exposure conditions.

In the whole-animal exposures, 2 hours after the rats were injected with $^{45}Ca^{++}$, groups were exposed for 20 minutes to 2.06-GHz RFR with their long axes parallel to the E-field, one group each to CW at 0.5, 1.0, 5.0, or 10.0 mW/cm^2 and one group each to 10-ms pulses at 8, 16, or 32 pps at an average power density of 0.5, 1.0, 5.0, or 10.0 mW/cm^2. The normalized SAR was 0.24 W/kg per mW/cm^2. The rats were euthanized after exposure, and their brains were removed and processed appropriately for $^{45}Ca^{++}$ assays.

Statistical tests on the 17 treatment combinations (4x4 RFR-exposures, 1 sham-exposure) showed that the difference between the results for the sham group and the combined RFR groups, and the differences between the results for the sham group and each of the RFR groups were not significant.

Adey et al.[124] exposed paralyzed awake cats for 60 minutes to 450-MHz RFR, amplitude-modulated at 16 Hz, after incubating the brain cortex of each cat with $^{45}Ca^{++}$ through a hole in the skull. The average power density was 3.0 mW/cm^2 (SAR about 0.29 W/kg in the interhemispheric fissure). The authors used the experimental data for curve fitting, but did not present any actual data. They stated that there was no difference between the means of the exposed and control values, but that the standard deviations were greater for all of the exposed data points than for the controls. It is difficult to interpret these second-order statistical findings as indicating the existence of the effect.

Blackman et al.,[125] noted that the currents in the walls of the TEM line yielded an alternating magnetic component in addition to the TEM field, in contrast to the solely electric field produced between the parallel plates used by Bawin and coworkers, which led them to hypothesize that the magnetic component could influence changes in calcium efflux and to suggest that the earth's DC magnetic field might also be involved in the effect. Therefore, they did exposures in a transmission line that permitted use of an alternating electric field either alone or together with an alternating magnetic field. They also placed the transmission line within a pair of large Helmholtz coils oriented to produce a DC magnetic field parallel to the earth's field, that could be varied to alter the local magnitude and polarity of that field.

Mixed results were obtained, from which it was difficult to relate the occurrence of calcium efflux or its absence to changes in local geomagnetic field. More recently, Blackman et al.[126] reported that calcium efflux can be reduced, enhanced, or nullified by appropriately varying the temperature of the chick-brain samples before and during exposure. The authors interpreted these results as indicating the existence of a temperature window analogous to the RFR intensity window, but which may also be interpreted as an unrecognized artifact in this and previous studies.

In summary, although researchers in several laboratories have reported obtaining the calcium-efflux effect, researchers in other laboratories have been unable to confirm its existence. Several of the recent studies that report positive findings suggest that magnetic fields, primarily at powerline frequencies as well as the earth's DC field, could contribute significantly to the effect, with ion cyclotron resonance as the primary mechanism. However, quantitative analyses by others indicate that it is most unlikely that ion cyclotron resonance could occur under the proposed conditions of the cell membrane. In any case, there is no experimental evidence that the effect, if it does exist, would occur in or be harmful to humans or intact animals.

Immunology and Hematology. *Studies in Vitro.* Smialowicz[127] compared suspensions of mouse-spleen cells exposed to 2.45-GHz RFR at 10 mW/cm^2 (19 W/kg) for up to 4 hours with suspensions held at 37°C for the same durations as controls. After treatment, suspensions were cultured with or without each of four different mitogens. No significant differences in percentages of cells undergoing mitosis or in percentages of viable cells were found between RFR-exposed and control specimens treated for corresponding durations. Hamrick and Fox[128] also obtained negative results for rat lymphocytes cultured with or without the mitogen phytohemagglutinen (PHA) and exposed to 2.45-GHz RFR at up to 20 mW/cm^2 (2.8 W/kg) for up to 44 hours.

Roberts et al.[129] found that the viability of cultures of human mononuclear leukocytes was unaffected by exposure to 2.45-GHz RFR for 2 hours at SARs in the range 0.5 to 4 W/kg. There were also no significant effects on DNA, RNA, and total protein synthesis, or in assays for spontaneous production of interferon, influenza-virus-induced production of

alpha-interferon, and mitogen-induced production of gamma-interferon. Subsequently, Roberts et al.[130] infected human mononuclear leukocyte cultures with influenza virus and then exposed them to 2.45-GHz RFR, either CW or pulsed at 60 or 16 Hz, all at 4 W/kg. No significant differences were found in leukocyte viability between RFR-exposed and sham-exposed virus-infected or uninfected cultures, or in DNA synthesis from mitogen stimulation.

Lyle et al.[131] sought effects for 60-Hz-amplitude-modulated 450-MHz RFR at 1.5 mW/cm^2 (SAR not stated) on the effectiveness of a specific class of rodent T-lymphocytes (effector cells) against a specific class of lymphoma (target) cells. Mixtures of T-lymphocytes and lymphoma cells exposed to the RFR showed reductions of 17-24% in mean effectiveness of T-lymphocytes against lymphoma cells relative to unexposed mixtures. Similar percentages (15-25%) were obtained in mixtures not exposed to the RFR but in which T-lymphocytes were exposed to RFR before mixing them with lymphoma cells, thus indicating that the effect of the RFR was on the T-lymphocytes. However, the effect on the T-lymphocytes decreased with elapsed time after exposure.

Effects were also sought for 450-MHz RFR modulated at 3, 16, 40, 80, and 100 Hz. The reduction of effectiveness was negligible with unmodulated RFR; nonsignificant with 3, 16, and 40 Hz; maximal with 60 Hz; and significant (but smaller) with 80 and 100 Hz, an indication that the effect was due to the amplitude modulation itself.

Sultan et al.[132] studied the effects of combined RFR-exposure and hyperthermia on the effectiveness of normal mouse B-lymphocytes against a specific antigen (anti-mouse immunoglobulin from the goat). They exposed suspensions of normal mouse B-lymphocytes at 37, 41, and 42.5°C to 2.45-GHz CW RFR for 30 minutes at levels in the range 5-100 mW/cm^2 (2.25-45 W/kg). Control suspensions at each temperature were sham-exposed. The effectiveness of B-lymphocytes against the antigen was more than 90% for cells heat-treated at 37°C, but less than 60% of those treated at 41°C, and less than 5% of those treated at 42.5°C. The authors concluded that the mechanisms involved are thermally based, with no apparent effects of the RFR *per se* if RFR-exposed and control samples are held at the same temperature.

Sultan et al.[133] reported similar negative findings with cell suspensions exposed for 30 minutes to 147-MHz RFR amplitude-modulated at 9, 16, or 60 Hz at average power densities from 0.1 to 48 mW/cm^2 (0.004-2.0 W/kg). Thus, their results showed no amplitude-modulation effect. They also found that for temperatures not exceeding 42°C, the effectiveness of mouse B-lymphocytes returned to normal 2 hours after heat treatment.

Cleary et al.[134] obtained negative findings on the viability and phagocytotic ability of rabbit neutrophils exposed to 100-MHz CW RFR for 30 or 60 minutes at electric field strengths ranging from 250 to 410 V/m (120 to 341 W/kg), or for 60 minutes to 100-MHz RFR amplitude-modulated at 20 Hz (331 W/kg). However, the credibility of these findings is diminished by the relatively large variabilities among the control groups in each case, an indication of the possible presence of uncontrolled non-RFR factors.

Kiel et al.[135] sought effects of RFR on oxidative metabolism of human peripheral mononuclear leukocytes. They noted that chemiluminescence (CL) occurs in the production of oxygen metabolites and that CL can be enhanced and used as a sensitive detector of such effects. They collected blood samples from human volunteers, and separated, washed, and resuspended the leukocytes. Samples were paired, and one of each pair was exposed for 30 minutes to 2.45-GHz CW RFR at 104 W/kg with the sample held at 37°C and the other was held at 37°C in an incubator without RFR exposure. Following treatment, half of each sample was used for measuring CL activity, and the other half for determining cell counts and viability. The differences between the RFR-exposed and sham-exposed samples were not significant.

In studies of possible effects of RFR interactions with samples of red blood cells (RBCs) taken from animals or humans, alterations of cell membrane function, particularly any effects on the movement of sodium ions (Na^+) and potassium ions (K^+) across the membrane were sought. In early studies done in Eastern Europe, increases in cell breakdown and efflux of K^+ from rabbit red blood cells (RBCs) were reported for exposure to 1-GHz or 3-GHz RFR at levels as low as 1 mW/cm^2.

Peterson et al.[136] found that heating suspensions of rabbit RBCs with 2.45-GHz RFR at 10-140 mW/cm^2 (46-644 W/kg) yielded higher hemoglobin (Hb) or K^+ losses than conventionally heating suspensions. However, in all experiments in which RFR-heated and conventionally-heated RBCs were warmed at the same rate to the same final temperature, both Hb and K^+ were lost in equal amounts, indicating the thermal basis for the effect. However, the authors also used 2.45-GHz RFR to heat samples of human RBCs to 37°C and maintain them there, and found no significant differences in either hemolysis or K^+ release relative to conventionally heated samples.

Brown and Marshall[137] sought nonthermal effects of RFR on growth and differentiation of the murine erythroleukemic (MEL) cell line. They exposed MEL cells cultured with HMBA (an inducer of MEL cells to differentiate and form hemoglobin) to 1.18-GHz RFR at SARs up to 69.2 W/kg, with incubation temperature held at 37.4°C. There were no significant differences in any of the endpoints between cultures exposed at each RFR level and corresponding control cultures.

Studies in Vivo. Huang et al.[138] exposed hamsters to 2.45-GHz RFR at levels in the range 5-45 mW/cm^2 (2.3-20.7 W/kg), and cultured leukocyte suspensions with or without the mitogen PHA. For cultures not stimulated with PHA, the percentage of transformed cells increased with RFR level to a maximum at 30 mW/cm^2 (13.8 W/kg), clearly a thermal effect. For unclear reasons, however, the percentage decreased at still higher RFR levels. There were no significant changes in differential leukocyte counts, thus supporting the contention that RFR does not cause lymphocytosis. For PHA-stimulated cultures exposed at 30 and 45 mW/cm^2 (13.8 and 20.7 W/kg), the percentage of cells in mitosis diminished significantly relative to the total number of lymphocytes.

Huang and Mold[139] exposed mice to 2.45-GHz RFR at 5-15 mW/cm^2 (3.7-ll W/kg), after which they cultured spleen cells with tritiated thymidine and with or without a T-lymphocyte mitogen or a B-lymphocyte mitogen. Then the cells were assayed for thymidine uptake, a measure of DNA synthesis during cell proliferation. The results for the RFR-exposed mice showed cyclic time variations of thymidine uptake for the mitogen-stimulated and nonstimulated cultures, but also for the sham-exposed mice, apparently due to factors other than RFR, thus rendering the findings of this study questionable.

Lin et al.[140] exposed mice to 148-MHz RFR at 0.5 mW/cm^2 (0.013 W/kg) for 10 weeks beginning on postpartum day 4, 5, 6, or 7, and weighed them periodically up to age 600 days. The mean weights of the mice did not differ significantly from those of sham-exposed mice at corresponding ages. In blood samples drawn periodically up to age 600 days, no significant differences were seen between RFR-exposed and sham-exposed mice in any of the blood parameters.

Wiktor-Jedrzejczak et al.[141] exposed mice to 2.45-GHz RFR at 14 W/kg in a single 30-minute session or in three such sessions, one per day, three days apart. Total T-lymphocyte populations were unaffected by either single-session or triple-session exposures. However, single sessions significantly increased the population of one subclass of splenic B-lymphocytes (CR^+) and not of another subclass (Ig^+), but triple sessions increased both subclasses. Also, splenic cells from mice given single or triple exposures and cultured with various T-cell or B-cell mitogens showed significant increases in blastic transformation of B-cells but nonsignificant effects on T-cells. Mice were inoculated with either of two types of antigens and were exposed to the RFR. Antibody production to either

antigen was reduced by RFR-exposure, but only the difference for one of the antigens was statistically significant. Taken together, the results of this study show that thermogenic RFR levels (e.g., 14 W/kg) can have weak stimulatory effects on splenic B-lymphocytes but none on T-lymphocytes.

Sulek et al.[142] found that the threshold for increases in CR^+ B-cells was about 5 W/kg for 30-minute exposures to 2.45-GHz RFR, yielding an energy-absorption threshold of 10 J/g. They noted that multiple exposures at levels below that threshold were cumulative if done within one hour of one another, but not if spaced 24 hours apart. However, Schlagel et al.[143] presented results showing that RFR-induced increases in CR^+ B-cells depend on genetic factors. For example, mice of strain CBA/J that have a specific histocompatibility haplotype showed marked increases in CR^+ cells due to RFR-exposure, but mice having other haplotypes did not.

Wiktor-Jedrzejczak et al.[144] noted the findings of Schlagel et al.[143] and suggested that the effect might be mediated by a humoral factor. As a test, they performed experiments involving implantation of spleen cells derived from RFR-exposed CBA/J mice into sham-exposed CBA/J mice and vice versa, and assayed both groups for circulating CR^+ spleen lymphocytes. For both donor and recipient mice exposed to RFR, the counts of circulating CR^+ spleen lymphocytes were higher than for their respective controls, thus supporting their hypothesis.

Smialowicz et al.[145] exposed CBA/J mice 10-12 weeks old to 2.45-GHz CW RFR at 15-40 mW/cm^2 (11-29 W/kg) for 30 minutes. Six days later, assays of the percentages of CR^+ splenic cells and the numbers of nucleated splenic cells showed no significant differences in those two endpoints between sham-exposed mice and mice exposed at any of the RFR levels. Thus, these authors could not confirm the RFR-induced CR^+ increases found by Wiktor-Jedrzejczak and coworkers. Assuming that older mice may be more responsive, Smialowicz et al.[145] also exposed mice 14, 16, and 24 weeks old at 30 or 40 mW/cm^2. Only the 16-week-old mice exposed at 40 mW/cm^2 (29 W/kg) showed significantly higher percentages of CR^+ cells and smaller numbers of nucleated spleen cells.

Liburdy[146] exposed mice for 15 minutes to 26-MHz RFR at 80 mW/cm^2 (5.6 W/kg), which produced core-temperature rises of 2-3°C. Other mice were heated in an oven at 63°C for the same duration to obtain the same rises in core temperature. A decrease in the mean lymphocyte count (lymphopenia) and a rise in the mean neutrophil count (neutrophilia) were seen in the RFR-exposed mice, which persisted for 12 hours after exposure. Additional RFR-exposures at 3-hour intervals sustained the effects and prolonged the recovery period. By contrast, the oven-heated mice showed only slight effects. Those effects were absent for mice exposed to 26-MHz RFR at 50 mW/cm^2 or to 5-MHz RFR at 800 mW/cm^2, both of which yielded a whole-body SAR of about 0.4 W/kg (the basis for the 1982 ANSI standard[73]).

Smialowicz et al.[147] exposed 16 rats almost continuously for 69-70 consecutive days to 970-MHz RFR at 2.5 W/kg. Blood samples after exposure showed no significant differences from sham-exposed rats in erythrocyte count, total or differential leukocyte counts, mean cell volume of erythrocytes, hemoglobin concentration, or hematocrit. Splenic cells cultured with various mitogens exhibited no significant differences in responses from sham-exposed rats. However, the results of blood-serum analysis and the absence of changes in erythrocyte assays of the RFR-exposed group indicated that the rats may have been dehydrated. The authors noted that 2.5 W/kg is about half the basal metabolic rate of the rat, and that at 970 MHz, there probably were regions within the rat where local SARs were much higher than 2.5 W/kg, and that such higher SARs could have affected the endocrinologic system of the rat.

Smialowicz et al.[148] exposed pregnant mice to 2.45-GHz RFR at 28 mW/cm^2 (16.5 W/kg) for 100 minutes daily. At 3 and 6 weeks of age, the pups were assessed for development of primary immune response to an antigen (SRBC), proliferation of lymphocytes *in vitro* by stimulation with mitogens, and *in vitro* activity of natural killer (NK) cells against lymphoma cells. No consistent significant differences were found between RFR-exposed and sham-exposed mice in any of the endpoints.

Smialowicz et al.[149] exposed mice to CW or pulsed 425-MHz RFR at up to 8.6 W/kg. No differences were seen in mitogen-stimulated responses of lymphocytes or in primary antibody response to sensitization with SRBC or another antigen (PVP) between RFR-exposed and sham-exposed mice, or between mice exposed to the CW or pulse-modulated RFR.

Smialowicz et al.[150] exposed mice for 1.5 hours per day to 2.45-GHz RFR at several levels. For positive controls, other mice were injected with either hydrocortisone or saline. Splenic cells were then assayed *in vitro* for NK-cell activity by their cytotoxicity against mouse-lymphoma cells. NK-cell activity was significantly suppressed for 30 mW/cm^2 (21 W/kg), but activity returned to normal within 24 hours after the last RFR-exposure. However, this transient effect was not seen at 15 or 5 mW/cm^2 (10.5 or 3.5 W/kg). NK-cell activity was also assayed *in vivo*. Suppression of activity was seen in mice exposed at 30 mW/cm^2, but with return to normal several days after the last exposure. Hydrocortisone injection caused activity suppression both *in vitro* and *in vivo*.

Ortner et al.[151] exposed rats to 2.45-GHz RFR for 8 hours at 2 or 10 mW/cm^2 (0.44 or 2.2 W/kg). Within 5 to 15 minutes after exposure, peritoneal mast cells were extracted, and histamine releases from the cells induced by a chemical stimulant were determined. No significant differences from controls were observed in percentage of cell viability, percentage of cells, amount of histamine per cell, and cell diameter. For other rats similarly exposed, the counts of total red and white cells were not affected by exposure at either RFR level, nor were the blood-hemoglobin levels or percentages of lymphocytes and neutrophils relative to those for sham-exposed rats. The other types of cells were also unchanged by the RFR, and serum biochemistry parameters were not affected by either RFR level.

Wong et al.[152] sought possible effects of prolonged exposure of rats to low RFR levels in the HF band (3-30 MHz). In one of two experiments, 20 groups of 5 rats each were exposed to 20-MHz RFR at 1,920 mW/cm^2 (about 0.3 W/kg) for 6 hours per day, 5 days per week, and another 20 groups were sham-exposed as controls. After 8 days of treatment, 6 groups each of the exposed and control rats were euthanized and examined for histopathology. This was also done for 7 groups each after 22 days and for the remaining 7 groups each after 39 days. The results showed a significantly higher mean count of red blood cells and a significantly lower mean hemoglobin content for the rats terminated after 39 days of exposure than for the control group. However, statistical analysis showed that those differences were not RFR-related.

In the second experiment, 12 rats were exposed to the RFR and 12 other rats were sham-exposed, but each rat was housed separately and all rats were euthanized after 6 weeks. On termination, blood samples were collected and the routine counts and blood-chemistry assays were done, the spleens were excised and weighed, and suspensions of splenic cells were prepared. Also various other tissues were examined for histopathology. Unlike the previous results, no significant differences were seen in mean red blood cell count, hemoglobin, or any blood-chemistry parameters. All of the 24 rats were found to be histologically normal.

Studies in Vivo On The Effects Of Chronic Exposure On Health, Longevity, And Resistance To Disease. As discussed previously, Prausnitz and Susskind[58] exposed 100 mice to 9.3-GHz pulsed RFR at average power density of 100 mW/cm^2 (roughly 45 W/kg) for 4.5 minutes daily, which yielded a mean rise in body temperature of 3.3°C, 5 days per

week for 59 weeks. Controls were 100 sham-exposed mice. Some deaths occurred in both groups, which were attributed to a pneumonia infection introduced accidentally into the colony. However, the death rate was found to be lower in the RFR-exposed mice than in the sham-exposed mice, a finding that could be ascribed to the protective effect of the daily rise in temperature ("fever") induced by the RFR. On necropsy, liver abscesses were found in some mice, but because of tissue breakdown, the relative incidence in RFR-exposed and control mice could not be determined. The authors mistakenly described the occurrence of leukosis (increase of circulating lymphocytes) as "cancer of the white blood cells."

Szmigielski et al.[153] injected mice with staphylococcal bacteria at a dose selected to yield a 60% survival rate on day 3 after injection. Before injection, the mice were sham-exposed or exposed to CW or pulsed 2.45-GHz RFR at 5 or 15 mW/cm^2 (2-3 or 6-9 W/kg) 2 hours per day for 6 or 12 weeks. For the mice exposed at 5 mW/cm^2 for 6 weeks, the survival rate was 80%; for those exposed at 5 mW/cm^2 for 12 weeks, it was 45%. The differences among those two RFR groups and the sham-exposed group were not statistically significant. The survival rates at 15 mW/cm^2 for 6 or 12 weeks respectively were 25% and 5%.

Liddle et al.[154] sought effects of RFR-exposure at various ambient temperatures on the survival of mice given an LD_{50} dose of another strain of staphylococcus. They injected mice with that strain and exposed them to 2.45-GHz RFR at 10 mW/cm^2 (6.8 W/kg) for 5 days (4 hours per day) at 8 different ambient temperatures in the range 19-40°C. For temperatures up to 31°C, the percentages of RFR-exposed mice that survived the challenge were significantly higher than for sham-exposed mice. The results also indicated that most of the deaths of exposed animals were due to hyperthermia, and indicated that RFR-exposure may be beneficial to infected animals at low and moderate ambient temperatures, in consonance with the findings of several other studies.

In the previously discussed study at the University of Washington, 100 rats were exposed to 2.45-GHz RFR at 0.5 mW/cm^2 and sham-exposed 100 other rats for 25 month, and 10 each of the two groups were euthanized after 13 months (the interim kill), as were 10 of the 12 RFR-exposed rats and 10 of the 11 sham-exposed rats that survived the 25-month regimen (the final kill). In the immunologic aspects of the study, assays of suspensions of splenic cells at the interim kill showed significantly higher counts of T- and B-lymphocytes for the RFR-exposed rats than the sham-exposed rats, indicating that the RFR had stimulated the lymphoid system. However, there were no significant differences in T-cell and B-cell populations between the RFR and sham groups of the final kill, a possible indication of the onset of immunosenescence.

The values of CR^+ for the RFR groups of both kills were lower than for the sham groups, but the differences were not significant, indicating no differences between RFR and sham groups in lymphocyte maturation. For both kills, there were no significant differences in percentages of plaque-forming cells in response to immunization with sheep red blood cells. Stimulation of splenic-cell suspensions with various mitogens yielded mixed results at the interim kill. No mitogen results were obtained for the terminal kill because the lymphocyte cultures failed to grow and respond to any of the mitogens.

Blood samples drawn periodically from all of the rats were assayed for various hematologic parameters and serum chemistry. By multivariate analyses, there were no overall significant differences between RFR-exposed and sham-exposed rats in the hematologic parameters. Differences in thyroxine (T_4) levels between the RFR-exposed and sham-exposed rats were nonsignificant, indicating that the RFR had no effect on the hypothalamic-pituitary-thyroid feedback mechanism. As expected, however, the T_4 levels of both groups decreased significantly with age.

Toler et al.[155] implanted cannulas in the aortas of 200 rats. After the rats recovered, 100 of them were concurrently exposed to 435-MHz pulsed RFR. Exposures were at 1

mW/cm^2 average power density for about 22 hours daily, 7 days a week, for 6 months. The whole-body SARs varied with time, ranging from 0.04 to 0.4 W/kg, with a mean of about 0.3 W/kg. The other 100 rats were concurrently sham-exposed. Blood samples drawn cyclically without rat restraint or anesthesia were assayed for hormones ACTH, corticosterone, and prolactin in the plasma; for plasma catecholamines; and for hematologic parameters, including hematocrit and various blood cell counts. Heart rates and arterial blood pressure were also monitored. There were no significant RFR-induced differences between groups in any of the endpoints.

Overall, many of the early studies seeking possible effects of RFR on suspensions of various classes of leukocytes exposed *in vitro* suffered from the lack of adequate control of cell temperature during exposure. In later studies in which effective control over culture temperature was exercised, nonsignificant differences were obtained with exposed cultures held at the same temperature as control cultures for the same durations. In studies where elevation of culture temperature by RFR or conventional means did affect leukocytes adversely, the effects were clearly of thermal origin.

In early studies of RFR exposure of erythrocytes *in vitro*, hemolysis and potassium-ion (K^+) efflux were found for rabbit erythrocytes that were exposed at average power densities as low as 1 mW/cm^2. In subsequent investigations, however, the hemoglobin and K^+ losses from rabbit erythrocytes resulting from heating with RFR to 37°C did not differ significantly from losses from conventional heating; the threshold for effect was found to exceed 46 W/kg.

Studies seeking immunological effects of exposing animals to RFR *in vivo* yielded mixed results. Some investigators found that RFR-exposure of mammals increased the proliferation of leukocytes or the production of antibodies (relative to controls), but with few exceptions, the measured or estimated SARs were well in excess of 1 W/kg. More subtle effects on mammalian immune systems were sought in more recent studies, making use of significant advances in assay methods, and with attention to possible effects of non-RFR stress. Some of those investigations were directed toward the effects of RFR on the activity of natural killer (NK) cells, the results of which again showed that SARs much higher than 1 W/kg were necessary for effect. On the other hand, some studies indicated that animals exposed for short periods to relatively high RFR levels can withstand bacterial infection better than sham-exposed animals.

More directly relevant to possible effects of RFR on the human immune system would be studies in which animals are chronically exposed to RFR (preferably over virtually their entire lifetimes), to determine whether such exposure adversely affects their health, longevity, and resistance to natural disease or to experimental challenge with various microorganisms or toxins therefrom. The previously discussed University of Washington study, though with some findings open to question, is an example. Also noteworthy is the study of rats by Toler et al.[155] for the number of animals involved and the long exposure duration. As remarked by the authors, the absence of RFR-induced effects complement those of Guy and coworkers at the University of Washington. However, because of funding limitations, relatively few such studies have been carried out or repeated by other laboratories.

Physiology and Biochemistry. *Metabolism and Thermoregulation.* Bollinger[156] exposed rhesus monkeys to 10.5-MHz or 19.3-MHz RFR at successively higher power densities up to 600 mW/cm^2 (about 0.2 or 0.6 W/kg, respectively), or to 26.6-MHz RFR at up to 300 mW/cm^2 (0.6 W/kg). Colonic temperatures and electrocardiograms (EKGs) taken during exposure indicated no obvious indications of thermal stress, heart-rate increases, or other influences on the electrical events of the heart cycle due to the RFR. Also, rhesus monkeys were exposed to 10.5- or 26.6-MHz RFR for 1 hour at 200 or 105 mW/cm^2 (0.06

or 0.2 W/kg), or to 19.3-MHz RFR for 14 days (4 hours per day) at 115 mW/cm^2 (0.1 W/kg). Hematologic and blood-chemistry analyses done before and after exposure showed no significant differences between exposed and control monkeys for most of the cellular components of blood. Unrelated to RFR were significant differences in mean counts of monocytes and eosinophils. No abnormalities ascribable to exposure were seen in gross pathological and histopathological examinations.

Frazer et al.[157] exposed rhesus monkeys to 26-MHz RFR at up to 1,000 mW/cm^2 (2.0 W/kg) for 6 hours, during which skin and rectal temperatures were measured. The monkeys remained in thermal equilibrium even at the highest RFR level; their thermoregulatory mechanisms were able to efficiently dissipate the additional heat from the RFR. Krupp[158] exposed rhesus monkeys for 3 hours to 15 or 20 MHz RFR at levels up to 1,270 mW/cm^2 (1.3 W/kg). Again, the thermoregulatory mechanisms of the monkeys readily accommodated the additional heat. Krupp[159] followed up on 18 rhesus monkeys that had been exposed 1-2 years previously to 15-, 20- or 26-MHz RFR for up to 6 hours at least twice at levels in the range 500-1270 mW/cm^2. No RFR-related variations from normal values of hematologic and biochemical blood indices or of physical conditions were found.

Ho and Edwards[160] exposed mice for 30 minutes to 2.45-GHz RFR in a waveguide system that permitted measurement of oxygen-consumption rates and SAR during exposure. Such measurements were done at 5-minute intervals during exposure. Oxygen-consumption rates were also measured at 5-minute intervals for 30 minutes before and after exposure. The oxygen-consumption rates were converted into specific metabolic rates (SMRs) and expressed in the same units as the SARs (W/kg). At the highest RFR level, both the mean SAR and mean SMR had steadily decreased during exposure, thereby decreasing the total thermal burden of the mice. Apparently, they sought to diminish their thermal burdens by altering their body configurations during exposure to minimize their RFR-absorption rates, and they reduced their oxygen consumption. After exposure completion, oxygen consumption rates returned to normal.

To study voluntary thermoregulation, Stern et al.[161] trained fur-clipped rats in a cold chamber to press a lever that turned on an infrared lamp. The rats were then exposed to 2.45-GHz RFR for 15-minute periods at increasing levels, ranging from 5 to 20 mW/cm^2 (1-4 W/kg). As the RFR level was raised, the rats responded to maintain a nearly constant thermal state by decreasing the rate at which they turned on the lamp.

Adair and Adams[162] trained three squirrel monkeys to regulate their environmental temperature (T_a) behaviorally by adjusting the flows of air at various temperatures into an exposure chamber. The monkeys were then exposed to 2.45-GHz RFR for 10-minute periods at levels in the range 1-22 mW/cm^2 (0.15-3.3 W/kg). The monkeys were also exposed to infrared radiation (IR) of equivalent power densities while being sham-exposed to RFR. For the RFR at about 7 mW/cm^2 (1.05 W/kg) and higher, all were stimulated to select a lower T_a, indicating the existence of a threshold of 1.1 W/kg whole-body SAR or 20% of the resting metabolic rate of the squirrel monkey. Comparable reductions in selected T_a did not occur for exposure to the IR.

Bruce-Wolfe and Adair[163] studied the ability of squirrel monkeys to vary the level of 2.45-GHz RFR as a thermal energy source. They trained four monkeys to successively select air streams having temperatures of 10°C and 50°C (30° ± 20°C, the thermoneutral temperature in the exposure chamber). The resulting mean T_a was about 35°C. Then the 50-°C air source was replaced with 2.45-GHz RFR at 20 mW/cm^2 (3 W/kg) and 30-°C air. Thus, only the latter two sources were activated whenever the monkeys demanded heat, and only the 10-°C air source was activated whenever they demanded cooling. The results indicated that the monkeys were readily able to use the thermal energy from the RFR for thermoregulation instead of the 50-°C air source, and were thereby able to maintain normal rectal temperature.

Adair et al.[164] did similar experiments to determine the effects of long-term RFR exposure on thermoregulation. The exposures were for 15 weeks, 40 hours/week, to 2.45-GHz CW RFR at 1 or 5 mW/cm^2 (0.16 or 0.8 W/kg) at T_as of 25, 30, or 35°C. Fourteen monkeys were trained to select a preferred T_a, and were treated concurrently in fours, one pair each for RFR-exposure and sham exposure. The results for 25°C or 30°C showed no changes in preferred T_a during exposure at 1 mW/cm^2. However, at 35°C and 1 mW/cm^2 (0.16 W/kg), or at all three T_as and 5 mW/cm^2 (0.8 W/kg), the monkeys selected cooler T_as (1 to 3°C lower). Their colonic temperatures were not affected, but their skin temperatures varied with T_a and RFR-exposure in an unreliable way.

Lotz and Saxton[165] studied the vasomotor and metabolic responses of five rhesus monkeys exposed to 225-MHz CW RFR with body axis parallel to the electric component. In the first of two protocols, each monkey was given 10-minute RFR exposures at successively higher levels, with enough time after each exposure for the monkey to return to its pre-exposure equilibrium, until a marked vasomotor response was evidenced by a rapid change in tail-skin temperature. RFR levels in the range 1.2-12.5 mW/cm^2 (0.3-3.6 W/kg) were used. At 20°C, metabolic heat production was not altered at 1.2 mW/cm^2 (0.3 W/kg) but declined with increasing RFR level. At 26°C, the rate of metabolic heat production before exposure was well below that at 20°C, and was not altered by the 10-minute RFR-exposures. The lowest RFR level that reliably altered metabolic heat production during such 10-minute exposures was in the range 5-7.5 mW/cm^2 (1.4-2.1 W/kg).

In the second protocol, the monkeys were equilibrated at 20°C and then given single 120-minute exposures at levels in the range 0-10 mW/cm^2 (0-2.9 W/kg). The monkeys were also similarly treated at 26°C, but at RFR levels up to 7.5 mW/cm^2 (2.1 W/kg). The mean metabolic heat production dropped sharply during RFR exposure at 20°C, but remained essentially unchanged during RFR exposure at 26°C. Also evident was progressive recruitment of metabolic and vasomotor responses at 20°C. At both ambient temperatures, the mean colonic temperature during the last 30 minutes of RFR-exposure was higher than for the last 30 minutes of sham-exposure, even at 2.5 mW/cm^2 (0.7 W/kg), which was below the threshold for thermoregulatory action. This result indicated that thermoregulatory responses could not fully compensate for the heat generated by the RFR even in the cooler environment (20°C).

In summary, the thermal basis for effects of RFR on the autonomic thermoregulatory systems of mammals and on their behavioral thermoregulatory responses to RFR is evident. Especially noteworthy are results for primates because of their far greater similarities to humans than other animals.

Endocrinology. Cairnie et al.[166] exposed unanesthetized male mice for 16 hours to 2.45-GHz RFR at 50 mW/cm^2 (60 W/kg) and determined their rectal and testis temperatures at exposure end. The mean rectal temperature was significantly higher than for sham-exposed mice, but the mean testis temperature did not differ significantly, showing that the thermoregulatory system of the testes was able to compensate fully for the increased thermal burden from RFR at close to lethal level. Also, conscious mice exposed for various durations to 2.45-GHz RFR in the range 21-37 mW/cm^2 exhibited no testicular cell damage or abnormal sperm counts. The corresponding ranges of whole-body and testicular SARs were 25.3-44.5 W/kg and 8.4-14.8 W/kg.

Lebovitz and Johnson[167] exposed unanesthetized male rats to 1.3-GHz RFR for 9 days (6 hours per day) at a whole-body SAR of 6.3 W/kg, resulting in a mean core-temperature rise of 1.5°C. On exposure completion, groups of rats were weighed and euthanized at intervals corresponding to 1/2, 1, 2, and 4 cycles of spermatogenesis. The RFR-exposed rats yielded 87.6% normal sperm after a half-cycle of spermatogenesis versus 95.8% for

sham-exposed rats. The difference was significant, but one rat of the RFR-exposed group contributed most of their abnormal sperm, which rendered the finding suspect. There was no significant difference in the mean weight of seminal vesicles, indicating that exposure at 6.3 W/kg was not deleterious to testosterone production, a finding supported by histologic evaluations by light microscopy.

Lebovitz and Johnson[168] exposed unrestrained male rats for 8 hours to 1.3-GHz CW RFR at 9 W/kg, selected to yield a core-temperature rise of 4.5°C and stated to be lethal for chronic exposure. Subgroups at 1/2, 1, 2, and 4 spermatogenesis cycles following exposure were analyzed for testis mass and daily sperm production as in the previous study. Trunk blood was assayed for follicle-stimulating hormone (FSH) and leutinizing hormone (LH). There were no significant differences between RFR-exposed and sham-exposed rats in any of the endpoints, except for a decline in sperm count 2 cycles of spermatogenesis after RFR-exposure. However, the authors remarked that a single positive result among the negative results for all of the many other endpoints studied is highly questionable. They also noted that a differential sensitivity of germ cells at this stage of maturation had been reported for conventional heating of the testes.

Lotz and Michaelson[169] first "gentled" rats for 2 weeks before RFR exposure by weighing and handling them at least four times a week, and behaviorally equilibrating each rat by taking its colonic temperature and putting it into an exposure cage for 3-5 hours for several days before use. They had observed a rapid rise in colonic temperature and corticosterone (CS) levels in the blood of the rat in the first 30 minutes of occupancy in the exposure chamber, followed by return to baseline values by the end of 180 minutes, thus proving the need for such equilibration before exposure.

The authors then exposed unanesthetized gentled rats to 2.45-GHz RFR at up to 60 mW/cm^2 (9.6 W/kg) for up to 120 minutes and measured their colonic temperatures and CS levels after exposure. The mean colonic temperature showed a small but significant rise after 30 minutes at 13 mW/cm^2 (2.1 W/kg), with 30-minute exposures at higher levels yielding mean temperature rises approximately proportional to the RFR level. The mean CS level increased nonsignificantly for durations up to 120 minutes at 13 mW/cm^2 (2.1 W/kg), up to 60 minutes at 20 mW/cm^2 (3.2 W/kg), and 30 minutes at 30 mW/cm^2 (4.8 W/kg). The results yielded threshold values for adrenal-axis stimulation of 30-50 mW/cm^2 (4.8-8.0 W/kg) for 60-minute exposures and 15-20 (2.4-3.2 W/kg) mW/cm^2 for 120-minute exposures. The latter range is somewhat less than half the resting metabolic rate of the rat.

Lu et al.[170] investigated the effects of 2.45-GHz RFR on serum thyroxine (T_4) concentration in male Long-Evans rats from two suppliers, Blue-Spruce (BS) and Charles River (CR). The normalized SAR was 0.19 W/kg per mW/cm^2. The rats were acclimated and gentled before exposure. Seven protocols were used that involved exposures at various RFR levels up to 70 mW/cm^2 for up to 8 hours. At the end of treatment, the rats were euthanized and assayed for serum T_4 concentration.

The results for 1-hour exposures at up to 70 mW/cm^2 showed no dependence of T_4 concentration on RFR level. Although some significant changes were seen for 2-hour exposures, those results were supplier-dependent. Thus, when the T_4 levels for the RFR-exposed rats from each supplier were compared with the sham-exposed rats from the same supplier, no significant RFR-induced changes in T_4 concentration were seen.

For the 4-hour exposures, only the results for BS rats exposed at up to 20 mW/cm^2 were shown. Their T_4 levels, compared to those for sham-exposure, were significantly higher at 1 mW/cm^2, not significantly changed at 5 or 10 mW/cm^2, and were significantly lower at 20 mW/cm^2. The results for the other protocols showed no significant RFR-induced alterations in T_4 level except for CR rats given 3 consecutive daily 4-hour exposures at 40 mW/cm^2, for which the T_4 level was significantly lower than for shams.

Lu et al.[171] endeavored to determine the influence of confounding factors in studies of effects of RFR on the adrenal cortex, with serum CS concentration used as the index of adrenocortical function. Gentled rats were subjected to 10 protocols involving exposures to 2.45-GHz RFR for 2 or 4 hours at up to 55 mW/cm^2 (11 W/kg). Colonic-temperature and CS-concentration rises were found to be dependent on RFR level with distinct thresholds, but the effect diminished with repetition. For example, the threshold for change of CS concentration was 40 mW/cm^2 (8 W/kg) at the first exposure, but no changes were observed in rats exposed 10 times at levels up to 40 mW/cm^2 (8 W/kg).

Injection of ethanol lowered baseline colonic temperatures and raised CS concentrations, effects not observed for saline-injected controls. Ethanol injection after RFR-exposure at 10 or 20 mW/cm^2 yielded higher concentrations of CS than in rats injected with saline after exposure. Removal of hair from the rats did not affect the baseline colonic temperatures and concentrations of CS significantly, but it decreased the RFR-induced hyperthermia and CS stimulation. The authors concluded that no adrenal response to RFR is evident without a colonic temperature rise of at least 0.7°C (20 mW/cm^2 or 4 W/kg).

Lotz and Podgorski[172] collected blood samples hourly for 24 hours from 6 rhesus monkeys and assayed them for cortisol, T_4, and growth hormone (GH). At the same clock time during those 24 hours, they exposed each monkey for 8 hours to 1.29-GHz RFR at 20, 28, or 38 mW/cm^2 (2.1, 3.0, or 4.1 W/kg). Three sessions at each RFR level were alternated with sessions of sham-exposure at intervals of 10-14 days for recovery. The data collected for the corresponding clock periods of the three sessions at each RFR level were averaged, to yield a 24-hour temporal series of mean values for each condition.

The mean rectal temperature rose within 2 hours after the start of RFR exposure to plateaus that were dependent on RFR level, but returned to control level within 2 hours after exposure end. For sessions at 20 and 28 mW/cm^2, the mean cortisol levels did not differ significantly from those for sham-exposure sessions, but rose significantly during sessions at 38 mW/cm^2. The levels then diminished to control values, indicating that the effect was transient. The authors suggested the existence of a threshold between 28 and 38 mW/cm^2 (3.0 and 4.1 W/kg) for rises in cortisol levels and associated such rises with rectal-temperature elevations of about 1.7°C, thus supporting the hypothesis that adrenocortical effects of RFR are thermally induced. For all RFR levels, no significant differences in mean GH or T_4 levels were seen.

In summary, although some effects of exposure to RFR on the endocrine system seem to be predictable from physiological considerations, other, more subtle effects may be worthy of additional study, such as those related to the interactions among the pituitary, adrenal, thyroid, and hypothalamus glands and/or their secretions. Part of the problem appears to be related to the uncertainties about stress mechanisms and various accommodations to such mechanisms. Animals placed in novel situations are more prone to exhibit stress responses than those adapted to experimental situations.

Because the effects of RFR on the endocrine systems of animals are largely ascribable to increased thermal burdens, to stresses engendered by the experimental situation, or to both, there is no clear evidence that such effects would occur in humans exposed to RFR at levels that do not produce significant increases in body temperature.

Cardiovascular Effects. Frey and Seifert[173] exposed excised beating frog hearts to 10-μs pulses of 1.425-GHz RFR at 60 mW/cm^2 peak. The pulses were triggered at the peak of the electrocardiogram (EKG) P wave and at 100 and 200 ms after that peak. The results for zero and 100-ms delays were inconclusive, but a significant rise in heart rate (tachycardia) was seen for the 200-ms delay.

Clapman and Cain[174] were unable to obtain those results. They also exposed groups of frog hearts to 2-μs or 10-μs pulses of 3-GHz RFR at 5,500 mW/cm^2 peak; for one 2-μs group, the pulses were triggered at the initial rise of the EKG's QRS complex and another 2-μs group was exposed to unsynchronized pulses at 500 pps (5.5 mW/cm^2 average power density). Again, no significant differences in heart rate were seen between any of the RFR-exposed groups and a control group, in contrast with those of Frey and Seifert.[173] and Liu et al.[175] also sought similar effects with excised hearts, but their findings were negative. Those authors also opened the thorax of frogs to expose the heart *in situ* to 100-μs pulses of 1.42-GHz or 10-GHz RFR, and again obtained negative findings.

Galvin et al.[176] isolated cardiac muscle cells from the quail heart and exposed them in suspension to 2.45-GHz RFR at 37°C for 90 minutes at SARs up to 100 W/kg. After exposure, samples of the suspensions were examined for integrity of the cells by their exclusion of trypan blue, a vital stain. Cell integrity was unaffected by exposure at 1 W/kg, but suspensions exposed at 10, 50, and 100 W/kg yielded successively larger increases in percentages of cells permeable to trypan blue relative to control suspensions. Suspensions were assayed for release of the enzymes creatine phosphokinase (CPK) and lactic acid dehydrogenase (LDH). CPK release was unaffected at any SAR. Release of LDH rose with SAR, but the increases were nonsignificant relative to controls except at 100 W/kg. By electron microscopy, the structures of heart cells exposed at 1, 10, and 50 W/kg, as well as control cells, were normal. Cells exposed at 100 W/kg showed increased intracellular changes, but intercellular junctions remained intact.

Yee et al.,[177] concerned about possible electrode artifacts, tested several types of electrodes for recording beat rates from isolated frog hearts during RFR-exposure. Faster than the usual decreases in heart rate after excision were seen only with glass electrodes containing either potassium chloride or a metal wire, results showing that bradycardia can be induced by field intensification caused by such electrodes.

Yee et al.[178] mounted isolated frog hearts within a waveguide filled with physiologic (Ringer's) solution. Each heart's beat rate was monitored for 60 minutes at 5-minute intervals. For those exposed to RFR, exposure was begun 10 minutes into the monitoring period and was terminated 30 minutes later. At the end of the period, the mean heart rate of the group of control hearts decreased linearly to 67% of the initial rate.

Other hearts were exposed to trains of 10-μs 2.45-GHz pulses at SARs up to 200 W/kg, either triggered or not triggered by the EKG. Most of the RFR-exposed groups exhibited decreases in heart rates similar to those of the controls. Noteworthy were the results for two groups exposed to RFR, both continuously at 200 W/kg, but only one cooled by circulating bathing solution. The uncooled group yielded heart-temperature increases of 2.5, 5.5 and 8°C at 5, 15, and 30 minutes of exposure, with concomitant decreases in mean heart rate relative to controls. By contrast, the cooled RFR-exposed group showed no significant differences from controls. Moreover, a group heated with circulating bathing solution to obtain a temperature-versus-time rise similar to the 200-W/kg group showed a linear decrease in heart rate to final values comparable to those of that group.

Yee et al.[178] also noted that Schwartz et al.[179] had reported a 19% increase in calcium efflux from isolated frog hearts that were exposed for 30 minutes to 16-Hz-modulated, 1-GHz RFR at 0.15-3 W/kg, an effect not seen for CW RFR or RFR amplitude-modulated at natural heart-beat rates. They tried to reproduce that finding by exposed one group each to CW and pulsed 2.45-GHz RFR, both amplitude-modulated at 16 Hz, at 3 W/kg. Their results did not significantly differ from those for controls. They remarked that an increase in concentration of free Ca^{++} in the cytoplasm of heart cells triggers rapid changes in heartbeat, so the absence of such changes in these groups does not support the findings of Schwartz et al.[179]

Yee et al.[180] did a study similar to their earlier one but with rat hearts. As in the frog-heart study, the beat rate of each heart was monitored for 60 minutes at 5-minute intervals, but exposure was begun 20 minutes into the monitoring period and was terminated at 50 minutes. The results led the authors to conclude that exposure to pulsed 2.45-GHz RFR at 2 or 10 W/kg had no specific influence on the myocardium or its neural components.

Presman and Levitina[181,182] had done two heart studies on the live rabbit, one with 2.4-GHz CW RFR at 7-12 mW/cm^2 and the other with 3-GHz pulsed RFR at 3-5 mW/cm^2 average power density, 4.3-7.1 W/cm^2 pulse power density. In both studies, rabbits were exposed for 20-minute periods each in various orientations. During each exposure and for 10 minutes before and afterward, the EKGs of the rabbits were recorded with plate electrodes.

Exposure of the entire top surface of the rabbit produced neither tachycardia nor bradycardia during RFR exposure. However, tachycardia was seen during the first 5 minutes postexposure, changing to bradycardia during the remaining 5 minutes postexposure. By contrast, exposing the top of the head only or the rear only produced significant tachycardia during exposure, with the head exposure yielding the greater effect. Exposing the underside of the rabbit yielded bradycardia during exposure, which was followed by returns toward normal heart rates during postexposure. The findings were difficult to assess because no data were given, only the relative differences among mean values. Also, artifact may have been present from use of metal electrodes.

Kaplan et al.[183] and Birenbaum et al.[184] tried to reproduce the results of those studies. Their findings were negative except for RFR levels that were clearly hyperthermic.

Phillips et al.[185] exposed rats to 2.45-GHz RFR for 30 minutes at 0, 4.5, 6.5, or 11.5 W/kg. Nonsignificant bradycardia was seen at 4.5 W/kg; mild but significant bradycardia developed within 20 minutes at 6.5 W/kg, followed by recovery in 2 hours; pronounced bradycardia occurred abruptly at 11.1 W/kg, after which heart rates rose to values well above those of controls, and they persisted at the higher levels to the end of the test period. The authors surmised that the heart block was caused by release of toxic materials, elevated serum potassium, or myocardial ischemia, all from excessive heat.

Galvin and McRee[186] studied the effect of RFR-exposure *in vivo* on the functioning of cat hearts with and without surgically produced myocardial ischemia (MI). One group of MI hearts was exposed to 2.45-GHz CW RFR at 30 W/kg with an applicator for 5 hours, and another MI group was sham-exposed. For comparison, two groups of non-MI hearts were similarly treated. Before and during the treatment, mean arterial blood pressure, cardiac output, heart rate, and EKG were measured, and blood samples were assayed for plasma protein concentration and creatine phosphokinase (CPK) activity. After the treatment, the hearts were excised and assayed for tissue CPK activity.

The results for both the MI and non-MI cats indicated no significant differences in mean arterial blood pressure, cardiac output, or heart rate between RFR-exposed and sham-exposed groups, and no synergism of ischemia and RFR-exposure for those cardiovascular indices. Also, the RFR and sham groups showed no significant differences in plasma or tissue CPK activity. Thus, localized exposure of the undamaged or ischemic heart to the RFR *in vivo* had no effect on the myocardium or its neural components, results at variance with those for excised hearts exposed to RFR.

Galvin and McRee[187] exposed conscious rats from below for 6 hours to 2.45-GHz CW RFR at 10 mW/cm^2 (3.7 W/kg) and assayed various cardiovascular, biochemical, and hematologic indices. No significant differences between RFR-exposed and sham-exposed rats were seen in any of the blood parameters. The initial mean heart rates of the two groups did not differ significantly, but the mean heart rate of the RFR group decreased to about 90% during the first hour and remained there (with smaller variations) during the rest of the period. Based on the results of a followup experiment, the authors surmised that the

bradycardia was due to reduction of metabolic rate to compensate for the heat from the RFR.

In summary of the physiologic and biochemical effects of RFR, there are scientifically credible experimental data to show that the thermoregulatory systems of nonhuman primates can readily compensate for high RFR levels, an especially important finding because of the greater similarities in anatomy and physiology between human and nonhuman primates than between humans and the various species of laboratory mammals.

Most of the studies of possible effects of RFR on endocrine systems were conducted on rodents. Studies that reported positive findings also yielded indications that the effects were largely due to increases in the thermal burdens of the animals. In many studies, observed alterations in endocrine function may have been significantly influenced by stresses in the animals. For this reason, the results of those studies that involved stress reduction by acclimating animals to handling and the experimental situation are notable. Nevertheless, some of the more subtle effects are worthy of further study.

Regarding cardiovascular effects, the positive findings reported in early studies (bradycardia, tachycardia, or both) were suspect because of the use of attached or indwelling electrodes that probably introduced artifact. Various kinds of electrodes were investigated, and several special types were developed that were not perturbed by RFR or did not perturb the local RFR fields. Studies involving use of such electrodes showed that heart rates were altered only at RFR levels that produced rises in temperature or otherwise added thermal burdens to the animal. Also investigated were possible effects of RFR pulses at repetition rates synchronous with periodic characteristics of the EKG. Specifically, the authors of an early study reported induction of tachycardia in excised hearts by RFR pulses in synchrony with the EKG, but others could not confirm this finding in excised hearts or in live animals.

Several researchers showed that for CW RFR, levels well in excess of 1 mW/cm^2 or 1 W/kg were necessary for significant alterations of heart rate. Small decreases in heart rate were seen in equilibrated conscious rats exposed for 6 hours at 3.7 W/kg, a finding ascribed to a compensating reduction in metabolic rate. The results of another study indicated that the functioning of hearts damaged from other causes (e.g., rendered ischemic) is not affected by exposure to CW RFR at 10 mW/cm^2 or lower.

RFR and Behavior. *Behavioral Studies in Rodents.* Justesen and King[188] trained food-deprived rats to lick a nozzle 40 successive times to obtain a drop of dextrose-water solution. They then added the presentation of an audio tone at random intervals to signal availability of the reward. After such training, the rats were exposed to 2.45-GHz RFR at up to 1.5 mW/cm^2 (4.6 W/kg) in 1-hour sessions consisting of alternating 5-minute intervals of RFR and no RFR. The mean number of responses by the rats diminished with increasing RFR level, but the decreases at higher levels were related to the cessation of responding rather than lower licking rates, most likely associated with warming.

In a three-part study, Hunt et al.[189] exposed rats to 2.45-GHz RFR for 30 minutes. In the first part, each rat was exposed to the RFR at 6.3 W/kg or sham-exposed, after which its exploratory movements within a test chamber were recorded. The mean activities after either treatment decreased with time, but the values were generally lower during most of the period after RFR-exposure than after sham-exposure and became comparable for the treatments toward session end. The RFR-exposed rats were often seen sleeping during the middle parts of sessions.

In the second part, the authors trained rats to repeatedly swim a 6-meter channel for 24 hours, and scored each rat's performance versus time as its median swim speed for each block of 20 traverses. In one experiment, rats were sham-exposed or exposed at 6.3 W/kg for 30 minutes and tested immediately after treatment to determine any prompt effects.

Their mean performance was similar to that of their sham group for about 200 traverses, but was below mean control speed for about the next 100 traverses, after which both groups again performed comparably. In two other similar experiments, the RFR level was 11 W/kg, and the rats were tested immediately or after 1 day of delay. Measurements of colonic temperatures immediately after treatment showed that the rats had been rendered severely hyperthermic. The performance of the group tested right after exposure was clearly impaired by the hyperthermia, but the group tested 1 day later showed recovery and yielded results similar to those of the 6.3-W/kg group.

In the third part, the authors trained water-deprived rats to press a lever in a complex vigilance-discrimination task to obtain small quantities of saccharin-flavored water. On the first day, all groups were sham-exposed. During the next four days, each group was exposed for one 30-minute period each at 6.5 and 11 W/kg and two periods of sham-exposure. Performance was tested for 30 minutes after each treatment. The mean error rate 5 minutes after exposure at 6.5 W/kg was significantly higher than after sham-exposure, but dropped to the latter range at 15 and 25 minutes. After exposure at 11 W/kg, however, the mean error rates were all much higher than after sham-exposure. The mean number of responses diminished with increasing RFR level, but the decreases at higher levels were related to response cessation rather than to lower licking rates, an effect most likely associated with warming.

Monahan and Ho[190] exposed mice for 15 minutes within a waveguide to 2.45-GHz CW RFR at forward powers up to 4.8 W in an ambient temperature held at 24°C by air flow through the waveguide. During exposure, the mean SAR and the mean percentage of forward energy absorbed were measured at 5-minute intervals. In another experiment, exposures were limited to 10 minutes and the absorptions were recorded at 12-second intervals. The mice could not be watched within the waveguide, but the results of both experiments showed that they had oriented themselves to reduce the percentages of RFR energy absorbed and the SARs when the forward power was 1.7 W (initial SAR 28 W/kg) or higher.

Lin et al.[191] sham-exposed or exposed food-deprived rats to 918-MHz RFR at 10, 20, or 40 mW/cm^2 (2.1, 4.2, or 8.4 W/kg) during 30-minute sessions. The rat holder was a truncated cone of rods designed to allow the rat to poke its head through the narrower end and move it freely. A small upward head movement interrupted a horizontal light beam, thereby registering a count. The rat was required to do 30 such movements rapidly and regularly for a food pellet. A downward head movement gave access to the pellet delivered.

One of three rats was exposed for 30 minutes each at 2.1, 4.2, and 8.4 W/kg on consecutive days, another rat was exposed on alternate days at the same levels, and a third was given 30-minute sessions of sham-exposure. No significant effects on performance were seen at 2.1 or 4.2 W/kg. At 8.4 W/kg, the performance of the two RFR-exposed rats did not change during the first 5 minutes. However, both rats displayed heat stress and diminished performance during the remaining 25 minutes. Yet another rat was exposed at successively higher levels up to 32 mW/cm^2 (6.7 W/kg), at which it exhibited similar signs of heat stress. Its performance rates indicated a threshold between 30 and 40 mW/cm^2 (6.3 and 8.4 W/kg).

Schrot et. al.[192] trained 3 rats to respond to auditory stimuli with four presses on three levers in a specific sequence that was changed for each session. Just before each session, the rats were sham-exposed or exposed to pulsed 2.8-GHz RFR at a specific average power density in the range 0.25-10 mW/cm^2 (0.04-1.7 W/kg) for 30 minutes. Sessions were conducted daily, 5 days a week. Exposure at 10 mW/cm^2 (1.7 W/kg) of all three rats yielded higher error-responding rates, lower sequence-completion rates, and alterations in the normal acquisition pattern. Similar effects were seen at 5 mW/cm^2 (0.7 W/kg) but to a

lesser extent. Below 5 mW/cm^2, most data points were within the control range, but a few were outside that range. The significance of the latter points is uncertain.

In a study by Gage and Guyer,[193] they trained rats to perform on a reinforcement schedule in which the opportunity to obtain a food pellet was presented on the average of once each minute in a preplanned sequence of intervals without cueing. After training, groups were exposed to 2.45-GHz RFR for 15.5 hours at 8 or 14 mW/cm^2 (1.6 or 2.8 W/kg) in 22°, 26°, or 30°C ambient temperature. The response rates at each temperature diminished directly with increasing RFR level, but the effects of ambient temperature itself were not consistent.

Lebovitz[194] exposed groups of unrestrained rats to 1.3-GHz pulsed RFR in individual waveguides. In each waveguide were a vertical displacement bar (behavioral operandum), a means for illuminating the operandum as a cue, and a means for delivering food pellets. Before exposure, groups of food-deprived rats were trained to press the bar for food pellets until they learned to press the lever 5 successive times to obtain a pellet (a fixed-ratio-5, or FR-5 schedule). Those that performed at the highest and most stable rates were trained further to respond only when the operandum was illuminated (a multiple fixed-ratio, extinction schedule of reinforcement). During training, the FR schedule was gradually raised to FR-25. The responses when the operandum was illuminated (SD) and when it was not illuminated (Sd, which yielded no pellets) were counted separately. Exposures were for 3 hours daily, 5 days a week. Behavioral sessions were initiated 15 minutes after the start of exposure and were halted 15 minutes before exposure end. Each behavioral session was divided into 6 equal sequential blocks to evaluate intrasession changes.

The results for 8 weeks of exposure at 1.5 W/kg showed no significant SD differences between the RFR and sham groups. Slight declines in rates during sessions were seen in both groups. The response rates for Sd were more variable than for SD. The intrasession declines in Sd rates, which were also evident for baseline and recovery weeks, was sharper than for SD, but there were no significant differences between the RFR and sham groups.

Groups exposed for 9 weeks at 3.6 W/kg also showed no significant differences in SD response rates. Marginally significant changes in weekly rates were seen, and the rate of intrasession decline was significant. The Sd response rates of both groups showed sharp intrasession declines again. The results for 6 weeks of exposure at 6.7 W/kg showed no significant differences between groups in overall SD response rates.

From the negative results for SD and Sd at 1.5 W/kg and the doubtfully significant decline in Sd rate at 3.6 W/kg, the author suggested that the latter RFR level could be the approximate threshold for modifying the rat behavioral paradigm studied. The author also indicated that 6.7 W/kg is the approximate resting metabolic rate for a rat, so that RFR level represents a doubling of the heat dissipation requirements of the rat. They therefore concluded that thermal factors were likely involved in the positive results.

Lebovitz[195] exposed similarly trained groups of rats to CW or pulsed 1.3-GHz RFR and tabulated their SD and Sd response rates, called S+ and S- in this paper. The results for 3.6 W/kg and 5.9 W/kg were consonant with those of the previous study. Also, the S+ and S- rates for pulsed RFR at 6.7 W/kg were similar to those with CW RFR at 5.9 W/kg. The author concluded that the differences in rates between the pulsed-RFR and sham groups could not be ascribed to the pulsed character of the RFR *per se*. Core temperatures were measured in other rats. Exposures to CW or pulsed RFR at 3.5 W/kg, the approximate threshold above, yielded no significant differences in rectal-temperature changes compared with rats similarly sham-exposed. However, exposures at 6.3 W/kg yielded increases of 0.5° to 1°C, with no significant duration-dependent differences. Thus, the thermal basis for the behavioral changes above is evident.

D'Andrea et al.[196] exposed a group of 14 chamber-adapted rats to 2.45-GHz CW RFR at 0.5 mW/cm^2 (0.14 W/kg) 7 hours daily for 90 days. Body masses and intake of food and

water were measured daily. Each rat was tested monthly for its threshold reactivity to footshock by observing movements of its paws in response to electric shocks of varied intensity within a gridded-floor chamber. Differences in body mass, food and water intake, or threshold footshock reactivities relative to those for the 14 sham-exposed rats were not significant. Right after such treatment, 7 rats of each group were assessed for open-field behavior, shuttlebox performance, and lever pressing for food pellets on an interresponse time schedule. The rest of the rats were examined for gross pathology; no significant differences ascribable to the RFR were seen. Major changes were observed in the open-field tests, but none of the differences were related to RFR-exposure. Open-field tests done 60 days after treatment yielded similar results.

Shuttlebox performance was tested for responses to an electric shock given right after presentation of a tone and white light as a warning. The rat could prevent presentation of the shock by crossing to the other side of the shuttlebox during 10 seconds of the warning (an avoidance response), or could cross while receiving the shock (an escape response). The time lags (latencies) for avoidance and escape responses were recorded. Overall, there were no significant differences between RFR-exposed and sham-exposed rats. A shuttlebox test done 60 days after treatment showed no significant differences in mean latencies or their variances.

Two days after the shuttlebox testing, the rats were deprived of food and trained daily to press a lever twice for a food pellet, with a specific time interval between the presses. The training was rendered progressively more difficult until the rats were required to do the second press only between 12 and 18 seconds after the first press to obtain a food pellet. During the training, the RFR group earned fewer pellets than the sham group, but the differences at corresponding times and overall were not significant.

The results of this study and of two similar studies in the same laboratory[197,198] were not fully consistent and showed little if any statistically significant differences between RFR-exposed and sham-exposed rats, but suggested that the threshold for behavioral responses to 2.45-GHz RFR in rats may be in the range 0.5-2.5 mW/cm^2 (0.14-0.70 W/kg).

Mitchell et al.[199] exposed chamber-adapted rats to 2.45-GHz CW RFR at 10 mW/cm^2 (2.7 W/kg) for 7 hours. Right after treatment, each rat was tested for spontaneous locomotor activity, acoustic startle response, and retention of a shock-motivated passive avoidance task. Lower spontaneous activity was seen in RFR-exposed rats than in sham-exposed rats. In the startle-response test, each rat was subjected to 20 intense acoustic pulses at variable intervals in the range 20-60 seconds, and the response of the rat during each acoustic pulse was determined. The startle responses of the RFR-exposed rats were significantly lower than for the sham-exposed rats. Each rat was then placed in the lighted smaller part of a gated, two-chamber shuttle box, the larger part of which was dark and equipped to deliver an electric shock. The gate was opened after 1 minute, and if the rat moved into the larger chamber within 2 minutes, it was given a shock, but if it remained in the smaller chamber for more than 2 minutes, it was removed and not tested further. One week later, retention of the shock experience was tested in the box by allowing each rat 5 minutes (instead of 2 minutes) within the smaller chamber to react. The differences in passive avoidance activity between the RFR and sham groups were not significant.

Akyel et al.[200] trained groups of 4 rats each on three different behavioral schedules to obtain food pellets. After training, each rat was exposed once a week for 10 minutes to 10-μs pulses of 1.25-GHz RFR at 1-MW peak forward power, with its long axis parallel to the electric component of the RFR. During sessions, the average forward power was held constant at 4, 12, 36, or 108 W, obtained by using a pulse repetition frequency of 240, 720, 2160, or 6480 pps. Each rat was administered all four RFR levels in a quasi-random weekly order. The whole-body specific absorptions (SAs) and whole-body specific absorption rates

(SARs) respectively ranged from 0.5 to 14.0 kJ/kg and 0.84 to 23.0 W/kg. Each rat was tested starting right after exposure end.

At the three lower RFR levels, no significant differences in any of the three behavior schedules were seen. At the highest level (14.0 kJ/kg, 23.0 W/kg), however, the rats trained on two of the schedules failed to reach baseline performance, and those on the third schedule exhibited variable effects. Exposures at that level caused an average colonic-temperature rise of 2.5°C, and the rats did not respond at all for about 13 minutes after exposure completion. The authors concluded that those behavioral changes were thermally induced.

Behavioral Studies in Nonhuman Primates. Galloway[201] trained rhesus monkeys to press one of three levers when that lever was lit in order to obtain a food pellet (discriminative behavior). After the training, the head of each monkey was exposed with an applicator to 2.45-GHz RFR at estimated mean head SARs of 7, 13, 20, 27, and 33 W/kg, and the effects on their performance were examined. The RFR was administered for 2 minutes just before each behavioral session but was stopped earlier if the monkey began to convulse. Convulsions occurred for all exposures at 33 W/kg, and often at 20 W/kg. Each monkey was exposed at least twice at each level during a 9-month period. Also, three of them were exposed at 13 W/kg for 5 daily 1-hour schedules of 2 minutes on and 1 minute off, totaling 40 minutes of exposure per day. No effects on that discriminative task were evident for either exposure regimen.

In a repeated-acquisition test, the three levers were lit sequentially four times in a specific order, and each monkey had to press the levers in the correct sequence to obtain a pellet. Daily sessions of 60 trials each were conducted before exposure, with the correct sequence changed each day. In the sessions just preceding RFR-exposure, a slight learning trend (decrease in error rate) was seen, but the changes were too small to ascribe significance. This was also true for the results at all RFR levels except 33 W/kg, for which the error rate at session start was highest. Thus, except possibly for the latter result, the RFR had no effect on this behavioral paradigm.

Cunitz et al.[202] inserted the head of a 3-kg or a 5-kg rhesus monkey through a hole in the bottom of a 383-MHz resonant cavity, with the monkey facing a diamond array consisting of the ends of four light pipes mounted through the cavity's side wall. Each monkey was trained to move a lever to the left, right, up, or down when any of the pipes was lit, to indicate the position of the lit pipe end in the array. For criterion performance, the monkey had to do 100 correct lever presses to obtain a food pellet. During testing, the pipes were lit in random order. A correct response caused the lighting of another pipe plus a tone for 0.75 second. An incorrect response yielded a 3-second timeout during which all of the lights were off and lever movements had no consequences.

In each session, the monkey's head was exposed to 383-MHz RFR for 2 hours at a specific input to the cavity in the range 0-15.0 W. The head SARs were estimated to range up to 33 W/kg and 20 W/kg respectively for the 3-kg and 5-kg monkey. The larger one was also exposed at 17.5 W (23 W/kg). Each monkey was restrained in a chair during the first hour, and the testing was done for the second hour. Sessions were conducted on 5 consecutive days at each successive level, with sham-exposure sessions before the RFR was raised to the next level. The lowest head SARs for diminished performance by the two monkeys were about the same: 22 and 23 W/kg.

Scholl and Allen[203] trained 3 rhesus monkeys in a visual-tracking task that required each monkey to move a lever so as to hold a continuously moving spot within a prescribed clear area on the screen of a display monitor. The spot was moved electronically in a specific pattern, and the responses generated continuous difference signals (errors). The central 15% of the screen was clear and comprised the on-target area. The monkeys received a brief electric shock for each 1 second accumulated outside the on-target area.

After training, the monkeys were exposed to horizontally polarized, 1.2-GHz CW RFR at 10 and 20 mW/cm^2 (measured at the center of the head in the absence of the monkey) for 2 hours each at one level and at the other level 2 days later. This polarization and frequency were chosen to provide half-wave resonant absorption in the monkey head. The corresponding head SARs were 0.8 and 1.6 W/kg. The data on mean tracking errors clearly showed that their performance was not diminished by the RFR-exposures.

De Lorge[204] trained five rhesus monkeys to perform the following task while seated: Each monkey was required to press a lever in front of its right arm, thus producing either a low-frequency tone to signal that no food pellet will be coming, or a higher-frequency tone for which the monkey had to press a lever in front of its left arm to receive a pellet. During 1-hour training sessions, pellets were made available at variable intervals averaging about 30 seconds. During 2-hour training sessions, pellets were made available at about 60-second intervals. The monkeys were exposed frontally to 2.45-GHz RFR at levels in the range 4-72 mW/cm^2 at head height. The estimated head SARs were 0.4-7.2 W/kg (0.1 W/kg per mW/cm^2).

After the monkeys achieved stable performance, they were tested on the variable 30-second delivery schedule in 1-hour sessions during which each was exposed to the RFR at 4 or 16 mW/cm^2 (0.4 or 1.6 W/kg head SAR) for 30 minutes. Their performances were not affected by either RFR level. Only three of them were tested on the variable 60-second delivery schedule in 2-hour sessions, during which they were exposed for 1 hour at levels in the range 16-72 mW/cm^2 (1.6-7.2 W/kg head SAR). One of them was also exposed at 16 mW/cm^2 during entire 2-hour test sessions. The performances of all three monkeys showed no significant departures from control rates for up to 52 mW/cm^2 (5.2 W/kg) and for two of them at 62 mW/cm^2 (6.2 W/kg); at the latter level, the performance of the third monkey was about 80% of its mean control rate. At 72 mW/cm^2 (7.2 W/kg), all three performed at about 50% of their mean control values. Those results suggested that the monkeys had reacted to body heating by the RFR at the higher levels, which diminished their performance.

De Lorge[205] trained four squirrel monkeys to press either the right or the left lever on top of a chair to obtain a food pellet. At first, a red light and a blue light were turned on alternately with each successive press of the levers. Next, the contingencies were changed such that presses of the right lever continued to alternate the red and blue lights (without reward) but a press of the left lever was rewarded only when the blue light was on. Each of the next stages of training required a higher number of right-lever presses to turn on the blue light. The last stage was a schedule in which each right-lever press yielded either a half-second of red light or 10 seconds of blue light, with a left-lever press during the latter yielding a pellet.

Each monkey was then exposed from above to 2.45-GHz RFR at levels in the range 10-75 mW/cm^2. SARs were estimated to have been 0.5 to 3.75 W/kg. No consistent behavioral changes occurred below 50 mW/cm^2 (2.5 W/kg); above that level, the effects increased with RFR level. During the 1-hour sessions, only the rate of right-lever responses exhibited a slight trend toward lower rates with increasing RFR level. The results for the 2-hour sessions were similar, but more pronounced. The right-lever-response rate versus RFR level varied widely among the monkeys, but at 60 mW/cm^2 (3.0 W/kg), all showed decrements to about 60%.

The author concluded that the observed behavioral changes were temporary and clearly related to hyperthermia. Consistent changes were seen when rises in rectal temperature exceeded 1°C, which corresponded to a threshold between 40 and 50 mW/cm^2 (2.0-2.5 W/kg). The author noted that similar results had been obtained with rhesus monkeys tested for the same behavioral task during exposure to 2.45-GHz RFR, but with a threshold 10 to

20 mW/cm^2 higher, and suggested that RFR-induced behavioral changes in different species may be scaled on the basis of body mass.

The findings of this study, reinforced by the similar results with rhesus monkeys, are important because the measurements of performance of a complex behavioral task during exposure to RFR were carried out with two species much closer to human physiology and intelligence than more commonly used nonprimate laboratory animals, and because reasonably accurate RFR thresholds for each primate species were determined.

De Lorge[206] similarly trained rhesus monkeys to perform a task in which each monkey was to press a lever in front of its right hand (called an observing response), which produced a brief low tone to signal that no food pellet will be delivered, or a longer high tone to signal the availability of a pellet. If the monkey pressed a lever in front of its left hand while the high tone was on (a detection response), the tone would cease and a pellet would be delivered. A response on the left lever at other times caused a 5-second interval in which presses of the right lever yielded only the low tone.

After stable performance was reached, each monkey was frontally exposed, during 1-hour sessions, to vertically polarized 225-MHz CW RFR (near the whole-body resonant frequency), or to pulsed RFR at 1.3 GHz or 5.8 GHz (both above whole-body resonance). The exposures to 225 MHz were at 5-11 mW/cm^2 (2.0-4.4 W/kg); those to 1.3-GHz RFR were at 20-95 mW/cm^2 average (2.6-12.4 W/kg); and those to 5.8-GHz RFR were at 11-150 mW/cm^2 (0.34-4.7 W/kg). The results yielded average-power-density thresholds for behavioral effects that increased with frequency: 7.5 mW/cm^2 at 225 MHz, 63 mW/cm^2 at 1.3 GHz, and 140 mW/cm^2 at 5.8 GHz. However, the corresponding whole-body SARs varied up and down with frequency: respectively 3.0, 8.2, and 4.3 W/kg, presumably because of differences in penetration depth. The detection-response rate on the food lever was not consistently affected by RFR-exposure at any of the frequencies: No effect was observed for 225 MHz or 5.8 GHz; a decreased response rate was occasionally seen for 1.3 GHz, but only at 83 mW/cm^2 (10.8 W/kg) or higher.

Exposure to 5.8-GHz RFR at 150 mW/cm^2 (4.7 W/kg) also produced minor burns on the faces of three of the monkeys, with the worst burns occurring between the eyes and along the orbitonasal area. The erythema disappeared within a few days except in one monkey who continually irritated the burned skin by removing scabious material. No burns occurred at 140 mW/cm^2 (4.4 W/kg), the behavioral threshold for this frequency, or at the highest levels of the other frequencies. The small penetration depth for 5.8 GHz (about 0.8 cm) probably was an important factor.

D'Andrea et al.[207] trained five rhesus monkeys to operate three levers (left, right, center) in various sequences to obtain food pellets. The sessions were 60 minutes long. The task during each session comprised three successive 10-minute schedules of lever presses, followed by a repeat of the same three schedules.

In the first 10-minute schedule, the monkey was required to withhold responses for 8 seconds after the start of an audio tone, and then to respond only within the next 4 seconds; the correct response during those 4 seconds was two presses of the left lever within 2 seconds of each other. The authors called this an interresponse-time (IRT) schedule. The second 10-minute period involved a time-discrimination (TD) schedule in which a press of the center lever in the presence of blue light yielded white light of either short or long duration in random fashion; at the end of either duration, the white light was replaced with red and green light. For the monkey to obtain a pellet when the red and green light was present, it had to press the right lever if the preceding white light was of short duration or the left lever if the preceding white light was of long duration.

During the third 10-minute period, a fixed-interval (FI) schedule was used: The monkey was presented with a continuous high tone, and its first press of the right lever after 55 seconds yielded a pellet.

During the 60-minute behavioral test sessions, each monkey was sham-exposed or exposed from above to 3-μs pulses of 1.3-GHz RFR at a root-mean-square pulse power density of 131.8 W/cm^2. The peak SAR was 15.0 W/kg in the head and 8.3 W/kg whole-body. The pulse repetition rate was 2, 4, 8, 16, or 32 pps, with corresponding average power densities of 0.92, 1.85, 3.70, 7.40, or 14.80 mW/cm^2.

The results showed no significant differences between sham-exposures and RFR-exposures in any of the behavioral responses. The authors noted that the the energy absorbed in the head by each pulse (280 mJ/kg) was well above the threshold for the RFR-auditory effect, and they remarked that if such auditory stimulation did occur, it produced no obvious effect on the trained behavior.

In summary, many of the studies on avoidance behavior indicate that RFR could be a noxious or unpleasant stimulus. There is much evidence, however, that changes in behavioral patterns induced by RFR are responses by their thermoregulatory systems, either to minimize absorption of heat in normal or warm ambient environments (including high levels of humidity) or to obtain warmth in relatively cold environments. Thus, other than possible auditory perception of RFR pulses, animals do not appear to directly sense RFR.

The results of studies on disruption of performance or learned behavior by RFR were variable; however, most of the findings showed that the behavioral changes were ascribable to the added thermal burden imposed by the RFR, and were significant at whole-body SARs well in excess of 1 W/kg.

It is worth emphasizing that the behavioral findings of the primate studies are more relevant than those with the other animal species with regard to possible effects of RFR on human behavior, because the tasks the primates had to learn were far more complex, and their physiologies and intelligence are much closer to those of humans. It is also noteworthy that reasonably accurate thresholds for RFR-induced behavioral changes were determined for each primate species studied, and that those thresholds served as a basis for the ANSI/IEEE[24] human-exposure standard.

RFR and Drugs. Various studies have been conducted on possible interactive effects of exposure to RFR and medications or other drugs taken or administered. Those discussed below are representative.

Thomas et al.[208] trained rats on a fixed-interval, 1-minute (FI-1) schedule to press a bar for a pellet. After stable baseline patterns were achieved, an effect-versus-dose function for chlordiazepoxide, a psychoactive drug (tradename *Librium*), given 30 minutes before a session, was established. This function showed that the responding rate rose with increased drug dose up to 10 mg/kg of body weight, attaining 2 to 3 times the baseline rate at that dose. At still higher doses, the responding rate decreased; it attained zero at 40 mg/kg.

After training, the rats were exposed to 2.45-GHz RFR at 1-W/cm^2 peak, 1-mW/cm^2 average (0.2 W/kg) during the 30 minutes before each bar-pressing session. RFR-exposure yielded the same shape of effect-versus-dose function, but the magnitudes were generally higher by a factor of about 2. By contrast, RFR-exposure without drug injection produced no difference in responding rate. The results of this study are unequivocal, but the mechanisms are obscure. For example, although the average power density and whole-body SAR were low, local SARs in brain regions that are target areas for central actions of chlordiazepoxide may have been high enough for a thermally potentiating effect. It is also possible that the pulse parameters produced the RFR-hearing effect during the 30-minute exposure preceding the bar-pressing session. If so, it is not clear whether or how this effect would have influenced the testing sessions themselves, during which RFR was absent.

Thomas and Maitland[209] trained six rats to depress a lever on a schedule in which a second response at least 18 seconds after a first response was rewarded with a pellet, but a second response in less than that interval reset the timing period. After such training,

effects of exposure to the pulsed RFR (0.2 W/kg) used in the previous study were sought on the dose-versus-response function of the psychoactive drug d-amphetamine.

Three of the rats were dosed with the drug once per week and exposed for 30 minutes (single-exposure condition). Their behavior was studied for 1 hour right after exposure for any direct drug-RFR interaction. To detect possible cumulative action of RFR, the other three rats were dosed with d-amphetamine once a week and exposed for 4 days a week, 30 minutes daily (multiple-exposure condition), except on drug-injection days. On the latter days, their behavior was observed for the 30 minutes after injection. The sessions were conducted for 13 weeks, and included sham-exposures and saline injections.

For the single-exposure condition, the mean response rates after saline injection and sham-exposure and after saline injection and RFR-exposure were comparable to baseline performances. When those rats were given d-amphetamine and sham-exposed, their mean response rates rose with drug dose to a maximum at 2.0 mg/kg, with consequent reductions in the frequency of correct responses that yielded reinforcement. At higher drug doses, the mean response rates dropped sharply, to zero for 4.5 mg/kg. By contrast, the mean response rate of drug-injected rats and exposed to the RFR rose to values significantly higher than for the corresponding drug doses and sham-exposure, with maximum response rate at 0.5 mg/kg. Above 0.5 mg/kg, the mean response rate declined sharply, to zero for 1.5 mg/kg. Those results show that exposure to the RFR after injection of a given dose of d-amphetamine yielded behavior similar to that obtained with a larger dose without the RFR exposure.

The dose-response functions of the rats with and without multiple RFR-exposures were qualitatively similar to those with and without single RFR-exposures. With the multiple sham-exposures, maximum responses were obtained for 2.0 mg/kg, with a sharp decline to zero for 4.5 mg/kg. The maximum responses for the multiple RFR-exposures were obtained with 0.5 mg/kg, and the responses declined sharply to zero for 2.0 mg/kg.

Thomas et al.[210] did similar research with the drugs diazepam and chlorpromazine. Diazepam (*Valium*) has been widely used as a tranquilizer and muscle relaxant. Chlorpromazine is a sedative and an antiemetic. Four rats each of two different strains were trained on a fixed-interval, 1-minute (FI-1) schedule of reinforcement. After training, the dose-effect functions for diazepam were determined in one strain and for chlorpromazine in the other strain. For each drug, one dose was injected into rats 30 minutes before each session, and their response rates were compared with their respective baseline performances. Chlorpromazine diminished the performance of all four rats for doses above about 1 mg/kg. For those given diazepam, the drug caused slight increases in response rate at up to 2.5 mg/kg and declines at higher doses.

After determining the dose-effect functions for the drugs, the authors exposed each rat for 30 minutes to 2.8-GHz pulsed RFR at 0.2 W/kg right after administering each drug, and tested the rats at exposure end. The RFR did not alter the effects of either chlorpromazine or diazepam, in contrast with the results with chlordiazepoxide and d-amphetamine. Such differences in findings are difficult to reconcile.

Ashani et al.[211] sought possible changes of the hypothermic effects of anticholinesterase drugs in rats and the influence of antidotes for such drugs. Mixed results were obtained for 10-minute exposures to 2.8-GHz RFR at 10 mW/cm^2 (about 2 W/kg). Since few people ingest such drugs or their antidotes, there appears to be no direct significance of the findings with regard to possible effects of RFR on human health.

Pappas et al.[212] conducted experiments to determine whether RFR alters seizures induced by the drug pentylenetetrazol (PTZ), and to study the effects of RFR on the efficacy of chlordiazepoxide (CDZ) for counteracting such seizures. In one experiment, rats were exposed for 30 minutes to 2.7-GHz pulsed RFR (2-μs pulses at 500 pps) at average power densities up to 20 mW/cm^2 (2.25 W/kg). After exposure, the rats were given PTZ at

doses up to 80 mg/kg, and their seizure activity was studied. In another experiment, each rat was injected with anti-seizure CDZ at doses up to 15.0 mg/kg before exposure. After exposure, 70 mg/kg of PTZ was injected, and the degree of inhibition of PTZ-induced seizures by the CDZ was studied.

In both experiments, the rats were watched for signs of seizure activity for 8 minutes after injection of PTZ. The latency interval to the onset of the first sign was recorded, and the seizure intensity was rated from 0 (no seizure, normal exploratory activity) to 4 (wild running and convulsions with 99% mortality).

In the first experiment, the latency times to seizure following PTZ injection decreased with increasing PTZ dose for all RFR levels. The rats exposed at 15 mW/cm^2 (2.25 W/kg) exhibited significantly shorter latencies than the sham-exposed rats. The mean score of seizure intensity increased with PTZ dose, with no apparent effect of RFR level. Although the mean score was significantly higher for 15 mW/cm^2 (2.25 W/kg) than 5 mW/cm^2 (0.75 W/kg), the values for the RFR groups did not differ significantly from those of the sham group. The authors suggested that the decreases in seizure latency and the increases in seizure intensity at 15 mW/cm^2 could have resulted from local brain hyperthermia rather than alteration of the PTZ effect on brain neuronal activity.

The results of the second experiment were unclear. Rats given 7.5 mg/kg of CDZ had longer latencies after exposure at 15 mW/cm^2 (2.25 W/kg) than at lower levels, and the latencies of rats given 15 mg/kg were shorter after exposure at 5 mW/cm^2 (0.75 W/kg) than for sham-exposure or exposure at 10 or 15 mW/cm^2 (1.5, or 2.25 W/kg). The authors suggested that those positive findings may be ascribable to a random Type II statistical error. Therefore, they did a third experiment, the results of which did not support the earlier findings that 15 mW/cm^2 (2.25 W/kg) or even 20 mW/cm^2 (3 W/kg) enhanced PTZ seizures and increased the antiseizure protection of CDZ at 7.5 mg/kg. Thus, they regarded the few apparently positive results as spurious, Type II errors, but did not rule out possible experimenter bias.

Lai et al.[213] exposed unrestrained rats to circularly-polarized pulsed 2.45-GHz RFR in cylindrical waveguides for 45 minutes at a spatially-averaged power density of 1 mW/cm^2 (0.6 W/kg whole-body SAR). The authors noted that at this SAR, the range of average power densities for linearly polarized RFR would be 3-6 mW/cm^2. Control rats were sham exposed. The effects of each of several drugs given right after exposure were investigated.

In the first experiment, the effect of the RFR on the stereotypy induced by subcutaneous apomorphine injection was studied: Rats were scored on which of five different forms of behavior they exhibited during 5-minute observation periods right after apomorphine injection and after four 15-minute intervals. The results yielded a higher mean score for RFR-exposed rats than for sham-exposed rats, indicating that RFR-exposure produced more intense stereotypy, with biting and clawing, than did sham-exposure. Also studied was the effect of the RFR on the hypothermia induced by apomorphine: Colonic temperatures were measured right after RFR- or sham-exposure, after which the rats were injected with apomorphine and their colonic temperatures were recorded four more times at 15-minute intervals. At 15 minutes after drug injection, the mean temperature of the RFR group had decreased by 0.95°C, but the decrease was only 0.63°C for the sham group, a significant difference showing that the hypothermic effect of apomorphine was enhanced by the RFR.

Next, the effect of the RFR on stereotypy induced by d-amphetamine was studied. Each rat was injected with the drug right after exposure and was watched for the presence of any of three normal behaviors and three abnormal behaviors. Starting 4 minutes after injection, the rats were observed for 1 minute every 5 minutes for an hour. The difference in average score between the RFR and sham groups for the six behaviors was nonsignificant. The effect of the RFR on the hyperthermia induced by d-amphetamine was

also recorded: Colonic temperatures were measured right after exposure as before, but at 15-minute intervals for 90 minutes after drug injection. The results were shown as the mean colonic temperature change for each group versus time interval after injection. At zero time, the mean temperature was 38.3°C for the RFR group and 38.2°C for the sham group. The mean temperature of the sham group reached its maximum at 45 minutes, at which time it had increased by 1.4°C, and then it diminished to about 39.3°C at the end of the 90-minute period. By contrast, the temperature of the RFR group reached its maximum at 60 minutes, an increase of 1.2°C, and diminished to about 39.2°C at 90 minutes. These differences in time-dependent increases were significant.

In the final experiment, rats were injected with morphine at doses of 1 to 20 mg/kg right after exposure. The number of rats that exhibited catalepsy (general muscular rigidity and a certain posture for more than 1 minute) 30 minutes after injection and the number of rats that died within 2 hours after injection were recorded.

The differences in the percentages of RFR-exposed and sham-exposed rats that exhibited catalepsy were reported to be significant. However, in an erratum,[214] the authors indicated that the statistical method used was inappropriate. Their use of another statistical method showed that the responses of the sham group increased with drug dose, but the responses of the RFR group were not dose-dependent. Also, the original paper reported that no deaths had occurred in the RFR or sham group at doses of 1 or 5 mg/kg, but at the larger doses, the percentages of deaths were significantly higher in the RFR group. In the erratum, however, the authors noted that they found no significant differences in deaths due to the RFR.

It is interesting that the mean colonic temperatures of the apomorphine-injected and amphetamine-injected groups immediately after RFR-exposure were both 0.1°C higher than for their respective sham-exposed groups (38.3 versus 38.2°C in both cases). Not clear was the influence of thermoregulation on those results. Would similar results be obtained if pre-injection colonic temperatures were raised by the same amount by an agent other than RFR?

Lacking in this investigation were data on animals administered saline instead of the drugs. Such control data might have more clearly delineated subtle non-RFR factors that may have influenced the results.

In a similar study, Lai et al.[215] examined the effects of the same RFR on the actions of pentobarbital in the rat. In the first of two series, unanesthetized, unrestrained rats were exposed to the RFR. Each rat's colonic temperature was taken right after exposure, after which the rat was injected with sodium pentobarbital at a dose sufficient to induce surgical anesthesia. After the rat lost its righting reflex, its colonic temperature was recorded at 15-minute intervals for 150 minutes, and the time interval after injection to regain the righting reflex was noted. A group of rats was sham-exposed and similarly treated.

In the second series, baseline colonic temperatures were measured, and the rats were injected with pentobarbital. After 15 minutes, by which time all of the rats had lost their righting reflex, some rats were exposed to the RFR for 45 minutes anteriorly (head toward source) and other rats posteriorly (rear toward source). Their colonic temperatures were recorded for 90 minutes after exposure as were their time intervals for regaining the righting reflex.

The (conscious) rats in the first series did not show any preferred orientation during RFR-exposure and there was no significant difference in mean colonic temperature between the RFR and sham groups immediately after exposure (37.8°C for both). The mean colonic-temperature changes for each group versus time after pentobarbital injection indicated that both groups reached maximal hypothermia (about -3°C) at 75 minutes. Mean temperature depressions of the RFR group did not differ significantly from those of the sham group at corresponding times up to 105 minutes. During the interval from 105 to 150

minutes, the mean depressions for the RFR group were significantly larger than for the sham group, indicating that the recovery of the RFR group from the hypothermia was slower. Also, the mean time to righting-reflex recovery for the RFR group was significantly longer than for the sham group.

In the second series, the baseline mean colonic temperatures of the pentobarbital-injected rats before RFR- or sham-exposure were 37.9°C. Right after anterior-exposure or posterior-exposure, the mean temperatures of the RFR groups were respectively 34.6 and 34.7°C, and was 34.1°C for the sham group. The difference between the two RFR groups was not significant, but both values were significantly higher than for the sham group. All three groups attained maximal hypothermia 30 minutes after exposure (45 minutes after injection). The changes at that time for the posterior-RFR, anterior-RFR, and sham groups were respectively about -3.8, -4.2, and -4.5°C, but only the difference between the posterior-RFR and sham groups was significant.

From 30 to 90 minutes, all three groups showed recovery toward baseline temperatures. However, the posterior-RFR group recovered from the hypothermia earlier and recovered their righting reflex more quickly than the anterior-RFR and sham groups. The authors surmised that those findings were due to the differences in local energy deposition in the two orientations, which could yield differences in drug metabolism or kinetics.

Lai et al.[216] also did experiments to determined the effects of the same RFR on ethanol-induced hypothermia and consumption of ethanol. For the ethanol-hypothermia experiment, 15 rats were RFR-exposed and 14 were sham-exposed for 45 minutes. Right after exposure, each rat was removed from its waveguide, its colonic temperature was measured, and it was injected with a solution of ethanol (3 μg/kg of body weight in 25% of water by volume). After injection, the colonic temperatures of the rats were measured at 15-minute intervals for 120 minutes.

The mean colonic temperature of the RFR-exposed rats at exposure end did not differ significantly from that of the sham-exposed rats. Ataxia developed within 5 minutes of ethanol injection, but righting reflex remained intact. The mean colonic-temperature changes versus time after ethanol injection for the two groups showed that hypothermia had occurred in the RFR group at a significantly slower rate than the sham group, but the temperature depressions of both groups became about the same 90 minutes after injection.

In the ethanol-consumption part, rats were given 90-minute sessions in the waveguides daily for 9 days. On the first three days, the rats were placed within the waveguides for the first 45 minutes with the RFR source on "standby." At this time, a bottle of 10% sucrose solution was inserted in each waveguide and the amount consumed during the remaining 45 minutes was measured. On the fourth day, the procedure was the same but half the rats (24) were exposed to the RFR for the full 90 minutes, and the other half were sham-exposed. The procedure was the same on the next three days, but a bottle containing 15% ethanol + 10% sucrose was used instead (the latter to render the ethanol more palatable). On the eighth day, half the rats were exposed to the RFR for 90 minutes, the remaining were similarly sham-exposed, and the amounts of sucrose-ethanol solution consumed were measured. On the ninth day, the roles of the two groups were reversed: the first group was sham-exposed, and the second was RFR-exposed. The results indicated that the RFR had no apparent effect on sucrose consumption, but that it increased the consumption of the sucrose + ethanol solution.

It should be noted that there were significant differences in the mean baseline temperatures among the groups given different dosages, and that there was an apparent discrepancy in the mean baseline temperatures between the sham-saline groups. Such differences among control groups, as well as the significant colonic-temperature rises

during both RFR- and sham exposure, indicate the presence of large uncontrolled factors and render questionable most of the findings of this study.

Lai et al.[217] exposed rats to the same pulsed RFR (2-μs pulses of 2.45-GHz RFR at 500 pps) at a whole-body SAR of 0.6 W/kg either once for 45 minutes or for 10 daily 45-minute sessions to determine the effects of the RFR on the concentration and affinity of benzodiazepine receptors in the cerebral cortex, hippocampus, and cerebellum. The results of the single exposures showed a significant increase of receptor concentration in the cerebral cortex but not in the hippocampus or cerebellum. There were no significant changes in the binding affinity of the receptors in any of the three regions. The multiple exposures yielded no change in receptor concentration right after the last exposure, a possible indication of adaptation to repeated exposures.

In another experiment, rats adapted first to the experimental situation and then exposed once to the RFR showed significantly higher benzodiazepine-receptor concentrations in the cerebral cortex than did similarly treated sham-exposed rats. Those results led the authors to suggest that because such receptors are responsive to anxiety and stress, low-level RFR can be a source of stress.

In overall summary, the studies on possible synergism between RFR and psychoactive drugs such as diazepam, chlorpromazine, chlordiazepoxide, and dextroamphetamine, yielded unclear or inconsistent results. In some studies, the changes in drug dose-response relationship were subtle and not necessarily induced by the RFR. In most of the studies that yielded RFR-induced changes in drug response, whole-body SARs of 0.6 W/kg or average power densities of 1 mW/cm^2 or higher coupled with relatively high drug dosages were necessary. In still other studies, the results were negative (no effects). At relatively low RFR levels, the role of thermoregulation in the results is unclear. Also the occurrence of relatively high local SARs in the brain cannot be ruled out, a point applicable to the studies above by Lai et al., in which the whole-body SARs were only about 0.6 W/kg.

In general, it seems unlikely that the effects of psychoactive drugs prescribed by physicians or the effects of recreationally consumed alcohol would be altered by exposure to environmental levels of RFR.

Cellular and Subcellular Effects. Webb and Dodds[218] sought effects of RFR at specific frequencies above 30 GHz (in the millimeter-wave region) on growth of *E. coli* bacteria. Their results appeared to show that bacterial growth was inhibited by 136-GHz RFR, but from examination of their data, the presence of non-RFR factors was likely. Webb and Booth[219] also reported the absorption of RFR by *E. coli* cells and by preparations of protein, RNA, and DNA derived from *E. coli* at specific (resonant) frequencies within the range 65-75 GHz. The latter findings were difficult to evaluate because the information on methodology, instrumentation, and statistical treatment was inadequate.

Several investigations sought to confirm predictions by Fröhlich[220] of resonances above 30 GHz. For example, Webb and Stoneham[221] reported the detection of resonances in the range 70-5000 GHz in active cells of *E. coli* and B. megaterium, using laser Raman spectroscopy. The authors found no resonances in resting cells, cell homogenates, or nutrient solutions, and they therefore associated the observed active-cell resonances with metabolic processes. Cooper and Amer[222] disputed those findings. They indicated that cell suspensions yield spurious Raman lines in the frequency range of interest under certain conditions, notably by Mie scattering from cell clumps, and that they thereby were able to reproduce many of the spectra.

Gandhi et al.[223] used a stable computer-controlled system to measure RFR absorption in various biological specimens at discrete frequencies in the range 26.5-90.0 GHz in small steps. They studied solutions of DNA from salmon sperm and RNA from whole yeast and

yeast-like fungi, and suspensions of *E. coli* cells and baby-hamster-kidney cells transformed with mouse sarcoma virus. They found no resonances at any of the frequencies sampled, and they strongly suggested that none of the biological materials studied absorb significant RFR energy in that frequency range.

Swicord and Davis[224] used a novel method for measuring absorption by optically transparent liquids and for studying interactions between cellular constituents and RFR at frequencies below (as well as above) the millimeter-wave range. They measured RFR absorption in the range 8-12 GHz by aqueous solutions of DNA extracted from *E. coli*. A plot of attenuation coefficient for DNA versus frequency showed no resonances, but the attenuation increased linearly with frequency and its values were much higher than for physiologic (Ringer's) solution or deionized water at the same frequencies.

Edwards et al.[225] noted that biochemical analysis of the DNA solution used in the Swicord and Davis[224] study indicated the presence of significant amounts of RNA and protein impurities, and that the DNA had been sheared extensively by improper handling. In addition, the enhanced absorption observed for such samples in the range 8-12 GHz was absent for carefully prepared DNA samples of high molecular weight that were free of protein and RNA.

Gabriel et al.[226] described the efforts in two laboratories (London and Uppsala), that respectively used a reflection technique and a transmission technique to detect resonances in the range 1-10 GHz for aqueous solutions of circular DNA molecules of the form studied by Edwards et al.[225] Gabriel et al.[226] noted that a most important feature of both of those techniques is the use of a reference sample to normalize the reflection or transmission coefficients to eliminate systematic experimental artifacts, such as slight impedance mismatches. Their plots of relative permittivity and loss factor of a plasmid DNA solution versus frequency yielded values close to those for pure water. Moreover, similar plots of attenuation coefficient and incremental attenuation coefficient relative to water did not show any of the resonances reported by Edwards et al.[225]

Foster et al.[227] also tried to reproduce the findings of Edwards et al.[225] They suggested that the apparent resonances might be due to a reflection artifact from the coaxial connector to the probe used by Edwards et al.[225] for the measurements and to their lack of consideration of possible radiation from the probe. Accordingly, Foster et al.[227] carried out such measurements with and without the use of a time-domain-gating procedure for removing connector artifacts. Without using time-domain gating, they obtained reflection-coefficient oscillations crudely resembling the resonances reported by Edwards et al.[225] in DNA solutions of threefold higher concentration than the latter authors had used. Moreover, the oscillations were eliminated by time-domain gating. In addition, they saw no resonances when radiation from the probe was eliminated.

Sagripanti et al.[228] reported that plasmid DNA, when exposed to low-levels of RFR in the frequency range 2.00 to 8.75 GHz, exhibited both single-strand and double-strand breaks, but only if small quantities of copper ions (cuprous but not cupric) were present. The samples consisted of 10 μg of plasmid DNA in 28 μl of buffer within a 1.5-ml micro test tube. For exposure, a coaxial probe with both inner and outer conductors of copper was immersed in each sample. Attenuation and standing-wave ratio were measured, to determine the maximum and minimum SARs (SAR_{max} and SAR_{min}). Those data indicated that SAR_{max} was about five times larger than SAR_{min}, with SAR_{true} somewhere between the two. The experimental results were referenced to the values of SAR_{max}.

First, samples were sham-exposed or exposed for 20 minutes to 2.55-GHz RFR at an SAR_{max} of 10 W/kg. Those results showed that the mean number of double-strand breaks in the RFR-exposed samples was significantly higher than in the sham-exposed samples. The authors characterized such exposures as nonthermal, because of the large surface-to-volume ratio of the samples and their consequent ability to dissipate heat readily. They also

noted that exposures at levels of about 1 kW/kg were needed to detect any significant temperature rises in the samples.

Next, in experiments toward seeking frequency specificity of the effect, samples were exposed to 8.75 GHz-RFR, which was previously found by Edwards et al.[225] to be one of the frequencies of maximum resonant absorption by DNA. They also exposed samples to 2.00-GHz, 3.45-GHz, and 7.64-GHz RFR, which were frequencies of minimum absorption reported by Edwards et al.[225] Their results showed no variations in double-strand breaks attributable to resonant absorption by DNA.

For statistical analysis, the authors pooled data on 12 experiments at the five frequencies above. The results showed a significantly higher mean percentage of double-strand breaks for the RFR-exposed samples than the sham-exposed samples. However, the mean percentage of double-strand breaks for the sham-exposed samples was also significantly higher than for control samples, for which the copper probe was close to the sample but not in contact with it. When the probe was covered with a thin plastic coating, the difference between sham-exposed and control samples vanished, but also no strand breaks were detected in RFR-exposed samples.

In other experiments, samples were incubated in either cupric or cuprous chloride, or in the storage buffer (controls), and not RFR- or sham-exposed. The results indicated that only incubation in cuprous chloride mimicked the strand breaking seen with RFR-exposure. Based on linear increases of damage with exposure duration, the authors concluded that the presence of cuprous chloride caused the strand breaking and that the RFR increased the effect.

In summary, many of the early studies on microorganisms and subcellular preparations yielded results that were taken as evidence of nonthermal effects of RFR. The existence of resonances at frequencies above about 30 GHz was postulated on theoretical grounds, and several studies were done that appeared to confirm that hypothesis. However, subsequent studies with the use of more sophisticated engineering and biological techniques and in which artifacts were reduced markedly, yielded results that did not confirm earlier findings of resonances or other evidence of nonthermal effects at such frequencies.

Specifically, the apparent absorption resonances in the range 2-9 GHz reported for aqueous solutions of DNA molecules derived from *E. coli* were regarded as indicative of direct action of RFR with such molecules. Later endeavors to reproduce such findings, however, yielded negative results. In addition, analytical and experimental results were obtained indicating that such resonances were most likely artifactual, associated with the probes and measurement methodology used.

In general, research on possible RFR effects on microorganisms or of RFR-exposure *in vitro* of cell preparations derived from macroorganisms is important toward eliciting possible mechanisms of direct interaction of RFR with such biological entities or their constituents at levels that can be characterized as nonthermal. However, whatever the findings of such research, their relevance to possible effects of exposure of intact animals to RFR, and ultimately the significance of such findings with regard to possible hazards of RFR to humans would have to be established.

UNRESOLVED ISSUES

The potential biological effects of RFR at frequencies up to 300 GHz have been assessed from representative peer-reviewed studies published in the scientific literature.

The preponderance of evidence indicates that chronic exposure to the RFR levels generally prevailing in the environment is not hazardous to human health. Nevertheless, there are several basic uncertainties, summarized below, regarding biological effects of RFR.

(1) Many of the epidemiologic studies on possible bioeffects of RFR were extensive and well done, but contained defects or uncertainties in varying degrees, such as imprecise assignment of individuals to exposure and control groups; difficulties in obtaining accurate medical records, death certificates, or responses to health questionnaires for individuals included in both the exposure and control groups; and most important, the large uncertainties about the frequencies, levels, and exposure durations for those selected for inclusion in exposure groups and the amount of exposure received by those selected for inclusion in control groups.

(2) Applying results on laboratory animals to humans, though essential, is an expedient that contains fundamental problems and uncertainties due to the basic differences between humans and other species. Investigations with nonhuman primates may narrow some of the interspecies gaps considerably, but at costs that are often prohibitive. Thus, it seems unlikely that major reductions in such uncertainties will occur in the near future.

(3) The results of many studies indicate the existence of threshold RFR levels for various bioeffects, thus providing confidence that exposure to levels that are appreciably below the thresholds are most unlikely to be deleterious. However, most experimental data that indicate the existence of thresholds were obtained by the use of single or repetitive exposures of relatively short durations. Although it is hard to conceive of mechanisms whereby RFR-exposures at well below threshold values over a long time are cumulative, very few investigations have been done that involve essentially continuous exposure of animals to low-level RFR (below threshold levels or those that can cause significant heating) during most of their lifetimes. The high costs of such chronic studies and the low probability that any positive effects will be found are major reasons why such studies are not given high priority by funding agencies.

(4) Regarding basic mechanisms of interaction between RFR and various biological entities, many important discoveries have been made, notably by exposure of cells and subcellular structures and constituents *in vitro* to relatively low RFR levels. The effects on such entities can be characterized as subthermal, but the gap between such effects and possibly hazardous effects on intact humans or animals from exposure to such RFR levels is enormous. Factors such as large body masses, penetration depth and internal field distributions, and changes in body orientation during exposures to RFR *in vivo* can vastly moderate such interactions or remove them entirely. Moreover, life processes *per se* are extremely complex. For these reasons, this gap is not likely to be reduced to any great extent.

It is necessary to distinguish between a bioeffect and a hazard. For example, a person's metabolism can be increased harmlessly by mild exercise. Analogously, an effect produced at RFR intensities that yield heat that can be easily accommodated within the thermoregulatory capabilities of an individual may not necessarily be deleterious. Moreover, any effects produced thereby are generally reversible. However, the thermoregulatory capabilities of any given species may be exceeded at high RFR intensities, so compensation for such effects may be inadequate. Thus, exposure at such intensities can cause thermal distress or even irreversible thermal damage.

It is scientifically impossible to guarantee that low levels of RFR that do not cause deleterious effects for relatively short exposures will not cause the appearance of deleterious effects many years in the future. As indicated previously, however, the weight of the present scientific evidence indicates the existence of threshold levels for various RFR bioeffects and that low-level RFR-exposures are not cumulative.

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