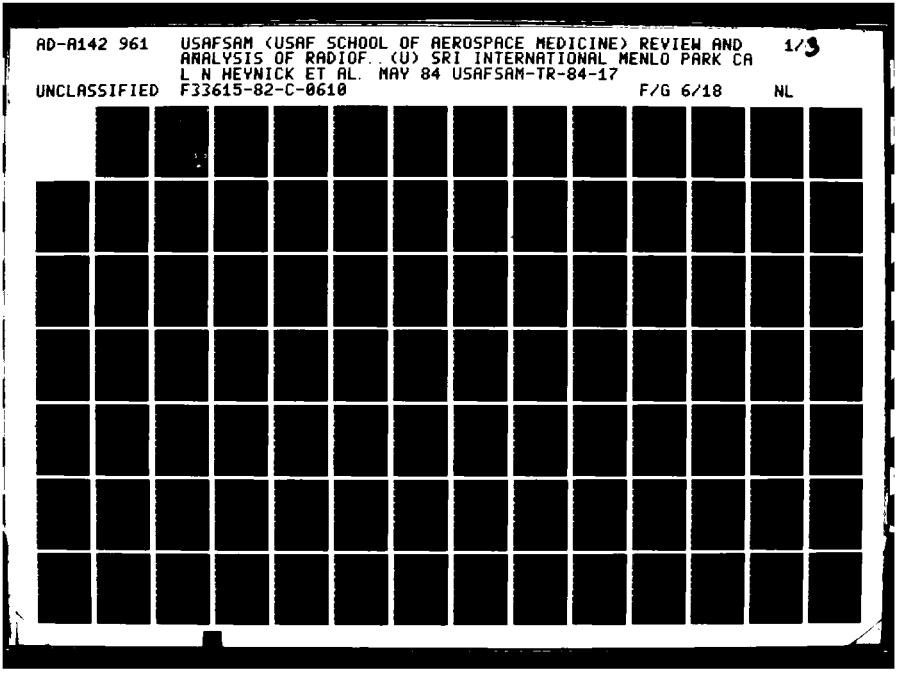
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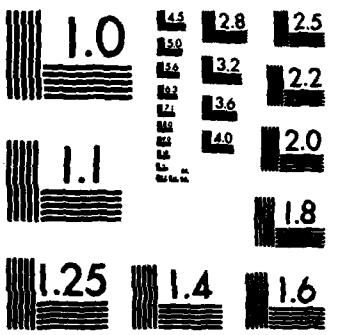

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Report USAFSAM-TR-84-17

USAFSAM REVIEW AND ANALYSIS OF RADIOFREQUENCY RADIATION BIOEFFECTS LITERATURE: FOURTH REPORT

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May 1984

Interim Report for Period 17 June 1983 - 16 March 1984

Prepared for
USAF SCHOOL OF AEROSPACE MEDICINE
Aerospace Medical Division (AFSC)
Brooks Air Force Base, Texas 78235

NOTICES

This interim report was submitted by SRI International, 333 Ravenswood Avenue, Menlo Park, California, under contract F33615-82-C-0610, job order 7757-01-87, with the USAF School of Aerospace Medicine, Aerospace Medical Division, AFSC, Brooks Air Force Base, Texas. James H. Merritt (USAFSAM/RZP) was the Laboratory Project Scientist-in-Charge.

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The Office of Public Affairs has reviewed this report, and it is releasable to the National Technical Information Service, where it will be available to the general public, including foreign nationals.

This report has been reviewed and is approved for publication.

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Nonionizing electromagnetic radiation; Radiofrequency radiation; Microwaves; and Biological effects.

19. ABSTRACT (Continue on reverse if necessary and identify by block number)
The objectives of this project are to acquire, review, and analyze on an ongoing basis, information on research pertaining to the biological effects of radiofrequency radiation (RFR) for the preparation of a computer data base of analyses at the USAF School of Aerospace Medicine (USAFSAM). The method in use is to: (1) select documents judged to be representative of prior and current research on various RFR-bioeffects topics, (2) analyze in detail the contents of each such document, and (3) assess the validity and significance of the results presented. In this fourth report, the major RFR-bioeffects topics are listed and the format used for analyzing each selected document is described. During the period covered by this report, 42 additional analyses were completed, for a total of 160 analyses. The texts of the additional analyses are presented in Appendix A. In addition to the text, each analysis includes information for computer retrieval by authors, key words, year of publication, and RFR parameters. Appendixes B and C are two cumulative indexes to reference citations for all of the analyses completed thus far. In Appendix B, each

20. DISTRIBUTION/AVAILABILITY OF ABSTRACT UNCLASSIFIED/UNLIMITED ☒ SAME AS RPT. ☐ DTIC USERS ☐	21. ABSTRACT SECURITY CLASSIFICATION UNCLASSIFIED
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20. ABSTRACT (Continued)

citation is listed under each pertinent major topic. Appendix C comprises a cumulative list of citations in alphabetical order by first author and without regard to topic.

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USAFSAM REVIEW AND ANALYSIS OF RADIOFREQUENCY RADIATION BIOEFFECTS LITERATURE: FOURTH REPORT

INTRODUCTION

The objectives of this project are to acquire, review, and analyze, on an ongoing basis, information on research pertaining to the biological effects of radiofrequency radiation (RFR), and to provide periodic technical reports of our findings and assessments to the USAF School of Aerospace Medicine (USAFSAM) in specified formats.

The first technical report was for the period from 1 March through 31 August 1980 and was issued as Report SAM-TR-81-24 (November 1981). The second technical report was for the period from 1 September 1980 through 30 June 1981 and was issued as Report SAM-TR-82-16 (May 1982). These two reports were prepared on Contract Number F33615-80-C-0608 (SRI Project 1485). The work is continuing on Contract Number F33615-82-C-0610 (SRI Project 4472), under which the third report, for the period from 17 May 1982 through 16 June 1983, was issued as Report USAFSAM-TR-84-6 (March 1984). The present (fourth) report is for the period from 17 June 1983 through 16 March 1984.

METHODOLOGY

Thousands of scientific papers, reports, books, summaries, and abstracts (referred to collectively herein as "documents") have been published on the bioeffects of RFR and related fields. Because references to most of these documents are readily available through various abstracting services and data bases, we are endeavoring to avoid needless duplication of such services and information. Instead, we are selecting documents judged to be representative of prior and current research on various RFR-bioeffects topics, analyzing the contents of each such document in detail, and assessing the validity and the significance of the results presented.

A major aspect of the project is to prepare the analysis of each selected document in a format that permits easy storage of the information in a computer at USAFSAM and retrieval by any of a variety of designators: analysis number, major bioeffects topic, author(s), year of publication, frequency, power density, modulation, duty cycle, specific absorption rate (SAR), species, and a list of special key words. The software for such retrieval is being developed by USAFSAM.

The analyses in the first two technical reports were printed with an optical-character-recognition (OCR-B) font, to permit direct storage of the information without retyping. This practice was found to be unsatisfactory and essentially redundant, so it was discontinued for the third and subsequent reports. Inclusion of International Standard Serial Numbers (ISSNs) in the citations was also discontinued. In addition, the analysis format was modified to conform with USAFSAM's retrieval software. The outline form now in use for analyses is displayed in Figure 1.

Analysis number
Authors
Title
Citation

*

Author abstract (or reviewer summary)

**

Study type (bioeffects topic; in vivo/in vitro; species)
Effect type
Frequency
Modulation
Power density
SAR

Exposure conditions

Other information

Critique

References

***

[Retrieval information (one entry per line):]
Authors (last names only)

/

Key words

//

Year of publication
Frequency--value or range in MHz (0=unknown)
Duty cycle--value or range (CW=1; 0=unknown)
Power density--average value or range in mW/sq cm (0=unknown)
SAR--average value or range in W/kg (0=unknown)

///

Figure 1. Outline form for analyses.

To conform with the modified format, numbers 1 through 80 were assigned serially to the analyses in the first two reports, and the sequence was continued in the third and present reports. The authors, title (in upper case), and reference are given immediately after the analysis number. The single asterisk after the citation is a flag to permit retrieval of only the citation part of the analysis, if desired.

As part of each analysis, the abstract or summary provided by the authors is reproduced without comment directly after the citation (a change from the previous outline) and the heading "AUTHOR ABSTRACT" or "AUTHOR SUMMARY" is used. If the document does not contain an abstract or summary, its important contents are summarized without comment, and the heading "REVIEWER SUMMARY" is used to indicate this fact. The two asterisks following the abstract or summary comprise a flag to permit retrieval of only the citation and abstract or summary, if desired.

Next, for each document reviewed, one or more pertinent major topics are listed under "Study type." To conform better with current usage, the list of topics was modified as shown in Figure 2, beginning with the third report. Also indicated under this heading are whether the study was done in vivo or in vitro and the species involved.

Auditory Effects
Behavior
Biorhythms
Cardiovascular Effects
Cellular and Subcellular Effects
Endocrinology
Environmental Factors
Exposure Methods, Dosimetry, and Modeling
Human Studies
Immunology and Hematology
Mechanisms of Interaction
Medical Applications
Metabolism and Thermoregulation
Multiagent Interactions
Mutagenesis, Carcinogenesis, and Cytogenetic Effects
Nervous System
Ocular Effects
Physiology and Biochemistry
Teratology and Developmental Abnormalities

Figure 2. Type of study.

Under the heading "Effect type," the specific effects, phenomena, biological endpoints, or other characteristics sought or studied are listed briefly. The frequencies, modulation characteristics (CW, amplitude-modulation, or pulse parameters), power densities, and SARs are given under their respective headings.

In the next section, "EXPOSURE CONDITIONS," the salient features of the exposure arrangements and parameters are briefly summarized.

Under "OTHER INFORMATION," any important information in the text of the document that was not included in the author abstract or summary or that is not appropriate for the reviewer summmary is summarized, again without comment.

Our analysis of the document is given under "CRITIQUE." To the extent possible or appropriate, each critique includes evaluation of the data presented (including the statistical aspects if the data presented are adequate), the biological and engineering methodology used, the validity of the results, how the findings compare with those of other studies, and the significance of the findings with respect to the health of humans (and/or other species) exposed to RFR.

It should be noted that critiques are no longer labeled "INITIAL" or "FINAL," in consonance with the view that any critique should be subject to possible revision, e.g., updates based on subsequent information or comments from the authors of the document. Concerning the latter point, written comments from authors or others regarding any analysis are welcome and will be treated as addenda to the critique thereof.

Any literature citations mentioned in the analysis are shown under "REFERENCES." The three asterisks after the references section mark the end of the analysis proper and comprise a flag to permit retrieval of the full text of the analysis proper.

The following items are solely for retrieval of the analysis by any of various designators. Listed first are the last names individually of all the authors of the document, followed by a single slant sign (/) to indicate the end of this form of designator. The next set of designators are key words derived from the analysis of the document. Such key words are not necessarily those provided by the authors, but are from a list designed expressly for USAFSAM retrieval use. The current list is displayed in Figure 3. It includes three additions, "EKG," "FROG," and "TURTLE" to the previous list. Other additions to the list may be made as appropriate. This designator section is terminated with two slant lines (//).

ANTIGEN
ANTIBODY
AUDITORY
BACTERIA
BEHAVIOR
BIOCHEMISTRY
BIORHYTHM
BBB
BRAIN-UPTAKE-INDEX
CALCIUM
CARCINOGENIC
CARDIOVASCULAR
CAT
CELLULAR
CHICKEN
CHINCHILLA
CHRONIC
CIRCADIAN
COMPLEMENT
CORTICOSTEROID
CW
CYTOGENETIC
DEVELOPMENT
DIELECTRIC
DOG
DOSIMETRY
DROSOPHILA
DRUG-RFR
E-COLI
ECOLOGICAL
EEG
EFFLUX
EKG
EMBRYO
ENDOCRINOLOGIC
ENVIRONMENTAL
EPIDEMIOLOGIC
ESTRUS
EVOKED-POTENTIAL
EXPOSURE-SYSTEM
FROG
GUINEA-PIG
HAMSTER
HAPLOTYPE
HEMATOLOGY
HISTOLOGY
HUMAN
HYPERTHERMIA
HYPOTHERMIA
IMMUNOLOGY
INFLAMMATION
INSTRUMENTATION
IN-VITRO
IN-VIVO
LETHALITY
LEUKOCYTE
LYMPHOCYTE
MECHANISMS
MEDICAL
METABOLISM
MICROSCOPY
MITOGEN
MODEL
MODULATED
MONKEY
MORBIDITY
MORTALITY
MOUSE
MULTIAGENT
MUTAGENIC
NERVOUS-SYSTEM
OCCUPATIONAL
OCULAR
PHYSIOLOGY
POSITIVE-CONTROL
PRIMATE
PULSED
QUAIL
RABBIT
RAT
RECTAL
REPEATED-ACQUISITION
REVIEW
RFR
STRESS
TENEBRIO
TERATOGENIC
THERMOREGULATION
THRESHOLD
TRACER
TURTLE
WEIGHT
YEAST

Figure 3. List of key words.

In the final section of designators, the following numerical information is presented in sequence: the year of publication of the document; the frequency or frequency range of the RFR in MHz; the duty cycle or range thereof, with "1" representing continuous-wave (CW) RFR or amplitude-modulated RFR when appropriate; the average power density or its range; and the average SAR or its range. (Peak power density is not included as a designator because values thereof can be calculated from duty cycles and average power densities.) The symbol "0" is used to signify "unknown" or "not specified". This designator section is terminated with three slant lines (///), which also indicate the end of the entire analysis, including the retrieval information.

The analyses in Appendix A to this report illustrate this methodology.

PROGRESS DURING THIS PERIOD

By the end of this period, 42 additional analyses were completed, for a total of 160 in the four reports. The texts of these 42 analyses (including the retrieval data) are presented in sequence by analysis number in Appendix A.

Two cumulative indexes to reference citations for all of the analyses completed thus far are included in this report. In Appendix B, each citation is listed under each of the pertinent major topics (selected from Figure 2) shown under "Study Type" in the analysis. Appendix C comprises a cumulative list of citations, in alphabetical order by first author and without regard to topic, for the analyses completed. For ease in finding the text of any analysis from either list, the four reports are referred to by Roman numerals in chronological succession, and the end of each reference citation in each cumulative list is annotated with the Roman numeral of the report containing the text of the analysis, followed by the first page number of the text. This indexing method is illustrated below:

87
Galvin, M.J., D.I. McRee, and M. Lieberman
EFFECTS OF 2.45-GHZ MICROWAVE RADIATION ON EMBRYONIC QUAIL HEARTS
Bioelectromagnetics, Vol. 1, No. 4, pp. 389-396 (1980) (III-35)

This citation is listed in Appendix B under the topic headings Cardiovascular Effects, Physiology and Biochemistry, and Teratology and Developmental Abnormalities, and is shown under "Galvin..." in Appendix C. The annotation "III-35" indicates that the analysis of this document can be found on page 35 of the third report.

PROPOSED PLANS FOR THE FUTURE

Preparation of detailed analyses of important documents on biological effects of RFR will continue, to augment the data base produced thus far under this project.

SRI had prepared a review of the literature entitled "Bioeffects of Radiofrequency Radiation: A Review Pertinent to Air Force Operations," by L.N. Heynick and P. Polson, which was issued as USAFSAM Report SAM-TR-83-1 (March 1983). A revision of that review, to include analyses of publications and other pertinent information that postdate the issuance of the review, will be started during the forthcoming period.

ACKNOWLEDGMENTS

The contributions of Barrett P. Eynon, Statistician (par excellence), Data Design and Analysis Department of SRI's Health and Social Systems Division, to this project are much appreciated. We thank Judith Bull, Senior Library Assistant, for her efforts in obtaining copies of not readily available documents. The guidance furnished by Jacqueline Bremer, Administrative Assistant, in the techniques of word processing is also appreciated very much.

APPENDIX A

TEXTS OF ANALYSES COMPLETED DURING THE FOURTH PERIOD

119
Sultan, M.F., C.A. Cain, and W.A.F. Tomkins
EFFECTS OF MICROWAVES AND HYPERTHERMIA ON CAPPING OF ANTIGEN-ANTIBODY COMPLEXES ON THE SURFACE OF NORMAL MOUSE B LYMPHOCYTES
Bioelectromagnetics, Vol. 4, No. 2, pp. 115-122 (1983a)

*

AUTHOR ABSTRACT: Normal mouse B lymphocytes were tested for the ability to cap plasma antigen-antibody complexes following exposure to 2.45-GHz continuous wave (CW) microwaves at power densities up to 100 mW/sq cm (45 W/kg specific absorption rate), at 37, 41, and 42.5 deg C. After a 30-minute treatment, the irradiated cells and the nonirradiated controls were tested for capping by the direct immunofluorescence technique. First, the cells were incubated for nine minutes at 37 deg C with fluorescein isothiocyanate-conjugated goat anti-mouse immunoglobulin. After fixing and washing, the percentage of capped cells was determined under a fluorescence microscope.

The results show that for the nonirradiated controls, capping is reduced from 90% at 37 deg C, to 52% at 41 deg C, to less than 5% for cells that were pretreated at 42.5 deg C. There was no significant difference between the microwave-treated cells and the controls when both were maintained at the same temperature. In another experiment, there was no significant difference in the percentage of capping between controls and cells that were exposed to microwave radiation during capping, when the temperature in both preparations was kept at 38.5 deg C. The results demonstrate that B-lymphocyte capping is sensitive to temperature in the range that is proposed for use in tumor therapy.

**

Study Type: Immunology and Hematology; IN VITRO; MOUSE
Effect Type: RFR- and temperature effects on capping of antigen-antibody complexes on B lymphocytes
Frequency: 2.45 GHz
Modulation: CW
Power Density: 5, 10, 25, 50, or 100 mW/sq cm
SAR: 2.25, 4.5, 11.25, 22.5 or 45 W/kg

EXPOSURE CONDITIONS: Aliquots of mouse-spleen-lymphocyte suspensions in cellulose-nitrate test tubes were exposed to RFR in groups of 3 tubes spaced 6 cm apart transverse to the RFR in an anechoic chamber. Control suspensions outside the chamber were maintained at the same temperature (to within 0.1 deg C) as the RFR-exposed suspensions by a common temperature-controlled saline flow system. Both RFR- and control tubes were mechanically agitated transversely at 75 cycles/min by the same shaker. RFR exposures were done for 30 min at a temperature of 37, 41, or 42.5 deg C.

OTHER INFORMATION: "Each B lymphocyte expresses a homogeneous set of membrane-bound immunoglobulin (Ig) molecules with a single specificity

for antigen. Upon binding of antigen to surface Ig, B cells proliferate and differentiate into plasma cells, which produce antigen-specific antibodies. Initially, membrane-bound Ig molecules are diffusely scattered over the entire surface of the B cell. Binding of anti-Ig initiates a redistribution of the antigen-antibody (Ag-Ab) complexes on the cell surface. The redistribution begins with a regrouping of the complexes into patches. Patching is followed by capping of the complexes wherein all patches coalesce into a polar cap, and the remaining part of the plasma membrane is devoid of Ig-anti-Ig complexes". The objective of this investigation was to study the effects of combined in-vitro RFR-exposure and hyperthermia on the ability of normal B lymphocytes to cap surface Ig following binding of specific anti-Ig molecules.

For SAR measurements, suspensions in tubes thermally isolated from the saline bath were exposed to RFR for 15, 30, and 60 s, and their temperature rises were noted. The slopes of the heating curves yielded 0.45 W/kg per mW/sq cm.

Suspensions of splenic B lymphocytes were derived from mice 4-8 weeks old killed by cervical dislocation. Before the RFR experiments, the rate of capping at 37 deg C was determined following heat treatment for 30 min at 37, 41, and 42.5 deg C. Viability after such treatments, determined by trypan-blue exclusion, was better than 90%. Immediately after treatment, the cells were washed in cold phosphate-buffered saline (PBS) and centrifuged. They were then incubated at 4 deg C for 10 min with fluorescein-isothiocyanate-labeled goat anti-mouse immunoglobulin (FITC-anti-Ig) to permit antibody binding to surface Ig, and transferred to a 37-deg-C environment to allow for capping. After 2, 4, 8, or 12 min at 37 deg C, the reaction was stopped by paraformaldehyde fixation, and 200 fluorescent cells (Ig+ cells) of each sample were randomly selected and scored (under a fluorescence microscope) for capping. It was found that after the samples were transferred from incubation at 4 deg C to the 37 deg C bath, they reached equilibrium at 37 deg C within 1 min, and that completion of capping occurred in less than 4 min for those cells having surface-Ig capping ability. The results were that more than 90% of the cells heat-treated at 37 deg C showed capping, whereas less than 60% of those treated at 41 deg C and less than 5% of those treated at 42.5 deg C exhibited capping.

Cell suspensions were exposed to 2.45-GHz CW RFR for 30 min at 5, 10, 25, 50, or 100 mW/sq cm concurrently with control suspensions while the suspension temperature was maintained constant at 37, 41, or 42.5 deg C by the saline flow system. Following exposure, RFR-exposed and control samples were incubated with FITC-anti-Ig at 37 deg C for 9 min and tested for capping as described above. The results were similar to those above: 90% of the RFR-exposed and control cells treated at 37 deg C exhibited capping; 52% of the cells treated at 41 deg C and less than 5% of those treated at 42.5 deg C showed capping. There was no dependence on power density and there were no statistically significant differences between corresponding results for RFR-exposed and control samples.

In another experiment, the possibility of direct RFR action on capping was investigated. The incubation with FITC-anti-Ig was done at 4 deg C for 10 min to produce antibody binding to surface Ig without capping (instead of at 37 deg C for 9 min). Samples were then immediately transferred to the exposure chamber and control bath, both held at 38.5 deg C. After 10 min of treatment, capping was again about 90% for RFR-exposed and control samples, with no dependence on power density and no significant differences between samples.

The investigators concluded that the mechanisms responsible for inhibition of capping in their experiments are thermal in origin, with no apparent effects of 2.45-GHz CW-RFR exposure if RFR-exposed and control samples are held at the same temperature.

CRITIQUE: The biological and engineering aspects of this investigation appear to be straightforward and carefully performed, and the results unequivocal. If positive results had been obtained, it would have been important to perform sham-exposures with concurrent controls. Perhaps the only obscure point is why the investigators would expect that the second RFR-exposure experiments would yield results basically different from those in their first RFR-exposure experiments. The point is that in their initial hyperthermia experiments (without RFR), they found that their samples reached temperature equilibrium with the saline bath within 1 min and that capping was completed in less than 4 min, both well within the 10-min exposures used in the second set of experiments. However, this point does not alter their conclusion.

It should be noted that these investigators also studied the effects of 147-MHz RFR amplitude-modulated at 9, 16, and 60 Hz on B-cell capping (Sultan et al., 1983b) and again obtained negative results.

REFERENCES:

Sultan, M.F., C.A. Cain, and W.A.F. Tomkins
IMMUNOLOGICAL EFFECTS OF AMPLITUDE-MODULATED RADIO FREQUENCY RADIATION: B LYMPHOCYTE CAPPING
Bioelectromagnetics, Vol. 4, No. 2, pp. 157-165 (1983b)

***

SULTAN
CAIN
TOMKINS

/

ANTIGEN
ANTIBODY
CANCER
CELLULAR
CW
HYPERTHERMIA
IMMUNOLOGY
IN-VITRO
LEUKOCYTE
LYMPHOCYTE
MOUSE
RFR

//

1983
2450
1
5-100
2.25-45

///

120
Sultan, M.F., C.A. Cain, and W.A.F. Tomkins
IMMUNOLOGICAL EFFECTS OF AMPLITUDE-MODULATED RADIO FREQUENCY RADIATION: B LYMPHOCYTE CAPPING
Bioelectromagnetics, Vol. 4, No. 2, pp. 157-165 (1983b)

*

AUTHOR ABSTRACT: B lymphocytes collected from normal ICR Swiss mouse spleens were exposed in vitro in a Crawford cell to 147-MHz radiofrequency (RF) radiation, amplitude modulated by a 9-, 16-, or 60-Hz sine wave. The power densities ranged between 0.11 and 48 mW/sq cm. The irradiated samples and the controls were maintained at 37 deg C or 42 deg C, with temperature variations less than 0.1 deg C. Immediately after a 30-minute exposure, the distribution of antigen-antibody (Ag-Ab) complexes on the cell surface was evaluated at 37 deg C by immunofluorescence. Under normal conditions (37 deg C, no RF), Ag-Ab complexes are regrouped into a polar cap by an energy-dependent process.

Our results demonstrate that the irradiated cells and the nonirradiated controls capped Ag-Ab complexes equally well after exposure at 37 deg C. Capping was equally inhibited at 42 deg C in both the controls and irradiated cells. No statistically significant differences in capping were observed between the RF-exposed and control samples at any of the modulation frequencies and power densities employed as long as both preparations were maintained at the same temperature.

**

Study Type: Immunology and Hematology; IN VITRO; MOUSE
Effect Type: Effects of amplitude-modulated RFR on capping of antigen-antibody complexes on B lymphocytes
Frequency: 147 MHz
Modulation: 9, 16, or 60 Hz (90%)
Power Density: 0.11 to 48 mW/sq cm
SAR: 0.042 W/kg per mW/sq cm max

EXPOSURE CONDITIONS: Aliquots of mouse-spleen-lymphocyte suspensions in thin-walled cellulose-nitrate test tubes were exposed to RFR in pairs spaced 6.4 cm apart within a transverse electromagnetic (TEM) cell. A pair of control suspensions outside the TEM cell was maintained at the same temperature (to within 0.1 deg C) as the RFR-exposed pair by a common temperature-controlled saline flow system. The RFR- and control tubes were mechanically agitated transversely to the propagation direction at 1.25 Hz by the same shaker. RFR exposures were done for 30 min at a temperature of 37 or 42 deg C.

OTHER INFORMATION: "One particular event that is altered by a change of intracellular concentration is the redistribution and capping of antigen-antibody (Ag-Ab) complexes on the surface of B lymphocytes following the binding of antibody molecules to surface immunoglobulins.

Capping is an active process in which membrane-bound Ag-Ab complexes coalesce into a polar cap on the cell surface. The capping of Ag-Ab complexes has been well characterized on the surface of B lymphocytes [Schreiner and Unanue, 1976]. Schreiner and Unanue reported that the introduction of a calcium ionophore into the plasma membrane of B lymphocytes completely suppress capping. If the ionophore-mediated calcium influx occurs after cap formation, the cap is completely disrupted by a metabolically active process that involves the lymphocyte's cytoskeletal system. These observations and other considerations led Schreiner and Unanue to suggest a model for capping whereby a calcium-dependent bond between antigen receptors and calcium-responsive cytoplasmic microfilaments effects the transport of the receptors through the plane of the membrane without affecting other components of the membrane."

This investigation was directed toward testing the hypothesis that RFR amplitude-modulated at frequencies associated with the electroencephalogram can induce changes in intracellular calcium concentrations in B lymphocytes, and thereby affect the capping of membrane-bound Ag-Ab complexes. The carrier frequency (147 MHz) and two of the modulation frequencies (9, 16 Hz) were selected to correspond with those reported by Bawin et al. (1975) to enhance calcium-ion efflux from brain tissue exposed in vitro.

Determinations of SAR from measurements of forward, reflected, and transmitted powers proved difficult because the quantities of energy absorbed by the suspensions at 147 MHz were within the standard deviations of such measurements. The maximum standard deviation per se was used as an estimate that the upper bound of SAR was 0.042 W/kg per mW/sq cm. Confirmation of this upper limit was obtained by thermally isolating suspension tubes from the saline bath, exposing the tubes at 1 kW for 60 s, and measuring suspension-temperature rises using a thermocouple with its leads perpendicular to the E-field. This technique yielded an upper bound of 0.035 W/kg per mW/sq cm.

Suspensions of splenic B lymphocytes were derived from mice 12-16 weeks old killed by cervical dislocation. After preparation, pairs of suspensions were exposed to modulated RFR for 30 min at power densities from 0.1 to 48 mW/sq cm concurrently with pairs of control suspensions while the suspension temperature was maintained constant at 37 or 42 deg C by the saline flow system. Following exposure, RFR-exposed and control samples were incubated at 4 deg C for 5 min with fluorescein-isothiocyanate-labeled goat antimouse-immunoglobulin (FITC-anti-Ig) to permit antibody binding to surface Ig, and transferred to a 37-deg-C water bath to allow for capping. After 9 min at this temperature, the cells were fixed with paraformaldehyde, and 200 fluorescent cells of each sample were randomly selected and scored (under a fluorescent microscope) for capping. The results showed that more than 90% of the RFR-exposed and control cells treated at 37 deg C exhibited capping, whereas less than 10% of those treated at 42 deg C showed capping. There was no dependence of capping on modulation frequency or power density and there were no statistically significant differences between corresponding results for RFR-exposed and control samples.

The investigators concluded that these results do not provide any support for the hypothesis that amplitude-modulated RFR alters the capping ability of B lymphocytes. However, the inhibition of capping at elevated temperature observed in these experiments is consistent with prior results without RFR (Sultan et al., 1983a).

CRITIQUE: Regarding the dosimetry, the investigators obtained an upper SAR limit of about 0.04 W/kg per mW/sq cm at 147 MHz and a value of 0.45 W/kg per mW/sq cm at 2.45 GHz (Sultan et al., 1983a). Qualitatively, these values are consonant with the dependence of SAR on the square of the frequency below resonance (Durney et al., 1978).

The negative results of this investigation supplement those obtained in a similar study with unmodulated 2.45-GHz RFR (Sultan et al., 1983a) and support the conclusion that modulated RFR has no apparent effect on B-lymphocyte membranes as determined by alterations of their capping ability.

As stated by the investigators, the carrier frequency used (147 MHz) and the modulation frequencies and range of incident power densities were selected to span those for which Bawin et al. (1975) reported maximum calcium-efflux increase from chick brains. However, the relevance of this work on lymphocytes to that of Bawin et al. is questionable in view of the differences of cell types studied and the widely different biological methodologies used in processing them.

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EFFECTS OF MICROWAVES AND HYPERTHERMIA ON CAPPING OF ANTIGEN-ANTIBODY COMPLEXES ON THE SURFACE OF NORMAL MOUSE B LYMPHOCYTES
Bioelectromagnetics, Vol. 4, No. 2, pp. 115-122 (1983a)

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Smialowicz, R.J., K.L. Compton, M.M. Riddle, R.R. Rogers, and P.L. Brugnolotti
MICROWAVE RADIATION (2450 MHZ) ALTERS THE ENDOTOXIN-INDUCED HYPOTHERMIC RESPONSE OF RATS
Bioelectromagnetics, Vol. 1, No. 4, pp. 353-361 (1980)

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AUTHOR ABSTRACT: The parenteral administration of bacterial endotoxin to rats causes a hypothermia that is maximal after approximately 90 minutes. When endotoxin-injected rats were held in a controlled environment at 22 deg C and 50% relative humidity and exposed for 90 minutes to microwaves (2450 MHz, CW) at 1 mW/sq cm, significant increases were observed in body temperature compared with endotoxin-treated, sham-irradiated rats. The magnitude of the response was related to power density (10 mW/sq cm > 5 mW/sq cm > 1 mW/sq cm). Saline-injected rats exposed for 90 minutes at 5 mW/sq cm (specific absorption rate approximately 1.0 mW/g) showed no significant increase in body temperature compared with saline-injected, sham-irradiated rats. The hypothermia induced by endotoxin in rats was also found to be affected by ambient temperature alone. Increases in ambient temperature above 22 deg C in the absence of microwaves caused a concomitant increase in body temperature.

This study reveals that subtle microwave heating is detectable in endotoxin-treated rats that have an impaired thermoregulatory capability. These results indicate that the interpretation of microwave-induced biological effects observed in animals at comparable rates and levels of energy absorption should include a consideration of the thermogenic potential of microwaves.

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Study Type: Endocrinology, Physiology and Biochemistry, Metabolism and Thermoregulation, Multiagent Interactions; IN VIVO; RAT
Effect Type: RFR-induced body-temperature increases in rats rendered hypothermic by endotoxin injection
Frequency: 2.45 GHz
Modulation: CW (11% ripple)
Power Density: 1, 5, or 10 mW/sq cm
SAR: 0.2, 1.0, or 2.0 W/kg

EXPOSURE CONDITIONS: Rats in individual vented acrylic restrainers were sham-exposed or exposed for 90 min to far-field RFR in groups of 4 arranged in a diamond pattern within an anechoic chamber, with their long axes parallel to the H-vector and a spacing of 19 cm between adjacent rats. The chamber was maintained at 22 deg C, 50% relative humidity, and 0.31 m/s air flow.

OTHER INFORMATION: In the first experiment, rats were injected with 1 of 3 doses of Salmonella typhimurium W lipopolysaccharide (LPS), to

determine the effect of dose on the time course of endotoxin-induced hypothermia. Following injection, colonic temperatures were monitored continuously for 2.5 hr and recorded every 30 min in a controlled environment. For controls, other rats were injected with saline. Colonic-temperature depression was LPS-dose-dependent, and maximal hypothermia was attained at about 90 min for all 3 doses. The controls showed no hypothermia.

In the second experiment, groups of 4 rats were injected with the largest of the 3 LPS doses or saline and were sham-exposed or exposed at 10, 5, or 1 mW/sq cm (SAR of 2.0, 1.0, or 0.2 W/kg) for 90 min, after which time their colonic temperatures were measured. The mean colonic temperatures of the LPS-injected rats were lower than those of the saline-injected rats at corresponding power densities, with the greatest difference at 0 mW/sq cm. The values for the LPS-injected rats exposed to the 3 RFR levels were significantly larger than for the sham-exposed LPS-injected rats. Also, the changes in temperature (temperature at 90 min minus temperature immediately after LPS injection) for 0, 1, 5, and 10 mW/sq cm were -1.6, -0.2, -0.5, and +0.1 deg C, respectively, and were statistically significant.

The third experiment was designed to examine the effect of procedural acclimatization on RFR response. Six groups of 4 rats were handled in a manner similar to that required for LPS injection. The rats were then placed in restrainers, their colonic temperatures were measured before and after 90 min in a controlled environment at 22 deg C and 50% relative humidity, and the rats were returned to their home cages. This acclimatization procedure was performed each day for 2 weeks. The rats were then injected with LPS and exposed in groups of 4 at 0.5 mW/sq cm concurrently with sham-exposed groups for 90 min. The results showed that the mean final colonic temperature of the RFR-exposed rats was nonsignificantly higher than for the sham-exposed rats. However, the mean initial temperature (after LPS injection but prior to RFR- or sham-exposure) was significantly lower for the RFR groups than for the sham groups, thus yielding a statistically significant difference in temperature depression between RFR- and sham-exposed rats.

In the fourth experiment, LPS- and saline-injected rats were maintained at an ambient temperature of 18, 22, 26, 30, or 34 deg C (without RFR exposure) while their colonic temperatures were monitored for 2.5 hr. Only mean colonic temperatures after 90 min in these ambients were presented.

For the saline-injected rats, the values rose only from about 37.5 to 38.4 deg C for the ambient range 18 to 34 deg C, whereas the corresponding values for the LPS-injected rats were about 34.9 to 39.0 deg C. The mean colonic temperature for the saline-injected rats at 22 deg C was about 37.5 deg C, approximately the same as for the saline-injected sham-exposed rats and the LPS-injected rats exposed at 10 mW/sq cm. At this power density, the colonic temperature of the LPS-injected rats fell between that of the LPS-injected rats held at 26 and 30 deg C ambient, which delimit the thermoneutrality zone for the rat. Above 30

deg C, a febrile response to endotoxin by rats occurs. Consequently, above 10 mW/sq cm or 30 deg C, core-temperature increases would result from the rat's pyrogenic response to endotoxin as well as from the increased thermal load from RFR or elevated ambient temperature. Based on these results, the investigators concluded that the RFR produced generalized body heating rather than direct interaction with the nervous system.

In their discussion, the investigators indicated that endotoxin injection in the rat, unlike other species, produces hypothermia, and that the alteration of its thermoregulatory capacity allows the rat to respond to ambient-temperature changes much like ectotherms. For this reason, significant core-temperature increases in rats rendered hypothermic by this means may be detected at lower power densities than in rats not so treated, i.e., the technique can be used in whole-body SAR dosimetry of the rat (Putthoff et al., 1977).

CRITIQUE: The results of this investigation indicate that relatively low RFR power densities or SARs (of the order of 1 mW/sq cm or 0.2 W/kg) are absorbed in the rat as heat that is not manifested as an increase in colonic temperature in normothermic animals but that can be detected in rats rendered hypothermic by endotoxin injection. These findings support the contention that RFR interaction at such RFR levels is primarily thermal.

Putthoff et al. (1977) obtained similar responses in rats rendered hypothermic by cortisone injection, but at higher SARs (about 40 W/kg).

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Radio Sci., Vol. 12, No. 6S, pp. 73-80 (1977)

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Lu, S.-T., N. Lebda, S. Pettit, and S.M. Michaelson
DELINEATING ACUTE NEUROENDOCRINE RESPONSES IN MICROWAVE-EXPOSED RATS
J. Appl. Physiol.: Respiratory, Environmental, and Exercise Physiol., Vol. 48, No. 6, pp. 927-932 (1980b)

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AUTHOR ABSTRACT: One hundred and eighteen male rats (Long-Evans) were acclimated to experimental procedures (i.e., handling, transferring from and back to "home" cage, body weight and colonic temperature determinations) for 2 wk and then subjected to cage confinement for 3 days before microwave (MW) exposure to 2,450 MHz for 1-8 h, at 1-70 mW/sq cm or sham exposure at ambient temperature of 24 +/- 1 deg C.

Colonic temperature increased after exposure to power densities equal to or greater than 20 mW/sq cm and was the most sensitive parameter measured. Inverse relationships between corticosterone and thyrotropin or growth hormone were noted after exposure for 1 h at 50 mW/sq cm and above. Pituitary-thyroid function was inhibited after exposure to 20 mW/sq cm for 2-8 h. Changes in other hormones were transient or inconsistent.

Corticosterone, thyrotropin, and growth hormone levels could be correlated with power density or colonic temperature in rats exposed to MW for 1 h; corticosterone and thyrotropin levels correlated with colonic temperatures in shams. Body temperature influences adenohypophysial hormones in studies of MW biological effects.

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Study Type: Endocrinology, Physiology and Biochemistry; IN VIVO; RAT
Effect Type: RFR-induced effects on hormonal levels in rats acclimated to the experimental situation prior to exposure
Frequency: 2.45 GHz
Modulation: Amplitude-modulated at 120 Hz
Power Density: 1-70 mW/sq cm
SAR: 0.21 W/kg per mW/sq cm

EXPOSURE CONDITIONS: After they were acclimated, rats in individual Styrofoam cages were sham-exposed or exposed concurrently in groups of 4 to far-field RFR at a power density in the range 1-70 mW/sq cm for 1, 2, 4, or 8 hr in an anechoic chamber maintained at 24 deg C.

OTHER INFORMATION: The procedure for acclimating rats involved the following: For 2 weeks, the rats were removed daily from their home cages, their colonic temperatures and body weights were measured, and they were returned to their home cages. On Mon, Tue, and Wed of the third week, after body weight and colonic temperature were measured, the rats were transferred to exposure cages for a 3-hr equilibration period and sham-exposure session. Following sham exposure, their body weights and colonic temperatures were measured again and the rats were returned to their home cages.

On Thurs of the third week, following measurements of colonic temperature (Tco) and body weight and a 3-hr equilibration period, groups of 4 rats were sham-exposed or exposed at 1, 5, 10, 20, 40, 50, 60, or 70 mW/sq cm for 1 hr, or at 1, 5, 10, or 20 mW/sq cm for 2, 4, or 8 hr. The rats were then decapitated, Tco was measured again, and blood was collected for serum assays of corticosterone (CS), thyrotropin (TSH), growth hormone (GH), and thyroxine (T4).

The results for 1-hr exposures (Table 1), analyzed by t-test, showed a consistent, statistically significant increase of mean Tco with power density from 20 mW/sq cm upward. Below this value, Tco rose with power density, but the differences were not significant. Mean values of CS, TSH, and GH varied widely and non-monotonically up to about 50 mW/sq cm, but consistent CS elevation and TSH and GH depression were obtained in the range 50-70 mW/sq cm. In contrast, the variation of mean T4 was non-monotonic over the entire power-density range, but significant elevation of T4 was observed at 40 and 70 mW/sq cm. Functional dependence of CS, TSH, GH, and T4 on power density and Tco, evaluated by regression analysis, yielded significant nonzero slopes for all but T4 vs Tco.

The exposures for 2, 4, and 8 hr included substantial parts of the circadian cycle, so analysis of variance was used to test for duration-dependent effects in the sham-exposed rats. Significant effect of duration was noted in Tco but not in T4. The variance among groups for CS, TSH, and GH were significantly inhomogeneous, so a t-test with Cochran and Cox's correction was used. The results indicated monotonic increase of CS and monotonic decrease of GH with sham-exposure duration. The level of TSH after 4 hr of sham-exposure was higher than after 2 hr and 8 hr (the latter lower than the former), but the differences were nonsignificant.

Regarding the exposures to RFR for 2, 4, and 8 hr, the increases in Tco, relative to the values for the shams, were significant at 20 mW/sq cm for all 3 durations, and were significant at 10 mW/sq cm for 4 and 8 hr and at 1 mW/sq cm for 4 hr (but not for 2 or 8 hr). Significant decreases of TSH, GH, and T4 were seen mostly at 20 mW/sq cm and the changes in CS level were nonsignificant, but these results were not fully consistent with regard to duration, power density, or circadian influence.

CRITIQUE: This study appears to be similar to that of Lotz and Michaelson (1978), but covering broader ranges of power density and exposure duration and other hormones besides CS. To the extent that they overlap, the results of the two investigations are reasonably consistent.

In the present study, the hormonal-level changes observed at power densities in the 20-70 mW/sq-cm range appear to be unequivocal, especially those for the 1-hr exposures. Most of the results for the

1-hr exposures at power densities in the 1-20 mW/sq-cm range showed no statistically significant changes in hormonal levels, but there were some large differences in variances among groups.

The results for the longer-duration exposures and lower power densities are even less clear, in part because of the superposed circadian alterations. Particularly noteworthy are the large differences in hormonal levels among the rats sham-exposed for the various durations.

In general, the inconsistencies in results and the inhomogeneities in variances indicate that the use of only 4 rats per treatment group was inadequate, so the powers of the statistical tests used were too low to draw strong inferences or to separate the influences of uncontrolled factors in the experiments.

Despite these shortcomings, the use of a procedure for acclimating animals to the experimental situation prior to treatment appears to be essential in studies of hormonal changes or other biochemical endpoints that can be affected by non-RFR-induced stress. This point was clearly demonstrated in the previous investigation (Lotz and Michaelson, 1978), in which Tco and the CS level rose rapidly during the first half hour of the 3-hr equilibration period and returned to baseline values by the end of the period.

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Lotz, W.G. and S.M. Michaelson
TEMPERATURE AND CORTICOSTERONE RELATIONSHIPS IN MICROWAVE-EXPOSED RATS
J. Appl. Physiol.: Respiratory, Environmental, and Exercise Physiol., Vol. 44, No. 3, pp. 438-445 (1978)

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Lotz, W.G. and S.M. Michaelson
EFFECTS OF HYPOPHYSECTOMY AND DEXAMETHASONE ON RAT ADRENAL RESPONSE TO MICROWAVES
J. Appl. Physiol.: Respiratory, Environmental, and Exercise Physiol., Vol. 47, No. 6, pp. 1284-1288 (1979)

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AUTHOR ABSTRACT: Circulating corticosterone levels were measured to compare the adrenocortical response to acute microwave exposure of normal, hypophysectomized, or sham-hypophysectomized rats. Plasma corticosterone levels in acutely hypophysectomized rats exposed to 60 mW/sq cm for 60 min were below control levels, indicating that the microwave-induced corticosterone response observed in normal, intact rats is dependent on ACTH secretion by the pituitary.

In other groups of rats pretreated with dexamethasone before being exposed to microwaves for 60 min, the corticosterone response to a 50-mW/sq-cm exposure was completely suppressed by doses equal to or greater that 3.2 microgram dexamethasone/100 g body weight. However, the corticosterone response to a 70-mW/sq-cm exposure was only partially suppressed by prior administration of 3.2 or 5.6 microgram dexamethasone/100 g BW.

The evidence obtained in these experiments, in conjunction with the results of other experiments previously reported, is consistent with the hypothesis that the stimulation of the adrenal axis in the microwave-exposed rat is a systemic, integrative process due to a general hyperthermia.

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Study Type: Endocrinology, Physiology and Biochemistry, Multiagent Interactions; IN VIVO; RAT
Effect Type: RFR-induced alterations of corticosterone levels in intact or hypophysectomized rats, with or without pretreatment with dexamethasone
Frequency: 2.45 GHz
Modulation: Amplitude-modulated at 120 Hz
Power Density: 50, 60, or 70 mW/sq cm
SAR: 0.16 W/kg per mW/sq cm

EXPOSURE CONDITIONS: After they were "gentled" and "conditioned" for 2 weeks, intact-, hypophysectomized-, or sham-hypophysectomized rats in individual cages were exposed concurrently in groups of 4 to far-field RFR in a temperature-controlled anechoic chamber for 60 min at 60 mW/sq cm, or were injected with ACTH to verify adrenal functional integrity. Other groups pretreated with various doses of dexamethasone were exposed for 60 min at 50 or 70 mW/sq cm. Two rats were sham-exposed for each RFR-exposed group.

OTHER INFORMATION: Gentling of rats consisted of weighing and handling them at least 4 times/week for 2 weeks before use; conditioning them involved taking each rat's colonic temperature and placing the rat in an exposure cage for 3-5 hr on 3 of the last 4 days before use.

Hypophysectomy ("hypox") was done under anesthesia, with subsequent inspection to ensure complete removal of the pituitary. The surgical procedure for sham-hypophysectomy ("sham-hypox") was the same except that the pituitary was not removed. Following surgery and full recovery from anesthesia, groups of rats were equilibrated within the exposure chamber for 3 hr prior to exposure, a step found necessary to ensure return of colonic temperature and serum corticosterone to baseline levels in unmanipulated rats (Lotz and Michaelson, 1978). At the end of RFR- or sham-exposure, the rats were decapitated, trunk blood was collected, and plasma was assayed for corticosterone (CS). Colonic temperature was taken within 60 seconds after decapitation.

In addition to the groups above, unexposed hypox rats were injected with ACTH, returned to their cages, decapitated 1 hr later, and assayed for blood-plasma CS. Groups of unexposed intact and sham-hypox rats were similarly treated for comparison. The objective of this test was to verify adrenal functional integrity during RFR exposure of hypox rats, because the response of the rat adrenal gland to exogenous ACTH stimulation declines 4-24 hr after hypophysectomy.

The mean colonic temperatures of the sham-exposed (control) rats showed nonsignificant differences ($p>0.05$, Student t-test) among the intact, hypox, and sham-hypox groups. For the RFR-exposed groups, the only significant difference in mean colonic temperature was between the hypox and sham-hypox rats, with the value for the latter lower than for the former.

For the sham-exposed groups, the mean plasma-CS level of the hypox rats was barely detectable and significantly lower than the values of the intact or sham-hypox rats; the difference between the latter two groups was not significant. For the RFR-exposed groups, the mean CS levels of the intact and sham-hypox rats were both much higher than the values of their respective sham-exposed groups and did not differ significantly from each other, but again the level for the hypox rats was barely detectable. For the groups injected with ACTH, the CS levels of the intact and sham-hypox rats were also much higher than the corresponding values of the sham-exposed rats. However, the level for the hypox rats was also high and did not differ significantly from the values for the other two groups.

To determine whether RFR-induced stimulation of the rat pituitary-adrenal axis could be blocked by pretreatment with exogenous glucocorticoids, dexamethasone in doses of 0.56, 1.0, 1.8, 3.2, 5.6, or 10 microgram/100 g of body weight were injected into groups of intact rats, after which the rats were exposed at 50 mW/sq cm for 60 min and assayed for CS. Doses of 3.2 or 5.6 microgram/100 g were injected into other groups followed by exposure at 70 mW/sq cm for 60 min. For controls, other rats were injected with saline and exposed.

Each dose of dexamethasone yielded a highly significant reduction in CS level after exposure at 50 mW/sq cm ($p<0.01$ for 0.56; $p<0.001$ for all other doses). The dose-response relationship was evident, and doses of 3.2 or larger almost totally blocked any increases in CS level during exposure. However, doses of 3.2 or 5.6 did not fully suppress increases of CS level during subsequent exposure at 70 mW/sq cm. Mean colonic temperatures for the rats exposed at 50 mW/sq cm after dexamethasone injection ranged from 39.8 to 40.1 deg C; the differences among the various dose groups were not statistically signiiicant.

From the barely detectable CS levels in the hypox rats exposed at 60 mW/sq cm for 60 min and the high levels in the hypox rats injected with ACTH (which stimulated adrenal secretion of CS in these rats), the investigators concluded that RFR exposure does not stimulate the adrenal gland primarily. Instead, the observed increases in CS level in intact rats during RFR exposure must have been due to adrenal stimulation by endogenous ACTH from the pituitary during RFR exposure. The results of the dexamethasone experiments comprise additional evidence that adrenal response is due to ACTH stimulation during exposure, and the moderate doses needed to inhibit RFR-induced ACTH secretion indicate that exposure at 50 mW/sq cm for 60 min is a relatively mild stimulus for such secretion.

Based on these results and those of Lotz and Michaelson (1978), the investigators hypothesized that RFR-induced secretion of ACTH is a systemic integrative process due to general hyperthermia.

CRITIQUE: As in other studies by this group, pains were taken to "gentle" and equilibrate the rats prior to exposure, to minimize uncontrolled non-RFR-induced changes in CS levels.

In the experiments with the intact, hypox, and sham-hypox rats, the standard errors about the mean CS levels were small relative to the observed changes in CS levels, thus providing biological credence to the findings. The increased CS levels for the intact rats exposed at 60 mW/sq cm for 60 min are consonant with the results obtained by Lu et al. (1977) and Lotz and Michaelson (1978) that indicated the existence of a threshold of about 20 mW/sq cm for CS elevation in the rat.

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Lotz, W.G. and S.M. Michaelson
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Lu, S.-T., N. Lebda, S.M. Michaelson, S. Pettit, and D. Rivera
THERMAL AND ENDOCRINOLOGICAL EFFECTS OF PROTRACTED IRRADIATION OF RATS BY 2450-MHZ MICROWAVES
Radio Sci., Vol. 12, No. 6S, pp. 147-156 (1977)

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Smialowicz, R.J., M.M. Riddle, P.L. Brugnolotti, R.R. Rogers, and K.L. Compton
DETECTION OF MICROWAVE HEATING IN 5-HYDROXYTRYPTAMINE-INDUCED HYPOTHERMIC MICE
Radiat. Res., Vol. 88, No. 1, pp. 108-117 (1981a)

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AUTHOR ABSTRACT: The intraperitoneal injection of 5-hydroxytryptamine (5-HT) in unrestrained and unanesthetized mice held at 22 deg C causes a hypothermia which is maximal after approximately 15 min. When mice injected with 5-HT were held in a controlled environment of 22 deg C and 50% relative humidity and exposed to microwaves (2450 MHz, cw) at 1 mW/sq cm for 15 min, significant increases were observed in the body temperature of these mice compared to 5-HT-treated sham-irradiated mice. The magnitude of the response was related to power density (10 > 5 > 1 mW/sq cm).

Saline-injected mice exposed for 15 min at 10 mW/sq cm (specific absorption rate = 7.2 mW/g) showed no significant increase in body temperature compared to saline-injected sham-irradiated mice. The hypothermia induced by 5-HT in mice was also found to be affected by ambient temperature alone. Increases in ambient temperature above 22 deg C, in the absence of microwaves, caused a concomitant increase in body temperature. By altering the thermoregulatory capacity of mice with 5-HT, subtle heating by microwaves was detected. These results indicate that the interpretation of microwave-induced biological effects observed in animals at comparable power and absorption levels should include a consideration of the thermogenic potential of microwave radiation.

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Study Type: Endocrinology, Physiology and Biochemistry, Metabolism and Thermoregulation, Multiagent Interactions; IN VIVO; MOUSE
Effect Type: RFR-induced body-temperature increases in mice rendered hypothermic by injection with 5-hydroxytryptamine
Frequency: 2.45 GHz
Modulation: CW (11% ripple)
Power Density: 0.5, 1, 5, or 10 mW/sq cm
SAR: 0.36, 0.7, 3.6, or 7.2 W/kg

EXPOSURE CONDITIONS: Mice in individual vented polycarbonate boxes large enough to permit free movement were sham-exposed or exposed for 15 min to far-field RFR in groups of 4 arranged in a diamond pattern at a spacing of 19 cm in an environmental chamber. The environmental chamber was mounted within an anechoic chamber and held at 22 deg C and 50% relative humidity.

OTHER INFORMATION: Female BALB/C mice weighing 20-25 g and male CBA/J mice weighing 24-28 g were used. Treated mice were injected i.p. with

0.5 mg of 5-hydroxytryptamine creatinine sulfate complex (5-HT) in 0.2 ml pyrogen-free saline, corresponding to a dose of 20-25 mg/kg. Control mice were injected with 0.2 ml of saline.

Colonic temperatures of unexposed BALB/C mice in an ambient temperature of 22 deg C, taken at 15-min intervals for 90 min after 5-HT injection, showed maximum depression (from about 38.2 to 34.9 deg C) at the first 15-min epoch, with gradual recovery (to about 37.8 deg C) at 90 min. A similar response was obtained for CBA/J mice (data not presented). Therefore, 15 min was chosen as the optimum exposure duration.

The effect of ambient temperature on unexposed CBA/J mice 15 min after rendering them hypothermic with 5-HT was determined. The mean colonic temperature rose from about 32.3 deg C at an ambient of 18 deg C to about 37.1 deg C at 34 deg C. Also, the latter colonic temperature was lower than for saline-injected mice (38.8 deg C) at 34 deg C. A linear-regression plot of colonic temperature of the hypothermic CBA/J mice vs ambient temperature yielded a correlation coefficient of 0.8985. The slope was 0.2775.

Following injection with 5-HT or saline, BALB/C mice in groups of 4 were sham-exposed or exposed for 15 min at 10, 5, or 1 mW/sq cm, corresponding respectively to 7.2, 3.6, or 0.7 W/kg, as determined by twin-well calorimetry. Though not stated explicitly, CBA/J mice were exposed at 0.5 mW/sq cm as well as at the other 3 power densities. Colonic temperatures were taken immediately before and after exposure. Comparisons of final temperatures and temperature changes (final minus initial temperatures) were analyzed by Student's t-test.

The mean final colonic temperature of the saline-injected BALB/C mice exposed at 10 mW/sq cm (about 38.7 deg C) did not differ significantly from that of the saline-injected sham-exposed mice (about 38.5 deg C). For the mice rendered hypothermic with 5-HT, however, the mean value at each power density was significantly higher ($p<0.05$) than for the corresponding sham-exposed group. For the mice exposed at 10, 5, and 1 mW/sq cm, the mean final colonic temperatures were about 36.5, 35.7, and 34.8 deg C, respectively, all of which were significantly lower than for the saline-injected mice. The differences among the values for the 3 sham-exposed hypothermic groups were not significant.

For the hypothermic CBA/J mice, the mean colonic temperatures for those exposed at 10 or 5 mW/sq cm were significantly higher than for the corresponding sham-exposed mice. Higher temperatures were also obtained for those exposed at 1 or 0.5 mW/sq cm, but the differences in mean temperatures were not statistically significant. All the mean values were significantly lower than for the saline-injected mice exposed at 10 mW/sq cm or sham-exposed. The values for the 2 saline-injected groups did not differ significantly. A linear regression plot of colonic temperature of the CBA/J hypothermic mice vs power density yielded a correlation coefficient of 0.6557 ($p<0.05$). The slope was 0.1553.

For the BALB/C mice, the mean colonic-temperature depression for the saline-injected mice exposed at 10 mW/sq cm (-0.22 deg C) was not significantly different from the depression for the saline-injected sham-exposed mice (-0.26 deg C). However, the depressions for the mice exposed at 1, 5, and 10 mW/sq cm (-3.53, -3.26, and -2.43 deg C) were all significantly smaller than for the corresponding sham-exposed groups (-4.45, -4.01, -4.46 deg C). For the CBA/J mice, the differences were not significant for the saline-injected mice or for those exposed at 0.5 mW/sq cm. However, again the depressions for those exposed at 1, 5, and 10 mW/sq cm were significantly smaller than for their sham-exposed groups.

The slopes (m) of the linear-regression plots of colonic temperature of the hypothermic CBA/J mice vs ambient temperature (m=0.2775) and power density (m=0.1553) were used to derive an equivalence between power density and ambient temperature elevation. Dividing the former slope by the latter slope yielded 1.79. This was interpreted to mean that, under the experimental conditions described, exposure of 5-HT-treated CBA/J mice to 2.45-GHz RFR at 1.79 mW/sq cm would cause the same increase in colonic temperature (0.3 deg C) as a 1-deg-C increase in ambient temperature.

A colonic temperature increase of 0.3 deg C at 1.79 mW/sq cm corresponds to an increase of 0.17 deg C at 1 mW/sq cm, which agrees well with predictions based on the SAR data. Specifically, the calorimetrically measured SAR of the mouse at 1 mW/sq cm was 0.7 W/kg. For comparison, absorption of energy (as heat) for 15 min at an SAR of 0.7 W/kg in a body having a specific heat of 1 and no heat losses would produce a temperature increase of about 0.15 deg C.

CRITIQUE: The reasons for using female BALB/C and male CBA/J mice or whether the strain and sex differences were important were not discussed.

The investigators noted that they had obtained similar results with rats rendered hypothermic with the endotoxin Salmonella typhimurium W lipopolysaccharide and exposed to RFR at power densities or SARs of the order of 1 mW/sq cm or 0.2 W/kg (Smialowicz et al., 1980). Such findings in both species indicate that RFR at such relatively low power densities can be absorbed as heat in normothermic animals without evoking colonic temperature increases, and that effects observed at such RFR levels are not necessarily nonthermal. Instead, the absence of a colonic temperature rise from exposure to RFR is an indication that the energy absorbed as heat is within the thermoregulatory capabilities of the animal.

The equivalence derived from the slopes of the colonic-temperature rise vs power density and ambient temperature in hypothermic animals is an interesting result. The investigators noted that based on such comparisons, animals rendered hypothermic may be useful as biological dosimeters for RFR, and cited Putthoff et al. (1977), who used cortisone or salicylate to produce hypothermia in rats.

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Abhold, R.H., M.J. Ortner, M.J. Galvin, and D.I. McRee
STUDIES ON ACUTE IN VIVO EXPOSURE OF RATS TO 2450-MHZ MICROWAVE RADIATION: II. EFFECTS ON THYROID AND ADRENAL AXES HORMONES
Radiat. Res., Vol. 88, No. 3, pp. 448-455 (1981)

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AUTHOR ABSTRACT: The effects of 8 hr continuous exposure of rats to 2450-MHz (cw) microwave radiation were studied at incident power densities of 0, 2, and 10 mW/sq cm. Thyroid axis function, as measured by serum thyroxine (T4) and triiodothyronine (T3) as well as T3 uptake, free thyroxine index, and adjusted-T4 values was not altered by the experimental conditions. Adrenal axis activity was also unaffected in rats exposed to 2 mW/sq cm microwave radiation for 8 hr. In the 10 mW/sq cm group, the serum corticosterone levels were less than in the 0 or 2 mW/sq cm groups (7.8 versus 9.9 microgram/dl). The experimental protocol increased serum corticosterone levels (7.0 versus 9.9 microgram/dl for the nonhandled and sham-exposed groups, respectively); however, the corticosterone concentration in the 10 mW/sq cm group was similar to that in the untreated (nonhandled) controls (7.8 versus 7.0 microgram/dl). This modified adrenal axis function gives further support to the concept that microwave radiation affects endocrine function.

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Study Type: Endocrinology, Physiology and Biochemistry; IN VIVO; RAT
Effect Type: RFR-induced effects on concentrations of various hormones
Frequency: 2.45 GHz
Modulation: CW
Power Density: 2 or 10 mW/sq cm
SAR: 0.44 or 2.2 W/kg

EXPOSURE CONDITIONS: Rats in individual vented 10x20 cm Styrofoam cages were sham-exposed or exposed concurrently to far-field RFR at 2 or 10 mW/sq cm in groups of 8 arranged in a circular pattern with their long axes parallel to the E-vector. Exposures were done for 8 hr within an anechoic chamber maintained at 23 deg C and 65% relative humidity. Food and water were not provided during exposure.

OTHER INFORMATION: Exposures were done for 8 hr within the period from about 0030 to about 0845. The rats were then decapitated and blood was collected in centrifuge tubes and allowed to clot. After centrifugation, sera from pairs of similarly treated rats were combined, thereby yielding 4 pooled samples each from 8 untreated rats (cage controls), 8 sham-exposed rats, and 8 rats exposed at each power density (a total of 16 pooled samples from 32 rats). Aliquots of the pooled samples were assayed for thyroxine (T4), triiodothyronine (T3), the percentage of T3-uptake (T3u), and corticosterone (CS). The free thyroxine index (FTI), defined as the product of T4 and T3u, and adjusted thyroxine (AT4), defined as the product of T4 and the ratio of T3u for treated rats to T3u for control rats, were calculated.

The differences in mean T4, T3, T3u, FTI, and AT4 were not statistically significant ($p>0.05$) among the cage-control, sham-exposed, and RFR-exposed groups. The mean serum CS value for the rats exposed at 2 mW/sq cm was almost the same as for the sham-exposed rats, and both were significantly higher ($p<0.01$) than for the cage controls. However, the mean CS for the rats exposed at 10 mW/sq cm did not differ significantly from the value for the cage controls.

CRITIQUE: The investigators did not state the rationale for pooling the sera from pairs of similarly treated rats instead of assaying each sample separately.

As the investigators indicated, the negative findings for T4, T3, T3u, FTI, and AT4 were consistent with those of Lu et al. (1977), who found no effect of 8-hr exposure to 2.45-GHz RFR at 1, 5, or 10 mW/sq cm on the level of T4. In a later investigation, Lu et al. (1980b) obtained nonmonotonic variations (increases and decreases) of mean T4 level vs power density in rats exposed to 2.45-GHz RFR at 0, 5, 10, or 20 mW/sq cm for 2, 4, or 8 hr; the changes were statistically significant mostly at 20 mW/sq cm, and were depressions of T4 (and of thyrotropin and growth hormone as well).

Regarding the CS results, Abhold et al. stated: "Although the 10 mW/sq-cm exposure group had lower corticosterone levels than either the 2 or 0 mW/sq-cm group, the 10 mW/sq-cm group had levels which were similar to those of the untreated controls. These data suggest that the experimental protocol stimulated the HHA [hypothalamo-hypophyseal-adrenal] axis, and that this effect could be counteracted by exposure to microwaves at 10 mW/sq cm." The meaning of "experimental protocol" in the latter statement is unclear; presumably it is used to account for the significant difference between the mean CS values for the untreated-control and sham-exposed groups, which is an indication of the presence of uncontrolled factors. Also, their results are not consonant with the the later CS results of Lu et al. (1980b), who controlled for stress factors that would otherwise have elevated CS levels, and obtained nonmonotonic, nonsignificant CS changes in the rats exposed for 8 hr at 5 or 10 mW/sq cm (relative to sham-exposed rats) and a significant CS depression for the rats exposed for 8 hr at 20 mW/sq cm.

In general, it appears difficult to distinguish alterations of hormonal levels ascribable to exposure to RFR in the 0-10 mW/sq-cm range for durations that are substantial fractions of the circadian cycle from alterations associated with the circadian cycle and/or with other uncontrolled factors. Perhaps use of larger numbers of animals per group would aid in separating RFR-induced changes from those produced by such other factors.

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EFFECTS OF 2.45 GHZ MICROWAVE RADIATION AND HEAT ON MOUSE SPERMATOGENIC EPITHELIUM
Int. J. Radiat. Biol., Vol. 40, No. 6, pp. 623-632 (1981)

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AUTHOR ABSTRACT: The rear halves of the bodies of anesthetized male C3H mice were exposed for 30 min to 2.45 GHz microwave radiation and the effects on the testes were compared to those produced by direct heating. Effects were observed which are consistent with the hypothesis that heat damage is the primary effect of microwave exposure. Damage measured six days after exposure ranged in severity from depletion of the spermatocytes to extensive necrosis of the germinal epithelium.

Temperature-sensitive probes implanted in the testes revealed a threshold effect for depletion of the spermatocytes of approximately 39 deg C and an LD6-50 (50 per cent cell death after 6 days) of about 41 deg C after microwave exposure or direct heating. The corresponding effective threshold effect and LD6-50 expressed in terms of absorbed microwave power were 20 W/kg and 30 W/kg. However, it is probable that a conscious animal is better able to regulate testicular temperature and hence adjust to higher dose-rates.

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Study Type: Endocrinology, Physiology and Biochemistry, Metabolism and Thermoregulation; IN VIVO; MOUSE
Effect Type: RFR- and heat-induced effects on mouse testes
Frequency: 2.45 GHz
Modulation: CW
Power Density: Not measured
SAR: "Half-body" values in the range 18-75 W/kg (whole-body values during exposure of the rear half)

EXPOSURE CONDITIONS: Rear halves of anesthetized mice were sham-exposed or exposed for 30 min in a waveguide at various values of forward power. Measurements of forward, reflected, and transmitted powers were used to determine SARs. Rear halves of other mice were heated for 30 min in a dry copper well that was immersed in water baths at various temperatures. The room in which the mice were housed and treated was maintained at 22 deg C.

OTHER INFORMATION: A group of 3-5 mice was used for each treatment. The mice used for the following temperature measurements were not subjected to histological examination. Other mice were used for the latter purpose, and the temperature measurements were used to estimate temperatures in those mice.

Rectal temperatures were measured with thermocouples before and immediately after RFR exposure or heating in the well. In other mice,

calibrated liquid-crystal fiber-optic probes surgically inserted into the testes were used to measure testicular temperatures during exposure at 0 (sham), 22, 43, and 57 W/kg, and surgically inserted thermocouples were similarly used during well heating in water baths at temperatures of 37, 41, 43, and 45 deg C.

For the mice exposed to RFR, the mean rectal temperature varied from 32.6 deg C at 0 W/kg to 42.2 deg C at 74.4 W/kg. The temperature for the sham-exposed mice was 4-5 deg C below that for unanesthetized mice. The mean pre-exposure testicular temperature for the sham-exposed and the 3 RFR-exposed groups was 34 +/- 1 deg C. During the 30-min treatment period, the testicular temperature of the sham-exposed mice decreased to about 30 deg C. For the mice exposed to RFR, approximate plateaus were reached after about 15 min of exposure. The final mean values were 37.0, 40.0, and 42.4 deg C for 22, 43, and 57 W/kg, respectively.

For the mice heated in the copper well, the mean rectal temperatures at the end of the 30-min period ranged from 36.4 deg C at a water-bath temperature of 37 deg C to 40.7 deg C at 45 deg C. The corresponding testicular temperatures were 37 and 43.2 deg C, respectively.

The mice were killed 6 days after RFR exposure or well heating; sections of the testes were prepared, examined, and scored; and sperm counts were made of samples taken from the epididymides. The 6-day interval allowed the cells to be studied to progress to early primary spermatocytes, intermediate primary spermatocytes, and spermatids, all three of which were readily identifiable for scoring.

The histological results showed that extensive degeneration of the spermatogenic epithelium was produced at an SAR of 75 W/kg or a water-bath temperature of 45 deg C. The interstitial cells and Sertoli cells appeared unaffected. At SARs of 57 and 46 W/kg or bath temperatures of 43 and 41 deg C, marked depletions of spermatids and spermatocytes but not spermatogonia were obtained. No effects were seen at SARs of 37, 30, 18, or 0 W/kg or at a bath temperature of 37 deg C.

A plot of the early-primary-spermatocyte counts (per tubule) vs testicular temperature (derived from both RFR-exposure and well heating) showed no significant depletion up to 42.5 deg C. For the RFR-exposed mice, this testicular temperature corresponds to an SAR of 57 W/kg and represents the threshold for no effect. The similar plot for the intermediate-primary-spermatocyte counts revealed a threshold for depletion at about 39 deg C, with an LD6-50 (50% depletion at the end of the 6-day period) at 40 deg C. For the RFR-exposed mice, the corresponding threshold- and LD6-50 SARs were 35 and 41 W/kg. The plot for the spermatid counts showed an ill-defined depletion threshold at about 38 deg C and an LD6-50 at 41 deg C; the corresponding SARs were about 30 and 46 W/kg.

Because the mice were anesthetized, their thermoregulatory function was impaired and they lost heat by peripheral vasodilation, resulting in

lower than normal body temperature. From the data, the investigators calculated that it required absorption of heat at a rate of 12 W/kg to maintain testicular temperature at 34 deg C. Consequently, they stated that the foregoing threshold SARs should be corrected by subtracting 12 W/kg, to yield "effective" thresholds. The investigators do suggest that mice with unimpaired thermoregulatory function may have higher thresholds for testicular damage than anesthetized mice.

Based on their results, the investigators concluded that the deleterious effects of RFR on the testes can be ascribed entirely to the heat produced by the RFR.

CRITIQUE: The findings of this investigation are consonant with those of Prausnitz and Susskind (1962), who exposed unanesthetized mice to 9.27-GHz RFR at 100 mW/sq cm for 4.5 min/day (which increased mean body temperatures by 3.3 deg C) for 5 days/week over 59 weeks. Testicular degeneration was found in 40% of the RFR-exposed and in 8% of the control mice that had died during the course of the experiment.

Cairnie and Harding (1979) reported that exposure of unanesthetized mice to 2.45-GHz RFR at 20 to 32 mW/sq cm for 16 hr/day for 4 days had no effect on sperm count or percentages of abnormal sperm. In a subsequent investigation, Cairnie et al.(1980) found that exposure of mice to 2.45-GHz RFR for 16 hr at 50 mW/sq cm elevated rectal but not testis temperature and that the local SAR in the testis was considerably lower than the whole-body SAR, thereby demonstrating the ability of the conscious mouse to regulate its testes temperature.

The results of Saunders and Kowalczuk were also similar to those of Muraca et al. (1976) with anesthetized rats exposed to 2.45-GHz RFR or heated by immersing their scrota in warm water. Exposures at about 80 mW/sq cm for 10 to 73 min, or immersion in water that yielded an intratesticular temperature rise to 40 deg C produced degenerative changes in less than 30% of the rats.

In contrast, Lancranjan et al. (1975) reported that men occupationally exposed to RFR in the 3.6- to 10-GHz range at power densities of tenths to hundredths of a mW/sq cm for 1 to 17 years (a mean of 8 years) showed slightly reduced sperm counts, but normal plasma levels of 17-ketosteroid and gonadotropic hormone. However, because non-RFR factors in the occupational environment may have been involved, the significance of these findings of Lancranjan et al. (1975) is open to question.

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Lebovitz, R.M. and L. Johnson
TESTICULAR FUNCTION OF RATS FOLLOWING EXPOSURE TO MICROWAVE RADIATION
Bioelectromagnetics, Vol. 4, No. 2, pp. 107-114 (1983)

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AUTHOR ABSTRACT: Male Sprague-Dawley rats were exposed for 6 h per day for nine days to pulse-modulated microwave radiation (1.3 GHz, at 1-microsecond pulse width, 600 pulses per second). Exposures were carried out in cylindrical waveguide sections at a mean dose rate of 6.3 mW/g; sham controls were treated similarly and received no irradiation. At time periods corresponding to 0.5, 1.0, 2.0, and 4.0 cycles of the seminiferous epithelium, groups of four sham-irradiated and four irradiated rats were killed and the testes removed for analysis. Net mass of the testes, epididymides, and seminal vesicles; daily sperm production (DSP) per testis and per gram of testis; sperm morphology; and the number of epididymal sperm were determined.

There were no statistically significant differences between the sham-irradiated and irradiated groups with respect to any measured variable. In a group of seven surrogate animals of similar body mass, the dose rate of 6.3 mW/g caused a net change in body temperature (via rectal probe) of 1.5 deg C.

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Study Type: Endocrinology, Physiology and Biochemistry, Metabolism and Thermoregulation; IN VIVO; RAT
Effect Type: Effects of RFR exposure on testes mass and on sperm production, morphology, and counts of epididymal sperm
Frequency: 1.3 GHz
Modulation: 1-microsecond pulses, 600 pps (0.0006 duty cycle)
Power Density: Not stated
SAR: 6.3 W/kg

EXPOSURE CONDITIONS: A group of 14 unanesthetized rats was exposed to circularly polarized RFR concurrently in individual cylindrical waveguides for 6 hr/day on 9 days during a 2-week period. A group of 15 rats was similarly sham-exposed. The ambient temperature was 23 +/- 1.5 deg C and the relative humidity ranged from 32% to 45% with a mean of 37%. A separate group of 4 similarly handled RFR- and 3 sham-exposed rats was used for measuring rectal temperatures following treatment.

OTHER INFORMATION: The rats were weighed and decapitated at 6.5, 13.0, 26.0, or 52 days after the last treatment day, corresponding to 0.5, 1.0, 2.0, or 4.0 cycles of spermatogenesis. The testes were separated from the epididymides before weighing each testis and epididymis and the seminal vesicles. The right testis was decapsulated, the tunica albuginea weighed, and the testis homogenized. The spermatids resistant to homogenization were enumerated by phase-contrast cytometry. Daily sperm production (DSP) per testis was calculated by dividing the number

of resistant spermatids by the 6.3-day life span. DSP/g of parenchyma was obtained by dividing DSP/testis by the difference between testis and tunic weights. The number of sperm in the right epididymis was determined from epididymal homogenates.

The left testis was fixed, osmicated, sectioned, stained, and examined by bright-field microscopy. Spermatogenesis was qualitatively evaluated by dicing the tail of the left epididymis in 2 ml of Minimum Essential Medium at room temperature and allowing sperm to swim from the tissue into the medium. Stained sperm smears on glass slides were examined by bright-field microscopy; at least 100 sperm per slide were selected and classified as either normal or abnormal.

The results showed some trend toward lower testicular and epididymal weights and toward lower levels of DSP/testis for the RFR-exposed rats compared to the sham-exposed rats, but the differences were not significant by 2-way analysis of variance. No RFR- vs sham-exposure differences were found nor were there any significant interactions between treatment and spermatogenic cycle.

The RFR-exposed rats examined on day 6.5 after treatment (0.5 cycle of spermatogenesis) yielded 87.6% normal sperm compared with 95.8% for the corresponding sham-exposed rats, a statistically significant difference. However, most of the abnormal sperm in the former group were derived from 1 rat that yielded 45.5% abnormal sperm, rendering the finding suspect.

Because spermatogenic cycle at time of sacrifice was not a significant factor, the data for all cycles were pooled. There were no statistically significant differences in pooled values from RFR- and sham-exposed rats of any endpoint.

The weight of the seminal vesicles was taken as a measure of testicular endocrine function. The nonsignificant differences between values for the RFR- and sham-exposed rats indicated that RFR exposure at 6.3 W/kg was not deleterious to the production of testosterone. This finding was supported by the histological evaluations by light microscopy, which showed similarities in: structure of seminiferous tubules, abundance of all types of developing germ cells, and structure of Leydig cells.

Deep rectal temperatures were measured hourly in 3 sham-exposed rats and 4 rats exposed at 6.3 or 6.9 W/kg for 6 hr and otherwise handled in the same fashion as those used in the testicular experiments. The mean elevation of core temperature for the 6.3-W/kg group was 1.5 to 2 deg C. Elevation was attained during the first hr and was stable for the remainder of the period. The mean elevation for the 6.9-W/kg group was slightly but not significantly higher than for the 6.3-W/kg group. Clearly, the rats were able to accommodate (attain an elevated but stable body temperature) to these levels of exogenous thermogenesis. There were no significant core-temperature changes in the sham-exposed rats.

The investigators noted that exposure of rats at 6.3 W/kg yielded not only an unambiguous elevation of core temperature, but also immediate alterations in food-reinforced behavior (Lebovitz, 1981), and that both effects were fully reversible. They suggested that if these effects were due solely to the RFR acting as an added thermal burden and a sensory stimulus or cue, then there would be little basis for considering such exposures as harmful.

CRITIQUE: The results of this investigation with unanesthetized rats as well as those of Saunders and Kowalczuk (1981) with anesthetized mice indicate that there exist threshold levels of RFR exposure for testicular damage (well above 6.3 W/kg for rats and about 37 W/kg in mice). Both groups of investigators characterized the effects observed as due to thermogenesis by the RFR. Qualitatively similar results were also obtained by Cairnie and Harding (1979) with mice and by Muraca et al. (1976) with rats.

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J. Microwave Power, Vol. 17, No. 2, pp. 145-150 (1982)

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AUTHOR ABSTRACT: Whole-body evaporative heat loss was measured as whole-body evaporative water loss in mice during a 90-min exposure to 2450-MHz microwave radiation at an ambient temperature of 20 deg C and in non-exposed mice maintained at ambient temperatures of 20, 25, 30, 33, and 35 deg C. The ambient-temperature threshold for increasing evaporative water loss was between 30 and 33 deg C. A specific absorption rate of microwave radiation in excess of 29 W/kg was required to produce an increase in heat loss. For absorption rates ranging from 29 to 44 W/kg, the mouse dissipated 65% of the total absorbed heat by water evaporation; the remainder was dissipated passively.

The data collected in the mouse may be extrapolated to larger species, such as man, but only by an exponential relationship. Using this relationship, it was shown that a threshold specific absorption rate of 29 W/kg in a 0.033-kg mouse was equivalent to approximately 0.25 W/kg in a 70-kg human.

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Study Type: Metabolism and Thermoregulation, Physiology and Biochemistry; IN VIVO; MOUSE
Effect Type: RFR- and ambient-temperature-induced alterations of thermoregulation
Frequency: 2.45 GHz
Modulation: CW
Power Density: Not measured
SAR: 0-44 W/kg

EXPOSURE CONDITIONS: Mice were exposed individually in a perforated cylindrical Plexiglas cage inserted within a closed section of rectangular waveguide that was totally immersed in a water bath thermostatically controlled to within +/- 0.5 deg C. A perforated flat plate of Plexiglas within the cage supported the mouse with freedom of movement. A layer of mineral oil beneath the plate was used to trap urine and feces. SARs were determined from measurements of forward, reflected, and transmitted powers. Ambient air dried in calcium sulfate and equilibrated with the temperature of the water bath was passed into the waveguide, and the exhaust air from the waveguide was drawn at 60 ml/min through a cannister filled with color-indicating anhydrous calcium sulfate.

After a 90-min equilibration period, each mouse was exposed at a forward power in the range from 0.25 to 2.75 W for 90 min at a water-bath temperature of 20 deg C. Evaporative water loss (EWL) during the

exposure period was determined from the increase in weight of the calcium sulfate in the exhaust line. After equilibration for 90 min at a water-bath temperature of 20, 25, 30, 33, or 35 deg C, the EWLs of other mice not exposed to RFR were measured for a 90-min period at each temperature.

OTHER INFORMATION: The investigator indicated that varying the volume of the mineral oil used to trap urine and feces did not affect power absorption.

The mean SAR at each forward power was determined by integrating the absorbed-power-vs-time curve over the 90-min exposure with a planimeter and ascertaining the SAR that would yield the same integral. At 2.75 W, the highest forward power used, the mean SAR was 44 W/kg, with large fluctuations about the mean, an indication of extensive animal movement from discomfort. The fluctuations were much smaller at 1.0 W (mean SAR of about 14 W/kg).

The evaporative water loss (EWL) was taken as a measure of the evaporative heat loss (EHL). Specifically, EHL (W/kg) for each treatment was calculated by multiplying the whole-body EWL (mg of water per g of body weight per second) by 2.426 J per mg of water (heat removed by evaporating 1 mg of water at 37 deg C). The data were analyzed by linear regression.

Plots of EWL (in both W/kg and in mg of water per g of body weight per min) vs SAR (W/kg) for 27 points taken with 9 mice were displayed in Fig. 3 of the paper. (For mice used more than once, recovery for at least 48 hr between determinations was allowed.) Two regression-line segments were drawn by the investigator, one that was essentially horizontal for the EWL points at SARs below 23 W/kg and the other with an indicated positive slope of 0.68 for the EWL points at SARs from 28 to 44 W/kg. (There were no points in the range 23-28 W/kg.) The intersection of the two segments was at about 26 W/kg.

From the results above, the investigator stated: "At an ambient temperature of 20 deg C, whole-body EHL was relatively constant between SARs ranging from 0 to 29 W/kg. Above 29 W/kg, the mouse underwent a linear elevation in EHL with increasing SAR. The slope of the regression line--0.65 W/kg evaporated heat per W/kg absorbed heat--implies that 65% of the absorbed microwave energy was dissipated by evaporation while the remaining 35% was dissipated passively."

His conclusion was that: "When mice were maintained at 20 deg C, an SAR of 29 W/kg was required to significantly raise EHL (the microwave heat load was equivalent to approximately 290% of the resting metabolic heat production of the mouse). That EHL did not increase below 29 W/kg indicates that thermal homeostasis can be maintained by passive dissipation of the entire absorbed microwave energy, mainly through radiative and convective heat loss." Thus, the value 29 W/kg represents a thermoregulatory threshold at 20 deg C.

The results for the mice maintained at 20, 25, 30, 33, and 35 deg C but not exposed to RFR (Table 1 of the paper) were EHL values of 2.49, 2.10, 2.02, 6.06, and 5.25 W/kg, respectively. The first three values did not differ significantly from each other; the last two values were significantly higher than the value for 30 deg C, and corresponded to an approximate doubling of resting EHL. Thus, about 30 deg C represents an ambient-temperature thermoregulatory threshold.

The investigator then addressed the question of how to extrapolate data from laboratory animals about RFR-induced effects on thermoregulation and the endocrine system to predict similar effects in humans. Using results, derived from 10 references, for a number of endpoints in various species studied at ambient temperatures in the range 20-30 deg C and mostly at 2.45 GHz, he abstracted the minimum SAR necessary to alter the physiological response studied and the representative body mass of each species used. He found that the best fit of a regression line was obtained for log of threshold SAR vs log of body weight that indicated an inverse relationship between them, from which it is possible to extrapolate a threshold SAR measured in a small mammal to a threshold SAR for humans. He gave as an example that a threshold SAR of 29 W/kg in a 0.034-kg mouse is predicted to be equivalent to an SAR of 0.25 W/kg in a 70-kg man.

CRITIQUE: A critique of this paper was published by Adair et al. (1983). The major comments made by Adair et al. were analyzed and are summarized below.

1. Because the threshold for any thermoregulatory response depends directly on the ambient temperature (Ta) as well as the internal body temperature, there is no single threshold SAR for EHL and attempts to generalize measurements of RFR-induced EHL at one Ta (20 deg C) are in error.

2. There are several inconsistencies regarding Fig. 3: The two regression segments intersect at 25 W/kg, not at 29 W/kg, and the slope of the segment above 25 W/kg, stated as 0.65 in the text and as 0.68 on Fig. 3, is actually 0.58.

3. The interpretation of the 0.65 slope ignored the resting metabolic heat production (M) of the adult mouse, conservatively estimated as about 10 W/kg at thermoneutrality, and therefore yielded much higher than actual percentages of aggregate heat dissipation by evaporative water loss. At SARs of 25 and 40 W/kg, the percentages should have been 8.6 and 22%, respectively. That the measurements of EWL at 40 W/kg could account for only 22% of a heat load that was about 5 times the resting metabolism indicates that the mice were undergoing substantial heat stress, which would have elevated body temperatures considerably (not measured).

4. The extrapolation scheme based on thresholds derived from the 10 references to studies (Fig. 5 of the paper) is deceptively simple in concept in that a variety of not necessarily related physiological

responses were involved. Some of the investigators cited had presented rigorous definitions of "threshold" but others gave no definition. Some of the values were incorrectly used or were of questionable relevance (e.g., outside the stated range of ambient temperatures) and other values that were relevant were not included. (Citations were provided for each case.) Among the problems with the concept is the great variability among different assessments of threshold, even in the same species, and that such threshold values depend strongly on ambient temperature.

Adair et al. (1983) concluded that: "Since we understand the thermoregulatory responses of man far better than the responses of any other species, and can quantify them more accurately, it is clear that no relation such as that depicted in Fig. 5 of Gordon (1982) can possibly exist."

In a subsequently published note, Gordon (1983) responded to the comments of Adair et al. (1983) as summarized below.

Regarding comment 1, he stated: "It is well known that the sensitivity of most thermoregulatory effectors is dependent on Ta. However, my study was designed to assess the effects of RFR at room temperature (20 deg C), not at Ta's far above or below this level. Any generalization of the EHL data collected at 20 deg C to other Ta's was never intended or stated. Indeed, I stated in the second-to-last sentence of my paper that variables such as ambient temperature will affect EHL." (The sentence cited was: "Variables such as physiological parameter, ambient temperature, humidity, and age, to name a few, should affect the slope and intercept of the regression line.")

Gordon responded to comment 2 with the following footnotes: "The regression line intersected at 25 W/kg but actual data points indicate EHL stability to 29 W/kg." and "The slope of the regression line in Fig. 3 is 0.65 not 0.68. Redrawing of the figure by artist was slightly inaccurate in final print. This has been corrected in an erratum."

In response to comment 3, he stated: "In my study metabolic compensation was not included in the analysis because the purpose of the paper was to relate the effects of SAR on EHL, not the combined effects of SAR and metabolic rate."

The major rebuttal offered by Gordon was to comment 4 regarding Fig. 5 of the original paper. He replotted (Fig. 2 of the response) threshold SARs for effects on fetal mass, body temperature, vasodilation, and metabolism separately vs body mass (corresponding to various species) on a log-log basis as before, using data, from a variety of sources, taken over a Ta span of 4 deg instead of the 15 deg used previously. The sources cited included several omitted previously and new ones. He stated: "The inverse relation between body mass and threshold SAR is obvious under all endpoints measured. It is interesting to note that for a 70-kg human being, the regression lines extrapolate to a threshold SAR of 0.06 to 0.3 W/kg, depending on the physiological endpoint."

The regression lines shown in Fig. 2 do indicate inverse relationships as stated. The lines for fetal mass, body temperature, and vasodilation were almost coincident with one another. For a 70-kg human, they yielded a threshold SAR of about 0.3 W/kg, the upper limit of the range cited above. The line for metabolism had a much steeper slope than the others, yielding the lower threshold limit 0.06 W/kg.

These regression lines do support the idea that threshold SARs decrease with increasing body mass, but the statistical reliability of the lines is open to question because of the paucity of the data (no correlation coefficients were presented). Moreover, in the absence of data for body masses exceeding 4 kg, the extent to which the relationships can be extrapolated to larger body masses (such as those of humans) and even whether the relationship is linear in the latter range are highly questionable. Specifically, Fig. 2 shows: 4 data points for fetal mass, with body masses ranging up to only about 0.4 kg (rat); 6 points for body temperature, with body masses up to 4 kg (rhesus), which was the highest of all the values; and 3 and 5 points for vasodilation and metabolism, respectively, with largest body mass of 1 kg (squirrel monkey). Thus, the validity of scaling such SAR thresholds to humans by these regression lines is open to question.

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MICROWAVE RADIATION AND DEXTROAMPHETAMINE: EVIDENCE OF COMBINED EFFECTS ON BEHAVIOR OF RATS
Radio Sci., Vol. 14, No. 6S, pp. 253-258 (1979)

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AUTHOR ABSTRACT: Combined effects of microwave radiation and dextroamphetamine were investigated in studies of six male albino rats that performed on a temporal reinforcement (DRL) schedule. During one-hour sessions, only a response that was delayed by 18 s or more after a preceding response was reinforced by a food pellet. Thirteen weeks of pretraining on the schedule generated a low-and-steady rate of responding, with the largest proportion of responses occurring after the appropriate interval of time.

Initially, a dose-effect function was obtained in the absence of radiation for a range of doses of dextroamphetamine (0.25 to 5.0 mg/kg). Smaller doses of the drug produced an increase in response rate with consequent reduction in frequency of reinforcement. Maximal rates of responding occurred at 1.0 to 2.0 mg/kg. Yet higher doses of the drug produced a decline of rate, then complete cessation of responding. A dose-effect function was then obtained for the same doses of the drug during three-month regimens involving single or multiple exposures to radiation.

In the single-exposure condition, after the drug was administered to a subject, it was exposed for one-half hour to 2.45-GHz pulsed microwaves at an averaged power density of 1 mW/sq cm (2 microsecond pulses; 500 pulses/s). The resulting whole-body dose rate was near 0.2 mW/g. In the multiple-exposure condition, a subject was exposed daily to microwaves under the same field conditions, except on days when dextroamphetamine was administered and behavior was observed. Under both conditions of radiation, the dose-effect function was displaced such that the maximal behavioral effect was obtained at doses lower than those without radiation.

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Study Type: Behavior, Multiagent Interactions; IN VIVO; RAT
Effect Type: Synergistic effects of RFR and dextroamphetamine on behavioral-response rates
Frequency: 2.45 GHz
Modulation: 2-microsecond pulses at 500 pps (0.001 duty)
Power Density: 1 mW/sq cm Av; 1 W/sq cm Pk
SAR: 0.2 W/kg

EXPOSURE CONDITIONS: Exposures were for 30 min to pulsed RFR in the near field of a standard-gain horn within an anechoic chamber at an ambient temperature of 23 deg C. The field was vertically polarized, with the H-vector parallel to the rat's long axis. A loosely fitting sleeve holder of fine plastic mesh was used to restrict excessive movement of the rat during exposure.

OTHER INFORMATION: The whole-body SAR was determined analytically and also calorimetrically by measuring the temperature rise in a Styrofoam-encased water model exposed at 1 mW/sq cm for 30 min. The model was enclosed in the plastic mesh sleeve used for holding the rats.

Six male albino rats (Nmri:O[SD]CV) maintained at 80% of their free-feeding weights were studied. The rats were trained to depress a small lever to produce food pellets on a differential-reinforcement-of-low-rate (DRL) schedule. Only responses that followed a preceding response by 18 s or more produced pellets. Responses that occurred within 18 s reset the timing period (DRL 18 s). Stable baseline performances were achieved in 13 weeks of daily, 5-days-a-week training.

Prior to initiating the RFR-exposure regimens, dextroamphetamine in saline was injected i.p. once weekly and the effect of the drug on rat performance was ascertained 30 min after injection. This was done at 10 doses ranging from 0.25 to 5.0 mg/kg. The dose-effect function was determined, using a minimum of 3 injections at each dose. The results were compared with those of control sessions that immediately preceded and later followed sessions of RFR exposure.

Three of the rats were each dosed with the drug once per week, exposed to the RFR for 30 min, and immediately observed for operant behavior for 1 hr, to ascertain any direct interaction of the drug with the RFR (single-exposure condition). To seek for possible cumulative action of the RFR, each of the other 3 rats was exposed for 30 min daily on 4 days per week except on days (usually Thursday) when the drug was injected (multiple-exposure condition). Operant behavior was observed 30 min after injection. Sessions were conducted for 13 weeks, and included sham-RFR exposures and control injections of saline for all 6 rats.

The baseline performance, in correct responses/min, of the 3 rats studied under the single-exposure condition was a mean of about 3.3 with a standard deviation (SD) of about 0.5. The performance of these rats after saline injection was 3.0 after sham exposure and 3.7 after RFR exposure (no SDs given), both of which were within the SD limits of the baseline value.

The mean performance of these rats under sham exposure rose from 3.3 (0.6 SD) for a dose of 0.25 mg/kg to a maximum of 8.9 (2.4 SD) for 2.0 mg/kg. Although the SDs were larger at the higher doses in this range, the increases in performance appeared to be statistically significant. For doses larger than 2.0 mg/kg, their performance declined sharply (with large SDs) to 0 for 4.5 mg/kg.

By contrast, the mean dose response of these rats under RFR exposure rose from 5.1 for 0.25 mg/kg to a maximum of 7.1 for 0.5 mg/kg, values that were significantly higher than those for the corresponding doses without RFR exposure. Above 0.5 mg/kg, the performance declined sharply to 0.6 for 1.0 mg/kg and 0 for 1.5 mg/kg. These results indicate that

RFR exposure after injecting a given dose of dextroamphetamine produced a behavioral effect similar to that obtained with a larger dose without RFR exposure.

For the 3 rats studied under the multiple-exposure condition, the mean baseline performance and the performances for saline injection followed by sham- or RFR exposure were not significantly different from the values for the other rats. Also, the dose-response functions with and without multiple RFR exposures were qualitatively similar to the functions with and without single exposures even though the performances of the former group were determined 24 hr after the last exposure. Maximum responses with multiple sham exposures were obtained for 2.0 mg/kg, with a sharp decline to 0 for 4.5 mg/kg. The mean response for 0.25 mg/kg was significantly higher with multiple RFR exposures than with sham exposures, rose to a maximum for 0.5 mg/kg, and declined sharply to 0 for 2.0 mg/kg.

Representative cumulative-response records for rats from both groups were displayed. The slope of such a record for any sub-interval of time indicated the mean rate of lever pressing during that period (including the incorrect presses that did not yield a food pellet) and was a measure of the activity of the rat; the number of downward deflections during that period represented the number of correct (pellet-producing) responses. As an example, a presumably typical record for one of the single-exposure rats (their Fig. 3) shows that for a dose of 0.5 mg/kg, the slope during the first half-hour of the test period after RFR exposure was about twice that for the same rat after sham exposure, but that the number of correct responses with RFR was only about a third of the number without RFR. For 1.0 mg/kg, this rat performed rapidly but poorly after sham exposure, but hardly at all after RFR exposure.

In their discussion, the investigators indicated that the modest average power density, measured in the near field, may have produced relatively high local SARs, particularly in the head by resonant absorption, which could have selectively heated the brain. In addition, although the estimated energy content of each pulse is below the threshold for acoustic perception, head resonance could have raised the energy to values above the threshold. However, these possibilities were discounted because they would not account for the persistence of the behavioral effects for 1 hr after cessation of the single exposures and 25 hr after the last of the multiple exposures.

The effects of body restraint, which can synergize with modest RFR levels and stressful events to produce sizable body-temperature elevations, were considered, but also discounted because restraint of the rats injected with saline and exposed to RFR produced no significant deviation from baseline values and the dose-effect functions of restrained and unrestrained rats not exposed to RFR were the same.

CRITIQUE: The dose-response plots for the single- and multiple-exposure conditions (their Figs. 1 and 4) provided SD bars that permitted rough verification of statistical significance of the results, even though the

numbers of values contributing to each mean were not clearly stated. (Presumably, a minimum of 3 replications per drug dose for each rat yielded at least 9 values per point.) However, it is not clear why SD bars for the saline-injected rats (with and without RFR exposure) were not included for either condition.

The data presented on the numbers of responses per min in a DRL-18-s schedule do indicate that RFR at the level used can alter the effects of various doses of dextroamphetamine in the rat. However, comparisons between the two groups of rats are open to question, because the rats in the multiple-exposure group were handled 5 days per week whereas those in the other group were presumably handled only once per week.

Also of importance were the cumulative-response records displayed for individual rats, which were discussed only semiquantitatively. Inclusion of data that were normalized to the total numbers of responses might have provided additional insight regarding the nature of the effects observed.

The discussion by the investigators regarding head resonances, auditory responses to pulsed RFR, selective brain heating, and the possible stresses induced by restraint appears to be well taken, an indication that additional research would be necessary to elicit the nature of the interaction between the drug and RFR. In this context, since dextroamphetamine has been reported anecdotally to heighten human perception with the various senses, it could be hypothesized that rats can perceive lower levels of RFR under the influence of the drug than in its absence, and that such perception would alter their behavior. However, this hypothesis would not account for the 24-hr persistence of the RFR influence seen in the multiple-exposure group.

Similar effects were obtained by Thomas et al. (1979) in rats trained to respond on a 1-min-fixed-interval schedule (FI 1), injected with the drug chlordiazepoxide (Librium) and exposed at the same level of RFR. In a later investigation (Thomas et al., 1980) however, negative results were obtained in rats on a FI-1 schedule with the drug diazepam (Valium) and also with chlorpromazine. Since chlordiazepoxide and diazepam are in the same class of drugs, such apparently contradictory findings are difficult to reconcile.

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de Lorge, J.O.
THE EFFECTS OF MICROWAVE RADIATION ON BEHAVIOR AND TEMPERATURE IN RHESUS MONKEYS
In C.C. Johnson and M. Shore (eds.), BIOLOGICAL EFFECTS OF ELECTROMAGNETIC WAVES, U.S. Dept. of Health, Education, and Welfare, Washington, D.C., HEW Publication (FDA) 77-8010, pp. 158-174 (1976)

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AUTHOR ABSTRACT: Male rhesus monkeys, trained to respond on an auditory vigilance task, were exposed to vertically polarized 2450 MHz microwaves in an anechoic room. Power densities of 4, 16, 32, 42, 52, 62, and 72 mW/sq cm, and exposure times of 30, 60, and 120 minutes were used. The monkeys performed the vigilance task in a Styrofoam restraint chair while irradiated from the front. Body temperature was monitored during exposure at all but the lowest power density.

Vigilance performance was not affected until 72 mW/sq cm illuminations occurred. Colonic temperature increase appeared to be a logarithmic function of power density from 16 to 72 mW/sq cm, whereas no such relationship was observed with behavioral indices. The animals showed adaptation to the microwaves in both behavioral and thermal measures, and thermal equilibrium was obtained except at 72 mW/sq cm.

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Study Type: Behavior, Physiology and Biochemistry, Ocular Effects, Metabolism and Thermoregulation; IN VIVO; RHESUS MONKEY
Effect Type: RFR-induced alterations of vigilance performance and rectal temperature during exposure
Frequency: 2.45 GHz
Modulation: Amplitude-modulated at 120 Hz, with 0.1-second pulses at 1 Hz superposed in some experiments
Power Density: 4 to 72 mW/sq cm
SAR: Not measured

EXPOSURE CONDITIONS: Each of 5 monkeys in a Styrofoam restraining chair was exposed frontally with vertically-polarized, far-field, 120-Hz-modulated RFR from a parabolic reflector or a standard-gain horn within an anechoic chamber at ambient temperature of 21-24 deg C and relative humidity of 55-70% and with white noise of about 79 dB present. All 5 monkeys were exposed to the modulated RFR (pulsed and unpulsed) at 4 and 16 mW/sq cm for 30 min during 1-hr behavioral-test sessions; 3 of them were also exposed to the unpulsed RFR at 16, 32, 42, 52, 62, and 72 mW/sq cm for 60 min during 2-hr test sessions; and 1 monkey was exposed to the unpulsed RFR at 16 mW/sq cm for entire 2-hr test sessions.

OTHER INFORMATION: The unpulsed 2.45-GHz RFR was modulated at 120 Hz (modulation percentage not stated). Discrete 0.1-second pulses superposed on the modulated RFR at a rate of 1 Hz by relay closures constituted the pulsed RFR. Relative power-density measurements were

made in horizontal planes at the levels of the head, chest, and abdomen (with the monkey absent). The results indicated that the highest values at any given incident power density were in the plane of the head. The mean values cited are for the head.

Each monkey was food-deprived to maintain it at 90-100% of its free-feeding weight and was trained in a Plexiglas chair for 70 sessions to perform a vigilance or observing-response task. In this task, the monkey was to press a Teflon lever in front of its right arm, which produced either a 1070-Hz, 85-dB tone for 0.5 second, to signal that no food pellet will be delivered, or a 2740-Hz, 82-dB tone that remained on until the monkey pressed a similar lever in front of its left arm. The latter response produced a pellet and extinguished the 2740-Hz tone.

Food was made available on a variable-interval 30-second (VI 30 s) schedule during 1-hr sessions and on a VI-60-s schedule during 2-hr sessions. This meant that during a 2-hr session, right-lever presses would produce the high tone about once per min and the low tone for the other right-lever presses. A left-lever response during the high tone yielded a pellet. Left-lever responses at other times caused a 10-s period during which right-lever presses only produced the low tone, a condition that inhibited extraneous left-lever responses.

After stable behavior was achieved and 122 subsequent sessions on the VI-30-s schedule were performed, the 5 monkeys were each exposed to the pulsed RFR at 4 and 16 mW/sq cm for 30 min in 1-hr sessions, during which they performed on the VI-30-s schedule. Similar sessions were conducted with the unpulsed RFR and with no RFR. No detectable effect on the performances of the monkeys was obtained with either the unpulsed or the pulsed RFR.

The negative results above led to use of only unpulsed RFR and of the VI-60-s schedule during 2-hr sessions of performance for the remainder of the investigation. Only 3 of the monkeys were used for these tests. Colonic temperatures were measured during some of the sessions.

Exposures at 16 mW/sq cm for 1 hr or 2 hr had no differential effect on the behavior of these 3 monkeys. The rectal temperature was measured on only 1 monkey, and the mean value showed no significant difference from control values.

Colonic temperatures were measured on all 3 monkeys during 1-hr exposures at 32, 42, 52, 62, and 72 mW/sq cm. Up to 62 mW/sq cm, increases to plateaus of about 0.3- to 1.0 deg C during the exposure period were obtained in all 3 monkeys. At 72 mW/sq cm, the increases were about 2 deg C at the end of the period, but the temperatures continued to rise, thus precluding studies at higher power densities.

The performances on the VI-60-s schedule, as measured by the right-lever response rate, showed no significant departures from control rates for all 3 monkeys up to 52 mW/sq cm and for 2 of the monkeys at 62 mW/sq cm. The mean performance of the third monkey at 62 mW/sq cm was about 80% of

its mean control performance. At 72 mW/sq cm, all 3 performed at about 50% of control values. Typically, a monkey at 72 mW/sq cm would accelerate its movements in the chair after about 20 min of exposure, take short naps after about 30 min, and sometimes appear to be deep asleep. Resumption of activity occurred about 10 min after cessation of exposure.

The latency time to make a pellet-producing left-lever response (reinforcement reaction time) was not significantly increased in 2 of the monkeys up to 62 mW/sq cm. For the third monkey, the latency time increased by about 20% at 52 and 62 mW/sq cm. At 72 mW/sq cm, the mean latency time of this animal was about 800% of control values. The corresponding values for the other 2 were 200% and 130%.

These results suggest that the monkeys reacted to the subtle body heating by the RFR at the higher power densities and that their performances were diminished because of such heat. Their behavior was further inhibited for about 10 min after cessation of the RFR, more so for the higher than the lower power densities. Such observations led the investigators to speculate that this post-exposure behavior may have been due to the relatively larger drops in colonic temperature after removal of the higher levels of RFR, and thereby indicating the need to measure behavioral performance during RFR exposure.

The 5 monkeys were also given standard physical examinations including ophthalmoscopy before and after each series of exposures. No clinically detectable abnormalities in eye structure or blood chemistry (no data presented) were found in any of them.

CRITIQUE: The description of the pulsed RFR used was obscure. It could be interpreted that the peak levels were much higher than the maxima for the modulated RFR, in which case the peak levels should have been stated. Alternatively, the description could be interpreted to mean that the RFR was on only during the pulsing periods, with no change in maximum levels but significant changes in average power density. However, this point is moot because no significant differences in behavior were found between the two forms of RFR.

The findings of this investigation are important because the study was one of the few carried out up to that time on primates trained to perform a complex vigilance or observing-response task during RFR exposure. A similar investigation on squirrel monkeys was performed subsequently by de Lorge (1979), with comparable findings.

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Radio Sci., Vol. 14, No. 6S, pp. 217-225 (1979)

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Radio Sci., Vol. 14, No. 6S, pp. 217-225 (1979)

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AUTHOR ABSTRACT: Squirrel monkeys, SAIMIRI SCIUREUS, were trained to respond on a two-lever observing-response task for food pellets. After stable response rates developed, the monkeys were exposed in the far field to 2.45-GHz microwaves (100% sinusoidally modulated at 120 Hz) at average power densities from 10 to 75 mW/sq cm of incident radiation. Four monkeys were exposed to irradiation for 30 minutes during two-hour sessions. Three of the monkeys were also exposed for 60 minutes during two-hour sessions. The animals were restrained in Styrofoam chairs during experimental sessions and exposures occurred in a microwave-anechoic chamber.

The behavior of the monkeys on the observing-response task was disrupted during the 30- or 60-minute exposures to irradiation but only at power densities that were 50 mW/sq cm or higher. This disruption was increasingly evident as power density increased. Under both durations of exposure, behavior was not consistently perturbed until rectal temperatures increased more than 1 deg C. Rectal temperature was slightly but reliably elevated at 10 mW/sq cm, was a monotonic function of the power density, and was markedly increased at power densities between 40 and 50 mW/sq cm.

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Study Type: Behavior, Physiology and Biochemistry, Metabolism and Thermoregulation; IN VIVO; SQUIRREL MONKEY
Effect Type: RFR-induced disruptions of observing-response task
Frequency: 2.45 GHz
Modulation: Amplitude-modulated at 120 Hz
Power Density: 10-75 mW/sq cm
SAR: Not given

EXPOSURE CONDITIONS: Each of 4 monkeys in a Styrofoam restraining chair was exposed from above for 0.5-hr periods to far-field, 120-Hz-modulated RFR at constant power densities in the range 10-70 mW/sq cm from a standard-gain horn within an anechoic chamber at an ambient temperature of about 23 deg C, with about 79 dB of white noise present. Three of the monkeys were also exposed for 1-hr periods. The mean relative humidity was about 57% during 0.5-hr exposures and 74% for 1-hr exposures.

OTHER INFORMATION: Each monkey was food-deprived and performed at 74-77% of free-feeding body mass. It was initially trained, in 1-hr sessions, to press either the right or the left of 2 Teflon levers on top of the chair to obtain a food pellet. Red and blue incandescent lights in front of the monkey were turned on alternately with each lever

press. After each animal pressed one or both levers consistently during at least 3 sessions, the contingencies were changed so that a left-lever press only during blue-light illumination was rewarded; depression of the right lever continued to alternate the red and blue lights.

Training progressed in stages, during subsequent sessions, each stage consisting of increasing the number of right-lever responses needed to turn on the blue light. The end result was performance on a schedule in which each right-lever response yielded either 0.5 second of red light or 10 seconds of blue light. Only a left-lever press during the latter yielded a pellet. The blue light occurred on an average of once per min (a variable interval of 1 min, or VI-1-min schedule) contingent on a right-lever response.

After stable behavior was achieved, each monkey was exposed to RFR at constant power density in the range 10-70 mW/sq cm in 10-mW/sq-cm increments. The exposures were done daily during the middle 30 min of 1-hr testing sessions, with the other 15-min periods providing baseline data. Twenty such sessions were conducted without rectal-temperature measurements. Sham exposures were conducted between sessions at each power density. The next 21 RFR-exposure sessions were similar but included rectal-temperature measurements with a commercial probe. (Field perturbation of or by the probe was discounted because the RFR was incident from above the head.)

For the remaining 53 sessions, only 3 of the monkeys were tested, the session duration was 2 hr, and the exposures were for the middle 60 min. The number of sessions at each power density ranged from 2 to 5, with sham exposures between sets. The order of exposure with respect to power density was varied, but most exposures at 60 and 70 mW/sq cm were done near the end of the sequence, and the last 3 sessions involved exposures at 75 mW/sq cm. Rectal temperatures were measured during all sessions.

Neither of the RFR-exposure regimens caused any obvious permanent physical changes in any of the monkeys.

The behavioral results for the 30-min exposures without rectal-temperature probes were similar to those with such probes. Among the variety of performance measures on the observing-response task, only the right-lever-response rate indicated an RFR-induced change. This measure, expressed in percentage of mean control value, showed a slight trend toward lower rates with increasing power density to a minimum of about 90% at 60 mW/sq cm and a slightly higher value (92%) at 70 mW/sq cm. However, the mean response rate (%) was never larger than 1 standard deviation from 100%.

A recurring effect on response rate was a cessation of responses within 30 seconds of commencement or termination of RFR exposure even though no mechanical vibrations or audible noises were detected by the investigator at such times. The "on effect" was noticed first in 1 monkey at 40 mW/sq cm but was less evident at higher levels. The "off effect" did not occur consistently in all animals except at 70 mW/sq cm, where the pause was more pronounced than for the on effect.

The pre-exposure mean rectal temperature was 38.8 +/- 0.3 deg C, or about 0.8 deg C lower than the norm for the restrained squirrel monkey, presumably due to extensive chairing and handling. The mean rectal-temperature rise (difference between rectal temperature at termination and initiation of exposure) for the 30-min exposures showed a significant ($p<0.001$) linear trend from sham exposure to 70 mW/sq cm. However, the differences between the mean for sham exposure and the means for power densities to 30 mW/sq cm were not significant ($p>0.05$), whereas those for 50, 60, and 70 mW/sq cm differed significantly from each other and from those below 40 mW/sq cm.

Presumably but not explicitly stated, the temperatures at termination of RFR exposure were plateau values reached before the end of the exposure period. However, sham exposures yielded small temperature rises (0.36 +/- 0.13 deg C) with no indication of plateaus, which compounded the difficulty of removing the contributions of restraint to the results for RFR exposure.

The mean rectal-temperature rises for the 1-hr exposures at 30 mW/sq cm or less (during the 2-hr test sessions) were lower than the rises for the 30-min exposures (during the 1-hr sessions) at the corresponding levels, an indication that the contributions from restraint per se were smaller during the longer than the shorter exposures.

Plots of mean rectal-temperature rise (on a logarithmic scale) vs power density (on a linear scale) for the 3 monkeys exposed for 1 hr showed qualitatively similar nonmonotonic increases for power densities up to about 40 mW/sq cm, an abrupt shift upward between 40 and 50 mW/sq cm, and monotonic increases for the higher power densities. The investigator surmised that the monkeys were able to equilibrate to the heat loads below 50 mW/sq cm and that the animals were unable to dissipate the loads at the higher levels within the 60-min exposure periods. The temperature rises at 75 mW/sq cm were close to lethality, so exposure was terminated whenever a monkey's temperature exceeded 42.5 deg C.

The general behavioral effects of 1-hr exposures were similar to those of 30-min exposures but were more pronounced. No consistent behavioral changes occurred below 50 mW/sq cm, and above that level, the effects increased with power density. The right-lever-response rate vs power density varied widely among the 3 animals, but at 60 mW/sq cm, all showed decrements to about 60% of control values.

Two monkeys exhibited the off effect (pauses following termination of RFR exposure) only. The third monkey exhibited the on effect (pauses on initiation of exposure) as well. Occasionally, this monkey also would respond by spuriously pressing both levers simultaneously, which precluded reinforcement, but such responses apparently were not RFR-induced because they also occurred during control sessions. The off-effect pauses, which were longer than those for the on effect, were ascribed to the larger differential rates of rectal-temperature change

at the end than at the beginning of exposure. An effect similar to the off effect was observed in an earlier study with the rhesus monkey exposed frontally (de Lorge, 1976).

Below 60 mW/sq cm, there were no large differences from control values of the time delay in food-lever response when food was available (reinforcement reaction time), but large increases were evident above that level, with wide differences among the animals.

One other effect, obvious in only 1 monkey, was a reliable increase in the number of incorrect left-lever responses with power density. Other behavioral indices, including post-reinforcement time and left-lever-response rate, showed no consistent RFR-induced changes.

The investigator concluded that the behavioral changes observed were temporary and obviously related to hyperthermia, with consistent results when the rise in rectal temperature exceeded 1 deg C, corresponding to a power-density threshold between 40 and 50 mW/sq cm. He also obtained similar results with the rhesus monkey tested for the same behavioral task during exposure to 2.45-GHz RFR, but with a threshold 10 to 20 mW/sq cm higher (de Lorge, 1976), and suggested that RFR-induced behavioral changes in different species may be scaled on the basis of body mass.

CRITIQUE: From the description of the same RFR source in Sanza and de Lorge (1977), the magnetron presumably was powered from an unfiltered full-wave rectifier. This type of power supply does yield sinusoidal voltage half-cycles of one polarity at 120 Hz. However, although the magnetron was turned on and off at 120 Hz, the RFR modulation was not sinusoidal because a threshold voltage well above zero must be attained during each half-cycle for the magnetron to start oscillating, and the magnetron ceases oscillating well before reaching zero on the downward part of the half-cycle.

For these reasons, a magnetron with this type of power supply operates only during part of each half-cycle, yielding a duty cycle significantly less than 1. Moreover, if the desired level of average output RFR power is set by phase control (altering the fraction of the half-cycle during which the magnetron is oscillating), then the duty cycle varies directly with the output-power setting. At low average-power settings (small duty cycles) the peak powers would be correspondingly high. Although the information provided in this paper was inadequate to determine the duty cycles, it appears unlikely that the peak powers used were high enough to produce the auditory-RFR effect.

Lacking in this investigation were measurements or estimates of mean SAR for the head and/or whole body. Nevertheless, the findings of this study, reinforced by the similar results with the rhesus monkey (de Lorge, 1976), are important because the performance measurements of a complex behavioral task during RFR exposure were carried out with two species much closer to human physiology and intelligence than the more commonly used laboratory animals and because reasonably accurate power-

density thresholds for each species were determined. However, the body-mass scaling concept may be too simplistic, and additional experimental evidence with other species would be needed to support it.

Regarding the on effect and the off effect, it is possible that the pauses by the monkeys were behavioral responses to sensory cues of sudden onset or removal of a heat source.

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Sanza, J.N. and J. de Lorge
FIXED INTERVAL BEHAVIOR OF RATS EXPOSED TO MICROWAVES AT LOW POWER DENSITIES
Radio Sci., Vol. 12, No. 6S, pp. 273-277 (1977)

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DELORGE

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BEHAVIOR
IN-VIVO
METABOLISM
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Sanza, J.N. and J. de Lorge
FIXED INTERVAL BEHAVIOR OF RATS EXPOSED TO MICROWAVES AT LOW POWER DENSITIES
Radio Sci., Vol. 12, No. 6S, pp. 273-277 (1977)

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AUTHOR ABSTRACT: Behavioral effects of 2.45-GHz microwaves (100% amplitude modulated at 120 Hz) were studied at averaged power densities of 8.8, 18.4 and 37.5 mW/sq cm of incident radiation as measured in the absence of the animal subjects. Four rats were exposed for 60 min while performing in a response chamber of Styrofoam. Lever pressing was reinforced by food pellets on a fixed-interval 50-sec schedule and produced high rates of responding in two rats and low rates of responding in the other two rats.

Radiation at 37.5 mW/sq cm disrupted the lever response in the two rats that responded at high rates during baseline measures. Radiation at the two lowest power densities had no observable effect on the rate of responding. For the two rats that responded at low rates, no effects on rate of lever pressing were observed at any of the power densities. However, ambulatory activity of all rats decreased during radiation at 18.4 and 37.5 mW/sq cm. This decrease was associated with a tendency of the rats to remain in areas of the response chamber with lower power densities of irradiation. The results are believed to be due to an interaction between higher metabolic rates in the more frequently responding rats and exogenous heating by microwave radiation.

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Study Type: Behavior, Metabolism and Thermoregulation; IN VIVO; RAT
Effect Type: RFR-induced alterations of behavior on a fixed-interval schedule
Frequency: 2.45 GHz
Modulation: Amplitude-modulated at 120 Hz
Power Density: 8.8, 18.4, and 37.5 mW/sq cm
SAR: Not given

EXPOSURE CONDITIONS: Each of 4 rats in a Styrofoam operant-conditioning chamber 24x30 cm horizontally and 30 cm vertically was sham-exposed or exposed to RFR from above with a standard-gain horn within an enclosure lined with RFR absorber and maintained at an ambient temperature of 24 deg C, relative humidity of about 70%, and air-flow rate of 1.52-3.44 m/min. With exhaust fans operating, the white-noise sound-pressure level (SPL) within the closed chamber was 76 dB. Exposures were for 60 min, with the polarization parallel to the 30-cm horizontal dimension.

OTHER INFORMATION: The floor of the operant-conditioning chamber was a grid of polyethylene. Methylacetate was used as the chamber cover and wall lining to minimize damage from biting and scratching. The food cup and response lever were of Plexiglas, which was resistant to abrasion

from clawing and licking. The power densities within the chamber (in the absence of the rat) were mapped horizontally 4 cm above the floor with a probe. The variations (+/-) from the spatial averages used for exposure were (in mW/sq cm): 5 at 37.5, 3 at 18.4, and .2 at 8.8.

The magnetron used as the RFR source was powered from an unfiltered full-wave rectifier and the RFR forward power was detected and stabilized by a feedback loop to a control unit. The cooling fan for the unit was switched on during sham- as well as RFR exposures, and the slight increase in noise (above the white-noise level) startled the rats.

The 4 rats studied were screened from 11 rats; the other 7 were not used because of their tendency to bite and claw at the chamber during training. Each rat was maintained at 80% of its free-feeding body mass.

In 3 daily practice sessions of 1-2 hr, each rat was trained to press the lever to obtain a food pellet. The rats were then trained, in 3-day sequences of 1-hr-daily sessions, to respond on a fixed-interval 15-second (FI-15-s) schedule the first day, a FI-30-s schedule the second day, and a FI-50-s schedule the third day. After 5 such sessions per week for 6 weeks, the rats were able to respond on the FI-50-s schedule for at least 22 consecutive daily 1-hr sessions at the same time each day (between 0900 and 0930 for the first rat, followed by the others at about 90-min intervals). No water was provided during sessions.

After completion of training, the rats were sham-exposed or exposed for 60 min at 8.8, 18.4, or 37.5 mW/sq cm. To minimize carry-over effects, the sequence of exposure at each power density was varied from rat to rat and each value was used only once in each sequence. In addition, sham-exposure sessions were conducted after each day of RFR exposure and when necessary to restabilize training. Water was provided between sessions.

Throughout the baseline sessions, 2 of the rats responded with lever presses at relatively high rates and the other 2 at relatively low rates, performances that were generally maintained except at the highest power density. No obvious changes were caused by RFR exposure in distribution of responses within the FI or in the interresponse-time distributions. Also, the lever-response rate of none of the rats was altered significantly at 8.8 or 18.4 mW/sq cm. However, the response rates of the high-performance rats diminished greatly at 37.5 mW/sq cm, and these rats showed signs of overheating on removal from the chamber. The low-performance rats did not exhibit such changes or overheating.

For sham exposures, the mean pause times were 40.7 and 30.6 seconds for the low- and high-rate rats, respectively. At 8.8 and 18.4 mW/sq cm, none of the rats showed statistically significant changes from these values. At 37.5 mW/sq cm, the mean value for the low-rate rats was 45.4 seconds, an increase that was not statistically significant; for the high-rate rats, the mean value was 48.4 seconds, a highly significant increase.

The pause times for the high-rate rats became progressively longer across successive fixed intervals, and were related to the apparent preference for the one area (near the wall on the opposite side of the food cup) found to have a relative power-density depression in the horizontal mapping of the chamber. The behavior of the low-rate rats was similar but less pronounced. During sham exposure or exposure at 8.8 mW/sq cm, all 4 rats spent only 3-4% of the session time in that area. However, at 18.4 and 37.5 mW/sq cm, the low-rate rats spent about 56% and 78% of the time there, respectively, whereas the corresponding values for the high-rate rats were 81% and 92%.

The investigators ascribed their results to "workload-microwave interaction," and indicated that the pronounced effects at 37.5 mW/sq cm on the high-rate rats were due to the significant thermal burden added to their high metabolic rates. The investigators also stated that "well-practiced operant behaviors, such as we used, may be impervious to the influence of radiation at low power densities."

CRITIQUE: Characterization of the RFR as 100% amplitude modulated at 120 Hz appears to be in error, because the magnetron used as the RFR source was powered from an unfiltered full-wave rectifier. This type of power supply does yield sinusoidal voltage half-cycles of one polarity at 120 Hz. However, although the magnetron was turned on and off at 120 Hz, the RFR modulation was not sinusoidal because a threshold voltage well above zero must be attained during each half-cycle for the magnetron to start oscillating, and the magnetron ceases oscillating well before reaching zero on the downward part of the half-cycle.

For these reasons, a magnetron with this type of power supply operates only during part of each half-cycle, yielding a duty cycle significantly less than 1. Moreover, if the desired level of average output RFR power is set by phase control (altering the fraction of the half-cycle during which the magnetron is oscillating), then the duty cycle varies directly with the output-power setting. At low average-power settings (small duty cycles) the peak powers would be correspondingly high. Although the information provided in this paper was inadequate to determine the duty cycles, it appears unlikely that the peak powers used were high enough to produce the auditory-RFR effect.

The description of the exposure facility did not include details regarding how the ambient temperature within the operant-conditioning chamber was maintained at 24 +/- 0.6 deg C. Not clear is whether the temperature within the chamber was monitored during RFR exposures and used for feedback control. If not, then the heat rising through the grid floor from the RFR-absorbent walls of the enclosure beneath the chamber may have significantly increased the temperature within the chamber, especially at the higher power densities.

As indicated by the results at the higher power densities, the rats endeavored to minimize their heat loads by spending more than 50% of the session periods in the area of minimum power density. Even though such behavior is an indication of the thermal basis of the effects observed,

the existence of such power-density minima may have artifactually altered the numerical values of response rate and pause duration, a point implied by the investigators.

Their suggestion that behavioral measures other than those used may be more sensitive to low RFR power densities is open to experimental verification. In two other studies by de Lorge (1976, 1979), the subjects were primates and the behavioral measures used were variable-interval (VI) schedules. Both yielded positive results at the higher power densities used and were clearly thermally based, and negative results at the lower power densities.

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OPERANT BEHAVIOR AND RECTAL TEMPERATURE OF SQUIRREL MONKEYS DURING 2.45-GHZ MICROWAVE IRRADIATION
Radio Sci., Vol. 14, No. 6S, pp. 217-225 (1979)

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BEHAVIOR
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de Lorge, J.O. and C.S. Ezell
OBSERVING-RESPONSES OF RATS EXPOSED TO 1.28- and 5.62-GHZ MICROWAVES
Bioelectromagnetics, Vol. 1, No. 2, pp. 183-198 (1980)

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AUTHOR ABSTRACT: The effects of microwave irradiation at two different frequencies (1.28 and 5.62 GHz) on observing-behavior of rodents were investigated. During daily irradiation, eight male hooded rats performed on a two-lever task; depression of one lever produced one of two different tones and the other lever produced food when depressed in the presence of the appropriate tone. At 5.62 GHz, the observing-response rate was not consistently affected until the power density approximated 26 mW/sq cm; at 1.28 GHz, the observing-response rate of all rats was consistently affected at a power density of 15 mW/sq cm. The respective whole-body specific absorption rates (SARs) were 4.94 and 3.75 W/Kg.

Measurements of localized SAR in a rat-shaped model of simulated muscle tissue revealed marked differences in the absorption pattern between the two frequencies. The localized SAR in the model's head at 1.28 GHz was higher on the side distal to the source of radiation. At 5.62 GHz the localized SAR in the head was higher on the proximal side. It is concluded that the rat's observing behavior is disrupted at a lower power density at 1.28 than at 5.62 GHz because of deeper penetration of energy at the lower frequency, and because of frequency-dependent differences in anatomic distribution of the absorbed microwave energy.

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Study Type: Behavior; IN VIVO; RAT
Effect Type: Thresholds for alterations of rat behavior at 2 frequencies
Frequency: 1.28 and 5.62 GHz
Modulation: 3-microsecond pulses at 370 pps (0.0011 duty) for 1.28 GHz; 0.5- or 2-microsecond pulses at 662 pps (0.00033 or 0.0013 duty) for 5.62 GHz
Power Density: 0-15 mW/sq cm at 1.28 GHz; 0-48.5 mW/sq cm at 5.62 GHz
SAR: 0.25 and 7 W/kg per mW/sq cm whole-body and head at 1.28 GHz; 0.19 and 5.5 W/kg per mW/sq cm whole-body and head at 5.62 GHz

EXPOSURE CONDITIONS: Each rat, in a Styrofoam operant-conditioning box, was sham-exposed or exposed to RFR from a horizontally radiating horn in an anechoic chamber both of which were designed for each frequency. Power density was varied at 1.28 GHz by altering the distance between the box and the horn, and at 5.62 GHz by changing the duty cycle as well. All exposures were in the far field except at 16 mW/sq cm for 5.62 GHz, which was done at 90% of the conventional far-field distance from the horn.

Exposure sessions were for 40 min, with the E-vector vertical and with the right side of the rat toward the horn while the rat was performing. Masking noise of about 78 dB was present during exposures. Ambient temperature varied with building values in the range 23-26.5 deg C, with small increases within the box due to RFR-generated heat in the wall opposite the horn in each chamber. Relative humidities were about 50%.

OTHER INFORMATION: Power density measurements were made with no rat present. Whole-body SARs were measured for Styrofoam models in the shape of a standing rat and filled with saline. The mean values were 0.25 W/kg per mW/sq cm at 1.28 GHz and 0.19 W/kg per mW/sq cm at 5.62 GHz. Local SARs were measured in the head, shoulder, abdomen, and hip (12 locations) within the models filled with muscle-equivalent synthetic material. The results (per mW/sq cm) were a relative maximum SAR of about 5.5 W/kg in the left side of the head (the side away from the source) at 1.28 GHz and a relative maximum of about 7 W/kg in the right side of the head at 5.62 GHz.

The top of the operant-conditioning box was covered with a nylon screen, a polystyrene grid served as the floor, and a tray well below the floor was used for waste collection. Two levers and a food hopper between them were mounted in the right side wall (as seen from the source) of the box. Two speakers, a 100-W lamp for general illumination, and a video monitor were mounted on the wall behind the horn of each chamber. A 25-W stimulus lamp was mounted in each ceiling above the box.

Eight rats maintained at well below their free-feeding body masses were given 40-min training sessions daily for 5 days per week to achieve a behavioral response that required the rat to depress the right lever, thereby producing a 1-kHz tone for 0.7 s or a 1.25-kHz tone for 10 s. Right-lever responses that yielded the 1.25-kHz tone were reinforced on the average of once every 20 s (variable-interval of 20 s, or VI-20-s schedule); otherwise the 1-kHz tone was presented. Depression of the left lever during the 10 s of the higher tone resulted in its cessation and the delivery of a food pellet. Depression of the left lever in the absence of the higher tone yielded a 10-s period during which right-lever responses produced only 0.7-s intervals of the lower tone. If the left lever was not pressed by the end of the 10-s of the higher tone, the VI-20-s schedule would recycle.

After completing training (90 sessions), the rats were exposed to the 5.62-GHz RFR at power densities of 7.5, 11.5, 16, 26, 31.5, 38.5, 42, and 48.5 mW/sq cm, comprising a total of 183 sessions. The mean of their body masses during this phase was 362 g or about 88% of the mean free-feeding mass. The whole-body SARs ranged from 1.4 to 9.2 W/kg.

About 90 days later, the rats were exposed to the 1.28-GHz RFR at power densities of between 0.1 and 1 mW/sq cm (obtained by interposing a sheet of absorber between the horn and the box), 5.5, 9.5, 10, and 15 mW/sq cm, totaling 62 sessions. The mean of their body masses was 400 g or about 90% of the mean free-feeding mass. The whole-body SARs ranged from 0.025 to 3.75 W/kg.

Sham exposures were conducted on days preceding and following RFR-exposure sessions. No overt physiological signs of hyperthermia were evident for either RFR frequency.

The results showed consistent disruption of the behavior pattern, manifested as overall reductions of right-lever response rates during RFR exposure, long pauses unrelated to pellet delivery, and complete cessation of responding after 15-20 min of exposure. Behavior disruption was consistent in all 8 rats for 5.62 GHz at 38.5 mW/sq cm and higher, and for 7 of the rats at 26 mW/sq cm and higher. For 1.28 GHz, consistent disruption was obtained in all rats at 15 mW/sq cm, and a statistically significant drop in mean response rate to about 88% of the mean sham-exposure rate was evident at 10 mW/sq cm. However, some of the rats showed habituation (i.e., less disruption) to successive RFR exposures at the same power density (with interposed sham-exposure sessions).

Incorrect left-lever or false-detection responses (made in the absence of the food-availability tone) were affected in similar fashion. For 1.28-GHz RFR, their frequencies decreased substantially at 10 mW/sq cm and higher, and similarly for 5.62-GHz RFR at 26 mW/sq cm and higher. However, the ratio of false detections to total observing-responses increased significantly with increasing power density.

The pause time for a correct left-lever response following a right-lever reinforced detection-response was also found to increase with increasing power density, with reliable differences evident at 10 mW/sq cm for 1.28 GHz and at 16 mW/sq cm for 5.62 GHz.

The pause time for correct left-lever responses following reinforcement right-lever responses also increased with increasing power density, and were manifested at 10 mW/sq cm for 1.28 GHz and at 16 mW/sq cm for 5.62 GHz.

The investigators concluded that the observed behavioral disruptions were "almost certainly related to the thermal consequences of such radiation" and suggested that the widely different thresholds for behavioral disruption at the two frequencies, i.e., 10 mW/sq cm (whole-body SAR of 2.5 W/kg) for 1.28 GHz and 26 mW/sq cm (4.9 W/kg) for 5.62 GHz, may have been due to the different spatial SAR variations within the rats.

CRITIQUE: The pulsed RFR used may have yielded the auditory-RFR effect. If so, however, the investigators were probably correct in discounting this phenomenon as a factor in their results, because the pulse repetition rates used (370 and 662 pps) were much lower than the 1-kHz and 1.25-kHz tones used in the behavioral regimen.

The findings of this investigation with 1.28- and 5.62-GHz RFR were qualitatively similar to those of previous behavioral studies with 2.45-GHz RFR in this laboratory with rats on fixed-interval schedules (Sanza and de Lorge, 1977) and with primates on variable-interval schedules (de Lorge, 1976 and 1979).

It was unfortunate that the investigators had not measured rectal temperatures of the rats during RFR exposures in this study as had been done by Sanza and de Lorge (1977), because such measurements would have undoubtedly supported the conclusion regarding the thermal basis of the findings of the present study. Moreover, their measured values of local SAR in muscle-equivalent phantoms (5.5 and 7 W/kg per mW/sq cm) were clearly thermogenic.

Their suggestion that the apparent discrepancy in thresholds for behavioral disruption for the two frequencies was ascribable to differences in internal SAR distributions is well founded, because the SAR maxima in the head were 5.5 W/kg per mW/sq cm at 1.28 GHz and 7 W/kg per mW/sq cm at 5.62 GHz. The ratio of the latter to the former is only about 1.3 as compared with a ratio of 2 for the corresponding whole-body SARs. However, because of differences in spatial SAR distributions, frequency scaling of effects in a single species or comparisons of results in one species with those in another species on the basis of whole-body SARs appears unjustified.

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BEHAVIOR
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AUTHOR ABSTRACT: Behavioral and thermal effects of radiating an animal with differing wavelengths of microwave energy at the same power density were investigated in the first of two studies. Five Long-Evans rats were trained to perform a lever-pressing task and were rewarded with food on a variable interval schedule of reinforcement. Rats were individually exposed in random order to 400-, 500-, 600-, and 700-MHz CW radiation at a power density of 20 mW/sq cm with the long axis of the rat's body parallel to the vector of the electric field. Radiation at all wavelengths produced rises of body temperature and stoppage of lever pressing. The averaged rise in body temperature was greatest and work stoppage was most rapid during exposures at 600 MHz.

In the second study, six rats were exposed in random order to 600-MHz CW radiation at power densities of 5, 7.5, 10, and 20 mW/sq cm while performing the same behavioral task. Exposures at 10 and 20 mW/sq cm resulted in work stoppage, while exposures at 5 and 7.5 mW/sq cm did not. In addition, three of the rats were subsequently exposed while responding to 600-MHz pulsed radiation (1000 pps, 3- or 30-microsecond pulse durations at a peak power density of 170 mW/sq cm (averaged 0.51 and 5.1 mW/sq cm). No work stoppage occurred to pulsed radiation.

Taken in sum, the data show that the mature Long-Evans rat is resonant at a frequency near 600 MHz while work stoppage during short-term exposures to 600-MHz radiation occurs at a power density between 7.5 and 10 mW/sq cm.

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Study Type: Behavior; Exposure Methods, Dosimetry, and Modeling; Physiology and Biochemistry; IN VIVO; RAT
Effect Type: Frequency- and power-density dependence of behavior alteration in the rat.
Frequency: 400, 500, 600, and 700 MHz
Modulation: CW and 3- or 30-microsecond pulses at 1000 pps
Power Density: 5, 7.5, 10, and 20 mW/sq cm for CW; 170 mW/sq-cm Pk, 0.51 and 5.1 mW/sq-cm Av for pulsed
SAR: Not stated; estimated as 0.8 W/kg per mW/sq cm at 600 MHz.

EXPOSURE CONDITIONS: The RFR source was a quarter-wavelength monopole (for each frequency) inserted horizontally through one copper wall of the exposure chamber, with the wall serving as the ground plane. The monopole was 33 cm from one of the adjacent walls, on which was mounted a 45-deg copper reflector. A Plexiglas rat-response cage was mounted on the ground plane at the same height as, and 143 cm from, the monopole.

The wall opposite the monopole and the adjacent halves of the ceiling, floor, and remaining wall were covered with RFR-absorption material. The floor and ceiling of the rat-response cage were grids of equally spaced Plexiglas rods. The cage contained a plastic response-lever and food-pellet feeder, which were remotely instrumented. The cage was oriented such that the long axis of the rat would be parallel to the E-vector during operation of the lever.

The ambient temperature and relative humidity of the exposure chamber were maintained at 21-22 deg C and 27%, respectively. Closed-circuit TV was used to monitor the rats during exposure. Exposures to RFR were for 55 min unless the rat stopped performing sooner. Water was not provided during exposures.

OTHER INFORMATION: Eleven rats that had initial body masses ranging from 420 to 450 g and a mean length of 19 cm from snout to base of tail were used. They were maintained at 85% of their free-feeding body mass and were trained, during daily 1-hr sessions, to respond on a variable-interval schedule of reinforcement. The clock time was 3 seconds, with a 10% probability of pellet delivery, thus yielding delivery at 3-second intervals or longer with an average interval of 30 seconds. Training sessions were conducted until stable performance was achieved, defined as when the total number of lever presses during a session differed by less than 15% from the total of a previous session. An average of 14 sessions was necessary to achieve stability. In addition, the rats were given 4 to 6 retraining sessions after each RFR-exposure session.

After training, 5 rats were each exposed in random order to 400-, 500-, 600-, and 700-MHz CW RFR at 20 mW/sq cm, starting on the fifth min of each 1-hr session. Exposure was continued until the end of the session or was terminated at the instant of work stoppage, defined as the end of the first min during which its response rate fell below 33% of its normal rate. The primary behavioral measure was the time interval to such work stoppage. Colonic temperatures were measured just before and immediately after RFR exposure and some training sessions.

The results for these 5 rats showed that the most rapid stoppage of work and the highest elevations of body temperature occurred at 600 MHz. The mean duration to work stoppage at this frequency was about 23 min as compared with about 51 min at 400 MHz, 37 min at 500 MHz, and 33 min at 700 MHz. By analysis of variance, the differences between the value at 600 MHz and the values at 500 and 700 MHz were significant ($p<0.05$), and all three durations differed significantly ($p<0.01$) from the value at 400 MHz. Comparison of total numbers of responses obtained in training sessions before and after RFR-exposure sessions showed no significant influence of RFR on post-RFR sessions.

The rise of colonic temperature during exposure was divided by the exposure duration as an indicant of energy delivered. The mean value of this measure (in deg C/min) at 600 MHz was 0.088 as compared with 0.024 at 400 MHz, 0.049 at 500 MHz, 0.063 at 700 MHz, and about 0.01 for training sessions. All the differences were statistically significant ($p<0.01$).

As noted by the investigators, these results indicated experimentally that 600 MHz is close to the frequency of resonance absorption for rats weighing 380-400 g and 19-20 cm in length (exclusive of tail) exposed with their long axes parallel to the E-vector, thereby confirming analytical calculations for a prolate-spheroidal model of a medium rat in the same orientation (Gandhi, 1974; Johnson et al., 1976).

The coefficient of product-moment correlation between duration to work stoppage and colonic-temperature rise with frequency was -0.78 ($p<0.001$). At the end of RFR exposure (at 20 mW/sq cm), all the rats were clearly stressed by heat. On removal, they were prone and immobile, and exhibited signs of vasodilation, but recovered within minutes.

The other 6 trained rats were each exposed in random order to 600-MHz CW RFR at 20, 10, 7.5, and 5 mW/sq cm, and similarly evaluated. Work stoppage invariably occurred at 20 and 10 mW/sq cm; the mean durations to work stoppage were 25 and 44 min, respectively. The corresponding mean rates of colonic-temperature rise were 0.066 and 0.043 deg C/min. The durations at 7.5 and 5 mW/sq cm were not significantly different from 55 min, and the corresponding colonic-temperature rises were 0.019 and 0.017 deg C. The coefficient of product-moment correlation between duration to stoppage and temperature rise with power density was -0.83 ($p<0.01$), and again there was no significant influence of RFR exposure on post-exposure sessions.

Three of these 6 rats were also exposed to 600-MHz pulsed RFR at a peak power density of 170 mW/sq cm and similarly evaluated. Pulse durations of 3 and 30 microseconds at 1000 pulses per second were used, which yielded average power densities of 0.51 and 5.10 mW/sq cm. All 3 rats responded at near normal rates, and the colonic-temperature rises were small (no data presented). One rat stopped responding near the end of a session of 3-microsecond pulses. The investigators noted that the energy in each 30-microsecond pulse was about half that found necessary by Guy et al. (1975) as the energy threshold for the auditory-RFR effect.

CRITIQUE: Apparently no sham-exposure sessions were conducted, i.e., all training and retraining presumably were done outside the exposure chamber. Sham exposures are often necessary, especially in experimental protocols that may be stressful to the animals, to minimize the effects of uncontrolled non-RFR factors. However, perhaps no sham exposures were necessary in this investigation because the results appear to be clear and unequivocal.

No measurements or estimates of SAR were presented. However, since 600 MHz was close to the calculated resonance in Johnson et al. (1976) for the medium rat, the corresponding calculated normalized SAR, about 0.8 W/kg per mW/sq cm, can be taken as a reasonable estimate. At 20 mW/sq cm, therefore, the whole-body SAR was about 16 W/kg. Also, because significant decreases in duration to work stoppage were evident for 600 MHz at 10 mW/sq cm but not at 7.5 mW/sq cm, the SAR threshold for this behavioral measure is probably between 6 and 8 W/kg.

The investigators suggested that other behavioral tests may be more sensitive to low power densities. In subsequent studies of rats (D'Andrea et al., 1979 and 1980), alterations of locomotor activity on a rotating wheel were nonsignificant ($p>0.05$) after chronic exposure to 2450- or 915-MHz RFR at 5 mW/sq cm, but a significant depression of performance on a stabilimetric platform was reported for 2450 MHz. However, this effect was not a disruption of performance on a learned task during RFR exposure.

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J. Microwave Power, Vol. 14, No. 4, pp. 351-362 (1979)

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AUTHOR ABSTRACT: Long-Evans male adult rats were exposed for sixteen weeks to 2450-MHz CW microwaves at an average power density of 5 mW/sq cm. The resulting dose rate was 1.23 (+/- 0.25 SEM) mW/g. The animals were exposed eight hours a day, five days a week, for a total of 640 h in a monopole-above-ground radiation chamber while housed in Plexiglas holding cages. Daily measures of body mass and of food and water intakes indicated no statistically significant effects of microwave irradiation. Biweekly stabilimetric tests immediately after exposure revealed a significant depression of behavioral activity by 15 microwave-exposed rats as compared with 15 sham-exposed animals. Measures of locomotor activity based on revolutions of a running wheel, which were obtained during 12-h periods between each 8-h exposure, showed no significant effect of irradiation.

Blood sampled after 2, 6, 10, and 14 weeks of exposure indicated slight alterations of sulfhydryl groups, and of red and white blood-cell counts. Measures of levels of 17-ketosteroids in urine at weeks 1, 5, 9, and 12 of exposure, and mass of adrenals, heart, and liver at the end of the sixteen-week period of exposure, revealed no indications of stress.

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Study Type: Behavior, Physiology and Biochemistry, Immunology and Hematology; IN VIVO; RAT
Effect Type: Alterations of body and organ masses, food and water intake, locomotor activity, and blood chemistry by chronic RFR exposure
Frequency: 2.45 GHz
Modulation: CW
Power Density: 5 mW/sq cm
SAR: 0.9-1.23 W/kg

EXPOSURE CONDITIONS: Exposures to RFR were done in the 2 halves of a chamber lined with pyramidal RFR-absorption materials and partitioned vertically with aluminum-covered sheets that served as ground planes. A quarter-wavelength monopole was mounted horizontally on each ground plane, and 10 rectangular rat-holding cages were centered around each monopole in a circular array of 90-cm radius with their long horizontal axes parallel to the monopole. The floor and ceiling of each rat cage were grids of equally spaced Plexiglas rods, and the other walls were of Plexiglas sheet. A type 2M53 magnetron was the RFR source.

Sham-exposures were performed in a chamber of the same design and interior dimensions, but was lined with Styrofoam sheet and sprayed with gray acoustic material to match the lighting and sound conditions of the RFR chamber. Both chambers were equipped with houselight, ventilating fans, and loudspeakers for providing white noise of about 70 dB.

RFR- and sham-exposures were from 0900 to 1700 (8 hr/day), 5 days/week for 16 weeks. No food or water was provided in either chamber.

OTHER INFORMATION: The power density at each cage location in the 2 halves of the RFR-exposure chamber was measured in the absence of rats. The values for the 10 locations in the left chamber ranged from 4.92 to 5.84 mW/sq cm, with a mean of 5.42 +/- 0.21 (SEM). The range for the right chamber was 4.15 to 5.68 mW/sq cm, with a mean of 4.85 +/- 0.27. The mean SAR at 5 mW/sq cm, determined calorimetrically for 3 carcasses of representative body mass immediately after exposure for 15 min, was 1.23 +/- 0.25 W/kg.

The subjects were 30 rats of initial body mass ranging from 350 to 375 g. Adaptation to handling and to the chambers was done by placing each rat in a chamber from 0900 to 1700 (without food or water), 5 days/week for 4 weeks prior to exposure. At 1700, the rat was removed and usually placed in a Wahman rodent-activity cage with food and water ad libitum. Wheel revolutions, food and water consumption, and body mass were measured at 0800 the next day, prior to returning the rat to one of the chambers. The rats were then divided into 2 groups of 15 on a random basis and statistically assessed for equality of mean daily wheel revolutions during the last 2 weeks of adaptation. With nonsignificant differences in means for the 2 groups, 1 group was selected by chance to serve as controls and the other for RFR exposures.

RFR- and sham-exposures were done from 0900 to 1700 daily, 5 days/week for 16 weeks and wheel revolutions, food and water consumption, and body mass were measured as before. In addition, the rats were each placed on one of two BRS/LVE stabilimetric platforms from 1700 to 1800 once every 2 weeks, starting with the week preceding exposure. Each platform, which measured lateral movements of the rat, was placed in a sound-attenuating enclosure ventilated with an exhaust fan and illuminated with a 10-W bulb. The tests of each rat were done on the same platform.

Under ether anesthesia, blood samples were taken from a tail vein at 4-week intervals. One sham-exposed and 2 RFR-exposed rats died under anesthesia at the first blood collection. For the blood samples from the remaining rats, hematocrit was assayed and counts of red blood cells (RBC), white blood cells (WBC), and differential WBC were made. Plasma- and whole-blood cholinesterase activities and total sulfhydryls in plasma were determined. Also, 24-hr urine samples collected at 4-week intervals on Saturdays were analyzed for 17-ketosteroid levels.

At the end of the 16th week of exposure, 6 RFR-exposed and 7 sham-exposed rats were euthanized and examined for gross pathology. The adrenal glands, liver, and heart of each rat were weighed.

No statistically significant differences between RFR- and sham-exposed rats in body mass, food intake, or water intake were obtained. Their Figure 5 shows that the mean number of wheel revolutions for each group decreased approximately linearly with time, at a rate somewhat faster for the sham-exposed than the RFR-exposed group. At corresponding times, the mean values for the RFR-exposed rats were consistently higher than for the sham-exposed rats, but the differences were not statistically significant ($p>0.05$).

By contrast, their Fig. 4 shows that the mean numbers of activity responses on the stabilimetric platform, expressed as percentages of mean baseline responses, did not vary significantly with time, but the values for the RFR-exposed rats were consistently lower than for the sham-exposed rats at corresponding biweekly times. The differences were statistically significant ($p<0.05$) for test sessions 1, 3, and 4, but not for the other 5 biweekly sessions.

The hematological results (their Table 1) showed statistically significant differences between RFR and sham groups only for the samples taken at week 6. The mean RBC count for the RFR group at week 6 was significantly lower than at weeks 2 and 10, whereas the values for the sham group at these 3 times did not differ significantly from one another or from the week-2 and week-10 values for the RFR group. The RBC values for both groups at week 14 were significantly higher than their corresponding values at week 10, but did not differ significantly from each other. By contrast, the mean WBC count at week 6 for the sham group decreased while the count for the RFR group increased; both changes were significant, and so was the difference between groups at week 6.

Hemoglobin values for both groups at week 6 were higher than at week 2, but only the increase for the RFR group was significant, thus yielding a significant difference between groups. Hematocrit decreased in the sham group from week 2 to week 6, but remained unchanged in the RFR group, also yielding a significant intergroup difference. There were no significant differences between groups, or within each group with time, for the mean percentages of neutrophils or lymphocytes.

As seen in their Table 2, RBC cholinesterase activity increased with time in both groups but did not differ significantly between groups at corresponding times. Plasma cholinesterase varied nonmonotonically with time in both groups. The values for the RFR group were consistently lower than for the sham group at corresponding times, but the differences were significant initially (before exposure) and only at week 6. Sulfhydryl levels also varied nonmonotonically, with the values consistently higher for the RFR group, but only the difference at week 6 was significant.

From their Table 3, the mean level of 17-ketosteroids in urine, which also showed nonmonotonic variations, was significantly lower for the RFR group than the sham group only initially and at week 1.

The measurements of body mass and masses of liver, heart, and adrenal glands (Table 4), with the latter three expressed both in grams and as ratios to body masses, showed no significant differences between groups. No pathology data were presented.

CRITIQUE: Describing the controls in this investigation as "sham-exposed" is not accurate in a strict sense, because the rats were not sham-exposed in the actual chamber used for RFR exposures. The investigators endeavored to make the RFR and control chambers similar, but the chambers did differ in some respects, notably in the shape and composition of the inner surfaces (which could affect the optical and acoustic characteristics of each chamber) and conceivably the odor-bearing emissions from the walls (which could influence behavior). However, the degree of influence (if any) of such differences on the results cannot be assessed.

The RFR used was characterized as CW. However, the manner in which the output power of the type 2M53 magnetron was adjusted to yield the desired 5 mW/sq-cm power density was not described. Often an unfiltered half-wave or full-wave rectifier is used to power magnetrons. Such power supplies yield, respectively, 1 or 2 sinusoidal voltage half-cycles of 1 polarity for each 60-Hz alternation. Each such half-cycle activates the magnetron for only part of the half-cycle, and the average output power is often adjusted by varying the fraction of the half-cycle or the duration of magnetron operation per half-cycle. Consequently, such RFR is amplitude-modulated (at 60- or 120-Hz) and may have small duty cycles (ratios of average-to-peak output powers) at relatively low average powers, and could produce the auditory-RFR effect under appropriate conditions. It is not clear whether this consideration applies to this investigation.

The body masses of both groups increased substantially during the experiment, i.e., from 350-375 g to means of 570 and 535 g for the euthanized control- and RFR-exposed rats, respectively. However, based on prolate-spheroidal models in Durney et al., (1978), the whole-body SARs for 2.45 GHz at 5 mW/sq cm for the "medium" and "large" rat (weighing 320 and 520 g, respectively) are about 1.2 and 0.9 W/kg. Therefore, the SARs of the rats used in this study must have decreased significantly with time.

The primary RFR-induced effect reported, i.e., the lower mean activities of the RFR-exposed rats on the stabilimetric platforms, is open to question. Specifically, the curves of mean activity vs test session for the RFR- and control groups (Fig. 4) were nonmonotonic but surprisingly similar in shape, an indication that perhaps time-dependent factors other than exposure were affecting both groups in at least qualitatively similar fashion. Strong evidence for the existence of such factors is the significant decline with time of the mean wheel activity of both groups (Fig. 5), probably reflecting the tendency of male rats to decrease their activity rates as their body masses increase with time.

In addition, the stabilimetric data (expressed only in percentages of baseline performances) showed that during the first 4 sessions, the control group performed at 125-150% of their baseline values for reasons that were not discussed , and that the differences between the groups were statistically significant only for sessions 1, 3, and 4 (out of a total of 8 sessions). Lacking are the baseline results for each group.

Another point to be noted about the stabilimetric results is that a subsequent study by these investigators (D'Andrea et al., 1980), conducted with rats of the same strain and with essentially the same methodology but at 915 MHz, showed that the mean percentages of stabilimetric activity for test-session 1 were the same (about 175% of baseline values) for both groups, but that the levels for the RFR group rose, rather than declined, to about 400% and 475% for sessions 2 and 3, respectively, while the corresponding values for the control group showed no statistically significant changes. This apparent reversal of effect is difficult to explain, particularly since 915- and 2450 MHz are both above the whole-body resonance for the mature rat.

The findings that the significant differences between groups in hematological measures were mostly for the week-6 samples and that there were significant week-to-week (time-dependent) changes within each group may also be indications that non-RFR factors were involved.

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J. Microwave Power, Vol. 15, No. 2, pp. 123-134 (1980)

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AUTHOR ABSTRACT: Long-Evans male adult rats were exposed for 16 weeks to 915-MHz CW microwaves at an average power density of 5 mW/sq cm. The resulting dose rate was 2.46 (+/- 0.29 SEM) mW/g. The animals were exposed eight hours a day, five days a week, for a total of 640 h in a monopole-above-ground radiation chamber while housed in Plexiglas cages.

Daily measures of body mass and of food and water intakes indicated no statistically significant effects of microwave irradiation. Measures by activity wheels and stabilimetric platforms of spontaneous locomotion indicate that mean activity levels increased about 25% after microwave exposure, but the findings are [of] doubtful statistical significance (Ps < 0.10 but > .05). Studies of blood sampled after 2, 6, 10, and 14 weeks of exposure revealed alterations of free sulfhydryls. Measures of levels of urinary 17-ketosteroids at weeks 1, 5, 9, and 12 of exposure, and measures of brain hypothalamic tissue, and of mass of adrenals, heart, and liver at the end of the 16-week period, revealed no significant differences between irradiated and control animals. Cortical EEGs sampled after conclusion of microwave exposures also revealed no significant differences.

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Study Type: Behavior, Physiology and Biochemistry, Nervous System, Biorhythms; IN VIVO; RAT
Effect Type: Alterations of body and organ masses, food and water intake, locomotor activity, blood chemistry, hypothalamic morphology, and EEGs by chronic RFR exposure
Frequency: 915 MHz
Modulation: CW
Power Density: 5 mW/sq cm
SAR: 1.6-2.5 W/kg

EXPOSURE CONDITIONS: Exposures to RFR were done in the 2 halves of a chamber lined with pyramidal RFR-absorption materials and partitioned vertically with aluminum-covered sheets that served as ground planes. A quarter-wavelength monopole was mounted horizontally on each ground plane, and 10 rectangular rat-holding cages were centered around each monopole in a circular array of 110-cm radius with their long horizontal axes parallel to the monopole. The floor and ceiling of each rat cage were grids of equally spaced Plexiglas rods, and the other walls were of Plexiglas sheet. A type JC-300 magnetron was the RFR source.

Sham-exposures were performed in a chamber of the same design and interior dimensions, but was lined with Styrofoam sheet and sprayed with gray acoustic material to match the lighting and sound conditions of the RFR chamber. Both chambers were equipped with houselight, ventilating fans, and loudspeakers for providing white noise of about 70 dB.

RFR- and sham-exposures were from 0900 to 1700 (8 hr/day), 5 days/week for 16 weeks. No food or water was provided in either chamber.

OTHER INFORMATION: The power density at each cage location in the 2 halves of the RFR-exposure chamber was measured in the absence of rats. The values for the 10 locations in the left chamber ranged from 4.9 to 5.4 mW/sq cm, with a mean of 5.1 +/- 0.23 (SD). The range for the right chamber was 4.7 to 6.5 mW/sq cm, with a mean of 5.4 +/- 0.64. The mean SAR at 5 mW/sq cm, determined calorimetrically for 3 carcasses of representative body mass immediately after exposure for 15 min, was 2.46 +/- 0.29 (SEM) W/kg.

The subjects were 30 rats of initial body mass ranging from 350 to 375 g. Adaptation to handling and to the chambers was done by placing each rat in a chamber from 0900 to 1700 (without food or water), 5 days/week for 8 weeks prior to exposure. At 1700, the rat was removed and usually placed in a Wahman rodent-activity cage with food and water ad libitum. Wheel revolutions, food and water consumption, and body mass were measured at 0800 the next day, prior to returning the rat to one of the chambers. The rats were then divided into 2 groups of 15 on a random basis and statistically assessed for equality of mean daily wheel revolutions during the last 2 weeks of adaptation. With nonsignificant differences in means for the 2 groups, 1 group was selected by chance to serve as controls and the other for RFR exposures.

RFR- and sham-exposures were done from 0900 to 1700 daily, 5 days/week for 16 weeks and wheel revolutions, food and water consumption, and body mass were measured as before. In addition, lateral movements of the rats were measured by placing each rat on one of six BRS/LVE stabilimetric platforms from 1700 to 1800 once every 4 weeks, starting with the week preceding exposure. The tests of each rat were done on the same platform.

Under ether anesthesia, blood samples were taken from a tail vein at 4-week intervals. Two rats of each group died under anesthesia during blood collection at the eighth week of adaptation prior to initiation of exposures. For the blood samples from the remaining rats, hematocrit was assayed and counts of red blood cells (RBC), white blood cells (WBC), and differential WBC were made. Plasma- and whole-blood cholinesterase activities and total sulfhydryls in plasma were assayed. Also, 24-hr urine samples collected at 2-week intervals on Saturdays were analyzed for 17-ketosteroid levels.

Fourteen days after completion of the 16th week of exposure, 6 randomly selected rats of each group were euthanized, sections of hypothalamic tissue were prepared for light- and electron-microscopy, and the adrenal

glands, liver, and heart of each rat were weighed. The remaining 7 rats of each group were surgically fitted with stainless-steel recording electrodes placed bilaterally in calvarium over the visual cortices. For reference and grounding, electrodes were also placed in calvarium over the cerebellum and frontal sinus. One week after surgery, EEG recordings were made for 10 min. The rats were then euthanized and treated in the same manner as the other rats. (Although not explicitly stated, the EEGs presumably were taken 7 weeks after cessation of exposures.)

The mean values of the serum-chemistry measures, displayed in their Table 1, showed that only the level of sulfhydryl in plasma at week 2 was significantly affected; the level for the control group decreased from a mean baseline value of 9.1 to 8.8 mM at week 2, while the level for the RFR group rose from 9.4 to 11.1 mM.

Their Figure 4 showed that the mean number of wheel revolutions for the control group decreased approximately linearly with time, whereas the mean wheel activity of the RFR group rose monotonically through the fourth week of exposure and subsequently diminished approximately linearly in a manner similar to that of the control group.

The results of the tests on the stabilimetric platforms (their Fig. 5) showed that the mean activities of both groups at test-session 1 (fourth week of exposure) were 175% above their baseline values. However, the values for the RFR group rose to about 400% and 475% above baseline at sessions 2 (eighth week) and 3 (twelfth week), respectively, whereas the corresponding values for the control group were about 225% and 175%. However, the investigators stated that because of the large intragroup variations, the intergroup differences for both activity measures were of doubtful statistical significance.

The mean body masses of both groups rose with time (their Fig. 6), with the values for the RFR group slightly but nonsignificantly lower than the corresponding values for the control group. Food intake of both groups varied with time in nonmonotonic but similar fashion (their Fig. 7), with no significant differences between groups. The results for mean water intake, displayed in Fig. 8, indicated that the mean values for the RFR group were lower than for the control group for the last 2 months of the experiment, but the differences were characterized as unreliable.

The mean values of RBC, WBC, and hemoglobin, and the percentages of hematocrit, polymorphic neutrophils, and lymphocytes, displayed in their Table 2 for each week assayed, showed no significant differences between corresponding values for the RFR and control groups. Similarly, the results for urinary 17-ketosteroids (their Table 3) showed no significant intergroup differences.

The measurements of body mass and masses of liver, heart, and adrenal glands of the rats euthanized 2 and 7 weeks after cessation of RFR exposure, displayed in their Table 4, showed no significant differences between groups.

Spectral analyses of the 10-min EEG recordings of the rats were performed by computer. The frequency range (in Hz) was divided into 4 bins (subranges): 0.6-10.5, 10.6-20.5, 20.6-30.5, and 30.6-40.5; each recording was scanned 4 times; and the spectral content in each bin was averaged for the 4 scans. The results, presented in their Fig. 10, showed no significant differences between groups. The maximum spectral content for each group was in the 10.6-20.5 bin.

Examination, by light microscopy, of hypothalamic slices stained with methylene blue showed some degree of abnormal vacuolization and chromatolysis in specimens from both groups, but no significant differences between groups. Under electron microscopy, neuronal cells having such morphological changes showed a lack of endoplasmic reticulum, presence of deep enfolding of nuclei, and a larger number of mitochondria in some cells. Counting of such cells yielded no significant differences between groups. (Such abnormal cells were found in 7 of 12 control rats and in 6 of 10 RFR-exposed rats.)

CRITIQUE: This study was similar in many respects to an earlier one performed with rats at 2.45 GHz (D'Andrea et al., 1979), and the first three comments presented below, which were made regarding the earlier study, seem applicable here.

Describing the controls in this investigation as "sham-exposed" is not accurate in a strict sense, because the rats were not sham-exposed in the actual chamber used for RFR exposures. The investigators endeavored to make the RFR and control chambers similar, but the chambers did differ in some respects, notably in the shape and composition of the inner surfaces (which could affect the optical and acoustic characteristics of each chamber) and conceivably the odor-bearing emissions from the walls (which could influence behavior). However, the degree of influence (if any) of such differences on the results cannot be assessed.

The RFR used was characterized as CW. However, the manner in which the output power of the type JC-300 magnetron was adjusted to yield the desired 5 mW/sq-cm power density was not described. Often an unfiltered half-wave or full-wave rectifier is used to power magnetrons. Such power supplies yield, respectively, 1 or 2 sinusoidal voltage half-cycles of 1 polarity for each 60-Hz alternation. Each such half-cycle activates the magnetron for only part of the half-cycle, and the average output power is often adjusted by varying the fraction of the half-cycle or the duration of magnetron operation per half-cycle. Consequently, such RFR is amplitude-modulated (at 60- or 120-Hz) and may have small duty cycles (ratios of average-to-peak output powers) at relatively low average powers, and could produce the auditory-RFR effect under appropriate conditions. It is not clear whether this consideration applies to this investigation.

The cited mean whole-body SAR (2.46 W/kg) was determined for rats of "representative body mass," presumably in the range 350-375 g. However, the body masses of both groups increased substantially during the experiment (e.g., to means of 505 and 513 g respectively for the control- and RFR-exposed rats euthanized 2 weeks after cessation of exposures, as shown in Table 4). Based on prolate-spheroidal models in Durney et al., (1978), the whole-body SARs for 915 MHz at 5 mW/sq cm for the "medium" and "large" rat (weighing 320 and 520 g, respectively) are 2.5 and 1.6 W/kg, respectively. Therefore, the SARs of the rats used in this study must have decreased significantly with time.

The reported elevation of serum sulfhydryl levels for the RFR group at week 2 (their Table 1) and not for any other assay time may be statistically significant but is difficult to ascribe to RFR exposure, in the light of the variations of mean levels with time for both groups. Examination of the mean values for week 2 shows that the level for the RFR group rose nonsignificantly from its baseline while the level for the control group decreased nonsignificantly from its baseline. A t-test for the week-2 levels yielded 1.97, for which $p>0.05$.

The decline of mean wheel activity by the control group during the course of the experiment (their Fig. 4) is a non-RFR effect probably ascribable to the tendency of male rats to decrease their activity rate as their body masses increase with time. Consequently, the increase in activity by the RFR group during the first month or so (Fig. 4) probably was RFR-induced. The subsequent decline by this group for the remainder of the experiment at a rate comparable to that of the control group may have been due to the decrease of whole-body SARs with time.

The stabilimetric-platform results (their Fig. 5), which showed significant rises of activities for the RFR group during the course of the experiment, were at variance with the corresponding results of the earlier investigation (at 2.45 GHz), which showed a decline for the RFR group. It is noteworthy that the newer data (presented, as before, only in percentages of baseline performances) also showed that both groups performed well above their baseline levels (175% at week 4 of exposures), an indication that non-RFR factors may have contributed to the results. Again, the baseline results for each group were not presented.

The description of the EEG results was rather obscure, particularly with regard to the number of rats in each group, why 4 scans per recording were taken, and why any changes ascribable to RFR exposure would be expected to persist for so long after cessation of exposure. Moreover, the surgical procedure used to implant the electrodes could have had significant effects on the EEGs of both groups. Therefore, even though the results showed no apparent RFR-induced effects, the finding is of little value.

As indicated by the investigators, the morphological changes in hypothalamic tissue from both groups were likely due to animal aging.

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Lin, J.C., A.W. Guy, and L.R. Caldwell
THERMOGRAPHIC AND BEHAVIORAL STUDIES OF RATS IN THE NEAR FIELD OF 918-MHZ RADIATIONS
IEEE Trans. Microwave Theory and Tech., Vol. 25, No. 10, pp. 833-836 (1977)

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AUTHOR ABSTRACT: Patterns of thermalized energy of rat carcasses exposed to 918-MHz CW radiation in the near zone have been determined using a computerized thermograph. Peak absorption of energy in the body was estimated to be 0.9 W/kg per mW/sq cm of incident energy.

Operant responses of irradiated rats to schedules of fixed-ratio (food) reinforcement under the same conditions as the dosimetric test were observed to occur at averaged power densities of 30-40 mW/sq cm. This range of densities corresponds to absorbed peaks of energy of 27-36 W/kg. No change in behavior was observed for incident power densities and peaks of absorbed energy to 20-30 mW/sq cm and to 18-27 W/kg, respectively, and all changes at higher values were reversible.

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Study Type: Behavior; Exposure Methods, Dosimetry, and Modeling; IN VIVO; RAT
Effect Type: RFR-induced alterations of behavior on fixed-ratio reinforcement schedules
Frequency: 918 MHz
Modulation: CW
Power Density: 10, 20, and 40 mW/sq cm
SAR: 9, 18, and 36 W/kg Pk; 2.1, 4.2, and 8.4 W/kg Av

EXPOSURE CONDITIONS: Rats in acrylic restraining holders were sham-exposed or exposed individually in 30-min sessions to near-field RFR from a cavity-backed applicator of square aperture located above the longitudinal midpoint of the rat's body. For exposure, the rat in the holder was inserted in an acrylic receiver, and the assembly was placed within a modified cooler chest lined with RFR-absorbing material and equipped with a fan to provide forced air flow.

OTHER INFORMATION: The subjects were 200-g female white rats (Sprague-Dawley) that were deprived of food to maintain them at 80% of their free-feeding body masses. The holder was a truncated cone of acrylic rods designed for adequate ventilation and to allow the rat to poke its head through the narrower end, thus permitting free movement of the rat's neck and head while restraining the remainder of the body. After a few sessions, the rats learned to run into the holder and exterd their heads through the opening.

Power densities at the rat's position in the empty exposure chamber were measured with a National Bureau of Standards electric energy-density

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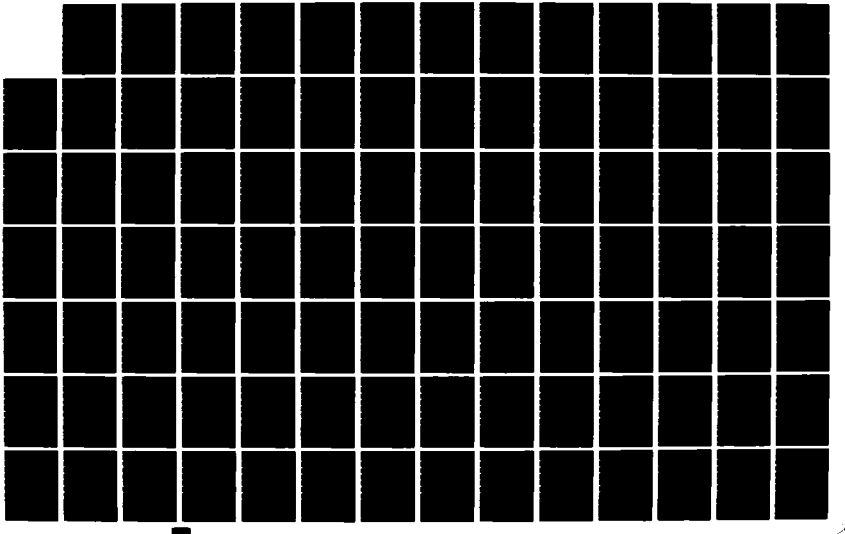

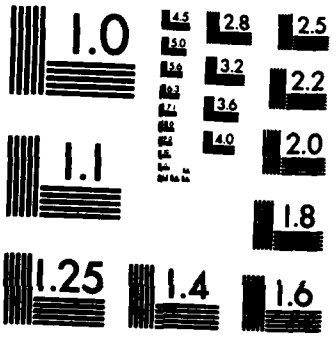

meter. Energy absorption rates in rat carcasses were determined by computerized thermography. Isothermal plots of energy absorption in the midsagittal plane yielded ranges (per mW/sq cm) of 0.1-0.9 W/kg in the tail and 0.1-0.8 W/kg in the body, values that were sensitive to the position of the tail. Consequently, exposures were performed with the tail immobilized. Mean whole-body SARs were not measured, but theoretical calculations for mass-equivalent muscle spheres yielded peak and average SARs of 0.85 and 0.21 W/kg per mW/sq cm at 918 MHz.

The receiver for the holder and the exposure chamber were designed so that a small upward movement of the rat's head interrupted a horizontal light beam. The task required of the rat was to execute 30 such movements rapidly and regularly in order to receive a food pellet (fixed-ratio, or FR-30 schedule). A slight downward movement of the head permitted the rat access to the pellets delivered. After the rats achieved stable performances, they typically responded 2000 times during each 30-min session.

Typical records of baseline cumulative responses vs time for 3 rats were presented in their Fig.4. Virtually uniform response rates of about 80/min were evident from the slopes. One of those rats was exposed for 30 min each at 10, 20, and 40 mW/sq cm on 5 consecutive days, another was exposed at the same levels on alternate days (Monday, Wednesday, and Friday), and the third rat was subjected to 30-min sessions of sham exposure. As seen in their Fig. 7, no significant alterations of performance rates were evident for the third rat or for the other 2 rats at 10 or 20 mW/sq cm.

At 40 mW/sq cm, the performance rate of the first rat was unchanged for the first 5 min, at which time its rate dropped to almost zero; the second rat performed at approximately baseline rate for the first 5 min or so, then at slowly decreasing rate for the next 15 min, and it essentially ceased performing for the remaining 10 min. The rats exposed at this power density exhibited physiological signs of heat stress, including panting, fatigue, and foaming of the mouth.

Another rat was subjected to exposures in power-density increments of 3 mW/sq cm up to 32 mW/sq cm, at which level the rat exhibited similar signs of heat stress. Its response rate (Fig. 8) remained at baseline through about the first 13-14 min, diminished slightly during the next 5-6 min, and then decreased significantly (but not to zero) for the remainder of the session. The calculated peak and average SARs at 32 mW/sq cm were 29 and 6.7 W/kg, respectively.

CRITIQUE: Although not explicitly stated, presumably the transverse component of the electric vector was parallel to the long axis of the rat's body. The calculated value of whole-body SAR based on muscle-equivalent spherical models (0.21 W/kg per mW/sq cm) for the rats used in this study (about 200 g) is consonant with estimates based on prolate-spheroidal models. For the latter models, the SARs at about 900 MHz for a "small" (110-g) rat and a "medium" (320-g) rat are about 0.1 and 0.5 W/kg per mW/sq cm, respectively (Durney et al., 1978).

Very significant is the large range of local SARs found by thermography in the rat carcass exposed at constant incident power density and animal orientation. As discussed by the investigators, there may be high local SAR values ("hot spots") within animals exposed to RFR at seemingly thermally insignificant power densities.

The behavioral results appear to be straightforward, and indicate the existence of a power-density threshold between 30 and 40 mW/sq cm at 918 MHz for the specific task studied. The corresponding whole-body SAR threshold is in the range 6.3 to 8.4 W/kg. The investigators stated that "recovery of the base-line performance for both exposed animals on the first day following exposure at 40 mW/sq cm is readily observed in Fig. 7." However, Fig. 7 did not include such data.

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Monahan, J.C. and W.W. Henton
MICROWAVE ABSORPTION AND TASTE AVERSION AS A FUNCTION OF 915 MHZ RADIATION
In D.G. Hazzard (ed.), SYMPOSIUM ON BIOLOGICAL EFFECTS AND MEASUREMENT OF RADIO FREQUENCY/MICROWAVES, U.S. Department of Health, Education, and Welfare, HEW Publication (FDA) 77-8026, pp. 34-40 (1977b)

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AUTHOR ABSTRACT: Thirty-two rats were divided into four subgroups and exposed to 915 CW radiation at different intensities immediately following 15 minutes access to a 10 percent sucrose solution. Absorption of microwave energy was relatively unchanged throughout a 15-minute exposure to forward power of 5.0 W. Microwave absorption progressively decreased during exposure at forward power levels of 9.1 and 19.0 W. Each subject was again given access to sucrose 24 hours later, with no indication of a conditioned taste aversion.

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Study Type: Behavior; IN VIVO; RAT
Effect Type: Taste aversion induced by RFR exposure; unconditioned alteration of percentage of RFR absorption as a noxious agent
Frequency: 915 MHz
Modulation: CW
Power Density: 16.3, 29.6, and 61.8 mW/sq cm (estimated)
SAR: 7.1, 9.6, and 17.3 W/kg

EXPOSURE CONDITIONS: Three groups of 8 rats, each restrained in a ventilated Plexiglas holder, were individually exposed to RFR within a waveguide system instrumented for continuously recording forward, reflected, and transmitted powers. One group was exposed at a forward power of 5.0 W, another at 9.1 W, and the third at 19.0 W. Air at 38 l/min was forced through the waveguide. The temperature of the exhaust air ranged from 23 to 26 deg C, and the relative humidity was 40-50%. A fourth group was sham-exposed. Exposures were for 15 min.

OTHER INFORMATION: The subjects (Sprague-Dawley male rats) were housed with food freely available. Water was provided for 15 min/day except on exposure days, when a 10% (by weight) sucrose solution was substituted. At the end of sucrose presentation, each rat was exposed for 15 min and immediately returned to its home cage. After 24 hr, each rat was given a 1-bottle preference test for 15 min, and the amount of sucrose consumed was compared with the quantity of liquid consumed on the previous day.

The average absorbed dose rate (SAR) was defined as the total energy absorbed by the rat during a specified period of exposure divided by that period and by the body mass of the rat. Presumably the continuous values of net power, defined as the forward power minus the reflected and transmitted powers, were integrated over the period to obtain the

total energy absorbed. The SARs were determined for the entire 15-min exposure period and for the 3 successive 5-min intervals thereof, and the latter were expressed as percentages of the incident (forward) power absorbed.

The results at 5.0 W were 7.1 W/kg for the entire period and for each 5-min interval, i.e., the SAR of the animal did not vary with time at this RFR level. From their Fig. 1, 7.1 W/kg represented about 50% of the forward power absorbed. (It should be noted that Fig. 1 presented only percentage absorptions, not the absolute values as stated in the text.)

At 9.1 W, the mean SAR was 9.6 W/kg for the entire period, and 10.5, 9.5, and 8.7 W/kg for the successive 5-min intervals, respectively. The latter values were about 49%, 44%, and 41% of the forward power. At 19.0 W, the mean SAR was 17.3 W/kg for the entire period, and 18.5, 16.9, and 16.5 W/kg for the 5-min intervals, the latter 3 values corresponding to 57%, 53%, and 52% of the forward power. The maximum relative SAR changes at 9.1 W and 19.0 W evidently occurred during the second 5-min interval (their Fig. 2).

Mean sucrose consumption 24 hr after exposure (their Fig. 3) at 0 (sham), 5.0 or 19.0 W was slightly higher than for the previous day in each case, and at 9.1 W was slightly lower than for the previous day, but none of the differences was significant. Thus, these results yielded no evidence of a conditioned aversion to sucrose engendered by RFR exposure.

CRITIQUE: The power densities used can be estimated by dividing the values of forward power by the cross-sectional area of the waveguide, which was 24.8 cm x 12.4 cm or about 308 sq cm. Thus, the power densities corresponding to 5.0, 9.1, and 19.0 W were about 16.3, 29.6, and 61.8 mW/sq cm.

As indicated by the investigators, their findings of time-dependent reduction of SAR in the rat are consonant with similar results in the mouse exposed to 2.45-GHz RFR (Monahan and Ho, 1976) and the dependence of the effect on the ambient temperature (Monahan and Ho, 1977). They suggested that the animals endeavored to alter their orientation and body configuration so as to decrease their SARs during exposure, a reasonable hypothesis, but no mention was made whether such endeavors were actually observed. The absence of the effect at 7.1 W/kg may indicate the existence of an SAR threshold between 7.1 and 9.6 W/kg, or a power-density threshold between 16 and 30 mW/sq cm.

No error bars and/or statistical analysis of the sucrose results were presented. Nevertheless, because the differences in mean consumption were small percentages of the consumption, the conclusion appears valid. Also, as indicated by the investigators, the finding of no conditioned aversion to sucrose in the rat was consistent with the absence of conditioned aversion to saccharin paired with exposure to pulsed 2.88-GHz RFR reported by Hjeresen et al. (1976).

In the discussion following the presentation of this paper, Dr. G.R. Session suggested that longer exposures (1-2 hr) at the higher RFR levels might conceivably have produced aversion. In this context, Dr. R.D. Phillips mentioned that an SAR of 17.3 W/kg corresponds to about 4 times the basal metabolic rate of a 450-g rat and therefore constitutes a "hefty" heat load on a rat.

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In C.C. Johnson and M. Shore (eds.), BIOLOGICAL EFFECTS OF ELECTROMAGNETIC WAVES, U.S. Department of Health, Education, and Welfare, HEW Publication (FDA) 77-8010, pp. 274-283 (1976)

Monahan, J.C. and H.S. Ho
THE EFFECT OF AMBIENT TEMPERATURE ON THE REDUCTION OF MICROWAVE ENERGY ABSORPTION BY MICE
Radio Sci., Vol. 12, No. 6S, pp. 257-262 (1977)

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Monahan, J.C. and H.S. Ho
THE EFFECT OF AMBIENT TEMPERATURE ON THE REDUCTION OF MICROWAVE ENERGY ABSORPTION BY MICE
Radio Sci., Vol. 12, No. 6S, pp. 257-262 (1977)

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AUTHOR ABSTRACT: Previous research has established that exposure to microwave radiation above a critical level is associated with a reduction across time in the rate at which the energy is absorbed by a mouse. Microwaves caused the animal effectively to decrease the radiation by decreasing the percent absorption of the incident energy. The current investigation sought to determine the effect of ambient temperature on this behavior. Male CF1 mice (30 to 34 g) were irradiated by 2450-MHz CW microwaves for 20 minutes in an environmentally controlled waveguide at temperatures of 20, 24, 30, or 35 deg C and at a relative humidity of 50 +/- 1.5 percent. Forward power in the waveguide ranged from 0.004 to 4 W, which resulted in averaged dose rates of 0.06 to 64 mW/g.

When the averaged dose rates were above a critical level, the percent absorption decreased after the initial five minutes of irradiation and remained lower for the duration of the exposure. The threshold level of power at which decreases in percent absorption were observed was found to decrease with an increase in the environmental temperature. The data suggest that the subjects were capable of detecting or otherwise reacting to microwave energy at dose rates as low as 0.6 mW/g when the environmental temperature was 35 deg C.

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Study Type: Behavior; Exposure Methods, Dosimetry, and Modeling; IN VIVO; MOUSE
Effect Type: Sensing of RFR absorption as heat by the mouse
Frequency: 2.45 GHz
Modulation: CW
Power Density: 0.07-64 mW/sq cm (estimated)
SAR: 0.06-64 W/kg

EXPOSURE CONDITIONS: Each mouse to be exposed to RFR was placed in a Plexiglas container ventilated for air flow and large enough to permit free movement. The container was placed in a waveguide system instrumented for continuous recording of forward, reflected, and transmitted powers. The waveguide system was housed within a chamber maintained at 50% relative humidity and ambient-temperature-controlled to +/- 0.5 deg C. Exposures were for 20 min at an ambient temperature of 20, 24, 30, or 35 deg C, 1 of 4 values of forward power for each temperature, and with 38 1/min of air flowing (unless otherwise noted).

OTHER INFORMATION: A total of 102 mice was divided into 17 groups of 6 each on the basis of body mass (30-34 g). After determining the mass of

each mouse, its rectal temperature was measured and it was exposed at one of the specified sets of conditions. Its rectal temperature was measured again as soon after exposure as possible.

The averaged dose rate (SAR) was defined as the total amount of energy absorbed by the mouse during a specified period of exposure divided by that period and by the mass of the mouse. The continuous values of net power, defined as the forward power minus the reflected and transmitted powers, were integrated over the period to obtain the total energy absorbed. The SARs were determined for the entire 20-min exposure period and for the 4 successive 5-min intervals thereof, and the latter were expressed as percentages of incident (forward) power absorbed. The results for all conditions were presented in Table 1.

Four groups were exposed at an ambient temperature of 20 deg C, one at a forward power of 1.62 W, another at 2.31 W, the third at 3.28 W, and the fourth at 3.84 W. The corresponding mean SARs for the entire 20-min exposure period were 30.7, 43.6, 56.3, and 63.8 W/kg.

At 30.7 W/kg (1.62 W), the percentages of incident power absorbed at successive 5-min intervals were not significantly different from one another, and no downward (or upward) trend with time was observed. At 43.6 W/kg, the differences in percentages of absorption between successive 5-min intervals were not statistically significant, but a highly significant ($p<0.001$) downward trend with time was evident; absorption had decreased from about 61% during the first 5 min to about 52% during the last 5 min. At 56.3 W/kg, the percentages for successive intervals were 57, 44, 50, and 44; the sequential differences were significant but no trend was discernible. At 63.8 W/kg, the successive percentages were 53, 39, 38, and 37, with only the first drop significant.

The next four groups were exposed at 24 deg C to forward powers of 1.12, 1.70, 2.39, and 3.11 W, corresponding to mean SARs of 20.6, 30.1, 43.5, and 51.3 W/kg. Significant downward trends were evident for all but the lowest level.

Four of the remaining groups were exposed at 30 deg C to forward powers of 0.415, 0.736, 1.62, and 2.45 W, yielding mean SARs of 7.3, 12.7, 25.8, and 40.2 W/kg. Significant downward trends were observed at all 4 levels. However, at the upper 3 levels, the largest drop occurred between the first and second 5-min intervals.

The last 5 groups were exposed at 35 deg C to forward powers ranging from 0.004 to 0.4 W, yielding mean SARs of 0.06, 0.6, 1.2, 3.7, and 6.7 W/kg. Downward trends were obtained at all levels, with significant differences at 0.6 W/kg and higher.

In summary, significant time-dependent reductions in percentage absorption were observed: at 20 and 24 deg C for mean SARs (averaged over the entire exposure period) of 43.6 W/kg and higher, at 30 deg C from 25.8 W/kg upward, and at 35 deg C for 0.6 W/kg or more.

Several of the mice of the last 5 groups were similarly exposed at 35 deg C, but with no flow of air, to ascertain whether the mice had been orienting themselves so as to minimize the flow of the hot air rather than their SARs. The results for 0.6 W/kg (Fig. 4) were about 56% and 57% absorption during the first and second 5-min intervals (a change that was nonsignificant), and about 49% and 38% respectively during the last 2 intervals. By contrast, the corresponding results at this ambient temperature and SAR but with air flowing were about 51%, 43%, 41%, and 39%. The investigators concluded that air flow does affect percentage absorption initially, but does not in the subsequent reduction of absorption.

Rectal temperatures before RFR exposure were highly variable, ascribed to handling and to the novelty of the experimental situation. The mean pre-exposure value for all of the mice was 37.0 +/- 1.1 deg C with a range from 34.6 to 39.4 deg C. As a consequence, no significant inferences were drawn.

CRITIQUE: The power densities used can be estimated by dividing the values of forward power by the cross-sectional area of the waveguide, which was 10.9 cm x 5.5 cm or about 60 sq cm. For example, for 1.12 W (which did not yield time-dependent SAR variations at 24 deg C), the calculated power density is 18.7 mW/sq cm. It is noteworthy that the corresponding experimental mean SAR, 20.6 W/kg, corroborates the SAR for the prolate-spheroidal model of a "large" mouse (25 g), i.e., from Durney et al. (1978), the SAR at 2.45 GHz is about 1.1 W/kg per mW/sq cm. Multiplying this value by 18.7 mW/sq cm yields 20.6 W/kg. (The exactitude of the agreement is probably fortuitous.)

The results of this study, showing that mice orient themselves to minimize their SARs, were consonant with those of another study by Monahan and Henton (1977) of rats exposed to 915-MHz RFR, as discussed below, and also demonstrated the strong dependence of the effect on ambient temperature.

In the present study with 2.45 GHz, the lowest RFR level at which the mouse altered its SAR at 24 deg C was about 1.70 W, corresponding to about 28 mW/sq cm and 30 W/kg. These and the values above for no effect indicate the existence of a power-density threshold between about 19 and 28 mW/sq cm and an SAR threshold between about 20 and 30 W/kg (at an ambient temperature of 24 deg C).

In the rat study, conducted at an ambient temperature in the range 23-26 deg C, the effect was absent at a forward power of 5.0 W corresponding to an estimated power density of 16.3 mW/sq cm and a mean SAR of 7.1 W/kg and the effect was observed at 9.1 W corresponding to 30 mW/sq cm and 9.6 W/kg (Monahan and Henton, 1977). Those results indicate the existence of power-density and SAR thresholds in the ranges 16-30 mW/sq cm and 7.1-9.6 W/kg at such ambient temperatures. It is interesting to note that the threshold SAR for the rat appears to be considerably lower than for the mouse. (The ranges straddling the power-density thresholds for the two species were comparable, but this result was probably fortuitous.)

The percentage of RFR absorption at 24 deg C (in the absence of the reorientation effect) for the mouse at 2.45 GHz (54%-59%) and the rat at 915 MHz (50%) were comparable. The body masses of the rats used were not stated, but if they were in the range denoted as "small" (about 110 g) in Durney et al. (1978), then this result would be expected, since the SAR of the prolate-spheroidal-rat model at 915 MHz is about 1.1 W/kg per mW/sq cm or about the same as for the model mouse at 2.45 GHz.

A point made by the investigators was that the behavior of the mice was not "avoidance" in the classic psychological sense, since the subjects could not escape the RFR (or high ambient temperatures), but could only endeavor to minimize their discomfort. Not explicitly discussed was that the amount of adjustment possible for the mouse to minimize its heat load was limited, which may account for the relatively large drops of absorption at the higher RFR levels and ambient temperatures during the initial time intervals.

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Lebovitz, R.M.
PROLONGED MICROWAVE IRRADIATION OF RATS: EFFECTS ON CONCURRENT OPERANT BEHAVIOR
Bioelectromagnetics, Vol. 2, No. 2, pp. 169-185 (1981)

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AUTHOR ABSTRACT: Two measures of performance were used to study the effects of pulse-modulated microwave radiation (PM MWR) on schedule-controlled operant behavior of rats: 1) cued (SD), fixed-ratio (FR) bar pressing for food reinforcement; and 2) noncued (Sd) bar pressing in the absence of food reinforcement. The animals were irradiated and the behavioral data were obtained concurrently, during daily three-hour sessions, five days per week for six to nine weeks. Each experiment began with a two to three-week baseline interval of sham irradiation; a two to three-week interval of sham irradiation followed the irradiation phase. The irradiated animals were exposed to 1.3-GHz PM MWR (pulse width of 1 microsecond at 600 pulses per second) at whole-body, average specific absorbed-dose rates of from 1.5-6.7 mW/g. Control and irradiated animals were tested in identical, cylindrical waveguide exposure/behavioral assemblies; different groups of irradiated and sham-irradiated were used for each dose rate.

At 1.5 mW/g, the levels of SD operant responding by control and irradiated animals were comparable, and showed similar progressive diminutions over the course of each daily session. Sd operant responding was more variable, but again comparable, with both groups showing similar, progressive declines in rate of responding during each session. At 3.6 mW/g, no specific effects on SD operant response rates were observed. However, there was an initial and transient increase in the rate of extinction of Sd responding. At 6.7 mW/g, SD response rates were slightly reduced, whereas there was a major reduction in noncued (Sd) operant responding followed by a sharp rebound during the first post-MWR week. This marked reduction in Sd operant responding at MWR onset was in contrast to the relative stability and persistence of FR responding for food reinforcement.

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Study Type: Behavior; IN VIVO; RAT
Effect Type: Alterations of cued, fixed-ratio bar pressing for food reinforcement and noncued bar pressing in the absence of food reinforcement during exposure to pulsed RFR
Frequency: 1.3 GHz
Modulation: 1-microsecond pulses at 600 pps (0.0006 duty cycle)
Power Density: 3.9, 9.2, or 17.2 mW/sq cm Av (estimated)
SAR: 1.5, 3.6, or 6.7 W/kg

EXPOSURE CONDITIONS: Groups of 15 rats each were concurrently sham-exposed and exposed to circularly polarized RFR (electric vector transverse to the propagation direction) in individual circular-

waveguide systems in a room maintained at 21 deg C but with unregulated relative humidity, which varied mostly between 40% and 60%. Exposures were for 3 hr/day, 5 days/week, for 6 to 9 weeks. Each system was equipped for automatically recording cued and noncued food-acquisition behavior during exposure. The rats, which were not restrained, were facing the RFR source during operant performance.

OTHER INFORMATION: The set of waveguide systems (which were similar to those designed by Guy and Chou, 1976) and the methods used to quantify the dosimetry were described in detail by Lebovitz and Seaman (1980). In brief, the SAR for a 300-g rat at 1.3 GHz was 2.2 W/kg per W of forward power, and dividing 1 W by the cross-sectional area of the waveguide (177 sq cm) yielded an estimated power density of 5.65 mW/sq cm. Thus, the approximate average power densities corresponding to SARs of 1.5, 3.6, and 6.7 W/kg were 3.9, 9.2, and 17.2 mW/sq cm. With a duty cycle of 0.0006, the corresponding peak power densities were 6.4, 15.4, and 28.7 W/sq cm.

Within each waveguide were a vertical displacement bar (behavioral operandum), a means for illuminating the operandum as a cue (visual discriminative stimulus), and means for delivering a food pellet when appropriate.

Groups of 46 female Long-Evans rats initially weighing 120-150 g were deprived of food for 2 days and were trained for 10 days to bar press for food pellets at increasing fixed-ratio (FR) schedules to FR-5 (requiring 5 successive lever presses to obtain a pellet). The 30 rats of each group that performed at the highest and most stable rates were selected and trained on a multiple fixed-ratio, extinction schedule of reinforcement using visual discriminative stimuli, in which only the responses when the operandum was illuminated (SD) were reinforced by pellet delivery. Such responses were tabulated. Reinforcement was done on a fixed-ratio schedule that was gradually increased to FR-25 during several weeks of training. Rat responses when the operandum was not illuminated (Sd), which yielded no pellets, were tabulated separately.

When not in their waveguides, the rats were kept in home cages with water available ad libitum. In addition, each rat was given 8 g of food daily irrespective of its operant performance. They were weighed three times per week. No rat failed to maintain a satisfactory growth curve.

Each set of 30 rats that achieved high and steady performance at FR-25 was given a baseline period of sham exposures and testing, after which half were randomly assigned to the RFR group and half to the sham group, with the 2 groups matched by baseline FR-25 performance. RFR- and sham exposures were for 3 hr at the same time each day for 5 days/week. The daily behavioral sessions were started 15 min after the beginning of exposure and were terminated 15 min before the end of exposure for a session duration of 150 min. The rats were also tested during a 2-week recovery period following the exposure regimen. Each behavioral session was divided into 6 sequential blocks of 25 min, each block consisting of a 15-min SD interval (when the operandum was illuminated) followed by a

10-min Sd interval (when the operandum illumination was extinguished). The response rates of each rat for SD and Sd during the baseline, exposure, and recovery periods were summed weekly by block number and the results for each group of rats were averaged. However, only the weekly group means for beginning-blocks 1, middle-blocks 3, and final-blocks 6 were presented.

The results for 8 weeks of exposure at 1.5 W/kg (Fig. 1) showed stable response rates for SD and no statistically significant differences between RFR and sham groups for corresponding blocks and weeks. Both groups showed a slight but significant increase in weekly SD response rate for blocks 1 and 3 and a slight decline for block 6. There were also modest declines in rates during sessions, i.e., over blocks 1-6. The response rates for Sd were much more variable than for SD. There was an increase in the block-1 weekly rate and declines in the block-3 and block-6 weekly rates, but the changes were not significant. Also, the decline in Sd rates during sessions, which was also evident for the baseline and recovery weeks, was sharper than for SD. However, there were no significant differences between the RFR and sham groups.

There were also no significant differences between groups in SD response rates for 9 weeks of exposure at 3.6 W/kg (Fig. 2). For both groups, the changes in weekly rates were marginally significant ($p=0.082$) and the intrasession decline in rate was significant ($p<0.0005$). Regarding Sd response rates, which again showed sharp intrasession declines for both groups during the baseline, exposure, and recovery periods, statistical analysis revealed an apparently transient difference between groups: the response rate by the RFR group was significantly lower than for the sham group only for blocks 2 and 3 of the first exposure week. The investigator stated that similar results were obtained with another group of rats exposed at 3.6 W/kg.

The results for 6 weeks of exposure at 6.7 W/kg (Fig. 3) showed no statistically significant differences between groups in overall SD response rates, but there were significant block-dependent differences between the groups. Specifically, the SD rates of both groups during blocks 1 and 3 did not change significantly week by week and the differences between groups for blocks 1, 3, and 6 during the baseline and recovery periods were not significant; however, the SD rate of the RFR group for block 6 was significantly lower ($p=0.012$) than the rate of the sham group for exposure-week 2 and was marginally significantly lower ($p=0.07$) for exposure-weeks 1, 3, and 4. These differences were ascribed to marked reductions in bar pressings near the end of behavioral sessions (blocks 4-6) during those weeks. An analysis by rat showed that the differences between SD rates during the last baseline week and the first exposure week were not significant, indicating that the decline was gradual rather than immediate.

Regarding the Sd rates for the 6.7-W/kg regimen, there were significant intrasession declines (differences in successive rates for blocks 1, 3, and 6) for both groups during the baseline period, with nonsignificant intergroup differences. However, the Sd rates for blocks 1, 3, and 6 of

the RFR group declined during the first week or so of exposure while the corresponding rates for the sham group rose (for unknown reasons), yielding significant intergroup differences for corresponding weeks and blocks. After about 3 weeks of exposure, the block-1 rate for the RFR group increased while it decreased for the sham group, so that the two rates became comparable again by the fifth week of exposure. The block-3 rate of the sham group also rose at the beginning of the exposure regimen and subsequently declined; concurrently, the rate for the RFR group dropped to very low values but showed sharp recovery at the end of the exposure regimen. The block-6 rates of both groups were already low during the baseline period; however, the Sd rate of the RFR group dropped to almost zero during exposures, with only slight increases evident during the recovery period.

Based on 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 investigator suggested that this SAR could be the approximate threshold for modifying the rate of operant responding in the absence of visual cue or food reinforcement. Regarding the results at 6.7 W/kg, although there were no significant differences in overall SD response rates between the RFR and sham groups, the decline rate of the intrasession SD response rate of the RFR group was higher than for the sham group. By contrast, the decline of the weekly and intrasession Sd rates of the RFR group were pronounced during exposure, with postexposure recovery to control levels.

The investigator noted that 6.7 W/kg is close to the resting metabolic rate for a 240-g rat, so that such RFR exposure represented a virtual doubling of the heat dissipation requirements of the animal. Thus, he concluded that thermal factors were likely involved in the behavioral effects observed. He also calculated that the energy deposited in the rat during each pulse was above the threshold for the RFR-auditory effect. However, he questioned whether the perceived loudness would be an adequate acoustic cue or how the presence of such a cue could account for the observation that the major decline in Sd responding was gradual rather than immediate with the onset of RFR exposure. He then indicated that other studies with CW RFR (manuscript then in preparation) yielded essentially the same findings as those reported for this investigation.

CRITIQUE: The engineering aspects of this investigation were excellent. Especially noteworthy were the exposure facilities, dosimetry, and automated execution of the behavioral paradigms and data acquisition. Also, the statistical treatment of the data was thorough.

As stated by the investigator, the operant data for each rat consisted of the number of bar presses during each of the 6 blocks or pairs of cued (SD) and uncued (Sd) response intervals sequentially numbered 1-6 daily. Not clear, however, was the rationale for summing the responses for correspondingly numbered blocks to obtain weekly block totals of SD and Sd responses as the "primary descriptive variables" for each rat, and why the time-dependent data for the successive blocks during daily sessions were not described or treated more explicitly, since even the

baseline results indicated intrasession diminutions of both SD and Sd response rates. Despite the extensive statistical treatment of the data, it is difficult to assess the contribution of this time-dependent non-RFR factor to the results of this investigation. However, this point does not gainsay the existence of the RFR-induced effects discussed.

The arguments presented by the investigator regarding the RFR-auditory effect as an ineffective behavioral cue seem weak. Incidentally, the peak power density corresponding to 6.7 W/kg was about 29 W/sq cm. More convincing are the since-published results with CW (and pulsed) RFR mentioned (Lebovitz, 1983).

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PULSE MODULATED AND CONTINUOUS WAVE MICROWAVE RADIATION YIELD EQUIVALENT CHANGES IN OPERANT BEHAVIOR OF RODENTS
Physiology and Behavior, Vol. 30, No. 6, pp. 891-898 (1983)

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MICROWAVE IRRADIATION AND INSTRUMENTAL BEHAVIOR IN RATS: UNITIZED IRRADIATION AND BEHAVIORAL EVALUATION FACILITY
Bioelectromagnetics, Vol. 1, No. 4, pp. 415-428 (1980)

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LEBOVITZ

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BEHAVIOR
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RAT
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PULSE MODULATED AND CONTINUOUS WAVE MICROWAVE RADIATION YIELD EQUIVALENT CHANGES IN OPERANT BEHAVIOR OF RODENTS
Physiology and Behavior, Vol. 30, No. 6, pp. 891-898 (1983)

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AUTHOR ABSTRACT: Long-Evans rats were trained to the point of stable performance on a multicomponent (fixed-ratio, timeout) operant task. Different groups were exposed to continuous wave (CW) and to pulse modulated (PM) microwave radiation (MWR) during daily three-hour behavioral sessions. The rates of responding under actual and sham exposure conditions were noted.

With comparable MWR dose rates, CW and PM MWR (5.8 and 6.7 mW/g, respectively) were equally effective in reducing response rates during both the fixed-ratio and the timeout components of the operant sessions. Dose rates of this order were associated with an elevation in body temperature of 0.5 to 1.0 deg C. At 3.6 mW/g, whereas the mean rates of fixed-ratio responding were unchanged, the rates of responding during timeout were reduced significantly. Again, CW and PM MWR yielded essentially equivalent results. This MWR dose rate was not accompanied by a measurable increment in whole body temperature.

It appears that (1) fixed-ratio operant responding of rats for food reward was more robust, that is, less subject to suppression by concurrent exposure to MWR than was bar-pressing during timeout, (2) PM and CW MWR, especially at the higher dose rate, effectively enhanced operant control over timeout responding and (3) the equivalent effects of CW and PM MWR support the hypothesis of a thermal basis for their effect despite the apparent inability to detect changes in whole body temperature.

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Study Type: Behavior; IN VIVO; RAT
Effect Type: Equivalence of pulsed and CW RFR at comparable SARs in altering cued, fixed-ratio bar-pressing rates for food and noncued timeout rates during RFR exposure
Frequency: 1.3 GHz
Modulation: CW or 1-microsecond pulses at 600 pps (0.0006 duty cycle)
Power Density: 9.2-17.2 mW/sq cm Av (estimated)
SAR: 3.6-5.9 W/kg CW; 3.6-6.7 W/kg pulsed

EXPOSURE CONDITIONS: Groups of 14 or 15 rats each were concurrently sham-exposed and exposed to circularly polarized (electric vector transverse to the propagation direction) CW or pulsed RFR in individual circular-waveguide systems in a room maintained at 23 deg C and about 50% relative humidity. Exposures were for 5 days, 3 hr/day. Each system was equipped for automatically recording cued and noncued food-acquisition behavior during exposure. The rats, which were not restrained, were facing the RFR source during operant performance.

OTHER INFORMATION: This investigation was similar to an earlier study in which only pulsed RFR was used (Lebovitz, 1981). The primary purpose of the later study was to ascertain whether pulsed and CW 1.3-GHz RFR at about the same whole-body SAR would have similar effects on the same behavioral paradigm as that used in the previous study.

The set of waveguide systems (which were similar to those designed by Guy and Chou, 1976) and the methods used to quantify the dosimetry were described in detail by Lebovitz and Seaman (1980). In brief, the SAR for a 300-g rat at 1.3 GHz was 2.2 W/kg per W of forward power, and dividing 1 W by the cross-sectional area of the waveguide (177 sq cm) yielded an estimated power density of 5.65 mW/sq cm. Thus, the range of average power densities corresponding to SARs in the range 3.6-6.7 W/kg was about 9.2-17.2 mW/sq cm. With a duty cycle of 0.0006 for the pulsed RFR, the peak power densities corresponding to 3.6 and 6.7 W/kg were 15.4 and 28.7 W/sq cm.

Within each waveguide were a vertical displacement bar, a spotlight for illuminating the bar as a cue (visual discriminative stimulus), and means for delivering a food pellet near the bar when appropriate.

Groups of Long-Evans hooded rats about 40-60 days old were deprived of food until they were 85% of their free-feeding weights and were trained to bar press for food pellets at fixed-ratio (FR) schedules increasing from FR-1 to FR-5 (requiring, respectively, 1 bar press or 5 successive presses to obtain a pellet). The rats were then trained daily on a multiple schedule starting with a 15-min (S+) interval during which the bar was illuminated with the spotlight and pellets were available at FR-25, followed by a 10-min timeout (S-) interval during which the spotlight was extinguished and bar presses were recorded but not rewarded with pellets. Each daily session consisted of 6 contiguous thus-paired 25-min periods, which were numbered 1-6 sequentially.

Groups of up to 30 rats were trained to achieve high and steady rates of performance at FR-25 during the S+ intervals. Following a 3-week baseline period, the rats were assigned to the RFR or sham group, with the 2 groups matched by baseline S+ performance. Exposures were for 180 min at the same time each day for 5 days. Recording of S+ and S- responses for the daily 150-min behavioral sessions was begun 15 min after the start of exposure and was terminated 15 min before the end of exposure.

The primary data for each rat were the numbers of bar-presses it did during each S+ and S- interval each day. The values of S+ responses for all correspondingly numbered intervals were summed weekly and the totals were converted into weekly individual response rates (bar-presses per min) for each interval. The values of S- responses were similarly treated. Distinct groups were used for exposures to the CW and pulsed RFR (and for their respective concurrent sham exposures) and the weekly results for each group were averaged and given appropriate statistical treatments.

When not in their waveguides, the rats were kept in home cages with water available ad libitum. In addition, each rat was given 6 g of food daily irrespective of its operant performance.

Fifteen rats were exposed to CW RFR at 5.9 W/kg. The S+ results for the baseline week preceding exposure (Fig. 1) showed a trend toward decreasing response rates of about 10% from period 1 to period 6 for both the RFR- and sham-exposed groups but no significant differences between the groups. The S+ rates of both groups for periods 1 and 2 were higher for the week of exposure than for the corresponding periods of the baseline week, and both groups exhibited a downward trend, but the decline in response rate was significantly faster for the RFR group.

Breakdown of those results by operant days and period numbers (Fig. 5) showed no significant differences in daily period-1 response rates between groups for the entire 2-week (baseline and exposure) duration. However, the daily S+ response rates of the RFR group for periods 3 and 6 declined significantly during the first 3 days of exposure, with a sharper decline for period 6. Recovery occurred during the last 2 days (to values comparable with those of the sham group).

During the baseline week, the S- response rates of this RFR group were initially higher than for the sham group, but declined faster between period 4 and period 5, so that the rates for the 2 groups were comparable for periods 5 and 6. During the week of exposure, the sham group exhibited higher rates for periods 1 and 2 than they did for the same periods of the baseline week, and approximately the same rate of decline. However, the rates of the RFR group dropped sharply for periods 1-3 to almost zero for periods 4-6.

Breakdown of the S- results by operant days and period numbers (Fig. 5) showed a significant decline of the period-1 response rates of the RFR group during the first 3 days of exposure, followed by recovery during the last 2 days. However, the response rates of this group for periods 3 and 6 dropped to, and remained at, extremely low values for the entire exposure week.

Response rates during both S+ and S- for 15 rats exposed to pulsed RFR at 6.7 W/kg and 15 sham-exposed rats (Fig. 2) were similar to those with CW RFR at 5.9 W/kg (Equipment limitations did not readily permit closer match of SARs.) The investigator noted that the results with pulsed RFR at this level were similar to those obtained in the previous study with pulsed RFR at the same level (Lebovitz, 1981), and that the occurrence of similar changes in S+ response rates with CW RFR at a comparable SAR indicated that the effect was not ascribable to the pulsed character of the RFR.

Another group was exposed to CW RFR at 3.6 W/kg (concurrently with a sham group). The S+ response rates (Fig. 3) of the RFR group during the baseline week were consistently lower than those of the sham group, but the rates of decline for periods 1-6 were essentially the same. Also,

similar results were obtained for the week of exposure except that the initial (period-1) response rates for both groups were higher than the initial rates for the baseline week. The S- response rates of the RFR group were consistently higher than the rates of the sham group for the baseline week, with comparable rates of decline, thus yielding no significant differences between the groups. The S- results for the week of exposure showed that the response rates of the RFR group were consistently higher than of the sham group; the response rates of both groups declined for periods 1-6, but the decline was significantly faster for the RFR group. Again, these results were consonant with those of the previous study. (Breakdowns of the S+ and S- results of these groups by operant days and period numbers were not presented.)

Separate groups of 5 rats each were used to determine rises of core temperature due to RFR exposure. The rectal temperature of each rat was measured immediately before placing the rat in the waveguide for 1 or 3 hr and was measured again within 10 min after removal of the rat from the waveguide. Exposures of 1 or 3 hr to CW RFR at 3.5 W/kg or pulsed RFR at 3.4 W/kg (approximate threshold for the foregoing behavioral effects) yielded no significant differences in rectal-temperature changes as compared with rats sham-exposed for the same durations. However, exposure at 6.3 W/kg (CW) or 6.4 W/kg (pulsed) yielded increases of 0.5 to 1 deg C, with no significant duration-dependent differences.

CRITIQUE: As was true for the previous investigation, the engineering aspects were excellent, and the statistical treatment of the data provided a sound basis for the conclusions reached. Moreover, the presentation of the data at 5.9 W/kg (CW) by operant days (a format lacking in the previous paper) provided greater insight into the time-dependent aspects of the results. Especially noteworthy in this context was that the daily S+ response rates for period 1 were not significantly affected by the entire week of RFR exposure, and that the declines in these rates occurred progressively in the subsequent periods of each session.

Also more clearly evident were the virtually immediate sharp declines in S- response rates for all periods at the onset of RFR exposure. In the absence of the light cue and pellet rewards, it is possible that the rats were thoroughly confused by the presence of the RFR. Another possibility, suggested by the investigator, was that without such reinforcement, the rats endeavored to reorient themselves so as to redistribute the thermal burden added by the RFR.

As indicated by the investigator, the thermal basis for the observed behavioral changes is evident, with a threshold SAR of about 3.5 W/kg irrespective of whether the RFR is CW or pulsed. Also, even though the pulse width (1 microsecond) and peak power density (estimated to be about 28.7 W/sq cm) were sufficient to produce the RFR-auditory effect, there is little doubt that perception of the pulses as sound (if it occurred) was not a factor in the results obtained.

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BEHAVIOR
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Monahan, J.C. and W.W. Henton
FREE-OPERANT AVOIDANCE AND ESCAPE FROM MICROWAVE RADIATION
In D.G. Hazzard (ed.), SYMPOSIUM ON BIOLOGICAL EFFECTS AND MEASUREMENT OF RADIO FREQUENCY/MICROWAVES, U.S. Department of Health, Education, and Welfare, HEW Publication (FDA) 77-8026, pp. 23-33 (1977a)

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AUTHOR ABSTRACT: Previous research has established that exposure to microwave radiation can effect both innate and acquired behaviors. The purpose of the present investigation was to determine the stimulus properties of microwaves in a free-operant avoidance-escape paradigm. Subjects were CF1 male mice weighing 35-42 g. Two groups of subjects were employed. Experimental subjects received microwaves (2.45 GHz CW) plus tone and sham subjects received tone only. During the session, all subjects could make responses by interrupting a light beam which passed through the animal holder. If no response was made, then the tone remained on for the entire session. Experimental subjects always received microwaves paired with the tone. If the subjects responded during the tone, it was terminated and remained off for 12 seconds. This was considered an escape response. If the subjects responded during this tone-off period, each response would delay the onset of tone for 12 seconds. This was considered an avoidance response.

The data show a clear difference in response patterns between experimental and control subjects both in frequency and variability of responding. Furthermore experimental subjects could be categorized by their response patterns into escape animals, avoidance animals, and mixed response animals. For a given subject his response pattern remained consistent across experimental sessions. These data show that microwave radiation can serve as a noxious stimulus which will maintain an active instrumental avoidance or escape behavior over repeated experimental sessions.

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Study Type: Behavior; IN VIVO; MOUSE
Effect Type: Avoidance and escape from RFR as a noxious stimulus
Frequency: 2.45 GHz
Modulation: CW
Power Density: 45 mW/sq cm (estimated)
SAR: 46 W/kg

EXPOSURE CONDITIONS: Five mice were exposed to RFR individually in a ventilated opaque Plexiglas container within a waveguide system maintained at 24 deg C and 40-50% relative humidity. The container was large enough to permit free movement of the mouse. Three mice were similarly sham-exposed. All RFR- and sham exposures were paired with a 2900-Hz tone. Behavioral sessions were for 30 min; exposure durations in each session were determined by the responses of the mouse.

OTHER INFORMATION: The waveguide system was instrumented for continuously recording forward, reflected, and transmitted powers. The values of net power (forward power minus the reflected and transmitted powers) when the RFR was on during each 30-min session, which varied with mouse movements, were integrated with time and divided by the sum of the RFR-on intervals to obtain the mean rate of energy absorption by the mouse during the session. Division of this rate by the mass of the mouse yielded its mean SAR (which averaged about 46 W/kg for the 5 mice). The forward power used was 2.7 W and the transverse dimensions of the waveguide were 10.9x5.5 sq cm, from which a power-density estimate of 45 mW/sq cm is obtained.

A beam of light was passed through an appropriately located set of aligned holes in the side walls of the waveguide and the mouse container to a photosensor, and interruption of the beam constituted the basic response of the mouse. Paired with the RFR (or sham exposure) was a 2900-Hz tone. The procedure was to turn on the RFR and tone (or tone alone) 12 seconds after the beginning of a session. These stimuli remained on as long as no response was made. Once on, a beam-interruption response terminated the stimuli, which remained off for 12 seconds in the absence of another response during the period. This behavior was characterized as an escape response. If the mouse responded again during the 12-second interval of no RFR and tone, each such response would delay their onset for 12 seconds. These actions were characterized as avoidance responses.

As seen in Fig. 3, the responses of mice 1 through 5 (which were exposed to RFR and tone) averaged over 8 sessions yielded mean cumulative exposures (total RFR-on durations) ranging between 10 and 20 seconds per session, with relatively small standard deviations. Whenever the RFR was turned on, these animals terminated the RFR by a response within 20 seconds. By contrast, for mice 6 through 8 (which were exposed only to the tone), the mean cumulative exposures were much larger and exhibited greater intersession variabilities than the RFR-exposed mice.

Figure 4 shows that representative total numbers of responses, during 30-min sessions, for RFR-exposed mice 1 through 5 were about 300, 350, 150, 450, and 470, respectively. (It should be noted that the captions for Figs. 4 and 5 are interchanged.) The values for sham-exposed mice 6 through 8 were about 50, 170, and 100; mouse 7 responded at a fairly constant rate during the session, but mouse 6 responded mostly during the first 15 min and mouse 8 mostly during the last 15 min.

Figure 5 presents the mean percentages of escape responses by mice 1-5, and Fig. 6 shows their mean number of avoidance responses per escape response. Mouse 3 exhibited nearly 30% escape responses and only 2 avoidance responses per escape response, so its behavior was categorized as primarily "escape." Mice 5 and 6 showed less than 10% escape responses, but 18 and 13 avoidance responses per escape response, respectively, so their behavior was classified as primarily "avoidance." The escape responses of mice 1 and 2 were about 15% of

their total responses and they performed about 6 avoidance responses per escape response, so their behavior was categorized as "mixed." Corresponding data for sham-exposed mice 6-8 were not presented, presumably because their total response rates were too low and inconsistent.

The investigators concluded that RFR can be an aversive stimulus.

CRITIQUE: The paper stated that the mice were used without any prior training. However, in response to a question following presentation of the paper, the presenter indicated that the behavior of the mice stabilized after 15-20 sessions and the results presented were for post-stabilization sessions.

In response to another question, the presenter stated that in a previous study (Monahan and Ho, 1976) in which mice could only lower their RFR absorption by body reorientation in the waveguide, continuous exposure at about the same SAR (46 W/kg) for 15 min caused a rectal temperature rise of about 0.5 deg C. Thus, it is most likely that the behavioral responses observed in the present investigation had a thermal basis.

As indicated, there were wide differences in behavior patterns among the 5 RFR-exposed mice. Because of such differences, it is difficult to interpret the significance of characterizing the mice, especially since so few mice were used. The investigators did state that each mouse maintained rather than altered its response pattern, but that the determinants governing the individual patterns were dependent on the available contingencies and not on the RFR itself. Nevertheless, it is clear that the mice were responding to the presence of the RFR in ways that tended to minimize their cumulative exposure times per session, which were significantly smaller than for the sham-exposed mice.

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Monahan, J.C. and W.W. Henton
THE EFFECT OF PSYCHOACTIVE DRUGS ON OPERANT BEHAVIOR INDUCED BY MICROWAVE RADIATION
Radio Sci., Vol. 14, No. 6S, pp. 233-238 (1979)

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AUTHOR ABSTRACT: A total of five male CF1 mice was trained to escape from or to avoid 2.45-GHz CW microwave radiation by emitting an operant response. The response consisted of an animal's interruption of a light beam passing through a conditioning chamber. If an animal responded while microwaves were on, radiation was terminated and remained off for 12 seconds (an escape response). If an animal responded during the off period, each response (constituting avoidance) would reset a timer that delayed the onset of microwaves for another 12 seconds. All responses involved discriminated cueing by a 2900-Hz sonic stimulus that was paired with microwave irradiation. Averaged dose rates were approximately 45 mW/g, but the duration of irradiation varied with the subjects' escape-avoidance behavior.

When stable baselines of responding were established, each subject was tested following administration of each of three psychoactive compounds at varying dosages: chlordiazepoxide, d-amphetamine, and chlorpromazine. Chlordiazepoxide resulted in a decreased percentage of avoidance responding coupled with an increased percentage of escape responding. A substantial increase in the animals' cumulative exposure to microwaves was also noted when they were dosed with this drug. The data based on administration of chlorpromazine and of d-amphetamine were highly variable both within and among subjects.

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Study Type: Behavior, Multiagent Interactions; IN VIVO; MOUSE
Effect Type: Avoidance and escape from RFR and the effects of psychoactive drugs on such responses
Frequency: 2.45 GHz
Modulation: CW
Power Density: 45 mW/sq cm (estimated)
SAR: 45 W/kg

EXPOSURE CONDITIONS: Five mice were exposed to RFR individually in a ventilated opaque Plexiglas container within a waveguide system maintained at 24 deg C and 50% relative humidity, with air flow at 38 l/min. The container was large enough to permit free movement of the mouse. Behavioral sessions were for 30 min; exposure durations in each session were determined by the responses of the mouse.

OTHER INFORMATION: The behavioral paradigm in this investigation was similar to that used in an earlier study (Monahan and Henton, 1977a), and the objective was to determine whether injection of the specified drugs would alter the behavioral responses to RFR found previously.

The waveguide system was instrumented for continuously recording forward, reflected, and transmitted powers. The values of net power (forward power minus the reflected and transmitted powers) when the RFR was on during each 30-min session, which varied with mouse movements, were integrated with time and divided by the sum of the RFR-on intervals to obtain the mean rate of energy absorption by the mouse during the session. Division of this rate by the mass of the mouse yielded its mean SAR (which averaged about 45 W/kg for the 5 mice). The forward power used was 2.7 W and the transverse dimensions of the waveguide were 10.9x5.5 sq cm, from which a power-density estimate of 45 mW/sq cm is obtained.

A beam of light was passed through an appropriately located set of aligned holes in the side walls of the waveguide and the mouse container to a photosensor, and interruption of the beam constituted the basic response of the mouse. Paired with the RFR was a 2900-Hz tone. The procedure was to turn on the RFR 12 seconds after the beginning of the session. These stimuli remained on as long as no response was made. Once on, a beam-interruption response terminated the stimuli, which remained off for 12 seconds in the absence of another response during the period. This behavior was characterized as an escape response. If the mouse responded again during the 12-second interval of no RFR and tone, each such response would delay their onset for 12 seconds. These actions were characterized as avoidance responses.

Stable baseline data were obtained for each mouse after 10 to 15 30-min RFR-plus-tone exposure sessions conducted daily. The baseline behavioral pattern of each subject was self-consistent, with only minor variability, but the patterns differed among the subjects and could be classified as primarily escape-response, primarily avoidance-response, or mixed-response. Representative patterns were shown in Fig. 3. The behavior of mouse 3 exemplified the escape-response pattern, which exhibited a relatively low response rate (about 100 responses per session), with most responses occurring after the onset of RFR, and yielding a mean RFR on-time of 30 seconds or more per session. Mouse 4 exhibited a primarily avoidance-response pattern consisting of a high response rate (about 700 responses per session), with most responses occurring during the RFR-off periods, and yielding a mean RFR on-time of 10 seconds or less. Mouse 2 yielded a mixed pattern of escape and avoidance responses occurring at an intermediate rate (about 400 per session). The patterns of mice 1 and 5 were of the mixed type but their response rates were not given in Fig. 3.

After the baseline period, the mice were intraperitoneally administered chlordiazepoxide (1, 5, 10 mg/kg), chlorpromazine hydrochloride (0.25, 0.5, 10 mg/kg), d-amphetamine sulfate (0.5, 1, 2 mg/kg), or saline in random sequence once a week 15 min before a session. Figure 4 showed that saline injection reduced the total response rate of mouse 1 to 80% of its mean control value, increased the total response rate of mouse 3 to 120%, and did not substantially affect the total rates of the other 3 mice.

Injection of 10 mg/kg of chlordiazepoxide into mouse 1 yielded the same reduction as saline (to about 80%), but doses of 1 and 5 mg/kg reduced the rate further, to about 60% and 40%, respectively. For mouse 2 (unaffected by saline), chlordiazepoxide reduced the rate to about 60% for doses of 1 and 5 mg/kg, and to about 40% for 10 mg/kg. For mouse 3, (rate increased to 120% by saline), 1 mg/kg of the drug yielded a rate comparable to its control value (i.e., 100%), but 5 and 10 mg/kg produced reductions to about 80%. Mouse 4 (unaffected by saline), showed substantial rate reductions from all 3 doses the drug, but the least reduction (to about 60%) was for 5 mg/kg, with successively larger reductions (to about 50% and 30%) for 1 and 10 mg/kg. Mouse 5 (rate slightly decreased by saline) showed successively larger reductions with dose: to about 80% for 1 mg/kg, to 40% at 5 mg/kg, and to 10% at 10 mg/kg.

The total numbers of responses of each mouse produced by injection of saline or chlordiazepoxide at each dose were divided into avoidance and escape responses and compared with the respective control values. The results were presented in terms of absolute numbers of avoidance responses (Fig. 5) and percentages of escape responses (Fig. 6). For mouse 1, the saline reduced both the avoidance and escape responses, but only slightly. The drug had little effect on avoidance or escape responses at 10 mg/kg, but reduced the avoidance rate and increased the percentage of escape responses at 1 and 5 mg/kg, with the latter effect higher for 1 than for 5 mg/kg. Qualitatively similar, but somewhat inconsistent results were obtained for mice 2, 3, and 5. The results for mouse 4 were less equivocal; with saline, the avoidance and escape response rates were comparable to their respective control means, whereas 10 mg/kg of the drug produced maximum reduction in the avoidance rate and maximum increase in the percentage of escape responses. However, such changes were least for 5 rather than 1 mg/kg.

Less equivocal were the cumulative exposure durations per session. The mean baseline values ranged from about 8 seconds (mouse 4) to about 30 seconds (mouse 3), and saline did not significantly alter the value for each mouse. However, chlordiazepoxide doses of 5 and 10 mg/kg increased the exposure durations of all 5 mice significantly with a clear dependence on dose.

Because of the high degree of variability of the results with the other drugs, no detailed data were presented. The investigators did state that chlorpromazine appeared to lower the response rate without increasing the cumulative exposure duration, and that d-amphetamine at the highest dose (2 mg/kg) increased the response rate in several subjects and decreased the exposure duration.

CRITIQUE: The baseline results were fairly similar to those obtained previously (Monahan and Henton, 1977a). However, the description of many of the results with saline and chlordiazepoxide was rather obscure. The investigators stated the following: "The means of total responses per session by subjects given either saline or chlordiazepoxide were

compared with the means of responses during control sessions (Figure 4)." Not clear in this statement is whether each mouse was treated with each dose of the drug (or saline) more than once. The absence of error bars in Fig. 4 (and in the subsequent figures) could be taken to mean that each treatment was administered prior to only one session. If this were the case, the credence given to the quantitative aspects of the results is diminished and it is more difficult to assess the dose-dependence involved, especially with so few animals having such large differences in baseline behavioral patterns. Use of the same mice for all of the drugs and dosages is also questionable, even though each mouse was given only one treatment per week and presumably recovered between treatments.

Also obscure in the statement above is the meaning of "control sessions." Presumably these were stabilized baseline RFR-exposure sessions prior to drug or saline injection rather than exposure sessions between the weekly drug treatments.

As in the previous investigation, it is difficult to interpret the significance of characterizing the baseline behavioral patterns of the mice, in view of the small number of mice used and the large differences among them. Nevertheless, the results do qualitatively indicate that chlordiazepoxide at the higher doses did increase the rates of escape responses and the cumulative exposure durations, but the dependence on dose is obscure.

It should be noted that Thomas et al. (1979) reported that pulsed 2.45-GHz RFR at an average power density of 1 mW/sq cm altered the effect of chlordiazepoxide on a fixed-interval behavioral paradigm of rats. However, the occurrence of the RFR-auditory effect could not be ruled out. The effects of RFR and dextroamphetamine on behavior were studied also by Thomas and Maitland (1979). Thomas et al. (1980) also investigated the effects of RFR in combination with chlorpromazine and diazepam.

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Gage, M.I., E. Berman, and J.B. Kinn
VIDEOTAPE OBSERVATIONS OF RATS AND MICE DURING AN EXPOSURE TO 2450-MHZ MICROWAVE RADIATION
Radio Sci., Vol. 14, No. 6S, pp. 227-232 (1979)

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AUTHOR ABSTRACT: Rats and mice were observed in a microwave field to see if they would take behavioral action to minimize absorption of microwave energy by altering their orientation with respect to the E field. Individual rats were housed either in a cylindrical or in a cuboidal container, and mice were housed in a cuboidal container. They were placed in an anechoic chamber at 22 or 28 deg C for a one-hour pre-exposure period. During the second hour, animals were exposed to 2450-MHz CW microwaves under far-field conditions at a power density of 15 mW/sq cm. Videotape samples of their positions were taken during a two-hour session. Six rats and six mice were observed at each level of temperature and were scored for orientation relative to the electric vector or to the magnetic vector of the microwave field.

Results indicate that 2450-MHz CW microwaves did not cause animals to alter their position from that adopted in response to environmental or caging conditions. The specific absorption rate (SAR) in rats was not dependent on their orientation in the field. The SAR in mice was dependent on their orientation in the field but their orientation did not show changes interpretable as attempts to reduce microwave absorption. Containers reduce the SAR and, depending on shape, may reduce the orientation-dependence of the SAR.

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Study Type: Behavior; IN-VIVO; RAT, MOUSE
Effect Type: Detection of RFR and reorientation in the field to minimize RFR absorption
Frequency: 2.45 GHz
Modulation: CW
Power Density: 15 mW/sq cm
SAR: For rat at 15 mW/sq cm: 2.1-2.6 W/kg in cylinder and 3.2-3.6 W/kg in cuboid; for mouse at 15 mW/sq cm in cuboid: 6.5-11.1 W/kg

EXPOSURE CONDITIONS: Rats and mice in ventilated Styrofoam cuboidal or Plexiglas cylindrical containers were individually exposed for 1 hr from above to far-field RFR (electric and magnetic vectors horizontal and perpendicular to one another) within an anechoic chamber illuminated with incandescent light and maintained at 22 or 28 deg C and 50% relative humidity while their movements were sampled and videotaped with a TV camera.

OTHER INFORMATION: Each animal was studied for 2 hr, during which its behavior was videotaped every 30 seconds for 2-3 seconds. The animals were exposed to the RFR only during the second hr of the session. The 6

rats used weighed 260-360 g and each was housed, during the session, within either a cuboidal container of Styrofoam (coated with quinine to prevent gnawing) or a cylindrical container of Plexiglas. The weights of the 6 mice were in the range 25-33 g and each was housed only in the cuboidal container during the session.

The videotaped records were scored by noting the numbers of times each animal assumed positions in which its long axis was parallel to the electric (E) vector, parallel to the magnetic (H) vector, and any other orientations during the 2-3 second sampling periods. These numbers were summed over each of the eight 15-min periods (30 samples per period) of its 2-hr session.

Observations of the rats at 22-deg-C ambient temperature showed that after exploratory activity during the first 15-min period, they generally became less active and usually adopted curled positions not preferentially oriented relative to either the E- or H-vector, and appeared to be sleeping in such curled positions. Turning on the RFR during the second hour did not alter their behavior significantly. The sums of occurrences of each orientation for each 15-min period were averaged for the 6 rats. For the rats in the cylindrical container, the mean values for the E- and H orientations ranged from 1 to 6 occurrences per period, with no statistically significant differences between the orientations or for RFR vs no RFR. By contrast, the mean numbers of occurrences of the "other" orientation ranged from 19 to 26, but again no significant RFR-induced differences. Comparable results were obtained for the rats in the cuboidal container.

At 28 deg C, the rats frequently stretched out on their backs during RFR exposure, but again with no preferential E- or H orientation.

The SARs of rat carcasses having a comparable range of body weights were measured without and within the two types of container at 10 mW/sq cm in both the E- and H orientations by twin-well calorimetry. The mean value without a container was 2.2 W/kg for both orientations. The presence of the cylindrical container reduced the SARs to 1.4 and 1.7 W/kg for the E- and H orientations, respectively, but these means did not differ from one another statistically. (The corresponding SARs at 15 mW/sq cm used in the behavioral sessions were 2.1 and 2.6 W/kg.) The presence of the cuboid container increased the E-orientation SAR to 2.4 W/kg and decreased the H-orientation SAR to 2.1 W/kg, but again the difference was not significant. (The corresponding values at 15 mW/sq cm were 3.6 and 3.2 W/kg.) Based on these measurements, the investigators indicated that the absence of a preferential-orientation effect might have been anticipated.

The behavioral results for the 6 mice (studied only in the cuboidal container) at 22 deg C were qualitatively similar to those for the rats. The mean number of occurrences for the E orientation rose approximately linearly from about 1 to 4 during the 2-hr session and the mean value for the H orientation was less than 1 for the first 5 periods, rose to 4 for period 6, and diminished linearly to 0 for periods 7 and 8. The

values for the "other" orientation diminished from 29 to 27 for periods 1-5, dropped to 22 for period 6, and rose to 25 for periods 7-8. The differences were not statistically significant.

Increasing the temperature to 28 deg C rendered the mice more active, and their means for both the E- and H orientations rose sharply during periods 3-6 from less than 3 to the range 8-12 while the mean for the "other" orientation dropped from 30 to within the 8-12 range. Analysis of variance indicated that although the presence of the RFR produced larger numbers of E- and H orientations, the differences between these two orientations were not significant.

Measurements of SARs for mouse carcasses at 10 mW/sq cm without the cuboidal container yielded 8.2 and 4.1 W/kg for the E- and H orientations, respectively (a 2:1 ratio). With the mice within the cuboidal container, the respective values were 7.4 and 4.3 W/kg (ratio 1.7). (The corresponding SARs at 15 mW/sq cm are 11.1 and 6.5 W/kg.) Thus, unlike for the rats, it was possible for the mice to reduce their SARs by aligning their long axes with the H-vector, but the results indicate that they did not do so. The investigators suggested that perhaps rodents do not sense or differentially respond to 2.45-GHz CW RFR during an initial 1-hr exposure.

CRITIQUE: The investigators did not state the angular limits within which the animals were considered to have taken the E- or H orientation; the size of these angular sectors would determine the counts of occurrences for each such orientation, and more importantly, the ratios of counts for these orientations to the counts for the "other" orientation. Also not discussed was the rationale for sampling the behavior of the rodents for only 2-3 seconds of every 30-second interval in the 2-hr sessions and how representative such samples were. However, these points are directed more toward the quantitative aspects of the results, rather than the qualitative findings.

The finding that the mice did not endeavor to reduce their whole-body SARs by appropriate orientation appears to be at variance with the results of Monahan and Ho (1976, 1977), who found that mice exposed to RFR in a waveguide system presumably altered their orientations and perhaps their body configurations, as evidenced by reductions of their energy-absorption rates (rather than by visual observations of the mice). However, unlike the far field used by Gage et al., the field within a waveguide is neither transverse-electromagnetic nor uniform over the waveguide cross-section. Also, the propagation direction in the latter system was parallel, rather than transverse, to the long axis of the mouse. Thus, whole-body SAR may be more sensitive to orientation in a waveguide than under far-field conditions. Moreover, the internal distribution of local SAR may differ significantly for the two exposure methods. For these reasons, the validity of such comparisons of findings is open to question.

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In C.C. Johnson and M. Shore (eds.), BIOLOGICAL EFFECTS OF ELECTROMAGNETIC WAVES, U.S. Department of Health, Education, and Welfare, HEW Publication (FDA) 77-8010, pp. 274-283 (1976)

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Thomas, J.R., J. Schrot, and R.A. Banvard
COMPARATIVE EFFECTS OF PULSED AND CONTINUOUS-WAVE 2.8-GHZ MICROWAVES ON TEMPORALLY DEFINED BEHAVIOR
Bioelectromagnetics, Vol. 3, No. 2, pp. 227-235 (1982)

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AUTHOR ABSTRACT: The effects of pulsed-(PW) and continuous-wave (CW) 2.8-GHz microwaves were compared on the performance of rodents maintained by a temporally defined schedule of positive reinforcement. The schedule involved food-pellet reinforcement of behavior according to a differential-reinforcement-of-low-rate (DRL) contingency. The rats were independently exposed to PW and to CW fields at power densities ranging from 1 to 15 mW/sq cm.

Alterations of normal performance were more pronounced after a 30-minute exposure to the PW field than to the CW field. The rate of emission of appropriately timed responses declined after exposure to PW at 10 and 15 mW/sq cm, whereas exposure at the same power levels to the CW field did not consistently affect the rate of responding. Change in performance associated with microwave exposure was not necessarily related to a general decline in responding; in some instances, increases in overall rates of responding were observed.

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Study Type: Behavior; IN VIVO; RAT
Effect Type: Relative alterations of a behavioral paradigm by pulsed and CW RFR at comparable average power densities
Frequency: 2.8 GHz
Modulation: CW or 2-microsecond pulses at 500 pps (0.001 duty cycle)
Power Density: 1, 5, 10, or 15 mW/sq cm Av
SAR: 0.2, 1.2, 2.5, or 3.6 W/kg

EXPOSURE CONDITIONS: Rats constrained in a plastic-mesh sleeve holder were exposed individually for 30 min to pulsed or CW RFR in the far field of a standard-gain horn within an anechoic chamber at an ambient temperature in the range 20-23 deg C. The field was vertically polarized, with the H-vector parallel to the rat's long axis. Sham exposures were also performed under similar conditions.

OTHER INFORMATION: Whole-body SARs were calculated from rises of rectal temperatures measured 5 min before and after 30 min of sham- and RFR-exposure at each power density. Mean temperature increases for 1, 5, 10, and 15 mW/sq cm were 0.1, 0.5, 1.1, and 1.5 deg C, which yielded SARs of 0.2, 1.2, 2.5, and 3.6 W/kg, respectively.

Four male albino rats (Nmri:O[SD]CV) maintained at approximately 80% of their free-feeding weights were studied. The rats were initially trained on a lever-pressing schedule in which a food pellet was delivered only when the interval between 2 successive responses (the

interresponse time or IRT) was not less than 1 second or more than 2 seconds. After about 20 sessions of continuous reinforcement, a differential-reinforcement-of-low-rate (DRL) schedule was instituted, in which the required 1-to-2-second IRT was reinforced only if the time interval between successive correct IRT responses was more than 8 seconds but less than 12 seconds. A correct DRL response was reinforced by delivery of a pellet, and the DRL interval was restarted. Incorrect IRT responses were not reinforced and had no effect on the timing of the 8-second DRL interval. A correct IRT response in less than 8 seconds or more that 12 seconds after a correct IRT response was not reinforced and the DRL interval was restarted. Daily sessions were conducted 5 days per week. Each session was terminated after delivery of 150 pellets or after 1 hr, whichever occurred first.

The rats were trained for 3 months to stabilize their baseline rates and to accustom them to the sleeve holder. After completion of training, the rats were exposed once a week to either CW or pulsed RFR at an average power density of 1, 5, 10, or 15 mW/sq cm for 30 min and tested following exposure. The levels were administered in mixed order, each rat was exposed at least three times at each level (except for 5 mW/sq cm), and the response rate (number of correct DRL responses per second) in each session was recorded. Comparison data were obtained for each rat by testing it, between RFR-exposure sessions, for baseline performance and for performance after sham exposure, the results of which were averaged.

As seen in Fig. 1, the response rates of the 4 rats for the CW RFR were comparable to their respective mean control values and there was no consistent variation of response rate with power density. The response rates for the pulsed RFR, however, were significantly lower than for the corresponding levels of CW RFR, and a statistically significant downward trend of rate with increasing average power density was observed.

In Fig. 2, the record of the cumulative number of correct DRL responses of one rat and its cumulative number of total responses were presented for a representative baseline session and for representative sessions involving exposure to pulsed RFR at 10 and 15 mW/sq cm and to CW RFR at 15 mW/sq cm. These records show that for the pulsed RFR at 10 mW/sq cm, the rate of correct responses was lower than the rate of correct baseline responses while the rate of total responses was higher than the baseline rate of total responses. For the pulsed RFR at 15 mW/sq cm, the rates of correct and total responses were both lower than the respective baseline rates and the corresponding rates at 10 mW/sq cm. By contrast, for the CW RFR at 15 mW/sq cm, the correct-response and total-response rates were only slightly lower than the corresponding baseline rates.

CRITIQUE: The use of rectal temperature rises to calculate whole-body SARs would be open to question, especially at low power densities (e.g., 1 mW/sq cm), because of thermoregulatory considerations. However, the results of such calculations were consistent with measurements of SAR in a water model of the rat made by Thomas and Maitland (1979).

Another point that may be open to question is the exposure of each animal to all of the RFR conditions, rather than the use of distinct animals for each condition. Presumably the response to this point is that the behavior of the rats returned to baseline values between RFR-exposure sessions. However, details were not presented.

The investigators suggested that the behavioral changes observed after exposure to the pulsed RFR may be the result of alterations in the stimulus control of very precise temporal discriminations, but that the mechanism was unclear. Also mentioned was that the RFR-auditory effect might have been involved. However, because the behavioral tests were performed after completion of the exposures, the persistence of the effect (whatever its nature) of the pulsed RFR for at least 1 hr after exposure is an important aspect to be considered, a point that was made by the investigators.

It should be noted that D'Andrea et al. (1977) reported that the behavior of rats trained to perform a lever-pressing task on a variable-interval schedule of reinforcement during exposure to 600-MHz pulsed RFR (3- or 30-microsecond pulses at 1000 pps) at a peak power density of 170 mW/sq cm and average power densities of 0.51 and 5.1 mW/sq cm was not disrupted. Thus, the findings of Thomas et al. (1982) with regard to pulsed RFR at 10 and 15 mW/sq cm are consonant with those of D'Andrea et al. (1977). However, also reported by D'Andrea et al. (1977) was that the power-density threshold for work stoppage on the task from exposure to CW RFR was between 7.5 and 10 mW/sq cm, a result that does not appear to support the absence of effect with CW RFR at 15 mW/sq cm found by Thomas et al. (1982).

On the other hand, Lebovitz (1983) reported that exposure of rats to 1.3-GHz CW RFR at SARs in the range 3.6-5.9 W/kg and to 1-microsecond pulses at 600 pps at SARs in the range 3.6-6.7 W/kg in a waveguide system were equally effective in reducing their performance on a multicomponent (fixed-ratio, timeout) operant task. The estimated equivalent average power densities ranged from 9.2 to 17.2 mW/sq cm.

Reasons for such contrary findings would be speculative at best.

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Ragan, H.A., R.D. Phillips, R.L. Buschbom, R.H. Busch, and J.E. Morris
HEMATOLOGIC AND IMMUNOLOGIC EFFECTS OF PULSED MICROWAVES IN MICE
Bioelectromagnetics, Vol. 4, No. 4, pp. 383-396 (1983)

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AUTHOR ABSTRACT: Mice were exposed in the far field in an anechoic chamber to 2,880-MHz pulsed microwaves 3 to 7.5 h daily, 5 days/week for 60 to 360 h. Three experiments were performed at average power densities of 5 mW/sq cm and six at 10 mW/sq cm, corresponding to averaged specific absorption rates (SARs) of 2.25 and 4.50 mW/g, respectively. Each experiment consisted of eight mice, with a concurrently sham-exposed group of eight.

In two of three studies at 5 mW/sq cm, there was a significant increase in bone marrow cellularity in the microwave-exposed groups compared to the sham-exposed groups. Significant differences were occasionally seen in erythrocyte, leukocyte, and platelet values from microwave-exposed groups, but were not consistently observed. In one of six groups exposed at 10 mW/sq cm, mean bone marrow cellularity was reduced significantly in the microwave-exposed mice; in another group, the lymphocyte count was increased. In only one exposure (10 mW/sq cm for 360 h) was any significant effect noted on serum proteins; a reduction to 5.1 +/- 0.3 g/dl in the exposed versus 5.6 +/- 0.4 g/dl in the sham-exposed mice. This was due to a decrease in alpha and beta globulins, with no effect on albumin or gamma globulin concentrations.

No effect on bone marrow granulocyte/macrophage colony-forming units (CFU) was revealed following exposure of mice to pulsed microwaves at 5 mW/sq cm. In one of four exposures at 10 mW/sq cm, there was a significant increase in CFU-agar colonies. No significant effects of exposures at 10 mW/sq cm were observed on in vivo and in vitro assays of cell-mediated immune functions. No exposure-related histopathologic lesions were found from examination of several tissues and organs.

Results of these series of exposures of mice at SARs of 2.25 and 4.50 mW/g indicated no consistent effects on the hematologic, immunologic, or histopathologic variables examined.

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Study Type: Immunology and Hematology; IN VIVO; MOUSE
Effect Type: Pulsed-RFR-induced effects on hematologic and immunologic parameters
Frequency: 2.88 GHz
Modulation: 2.3-microsecond pulses at 100 pps (0.00023 duty cycle)
Power Density: 5 or 10 mW/sq cm
SAR: 0.45 W/kg per mW/sq cm

EXPOSURE CONDITIONS: Mice in individual lucite tubes that permitted free movement were concurrently exposed in groups of 8 to far-field

pulsed RFR from a 16.1-dB-gain antenna in an anechoic chamber for 3 to 7.5 hr daily, 5 days/week, for 60 to 360 hr. The tubes were spaced 18 cm apart and were 3.1 m from the antenna for 5 mW/sq cm and 2.1 m for 10 mW/sq cm. Mean ambient temperature and relative humidity were 21.7 deg C and 42%. For exposures at 10 mW/sq cm, ambient temperature typically rose about 1.8 deg C during a 7-hr exposure day. For comparison, each group of 8 RFR-exposed mice was paired with a sham-exposed group of 8 mice. Food and water were not provided during exposure.

OTHER INFORMATION: In the absence of mice, power densities at 2.1 m from the antenna ranged from 7.8 to 11.6 mW/sq cm over the 8 positions, with a mean of 9.9 mW/sq cm. The values at each position with mice in the other 7 positions ranged from 6.7 to 13.6 mW/sq cm, with a mean of 10.2 mW/sq cm. By twin-well calorimetry, the spatial mean SARs were 0.63, 0.34, and 0.37 W/kg per mW/sq cm for the E, H, and K orientations, respectively, with an average of 0.45 W/kg per mW/sq cm or 2.25 and 4.50 W/kg at 5 and 10 mW/sq cm, respectively.

In Study 1, three groups of mice were exposed at 5 mW/sq cm (2.25 W/kg) for 7.5 hr daily for 10 days. After completion of the exposure schedule, blood samples were assayed for various hematologic and serum-chemistry indices, including volume of packed red cells; counts of red cells, white cells, reticulocytes, and platelets; hemoglobin, protein, and triglyceride concentrations; and the number of nucleated femoral bone marrow cells (femoral marrow cellularity). The results, presented in Table 1, indicated that two of the groups yielded higher values of femoral marrow cellularity than the corresponding sham-exposed groups, so the pooled mean for the three groups was significantly higher ($p<0.01$) than the mean for the sham-exposed groups. There were no significant differences between RFR- and sham-exposed groups in the other indices measured.

Similar data were obtained for six groups exposed at 10 mW/sq cm (4.50 W/kg): in Study 2, one group was exposed for 7.5 hr daily for a total of 75 hr; in Study 3, two groups were exposed for 3 hr daily for 60 hr; in Study 4, two groups were exposed for 7 hr daily for 190 hr, and in Study 5, one group was exposed for 7 hr daily for 360 hr. The data for the pairs of groups given the same treatment were pooled. The results, presented in Table 2, showed statistically significant differences in a few indices between some RFR-exposed groups and their corresponding sham-exposed groups, but no consistent pattern across groups. For example, the mean hemoglobin concentration for the group in Study 2 was significantly lower ($p<0.01$) than for its control group, but none of the differences for the other groups was significant. Also, this group yielded a significantly lower ($p<0.05$) mean femoral marrow cellularity, which was contrary to the finding at 5 mW/sq cm, but again the differences for the other groups were not significant. Comparisons of all of the sham-exposed groups indicated relatively large variations among their mean values for these indices.

Following exposure of mice at 5 or 10 mW/sq cm (for unstated durations), samples of nucleated bone-marrow cells were stimulated in vitro with

mouse postendotoxin serum or an extract of pregnant mouse uterus, and the numbers of colony-forming units (CFU) grown in agar were determined. Four culture plates were made from the marrow of each mouse. The results (Table 3) showed no significant differences among the two groups exposed at 5 mW/sq cm and their corresponding sham-exposed groups. Only one of four groups exposed at 10 mW/sq cm yielded a significant difference: the number of CFU for this group was larger than for its control group ($p<0.05$). However, as stated by the investigators, the coefficient of variation (about 40%) was more a reflection of large differences among animals than among plates from individual mice.

To evaluate the effects of RFR on cell-mediated immune (CMI) response, four groups of mice were exposed at 10 mW/sq cm for 3 hr daily, 5 days per week for 20 days. Two of these groups were injected subcutaneously with keyhole limpet hemocyanin (KLH) 13 days prior to termination of exposure, challenged with KLH after the last exposure, and the increases of skin thickness over the preinjection values were determined as a measure of the responses. The other two groups were sensitized to dinitrofluorobenzene (DNFB) by painting DNFB on the shaved abdomen on two successive days. Four days later, ear thicknesses were measured with a micrometer and a challenge dose of DNFB was painted on the dorsal side of the ear. Ear thicknesses were measured again 24 hr later, and the increases over the values prior to challenge were taken as an indication of the responses. Four sham-exposed groups were similarly treated. In addition, a control group of unexposed, unsensitized mice was used for each substance.

The results (Table 4) showed no significant differences in response to KLH between RFR- and sham-exposed groups. The mean response to DNFB of one of the two RFR-exposed groups was significantly higher ($p<0.05$) than for its corresponding sham-exposed group, but was nonsignificantly lower for the other RFR-exposed group. The responses of all four groups to DNFB were significantly higher than for the unexposed, unsensitized control group. Hematologic assays taken after these CMI evaluations (Table 5) showed no significant differences among RFR-exposed, sham-exposed, and unsensitized-control groups in leukocyte counts or serum protein values.

CMI response was also evaluated in vitro for mice, after 20 days of exposure at 10 mW/sq cm for 3 hr daily, by stimulating splenic cell cultures with the T-cell mitogens concanavalin A and phytohemagglutinin, the B-cell mitogen E-coli lipopolysaccharide, and pokeweed mitogen, and determining the uptake of I-125 in newly synthesized DNA. The results (Table 6), expressed as the stimulation index (ratio of counts/min after stimulation to counts/min of unstimulated sample), showed no significant differences between RFR- and sham-exposed groups for any of the mitogens.

Histologic examinations of liver, spleen, kidneys, adrenals, postcervical lymph node, thymus, heart, lung, brain, eyes, femoral bone, and marrow from mice exposed at 10 mW/sq cm for 200 or 360 hr showed no changes ascribable to RFR exposure. In addition, no differences were

detected histochemically in the amount of nonheme iron in the spleens or bone marrow.

CRITIQUE: As noted by the investigators, their results are consistent with those of Smialowicz et al. (1979), who found no significant differences in post-exposure leukocyte counts or hematologic indices, or in post-exposure responses of splenic lymphocytes to mitogen stimulation for mice exposed 15-30 min daily for 1-22 consecutive days to 2.45-GHz CW RFR at power densities in the range 5-35 mW/sq cm (SARs 4-25 W/kg). In addition, no significant immunologic effects were found in two later studies with mice by Smialowicz et al. (1982b, 1982c), the first with 2.45-GHz CW RFR at an SAR of 16.5 W/kg and the second with 425-MHz pulsed and CW RFR at SARs in the range 0.14-8.6 W/kg.

By contrast, as noted by Ragan et al., Huang and Mold (1980) reported that for mice exposed to 2.45-GHz CW RFR at 11 W/kg, the ability of bone-marrow cells to form myeloid and erythroid colonies in vitro was diminished by about 50%. However, the results of Huang and Mold (1980) are questionable because of the probable influence of non-RFR factors such as circadian rhythms and cyclic changes in female mice.

The statistically significant increase in bone marrow cellularity at 5 mW/sq cm found by Ragan et al. (1983) is also open to question because the difference was not significant in one of the three groups exposed at this power density and in five of the six groups exposed at 10 mW/sq cm. Moreover, the mean value for the sixth group was significantly lower than for its corresponding sham-exposed group.

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Yang, H.K., C.A. Cain, J. Lockwood, and W.A.F. Tompkins
EFFECTS OF MICROWAVE EXPOSURE ON THE HAMSTER IMMUNE SYSTEM. I. NATURAL KILLER CELL ACTIVITY
Bioelectromagnetics, Vol. 4, No. 2, pp. 123-139 (1983)

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AUTHOR ABSTRACT: Hamsters were exposed to repeated or single doses of microwave energy and monitored for changes in core body temperature, circulating leukocyte profiles, serum corticosteroid levels, and natural killer (NK) cell activity in various tissues. NK toxicity was measured in a Cr-51 release assay employing baby hamster kidney (BHK) targets or BHK infected with herpes simplex virus.

Repeated exposure of hamsters at 15 mW/sq cm for 60 min/day had no significant effect on natural levels of spleen-cell NK activity against BHK targets. Similarly, repeated exposure at 15 mW/sq cm over a 5-day period had no demonstrable effect on the induction of spleen NK activity by vaccinia virus immunization, that is, comparable levels of NK were induced in untreated and microwave-treated animals.

In contrast, treatment of hamsters with a single 60-min microwave exposure at 25 mW/sq cm caused a significant suppression in induced spleen NK activity. A similar but less marked decrease in NK activity was observed in sham-exposed animals. Moreover, the sham effects on NK activity were not predictable and appeared to represent large individual animal variations in the response to stress factors. Depressed spleen NK activity was evident as early as 4 h postmicrowave treatment and returned to normal levels by 8 h.

Hamsters exposed at 25 mW/sq cm showed an elevated temperature of 3.0-3.5 deg C that returned to normal within 60 min after termination of microwave exposure. These animals also showed a marked lymphopenia and neutrophilia by 1 h posttreatment that returned to normal by 8-10 h. Serum glucocorticosteroids were elevated between 1 and 8 h after microwave treatment. Sham-exposed animals did not demonstrate significant changes in core body temperature, peripheral blood leukocyte (PBL) profile, or glucocorticosteroid levels as compared to minimum-handling controls.

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Study Type: Immunology and Hematology; IN VIVO; HAMSTER
Effect Type: Alterations of natural-killer-cell activity by single and multiple exposures to thermogenic levels of RFR
Frequency: 2.45 GHz
Modulation: CW
Power Density: 15 or 25 mW/sq cm
SAR: 8.0 or 13.3 W/kg

EXPOSURE CONDITIONS: Groups of 1 to 6 hamsters, each hamster within a Mylar-lined Styrofoam cage, were concurrently exposed to far-field RFR 1 hr/day for 5 successive days at 15 mW/sq cm or once for 1 hr at 25 mW/sq cm in an anechoic chamber at 22.5 deg C ambient temperature and 55% relative humidity. Comparison groups were sham-exposed concurrently with the RFR groups in a nearby chamber of the same design. White noise at 72 dB was fed into both chambers to mask background noises.

OTHER INFORMATION: Cage locations within the exposure chamber were adjusted to obtain equal power densities. Calorimetric measurements of SARs for hamster carcasses with their long axes parallel to the E-vector yielded 0.53 W/kg per mW/sq cm or 7.95 and 13.25 W/kg for 15 and 25 mW/sq cm, respectively.

Some groups of hamsters given the 5 successive daily sham-exposures or exposures at 15 mW/sq cm were immunized intraperitoneally with vaccinia virus immediately after the second exposure. Some groups given the single sham-exposure or exposure at 25 mW/sq cm were immunized 4 days before the exposure. Other groups were not immunized. Following exposure, populations of natural killer (NK) cells were prepared from the spleens of the hamsters and were tested as effector cells against targets consisting of cultures of baby hamster kidney (BHK) cells, BHK cells infected with herpes simplex virus (BHK-H), transformed hamster embryo fibroblasts (PARA-7), or PARA-7 cells infected with herpes (PARA-7-H), all grown in Eagle's minimum essential medium (MEM) supplemented with fetal bovine serum, antibiotics, L-glutamine, HEPES, and sodium bicarbonate (complete MEM).

The splenic-NK-cell samples from the hamsters of each treatment group were pooled. For cytotoxicity assays, wells of microtiter plates were seeded with target cells in MEM containing the tracer Cr-51. Herpes simplex virus was added to the media of targets to be infected. Following incubation of the preparations, effector cells were added in quantities that yielded an effector/target ratio of 50. After further incubation and centrifugation, the supernatant was assayed for Cr-51 by gamma counting. Spontaneous and maximum Cr-51 releases were measured in target samples; the latter was promoted by the addition of detergent to the medium. NK-cell cytotoxicity was expressed as "percent specific Cr-51 release," defined as the percentage of net counts in test wells (corrected by subtraction of counts in spontaneous-release wells) to net counts in maximum release wells (similarly corrected). Triplicate wells were used for each assay, and the 2-tailed Student t-test was used for statistical comparisons.

Core temperatures were measured in hamsters under various conditions, but only once in each animal, to avoid repeated stress from the procedure. In one group of untreated hamsters, core temperatures were measured in the animal room. The temperatures of another group were taken immediately after transportation from the animal room to the exposure room for treatment. The temperatures of other groups that were RFR- or sham-exposed for 15, 20, 45, or 60 min were measured immediately after treatment. Subgroups treated for 60 min were removed at 90, 120,

and 180 min to determine cooling rates after cessation of treatment. The temperature change of a group transported to the exposure room but otherwise unhandled, denoted as a "normal" or "minimum-handling" control group, was followed and included in all experiments.

For normal and nonimmunized groups given multiple sham- or RFR exposures at 15 mW/sq cm, NK cytotoxicity against herpes-infected BHK (BHK-H) cells was assayed on days 0 and 2 postexposure. The results (Table 1), showed no significant differences among the three groups on day 0 or between the RFR and normal groups on day 2. However, the mean NK activity of the sham-exposed group on day 2 was significantly lower ($p<0.01$) than for the other groups, a result that was not reproducible and was ascribed to a lower NK level in that group.

Yang et al. (1982) had shown that immunization of hamsters with vaccinia virus increased splenic-NK activity, with maximum activity occurring between days 3 and 6 and return to baseline by day 10. Accordingly, for the normal group and the groups immunized with vaccinia on the second day of the multiple RFR- or sham exposures, NK cytotoxicity against BHK-H cells was assayed on days 3, 6, and 10 postimmunization. For baseline values, the activities of three treatment groups prior to immunization were also assayed.

The results (Table 2) showed that maximum NK activity occurred on day 3 for all three groups and smaller values on days 6 and 10. The authors stated: "There was no significant difference between NK cytotoxicity of microwave-exposed and sham-exposed hamsters 3 and 6 days post-immunization. On day 10 postimmunization, microwave-exposed animals demonstrated significantly reduced cytotoxicity ($P<.01$) compared to controls, but sham-exposed animals did not."

Although the quoted statements are statistically correct, the second sentence could be misinterpreted because, as seen in Table 2, the preimmunization mean values of the RFR and normal groups were comparable, but the value of the sham group was significantly lower ($p<0.05$) than of either group. On day 10, moreover, the value of the RFR group was comparable to its preimmunization value, but the values of the normal and sham groups were significantly higher than their corresponding preimmunization values. This point may be moot because the authors indicated that repetition of this experiment (data not presented) showed no significant differences among the three groups, which led them to conclude that the multiple exposures at 15 mW/sq cm did not reproducibly alter the induction by vaccinia virus of NK activity against herpes-infected BHK cells.

The NK cytotoxicities, against BHK-H cells, of immunized groups given a single 1-hr sham- or RFR exposure at 25 mW/sq cm were assayed 0, 2, 4, 6, 8, 10, and 12 hr after exposure. For reference, a group of immunized minimum-handling (normal) hamsters was assayed at 0 hr. The results were expressed graphically in Fig. 2 as percentage differences of mean cytotoxic activity between the RFR and control groups and between the sham and control groups. The means for the RFR and sham groups did not

differ significantly at 0 and 2 hr (the values for both groups at each epoch were a few percent above the control group). At 4 and 6 hr, however, the values for the RFR group were 15% and 14% below the control value while the corresponding values for the sham group were 4% below and 2% above control; the difference between the means for the RFR and sham groups was significant ($p<0.01$).

By contrast, the values for the RFR and sham groups at 8 hr were both 2% above control; at 10 hr, the value for the RFR group was 3% above control and no value was given for the sham group; and at 12 hr, the values for the RFR and sham groups were 5% above and 3% below control, respectively. Although the difference between the 12-hr values was called nonsignificant by the authors, use of the t-test yielded significance at the $p<0.01$ level, i.e., the NK activity of the RFR group was higher than of the sham group at that epoch.

The core-temperature measurements were displayed graphically in Fig. 3. For the groups exposed at 25 mW/sq cm for 15, 30, 45, and 60 min, the mean core temperatures at exposure cessation were 39.8, 40.1, 40.1, and 39.7 deg C, respectively. (Note that the highest mean temperature was 40.1, not 40.5 deg C as stated.) For a group exposed at this power density for 60 min, the temperatures 60 and 120 min after cessation were both about 37.9 deg C. For those exposed at 15 mW/sq cm for 1 hr, the temperatures at 0, 60, and 120 min after cessation were 38.5, 37.8, and 37.5 deg C, respectively. The temperatures of the sham and minimum-handling controls were comparable to each other; they both rose from 38.3 deg to peaks of 38.7 and 38.9 deg at 15 min after treatment and diminished to 37.7 and 37.6 deg, respectively, at 60 min. In addition, the temperature of the minimum-handling group increased from 37.9 deg in the animal room to 38.3 deg during transport to the exposure room.

To test whether the observed suppressive effect on NK activity of the single exposure at 25 mW/sq cm was due to direct heat on NK cytotoxic function, splenic lymphocytes were incubated for 60 min at 37.0, 40.5, 41.5, 42.5, and 43.5 deg C, and their cytotoxicities against transformed hamster embryo fibroblasts (PARA-7), PARA-7 cells infected with herpes (PARA-7-H), BHK cells, and BHK-H cells were assayed. The PARA and BHK results were similar so only those for PARA were presented (Table 3). The authors stated that for the cells incubated at 40.5 deg C, which corresponded to the core-temperature elevation obtained during RFR exposure, there was no significant decrease of NK activity against either PARA-7 or PARA-7-H relative to the values for incubation at 37.0 deg C. This statement was correct for PARA-7; however, by t-test of the values for PARA-7-H, the mean for 40.5 deg was significantly lower ($p<0.05$) than for 37.0 deg. In any case, there were progressive, significant diminutions of activity with temperature above 40.5 deg for both PARA-7 and PARA-7-H.

Because Liburdy (1979) showed that marked lymphopenia and neutrophilia were induced in mice by exposure to thermogenic RFR or injection of corticosteroids, peripheral-leukocyte and glucocorticosteroid-level changes in hamsters were determined at various intervals after sham

exposure or exposure at 25 mW/sq cm for 1 hr. Lymphopenia and neutrophilia (Fig. 4) were evident by 1 hr after RFR exposure, with return to baseline levels by 8 hr postexposure. The glucocorticosteroid results (Fig. 5) were much more variable, but the levels for the RFR group were consistently and significantly higher than for the sham group until 10 hr after treatment. Thus, the results were generally consistent with those of Liburdy (1979).

To determine whether there was a correlation between RFR-induced lymphopenia and redistribution of NK cells from peripheral blood to bone marrow, hamsters were immunized with vaccinia virus and exposed 4 days later at 25 mW/sq cm for 1 hr. Lymphocytes from peripheral blood and bone marrow were assayed for cytotoxicity against BHK and BHK-H targets 1 and 8 hr after exposure, corresponding to times of maximum lymphopenia and recovery. The results (Table 4) showed that the activities of peripheral-blood NK cells against both BHK and BHK-H targets at 1 hr were significantly lower than for sham-exposed animals, with recovery at 8 hr. There were no significant changes in activities of bone-marrow NK cells against either target, so the decrease in peripheral-blood NK activity was not from net flow of NK cells to bone marrow.

Effector cells from hamsters were treated in vitro with prednisolone sodium succinate at 5 (near physiologic), 20, and 50 ng/ml for 1 and 4 hr followed by a 16-hr assay for NK activity against BHK-H targets. The results (Table 5) showed that 1-hr treatment at the three doses reduced NK cytotoxity by 9.4%, 12.6%, and 14.1%, respectively, relative to untreated controls, but that none of the changes was significant. By contrast, the 4-hr treatment yielded decreases of 27.6%, 45.1%, and 38.6%, all of which were significant. It was noted that 20 ng/ml, which yielded the 45% reduction of NK cytotoxicity, was comparable to the corticosteroid levels in hamsters 4 hr after RFR exposure, which also suppressed NK function. Similar results were also obtained when the same doses of prednisolone were added to the effector-target mixtures during, rather than before, the 16-hr assay.

From these results, the authors concluded that glucocorticosteroid production above physiologic levels induced by RFR exposure could have a direct effect on NK activity and could explain the depressed NK cytotoxicity observed 4-6 hr after exposure, but that the lack of suppression after 1 hr of steroid treatment in vitro suggests that the decrease in circulating NK cells 1 hr after RFR exposure is not due to direct action of the drug.

CRITIQUE: This investigation involved evaluation, in hamsters immunized and nonimmunized with vaccinia virus, of the effects of RFR on splenic natural-killer-cell cytotoxity against baby-hamster-kidney cells and transformed-hamster-embryo-fibroblasts infected and noninfected with herpes simplex virus. A noteworthy variety of protocols was used in this and in a companion study by this group (Rama Rao et al., 1983) of the effects of hamster exposure to RFR on the in-vitro cytotoxic, phagocytotic, and NK-helper activities of their peritoneal macrophages.

Some of the statements derived from statistical treatment of the data are open to question, such as those mentioned in the previous section. Moreover, even though animal stress may have been minimized by not subjecting each hamster to more than one core-temperature measurement, the procedure did not permit tracking the temperature changes of each hamster with time. Also, the use of minimum-handling groups as controls is rather unclear, e.g., was a distinct group used for each experimental group? If so, how large were the variances among minimum-handling groups? In this context, little discussion was devoted to the biological significance of the differences between minimum-handling and sham-exposed groups.

A key finding of the temperature comparisons between control and sham-exposed hamsters is that apparently the sham-exposed and therefore the RFR-exposed hamsters were subjected to some degree of non-RFR-related stress that probably affected the functions of their immune systems by amounts that were difficult to quantify. For this reason, the effects of RFR-exposure per se must be deduced from comparisons of results from RFR- and sham-exposed animals only, under the tacit assumption that their non-RFR stresses were comparable.

Nevertheless, the results do indicate that a single exposure at 25 mW/sq cm (SAR of 13.3 W/kg) for 1 hr increases the core temperature by about 3 deg C and produces a transient increase in serum glucocorticosteroid level and a transient suppression of the activity of NK cells, which comprise a non-T, non-B population component. These effects were accompanied by transient lymphopenia and neutrophilia, which were also observed by Liburdy (1979) in mice exposed to thermogenic RFR levels.

In contrast, Smialowicz et al. (1982b) exposed pregnant mice to 2.45-GHz RFR at 28 mW/sq cm (SAR of 16.5 W/kg) for 100 min daily from gestational day 6 to 18, harvested NK cells from pups 3 and 6 weeks old, and assayed them for in-vitro splenic NK-cell activity against YAC-1 lymphoma cells at effector/target ratios of 25, 50, and 100. No significant differences in activity were found between 3-week-old RFR- and sham-exposed pups or for 6-week-old pups in one experiment. However, in a second experiment with 6-week-old pups, the NK activity of the RFR-exposed pups was significantly lower than for the sham-exposed pups, at least at an effector/target ratio of 100. (The statistical significance at ratio 50 is open to question.)

Huang and Mold (1980) also reported that exposure of mice to 2.45-GHz RFR at 15 or 30 mW/sq cm for 30 min per day, 5 days/week, did not alter the cytotoxicity of NK cells against transplantable allogeneic leukemic cells. However, the results of Huang and Mold (1980) are open to question because of the possible influence of non-RFR factors.

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EFFECTS OF MICROWAVE EXPOSURE ON THE HAMSTER IMMUNE SYSTEM. II. PERITONEAL MACROPHAGE FUNCTION
Bioelectromagnetics, Vol. 4, No. 2, pp. 141-155 (1983)

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AUTHOR ABSTRACT: Acute exposure of hamsters to microwave energy (2.45 GHz; 25 mW/sq cm for 60 min) resulted in activation of peritoneal macrophages that were significantly more viricidal to vaccinia virus as compared to sham-exposed or normal (minimum-handling) controls. Macrophages from microwave-exposed hamsters became activated as early as 6 h after exposure and remained activated for up to 12 days. The activation of macrophages by microwave exposure paralleled the macrophage activation after vaccinia virus immunization. Activated macrophages from vaccinia-immunized hamsters did not differ in their viricidal activity when the hamsters were microwave- or sham-exposed. Exposure for 60 min at 15 mW/sq cm did not activate the macrophages while 40 mW/sq cm was harmful to some hamsters.

Average maximum core temperatures in the exposed (25 mW/sq cm) and sham groups were 40.5 deg C (+/- 0.35 SD) and 38.4 deg C (+/- 0.5 SD), respectively. In vitro heating of macrophages to 40.5 deg C was not as effective as in vivo microwave exposure in activating macrophages to the viricidal state. Macrophages from normal, sham-exposed, and microwave-exposed hamsters were not morphologically different, and they all phagocytosed India ink particles. Moreover, immune macrophage cytotoxicity for virus-infected or noninfected target cells was not suppressed in the microwave-irradiated group (25 mW/sq cm, 1 h) as compared to sham-exposed controls, indicating that peritoneal macrophages were not functionally suppressed or injured by microwave hyperthermia.

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Study Type: Immunology and Hematology; IN VIVO; HAMSTER
Effect Type: Activation of peritoneal macrophages against vaccinia virus by in vivo acute exposure to RFR and in vitro heating
Frequency: 2.45 GHz
Modulation: CW
Power Density: 15, 25, or 40 mW/sq cm
SAR: 8.0, 13.3, or 21.2 W/kg

EXPOSURE CONDITIONS: Groups of 6 hamsters, each hamster within a Mylar-lined Styrofoam cage, were concurrently exposed to far-field RFR for 1 hr at 15, 25, or 40 mW/sq cm in an anechoic chamber at 22.5 deg C ambient temperature and 55% relative humidity. Comparison groups were sham-exposed concurrently with the RFR groups in a nearby chamber of the same design. White noise at 72 dB was fed into both chambers to mask background noises.

OTHER INFORMATION: As described in Yang et al. (1983), cage locations within the exposure chamber were adjusted to obtain equal power densities. Calorimetric measurements of SARs for hamster carcasses with their long axes parallel to the E-vector yielded 0.53 W/kg per mW/sq cm or 7.95, 13.25, and 21.2 W/kg for 15, 25, and 40 mW/sq cm, respectively.

Measurements of core temperatures were made in hamsters at 15-min intervals during 1 hr of sham- or RFR exposures at 15 and 25 mW/sq cm (Fig. 3 in Yang et al., 1983). To minimize stress, the temperature of each hamster was taken only once. The results showed rises from about 38.3 deg C to maximum mean values of 38.5 and 40.1 deg C (not 40.5 as stated), respectively. The mean temperature of a group transported from the animal room to the exposure room but otherwise unhandled or treated, denoted as a "normal" or "minimum-handling" control group, was found to increase from 37.9 to 38.3 deg C, the preexposure value for the RFR and sham groups. The temperatures of the sham and minimum-handling controls were comparable to each other; they both rose from 38.3 deg to peaks of 38.7 and 38.9 deg at 15 min after treatment and diminished to 37.7 and 37.6 deg, respectively, at 60 min. Thus, the mean temperature of the minimum-handling group increased by up to 1 deg C, an indication of non-RFR-induced stress.

Vaccinia virus was used to immunize some groups before RFR- or sham exposure and for macrophage viricidal studies. The virus was grown in Vero cells (a line of African green monkey kidney cells), released by disruption of the Vero cells by freeze-thawing, and assayed on monolayers of other Vero cells in terms of plaque-forming units (PFU).

Following hamster exposure, resident peritoneal macrophages (PM) were collected in Eagle's minimum essential medium (MEM) supplemented with newborn calf serum, antibiotics, L-glutamine, HEPES, and sodium bicarbonate (complete MEM) and counted. For vaccinia-growth studies, PM were inoculated and incubated with the virus prepared as described above, the PM were disrupted 48 hr after incubation by freeze-thawing, and the viral content was assayed by plaque formation. For each PM specimen, a duplicate specimen was freeze-thawed and assayed before incubation.

For phagocytosis assays, PM suspended in MEM were incubated in glass vials for 1 hr at 37 deg C, nonadherent cells were washed away, and the adherent cells were incubated for 24 or 48 hr in fresh MEM. The cells were then incubated for 2 hr with India ink, and phagocytosis was determined by counting the number of PM containing intracellular particles.

For assays of macrophage cytotoxicity, effector populations of PM were inserted in wells of microtest plates and nonadherent cells were washed away with MEM. Target cells consisted of either uninfected baby-hamster kidney (BHK) cells or BHK cells infected with herpes simplex virus (BHK-H cells), each type suspended in MEM containing the tracer Cr-51. After overnight incubation at 37 deg C, target cells were removed,

washed, and added to the test wells in quantities to yield an effector-to-target ratio of 50. After further incubation of the samples and centrifugation, the supernatant was assayed for Cr-51 release by gamma counting. Spontaneous Cr-51 release was determined in samples of uninfected BHK cells, and maximum Cr-51 release was promoted by adding detergent to the MEM and determined.

Macrophage cytotoxity was expressed as "percent specific Cr-51 release," defined as the percentage of net counts in test wells (corrected by subtraction of counts in spontaneous-release wells) to net counts in maximum release wells (similarly corrected). Each assay was performed in triplicate, and the 2-tailed Student t-test was used for statistical comparisons.

PM were incubated in humidified CO2 for 2 hr at 37 deg C and vigorously washed to form a strongly adherent monolayer. Nonadherent bone-marrow (BM) cells, prepared from femur cells dispersed in complete MEM and incubated for 1 hr in a flask to remove adherent cells, were added to the macrophage monolayers and incubated at 37 deg C for 20-24 hr. The residual nonadherent BM cells are denoted as BM-NK cells because they have been shown to possess natural-killer characteristics (Chapes et al., 1981). The cytotoxicities of BM-NK cells for BHK and BHK-H targets were assayed in terms of percent specific Cr-51 release.

Groups of hamsters were sham-exposed or exposed for 60 min at 25 mW/sq cm (SAR of 13.3 W/kg). Some groups were inoculated with vaccinia 4 days before exposure; other groups were not immunized. PM were collected from minimum-handling hamsters and from RFR and sham groups at various times from 0 to 12 hr postexposure, infected with vaccinia, and assayed for in-vitro viricidal activity as described above. The results (Fig. 1), expressed as the log of the change in input virus (PFU) vs postexposure hr, showed that PM from nonimmunized sham-exposed hamsters was partially viricidal as compared with PM from nonimmunized minimum-handling hamsters. Both groups yielded approximately constant titers for all postexposure times tested (a log of about +0.6 for the minimum-handling group and +0.2 for the sham group; a statistically significant difference). By contrast, the values for the nonimmunized RFR group diminished from +0.4 to +0.2 during the first 4 hr, dropped to -0.6 at 6 hr, and to about -0.8 during the remaining 6 hr. Moreover, the values for the immunized minimum-handling, sham, and RFR groups were all between -0.9 and -1.0 throughout the 12-hr test period. Thus, the viricidal activity, against vaccinia, of PM from nonimmunized hamsters exposed at 25 mW/sq cm is quantitatively similar to that of PM from vaccinia-immunized hamsters.

A possible alternative hypothesis for the decreases in virus titers for nonimmunized hamsters exposed at 25 mW/sq cm is that viral growth in the PM was inhibited by RFR-heat injury to the PM that lasted the entire 12-day interval. However, this hypothesis is not tenable because, as discussed later, the phagocytotic ability of PM for India ink particles was not diminished by exposure of hamsters at this power density.

The experiment was repeated, but with the viricidal activities of PM tested for 18 days postexposure. The results (Fig. 2) for the nonimmunized minimum-handling and sham groups for the entire test period were approximately the same constant values as before. Also, the nonimmunized RFR group yielded a sharp drop to -0.8 on day 1 and -0.9 on day 3, lesser viricidal activity for up to 12 days, and recovery to normal by day 15. The authors stated that the PM from the immunized groups showed viricidal activity by 4 days postimmunization (0 days postexposure), which would be consistent with the results of the previous experiment. However, Fig. 2 appears to indicate that maximum viricidal activity occurred at 4 days postexposure (i.e., 8 days postimmunization).

Results of similar experiments at 15 mW/sq cm (SAR of 8.0 W/kg), presented in Table 1, indicated that for the 12-hr postexposure test period, the viricidal activity of PM from nonimmunized RFR-exposed hamsters did not differ significantly from that of PM from nonimmunized sham-exposed hamsters. However, the activities of both groups were significantly lower than for the minimum-handling group, an indication of partial viricidal activity for the sham and RFR groups.

Exposures of hamsters at 40 mW/sq cm (SAR of 21.2 W/kg) for 1 hr (sublethal) yielded PM that exhibited uncharacteristic rounded morphology, did not adhere well to coverslips, and did not actively phagocytose India ink particles. Their viricidal activity (Table 1) was intermediate between the values for shams and hamsters exposed at 25 mW/sq cm.

Since the mean colonic temperature of hamsters after 1-hr exposure at 25 mW/sq cm was 40.5 deg C, an experiment was performed to ascertain whether PM from minimum-handling hamsters could be activated by in-vitro hyperthermia at this temperature. PM were collected and divided into two groups. The groups were incubated for 1 hr, one at 37 and the other at 40.5 deg C. Each group was then divided into two groups, one of which was immediately infected with vaccinia while the other was incubated for 6 hr and then infected. The results (Table 2) showed that the virus multiplied in the PM from all four groups and that the 6-hr incubation had no significant effect. However, the levels of growth for the hyperthermic groups were significantly lower than for the groups treated at 37 deg, but were comparable to those for the nonimmunized sham-exposed hamsters.

PM from minimum-handling hamsters, sham-exposed hamsters, and hamsters exposed at 15, 25, or 40 mW/sq cm were collected at various times after exposure, cultured in vitro for 24 and 48 hr, and tested for phagocytosis of India ink particles. The PM from all groups except those exposed at 40 mW/sq cm phagocytosed the particles well; Table 3 shows that most of these groups were able to phagocytose between 76% and 100% of the particles at all times tested (up to 8 days postexposure). However, phagocytosis was less than 25% for the 40 mW/sq-cm groups.

PM from hamsters immunized against vaccinia 4 days prior to sham exposure or exposure at 25 mW/sq cm for 1 hr were assayed on the day after exposure for cytotoxicity against BHK and BHK-H cells. The results (Table 4) showed no statistically significant cytotoxic differences between sham and RFR groups for either BHK or BHK-H cells; however, the cytotoxicities of both groups for both types of cells were significantly lower than for minimum-handling hamsters.

PM-NK cells were prepared as described above from minimum-handling, sham-exposed, and RFR-exposed (25 mW/sq cm) hamsters, and assayed for cytoxicity against BHK and BHK-H cells. Table 5 shows that the percentage cytotoxicities against BHK-H cells of the sham and RFR groups were comparable to one another and that both were significantly higher than for the minimum-handling hamsters. Against the BHK cells, however, the mean cytotoxicity of the RFR group was higher than for the sham and miminum-handling groups; the differences were both labeled as significant ($p < 0.01$), but use of the t-test shows that only the difference between the RFR and the sham groups was significant. Table 5 also indicates that the mean percentage cytotoxicities of all six groups were significantly higher than for PM or BM cells separately. Results of repeating these experiments, displayed in Fig. 3, were mostly similar.

Based on the results with BHK and BHK-H cells, the authors concluded that ..."a microwave exposure at 25 mW/sq cm for 1 h does not impair immune macrophage cytotoxic activity, nor does it inhibit macrophage NK helper function. On the contrary, microwave exposure appears to significantly enhance the ability of macrophages to induce NK activity from BM cells."

CRITIQUE: This investigation of the effects of RFR exposure of hamsters on the in-vitro cytotoxic, phagocytotic, and NK-helper activities of their peritoneal macrophages complements a companion study by this group (Yang et al., 1983) on the effects of RFR on splenic NK-cell cytotoxity. A noteworthy variety of protocols was used in both studies.

A few of the statements derived from statistical treatment of the data are open to question, such as those mentioned in the previous section, but the overall conclusions of the study were not affected thereby.

The comparisons of minimum-handling groups with sham-exposed groups by Yang et al. (1983) indicated that the sham-exposed and RFR-exposed hamsters were subjected to some degree of non-RFR-related stress that probably affected the functions of their immune systems, but to extents that were difficult to quantify. For this reason, the effects of RFR-exposure per se must be deduced from comparisons of results from RFR- and sham-exposed animals only, under the tacit assumption that their non-RFR stresses were comparable. On this basis, the results on PM viricidal activity, against vaccinia, of nonimmunized hamsters exposed at 25 mW/sq cm and the lack of effect at 15 mW/sq cm appear unequivocal.

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Chou, C.-K., A.W. Guy, L.E. Borneman, L.L. Kunz, and P. Kramar
CHRONIC EXPOSURE OF RABBITS TO 0.5 AND 5 mW/SQ-CM 2450-MHZ CW MICROWAVE RADIATION
Bioelectromagnetics, Vol. 4, No. 1, pp. 63-77 (1983)

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AUTHOR ABSTRACT: Two groups of 16 male New Zealand rabbits were exposed to 2450-MHz continuous wave microwave fields in two experiments of 90 days each. The incident power densities of the first and second experiment were 0.5 and 5 mW/sq cm, respectively. During each study, 16 animals were adapted to a miniature anechoic chamber exposure system for at least 2 weeks, then 8 of them were exposed for 7 h daily, 5 days a week for 13 weeks, and the other 8 animals were sham exposed. The rabbits were placed in acrylic cages, and each was exposed from the top in an individual miniature anechoic chamber. Thermography showed a maximum specific absorption rate of 5.5 W/kg in the head and 7 W/kg in the back at 5-mW/sq-cm incident power density. After each 7-h exposure session, the animals were returned to their home cages.

Food consumption in the exposure chamber and body mass were measured daily. Blood samples were taken before exposure and monthly thereafter for hematological, morphological, chemical, protein electrophoresis, and lymphocyte blast transformation studies. Eyes were examined for cataract formation. Finally, pathological examinations of 28 specimens of organs and tissues of each rabbit were performed. Statistically, there was a significant ($P<.01$) decrease only of food consumption during the 5-mW/sq-cm exposure; other variables were not significantly different between exposed and control groups.

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Study Type: Physiology and Biochemistry, Immunology and Hematology, Ocular Effects; IN VIVO; RABBIT
Effect Type: Effects of chronic RFR exposure on food consumption, body mass, immunologic and hematologic parameters, cataractogenesis, and histopathology of rabbits
Frequency: 2.45 GHz
Modulation: CW
Power Density: 0.5 or 5 mW/sq cm
SAR: 5.5 and 7 W/kg in the head and back, respectively, at 5 mW/sq cm

EXPOSURE CONDITIONS: Sixteen miniature anechoic chambers (Guy, 1979) were used to concurrently sham-expose 8 rabbits and expose 8 rabbits to far-field RFR at 0.5 or 5 mW/sq cm propagated vertically from above for 7 hr daily, 5 days/week, for 13 weeks. The chambers were in a room maintained at 21 deg C and 50% relative humidity. Each rabbit was confined in an acrylic cage that ensured exposure with its long axis parallel to the electric field most of the time. Each rabbit was provided with water and 200 g of dry food pellets during each 7-hr exposure period.

OTHER INFORMATION: The systems for food and water delivery and waste removal were designed to ensure minimal RFR absorption and field perturbation. SARs were measured in the sagittal plane of a rabbit carcass by thermography. Most of the energy was coupled to the back and head regions.

On receipt, rabbits were quarantined for 1 week, weighed, and determined to be disease-free and to possess clear lenses. The 16 rabbits used in the first study, which involved exposure at 0.5 mW/sq cm, were adapted to the exposure system by transferring them daily, 5 days a week, from their home cages to the exposure chambers before 9 am and returning them after 4 pm, for 2 weeks, after which they were divided into 2 groups matched in body mass and rate of weight gain. One group was then exposed to the RFR for 7 hr daily, 5 days a week, for 13 weeks and the other group was concurrently sham-exposed. The 16 rabbits used in the second study, at 5 mW/sq cm, were adapted for 4 weeks, similarly divided, and exposed.

The body mass of each rabbit was measured before daily transfer to the exposure chamber. The unconsumed portion of the 200 g of food pellets supplied during each 7-hr exposure was weighed and the amount of food consumed was calculated. Food was available ad libitum after return to the home cage, and consumption was not measured.

Blood was taken for hematology, chemistry, protein electrophoresis, and lymphocyte studies from the medial auricular artery before initial exposure and monthly thereafter. Monthly samples (15 ml) were obtained from 8 rabbits (4 RFR and 4 sham) each day on 2 successive days at 8 and 9 am to minimize differences caused by circadian variations. The first group of 16 rabbits had an average body mass of about 2 kg on receipt, and some difficulty was encountered in obtaining the first blood sample because of the smallness of the artery (but not for subsequent samples). Therefore, study of the second group was started at an average body mass of about 2.5 kg.

Samples were assayed for white-blood-cell (WBC) and red-blood-cell (RBC) counts, hemoglobin (Hg), and hematocrit (HCT). The mean-corpuscular-volume (MCV), hemoglobin (MCH), and hemoglobin concentration (MCHC) were calculated from the WBC, RBC, Hg, and HCT. Differential counts of segmented neutrophils, basophils, lymphocytes, monocytes, and eosinophils were done by light microscopy, which was also used for morphologic examination of WBC, RBC, and plasma.

Analyses included measurements of sodium, potassium, chloride, calcium, magnesium, phosphorus, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), lactate dehydrogenase (LDH), creatine phosphokinase (CPK), glucose, blood urea nitrogen (BUN), creatinine, cholesterol, alkaline phosphatase, and triglycerides. The concentrations of albumin and the globulins alpha-1, alpha-2, beta, and gamma were measured, and the electrophoretic serum protein fractions were determined by densitometry.

Leukocytes were separated from blood samples and suspended in minimum essential medium. The suspensions were stimulated with the mitogen phytohemagglutinin (PHA-P) and incubated. Further mitosis was blocked prior to cell harvest. Cells of each culture were then harvested, fixed, transferred to a glass slide, and stained. The percentage of blastogenesis (the percentage of blastoid-cell counts to total lymphocytic-cell counts) and the mitotic index (percentage of metaphase-cell counts to total lymphocytic counts) were determined by light microscopy. To determine the PHA-P stimulation index for each animal, tritiated thymidine was added to stimulated and unstimulated cultures, and its uptake (as a measure of DNA synthesis) was determined by liquid scintillation counting. The stimulation index was defined as the ratio of counts/min of stimulated cultures to the counts/min of unstimulated cultures, both corrected for background counts.

Both eyes of each rabbit were examined for cataracts with a slit lamp before and after the 13-week exposure period. At the end of the period, the animals were exsanguinated, necropsied, and evaluated for pathology.

Because the blood tests were large in number and interdependent, a multivariate analysis (MANOVA) on two groups of data in parallel profile was used. This type of analysis determines the equality of the graphical profiles of the two groups (RFR- and sham-exposed) as measured at different times. The two major hypotheses tested (at the assumed level of significance) are H-1: that the profiles are parallel; and H-2: that if parallel, they are at the same level, i.e., the treatment (RFR) had no significant effect. If the profiles are parallel but not at the same level, then the treatment produced a significant effect. However, if the analysis indicates rejection of H-1, then the analysis will not differentiate a treatment effect from a time effect, test of H-2 is not valid, and individual t-tests are necessary to ascertain any significant effect of treatment.

The daily body-mass and food-consumption measurements were made for the two weeks prior to exposure as well as during the 13-week exposure period. The results for each were averaged weekly. The weekly mean weights of the group exposed at 0.5 mW/sq cm and its concurrent sham group rose together linearly with time and leveled off in the later weeks of the exposure period with the sham group slightly heavier than the RFR group. The results for the 5-mW/sq-cm and sham groups were similar except that the RFR group was slightly heavier than the sham group. Profile analysis over the study period and for the last 5 weeks thereof indicated that the differences were nonsignificant ($p>0.05$).

In the 0.5-mW/sq-cm study, the graphs of weekly mean food consumption during exposure for the 2 groups were statistically parallel and equal, with a trend upward. There were disruptive increases in consumption after the 13th week of the study period, coinciding with a change of animal-handling technician. In the 5-mW/sq-cm study, there was a downward trend in food consumption by both groups. The consumption by the RFR group was slightly higher than for the sham group before and during the first week of exposure, but the converse was true for the

subsequent weeks. Moreover, the decrease for the RFR group was much faster than for the sham group, reaching a minimum on the 6th week of the study period and rising to the same level as the sham group by the 13th week. The differences between the 2 groups were significant at the $p<0.01$ level. Consumption by both groups rose sharply after the 14th week, again coinciding with a change of technician.

No blood-analyses data were presented. Instead, the authors stated the following results:

Profile analysis of the hematologic and blood-chemistry data showed no significant differences at the $p=0.05$ level between the RFR and sham groups at either power density. Glucose data from the 0.5-mW/sq-cm study failed the parallelism test. Therefore, separate t-tests were performed, which yielded no significant differences between groups.

The total protein of the RFR and sham groups both decreased in the 0.5-mW/sq-cm study but increased in the other study. However, no significant difference between groups was found. Parallelism of the beta-globulin data from the 0.5-mW/sq-cm study was rejected, but the differences were not significant by t-test.

No significant differences in lymphoblast-transformation data were found. This was also true for the mitotic and stimulation indexes from the 5-mW/sq-cm study. (Presumably these indexes were not determined for the other study.)

No cataracts developed in any of the rabbits during the 3-month period. The necropsies revealed various minor lesions (described in the paper) caused by bacterial infections, pinworms, and encephalitozoonosis in both RFR and sham animals, some of which were characterized as common occurrences in conventional colony rabbits. However, no RFR-induced pathological effects were observed.

CRITIQUE: The statistical methods used to analyze the data were appropriate and presumably thorough. However, presentation of the numerical results of the blood-test analyses would have provided quantitative support for the negative findings therefrom.

As indicated by the authors, the only RFR-related effect was the significantly lower food consumption by the rabbits exposed at 5 mW/sq cm (as compared with the corresponding sham-exposed group), a finding consistent with the results of Ferri and Hagan (1976), who obtained a 14% reduction in intake by rabbits exposed for 9 weeks to 2.45-GHz CW RFR at 10 mW/sq cm for 8 hr/day, 5 days/week, and of Moe et al. (1976) in rats exposed to 918-MHz RFR at 10 mW/sq cm for 10 hr/night, 7 nights/week, for 3 weeks.

Chou et al. suggested that the lower food consumption may be related to metabolism. From calculations based on SAR, they estimated that exposure at 5 mW/sq cm corresponds to about 16% of the resting metabolic rate of the rabbit, and they suggested that the absorbed energy may have

been "utilized" for part of the animal's metabolism in lieu of food. It would have been interesting to measure water consumption during exposure as well as food intake.

Because of the perturbations in food consumption coincident with changes of animal-handling technicians, the authors emphasized the necessity for consistent handling and treatment of both RFR- and sham-exposed animals, especially in studies involving chronic exposures.

The absence of ocular pathology is consistent with the findings of Ferri and Hagan (1976) and Guy et al. (1975, 1980b) in rabbits, and of Djordjevich and Kolak (1973) in rats. Some of the negative findings on the blood tests are contrary to those of other investigators, such as Shandala et al. (1979) in rats, as stated. However, it is interesting to note that in two sequential analyses of the same rabbits exposed to 2.45-GHz at 10 mW/sq cm for 23 hr/day for 6 months, Guy et al. (1980b) reported no significant effects on body mass, Hg, HCT, WBC, and other endpoints, and McRee et al. (1980) reported a significant decrease in albumin-to-total-globulin ratio, an increase in myeloid-to-erythroid ratio, and suppression in lymphocyte responsiveness to pokeweed mitogen (PWM) [but not to PHA or Concanavalin A (Con A)]. On the other hand, Smialowicz et al. obtained negative results with PHA, Con A, PWM, and lipopolysaccaride (LPS) for rats exposed to 2.45 GHz (1979a), 100 MHz (1981b), or 970 MHz (1981d). Large variabilities for such endpoints among animals of the same species, as well as differences in methodology and species, may account for such differences in findings.

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Bioelectromagnetics, Vol. 3, No. 1, pp. 117-125 (1982)

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AUTHOR ABSTRACT: Three male and three female adults individually placed the ventral surface of the right and upright forearm against a 15-cm-diameter aperture in a wall of microwave-absorbent material. Ten-second exposures occurred to E-vector-vertically polarized, 2450-MHz-CW microwave (MW) fields. Comparable exposure to infrared (IR) waves was repeated with four of the six observers. Thresholds of detection of just-noticeable warming by MW and IR radiation were determined by the double-staircase psychophysical method. Although the exposed surface areas of male observers' arm were larger than those of female observers, thresholds of warming by either source of energy overlapped; the pooled means of irradiance at threshold are 26.7 mW/sq cm (MW) and 1.7 mW/sq cm (IR).

Dosimetric measures on saline models indicated virtually perfect absorption of the incident IR, but nearly two-thirds of the MW energy was scattered. Accordingly, the 15-fold difference in means of MW and IR thresholds resolves to a 5-fold difference in the threshold quantities of absorbed energy. In the light of the high correlation between thresholds of IR and MW irradiation (r=.97), it is concluded that the same set of superficial thermoreceptors was being stimulated, only less efficiently so, by the more deeply penetrating, more diffusely absorbed MW energy.

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Study Type: Human Studies, Behavior, Nervous System; IN VIVO; HUMAN
Effect Type: Thresholds for detection of RFR and infrared radiation (IR) by superficially located thermoreceptors
Frequency: 2.45-GHz RFR; not stated for IR
Modulation: CW
Power Density: 0-70 mW/sq cm RFR; 0-5.5 mW/sq cm IR
SAR: 0.076 W/kg per mW/sq cm RFR; 0.59 W/kg per mW/sq cm IR

EXPOSURE CONDITIONS: The ventral surface of the forearm of each human subject was exposed to vertically polarized far-field RFR from a standard gain horn within an anechoic chamber for 10 seconds at aperiodic intervals averaging about 30 seconds. For exposure, each subject was behind an RFR-absorbing partition within the chamber, with forearm vertically placed against a 15-cm-diameter aperture in the partition. Exposures to infrared radiation (IR) from focused quartz lamps through a 15-cm-diameter aperture in a Styrofoam partition were similarly performed. The ambient temperature and relative humidity of the chamber were maintained at 25 +/- 2.0 deg C and 50 +/- 5%, respectively.

OTHER INFORMATION: Calibrations of RFR and IR power densities were done at the center of the aperture (in the absence of the subject) with a Narda Model 8316B E-field probe and a Hardy radiometer, respectively.

IR SARs were determined for a cylindrical red-latex-balloon model filled with 0.9% NaCl in distilled water and secured against the aperture. The length and diameter of the model were 15.5 and 2.25 cm, and the mass was 62 g. A Vitek temperature probe suspended at the geometric center of the model was used to measure the temperature rise. IR exposures for 10 min at 10.71 mW/sq cm yielded a temperature rise of 0.9 deg C, which corresponded to an SAR of about 6.3 W/kg or 0.59 W/kg per mW/sq cm.

RFR SARs were similarly determined, but with a saline-filled cylindrical model of length 15.9 cm, diameter 5.8 cm, and mass 420 g, which corresponded more closely to the mass and profile of the part of the human forearm exposed through the aperture. The model was exposed at 70 mW/sq cm until a rise of exactly 0.5 deg C was attained, which required 394 seconds. From these values, the calculated SAR was 5.31 W/kg or 0.076 W/kg per mW/sq cm (not 0.74 microwatt/g per mW/sq cm as stated in the text). The nominal power incident on the model, obtained by multiplying 70 mW/sq cm by the profile area exposed through the 15-cm aperture (about 88 sq cm), was 6.16 W. The rate of energy absorption in the model was 2.23 W or only about 36% of the incident power, i.e., about 64% of the incident power was scattered.

The 0-70 mW/sq-cm range of RFR power densities was divided into 5-mW/sq-cm steps and the 0-5.5 mW/sq-cm IR range into 0.5 mW/sq-cm steps. On-off switching of the RFR was done at the source. However, because of the long rise time of the IR, a human stationed near the quartz lamps operated a shutter.

Three men and three women were the subjects for the RFR experiment and two of each gender participated in the IR experiment. The profile of each subject's forearm within a 15-cm aperture was drawn, and the area within the profile was measured with a planimeter. Illumination of a bulb signaled the subject that a trial was to begin and to place the arm in the appropriate position. The 10-second exposure was done within 5-15 seconds of the signal, after which the experimenter elicited, via an intercom system, a yes or no from the subject regarding perception of the stimulus.

Thresholds for perception of RFR and IR were ascertained by the random double-staircase method (Cornsweet, 1962), in which the stimulus level presented during a given trial was determined as follows: If the subject reported perception of the stimulus during a trial, the stimulus was lowered by a randomly determined multiple of steps ("stairs") for the next trial; if the subject responded negatively, the stimulus was raised similarly. The randomness of the size of the multiples precluded discovery by the subject of any pattern of successive stimuli. This procedure was continued until 13 transitions (reversals of intensity direction) occurred, and the threshold was defined as the mean of stimulus intensities over the final 10 transitions.

The RFR and IR threshold data for each of the subjects and the profile areas of their forearms were presented in Table 1. From these data, the mean profile area and threshold power density were derived separately for the men and women, together with the respective standard deviations (SDs) and standard errors (SEs). The mean profile areas of the men and women were 122.67 (SD 8.14) and 91.33 (SD 3.06) sq cm, a significant difference. The mean threshold for RFR perception by the men and women were 25.27 (SD 10.92) and 28.22 (SD 13.91) mW/sq cm, respectively. The difference was nonsignificant ($p>0.05$). Also, the correlation coefficient, r, between RFR threshold power density and profile area was only 0.26. The mean threshold values for IR perception by the men and women were 1.48 (SD 0.04) and 2.00 (SD 0.78) mW/sq cm, respectively, again a nonsignificant difference, and r was 0.62. However, the RFR and IR thresholds for all 6 subjects (without regard to gender) were highly correlated ($r=0.97$, $p<0.02$).

The range of RFR thresholds over all 6 subjects was 15.40-44.25 mW/sq cm, with a grand mean of 26.74 (SD 11.30) mW/sq cm. The deviance, defined as the ratio of range to mean and used as an index of instability, was 108%. The IR thresholds for the 4 subjects ranged from 1.45 to 2.55 mW/sq cm, with a grand mean of 1.74 (SD 0.54) mW/sq cm and a deviance of 63%. Thus, the mean RFR power-density threshold was about 15 times higher than the mean IR power-density threshold. None of the 4 subjects exposed to both RFR and IR reported any difference in sensory quality between the two stimuli.

The RFR and IR threshold power densities of each subject and the dosimetric data from the saline models were used to calculate the threshold RFR and IR energies absorbed in the forearm of each subject during a 10-second exposure. From Table 2, the RFR threshold energy ranged from 6.43 to 15.58 J, with a mean of 10.16 (SD 4.02) J and a deviance of 90%, and the IR threshold range was 1.38-2.24 J, with a mean of 1.83 (SD 0.37) J and a deviance of 47%. Thus, the amount of RFR energy for threshold stimulation was about fivefold higher than for IR. Also, the threshold-energy deviance for each stimulus was smaller than its threshold-power-density deviance. Therefore, threshold energy absorption is a more stable predictor of just-noticeable warming by either RFR or IR than threshold power density.

Because of the high correlation between the RFR and IR thresholds, the warmth sensed is believed to be due to stimulation of the same superficially located thermoreceptors of the skin. The fifteenfold power-density difference and the fivefold absorbed-energy difference between RFR and IR thresholds for stimulation were ascribed in part to the large scatter (about 64%) of the incident RFR (vs virtually no IR scatter) and in part to the much larger penetration depth of the RFR.

The authors, citing Hendler and coworkers (Hendler and Hardy, 1960; Hendler et al., 1963), noted that exposure of 37 sq cm of the human forehead to a 3000-GHz (sic) field for 4 seconds required a mean threshold of 33.5 mW/sq cm for sensing warmth. Extrapolation to 10-second exposures yielded a threshold of about 27 mW/sq cm, which was

very close to the mean value, 26.7 mW/sq cm, found in this study. Also, the mean IR threshold (1.7 mW/sq cm obtained in the latter study) was comparable to the human-forehead IR threshold reported by Hendler and coworkers.

CRITIQUE: The adequacy of the SAR determinations is open to question because no mention was made of any measures taken to minimize heat loss from the saline models during exposure or of whether the saline was equilibrated (by stirring or other method) on completion of exposure before reading its temperature with the Vitek probe. If such precautions were not taken, then little credence can be given to the numerical values of energy-absorption thresholds in Table 2. On the other hand, even if the values are correct, their utility is unclear. Specifically, there is no evidence that the same values would be obtained if the subjects were exposed, for example, at half the threshold power density for twice as long. Thus, despite the smaller statistical deviances obtained for the energy-absorption thresholds, the power-density thresholds are biologically more significant.

The use of multiples of a fixed step (5 mW/sq cm for the RFR and 0.5 mW/sq cm for the IR) in the double-staircase paradigm precluded localizing the thresholds of any subject for the two stimuli to better than within one step in each case. Moreover, since presumably each subject was exposed to each paradigm only once, no data were obtained to assess the consistency or repeatability of the thresholds for each subject. Thus, the credence given to averaging the thresholds, for the women and men separately or as a group without regard to gender, is diminished, particularly regarding presentation of the results to three and four significant figures (two decimals).

The microwave source used by Hendler and coworkers furnished 0.4-microsecond pulses of 3-cm (10-GHz) RFR at 2500 pulses per second. The penetration depth in skin at this RFR frequency is only about 0.3 cm as compared with 1.7 cm at 2.45 GHz. Therefore, comparison of the RFR power-density threshold obtained in this study with that of Hendler et al. (1963) is inappropriate, and the close agreement reported is purely fortuitous.

An important sensory phenomenon is the almost immediate sensation of heat (or cold) that is experienced on the abrupt exposure to a source warmer (or colder) than the ambient or skin temperature, e.g., when moving from shadow to sunlight (or opening a refrigerator). Although power-density thresholds ascertained by the use of CW RFR (such as in this study) undoubtedly included the influence of this phenomenon, determination of thresholds by the use of RFR having pulse durations that span the time constants of this phenomenon may provide more appropriate and sensitive information in some kinds of RFR-behavioral studies, e.g., of avoidance or escape from noxious stimuli.

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Carroll, D.R., D.M. Levinson, D.R. Justesen, and R.L. Clarke
FAILURE OF RATS TO ESCAPE FROM A POTENTIALLY LETHAL MICROWAVE FIELD
Bioelectromagnetics, Vol. 1, No. 2, pp. 101-115 (1980)

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AUTHOR ABSTRACT: Ocularly pigmented rats, all mature females of the Long-Evans strain, were repeatedly presented an opportunity to escape from an intense 918-MHz field (whole-body dose rate = 60 mW/g) to a field of lower intensity (40, 30, 20, or 2 mW/g) by performing a simple locomotor response. Other rats could escape 800-microampere faradic shock to the feet and tail by performing the same response in the same milieu, a multimode cavity.

None of the 20 irradiated rats learned to associate entry into a visually well-demarcated area of the cavity with immediate reduction of dose rate, in spite of field-induced elevations in body temperature to levels that exceeded 41 deg C and would have been lethal but for a limit on durations of irradiation. In contrast, all of ten rats motivated by faradic shock rapidly learned to escape.

The failure of escape learning by irradiated animals probably arose from deficiencies of motivation and, especially, sensory feedback. Whole-body hyperthermia induced by a multipath field may lack the painful or directional sensory properties that optimally promote the motive to escape. Moreover, a decline of body temperature after an escape-response-contingent reduction of field strength will be relatively slow because of the large thermal time constants of mammalian tissues. Without timely sensory feedback, which is an essential element of negative reinforcement, stimulus-response associability would be impaired, which could retard or preclude learning of an escape response.

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Study Type: Behavior; IN VIVO; RAT
Effect Type: Comparison of escape responses from RFR at a potentially lethal level to lower levels and faradic shock
Frequency: 918 MHz
Modulation: 3-Hz amplitude modulation
Power Density: Not indicated
SAR: 50-120 W/kg for lethality; 60 and 40, 30, 20, or 2 W/kg for behavior

EXPOSURE CONDITIONS: RFR- and sham exposures were performed in a multimode cavity having a 3-element mode stirrer rotating once per second, thus amplitude-modulating the RFR at 3 Hz. The peak-to-average power ratio was about 5. The mean temperature and relative humidity of the laboratory were 21.1 deg C and 53%. Air flow through the cavity was at 0.1 m/second.

For the studies on escape from RFR, the cavity was equipped with a false floor of white, opaque Plexiglas on which the boundary of a rectangular "safer" region about 25% of the total floor area was marked with black tape. Movement from the unsafe to the safer area was the simple escape response required. It should be noted that the safer area was not a region of intrinsically lower intensity relative to the unsafe area, but was rendered safer by the experimenters who reduced the input RFR power to the cavity (including the safer area) by a predetermined percentage each time a rat entered that area.

Recrossing by a rat to the unsafe area triggered restoration of the initial RFR power to the cavity. A crossing in either direction was defined as when half the body traversed the boundary.

A false floor with a similarly marked boundary and with 22 parallel, equidistant 5-mm stripes of conductive silver painted on the "unsafe" area was used for the faradic-shock studies; alternate stripes were connected together to form 2 poles, to which the shock source was connected.

OTHER INFORMATION: Whole-body SARs were determined calorimetrically with saline solution in foamed polystyrene vessels exposed at the center of the unsafe area, and by measuring colonic temperature increases in similarly exposed live rats previously rendered hypothermic by i.p. injection of cortisone or pentobarbital.

In a pilot study on dose lethality, rats were exposed for a succession of five two-min periods timed two min apart, one rat at 50 W/kg, three at 60 W/kg, one at 75 W/kg, and one at 120 W/kg. The latter two rats expired. The four rats exposed at 60 W/kg or lower exhibited symptoms of severe hyperthermia but survived because of the cooling during the interexposure periods. For the three rats exposed at 60 W/kg, the mean colonic temperature increase at the end of the five exposures was 3.5 deg C. Based on this pilot study, 60 W/kg was adopted as the maximum SAR, and the exposure regimen used in the formal studies consisted of alternating 2-min exposures and respites, with a 2-min interval preceding and following the exposures for a total session time of 22 min per day for 6 days.

The first formal experiment was done in 3 consecutive phases of 6 days each at 40-hr intervals. In the first phase (involving 10 rats), each crossing of a rat to the safer area during each 2-min exposure resulted in a SAR reduction from 60 to 40 W/kg, with restoration of 60 W/kg for each reverse crossing and at the start of each period. In the second phase, the reduction was to 30 W/kg for 5 of the rats and to 20 W/kg for the other 5 rats. For the third phase (all 10 rats), the reduction was to 2 W/kg.

For each rat, the numbers of entries into the safer area during the periods of exposure and nonexposure and the times spent there were recorded independently by two observers. To obtain pre- and post-exposure baseline data, sham exposures were performed during the first

daily session of each phase and during an additional session 40 hr after completion of the 3 phases.

The numbers of entries per session by each rat into the safer area were measures of locomotor activity rather than of escape learning. For the periods of RFR exposure, the mean number and standard error (SE) of entries by all 10 rats in all 3 phases of experiment 1 was 7.9 +/- 0.7 per session or an average of about 1.6 times per rat per 2-min exposure, for a grand mean of 119 entries per rat during the 75 periods of RFR exposure. The corresponding values for the nonexposure periods were 5.6 +/- 0.4 per session, 0.93 times per period, and a grand mean of 70 entries per rat. The differences were significant ($p<0.05$), an indication of greater activity during RFR exposure. During the exposure periods, the rats exhibited hyperthermic behavior similar to that during the pilot study, i.e., initial hyperactive locomotion followed by salivary grooming and, usually, by immobilization and collapse. In general, complete recovery occurred within 30-60 seconds of the next nonexposure period.

For the nonexposure intervals, there were no statistically significant intraphase or interphase differences in mean entries; the means were comparable to baseline values. For the RFR-exposure intervals, there were no significant intraphase differences in activity, but the mean phase activity diminished significantly with successive phases.

Despite the significantly higher levels of activity during RFR exposure, the mean times spent in the safer area during the periods of exposure and nonexposure did not differ significantly from baseline values or from each other either intraphase or interphase. Irrespective of experimental conditions, the rats spent only about 10% of the session times in the safer area, and specifically, the absence of an interphase effect indicated that the level of RFR in the safer area (obtained by the respective percentages of SAR reduction from 60 W/kg) was not a significant factor.

In the second formal experiment, 10 of 20 naive rats were treated in a manner similar to that used for phase 3, i.e., each crossing into the safer area during exposure produced reduction of the SAR from 60 to 2 W/kg. The other 10 rats were similarly scheduled, but faradic shock (about 800 microamperes) was administered instead of RFR, and each crossing to the safer area while the source was activated produced reduction of the intensity to zero. Six daily sessions of 22 min each were performed, of which the first day was for acquiring baseline data.

The mean number of entries per session into the safer area by the RFR-exposed rats during the exposure periods (3.3 +/- 0.7) was significantly lower ($p<0.05$) than for the rats during phase 3 of experiment 1. Also, as seen in Fig. 4, the mean percentages of time spent in the safer area by the RFR-exposed rats (about 5% both during, and between, exposure periods of each daily session) were smaller than the baseline mean (about 10% for all 20 rats), with a trend toward diminution in successive sessions.

By contrast, during the first and second days of faradic shock, the rats so treated occupied the safer area for averages of 89% and 95% of the respective source-on periods, and most of the time spent in the unsafe area occurred during the source-off periods. In addition, the rats probed the unsafe area frequently during the first day and quickly retreated to the safer area; they also actively resisted placement within the cavity on the second day. Because of these decisive results, the remaining shock sessions were cancelled.

CRITIQUE: There was a flaw in the design of the exposure apparatus. The unsafe area of the false floor used for the faradic-shock aspects was readily distinguishable visually from the safer area by the conductive stripes, which may have also provided tactile differences. However, the safer area of the false floor used for the RFR aspects of the study was not visually distinguishable from the unsafe area; the only visual cue was the boundary per se. Thus, the rats exposed to RFR may have had a more difficult task to learn than those given faradic shock. This flaw could have been avoided by providing, on the unsafe area of the RFR floor, nonconductive stripes of reflective characteristics and texture approximating those of the faradic floor, a measure taken in a subsequent investigation (Levinson et al., 1982).

It should also be noted that although the safer area was 25% of the total floor area in each case, the data indicate that the rats actually spent only about 10% of the time there during baseline sessions. Was there some repelling factor associated with that region of the cavity?

Despite the points above, the finding that rats do not learn to escape from potentially lethal levels of RFR appears unequivocal. Experimental results obtained subsequently by Levinson et al. (1982) provide additional support for this finding.

In the discussion by the authors, they considered the argument that the use of the alternating 2-min cycles of RFR and respite might confuse the rats and preclude learning. Specifically, during the respite periods, the paradigm did not provide for escape reinforcement whenever a rat entered the safer area nor was punishment administered whenever a rat left the safer area. Their response was that the periodic respites might retard avoidance learning, but that escape learning is motivated primarily during application of noxious stimulation, an argument validated by their results with faradic shock.

Another point considered by the authors was their use of faradic shock as a positive control rather than infrared radiation (IR). Their response was that the rats used for RFR exposure and faradic shock served as their own controls, and that the findings with the two forms of stimulation were independent, with no comparison intended; the primary purpose of the use of faradic shock was to demonstrate that a simple escape task could be learned rapidly.

An important implication of the findings of this investigation is that humans who might be exposed to intense levels of RFR at frequencies that

penetrate deeply may not sense the internal heat generated in time to avoid tissue damage. This could be true under some conditions. It is also interesting to note that Justesen et al. (1982) reported that the power-density threshold for detecting 2.45-GHz RFR in the human forearm was about 15 times larger than for IR.

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Bioelectromagnetics, Vol. 3, No. 1, pp. 117-125 (1982)

Levinson, D.M., A.M. Grove, R.L. Clarke, and D.R. Justesen
PHOTIC CUING OF ESCAPE BY RATS FROM AN INTENSE MICROWAVE FIELD
Bioelectromagnetics, Vol. 3, No. 1, pp. 105-116 (1982)

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Levinson, D.M., A.M. Grove, R.L. Clarke, and D.R. Justesen
PHOTIC CUING OF ESCAPE BY RATS FROM AN INTENSE MICROWAVE FIELD
Bioelectromagnetics, Vol. 3, No. 1, pp. 105-116 (1982)

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AUTHOR ABSTRACT: A total of 16 female hooded rats was first observed for baseline behaviors and then they received 25 2-min trials of training, five trials per day, under one of four stimulus conditions (all ns=4): exposure to a highly intense 918-MHz field (dose rate, 60 mW/g); exposure to photic stimulation (about 350 lx); exposure to the field in synchrony with photic stimulation; or exposure to faradic shock (about 800 microamperes rms). During conditioning trials, which were separated by 2-min intertrial intervals, entry by a rat into a safe area of a multimode cavity resulted in immediate and complete cessation of stimulation; exit, in resumption.

Acquisition of the escape response was rapid and highly efficient for shocked animals and was less rapid and efficient but was reliably demonstrated by irradiated animals that were also signaled by light. In the absence of microwave irradiation, cessation of light did not reliably motivate escape behavior. Although there was weak evidence of escape learning by rats subjected only to microwave irradiation, their performances failed to differ reliably from those of rats in the light-only condition. These data confirm and extend those of Carroll et al, which indicate that potentially lethal, deeply penetrating, nonpulsed microwaves in a multipath field lack the sensory quality to motivate efficient aversive behavior by the rat.

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Study Type: Behavior; IN VIVO; RAT
Effect Type: Comparison of escape performances from a photically-cued or noncued, potentially-lethal RFR field and from faradic shock
Frequency: 918 MHz
Modulation: Nonpulsed (amplitude-modulated at 3 Hz)
Power density: Not measured
SAR: 60 W/kg

EXPOSURE CONDITIONS: RFR- and sham exposures were performed in a multimode cavity having a 3-element mode stirrer rotating once per second, thus amplitude-modulating the RFR at 3 Hz. The peak-to-average power ratio was about 5. The ambient temperature in the cavity ranged between 21 and 25 deg C and the mean relative humidity was 53%. Air flow through the cavity was at 0.1 m/second.

The cavity was equipped with either of two false floors of white, opaque Plexiglas on which the boundary of a rectangular "safe" region about 25% of the total floor area was marked with black tape. The "unsafe" area of one of the floors was painted with 22 parallel, equidistant 5-mm stripes of conductive silver for administering faradic shock; alternate

stripes were connected together to form 2 poles, to which the shock source was connected. A similar grid of nonconductive paint was applied to the unsafe area of the other floor, which was used in the RFR sessions.

Movement from the unsafe to the safe area was the simple escape response required. It should be noted that for RFR exposure, the safe area was not a region of intrinsically lower intensity relative to the unsafe area, but was rendered safe by removal of the input RFR to the cavity each time a rat entered that area.

A thermally sheltered 40-W white incandescent lamp in an upper rear corner of the cavity provided continuous illumination except when it was used for photic cuing. Two continuously operated 40-W red incandescent lamps in the top wall of the cavity permitted observation of animal behavior when the white lamp was extinguished.

OTHER INFORMATION: Whole-body SARs were determined calorimetrically with saline solution in foamed polystyrene vessels exposed at the center of the unsafe area, and by measuring colonic temperature increases in similarly exposed live rats previously rendered hypothermic (Carroll et al., 1980).

Four groups of 4 rats each were studied. One group, designated M, was exposed to RFR (60 W/kg) only; the second, designated L, was stimulated photically (350 lx) only; the third, ML, was concurrently stimulated with the RFR and photically; and the last, a positive-control group designated S, was given faradic shock (800 microamperes) only.

Daily sessions of 22-min duration were conducted with each group for 6 consecutive days. Baseline data (without stimulation) were obtained for each group in session 1, during which the number of entries by each rat into the safe area and the times spent therein were recorded. The criterion for entry into either area was traversal of the boundary by the entire head of the rat. Sessions 2-6 each comprised 11 serially numbered 2-min periods, with the respective stimuli available only during the 5 even-numbered periods. During stimulation-availability periods, the rats were continuously subjected to the stimuli except when they moved into the safe area. Rectal temperatures were taken just before the start of session 6 and immediately after completion of the last stimulation period.

To determine whether the rats had adequate opportunity to associate entry into the safe area with cessation of stimulation, the numbers of entries by each rat during the even periods of each session (10 min total time) were summed and the sums were averaged by group daily. Figure 2 showed that the mean numbers of entries for session 1 (baseline) were about 4 for the S group, 6 each for the M and L groups, and 8 for the ML group. No variances were given, but the authors stated that by analysis of variance, the differences among these baseline means were due to chance.

For the S group, after a small initial rise for session 2, the mean number of entries (during the 10 min of stimulation) decreased to a plateau of about 2 (cf. the baseline value 4) for sessions 3-6. For the M group, after successive rises for sessions 2 and 3, the mean diminished to a plateau comparable to the baseline value for sessions 4-6. The results for the L and ML groups were similar to those for the M group, but with plateaus of about 14 and 19, both higher than their respective baseline values.

The mean times spent in the safe area by each group during the entire 22-min (1320-second) session of each day, i.e. irrespective of the presence or absence of stimulation, were presented in Fig. 3 (with standard-error bars). The baseline values (session 1) for the 4 groups were all about 100 seconds or about 8% of the session time. For the L group, the mean times remained at baseline for sessions 2-6. The values for the M group also remained at baseline for sessions 2 and 3, but rose to about 300 seconds (23% of the session time) for sessions 4-6. By contrast, the mean times for the ML group rose linearly for sessions 2-3 to a plateau of about 650 seconds (49%) for sessions 4-6, and the values for the S group rose similarly to a plateau of about 1200 seconds (91%).

The mean times each group spent in the safe area during each of the five 2-min periods of stimulation and the six 2-min periods of nonstimulation each session were presented separately in Fig. 4. Photic stimulation only (L group) produced no significant differences among mean values for stimulation and nonstimulation periods either within each session or among all 6 sessions. RFR stimulation only (M group) yielded similar results for sessions 1-3, but the variations among period values increased progressively for sessions 4-6 with no clear differences between stimulation and nonstimulation, and a trend toward longer durations in the safe area was discernible (to a mean of about 25% per period for session 6 as compared with about 8% for session 1).

The combination of photic stimulation and RFR (ML group) yielded no significant differences between stimulation and nonstimulation values, but the variations among period values were much larger, as was the trend toward longer durations in the safe area (49% for session 6). By contrast with the other 3 groups, the faradic-shock (S) group spent about 75% of session 2 in the safe area (with no significant difference between mean values for stimulation and nonstimulation). Moreover, after the first two 2-min periods, the rats spent 100% of the remainders of sessions 3-6 in the safe area.

The results of the rectal temperature measurements in session 6 (Table 1) showed significant ($p<0.05$) increases for the M and S groups and nonsignificant ($p>0.05$) increases and decreases for the L group. For the ML group, the difference between the mean final and initial temperatures was not significant; however, the temperature of 1 of the 4 rats was above normal initially (40.6 deg C), presumably from excitation, and did not change. The temperature increases of the other 3 rats were significant.

CRITIQUE: Although the safe area of each false floor was about 25% of its total area, Fig. 3 indicated that even during baseline sessions, all 4 groups spent only about 8% of the session time there. Was there some repellent factor associated with that region of the cavity (or conversely an attractive factor in the unsafe area)? (A similar finding was obtained by Carroll et al., 1980.) If so, there is no assurance that such a factor would bias the results for each group equivalently so as to permit removal of the factor from consideration.

The statement that "the white, 40-W incandescent lamp...was continuously illuminated except during trials with animals for which it served as a photic cue" is unclear. A possible interpretation of the sentence is that the lamp was on continuously for the tests of the faradic-shock (S) and RFR-only (M) groups and was off during the sessions with the ML and M groups except during the even-numbered 2-min periods (with and without concurrent RFR, respectively). If this interpretation is correct, then the S and M groups were afforded brighter illumination for visually distinguishing between the unsafe and safe areas during the 2-min periods of nonstimulation than the ML and L groups. The extent to which this difference in illumination procedure could have affected the results is unknown.

The text accompanying Fig. 2 (mean entries to safe area during the 10 min of stimulation per session vs session number) was brief and obscure, with a confusing mention of Fig. 3. The authors stated that: "The relatively small mean number of entries by rats of the S group during Days 2 through 6 reflects very rapid learning of the escape response by these animals." It would be expected that animal activity would increase directly with the degree of stress induced by the stimulation and would be reflected in an increase in the number of entries to the safe area, i.e., that the latter is a measure of the former. However, presumably the faradic shock was so noxious that once the rats learned the location of the safe area, they did not necessarily reduce their activities but confined their movements to that area. Moreover, as discussed above, recall of the location of the safe area may have been better facilitated by the white light furnished.

Conversely, the increases in the numbers of entries to the safe area with the other stimuli appear to indicate that the rats were able to sense the stimuli, but had difficulties in locating the safe area or remembering its location. Thus, performance on this type of behavioral paradigm depends not only on the detection sensitivity of the animal to the stimulus but also on its ability to discern and remember the ameliorative measures available to it.

In their discussion, the authors compared their results with those of Monahan and Ho (1977a, b; 1979b) and with Monahan and Henton (1977) for mice exposed in a waveguide. It should be noted that there was no Monahan and Ho (1977a or 1979b) in their reference list. We surmised that for Monahan and Ho (1977a) they meant Monahan and Henton (1977a), and for Monahan and Ho (1979b) they meant Monahan and Ho (1977) in our reference list below. On the basis of their findings with rats,

Levinson et al. (1982), with due regard for the species difference, argued against the conclusion of Monahan and coworkers that mice could detect the RFR and endeavor to ameliorate their exposure by reorienting themselves in the waveguide to reduce their SARs.

One basis for the argument was that the SAR reductions were only about 30%, or too small to discriminate because of thermal inertia. This basis appears to be irrelevant because perhaps the 30% represented about the maximum reduction of SAR available to the mouse rather than a measure of its detection sensitivity. The other basis was that the detection sensitivity increased (rather than decreased) with increasing ambient temperature in the waveguide, citing a reliable SAR decline from an initial value of only 0.06 W/kg at 35 deg C. [Note that Table 1 of Monahan and Ho (1977) does show a trend toward SAR decline with time for exposure at 0.06 W/kg but that those results were not labeled as statistically significant; the SAR declines at 0.6 W/kg and higher at 35 deg C were significant.] This basis appears to ignore the lower thresholds for loss of thermoregulation at higher ambients.

Levinson et al. (1982) also suggested that the SAR declines with exposure time observed by Monahan and coworkers were more likely due to heat prostration of the mice to lower energy-absorption profiles in the waveguide, rather than to behavioral reorientations. However, the SAR declines with time were gradual rather than sudden. (It should be noted that qualitatively similar SAR reductions were obtained in rats by Monahan and Henton, 1977b, as well as in mice.)

There appears to be common agreement that under some exposure conditions, intense levels of deeply penetrating RFR may not be sensed in time to avoid tissue damage.

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Kaplan, I.T., W. Metlay, M.M. Zaret, L. Birenbaum, and S.W. Rosenthal
ABSENCE OF HEART-RATE EFFECTS IN RABBITS DURING LOW-LEVEL MICROWAVE IRRADIATION
IEEE Trans. Microwave Theory and Tech., Vol. 19, No. 2, pp. 168-173 (1971)

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AUTHOR ABSTRACT: Soviet studies have reported that low-level microwave irradiation alters the heart rate of humans and animals. In a replication of one such study, 16 rabbits were exposed to dorsal irradiation of the head by 2.4-GHz CW microwaves at a power density of 10 mW/sq cm for 20 min. The rest of the animal's body was shielded by absorbent material. There was no significant difference between the heart rate during or after irradiation and the heart rate of the same animals during a control condition in which they were not irradiated. Analysis of the variability in heart rate observed in this experiment suggested that the heart-rate effects reported in the original Soviet study might have been chance variations.

In a second experiment, heart rate, respiration rate, and body temperature were recorded simultaneously while each of two rabbits was irradiated as before, on the dorsal aspect of the head only, at various power densities from 0 to 100 mW/sq cm, in steps of 20 mW/sq cm. Respiration rate increased during irradiation at 40 mW/sq cm, body temperature rose at 80 mW/sq cm, and ultimately the heart rate also increased, but only at 100 mW/sq cm.

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Study Type: Cardiovascular Effects, Physiology and Biochemistry, Biorhythms; IN VIVO; RABBIT
Effect Type: RFR-induced effects on heart rate, respiration rate, and subcutaneous temperature
Frequency: 2.4 GHz
Modulation: CW
Power Density: 10-100 mW/sq cm
SAR: Not determined

EXPOSURE CONDITIONS: The heads of rabbits restrained in a wooden "squeeze box" were exposed from above to far-field RFR for 20 min in an anechoic chamber; a panel of RFR-absorbent material on top of the box was used to shield the rest of the body. Although not stated, the long axis of the rabbit presumably was parallel to the E-vector. RFR exposures were preceded and followed by 10 min each of no RFR (total session time of 40 min). Control sessions consisted of 40 min of no RFR. In the first experiment, the power density was 10 mW/sq cm; in the second, the power density was 20, 40, 60, 80, or 100 mW/sq cm.

OTHER INFORMATION: Presman and Levitina had exposed various regions of the rabbit with 2.4-GHz CW RFR at 7-12 mW/sq cm (1962a), and to 1-

microsecond pulses, 700 pps, at 3-5 mW/sq cm average power density (1962b). The maximum effect they obtained with the CW RFR was an increase in heart-beat rate during and after exposure of the dorsal aspect of the rabbit head. The procedure used by Kaplan et al. (1971) in their first experiment was similar to that of Presman and Levitina, in an endeavor to reproduce that effect.

Sixteen male albino rabbits weighing 2-3 kg were studied. Each was instrumented subcutaneously with small curved surgical needles in the left chest, right chest, and left hip for EKG recording. These needles and their lead wires were shielded from the incident RFR by the absorbent panel. The rabbits were placed in the chamber 15 min preceding each session. EKGs of 20-second durations were recorded every 2 min during the 20 min of RFR (or sham) exposure and once per min during the 10-min pre- and post-exposure periods. Each rabbit was given a control session followed by an RFR session on the same day; one week later, each was given an RFR session followed by a control session. Thus, 64 sessions were conducted.

The heart rates for the 16 rabbits were averaged over each 20-second EKG interval of the 32 RFR sessions and similarly for the 32 control sessions. The means (Fig. 6) ranged from a high of 65.4 beats per EKG interval at session start to 63.0 at session end, with a mean standard deviation of 7.6 and no statistically significant differences between means for corresponding intervals. The slight downward trend during RFR and control sessions was approximately linear with time, with no observable shift at either the pre-exposure/exposure or the exposure/post-exposure boundary. Analysis of variance showed that the trend was significant ($p<0.01$).

For comparison with the results of Presman and Levitina (1962a), changes in heart rate were calculated by their method. The mean heart rate for the 10-min pre-exposure period of each RFR session was determined, and the deviation from this mean was calculated for each 20-second EKG record during the exposure and post-exposure periods. Each control session was treated similarly. The relative change in rate was obtained for each EKG record of the RFR session by subtracting from its deviation the corresponding deviation for the control session.

The 16 rabbits were arbitrarily divided into 4 equal groups and the relative changes in rate were averaged over each group. The results (Fig. 4) were mixed; one group exhibited positive differences (relative increases in heart rate) during the RFR- and post-exposure periods; the second group showed negative differences (relative decreases) during the first 10 min of RFR and no consistent subsequent changes; the differences for the third group were negative for the first 8 min of RFR, zero for the next 4 min, and positive for the rest of the session; and the differences for the fourth group were negative for almost the entire session.

When the data for all 16 rabbits (32 RFR and 32 control sessions) were averaged (Fig. 5) there were no statistically significant relative

changes. The mean heart rate was about 64 beats per 20-second EKG interval and the average relative changes ranged from about -0.5 to + 1 beat per 20-second EKG interval with a standard deviation of 4.6 beats per interval. By contrast, the relative changes obtained by Presman and Levitina (1962a) for 16 RFR and 16 control sessions with 8 rabbits (also shown in Fig. 5) were positive, larger, and more variable, ranging from 0 to 8 beats per interval. Presman and Levitina had defined the "coefficient of chronotropic effect, K," as:

$$K = (100+mi)/(100+md),$$

where mi and md are the respective positive and negative relative changes in percent. Values of K>1 and K<1 signified increases and decreases of heart rate, respectively. Their results were K=1.3 during RFR exposure and K=1.42 post-exposure. By contrast, the corresponding values obtained by Kaplan et al. (1971) were 0.93 and 0.94, which did not differ significantly from 1. The latter authors opined that the variations found by Presman and Levitina were probably due to chance.

The second experiment was directed toward determining the power density needed to alter the rabbit heart rate. The head of each of 2 rabbits was exposed once weekly at 0, 20, 40, 60, 80, or 100 mW/sq cm for 20 min, with 10-min pre- and post-exposure periods as before. In addition to heart rate, the respiration rate was recorded with a strain gauge around the thorax, and the body temperature was measured with a hypodermic thermistor inserted subcutaneously near the midline of the lower back. These sensors and their lead wires were also shielded from the RFR by the absorption panel.

During the pre-exposure period, the mean heart rate was 192 beats/min (which corresponded to 64 beats per 20-second EKG interval), respiration averaged 373 breath/min, and mean body temperature was 39.1 deg C. The high respiration rate was ascribed to animal stress; the authors stated that the respiration rate of rabbits in the squeeze box but otherwise left undisturbed in the anechoic chamber for an hour or more gradually decreased to normal values in the range 38-60 breaths/min.

During RFR exposure, there was no consistent change in heart rate except at 100 mW/sq cm, for which the mean heart rate was 12 beats/min faster during the second 10 min of exposure than before exposure. However, respiration rate increased by about 50 breaths/min at 40 mW/sq cm and by over 100 breaths/min at 100 mW/sq cm. At 80 and 100 mW/sq cm, body temperature rose by about 0.5 deg C. Thus, the rabbits were under significant thermal burdens at power densities considerably lower than levels needed to increase their heart rates.

CRITIQUE: The use of metal electrodes, sensors, and lead wires for recording biorhythms such as the EKG and EEG during exposure to RFR may introduce field perturbations or recording artifacts. However, this did not appear to be a problem in this investigation, because only part-body (head) exposures were used and presumably the shielding of the devices and leads provided by the RFR-absorbing panel was adequate.

Birenbaum et al. (1975), in a similar study (with exposures of rabbits for 1 hr instead of 20 min), found that the heart rate, respiration rate, and subcutaneous temperature under sham exposure decreased approximately linearly with time (negative slopes), and that the slopes increased (became less negative and then increasingly positive) as the power density was increased from 0 to 100 mW/sq cm. These results generally indicated that the changes in heart and respiration rates were associated with the additional thermal burden imposed by the RFR.

Chou et al. (1980b) exposed rabbits for 20 min to far-field, 2.45-GHz RFR as follows: CW at 5 or 80 mW/sq cm; 1-microsecond pulses, 700 pps, at power densities of 7.1 W/sq cm peak and 5 mW/sq cm average; and 10-microsecond pulses at 13.7 W/sq cm synchronized with and triggered by the R wave of the EKG at delays of 0, 100, and 200 milliseconds. Non-perturbing carbon-loaded Teflon electrodes (Chou and Guy, 1979a) were used to record EKGs before, during, and after RFR exposure. Increases in heart-beat rate were observed at 80 mW/sq cm but not lower average power densities. A possible weak positive chronotropic effect was also reported for the pulsed RFR at delays of 100 and 200 milliseconds after the R wave. No cumulative effect was observed over a period of 4 months.

In summary, all of the findings above were at variance with those of Presman and Levitina (1962a).

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Birenbaum. L., I.T. Kaplan, W. Metlay, S.W. Rosenthal, and M.M. Zaret
MICROWAVE AND INFRA-RED EFFECTS ON HEART RATE, RESPIRATION RATE AND SUBCUTANEOUS TEMPERATURE OF THE RABBIT
J. Microwave Power, Vol. 10, No. 1, pp. 3-18 (1975)

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AUTHOR ABSTRACT: Microwaves (CW, 2.4 GHz) were used to irradiate the dorsal aspect of the head of unanesthetized rabbits at power levels from 0 to 80 mW/sq cm. Respiration rate, heart rate and subcutaneous temperature were monitored. Increases in all 3 indices resulted, with the greatest increases at the highest power levels. Respiration rate increases were 20 times greater than those in the heart rate.

CW and pulsed microwaves at 2.8 GHz, 20 mW/sq cm average power level, were used to irradiate the entire dorsal surface of the animal. No significant difference in any of the 3 indices between the CW and pulsed responses could be detected.

CW 2.4 GHz microwave and infra-red whole back irradiations were carried out at 0, 10, and 20 mW/sq cm levels. Although respiration and heart rate changes were substantially the same, subcutaneous temperature increased more rapidly and rose to higher values for the infra-red case.

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Study Type: Cardiovascular Effects, Physiology and Biochemistry, Biorhythms; IN VIVO; RABBIT
Effect Type: Effects of CW and pulsed RFR and of infrared radiation on heart rate, respiration rate, and subcutaneous temperature
Frequency: 2.4 GHz CW; 2.8 GHz CW and pulsed
Modulation: CW and 1.3-microsecond pulses at 1000 pps (0.0013 duty)
Power Density: 20-80 mW/sq cm at 2.4 GHz; 20 mW/sq cm Av at 2.8 GHz; 10, 20 mW/sq cm IR
SAR: Not determined

EXPOSURE CONDITIONS: Rabbits restrained in a wooden "squeeze box" were exposed from above to far-field RFR in an anechoic chamber. In experiment 1, exposures were for 1 hr per session to 2.4-GHz CW RFR at 0, 20, 40, 60, or 80 mW/sq cm, and a panel of RFR-absorbent material was used as a shield so as to expose only the dorsal aspect of the head. For experiment 2, exposures were for 20 min per session to 2.8-GHz CW or pulsed RFR at 20 mW/sq cm (Av), and the absorbent panel was not used, thus exposing the entire dorsal surface. In experiment 3, exposures were for 1 hr per session to 2.4-GHz CW RFR or infrared radiation (IR) at 0, 10, or 20 mW/sq cm without the absorbent panel. Although not stated, the long axis of the rabbit presumably was parallel to the E-vector of the RFR.

OTHER INFORMATION: The procedure used was similar to that followed in an earlier investigation (Kaplan et al., 1971). After confining the

rabbit in the squeeze box, the rabbit was instrumented with: subcutaneous small curved surgical needles in the left chest, right chest, and left hip for EKG recording; a strain gauge tied around the thorax to measure respiration rate; and a subcutaneous hypodermic thermistor near the midline of the lower back for temperature measurement.

The investigators had found that prior to treatment, heart and respiration rates of rabbits fluctuated widely in response to the experimental situation, which necessitated months of sham exposure for abatement of such fluctuations. Therefore, the same rabbits were studied repeatedly instead of using different rabbits for each set of experimental conditions. To acclimate the rabbit to the experimental situation, it was placed in the anechoic chamber 15 min before the beginning of each session.

Two rabbits were studied in experiment 1. The dorsal aspect of the head only of each was exposed to 2.4-GHz CW RFR at 0, 20, 40, 60, and 80 mW/sq cm for 2 sessions to each level at intervals at least 1 week apart. Each session consisted of 10 min of no exposure followed by 60 min of exposure. The EKG, respiration rate, and temperature were recorded at 1-min intervals during the session. Each EKG and respiration trace was for 10 seconds.

For each physiological indicator, the results for the two rabbits during each 10-min interval of the 2 exposure sessions at each level were averaged, and straight lines were fitted by least squares. In addition, variabilities were assessed by calculating the pre-exposure means of each indicator for each RFR level, and determining the average and standard deviation (SD) of the resulting 5 means for each indicator. These calculations yielded 193.6 +/- 4.6 beats/min for the heart rate, 292 +/- 22.4 breaths/min for the respiration rate, and 38.9 +/- 0.2deg C for subcutaneous temperature.

At 0 mW/sq cm (no RFR), the heart rate diminished slightly with time (from about 200/min during the pre-exposure interval to about 190/min for the last 10 min of the session), which corresponded to a slope of -0.17 (0.1%) change of mean heart rate per min. At 20 mW/sq cm, there was no diminution of rate with time (zero slope). At 40, 60, and 80 mW/sq cm, the slopes increased progressively with power density.

The results for the mean respiration rate and subcutaneous temperature were qualitatively similar to those for the heart rate. The no-RFR slopes were -2.66 breaths/min (a decrease of 0.9%) and -0.005 deg C/min (a decrease of 0.01%), and the slopes for 80 mW/sq cm were +9.39 breaths/min (a 3.2% increase) and +0.021 deg C/min (a 0.05% increase). Thus, the percentage changes of mean respiration rate with power density were considerably higher than the changes of heart rate and temperature. Also, at 40 mW/sq cm and higher, the variations of respiration rate with time tended to attain plateaus after about 30 min of exposure, whereas the other two indices rose linearly throughout the exposure period.

In experiment 2, the two rabbits used in experiment 1 and two additional ones were exposed to 2.8-GHz RFR at 20 mW/sq cm, each 4 times to CW and 4 times to 1.3-microsecond pulses at 1000 pps (0.0013 duty cycle), with the RFR-absorbent panel removed, thus exposing the entire dorsal surface. Sessions comprised 10 min of pre-exposure and 20 min of exposure, records were taken every 2 min, the 16 values per observation were averaged, and lines were fitted by least squares.

The mean heart rate was about the same for the CW and pulsed RFR and remained nearly constant with time. The mean temperatures for the two types of RFR were basically equal to one another at corresponding intervals; both remained constant during the pre-exposure period and rose linearly by about 1.4 deg C by the end of the 20-min exposure period. The mean respiration rates also were constant during the pre-exposure period and rose linearly during exposure, but appeared to be slightly higher for pulsed than CW RFR at corresponding times. However, t-tests indicated that all differences between responses to CW and pulsed RFR were nonsignificant ($p>0.05$).

In experiment 3, the two rabbits used in experiment 1 were exposed to 2.4-GHz CW RFR again, each twice at 0, 10, and 20 mW/sq cm but with the RFR-absorbent panel absent. After completion of these exposures, these rabbits were also exposed to infrared radiation (IR), each twice at 10 and 20 mW/sq cm. (The results for sham-RFR exposures were used in lieu of performing distinct sham-IR exposures.) The IR was from a bowl heater at 417 and 540 deg C, respectively. Assuming that black-body radiation was emitted, the peak wavelength and half-power band at 10 mW/sq cm were 4.15 and 2.55-7.60 micrometers; the corresponding values for 20 mW/sq cm were 3.55 and 2.17-6.47 micrometers. Exposures were for 60 min after a 10-min pre-exposure period, records were taken at 5-min intervals, and the averages were fitted with least-squares lines.

The mean heart and respiration rates under sham exposure diminished linearly as in experiment 1, and the initial mean temperature was about 38.0 deg C but exhibited an essentially zero (instead of negative) slope for the remainder of the session. At 10 mW/sq cm of RFR, the slopes for the heart and respiration rates were both zero, but the temperature during pre-exposure was about 38.6 deg C (i.e., about 0.6 deg higher than under sham exposure), and rose linearly during exposure to about 39.3 deg C. At 20 mW/sq cm of RFR, the slope for the heart rate was again zero, but the respiration rate increased from about 240 to 270 breaths/min and the temperature from about 38.6 to 39.9 deg C.

At 10 mW/sq cm of IR, the heart rate had essentially the same negative slope as for sham exposure, and increasing the power density to 20 mW/sq cm rendered the slope slightly less negative. The respiration rate, which had a negative slope under sham exposure, exhibited the same positive slope under 10 and 20 mW/sq cm of IR. The major differences between results for the RFR and IR were in the subcutaneous temperature. The slope of this index was zero under sham exposure. At 10 mW/sq cm, the temperature rose from about 37.4 during pre-exposure to about 39.0 deg C during the first 30 min of exposure, and leveled off

for the remaining 30 min. At 20 mW/sq cm, the temperature rose from about 37.3 to a plateau of about 40.7 deg C.

CRITIQUE: The results for exposure to CW RFR at 0-80 mW/sq cm confirm and extend those of the previous investigation (Kaplan et al., 1971) and are at variance with the findings of Presman and Levitina (1962a) with 2.40-GHz CW RFR at 7-12 mW/sq cm.

The authors noted that exposure of metal electrodes, sensors, and lead wires to RFR could cause field perturbations or recording artifacts, but believed that they had minimized the problems by locating the surgical needles used for EKG recordings and the strain gauge used for measuring respiration rates in the ventral region of the rabbit, thus using the body as an absorption shield. Also, the hypodermic-needle thermistor-temperature sensor was oriented perpendicular to the E-field direction. The absence of abrupt shifts in values at the boundaries between the pre-exposure and exposure intervals supports their belief.

The authors also commented that respiration rate is a more sensitive indicator of increases of RFR level than heart rate, a point that is consistent with the more efficient heat removal rate obtained by faster breathing. The rabbits were clearly under significant heat stress at 10 mW/sq cm, and the stress became successively more severe as the power density was increased, as evidenced by the increases in subcutaneous temperature. Monitoring core temperature instead of (or in addition to) subcutaneous temperature might have indicated the threshold power density at which the thermoregulatory capabilities of the animals were exceeded (if such occurred).

The absence of significant differences in responses to the CW and pulsed RFR is at variance with the findings of Presman and Levitina (1962b) with 1-microsecond pulses, 700 pps, 3-5 mW/sq cm (Av), and is an indication that the major changes in the physiological indices measured (irrespective of the type of RFR) were due to the absorption of the energy at 20 mW/sq cm as heat.

Chou et al. (1980b) exposed rabbits for 20 min to far-field, 2.45-GHz RFR as follows: CW at 5 or 80 mW/sq cm; 1-microsecond pulses, 700 pps, at power densities of 7.1 W/sq cm peak and 5 mW/sq cm average; and 10-microsecond pulses at 13.7 W/sq cm synchronized with and triggered by the R wave of the EKG at delays of 0, 100, and 200 milliseconds. Non-perturbing carbon-loaded Teflon electrodes (Chou and Guy, 1979a) were used to record EKGs before, during, and after RFR exposure. Increases in heart-beat rate were observed at 80 mW/sq cm but not lower average power densities. A possible weak positive chronotropic effect was also reported for the pulsed RFR at delays of 100 and 200 milliseconds after the R wave. No cumulative effect was observed over a period of 4 months.

Birenbaum et al. (1975) stated that the ratio of peak-to-average power density was about 770. Thus, the peak power density was 15.4 W/sq cm, a value (together with the 1.3-microsecond pulse duration) probably well

above the energy-density threshold for the RFR-auditory effect. However, no mention was made of the possible occurrence of this effect.

Subcutaneous temperature changes from exposure to RFR and IR at equal power densities were the largest differences in responses obtained in this investigation, results that would be expected from the use of subcutaneous sensors and the differences in reflection coefficients and penetration depths for the RFR and IR.

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Bull. Exp. Biol. Med., Vol. 53, No. 2, pp. 154-157 (1963b)
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Chou, C.-K., L.F. Han, and A.W. Guy
MICROWAVE RADIATION AND HEART-BEAT RATE OF RABBITS
J. Microwave Power, Vol. 15, No. 2, pp. 87-93 (1980b)

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AUTHOR ABSTRACT: Each of three adult New Zealand rabbits, 2 male and 1 female albinos, was exposed dorsally or ventrally, to 2450-MHz plane waves for 20 min under each of several field conditions: 1) to continuous waves (CW) at 5 mW/sq cm; 2) to pulsed waves (PW) of 1-microsecond width that recurred 700 pps at an average of 5 mW/sq cm and at a peak of 7.1 W/sq cm; 3) to PW of 10-microsecond width at a peak of 13.7 W/sq cm that were synchronized with and triggered by the R wave of the electrocardiogram (EKG) at various delay times (0, 100, and 200 ms); and 4) to CW at 80 mW/sq cm. Carbon-loaded Teflon electrodes were used to record the EKG from forelimbs of an animal before, during, and after irradiation whilst it was maintained in a constant exposure geometry in a wooden squeeze box.

Field induced changes in the heart-beat rate were observed at 80 mW/sq cm but not at lower average power densities, although a weak positive chronotropic effect might have been occasioned by PW introduced at 100 and 200 ms after the R wave peak. No cumulative effect was observed over a period of four months. Thermographic analysis revealed relatively little absorption of microwave energy by the myocardium irrespective of anatomical aspect of exposure.

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Study Type: Cardiovascular Effects, Biorhythms; IN VIVO; RABBIT
Effect Type: Alterations of heart-beat rate by CW and pulsed RFR
Frequency: 2.45 GHz
Modulation: CW, 1-microsecond pulses at 700 pps (0.0007 duty), and 10-microsecond pulses synchronized with the R wave of the EKG (0.00003 duty)
Power Density: 5 or 80 mW/sq cm for CW; 5 mW/sq cm average for 1-microsecond pulses; and 13.7 W/sq cm Pk, 0.43 mW/sq cm Av for 10-microsecond pulses
SAR: 0.09-1.79 W/kg for dorsal exposure at 5 mW/sq cm; 0.12-0.87 W/kg for ventral exposure at 5 mW/sq cm

EXPOSURE CONDITIONS: Three rabbits restrained individually in a wooden "squeeze box" were exposed 20 min/day for 10 days dorsally (from above) or ventrally (from below) in an anechoic chamber to far-field RFR of each set of values. The long axis of the rabbit was parallel to the electric vector. Each exposure was preceded by at least 15 min of no exposure to allow the heart rate to stabilize, and was followed by 15 min of no exposure. The squeeze box permitted the rabbit to move its head and to assume a comfortable position. Sham-exposure sessions of the same duration were conducted before and after each series of sessions involving RFR exposure.

OTHER INFORMATION: This investigation was another endeavor to repeat the work of Presman and Levitina (1962a, 1962b), who had exposed various regions of the rabbit to 2.40-GHz CW RFR at 7-12 mW/sq cm, and to 1-microsecond pulses, 700 pps, at 3-5 mW/sq cm average power density. They had found that exposure of the dorsal and ventral areas resulted respectively in tachycardia and bradycardia, and that the chronotropic effect of pulsed RFR was more pronounced than for CW RFR. Other endeavors for the same purpose included those of Kaplan et al. (1971) and Birenbaum et al. (1975).

Scanning thermography was used to determine SARs at various locations within a 3.3-kg rabbit carcass exposed dorsally and ventrally. The values corresponding to 5 mW/sq cm were tabulated. With dorsal exposure, most of the energy was deposited in the ears, back, neck, and brain, with relatively little in the heart. Among the values were 0.093 W/kg in the heart (the smallest of the values tabulated) and 0.86 W/kg in the brain; the largest value tabulated was for the edge of the back, 1.79 W/kg. With ventral exposure, the values for the heart and brain were 0.30 and 0.24 W/kg, respectively, and the smallest and largest values tabulated were 0.12 W/kg (at the center of the trunk) and 0.87 W/kg (at the ventral edge of the chest).

Three rabbits were studied, with each rabbit subjected to each set of exposure values. The rabbits were acclimated to the experimental situation for several weeks prior to the treatment sessions. For EKG recording, each rabbit was instrumented with two carbon-loaded Teflon (nonperturbing) electrodes (Chou and Guy, 1979a) tied around cleanly shaven forelegs. Recordings were made every min during the 20 min of exposure and the 15-min pre-exposure and post-exposure periods.

Changes in heart rate were calculated by the method of Presman and Levitina (1962a). The mean heart rate for the 15-min pre-exposure period of each RFR session was determined, and the deviation from this mean was calculated for the EKG obtained each min during the 20 min of RFR exposure and the 15-min post-exposure period. Each control session was treated similarly. The relative change in rate was obtained for each min of RFR exposure by subtracting from its deviation the deviation for the corresponding min of the control session.

The sessions of ventral exposure of the rabbits to CW and to the 1-microsecond pulses, 700 pps, at 5 mW/sq cm yielded relative positive and negative changes of heart rate that varied randomly with time during exposure. The mean pre-exposure heart rate was 188 beats per min. The total range of variation during each session seldom exceeded 15 beats per min and the maximum mean relative change was less than 2.5% of the mean rate. Similar results were obtained for dorsal exposure; the mean pre-exposure rate was 187 beats per min and the maximum mean relative change was only 3%.

Dorsal exposure to the CW RFR at 80 mW/sq cm engendered considerable heat stress, causing enough animal movement to render satisfactory EKG recording during exposure difficult. Heart-beat rates measured when the

rabbits began to settle down did increase after the exposure period, but returned to normal after about 20 min.

The rabbits were also exposed dorsally to 10-microsecond pulses of 13.7 W/sq cm peak, 0.43 mW/sq cm average. When the pulses were synchronized with the R-wave peak of the EKG (no time delay), the changes in heart rate were random, i.e., all three rabbits exhibited both increases and decreases in rate during exposure, with no apparent time-dependent pattern. When the pulses were triggered 100 or 200 ms later than the R-wave peak, two of the rabbits again showed both positive and negative changes during exposure for both delays. However, the third rabbit displayed variable but consistent increases in rate for both delays. Its maximal mean relative increase was only 4.8 beats/min, less than 3% of its mean rate before exposure. Thus, the mean relative change in rate for all three was found to be slightly positive.

The authors reported that the heart-beat rates of the rabbits after more than four months of study did not differ from those at the beginning of the investigation, indicating that no cumulative effects had occurred.

CRITIQUE: Among the illuminating results of this investigation were the thermographically determined local SARs. With only 0.019 W/kg per mW/sq cm in the heart for dorsal exposure and 0.060 for ventral exposure, the absence of a direct effect of the RFR on the heart rate is perhaps not surprising. On the other hand, as mentioned by the authors, Presman and Levitina (1962a) had ascribed the tachycardia they observed from dorsal exposure of only the head to direct action of the RFR on the brain. The SAR in the brain, 0.86 W/kg at 5 mW/sq cm (0.17 W/kg per mW/sq cm), might have been high enough to affect the brain, but the negative results obtained by Chou et al. (1980b) do not support that hypothesis.

The findings of this investigation were in agreement with those of Kaplan et al. (1971) and Birenbaum et al. (1975), and with their conclusion that the changes in heart-beat rate reported by Presman and Levitina (1962a, 1962b) may have been chance variations.

The negative results of Chou et al. (1980b) with RFR pulses triggered by the R-wave peak of the EKG (without and with time delays) provided confirmation in intact animals of the negative results of Clapman and Cain (1975) and Liu et al. (1976) with isolated frog hearts. All of these negative findings were at variance with those of Frey and Seifert (1968) with isolated frog hearts.

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BIORHYTHM
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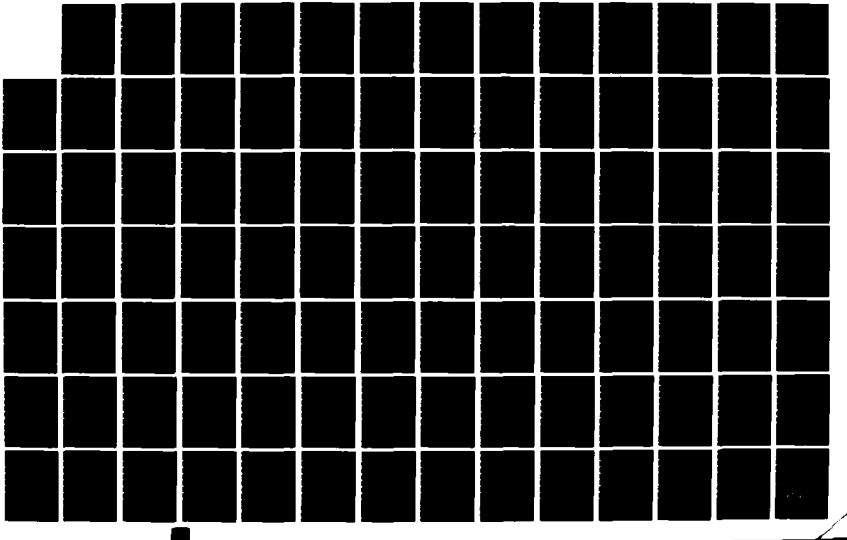

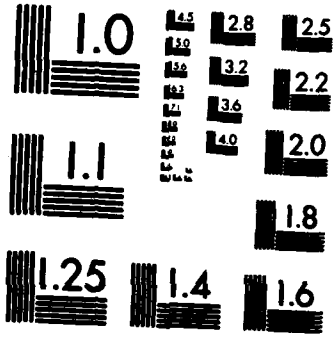

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Phillips, R.D., E.L. Hunt, R.D. Castro, and N.W. King
THERMOREGULATORY, METABOLIC, AND CARDIOVASCULAR RESPONSE OF RATS TO MICROWAVES
J. Appl. Physiol., Vol. 38, No. 4, pp. 630-635 (1975)

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AUTHOR ABSTRACT: This study was undertaken to determine the effects of 2,450-MHz microwave irradiation on thermoregulation, metabolism, and cardiovascular function of rats. Young adult male animals (430 g) were exposed for 30 min to 2,450-MHz microwaves in a cavity at absorbed dose rates of 0, 4.5, 6.5, or 11.1 mW/g. For animals of the size used in this study, these dose rates represent absorption of energy at the rate of 27.7, 40.1, and 68.2 cal/min, respectively. For a period of 5 h following exposure, measurements were made of colonic temperature, skin temperature, oxygen consumption, carbon dioxide production, respiratory quotient, and heart rate.

Rats that received 27.7 cal/min for 30 min exhibited an initial transient increase in colonic and skin temperatures but no alterations in other functions. The group irradiated at 40.1 cal/min had greater elevations in colonic and skin temperatures immediately after exposure, followed by overcompensation and lower than normal colonic temperatures for about 3 h. The metabolic rate was depressed in this group for 3 h. Bradycardia developed within 20 min after exposure and persisted for about 3 h. The group of rats that received 68.2 cal/min for 30 min had responses similar to those of the 40.1 cal/min group, but the changes were more severe and lasted longer.

In addition, a number of transient abnormalities were noted in the ECG tracings of rats that had received the highest dose, including irregular rhythms and incomplete heart block. The physiological changes observed in this study can be attributed to the heating induced by irradiation.

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Study Type: Metabolism and Thermoregulation, Cardiovascular Effects, Physiology and Biochemistry, Biorhythms; IN VIVO; RAT
Effect Type: Post-exposure effects of RFR on thermoregulation, metabolism, colonic and skin temperatures, and heart rate and performance
Frequency: 2.45 GHz
Modulation: 2.5-ms pulses at 120 pps (0.30 duty cycle)
Power Density: 21, 31, and 53 mW/sq-cm plane-wave equivalents of 4.5, 6.5, and 11.1 W/kg
SAR: 4.5, 6.5, or 11.1 W/kg

EXPOSURE CONDITIONS: Rats (mean weight of about 430 g) were exposed individually to RFR in a holder consisting of 2 Polyform endplates joined by Lucite rods to form a cylindrical space 18 cm long and 6.2 cm in diameter. Exposures were for 30 min in a multimode cavity powered

with a magnetron that provided 2.5-ms pulses at 120 pps. Three groups of 10 rats each were exposed, one at each SAR. A group of 30 other rats was sham-exposed for controls, half before exposure of each RFR group and half after RFR exposure.

OTHER INFORMATION: Twin-well calorimetry with rat carcasses was used to determine the SARs. The SARs were also expressed in cal/min; the values corresponding to 4.5, 6.5, and 11.1 W/kg for a 430-g rat were 27.7, 40.1, and 68.2 cal/min.

Immediately after exposure, the colonic temperature of each rat was measured with a thermistor probe, the rat was instrumented and placed in an all-Lucite holder, and measurements were begun within 10 min after completion of exposure. Skin temperature of the dorsal surface at the base of the tail was measured with a surface thermistor. EKG recordings were obtained from transthoracic surface electrodes. Continuous readings of heart rate were obtained with a tachograph.

One of the endplates of the Lucite holder had a hole through which the rat's head extended into a cone. Air was drawn with a pump into the cone through holes in the endplate and out through the nose of the cone at 100 ml/min; oxygen consumption and carbon dioxide production were calculated from measurements of the amounts of oxygen removed from, and carbon dioxide added to, air samples by the rat. Room temperature was held at 24.2 +/- 0.2 deg C.

The mean colonic temperature of the sham group was 38.6 deg C at the end of sham-exposure and decreased slowly to about 38.0 deg C over the 5-hr measurement period. For the 27.7-cal/min group, the temperature was 40.0 at the end of exposure, decreased to control levels by 40 min, was slightly below control values (an indication of overcompensation) during the next 3 hr, and was essentially the same as for the controls (recovery from overcompensation) during the remaining time. Although the overcompensation was clearly discernible, the differences of those mean values from those of the sham group at corresponding times were nonsignificant ($p>0.05$).

The results for the 40.1-cal/min group were more pronounced, ranging from 40.5 at the end of exposure to an overcompensation plateau of about 37.5 deg C 80 min later, which, unlike for the 27.7-cal/min group, persisted for the entire 5-hr period. Moreover, the differences in mean values at corresponding times were significant.

Still more pronounced were the results for the 68.2-cal/min group, which ranged from 42.4 deg C (a value near the upper survival limit for rats) to a minimum of about 37.0 deg C about 2 hr later. This level persisted for about 40 min, but was followed by slow recovery to control levels toward the end of the period.

The mean skin temperatures of the two lower-level RFR groups, which were higher than for the sham group at the end of exposure, returned to normal values (about 27 deg C) by 50 min and remained comparable to

those of the control group for the rest of the 5-hr period. The skin temperatures of the 68.2-cal/min group also returned to normal by 50 min, but continued to decrease, reaching a plateau of about 25.6 deg C by 100 min, which persisted for the remainder of the period.

Oxygen consumption by the sham group diminished slowly, during the 5 hr, from about 7.8 to 7.4 ml/min. The values for the 27.7-cal/min group were comparable. By contrast, the values for the 40.1-cal/min group were significantly lower initially (7.1 ml/min) and rose slowly but erratically to control values during the period. The 68.2-cal/min group yielded values that were significantly lower than for the other groups (a minimum of 6.3 ml/min during the first half of the period and a maximum of 7.0 ml/min near the end of the period).

Carbon dioxide production by the sham and 27.7-cal/min groups were comparable. Both yielded a mean of 6.6 ml/min initially and a maximum of 7.0 ml/min within 1 hr, followed by a slow diminution to 6.2 ml/min by the end of the 5 hr. The mean values for the 40.1-cal/min group varied widely around the 6.1-ml/min level, with no obvious upward or downward trend, thus becoming comparable to the level for the control group near the end of the period. The results for the 68.2-cal/min group were similar but at a lower level, about 5.7 ml/min, thus remaining lower than for the control group for the entire period.

Respiratory quotients, calculated from the results, were not altered by RFR exposure at any of the three levels and remained comparable to control values throughout the test period (no data presented).

The mean heart rate of the control group, about 450 beats/min initially, diminished slightly in linear fashion to about 430 beats/min at the end of the 5-hr test period. At all corresponding times during the test period, the mean heart rates of the 27.7-cal/min group were about 10 beats/min lower than those of the sham group, but the differences were not statistically significant. Mild bradycardia (to 410 beats/min) occurred within 20 min after exposure of the 40.1-cal/min group, with recovery within about 2 hr. Two of the 10 rats in this group exhibited irregular rhythms, whereas none in the sham and 27.7-cal/min groups did.

Pronounced bradycardia (to about 325 beats/min) developed within the first 20 min after exposure of the 68.2-cal/min group. The mean heart rate then increased approximately linearly, crossed mean control level about 2 hr after the minimum, rose to about 450 beats/min (slight tachycardia), and remained at about that level for the last 2 hr of the period. The abrupt bradycardia was accompanied by irregular heart rate and incomplete heart block in 7 of the 10 rats of this group, with complete recovery from the heart block within 60 min and no recurrence during the remainder of the period.

In their discussion, the authors indicated that the mean metabolic rate (based on oxygen comsumption) of the control rats, about 37 cal/min, was consistent with values in the literature. They also noted that the metabolic rates of the RFR-exposed rats were depressed even when the

colonic temperatures were elevated, findings similar to those obtained with mildly heat-stressed rats by Spielman and Lyman (1971), who also reported that the observed depressed metabolism was accompanied by bradycardia.

CRITIQUE: The finding of RFR-induced reductions of metabolic rate is supported by results of a subsequent study by Ho and Edwards (1977). In the latter study, mice were exposed for 30 min to 2.45-GHz RFR at SARs in the range 0-44 W/kg and their oxygen-consumption rates were monitored during, as well as before and after, exposure. SAR-dependent decreases of specific metabolic rate (SMR) were obtained for SARs of 10.4 W/kg and higher (and not for 5.5 W/kg and lower), and were ascribed to thermal stress.

On the other hand, no significant changes of heart rate were observed by Kaplan et al. (1971), Birenbaum et al. (1975), or Chou et al. (1980b) in intact rabbits exposed to far-field CW or pulsed RFR up to 80 mW/sq cm, at which the animals were obviously under severe thermal stress and tachycardia was detected. The much larger size of the rabbit (as well as its physiological differences from those of rats and mice) may account for the differences in findings.

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INFLUENCE OF ACUTE MICROWAVE RADIATION ON CARDIAC FUNCTION IN NORMAL AND MYOCARDIAL ISCHEMIC CATS
J. Appl. Physiol: Respiratory, Environmental, and Exercise Physiol., Vol. 50, No. 5, pp. 931-935 (1981a)

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AUTHOR ABSTRACT: Exposure of biological specimens to microwave radiation in vivo and in vitro has been reported to cause alterations in the cardiovascular system. In addition, microwave radiation may cause effects in damaged cardiac tissue that are not observed in normal tissue. In this study, we examined the influence of direct microwave irradiation (2.45 GHz, continuous wave) of the intact exposed heart on cardiac function in cats with and without myocardial ischemia. Myocardial ischemia was induced by occlusion of the left anterior descending coronary artery. In the sham-nonexposed and sham-plus-microwave exposed animals, the coronary artery was isolated but not occluded. The exposed hearts were either irradiated at a specific absorption rate (SAR) of 30 mW/g or not irradiated, and were monitored for 5 h.

At a SAR of 30 mW/g, the temperature of the exposed tissue increased at an initial rate of 0.43 deg C/min in dead cats. However, in live animals, no increases in aortic blood temperatures occurred during irradiation. Mean arterial blood pressure, cardiac output, heart rate, plasma and myocardial creatine phosphokinase, and S-T segment were not influenced by 5 h of microwave irradiation of the myocardium in cats with or without myocardial ischemia.

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Study Type: Cardiovascular Effects, Physiology and Biochemistry, Biorhythms; IN VIVO; CAT
Effect Type: RFR-induced alterations of cardiac function (blood pressure, output, rate, creatine-phosphokinase release, and EKG) of cats with and without surgically-induced myocardial ischemia
Frequency: 2.45 GHz
Modulation: CW
Power Density: Not measured
SAR: 30 W/kg

EXPOSURE CONDITIONS: After each cat was prepared and instrumented, and control values of various parameters were measured, a midsternal thoracotomy was performed, the pericardial sac was retracted, and the anterior surface of the heart was exposed to RFR for 5 hr with a dielectrically loaded waveguide 2 cm from the cardiac surface. The cat was on a heating pad at 37 deg C during the entire experiment.

OTHER INFORMATION: Heart SARs were calculated from initial slopes of time-temperature profiles measured with a thermistor probe inserted in

surgically-revealed and RFR-exposed hearts of cats euthanized and cooled to room temperature.

Each live cat studied was anesthetized, polyethylene catheters were inserted into the left external jugular and right femoral artery, a thermistor probe was inserted into the aortic arch via the right carotid artery to monitor arterial-blood temperature, and needle electrodes were inserted subcutaneously into the right forelimb and left hindlimb to monitor the scalar EKG. The trachea of the cat was intubated and intermittent positive-pressure respiration was established, after which the thoracotomy was performed to reveal the anterior surface of the heart.

Four groups of cats were studied. Myocardial ischemia (MI) was produced in two groups by ligating (occluding) the left anterior descending coronary artery 7-10 mm from the coronary ostium. For comparison, the coronary artery was isolated but not occluded (sham occlusion) in the other two groups. Following these surgical procedures, one MI group (11 cats) and one non-MI group (7 cats) were exposed at 30 W/kg, and the other MI group (8 cats) and non-MI group (7 cats) were sham-exposed. All exposures were for 5 hr.

Arterial blood samples (3 ml) were drawn (and replaced with saline) just prior to occlusion (or sham occlusion) and at hourly intervals of the exposure period, and the plasma was collected and assayed for protein concentration and creatine phosphokinase (CPK) activity. Also measured at these intervals were mean arterial blood pressure (MABP), cardiac output (CO), heart rate (HR), and the elevation of the S-T segment of the EKG.

The results for each group showed no statistically significant changes of MABP, CO, or HR with time. Moreover, the differences in these measurements among the four groups at corresponding times were nonsignificant ($p>0.05$), i.e., RFR exposure only, occlusion only, or the combination of these treatments had no significant effect. However, there were significant differences in elevation of the EKG S-T segment and in plasma CPK activity.

The elevation of the S-T segment of the EKG of all four groups was less than 0.05 mV initially and remained at that level for the RFR- and sham-exposed non-MI groups during the 5-hr exposure period. For both the RFR- and sham-exposed MI groups, the elevation rose to about 0.62 mV at 1 hr and gradually diminished to about 0.55 mV at 5 hr. At 2, 3, 4, and 5 hr, the values for the RFR group were lower than for the sham group, but the differences at corresponding times were not significant.

The CPK activity of all four groups were initially about 1 IU/mg of protein. For the RFR- and sham-exposed non-MI groups, the activity rose together to about 3.2 IU/mg at 5 hr, with no significant differences between the two groups. By contrast, the rises in activity of the RFR- and sham-exposed MI groups were again similar (with no significant

differences) but sharper, to about 8.8 IU/mg at 5 hr, and the differences between MI and non-MI groups were significant ($p<0.01$).

At the end of the 5-hr period, the hearts of the MI cats were excised, and the left ventricle was divided into ischemic-myocardium (IM) and nonischemic-myocardium (NIM) parts of the ischemic heart. Each such section was homogenized and centrifuged, and the supernatant was assayed for CPK activity. The results were expressed as the ratio of CPK activity in the IM part to the activity in the NIM part of each heart. Anatomically equivalent parts of the hearts of the non-MI cats were similarly treated. The mean ratios for the RFR- and sham-exposed nonischemic hearts were both about unity, and the difference between them was not significant. The mean ratios for the RFR- and sham-exposed ischemic hearts were both about 0.8, with the difference again nonsignificant. However, the differences in mean ratios between the ischemic and nonischemic hearts were significant ($p<0.05$).

CRITIQUE: The abstract stated that at an SAR of 30 W/kg, the heart temperature of the dead cats increased at an initial rate of 0.43 deg C, but that no increases were observed in the live cats. Presumably the difference was ascribable to the cooling of the heart by blood circulation in the latter. However, no time-temperature profile data were presented in the body of the paper.

An important objective of this study was to ascertain whether exposure to a clearly thermal level of RFR would exacerbate the pre-exposure presence of ischemia, with possible implications for qualitatively similar effects in humans that suffer from cardiac insufficiencies. The negative results do not support that hypothesis.

In their discussion, Galvin and McRee (1981a) noted that several groups of investigators, notably Levitina (1966), Lords et al. (1973), and Reed et al. (1977) had reported the occurrence of bradycardia in RFR-exposed isolated hearts of various animals. For example, Reed et al. (1977) had obtained bradycardia in isolated rat hearts exposed to 960-MHz CW RFR at about 1.5 W/kg, and found that the effect was absent when the parasympathetic and sympathetic nerves were simultaneously blocked with atropine and propranolol. Reed et al. (1977) had suggested that their results were due to an RFR-neuron interaction or RFR-synapse interaction by a mechanism other than generalized heating. However, the negative results in undamaged hearts obtained by Galvin and McRee (1981a) led them to surmise that the effects reported by Reed et al. (1977) in isolated hearts may have been due to the action of the RFR on the damaged nerve endings thereof.

The authors also noted that the absence of elevation of the S-T segments of nonischemic cats was contrary to the results of Paff et al. (1963), who reported changes in S and T deflections and shortening of the QT wave in the hearts of chicken embryos exposed to 24-GHz CW RFR at 74, 167, and 478 mW/sq cm.

Lastly, the authors indicated that plasma CPK activity is usually correlated with the degree of myocardial damage, which increases the release of CPK from the myocardium. This point was evident in the significantly higher CPK activities in the ischemic cats as compared with the nonischemic cats. The absence of differences in CPK activity between RFR- and sham exposure in either case indicates that RFR (at the level used) does not influence the release of this enzyme.

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BIOCHEMISTRY
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CARDIOVASCULAR
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Hamburger, S., J.N. Logue, and P.M. Silverman
OCCUPATIONAL EXPOSURE TO NON-IONIZING RADIATION AND AN ASSOCIATION WITH HEART DISEASE: AN EXPLORATORY STUDY
J. Chron. Dis., Vol. 36, No. 11, pp. 791-802 (1983)

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AUTHOR ABSTRACT: Exploratory analyses for dose-related exposure to non-ionizing radiation and adverse health effects among male physical therapists were done from a mail questionnaire survey. The cohort consisted of 3004 respondents who were stratified into subgroups according to exposure across and within the various types of non-ionizing radiation energy emitted from diathermy equipment. The radiation modalities considered were ultrasound, microwave, shortwave, and infrared. An association between heart disease and exposure to shortwave radiation was the only consistently significant finding when high and low exposure groups were compared.

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Study Type: Human Studies, Cardiovascular Effects; IN VIVO; HUMAN
Effect Type: Epidemiologic study of possible effects of "microwave" and "shortwave" RFR, and other forms of radiation on physical therapists who used diathermy or other treatment modalities occupationally
Frequency: 2.45 GHz (microwave); 27 MHz (shortwave)
Modulation: Not specified
Power Density: 0.08-1.3 mW/sq cm estimated range for microwave; 0.04-16.6 mW/sq cm estimated free-space-equivalent range for shortwave
SAR: Not determined

EXPOSURE CONDITIONS: A survey of 9 diathermy units, 3 microwave and 6 shortwave, was conducted by the Division of Electronic Products of the Bureau of Radiological Health (BRH), to quantify occupational exposure of diathermy operators. This survey yielded, for the microwave units, means of 0.65 and 0.71 mW/sq cm at the eyes and waist, respectively, and a range of 0.08-1.30 mW/sq cm. For the shortwave units, the mean free-space-equivalent power densities of the E- and H- fields at the eyes were 1.21 and 1.06 mW/sq cm, respectively; the corresponding values at the waist were 4.05 and 2.45 mW/sq cm; and the range of combined equivalent power density was 0.04-16.58 mW/sq cm.

Among the results of a questionnaire sent to diathermy operators was that for the under-35 age group of operators, the mean time spent within 3 ft of the equipment was 2.4 min per treatment with microwave diathermy and 2.7 min per treatment with shortwave diathermy. (Typically, the operator initiates the treatment, then leaves to attend to other matters for the remainder of the treatment.) Other factors considered were frequency of treatments, years of work experience, and the use of infrared and ultrasound diathermy.

OTHER INFORMATION: Physical therapists are known to use various diathermy modalities (microwave, shortwave, infrared, and ultrasound equipment) in the course of treating patients. This epidemiologic study took the form of statistical analyses of responses by male members of the American Physical Therapy Association (APTA) to a questionnaire. A brief measurement survey by BRH provided the estimates of the RFR-exposure levels given above.

A study population of approximately 5300 from the APTA Roster was identified by given (first) name as male physical therapists. A pretest of 200 subjects was conducted to: determine the effectiveness of the questionnaire, project a response rate, and characterize the group with regard to demographic, health, and occupational attributes. After making adjustments based on the results of the pretest, the full-scale study was initiated.

Although other factors were considered in the questionnaire, emphasis was placed on those health experiences reported in the literature as being associated with low-level exposure to radiofrequency/microwave electromagnetic radiation. The responses requested from each subject included the occupational history of diathermy utilization by length of employment in each position held since entering the clinical affiliation and the number of treatments of each modality administered per typical work week. Three mailings were made, to reduce the level of nonresponses. The final population sample was 3004 responses from a total of 5187 therapists solicited.

The four diathermy modalities were coded as U (ultrasound), I (infrared), M (microwave), and S (shortwave). Based on use of single modalities or combinations thereof, 15 independent subgroups plus 1 group with no exposure were constructed. The designations of these groups and the number of subjects in each were U (208), I (4), M (2), S (3), IU (60), MU (114), SU (509), IM (1), IS (11), MS (2), IMU (61), ISU (418), MSU (388), IMS (5), IMSU (1097), and "none" (121). Small sample size in some groups required stratification in a different manner for more meaningful statistical tests. It was reasoned that since ultrasound produces biological effects only by direct contact, this modality was inert as far as the therapists were concerned. This then yielded 9 new subgroups: MU (116)=MU+M, SU (512)=SU+S, IU (64)=IU+I, MSU (390)=MSU+MS, ISU (429)=ISU+IS, "other" (67)=IM+IMS+IMU, U (208)=U, IMSU (1097)=IMSU, and "none" (121)="none". (This detailed information of group sizes was not provided in the text, but was derived from the tabular footnotes.)

Age was closely related to cumulative exposure and is always related to certain pathologic, psychologic, and physiologic processes, so age adjustment was done by dividing each subgroup into an under-35 and a 35+ group. The 35+ group was further stratified into 3 subgroups: 35-44, 45-54, and 55+. Groups were dichotomized into high- and low-exposure categories using mean length of employment (less than 14 yr and 14 yr or more), mean frequency of treatments administered per week (less than 17 and 17 or more), and jointly by employment duration and treatment frequency.

Subgroup characterization was conducted before analysis for potential effects of microwave and/or shortwave RFR. The hypothesis that ultrasound and infrared do not contribute to potential effects was assessed as follows. The U (ultrasound only) subgroup was compared with the "none" subgroup to test for ultrasound effects, and the SU group was compared with ISU group to test for infrared effects. There were no statistically significant differences for any of the disorders. This was interpreted to indicate that neither ultrasound nor infrared alone contributed to any of the biological effects under consideration.

Tables were provided of selected characteristics of respondents (e.g., age, race, marital status, present work setting) by the 9 exposure subgroups. Likewise, prevalence of reported conditions by subgroups were tabulated. These included conditions such as blood disorder, cataracts, diabetes, heart disease, nervous breakdown, and others. The authors stated that, "for the entire cohort, the reported prevalence rates are below the population rate in all instances. Although the rates vary by subgroups for each condition, no one subgroup appears to exhibit markedly higher rates relative to total rates."

A new approach was tried. New subgroups were formed for microwave, shortwave, and joint microwave/shortwave exposure, and further divided into high- and low-exposure groups. A respondent with any exposure to microwave was included in the microwave group. Similar definitions were used for shortwave and for joint exposure. Thus, the subgroups were not mutually exclusive. This produced the anomalous situation where Table 6 of the paper presented "prevalence of conditions by frequency of exposure: respondents 35+ in high and low exposure categories," which yielded a total of 3079 subjects, whereas Table 5 indicated a total of 1373 microwave- or shortwave-exposed subjects in the 35+ category. Clearly there was considerable double-counting of subjects in this new approach.

Contingency tables were constructed for internal comparisons across subgroups. That is, for the three types of exposure (microwave only, shortwave only, and joint microwave/shortwave) and for the three high- vs low-exposure situations (by frequency of treatments/wk, length of employment, and combination thereof), 3x3 or nine separate contingency tables of condition vs modality were constructed. This was done for each of the 10 medical conditions, for a total of 90 contingency tables. The values in each table were tested with the chi-squared test for homogeneity. Some significant results were obtained. However, when age groups were combined by using the Mantel-Haenszel method (stratification by age group), which permits correction for age-related effects, most of those results became nonsignificant.

Odds ratios were also calculated for each contingency table, and confidence intervals were calculated for the odds ratios that remained statistically significant after age adjustment. Heart disease was the only condition that remained statistically significant in this new approach. Interestingly, for the 3x3 types of exposure-vs-high/low situations (described above), only four were significant. These were:

microwave x frequency of treatments/wk, shortwave x combined frequency of treatments/wk and length of employment, and joint microwave/shortwave x frequency of treatments/wk. The other five situations were not significant at the 5% level, i.e., all three of the cases where length of employment alone was a criterion of high- vs low exposure, the case for joint microwave/shortwave exposure x combined treatment frequency and employment duration, and the case for microwave exposure x combined treatment frequency and employment duration.

It should be noted that of the 90 contingency tables constructed and tested, only four were significant at the 5% level, a finding that is no better than chance.

None of the other nine medical conditions (including neoplasms, i.e., "cancer") was statistically significant.

The authors also assessed the possible confounding effects of diagnostic X-ray and diathermy treatments on individual respondents. After age adjustment, no conditions related to such treatments were found to be statistically significant.

CRITIQUE: This study most likely will be widely cited as proof that exposure to microwave/shortwave RFR causes heart disease. However, close examination of the paper does not provide convincing evidence that this is so. First, the paper illustrates well the problems associated with attempts to uncover causal relationships between a purported health-effects agent (RFR in this case) and medical conditions in an identified population by using the results of a mailed, self-administered questionnaire. The response rate to the mailings was 58%, so 42%, or 2183 persons did not respond. No mention was made of any attempt to contact a sample of nonrespondents by telephone or in person, to endeavor to characterize them as a group. (Statistical techniques exist to correct for bias in a large nonrespondent group.) Therefore, the 58% that did respond were self-selected in the sense that many of them may have responded because they had medical conditions and were curious about how such conditions may have arisen.

The difficulty in extrapolating from a pilot study of 3 microwave plus 6 shortwave RFR exposure situations to a cohort of 3004 respondents who had used diathermy equipment for up to 20 or more years is also not strong evidence of actual RFR exposure conditions.

As discussed above, a straightforward analysis of prevalence rates by subgroup for all reported medical conditions yielded nonsignificant differences between subgroups and the prevalence rates for the entire cohort. Furthermore, the reported prevalence rates for the entire cohort were below population rates in all instances (the "healthy worker" effect, plus the higher socioeconomic group effect--therapists are better off as a group than the general population). It was only after a regrouping of subjects into non-mutually-exclusive categories (i.e., double-counting of subjects in more than one category) that the analysis showed statistical significance, and then for only one medical condition

out of 10 tested. For blood disorder, cataracts, diabetes, endocrine disorder, hearing disorder, high blood pressure, nervous breakdown, and "other" (including cancer), neither of the two analytical approaches showed any statistically significant relationship between the condition and diathermy usage.

The major finding, that there is a statistical link between one aspect of occupational exposure (self-reported recollection of frequency or number of treatments/week, but not employment duration) and "heart disease" (defined here as "disorders of conduction/rhythm and ischemia, and 'others'," a combination of many symptoms of various etiologies) for shortwave and microwave radiation (the latter downplayed), but not joint shortwave/microwave exposure, is at best weak. If shortwave exposure, as defined, is a causal agent, but joint shortwave/microwave exposure is not, it could be inferred that microwave exposure protects against the possible adverse effects of shortwave exposure with respect to heart disease, a most unlikely conclusion. Furthermore, length of employment, which normally would be considered a factor in "cumulative exposure," showed no statistically significant role.

For heart disease, cigarette smoking is identified as a major risk factor and is widely known to be a strong predictor of heart disease in an aging population such as the 35+ group, for which the relationship with shortwave exposure was claimed in the present study. Inexplicably (but acknowledged by the authors), smoking history was not included in the questionnaire. The failure to consider this major biasing factor does not inspire great confidence in the sole positive result of this study.

Finally, the authors stated: "To our knowledge, no epidemiologic studies of cardiovascular effects associated with shortwave exposure have been reported." Overall, their study does not provide strong evidence of such an association either.

REFERENCES: None

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CARDIOVASCULAR
CHRONIC
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Hamrick, P.E. and D.I. McRee
THE EFFECT OF 2450 MHZ MICROWAVE IRRADIATION ON THE HEART RATE OF EMBRYONIC QUAIL
Health Phys., Vol. 38, pp. 261-268 (1980)

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AUTHOR ABSTRACT: Japanese quail (Coturnix coturnix japonica) embryos 8-13 days old were exposed to pulsed and CW 2450 MHz microwave radiation at specific absorption rates of 0.3-30 mW/g to investigate the effect of the radiation on heart rate. Although non-thermal effects (in particular bradycardia) caused by exposure to microwave radiation have been reported, no effects on heart rate were detected in this study which could not be attributed to temperature changes.

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Study Type: Cardiovascular Effects, Biorhythms; IN VIVO; QUAIL
Effect Type: Effects of CW and pulsed RFR on embryonic heart rates
Frequency: 2.45 GHz
Modulation: CW and 10-microsecond pulses at 10-50 pps (0.0001-0.0005 duty cycle)
Power Density: 0.25-25 mW/sq cm (estimated)
SAR: 0.3-30 W/kg

EXPOSURE CONDITIONS: The exposure chamber consisted of a vertical section of waveguide fed from below by either a CW- or pulsed-RFR source. Circulating water 3 cm in height above a partition across the waveguide served as a temperature-controlled bath in which eggs, with the tops of the shells removed (to permit insertion of EKG leads), were individually suspended with the embryo near the surface of the water. The partition was a quarter-wavelength section to match the impedance of the water to the air below. Most exposures at 15 W/kg or lower were for 5-10 min, those at higher than 15 W/kg were for 2 min or less, and a few at lower than 1.5 W/kg were for 1 hr.

OTHER INFORMATION: Measurements of forward and reflected powers on the source side of the partition indicated that less than 3% of the forward power was reflected. The SAR at the embryo location (water surface on the axis of the waveguide) was calculated (from Eq. 2 of the paper) for a specified value of the net input (forward-minus-reflected) power, the cross sectional area of the waveguide, an assumed attenuation factor of 0.59/cm at 2.45 GHz in tissues of high water content (penetration depth of 1.7 cm), and an assumed density of 1.05 g/cu cm. The area of the guide was not given, but the system was similar to that of Chou and Guy (1975), who used Type WR-284 waveguide, which has an internal cross section of 24.6 sq cm. The SAR was also determined from the slope of the cooling curve obtained by heating an egg above a given equilibrium temperature with RFR and measuring the temperature vs time while the egg cooled to equilibrium with the RFR off (McRee and Hamrick, 1977).

For Japanese quail, the normal incubation time is 16-17 days. Embryos 8-13 days old were used. Prior to exposure, the eggs were maintained at optimum incubation temperature and relative humidity, 37.5 +/- 0.30 deg C and 60 +/- 5%, respectively. Prior to exposure of each egg, the top of the shell was removed and non-perturbing carbon-loaded Teflon leads (Chou and Guy, 1979a) were inserted for measuring heart rate. Heart rates were determined just before exposure, just after beginning exposure, near the end of exposure, and just after completion of exposure.

A plot of mean heart rate vs temperature for 9-day-old embryos (Fig. 3 of the paper) showed 203 beats/min at 35 deg C and 211 beats/min at 36 deg C; from 36 deg C, the mean rate increased nearly linearly with embryo temperature to 279 beats/min at 39 deg C.

The results for 9-day-old embryos exposed to CW RFR at SARs in the range 0.3-30 W/kg were displayed, together with control values, in Table 1. At 30 W/kg, the mean heart rate ranged from 201 beats/min at 35.0 deg C to 232 beats/min at 38.0 deg C. The control values at this SAR ranged from 204 beats/min at 35.0 deg C to 232 beats/min at 38.0 deg C. By t-test, the differences of means at corresponding temperatures were not significant ($p>0.05$). At 15, 6, or 3 W/kg, the differences between exposed and control embryos at corresponding temperatures were also nonsignificant.

Embryos 8, 11, and 12 days old were also exposed to CW RFR. There were no significant differences between controls and embryos exposed at 30 or 15 W/kg (no data presented).

The mean heart rate vs temperature for 9-day-old embryos exposed to 10-microsecond pulses at SARs of 0.3, 1.5, and 3 W/kg were presented in Table 2. The pulse repetition rates used were 10, 13, 16, 20, 25, 30, and 50 pps, selected to span the range for which calcium efflux from chick brains was reported by Bawin et al. (1975). Presumably the input power was varied appropriately to obtain the stated SARs. Again there were no significant differences between exposed embryos and their corresponding controls. This was also true for 8- and 11-day embryos exposed to 10-microsecond pulses, 1000 pps, at 3 and 15 W/kg (no data presented).

CRITIQUE: Although the authors emphasized the importance of accurate dosimetry, their discussion of the subject is ambiguous. They stated: "Various exposure power levels from 0.1 to 10 W were employed giving estimated specific absorption rates ranging from 0.3 to 30 mW/g at the position of the embryo." This statement would imply an SAR of 3 W/kg per W of input power. However, the SAR calculated from the slope of the cooling curve for 5 W input was stated to be 37.7 W/kg, which yields 7.54 W/kg per W. (Incidentally, use of 0.011 deg C/sec for the slope of the cooling curve and 0.8 cal/deg C for the specific heat of the egg, both given in the paper, yields 36.8 not 37.7 W/kg, but the difference is relatively small.)

The remainder of the discussion of SAR estimations is also unclear. Specifically, the authors indicated that the SAR at the location of the embryo can be estimated from Eq. 2, but they presented an estimate of the power density instead. Performing the calculation with assumptions of 4W of input power and a waveguide cross-sectional area of 24.6 sq cm yields 5.31 W/kg for the SAR averaged over the waveguide area or 10.62 W/kg at the location of the embryo (twice the average). The latter on-axis value corresponds to 2.66 W/kg per W, which rounds to 3 W/kg per W but is only about 35% of the value from the cooling curve.

The authors also stated that integration of Eq. 2 over the egg yielded a mean SAR of 24.6 W/kg for 5 W input (an SAR that could not be checked because the integration limits were not given). The corresponding on-axis value, 49.2 W/kg, which is equivalent to 9.84 W/kg per W, is about 30% higher than the value from the cooling curve. Thus, neither estimate from Eq. 2 (directly or by integration) provided numerical confirmation of the cooling-curve result.

Another obscure point was whether the plot of mean heart rate vs temperature shown in Fig. 3 was obtained by heating the embryos with RFR or by a non-RFR method. The results for the control groups of embryos at various temperatures, displayed in Tables 1 and 2, would imply the latter. Still another unclear aspect was how the heart rates measured just before and after the start of exposure and just before and after the end of exposure were treated arithmetically to yield the stated results.

Although the findings of this investigation were negative, i.e., no effects on heart rate could be ascribed to either CW or pulsed RFR, these conclusions are weakened by the uncertainties indicated above.

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EXPOSURE OF JAPANESE QUAIL EMBRYOS TO 2.45-GHZ MICROWAVE RADIATION DURING DEVELOPMENT
Radiat. Res., Vol. 71, No. 2, pp. 355-366 (1977)

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BIORHYTHM
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Presman, A.S. and N.A. Levitina
NONTHERMAL ACTION OF MICROWAVES ON THE RHYTHM OF CARDIAC CONTRACTIONS IN ANIMALS--REP. II: INVESTIGATION OF THE ACTION OF IMPULSE MICROWAVES
Bull. Exp. Biol. Med., Vol. 53, No. 2, pp. 154-157 (1963b)
(Engl. Transl. of pp. 39-43 of 1962b Russ. publ.)

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AUTHOR ABSTRACT: In investigations we performed earlier (Presman and Levitina, 1962a), it was established that irradiation of different parts of the body of a rabbit with continuous microwaves (wavelength 12.5 cm) of nonthermal intensity (7-12 mW/sq cm) causes a "chronotropic effect" --a change in the sinus rhythm. The effect is rapidly reversible; it is manifested during the irradiation and for a short time afterward.

We considered it of unquestionable interest to carry out analogous investigations with impulse microwaves, whose biological activity is sometimes apparently different from the action of the continuous type. The thermal effect of impulse microwaves is determined by their mean intensity, which is considerably lower than the impulse intensity (proportionality coefficient--product of the duration of the impulse times the frequency of the impulses). Thus, at a nonthermogenic mean intensity, the impulse intensity will be considerably higher.

In this report, we describe an experiment involving investigation of the sinus rhythm in rabbits subsequent to irradiation of different portions of their bodies with impulse microwaves. The results of these experiments are compared to corresponding data, obtained earlier from trials employing irradiation with continuous microwaves.

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Study Type: Cardiovascular Effects, Biorhythms; IN VIVO; RABBIT
Effect Type: Alterations of heart-sinus rate by pulsed RFR
Frequency: 3 GHz (wavelength 10 cm)
Modulation: 1-microsecond pulses at 700 pps (0.0007 duty cycle)
Power Density: 3-5 mW/sq cm Av; 4.3-7.1 W/ sq cm Pk
SAR: Not measured

EXPOSURE CONDITIONS: For exposures of the dorsal aspects of the head only and back only and of the entire dorsal surface, each rabbit in a box lined with RFR-absorbent material was placed below a horn powered from a pulse generator. For exposures of the ventral aspects of the head only and belly only and of the entire ventral surface, each rabbit was placed on an RFR-transparent plate of foam polystyrene and exposed with the horn from below. RFR-absorbent plates were used to cover the areas not to be exposed. The subjects for all experiments were 8 male rabbits, each weighing 3-4 kg. Each rabbit was exposed for two 20-min periods at each aspect. For control data, the same rabbits were sham-exposed once prior to, and once after, the series of RFR exposures.

OTHER INFORMATION: The EKGs were recorded from plate electrodes. Each rabbit was placed below or above the horn 15 min before each EKG-recording session, which consisted of 20 min of RFR- or sham exposure and 10 min each of pre- and post-exposure. During each session, the EKG of each rabbit was recorded for 20 seconds at 1-min intervals and the heart rates were derived therefrom.

The data from these experiments were treated in the same manner as those obtained with rabbits exposed to 2.40-GHz CW RFR (Presman and Levitina, 1962a): For each RFR-exposure session, all of the heart rates of the rabbits during the 10-min pre-exposure period were averaged. Also, each set of 16 heart rates (2 per rabbit) for each 1-min interval of the 20-min RFR-exposure and 10-min post-exposure periods was averaged, and the deviation of this mean from the pre-exposure mean was calculated. The data from the sham-exposure sessions were similarly treated, and the relative change in rate, i.e., the difference of mean change in rate between the RFR and sham sessions at each corresponding 1-min interval, was calculated and plotted vs elapsed time from the start of exposure. The values of this quantity (positive for tachycardia, negative for bradycardia) were deemed significant if they exceeded twice the mean relative change in rate.

Exposure of the entire dorsal surface yielded no significant tachycardia or bradycardia during exposure. However, significant tachycardia was observed during the first half of the post-exposure period, changing to significant bradycardia toward the end of that period. By contrast, exposure of the dorsal aspect of the head and of the back produced significant tachycardia during the exposure period, with the former yielding the greater effect. The tachycardia increased to peak values at about 5 min post-exposure and declined to nonsignificant values by the end of that period.

Bradycardia occurred during exposure for all three ventral aspects, which persisted to the end of the exposure period, and was followed by returns toward normal heart rates during the first half of the post-exposure period. It was most pronounced and was manifested earliest for exposure of the head only.

In their previous paper on the effects of CW RFR (Presman and Levitina, 1962a), the authors stated: "We have tried to take into account changes of cardiac rhythm which were no greater than the random changes. For this purpose, we treated the experimental results as follows: a) in all the experiments with a given type of irradiation, we calculated the percentage of cases in which the rhythm was slowed or speeded, both during the treatment and subsequently, as compared with the mean value before treatment; b) similar calculations were made for the control experiments; c) we then calculated the difference between the percentages of cases of change in the experimental and control groups; the differences were taken as significant when they exceeded twice the mean error of the difference of the percentages; d) for a quantitative and qualitative description of the effect on the heart rate of irradiating different parts of the body we used the ratio K, which we

called the coefficient of the chronotropic effect.

$$K = (100+mi)/(100+md),$$

where mi and md are the respective significant values of changes in the percentage of cases with rates increased or decreased from the control values (with due account of sign). Insignificant values were recorded as zero." Thus, values of $K>1$ and $K<1$ signified tachycardia and bradycardia, respectively, and $K=1$ no effect. This statistical procedure was also used for the data obtained with pulsed RFR.

For dorsal exposure of the head only, the values of mi and md were both 23% during the exposure period and 31% during the post-exposure period, which yielded $K=1.6$ and $K=1.9$, respectively, thus signifying the occurrence of tachycardia during exposure and its persistence during post-exposure. For dorsal exposure of the back only, the values of mi and md were both zero during exposure but mi was 16% and md was 21% during post-exposure, yielding $K=1.47$. For exposure of the entire dorsal surface, $K=1$ for both the exposure and the post-exposure periods.

For all three ventral-exposure aspects, $K=1$ for the post-exposure period. However, the values of K were 0.89, 0.75, and 0.61 for ventral exposure of the entire surface, the stomach only, and the head only, respectively, all signifying the occurrence of bradycardia during exposure that did not persist after exposure.

The chronotropic effects of pulsed RFR were compared with those for rabbits exposed to 2.40-GHz CW RFR at 7-12 mW/sq cm (Presman and Levitina, 1962a). For the latter, tachycardia was observed during and after dorsal exposure of the head ($K=1.3$ and 1.42, respectively), results that were qualitatively similar to those for the pulsed RFR. Exposure of the entire dorsal surface to either type of RFR yielded $K=1$. Exposure of the dorsal aspect of the back to the CW RFR yielded $K=1$ for the exposure period, as did the pulsed RFR, but the CW RFR yielded post-exposure bradycardia ($K=0.76$) whereas the pulsed RFR produced post-exposure tachycardia ($K=1.47$).

Ventral exposure of the head and of the stomach and exposure of the entire ventral surface to the CW RFR all yielded bradycardia ($K=0.76$, 0.73, and 0.67, respectively) during the exposure period, results that were qualitatively similar to those for the pulsed RFR. Also, $K=1$ post-exposure for exposure of the head to either type of RFR. However, post-exposure bradycardia ($K= 0.91$ and 0.67) was evident for exposure of the stomach or the entire ventral surface to the CW but not the pulsed RFR.

CRITIQUE: The authors summarized the comparison of results for the CW and pulsed RFR as follows: "Above all, it must be noted that the effect of impulse irradiation was basically more manifest than the effect of continuous irradiation, despite the fact that the mean intensity with impulse irradiation was approximately half as great. This is not

difficult to understand if it is taken into consideration that the impulse intensity exceeded the mean by 1400 times." However, the comparisons presented above do not provide strong support for this conclusion. Specifically, the tachycardial values (K>1) for the dorsal exposures to the pulsed RFR were larger than (or equal to) the corresponding values for the CW RFR, an indication that the former was more effective than the latter. However, the bradycardial values (K<1) for the ventral exposures to the CW RFR were smaller than (or equal to) those for the pulsed RFR, indicating the greater effectiveness of the CW RFR.

The statistical treatment of the results is obscure. No actual data were presented, only relative differences of means, rendering it difficult to estimate variabilities among the rabbits or the time variations of the heart-beat rate of each rabbit in the absence of RFR.

A more fundamental question is whether the use of metal electrodes during RFR exposure introduced artifacts of sufficient magnitude to render the results meaningless because the presence of such electrodes and their conductive leads can alter the local fields significantly. This possibility is supported by subsequent investigations directed toward reproducing the results of these authors, notably the studies of Kaplan et al. (1971) and Birenbaum et al. (1975) with 2.4-GHz CW RFR, who used surgical needles subcutaneously implanted in regions well shielded from the RFR to record the EKGs, and of Chou et al. (1980b) with 2.45-GHz CW and pulsed RFR, who used nonperturbing electrodes (Chou and Guy, 1979a). All of these investigators found that exposure of rabbits to at least 80 mW/sq cm was necessary to affect their heart rates significantly.

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PRESMAN
LEVITINA

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BIORHYTHM
CARDIOVASCULAR
CW
EKG
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APPENDIX B

CUMULATIVE LIST OF ANALYSES BY TOPIC

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