



Assessment of the Possible
Health Effects of Ground Wave
Emergency Network

Assessment of the Possible Health Effects of Ground Wave Emergency Network

Committee on Assessment of the Possible Health
Effects of Ground Wave Emergency Network (GWEN),
Board on Radiation Effects Research, National
Research Council

ISBN: 0-309-59531-2, 182 pages, 8.5 x 11, (1993)

**This PDF is available from the National Academies Press at:
<http://www.nap.edu/catalog/2046.html>**

Visit the [National Academies Press](http://www.nap.edu) online, the authoritative source for all books from the [National Academy of Sciences](http://www.nap.edu), the [National Academy of Engineering](http://www.nap.edu), the [Institute of Medicine](http://www.nap.edu), and the [National Research Council](http://www.nap.edu):

- Download hundreds of free books in PDF
- Read thousands of books online for free
- Explore our innovative research tools – try the “[Research Dashboard](#)” now!
- [Sign up](#) to be notified when new books are published
- Purchase printed books and selected PDF files

Thank you for downloading this PDF. If you have comments, questions or just want more information about the books published by the National Academies Press, you may contact our customer service department toll-free at 888-624-8373, [visit us online](#), or send an email to feedback@nap.edu.

This book plus thousands more are available at <http://www.nap.edu>.

Copyright © National Academy of Sciences. All rights reserved.

Unless otherwise indicated, all materials in this PDF File are copyrighted by the National Academy of Sciences. Distribution, posting, or copying is strictly prohibited without written permission of the National Academies Press. [Request reprint permission for this book](#).

Assessment of the Possible Health Effects of Ground Wave Emergency Network

Committee on Assessment of the Possible Health Effects of Ground Wave Emergency Network
Board on Radiation Effects Research
Commission on Life Sciences
National Research Council

NATIONAL ACADEMY PRESS
Washington, D.C. 1993

NATIONAL ACADEMY PRESS 2101 CONSTITUTION AVENUE, N.W., WASHINGTON, D.C. 20418

The project that is the subject of this report was approved by the Governing Board of the National Research Council, whose members are drawn from the councils of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. The members of the committee responsible for the report were chosen for their special competencies and with regard for appropriate balance.

This report has been reviewed by a group other than the authors according to procedures approved by a Report Review Committee consisting of the members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

The National Academy of Sciences is a private, nonprofit, self-perpetuating society of distinguished scholars engaged in scientific and engineering research, dedicated to the furtherance of science and technology and to their use for the general welfare. Upon the authority of the charter granted to it by the Congress in 1863, the Academy has a mandate that requires it to advise the federal government on scientific and technical matters. Dr. Frank Press is president of the National Academy of Sciences.

The National Academy of Engineering was established in 1964, under the charter of the National Academy of Sciences, as a parallel organization of outstanding engineers. It is autonomous in its administration and in the selection of its members, sharing with the National Academy of Sciences the responsibility for advising the federal government. The National Academy of Engineering also sponsors engineering programs aimed at meeting national needs, encourages education and research, and recognizes the superior achievements of engineers. Dr. Robert M. White is the president of the National Academy of Engineering.

The Institute of Medicine was established in 1970 by the National Academy of Sciences to secure the services of eminent members of appropriate professions in the examination of policy matters pertaining to the health of the public. The Institute acts under the responsibility given to the National Academy of Sciences by its congressional charter to be an advisor to the federal government and, upon its own initiative, to identify issues of medical care, research, and education. Dr. Kenneth I. Shine is president of the Institute.

The National Research Council was organized by the National Academy of Sciences in 1916 to associate the broad community of science and technology with the Academy's purposes of furthering knowledge and advising the federal government. Functioning in accordance with general policies determined by the Academy, the Council has become the principal operating agency of both the National Academy of Sciences and the National Academy of Engineering in providing services to the government, the public, and the scientific and engineering communities. The Council is administered jointly by both Academies and the Institute of Medicine. Dr. Frank Press and Dr. Robert M. White are chairman and vice chairman, respectively, of the National Research Council.

This project was prepared under contract No. F49620-90-0012 between the National Academy of Sciences and the Department of the Air Force.

Library of Congress Catalog Card Number 92-60706
International Standard Book Number 0-309-04777-3

B-616
Limited number of copies available from the Board on Radiation Effects Research, Commission on Life Sciences, National Research Council, 2101 Constitution Avenue, N.W., Washington, D.C. 20418
Copyright 1993 by the National Academy of Sciences. All rights reserved.

Printed in the United States of America

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Committee on Assessment of the Possible Health Effects of Ground Wave Emergency Network

- THOMAS S. TENFORDE** (*Chairman*), Battelle Pacific Northwest Laboratories, Richland, Washington
CLAUDIO J. CONTI, M. D. Anderson Cancer Center, Smithville, Texas
H. KEITH FLORIG, Resources for the Future, Washington, D.C.
OM P. GANDHI, University of Utah, Salt Lake City, Utah
MICHAEL E. GINEVAN, Department of Energy, Washington, D.C.
GEORGE H. HARRISON, University of Maryland Medical School, Baltimore, Maryland
MAUREEN M. HENDERSON, Fred Hutchinson Cancer Research Center, University of Washington, Seattle, Washington
J. ROSS MACDONALD, University of North Carolina, Chapel Hill, North Carolina
REGINA M. SANTELLA, Columbia University, New York, New York
JAN A. J. STOLWIJK, Yale University, New Haven, Connecticut
HOWARD WACHTEL, University of Colorado, Boulder, Colorado

National Research Council Staff

- D. DENNIS MAHLUM**, Study Director until February 28, 1993
LARRY H. TOBUREN, Study Director as of February 1, 1993
DORIS E. TAYLOR, Administrative Assistant
NORMAN GROSSBLATT, Editor

Sponsor's Project Offices

- Major Robert Veal, U.S. Air Force
Lt. Col. Stephen Martin, U.S. Air Force

Board on Radiation Effects Research

- WARREN K. SINCLAIR** (Chairman), National Council on Radiation Protection & Measurements (ret.), Bethesda, Maryland
DOUGLAS GRAHN, Argonne National Laboratory (ret.), Madison, Indiana
ERIC J. HALL, Columbia University, New York, New York
MAUREEN M. HENDERSON, Fred Hutchinson Cancer Research Center, University of Washington, Seattle, Washington
LEONARD S. LERMAN, Massachusetts Institute of Technology, Cambridge, Massachusetts
JOHN B. LITTLE, Harvard University, Cambridge, Massachusetts
JONATHAN M. SAMET, New Mexico Tumor Registry, University of New Mexico Albuquerque, New Mexico
THOMAS S. TENFORDE, Battelle Pacific Northwest Laboratories, Richland, Washington
ARTHUR C. UPTON, New York University Medical Center (ret.), New York, New York

National Research Council Staff

- CHARLES W. EDINGTON**, Director
D. DENNIS MAHLUM, Senior Program Officer until February 28, 1993
EVAN B. DOUPLE, Senior Program Officer
LARRY H. TOBUREN, Senior Program Officer as of February 1, 1993
CATHERINE S. BERKLEY, Administrative Associate
MAURITA A. DOW, Project Assistant
DORIS E. TAYLOR, Administrative Assistant

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Commission on Life Sciences

- THOMAS D. POLLARD** (*Chairman*), Johns Hopkins Medical School, Baltimore, Maryland
BRUCE M. ALBERTS, University of California, San Francisco, California
BRUCE N. AMES, University of California, Berkeley, California
J. MICHAEL BISHOP, University of California Medical Center, San Francisco, California
DAVID BOTSTEIN, Stanford University School of Medicine, Stanford, California
MICHAEL T. CLEGG, University of California, Riverside, California
GLENN A. CROSBY, Washington State University, Pullman, Washington
LEROY E. HOOD, University of Washington, Seattle, Washington
MARIAN E. KOSHLAND, University of California, Berkeley, California
RICHARD E. LENSKI, University of Oxford, Oxford, United Kingdom
STEVEN P. PAKES, Southwestern Medical Center, Dallas, Texas
EMIL A. PFITZER, Hoffmann-La Roche Inc., Nutley, New Jersey
MALCOLM C. PIKE, USC School of Medicine, Los Angeles, California
PAUL G. RISSER, Miami University, Oxford, Ohio
JONATHAN M. SAMET, New Mexico Tumor Registry, Albuquerque, New Mexico
HAROLD M. SCHMECK, JR., Armonk, New York
CARLA J. SHATZ, University of California, Berkeley, California
SUSAN S. TAYLOR, University of California at San Diego, La Jolla, California
P. ROY VAGELOS, Merck & Co., Inc., Whitehouse Station, New Jersey
TORSTEN N. WIESEL, Rockefeller University, New York, New York

National Research Council Staff

- ALVIN G. LAZEN**, Acting Executive Director

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Preface

This report was prepared in response to a request from the U.S. Air Force for the National Academy of Sciences (NAS) to review the potential health effects of electromagnetic fields emitted by the Ground Wave Emergency Network (GWEN). This system was designed to protect strategic communication capabilities in the event of a high-altitude nuclear detonation. The GWEN communication system broadcasts UHF messages (225-400 MHz) that are transmitted at low altitudes by a network of low-frequency (150-175 kHz) relay nodes located throughout the United States. This mode of message transmission is immune to interference from the strong electromagnetic pulse produced by a high-altitude nuclear detonation.

An Environmental Impact Statement (EIS) for the GWEN system was issued in 1987. However, in 1990 members of Congress requested the Air Force to evaluate recent evidence for adverse health effects of electromagnetic fields and to assess the relevance of this information to the issue of possible health effects of GWEN emissions. The release of federal funds to bring the entire system of GWEN transmitters and relay nodes into operation was delayed until a response to this request for information was received by Congress. The Air Force subsequently established a contract with the National Research Council (NRC) to convene a committee of independent scientists to address the question of potential health effects of GWEN electromagnetic fields.

An NRC committee of eleven scientists who are recognized for expertise in the areas of dosimetry, biological interactions, epidemiology, and health effects of electromagnetic fields was appointed by the NAS Board on Radiation Effects Research and approved by the NRC Chairman, Dr. Frank Press. The GWEN committee met on five occasions for a total of nine days during the period December 14, 1990 to September 15, 1991. A draft report was prepared during this interval, and subsequently refined for submission to an NRC-appointed peer review committee in April, 1992.

The GWEN report was designed to be responsive to a series of questions raised by the Air Force on the potential health effects of electromagnetic fields, including risks of shocks and burns, effects of these fields on membrane processes in living cells, and the possible carcinogenic effects of these fields. Although relatively little information exists on the biological and health risks of electromagnetic fields in the frequency bands used for GWEN transmissions, the committee was nonetheless able to draw conclusions on the basis of available data for fields with frequencies below and above those of the GWEN system. Detailed dosimetric calculations were performed to characterize the physical interaction of GWEN fields with humans, and the existing biological and human health literature on the effects of electromagnetic fields was evaluated in this context. The levels of GWEN fields in public areas were also analyzed in relation to exposure standards and guidelines for electromagnetic fields that have been issued by governments and agencies throughout the

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

world. Finally, estimates of cancer risk imposed by public exposure to GWEN fields were made by comparison of GWEN field intensities with those of AM and FM communication systems, for which there is currently no evidence of adverse health impacts.

The overall conclusion of the committee was that no unacceptable risks to public health should result from full operation of the GWEN communication system. The committee also recommends that its report be used in conjunction with the original EIS as a definitive assessment of potential effects on public health of electromagnetic fields emitted by the GWEN system.

THOMAS S. TENFORDE
CHAIRMAN
COMMITTEE ON ASSESSMENT OF THE POSSIBLE HEALTH EFFECTS OF GROUND WAVE
EMERGENCY NETWORK

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Acknowledgments

The committee would like to extend its appreciation to Dr. D. Dennis Mahlum of the Board on Radiation Effects Research, who served as Study Director from January, 1991 through the conclusion of the report activities. We also thank Dr. Raymond D. Cooper, who was instrumental in establishing the Air Force contract in 1990 and served as the initial Study Director. The committee also thanks Mrs. Doris E. Taylor for her role in organizing the committee meetings and in preparing this report.

Appreciation is extended to Major Robert T. Veale and Lt. Col. Stephen Martin, who served as the Air Force project offices and provided extensive background information on the GWEN system to the committee; to Lt. Col. G. Andrew Mickley, Dr. James H. Merritt and Dr. Sidney Everett of the Armstrong Laboratory for Human Systems at Brooks Air Force Base, who provided perspectives and information on the biological effects of radiofrequency radiation; and to Dr. Steven M. Sussman and Dr. Roswell P. Barnes, Jr. of the MITRE Corporation in Bedford, MA for providing information on the physical characteristics of GWEN electromagnetic fields. Appreciation is also extended to Dr. John E. Burris, the Executive Director of the NRC Commission on Life Sciences, and Dr. Charles W. Edington, Director of the Board on Radiation Effects Research, for their support of the GWEN committee's activities.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

ACKNOWLEDGMENTS

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Contents

	Executive Summary	1
	Introduction	1
	Description of GWEN System	2
	Biological Interactions	2
	Field Interactions	3
	Organ and Tissue Systems	4
	Cellular and Subcellar Effects	5
	Human Evidence	5
	Thermal Effects	7
	Risk Assessment	7
	Exposure Reduction	9
	Conclusions	9
Chapter 1	Introduction	11
Chapter 2	Description of GWEN System	13
Chapter 3	Coupling of GWEN Electromagnetic Fields to the Human Body	25
	Low-Frequency (LF) Transmitter	25
	Ultra-High-Frequency (UHF) Transmitter	25
	Induced Fields and Currents in the Human Body	25
	Induced Currents and E Fields at 174.625 kHz	26
	SARs for UHF Electromagnetic Fields	28
	Microscopic Field Interactions at the Molecular, Cellular and Tissue Levels	32
	Indirect Coupling—Shock and Burns	34
APPENDIX A:	Anatomically Based Model and Numerical Procedure Used for Calculations	37
	Anatomically Based Model	37
	Finite-Difference Time-Domain Method	37
	References	41
Chapter 4	Perception and Behavioral Effects of Electromagnetic Fields	45
	References	49
Chapter 5	Effects of Electromagnetic Fields on Development	53
	References	61

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

CONTENTS		xii
Chapter 6	Effects of Electromagnetic Fields on Organs and Tissues	67
	Introduction	67
	Nervous System	67
	Visual System	69
	Endocrine System	70
	Immune System	71
	Hematologic and Cardiovascular Systems	71
	Animal Carcinogenesis	72
	Conclusions	74
	References	76
Chapter 7	In Vitro Cellular and Subcellular End Points	85
	Bone Healing	89
	Mutagenic Effects	89
	Cytogenetic Effects	90
	Cell Transformation	92
	Effects on Transcription	92
	Tumor Promotion	94
	Conclusions	98
	References	99
Chapter 8	Human Laboratory and Clinical Evidence of Effects of Electromagnetic Fields	109
	Cutaneous Perception	109
	Phosphenes	110
	Pacemaker Interference	110
	Microwave Auditory Effect	110
	Circadian Rhythms	110
	Brain Evoked Potentials	111
	Heart Rate	111
	Reaction Time	111
	Mood and Cognitive Function	113
	Blood Composition	113
	Bone Repair and Growth Stimulation	113
	Conclusions	114
	References	115
Chapter 9	Epidemiological Research Relevant to Identification of Health Hazards Associated with GWEN Fields	121
	Studies of General Environmental Exposure	121
	Occupational Studies	126
	Epidemiologic Studies of Health Effects of Microwave Exposure	127

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

CONTENTS		xiii
	Radio Broadcast Stations	128
	Amateur Radio Operations	129
	Conclusions	129
	References	131
Chapter 10	Standards and Guidelines for Exposure to Radiofrequency and Extremely Low Frequency Electromagnetic Fields	137
	References	141
Chapter 11	Risk Analysis and Management	143
	Risk Assessment	143
	General Description of GWEN Fields	144
	Coupling of Electromagnetic Fields to Human Body	145
	Shielding by Buildings	146
	Population Distribution Around Gwen Sites	146
	LF and UHF Exposures of Population Around Sites	149
	Exposure Comparisons with Existing Standards	150
	Exposure Comparisons with Other Sources	150
	Bounding GWEN Risks	154
	Historical Growth in Broadcast Activity	154
	Public Health Surveillance Around Broadcast Facilities	157
	Meadowlands Sports Complex	157
	Bounds on Excess Population Risk from GWEN Fields	158
	Limitations of GWEN Risk Assessment	162
	Risk Perception	162
	Exposure Reduction	163
	Research Needs	164
	References	165

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

ACKNOWLEDGMENTS

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

ASSESSMENT OF THE POSSIBLE HEALTH EFFECTS OF GROUND WAVE EMERGENCY NETWORK

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Executive Summary

INTRODUCTION

The Ground Wave Emergency Network (GWEN) is a nationwide system of radio transmitters and receivers intended to ensure adequate communication between command authorities and land-based strategic nuclear forces in the event of a nuclear attack on the United States mainland. The full GWEN system would consist of about 86 ground stations that would communicate in both the low-frequency (LF) radioband (150-175 kHz) and the ultrahigh-frequency (UHF) radioband (225-400 MHz) and 12 ground stations that would transmit only in the UHF band. The peak radiated power of the LF and UHF transmissions would be about 3,200 W and 20 W, respectively. Those power levels are modest, in comparison with those of most radio and television stations that broadcast at similar frequencies. During peacetime, the GWEN network would operate in a maintenance mode, each LF transmitter broadcasting about 1% of the time and each UHF transmitter turned on about 2% of the time.

A partial GWEN system involving about 60 broadcast sites is being constructed and tested. In support of the decision to begin construction, the U.S. Air Force completed an environmental impact statement (EIS) on the GWEN system in 1987. The EIS concluded that the impact of GWEN LF and UHF emissions on the health of people living near GWEN sites was likely to be negligible, because exposures would be smaller than safety guidelines established by scientific standard-setting organizations. The guidelines were based on a scientific consensus that harmful biological effects were not likely to occur at exposures smaller than those required to heat tissue measurably. Since publication of the EIS, new issues have arisen concerning the possible health effects of electromagnetic fields (EMFs). Most notable is the increase in epidemiological evidence of an association between cancer risk and exposure to extremely-low-frequency (ELF) fields that are too weak to cause tissue heating. In 1990, Congress asked the Air Force to postpone completion of the GWEN system, pending an evaluation of the new evidence on the biological effects of EMFs. The Air Force then asked the National Research Council to convene an independent scientific committee to address the issue. The Committee on Assessment of the Possible Health Effects of Ground Wave Emergency Network was formed in the Research Council's Board on Radiation Effects Research, and this is the final report of that committee. The report includes a description of the physical nature of GWEN fields and the electrical coupling of GWEN fields to humans, an analysis of current scientific literature bearing on the biological effects of GWEN fields, and evaluations of possible related human-health hazards and options for risk management. A particular effort has been made to interpret the controversial epidemiological data which suggest that a possible link exists between EMF exposure and cancer risk. The conclusions reached by the GWEN committee on this and other health-related aspects of exposure to EMFs are summarized in the

following sections of the Executive Summary. The literature review upon which this report is based extends to February 1992.

DESCRIPTION OF GWEN SYSTEM

GWEN is a radio communication system designed to withstand the damaging effects of electromagnetic-pulse energy surges produced by high-altitude nuclear detonations and other ionospheric disturbances. It uses ground waves, rather than the Earth's ionosphere, as a communication pathway, and it has three types of stations: input/output (I/O), receive-only (RO), and relay nodes (RNs). I/O stations, which use UHF, are capable of both sending and receiving messages. ROs would only receive messages transmitted through I/Os. RNs would provide continuous relay links between I/Os and ROs. The final operating system would consist of 12 I/Os, 130 ROs, and 123 RNs.

The I/Os would broadcast UHF signals in the 225-to 400-MHz band with an effective power of 20 W and a duty cycle of 0.024. A network of RNs would broadcast LF signals in the 150-to 175-kHz range modulated with minimum shift keying with a duty cycle of 0.014; the peak broadcasting power would be 2,000-3,200 W.

At 300 m from an RN, the peak LF electric and magnetic field values are 5 V/m and 165 μ G, respectively. The values decrease to 0.2 V/m and 9 μ G at a distance of 4 km. RNs would be located primarily in rural areas so surrounding population densities are typically low. It is estimated that the numbers of people living within 300 m and 4 km of all RNs in a completed GWEN system are roughly 80 and 37,000, respectively. For comparison, measurements of electric fields from AM radio broadcasts (535-1605 kHz) made at random outdoor locations in 15 U.S. metropolitan areas show that about 22 million people are exposed to fields stronger than 0.28 V/m and 1.3 million to fields stronger than 1.0 V/m.

BIOLOGICAL INTERACTIONS

The LF RNs and the UHF transmitters of GWEN emit signals that span more than a factor of 1,000 in frequency within the radiofrequency (RF) band. The LF fields emitted by the GWEN system are only weakly absorbed by the human body, and their interactions are best characterized by the current density and induced electric field, rather than by the power deposited in tissue (which depends on the square of the induced electric field). Because the wavelength and depth of penetration of LF fields are much larger than the maximum dimension of the human body, these fields interact in a manner that is more similar to power-frequency fields in the ELF band than to microwaves. In judging the potential biological effects of LF fields from GWEN transmitters, the committee has therefore analyzed both the substantial literature related to ELF fields and the literature on studies conducted with much higher frequencies in the RF band (primarily microwaves). The committee found only a few biological studies on humans or laboratory animals

conducted at frequencies in the LF band that were comparable to the transmitted signals from the GWEN system. Conclusions on the potential effects of LF fields from GWEN RNs have therefore been inferred from available information on the biological interactions of fields oscillating at both lower and higher frequencies, with recognition of the uncertainties associated with extrapolation across such a broad frequency range.

The UHF transmitters in the GWEN system emit signals that are close in frequency to FM radio transmitters and UHF television transmitters. The primary interaction mechanism of these fields involves electrical interactions with dipolar molecules in tissue, and the radiation is absorbed as a function of the depth of penetration inside the body. The potential biological effects of the UHF fields have been assessed by reviewing the literature on responses of humans and laboratory animals to microwave radiation of 300-30,000 MHz.

FIELD INTERACTIONS

The maximum induced currents in the body of a standing adult human optimally coupled to the ground (i.e., barefoot) were calculated with an anatomically based electrical model. For the maximum GWEN LF fields at the perimeter of a transmitter site, the calculated current passing through the feet to the ground was 2.89 mA, which is one-thirtieth the maximum permissible current, according to the Institute for Electrical and Electronic Engineers (IEEE) RF-protection guide. The peak current density is induced in the ankles in such an exposure and equals 700 mA/m² (70 μ A/cm²). In the brain and chest, the peak induced current densities are, respectively, 35 and 80 mA/m². The peak whole-body specific absorption rate (SAR) resulting from exposure to the UHF signal transmitted by the GWEN system is 97.9 μ W/kg, on the basis of calculations for a grounded, anatomically based model of a standing man. That SAR is lower by a factor of more than 10,000 than the maximum permissible levels for partial body exposures given in the IEEE radiofrequency protection guide.

Consideration was given to both thermal and nonthermal mechanisms through which the maximum fields induced in tissue could influence biological functions. The rate of energy deposition in tissue by GWEN fields was far below the rates that could produce a biologically important temperature rise. Similarly, on the basis of electrophysiological models, it was concluded that the fields induced in tissue were too low to significantly alter the transmembrane potential (i.e., the calculated change in potential was estimated to be less than 10 μ V). Nonthermal mechanisms of interaction of GWEN fields with living cells that might proceed under conditions in which the electrical properties of the cell membrane are not substantially altered were also considered. In general, they depend on a large ELF content of the applied fields, for example, through subharmonic-frequency content or LF amplitude modulation of the RF carrier wave. Apart from the pulse-group repetition frequency of approximately 1 Hz, the GWEN fields lack any ELF components, and it was concluded that such interactions are highly unlikely.

Indirect coupling of the LF fields from GWEN transmitters to persons in contact with ungrounded metallic objects, such as cars, was also considered. That form of coupling can lead to perception, shock, pain, and (at very high field levels) burns. From an analysis of the LF fields at the perimeter of a GWEN site, it was concluded that there would be no risk of electrical shocks or burns outside the site. A similar conclusion was drawn for the UHF fields emitted by GWEN transmitters.

ORGAN AND TISSUE SYSTEMS

The response of tissues to the RF fields emitted by GWEN LF RNs was assessed from a comprehensive review of literature on both ELF and microwave field effects. Potential effects of UHF fields were judged primarily on the basis of the biological literature on the effects of microwave exposures. Both thermal and nonthermal responses to RF radiation were reviewed in the context of possible effects of GWEN fields.

Tissue heating from exposure to RF radiation produces a series of physiological responses characteristic of stress, with a resulting activation of thermoregulatory mechanisms mediated through the neuroendocrine and cardiovascular systems. The threshold SAR for reproducible physiological and behavioral changes associated with tissue heating is approximately 1 W/kg, which greatly exceeds the SAR from GWEN fields. A detailed analysis of the literature on RF-field effects on major organ and tissue systems revealed no adverse responses of the nervous, ocular, endocrine, immune, hematologic, or cardiovascular systems to subthermal energy absorption. Similar conclusions were drawn for reproduction and development in mammals exposed to RF fields.

The responses of tissues to low-intensity, amplitude-modulated RF (AM-RF) fields and to sinusoidal and pulsed ELF fields were also reviewed in the context of possible physiological effects of LF fields from the GWEN system. Although changes in calcium ion binding to nerve cell surfaces and in lymphocyte immune functions have been reported after in vitro exposure to AM-RF fields, no such effects have been established for the weak LF fields produced by the GWEN system. Similarly, a variety of tissue responses to relatively high-intensity ELF fields have been reported, but there is no convincing evidence for adverse health effects of induced tissue currents comparable to those produced by GWEN fields. The effects of pulsed and sinusoidal ELF fields on reproduction and fetal development have been studied in both mammals and nonmammals. Although developmental abnormalities have reportedly occurred in avian embryos after exposure to pulsed magnetic fields with ELF repetition rates, there is no convincing evidence of teratogenic effects of high-intensity ELF fields in developing mammals.

CELLULAR AND SUBCELLULAR EFFECTS

It is difficult to draw firm conclusions with respect to GWEN emissions on the basis of available published data on the biological effects of EMFs, primarily because most of the information pertains to high-level fields with frequencies either higher than GWEN LF frequencies by a factor of about 1,000 (microwaves) or lower than GWEN frequencies by a factor of about 1,000 (ELF). However, a few papers related to bone healing have reported in vitro effects of 60-kHz electric fields. The GWEN UHF emissions oscillate at frequencies closer to the microwave region about which many biological effects data have been published. Positive microwave effects have been reported only after exposures considerably larger than those produced by GWEN emissions. The committee's review of cellular and subcellular data concentrated on ELF fields and bone-healing pulsed fields, because some of the relevant dose metrics were comparable with, or lower than, those associated with GWEN population exposures and also involved end points perceived to be important in risk assessment.

The more consistent low-level effects are related to changes in gene expression, alterations in Ca^{2+} balance at the cellular level, and changes in the activity of some enzymes. None of those effects has been clearly related to a particular health problem. Potential genotoxic effects of EMFs have also been addressed in numerous studies. Assays for mutagenicity have been consistently negative, but there have been some controversial results in the studies of chromosomal aberration. Most of the studies showing chromosomal clastogenic effects of EMFs have not been corroborated, and it can therefore be concluded that the data available do not support a direct genotoxic effect of EMFs. Because the genotoxicity studies on EMFs have been predominantly negative, it has been suggested that possible carcinogenic effects might be due to tumor promotion, rather than initiation. Several studies are currently attempting to determine whether EMFs act as a promoter or copromoter in tests with well-established in vitro and in vivo carcinogenicity models.

There are few experimental studies on the possible carcinogenic effect of EMFs with animal models and in vitro systems. In the case of ELF, a few negative results have been reported. At microwave frequencies, several studies have been published, some with positive indications of carcinogenic activity, including a dose-response curve for neoplastic transformation in vitro. All those studies were conducted at higher exposure levels than would be associated with GWEN.

HUMAN EVIDENCE

Direct evidence of biological effects of EMFs comes from three primary sources—laboratory studies of human volunteers, epidemiological studies of ELF-exposed populations, and epidemiological studies of populations exposed to AM radio frequencies or to radar or microwaves.

Studies of human volunteers have documented a variety of effects, including cutaneous perception, which is manifested at thresholds of 30-200 mA; phosphenes, which are visual effects resulting from induced currents in the retina; pacemaker interference; microwave auditory effects; circadian-rhythm alterations; behavioral changes; heart-rate changes; blood-chemistry changes; and changes in bone growth. The relevance of those observations to the GWEN system is for the most part small, because the exposures were at a much higher field level than would be associated with GWEN fields or at very different frequencies from those encountered in GWEN fields. The bone-growth changes are perhaps of some relevance, because the envelope of effective power and frequency might include expected GWEN emissions. However, the effects observed are restricted to accelerated healing of bone fractures that otherwise failed to heal, and the effects, if any, on healthy tissue are unknown.

Epidemiological studies of ELF-exposed populations can be divided into residential and occupational studies. Several of the dozen or so completed residential studies have reported excesses of childhood or adult cancer, particularly brain cancer and leukemia. The strength of those findings is limited by possible biases in the selection of study populations and by ambiguities in retrospectively assessing the magnetic-field exposure of study subjects. Excess cancer risk seems to be more strongly correlated with the configuration of residential power lines than with measured magnetic field levels in subjects' homes. That implies either that ELF-field exposure is not causally related to cancer risk, that measurements made in the present are worse indicators of past exposure than is power line configuration, or that biologically relevant aspects of field exposure are not captured by the measurements made to date.

A number of retrospective epidemiological studies have also reported excess cancer risk among workers thought, by virtue of their job titles, to work in unusual electromagnetic environments. Recent measurements of magnetic-field exposures in these electrically related occupations show that, as a group, such workers are indeed exposed to higher fields. However, correlations with cancer risk have generally proved stronger for job title than for measured field, suggesting either that the exposure measurements made to date do not capture the risk-relevant aspects of field exposure or that findings of elevated risk arise from some unknown confounder or from biases in the selection of comparison populations. Most studies of electrical workers have not controlled for possible exposures to hazardous chemical agents. Cancer rates noted in proportionate mortality studies might be higher simply because mortality from chronic diseases other than cancer is lower in electrically related occupations than in other comparison groups (i.e. the "healthy worker" effect).

Taken as a whole, the epidemiological literature indicates that there are not enough data to identify unequivocally, let alone to quantify, ELF-related cancer risks. Moreover, differences in frequencies and other characteristics between ELF and GWEN fields convinced the committee that the ELF data were of limited usefulness in assessing potential health risks from GWEN.

Epidemiological studies of populations exposed to electromagnetic fields from radio and television broadcasting are very few. One occupational study of the effect of exposures at AM radio frequencies found no elevated cancer risks among the study population, but the statistical power of that study to detect all but the shortest latency cancers was small. A study of proportionate cancer incidence in Hawaii reported elevated risks in census tracts near radio and television broadcast towers, but the study did not adjust for race, age, sex, socio-economic status, or urban-rural effects on cancer rates.

The last data source, epidemiological studies of radar and microwave-exposed populations, is sparse. Two populations, U.S. naval personnel exposed to radar and American embassy personnel in Moscow, dominate the literature. Neither has shown any adverse health effects, but both lack well-defined exposure data. Because the studies in question are of low statistical power and radar and microwave radiation have frequencies very different from those of GWEN, the findings do not offer much insight as to whether the GWEN system poses any risks to public health.

THERMAL EFFECTS

The calculated SARs for LF and UHF radiation for a person standing at the fence of a GWEN site are 0.001 and 0.0001 W/kg, respectively, compared with an IEEE radiation-protection guideline of 0.08 W/kg. The combined SAR of 0.0011 W/kg at the fence of a GWEN site would have a body-heating effect equivalent to raising the environmental temperature by 0.02°C. A temperature increase of that magnitude should not cause any health or environmental concerns.

RISK ASSESSMENT

Assessing risks from exposures to low levels of any environmental agent is an uncertain enterprise. Epidemiological and animal studies are limited in statistical power to risk levels that are well above those that society often considers important. Judgments about the amount of human exposure needed to produce very small risks must rely on extrapolations from higher levels, and often on extrapolations from effects measured in other species. Uncertainties in risk assessment are particularly troublesome for exposures to subthermal levels of nonionizing radiation, because the four usual steps in risk assessment are not complete:

- Hazard identification. The hazard is not clearly identified and still very controversial.
- Exposure characterization. There is no accepted exposure metric, and there are only categories of exposure of a proxy nature.

- Dose-effect relationships. Because there is no agreed on exposure metric, very few exposure measurements, and no clearly identified hazard, dose-effect relationships cannot be established.
- Risk characterization. Without quantitative population exposure assessments, and without dose-effect relationships, risks to any population cannot be established.

We concluded, therefore, that a traditional risk assessment could not be done. Consequently, we established upper bounds of risk for GWEN using data (basically negative data) from similar broadcast installations that had been in use for many years.

First, despite a large growth in radio and television broadcasting over the last several decades, concurrent time-series analyses reflect no parallel increase in overall cancer morbidity and mortality above the "noise" level of the data. Because the time-averaged electric fields induced in a body standing near the outer fence of a GWEN RN facility are comparable to or smaller than those induced by exposures to radio and television broadcasts in urban areas, GWEN risks are unlikely to be significantly greater than those associated with commercial broadcasting.

Second, systems for public-health surveillance regularly review cancer incidence and mortality data at the resolution of the census tract. This surveillance activity would be expected to detect any excess cancer risks around broadcast facilities that are larger than the detection threshold of the surveillance apparatus. That no convincing evidence has so far been found suggests that, whatever the public health risks related to broadcast exposure might be, they are smaller than the detection threshold. A recent epidemiological study of cancer risk in a population exposed to AM-broadcast fields as high as 16 V/m during work hours found no excess cancer risk. However, the statistical power to detect cancers other than short-latency ones, such as leukemia, was low. Only two cases of leukemia and no leukemia deaths were identified in a work population of 8,000. Both cases were among indoor workers who had the lowest exposure. Among outdoor workers, 1.11 cases of leukemia would have been expected. Despite the negative findings in this study, upper bound estimates from these data are close to the background rate, indicating the variability encountered in this type of analysis.

On the basis of the negative findings of epidemiological surveys related to AM broadcast stations, the committee estimates that the total excess number of cancer deaths associated with operation of the entire GWEN communication system over a 70-yr lifetime is less than one. This upper-bound estimate is well below the level that could be detected by an epidemiological survey system. The committee recognizes the inherent limitations in any effort to establish an upper bound estimate of cancer risk using negative data. However, such a bounding argument is likely to produce large numbers relative to the actual (unknown) risk. The upper bound cancer risk estimated here from negative epidemiological

findings on health effects of RF broadcast systems clearly indicates the absence of a significant cancer risk associated with GWEN emissions.

EXPOSURE REDUCTION

A number of actions might be taken to reduce population exposures to GWEN LF and UHF emissions. They include choosing from among candidate sites to avoid those with homes nearby (this is currently an important factor in site selection), placing the UHF-transmitting antenna in each site so as to minimize UHF exposures of those living nearby, placing the UHF-transmitting antennae on higher poles, increasing the size of the GWEN RN sites to reduce field strengths at the site boundaries, and reducing the number of GWEN RNs in the final GWEN system. Each of those options carries certain costs. The primary risk-management challenge is to decide which, if any, of the costs are justified.

CONCLUSIONS

The results of a critical evaluation of published literature on the biological and health effects of EMFs and of an analysis of the relevance of this information to the potential health effects of GWEN fields indicate that these fields should have only a minimal, and probably undetectable, impact on public health. In 1987, an environmental-impact statement on the effects of the GWEN system on humans and natural biota was published. The EIS focused on thermal mechanisms of interaction of GWEN fields with living systems and on the risks to humans posed by shock and burn phenomena associated with the charging of electrically conductive objects in the vicinity of GWEN towers.

Since the publication of the EIS, a number of new issues have arisen concerning the possible biological and health effects of EMFs, particularly those that might be associated with nonthermal events. The present report evaluates information from studies conducted at frequencies varying from ELF to microwaves in the context of public exposure to GWEN fields. Because there is little published information about the effects of LF signals, the committee used information from AM broadcasting to establish an upper-bound estimate of cancer risk to people living in the vicinity of GWEN stations. On the basis of its review of published literature on the biological and health effects of electromagnetic fields, the committee concludes that the excess risk of cancer death (that is, the risk that cannot be ruled out from health surveillance data) associated with exposure to GWEN fields is less than one additional death over a 70-yr period for persons living within 10 km of the entire system of GWEN sites.

The conclusions of this report reinforce those of the EIS, in that no evidence of adverse effects of GWEN fields on public health was found. The National Research Council committee recommends that this report be used in conjunction with the original EIS as a comprehensive assessment of the potential public health impact of the GWEN system.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Although the EIS published in 1987 is now somewhat outdated, the results of the present study obviate the need for its revision.

1

Introduction

The Ground Wave Emergency Network (GWEN) is a communication system under development by the Air Force for which a Final Environmental Impact Statement was issued in September 1987. In 1990, members of Congress asked the Air Force to evaluate recent evidence that exposure to electromagnetic fields results in adverse biological effects and to assess the significance of the evidence in the context of potential health effects of the low-frequency (LF) and ultra-high-frequency (UHF) radiation emitted by the GWEN system. The release of federal funds to bring the entire system of GWEN transmitters and relay stations into operation was delayed until the Air Force responded to the Congressional request for information. The Air Force established a contract with the National Research Council (NRC) to convene a committee of independent scientists to address the questions raised by Congress, and this report is the result of the work of the committee, the Committee on Assessment of the Possible Health Effects of Ground Wave Emergency Network.

In correspondence with NRC before establishment of the GWEN committee, the Air Force raised several questions related to the interaction of electromagnetic fields with living systems. Briefly stated, the questions raised by the Air Force were as follows:

- Is there epidemiological evidence relevant to the risk of significant environmental impact associated with GWEN electromagnetic fields?
- Is there evidence of tumor-promoting effects of electromagnetic fields, and if so, is it relevant to a carcinogenic risk to the public associated with the operation of GWEN?
- What is known about the effects of electromagnetic fields on membrane properties, biological signal transduction, Ca^{2+} binding to membranes, and Ca^{2+} transport into cells? Is there evidence of ion cyclotron resonance interactions or other physical interactions that could influence biosystems? If these interactions have been shown to exist, is the evidence strong and do the observed bioeffects constitute a health risk?
- What is the relevance of previous studies on shock and burn hazards to the operation of GWEN?

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

This report addresses all those questions. To accomplish its task within the broader framework of a comprehensive report, the committee has prepared a report that progresses from interaction mechanisms of electromagnetic fields to their biological and human health effects, and then to an analysis of risk and exposure reduction options. This report describes the physical characteristics of GWEN fields, mechanisms of electromagnetic field interactions with the human body, animal and tissue interactions of electromagnetic fields, human health effects of the fields, existing exposure standards and guidelines, and risk analysis and management. The committee has tried to relate the findings of laboratory studies and human epidemiological surveys to the possible health and environmental effects of GWEN fields. The committee has answered the Air Force's specific questions within the broader context of interaction mechanisms, biological effects and human health effects of electromagnetic fields.

2

Description of GWEN System

The Ground Wave Emergency Network (GWEN) system was designed to protect U.S. communications during a high-altitude nuclear explosion. Such an explosion could affect strategic communications in two important ways. First, the electromagnetic pulse (EMP) produced by a high-altitude nuclear detonation could, over a large area, produce a sudden power surge that would overload unprotected electronic equipment and render it inoperable; in recent years, the Air Force has pursued an extensive program to protect critical electrical components by making them immune to the effects of the EMP-produced power surge. Second, the EMP could interfere with radio transmissions that use the ionosphere for propagation of signals. GWEN uses a ground-hugging wave for propagation, not the ionosphere, and so would be unaffected by high-altitude nuclear detonations. It should be recognized that this ground-hugging wave propagation of the GWEN LF transmitter is no different than that for normal AM radio stations. For the latter sources, the electromagnetic fields also propagate with electric fields that are nearly vertical relative to the ground.

GWEN, developed by the Air Force Electronic Systems Division, calls for three types of stations: input/output stations (I/Os), receive-only stations (ROs), and relay nodes (RNs). I/O stations can both send and receive messages. ROs only receive messages transmitted through I/Os. Unmanned RNs, which would be dispersed throughout the conterminous 48 states, would provide continuous relay links between I/Os and ROs. I/Os and ROs would be at strategic Air Force locations, and RNs would be either on government land or on private land leased by the government. The ground wave used to transmit through the network requires RNs at intervals of approximately 150-200 miles.

I/Os would use 50-W transmitters to drive antennae mounted on 20-to 100-ft towers. They would broadcast ultra-high-frequency (UHF) signals in the 224-to 400-MHz band. I/O terminals would enter messages into the RNs by line-of-sight UHF radio links. Airborne I/Os would use the same basic equipment sets and operate at the same power and frequency band as fixed I/Os.

ROs would use 48-in. loop low-frequency (LF) receiving antennae, which would be generally on or near the roofs of existing military communication facilities as would fixed I/Os. Airborne I/Os and portable RO terminals would also use the system. ROs would be installed at fixed locations, and portable units would be stationed at alternate Strategic Air Command (SAC) operating locations. Launch control centers (LCCs) would also be equipped with RO terminals.

Most of the RNs would communicate primarily with other RNs, transmitting with LF antennae at 150-175 kHz. Eight of the RNs would receive messages from I/Os

via UHF transmission and transmit messages via the LF antenna to other RNs. All other RNs would have UHF antennae on 60-or 150-ft self-supporting towers for communicating with airborne I/Os.

The overall site area of an RN (see Figure 2-1) would be approximately 11 acres, measuring approximately 700 × 700 feet. A 299-ft-high LF (triangular in cross section, with 2-ft sides) transmitter tower structure with an 8 × 14 × 8-ft antenna-tuning unit (ATU) would be in the center of the site. The structure comprises a concrete foundation 2 ft above grade, a 3-ft-high insulator, a 290-ft steel tower, and 4-ft lightning rods enclosed by a 42 × 47-ft, 8-ft-high chain-link fence topped with barbed wire. The tower itself would be supported by 15 guy wires attached to the ground at six anchor locations. Surrounding the tower and attached to it at the top and anchored in the ground by concrete blocks would be 12 top-loading elements (TLEs). The purpose of the TLEs is to enhance the efficiency of the antenna. Anchors for the TLEs and guy wires would be within the site boundaries.

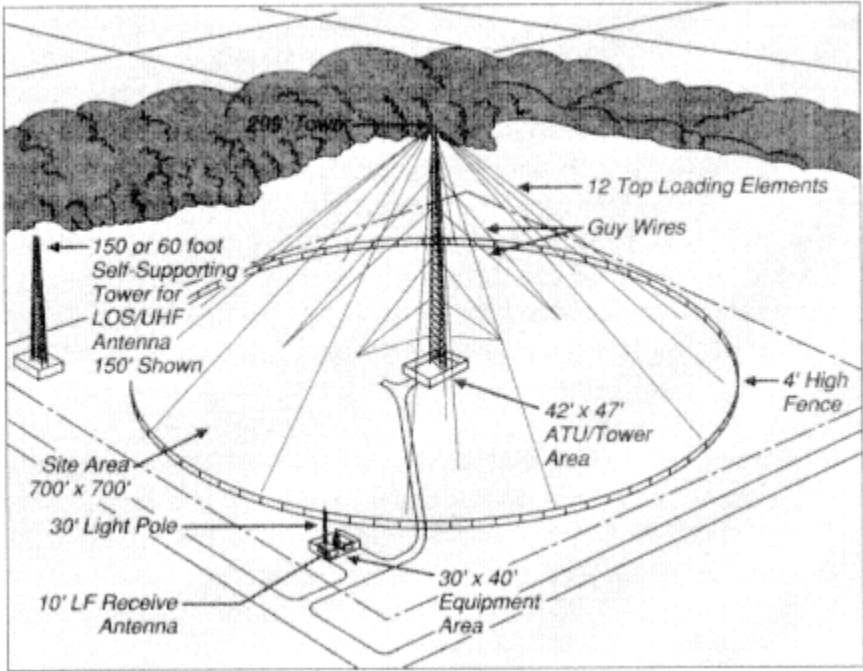


Figure 2-1.
Proposed layout of typical relay node (RN) station with line-of-sight capability (eight proposed).

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

A ground plane typically consisting of approximately 100 copper wires, 0.128-in. in diameter (about the thickness of pencil lead) and installed about 12 in. below grade, would radiate approximately 330 ft from the center of the tower. The number (50 150) and length of copper wires would vary from site to site, depending on soil conductivity. The whole tower area—including guy wires, TLEs, and the ground plane—would be enclosed in a circular 4-ft-high barbed-wire or woven-wire fence. A 30 × 40-ft equipment area enclosed by an 8-ft-high chain-link fence topped with barbed wire would also be on the site. It would be connected to the antenna area by a 10-ft-wide access and service road and to the adjacent road by a 12-ft-wide access and service road.

In addition to the 299-ft LF tower structure, each RN would have a UHF antenna and an LF receiving antenna on a 10-ft mast in the equipment area. The eight RNs within line of sight of the I/Os would have UHF antennae mounted on 60-or 150-ft self-supporting or guyed towers; these towers would be in their own fenced areas enclosed by 8-ft fences topped with barbed wire. The other RNs would have smaller, whip-like UHF antennae mounted on 30-ft light poles within the equipment area. Each RN would have only one type of UHF antenna.

The geographic distribution of GWEN stations is determined by the location of users and the system's technical capabilities. Fixed I/Os and ROs are determined by user needs and would be at existing military facilities. The network configuration of RNs is determined by the necessity to connect fixed and mobile I/Os and ROs and existing thin-line connectivity capability (TLCC) sites. Nominal locations for RNs are derived in part by using a system-wide allocation procedure based on system requirements, predicted signal strength, and ground conductivity. To maintain the required signal strength, actual RN sites should generally be within approximately 9 miles of the nominal locations; the distance will vary with site-specific technical performance factors, such as signal strength and signal-to-noise ratio.

A partial GWEN, called TLCC, is in the construction and testing phase. It contains eight I/Os, 30 ROs, and 56 tower RNs. The FOC phase of GWEN would involve adding four fixed I/Os, 107 fixed ROs, approximately 70 unmanned RNs, 34 airborne I/Os, and a number of portable ROs to the network.

Message injection into the network is by line-of-sight UHF radio links from I/Os to nearby RNs. Message reception via LF RO terminals occurs at key bases throughout the 48 states, including main operating bases of SAC, dispersal bases, and LCCs. I/O and RO terminals are co-located with other manned communications systems at existing military installations; RNs would be unmanned. In addition to transmitting vital messages, the system would be used by SAC and the North American Air Defense Command for exercises and daily status checks among different locations.

The main GWEN antenna would operate intermittently in the LF band at 150-175 kHz (for comparison, the bottom of the AM band is 530 kHz). The peak broadcasting power would be 2,000-3,200 W. The UHF antennae at RNs would operate at 20 W and 225-400 MHz. GWEN would not interfere with commercial television or radio broadcasts, amateur radio operations, garage-door openers, or pacemakers.

When broadcasting, the GWEN system would transmit for approximately 2/3 second, receive for 2/3 second, and receive a duplicate message for 2/3 second before repeating the cycle. In its ready status, the GWEN system would nominally make nine 2/3-second transmissions every hour, for a total of 6 seconds/hour. In a 24-hour period, the system would transmit for about 144 seconds or 2.4 min. If the system were to broadcast on a continuous basis for an exercise, it could transmit for a maximum of 6.7 hours in a 24-hour period (but this assumes a level of message input efficiency that does not yet exist).

After discussions with MITRE Corporation staff, we decided to use a duty cycle of 1.4% (0.014 of the time) for our calculations in [Chapter 11](#). This number is probably on the high side but allows for some use of the system in addition to the routine broadcasts (6 seconds/hour) used to check the system.

GWEN will use a procedure called minimum shift keying (MSK). The MSK spectrum, referred to the channel center frequency, has been plotted on a linear scale in [Figure 2-2](#) for the GWEN data rate of 1,200 bits/sec. The actual GWEN signal at the LF carrier frequency consists of intermittent transmissions of packets carrying 644 uncorrelated MSK bits. That is equivalent to a continuous MSK signal turned on and off as packets are transmitted. The resulting "envelope modulation" of the bit stream has an insignificant effect on the normalized signal spectrum of [Figure 2-2](#). There are no spectral lines in the radiated GWEN signal because the uncorrelated bit stream has no periodic components.

During instances of repeated signal transmission, the GWEN signal envelope will appear as a train of pulses of approximately 0.557 second with a pulse repetition period of 2 seconds. The nonlinear process of envelope detection operating on the packet train produces a periodic signal with a DC component, as well as the 0.5-Hz fundamental frequency and its harmonics. [Figure 2-3](#) shows the normalized spectrum of the GWEN packet envelope. The spectral amplitude is related to the radiated signal peak envelope through the proportionality constant At/T , where A is the signal peak envelope, in volts/meter or milligauss; t is the pulse duration; and T is the period. The fraction t/T is the maximum duty cycle of the GWEN LF signal. The DC component is $0.28A$, and the fundamental at 0.5 Hz is $0.19A$. The 16th harmonic at 8 Hz is more than 20 dB below the fundamental component. The characteristics of the GWEN LF signal and the GWEN transmitter and antenna are summarized in [Tables 2-1](#) and [2-2](#).

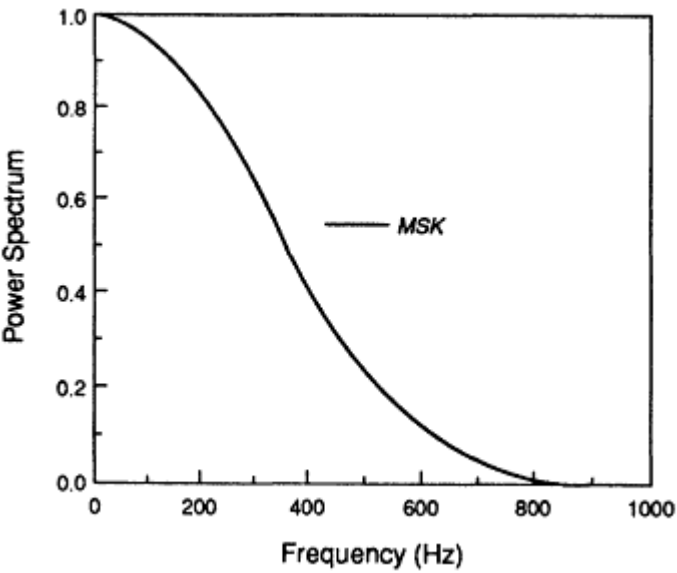


Figure 2-2.
Minimum shift keying (MSK), 1,200 bits/sec.

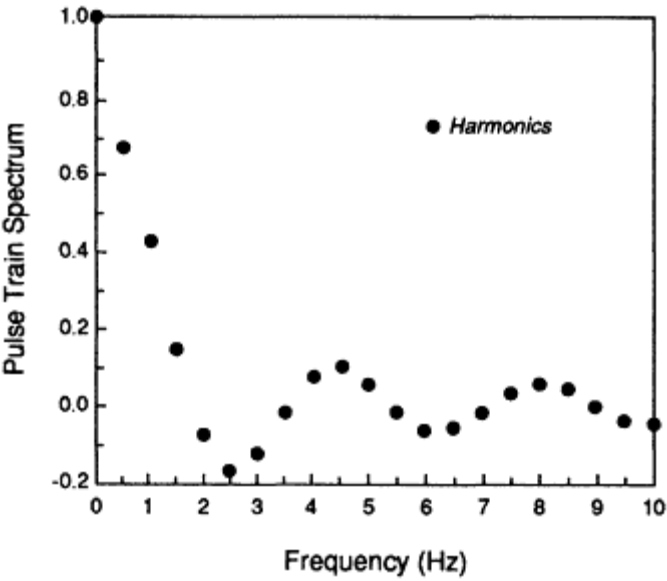


Figure 2-3.
Packet envelope spectrum (0.557-sec pulse, 2-sec period).

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 2-1. GWEN LF Signal Characteristics

Carrier frequency	150.625-174.625 kHz
Channel spacing	1, 25 Hz
Number of channels	20
Modulation	Minimum shift keying
Instantaneous data rate	1,200 bits/sec
Packet length	644 bits
Packet duration	537 msec
Minimum transmission interval	2 sec
Normal message transmission	3-packet repetition
Average transmitter duty cycle	0.0017 ^a

^a This number may double to support full implementation of mobile and portable GWEN stations.

TABLE 2-2. GWEN Transmitter and Antenna (Typical Values)

Antenna type	Base-fed, top-loaded monopole
Antenna height	299 ft
Number of top loading elements	12
Ground plane radius	330 ft
Number of buried copper radial wires	100
Input power	5 kW
Antenna system efficiency	40-65%
Radiated power	2-3.2 kW

The electric field strength depends on the power radiated by the transmitting antenna. GWEN is designed on the basis of a radiated power of 2 kW, so this value has generally been used in estimating exposure. However, some RNs radiate considerably less than 2 kW, and others considerably more. A reasonable upper limit of the power that can be radiated by a GWEN LF antenna is 3.2 kW, and this value is therefore recommended as a "worst case" for estimating exposures.

Two types of geometric dependencies influence electric field strength at a distance. The first is the usual 1/r spreading loss that occurs in the far field of the transmitting antenna as a simple result of the conservation of energy for propagation in a lossless medium. This dependence would be sufficient for describing the geometric effects if the earth were flat. But the 1/r dependence does not apply in the near field of the transmitting antenna. For example, in the case of the electric field strength near a GWEN LF transmitting antenna, the 1/r dependence should be applied only at distances greater than 0.2 mile.

The second dependence is the diffraction loss that results from the earth's being spherical instead of flat. Thus, at longer ranges from the transmitter, a receiving location will be in a geometric shadow zone (i.e., it will be below the horizon). Radiation can then reach the location only through diffraction effects. In the LF range used by GWEN, the shadowing effect reduces electric field strength by less than 1% for ranges less than about 75 miles and by less than 10% for ranges less than about 155 miles. At 300 miles, electric field strength can be reduced by about 35%.

A third influence on electric field strength with increasing distance is absorption loss. The surface of the earth provides a boundary for the propagating electromagnetic wave, and the physical boundary conditions lead to the absorption of energy by the earth. The lower the electrical conductivity of the earth's surface, the larger the loss. That effect leads to losses that are best described in terms of dB per mile. The highest anticipated rate of absorption is about 0.1 dB/mile; this occurs if the effective conductivity of the earth's surface is 0.5 millisiemen/meter (mS/m), and it reduces the electric field strength by an additional factor (beyond pure geometric effects) of 3 in about 95 miles and by a factor of 10 in 200 miles. But, there are portions of the United States where the effective conductivity is 30 mS/m; in this case, the rate of absorption is only 0.0042 dB/mile, which leads to an additional reduction (beyond pure geometric effects) by only 5% in 200 miles.

Figure 2-4 shows the relationship between distance and the electric and magnetic field strengths for the LF transmitter. Figure 2-5 is a similar diagram showing electric field strength vs. distance for the UHF transmitter. The contribution of the UHF antenna is much smaller than that of the LF transmitter. Detailed survey data around

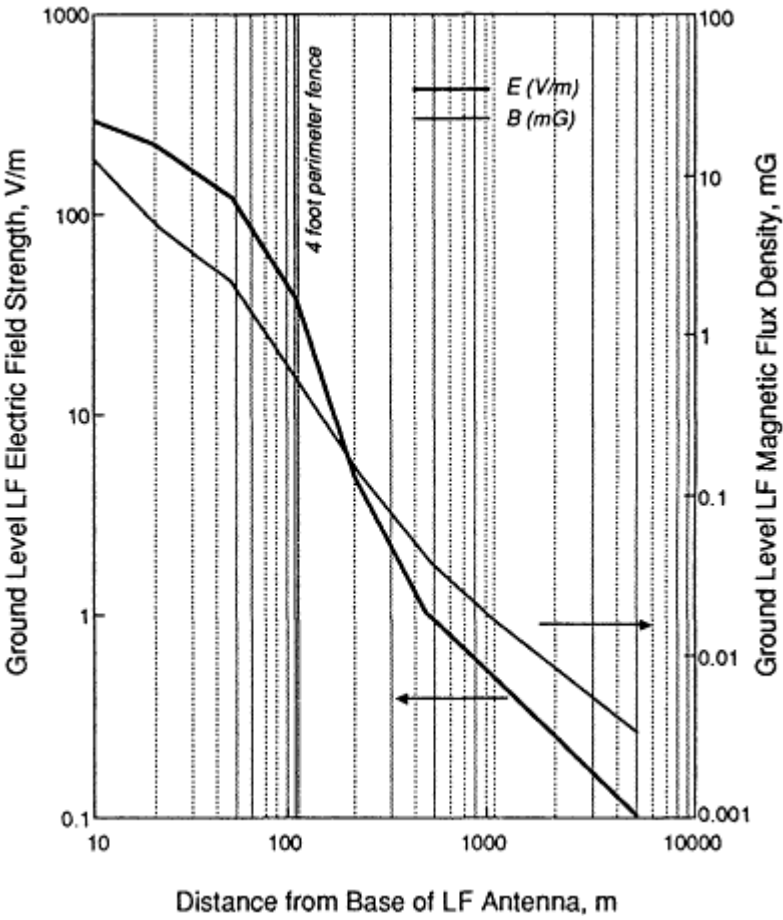


Figure 2-4.
Ground level electric and magnetic fields from GWEN LF antenna radiating the design maximum peak power of 3,200 W. Typical radiated peak and average powers are 2,000 W and 28 W, respectively.

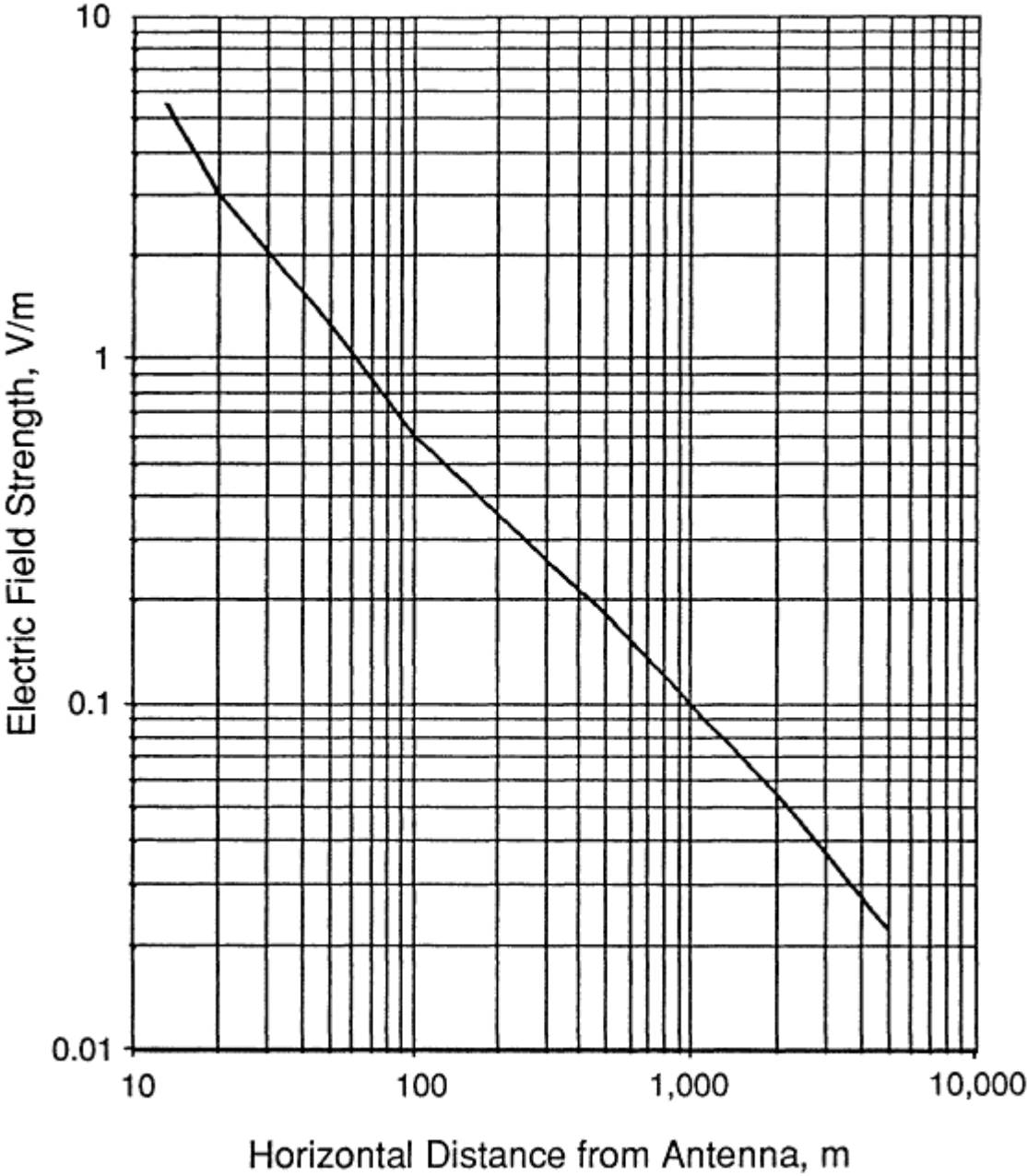


Figure 2-5.
Electric field strength at 10 m height vs. horizontal distance from GWEN UHF ground-to-air antenna radiating design maximum peak power of 70 W. Typical radiated peak and average powers are 50 W and 1 W, respectively. Ground-level fields will be smaller than those at 10m.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

candidate sites do not exist. However, because most candidate GWEN sites are relatively remote, the background levels are not expected to be high enough that the addition of the GWEN fields is going to push levels above the accepted guidelines for public exposure (see [Chapter 10](#)).

Populations around 40 proposed GWEN RN sites have been estimated by examining U.S. Geological Survey maps of randomly chosen candidate locations for each site. All structures, unless specifically identified otherwise, were counted as homes. Structures were counted in each of six contiguous concentric zones with outer boundaries of 0.2, 0.3, and 0.5, 1, 2, and 4 km. A value of 2.6 persons/structure was used to estimate populations. [Table 2-3](#) summarizes the data for these 40 sites and presents calculations of minimum and maximum E and B fields encountered in each zone. Population densities are low; it is estimated that approximately 800 people would reside within 1 km of the antenna. LF fields at the 1-km boundary are estimated to be 0.54 to 1.0 V/m for the E field and 1.8 to 4.2 μ G for the B field. For comparison, it has been estimated that in 15 metropolitan areas surveyed by the U.S. Environmental Protection Agency about 1.3 million people would be exposed to E-fields of more than 1.0 V/m.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

DESCRIPTION OF GWEN SYSTEM								23
TABLE 2-3. Numbers of Houses Within Six Contiguous Concentric Zones Around 40 RN Sites. Dmin is Distance from Center of Site to Nearest House.								
	Site	Region						D min. ft
		1	2	3	4	5	6	
		< 200 m	200-300 m	300-500 m	.5-1 km	1-2 km	2-4 km	
	901	0	1	0	2	6	46	1000
	902	1	1	0	5	11	69	500
	903	1	0	3	4	37	310	625
	904	1	0	0	3	28	152	500
	905	0	0	2	13	40	235	1125
	906	1	1	1	7	18	75	600
	907	3	2	9	17	57	139	375
	908	0	0	2	12	78	195	1250
	909	0	0	4	27	22	123	1000
	910	0	0	1	6	45	66	1250
	911	0	0	0	0	0	365	9000
	912	0	0	2	3	33	42	1125
	913	0	0	0	1	0	7	1750
	914	0	0	0	3	12	115	2000
	915	0	0	0	1	30	51	2400
	916	0	0	0	2	2	6	2375
	917	0	0	1	0	0	1	1000
	918	0	0	0	0	0	13	11500
	919	0	0	0	0	2	20	2001
	920	0	0	0	0	1	4	6250
	921	0	0	0	0	0	0	13120
	922	0	0	0	0	0	15	10250
	923	0	0	0	2	10	15	2250
	924	1	0	2	0	2	1	375
	925	0	0	0	5	55	417	2200
	926	2	0	0	6	20	51	1000
	927	0	2	2	5	11	19	875
	928	1	0	0	2	6	12	440
	929	1	0	0	0	1	3	435
	930	1	0	0	1	5	11	500
	931	0	0	0	0	3	5	3400
	932	0	0	0	1	4	18	2400
	933	0	0	1	13	28	110	1040
	934	0	0	1	1	10	23	2000
	935	0	0	0	3	11	32	1625
	936	0	0	0	4	15	52	2750
	937	0	1	2	11	42	90	625
	938	0	2	3	7	70	285	750
	939	0	0	0	0	15	355	5250
	940	0	3	2	18	57	79	625
AVG		0.325	0.325	0.95	4.625	19.675	90.675	2488.4
ST DEV		0.647592	0.7206768	1.6725729	5.965264	21.11799	112.0539	3115.651
SUM		13	13	38	185	787	3627	
CUM SUM		13	26	64	249	1036	4663	
Area, km ²		0.0801	0.237	0.7399	3.096	12.52	50.2	
Avg density (houses/km ²)		4.06	1.37	1.28	1.49	1.57	1.81	

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

DESCRIPTION OF GWEN SYSTEM	24
----------------------------	----

3

Coupling of GWEN Electromagnetic Fields to the Human Body

ELECTROMAGNETIC FIELDS FOR GWEN SITES

Low-Frequency (LF) Transmitter

The carrier frequency of the LF transmitter is 150.625-174.625 kHz. The largest electric (E) and magnetic (B) fields estimated for the GWEN relay nodes (RNs) in public-access areas are at the 4-ft-high perimeter fence typically 333 ft from the base of the LF antenna. Measurements have been conducted by the MITRE Corporation for E and B fields at various distances from the base of the LF antenna for an input power of 50 W, and the results have been scaled to a power of 5,000 W for an operating system. The maximum E and B fields at the GWEN RN perimeter fence obtained by this scaling procedure are 50 V/m and 0.7 mG (0.07 μ T), respectively. These values, therefore, have been used as nominal values for the calculations in this chapter. These are the highest E and B fields for areas of general public access, and both diminish fairly rapidly with increasing distance from the LF antenna.

ULTRA-HIGH-FREQUENCY (UHF) TRANSMITTER

The UHF transmitter would typically radiate about 20 W of power at 225-400 MHz. According to calculations by the MITRE Corporation, the maximum power density of exposure at the perimeter fence would be 0.001 mW/cm², and this would decrease rapidly with increasing distance from the antenna and the perimeter fence.

INDUCED FIELDS AND CURRENTS IN THE HUMAN BODY

We have used an anatomically based model of the human body^{1,2} to estimate induced fields and currents for both LF and UHF electromagnetic fields. For the LF fields, we have assumed a highest frequency of 174.625 kHz, because the induced currents are known to increase linearly with frequency and this frequency would be the highest used for the LF transmitter; an E field that is vertically polarized; a B field that is oriented from arm to arm of the hypothetically exposed person because these conditions are known to produce the highest internal fields; and a barefoot exposed person, because that is the worst-case condition. For electromagnetic fields due to the UHF transmitter, we have similarly assumed a vertically polarized E field, a B field oriented from arm to arm, and a grounded barefoot person. The salient features of the

anatomically based model and the numerical procedure used for the calculations are given in Appendix A to this chapter. Even though the exposure is in the "near field" of the LF transmitter, the E and B fields do not vary a great deal over the physical extent of the human body. As shown in the literature,³ coupling to the human body for such relatively uniform incident fields is no different than that for far-field conditions. Far-field exposure conditions have therefore been used to estimate coupling of the GWEN EM fields to the human body.

INDUCED CURRENTS AND E FIELDS AT 174.625 KHZ

For the calculations, we used an E field of 50 V/m (vertical) and a B field of 0.7 mG (oriented from arm to arm of the model). The value of 50 V/m is the highest electric field intensity at the perimeter fence of any GWEN facility, and a more typical value is 40 V/m at the perimeter fence.

The induced current for these exposure conditions is primarily vertical (z). The calculated z-directed current from the grounded anatomically based model is shown in Figure 3-1 as a function of height above ground for the various sections of the body. An induced current of 2.88 mA is calculated to be flowing through the feet of a standing person. The calculated current is in excellent agreement with that estimated with the empirical equation given previously by Gandhi et al.⁴ In the empirical equation, the foot current I_h for a vertically polarized E field flowing through a standing, barefoot person is given by

$$I_h = 0.108 h_m^2 f_{\text{MHz}} E, \tag{1}$$

where h_m is the height of the human in meters (1.75 m for our model), f_{MHz} is the frequency (for this case, 0.174625 MHz), and E is the incident electric field in V/m (for this case, 50 V/m). For the assumed exposure conditions, from Equation 1, $I_h = 2.89$ mA. That is in excellent agreement with the value calculated for the section through the feet of the anatomically based model (Figure 3-1).

The calculated current through the feet (2.88 mA) is considerably smaller than the maximum permissible current of 90 mA for the uncontrolled environment in the new radiofrequency (RF) protection guide suggested by the Institute for Electric and Electronic Engineering (IEEE) Standards Coordinating Committee.⁵

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

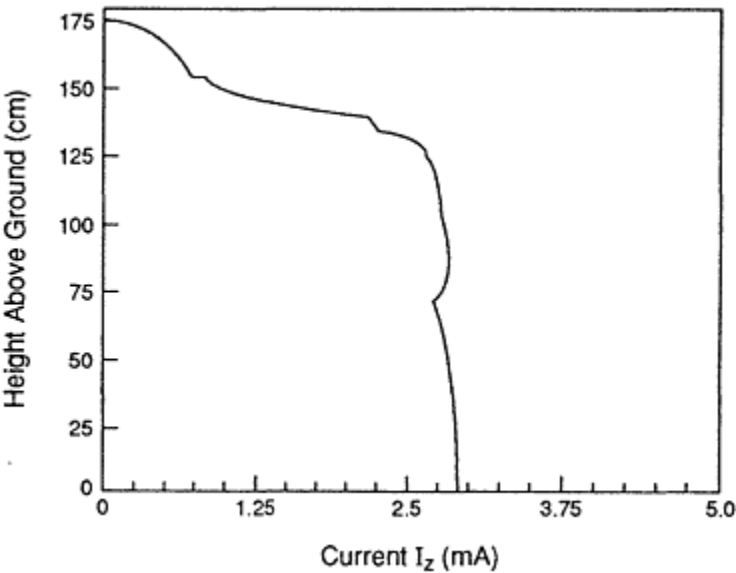


Figure 3-1.
Calculated section currents for grounded anatomically based model of human body for exposure to electromagnetic fields at 174.625 kHz. Fields assumed are at 4-ft perimeter fence. $E = 50$ V/m (vertical); $B = 0.7$ mG (from arm to arm model).

Table 3-1 shows maximum current densities calculated for some representative sections of the human body. The exposure dimensions are those measured for the public-access points closest to the LF transmitting antenna, i.e., at the 4-ft perimeter fence. For the numbers given in Table 3-1, we have taken $E = 50$ V/m (vertical) and $B = 0.7$ mG from arm to arm of the model.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 3-1 Calculated Maximum Values of Current Density ($J_{T,max}$) and Electric Field ($E_{T,max}$) for Some Representative Sections of Anatomically Based Human Model ($E = 50$ V/m; $B = 0.7$ mG)

Section of Body	Height above ground, cm	$J_{T,max}$, $\mu A/cm^2$	$E_{T,max}$, mV/m
Brain	165.7	3.5	170
Neck	155.2	8.1	380
Lung	140.8	7.2	475
Heart	133.0	8.4	250
Kidney	122.5	7.1	220
Liver	117.2	7.5	200
Bladder	89.7	10.4	405
Ankle	16.4	71.4	2,140

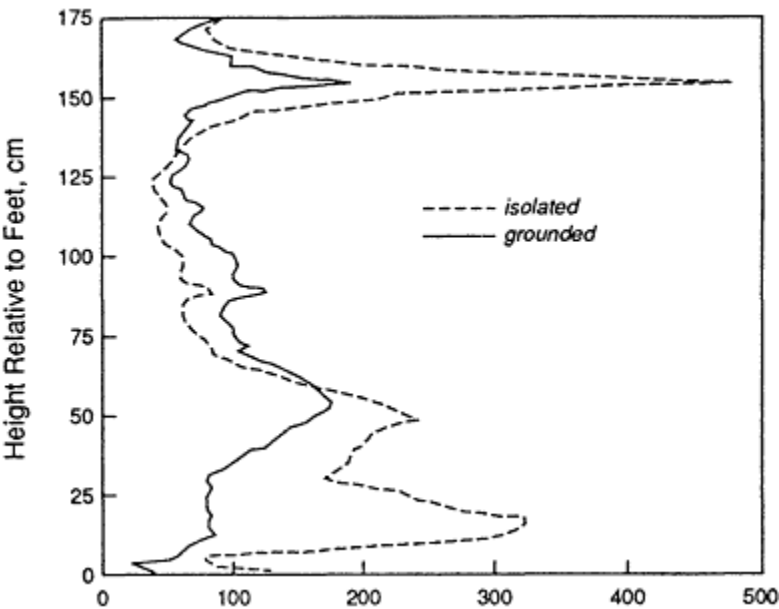
SARs FOR UHF ELECTROMAGNETIC FIELDS

According to calculations by the MITRE Corporation, the maximum power density for the general public would be at the 4-ft perimeter fence, where the power density is estimated to be 0.001 mW/cm². We have calculated the whole-body-averaged specific absorption rates (SARs) for some representative frequencies in the UHF band, assuming vertically polarized E fields and both the isolated and grounded anatomically based models of a human. The values calculated for various frequencies are given in Table 3-2. They are considerably lower than the 0.08 W/kg (80,000 μ W/kg) suggested in the new RF protection guide issued by IEEE.⁵ Shown in Figure 3-2 are the section averaged SAR distributions for grounded and isolated models of the human body. The highest SARs calculated for points in the body are smaller by a factor of several thousand than the local SARs of 1.6 W/kg permissible in the new RF protection guide.⁵ For all of the preceding calculations in this chapter, the effect of the ground plane has been considered. Reflections from any structures have, however, been ignored since they are so dependent on the size and shape of these structures. These reflections can cause interference with the incident EM fields resulting in a slight enhancement or decrease of the fields depending upon the physical location on the ground.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

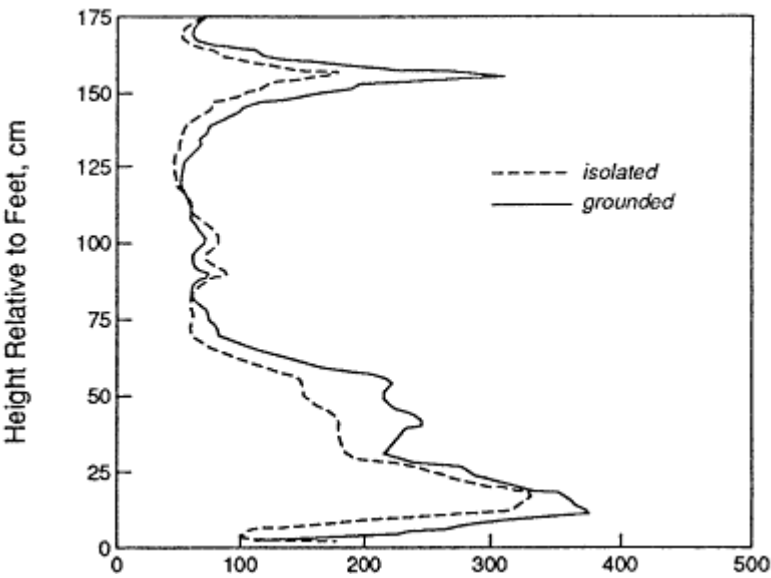
TABLE 3-2. Whole-Body-Averaged SARs Calculated for Anatomically Based Model of Human Body (Incident Power Density, 0.001 mW/cm²; Vertically Polarized E Field)

Frequency MHz	SAR μ W/kg Isolated Model	SAR μ W/kg Grounded Model
225	94.5	85.1
250	89.3	96.7
300	79.9	97.9
350	80.4	84.4

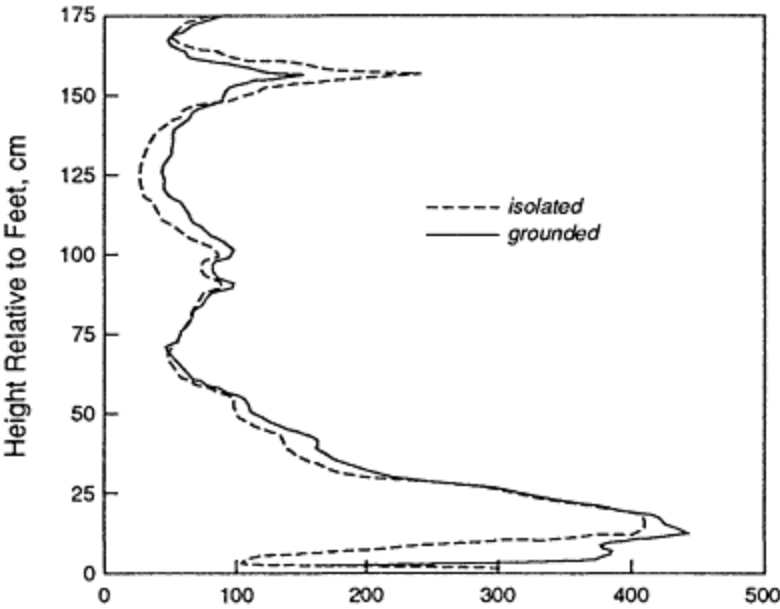


SAR, $\mu\text{W/kg}$
a. 225 MHz

Figure 3-2.
Calculated section-averaged SAR distributions for anatomically based model of human body at various ultra-high frequencies. Both isolated and grounded conditions of model are considered. Calculations assume incident power density of 0.001 mW/cm² that is estimated for 4-ft perimeter fence around GWEN antennae.

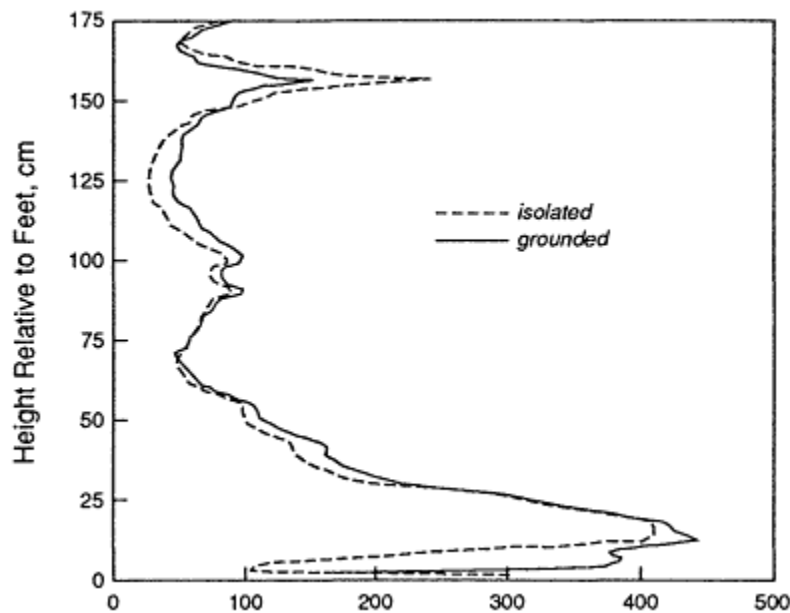


SAP, $\mu\text{W/kg}$
b. 250 Mhz



SAP, $\mu\text{W/kg}$
c. 300 MHz

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.



SAP, $\mu\text{W/kg}$
d. 350 Mhz

MICROSCOPIC FIELD INTERACTIONS AT THE MOLECULAR, CELLULAR, AND TISSUE LEVELS

As described in the preceding section of this report, the coupling of both LF and UHF signals from the GWEN system at locations beyond the site perimeter is too weak to produce measurable tissue heating. However, it has been reported that electromagnetic fields can produce biological effects through nonthermal interactions.^{6,7}

As discussed in Chapters 4-8, effects of nonthermal fields have been reported to occur in the nervous, cardiovascular, endocrine, immune, and reproductive systems

(for general reviews, see Tenforde and Budinger⁸ and Michaelson and Lin⁹). Possibly the most widely discussed nonthermal effect of electromagnetic fields is the reported change in Ca^{2+} binding to nerve cell surfaces as a result of exposure to RF radiation with amplitude modulation at extremely low frequencies (ELFs), reviewed in detail in Chapters 6 and 7. The most effective band of modulation frequencies has been found at 6-20 Hz, although some higher frequencies are also effective.¹⁰ In none of the studies on Ca^{2+} binding to cell surfaces has the unmodulated RF carrier wave itself been found effective. Modulation of GWEN signals by the minimum key shifting procedure produces a waveform very different from that of the sinusoidally amplitude-modulated RF signals that were used in most of the Ca^{2+} experiments. Specifically, the GWEN fields have the general characteristics of pulsed on-off RF signals during message transmission (see Chapter 2). It is therefore unlikely that the Ca^{2+} effects observed with sinusoidally amplitude-modulated RF fields have direct implications for biological or human health effects of the GWEN transmitter. That conclusion is supported by the inability of Merritt et al.¹¹ to alter Ca^{2+} binding to brain tissue using RF fields that were pulse modulated at 16 Hz.

RF fields from sources outside the body induce E and B fields in tissue that can be calculated with the procedures described earlier in this chapter. At frequencies below approximately 10^5 Hz, the induced fields can alter the electrical properties of cellular membranes and, at sufficiently high intensities, stimulate excitable tissues. At these frequencies, the impedance of the cell membrane is high and induced currents flow primarily in the extracellular fluids. At higher frequencies, the cell membrane poses less of a dielectric barrier to current flow and the electromagnetic field is more strongly absorbed by tissue. As described in Chapter 4, there is a distinct difference in human perception of fields with frequencies below and above 10^5 Hz. In the lower-frequency range, the perception is of tingling (typical of nerve stimulation); at higher frequencies, the sensation is of warmth. For the field intensities encountered at the perimeter of a GWEN site, the LF and UHF fields are not expected to produce either of those effects (see Chapter 4).

Extensive studies on the interactions of ELF fields with cells and tissues are yielding a growing body of evidence that membrane interactions of these fields can trigger intracellular responses, such as alterations in the transcription and translation of macromolecules (see Chapter 7 for a detailed discussion). The exact molecular mechanisms by which the cellular responses occur are poorly understood, but there is evidence of effects of ELF fields on the transmembrane flow of ions, such as Ca^{2+} , and on second-messenger signaling mechanisms that involve membrane receptors.¹²⁻¹⁵ Several of these reported effects could result from tangential electric field and currents acting on components of the cell surface (e.g., the extracellular portion of a transmembrane receptor protein), whereas other effects may be attributable to electric fields induced across the cell membrane. The threshold induced current density for reproducible cellular responses to ELF fields¹⁴ appears to be in the range of $0.1\text{-}1.0\ \mu\text{A}/\text{cm}^2$, which is comparable with the intrinsic current densities flowing in the body as

a result of endogenous electrical activity in excitable tissues.¹⁶ As described earlier in this chapter, the largest current density induced in most parts of the human body by the LF fields at the perimeter of a GWEN site is in the range of 3-10 $\mu\text{A}/\text{cm}^2$. Those LF current densities are therefore about 10 times the threshold of ELF current densities for eliciting membrane and cellular responses. However, the effective E field induced within the cell membrane will be substantially lower, by a factor of about 1,000, at low frequencies than at extremely low frequencies, because of the lower membrane impedance.^{17, 18} Consequently, the maximum currents induced in body tissues by the LF fields of a GWEN transmitter would be expected to establish electrical signals in cell membranes that are approximately one-hundredth the magnitude of the threshold values found to elicit reproducible cellular responses to ELF fields.

The shift in transmembrane potential associated with a 10- $\mu\text{A}/\text{cm}^2$ current density is expected to be about 10 μV at GWEN frequencies (150-175 kHz), where the effective membrane impedance is about 1 ohm/cm^2 . On the basis of both theoretical predictions and experimental measurements, approximately 0.01% of a transmembrane LF potential is rectified to a DC signal.^{19, 20} It can therefore be predicted that a 10- μV LF signal induced by GWEN fields would produce a DC transmembrane potential shift of about 1 nV, which is less by a factor of about 107 to 108 than the resting potential of eukaryotic cell membranes. A consideration of factors such as cell size, shape, and orientation relative to the field will not alter the conclusion that the transmembrane potential shift induced by GWEN fields is negligible.

Many possible interaction mechanisms that could lead to membrane transduction and amplification of weak electromagnetic signals have been discussed in theoretical terms.^{6, 7} The proposed mechanisms include cooperative phenomena (e.g., phase transitions) triggered by weak field interactions, strong dipolar oscillations induced in large membrane proteins, nonlinear wave excitations (e.g., solitons), and resonance interactions induced by the simultaneous presence of the geomagnetic field and a time-varying field with an appropriate frequency (e.g., nuclear magnetic resonance or ion-cyclotron resonance effects). Although such phenomena have been observed in model systems, they have not been demonstrated conclusively to produce substantial functional perturbations in biological systems. In addition, there are strong theoretical arguments against the existence of several classes of proposed interactions, especially the ion-cyclotron resonance model.

INDIRECT COUPLING—SHOCK AND BURNS

Commonly encountered ungrounded metallic objects—such as cars, vans, and buses—can develop open-circuit voltages from incident electric fields. Large currents can flow through a person who touches such an object, depending on the magnitude of the incident electric fields. If the incident fields are large enough, currents larger than those which produce perception, pain, or even burns can occur.

Gandhi and Chatterjee²¹ and Guy and Chou²² have studied the problem of shock hazard from ungrounded objects in RF fields for frequencies of 10-3,000 kHz. The body impedance and threshold currents needed to produce perception and pain were measured with 367 human subjects (197 male and 170 female; ages, 18-70 years).^{23, 24} Various types of contact with metallic electrodes were used to simulate situations in which a person would be in contact with a large ungrounded metallic object. It was found that the sensation is of tingling or pricking at frequencies below about 70-100 kHz and of warmth at frequencies higher than 100 kHz. For LF fields at 150.625-174.625 kHz, the sensation due to contact with ungrounded bodies would therefore be of warmth in the region of the contact. Both small-area contacts (25 and 144 mm²), such as finger contact, and larger-area contacts (cylindrical rod 1.5 cm in diameter and 14 cm long), such as a grasped handlebar of a vehicle, have been studied to establish the average threshold currents needed for perception and pain.^{23, 24} Measurements were performed only on adult subjects, but it was possible to predict thresholds for 10-year-old children by scaling the physical dimensions.

Gandhi et al.²³ and Chatterjee et al.²⁴ have estimated the threshold E fields that produce various sensations upon contact with various commonly metallic objects. Because somewhat larger currents would flow at 174.625 kHz than at 150.625 kHz and the threshold currents for various sensations are generally independent of frequency for frequencies higher than 100 kHz, somewhat smaller E fields can cause sensations at 174.625 kHz than at 150.625 kHz. Table 3-3 lists the estimated E fields for various sensations at 174.625 kHz related to contact with ungrounded metallic objects. The data could not be taken as threshold currents for pain under conditions of grasping contact because of the unavailability of high-power sources, but the currents are likely to be about 20% higher than those needed only for perception, as is the case for finger contact. The threshold E fields needed for pain under conditions of grasping contact would therefore be about 20% higher than the numbers given in the right-hand column of Table 3-3.

Table 3-3 shows that the vertical E fields needed for perception or pain are considerably larger than the 50 V/m measured for LF transmitters close to the 4-ft perimeter fence around the GWEN RNs. Inasmuch as both E and B fields diminish rapidly with increasing distance from the LF antenna, it is clear that the potential for shock and burns is low. An exception would be the erection of ungrounded metal towers or guy wires parallel to the E field within a few hundred meters of the antenna.

The phenomenon of perception, pain, or burns is peculiar to LF transmissions (less than about 50-100 MHz) and is relatively unimportant at UHF frequencies, where the dimensions of the ungrounded objects are larger than the wavelength and the concept of open-circuit voltage as a source of contact currents is irrelevant.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 3-3. Incident Vertical Threshold E Fields Needed to Produce Various Sensations in Contact with Ungrounded Metallic Objects

Object	Person	Finger contact, contact area = 144 mm ²		Grasping contact,
		E _{perception} , V/m	E _{pain} , V/m	E _{perception} , V/m
Compact car	Man	280	340	1,490
	Woman	300	400	1,200
Van	10-year-old child	205	255	950
	Man	155	180	735
	Woman	155	220	580
Bus	10-year-old child	120	140	430
	Man	160	200	480
	Woman	165	230	425
Fork-lift truck	10-year-old child	130	155	360
	Man	95	120	395
	Woman	90	125	330
50-ft fence	10-year-old child	65	80	260
	Man	420	450	2,700
	Woman	390	450	2,250
	10-year-old child	250	300	1,680

Source: O. P. Gandhi et al.²³; I. Chatterjee et al.²⁴

APPENDIX A: ANATOMICALLY BASED MODEL AND NUMERICAL PROCEDURE USED FOR CALCULATIONS

Anatomically Based Model

As described by Sullivan et al.,^{1,2} the inhomogeneous model of the human body is taken from A Cross-Section Anatomy²⁵ which contains cross-sectional diagrams of the human body that were obtained by making cross-sectional cuts at intervals of about 1 inch in human cadavers. The process for creating the data base of the man model was as follows: A 0.25 inch grid was taken for each cross-sectional diagram. Each cell on the grid was assigned a number corresponding to air or one of 16 tissue types—muscle, fat, bone, blood, intestine, cartilage, liver, kidney, pancreas, spleen, lung, heart, nerve, brain, skin, and eye. The data associated with a particular layer consisted of three numbers for each square cell: x and y positions relative to some anatomical reference point in the layer, usually the center of the spinal cord and an integer indicating which tissue the cell contained. Because the cross-sectional diagrams available in Eychleshymer and Schoemaker²⁵ are for variable separations, typically 2.3-2.7 cm, a new set of equispaced layers was defined at 0.25-inch (0.635-cm) intervals by interpolating the data onto the layers. Because the 0.25-inch cell size is too small for the memory space of readily accessible computers, the proportion of each tissue type was calculated for somewhat larger cells, 0.5 inch (1.27 cm) and the data on $2 \times 2 \times 2 = 8$ cells of the smaller dimension were combined. Without changes in the anatomy, the process allows some variability in the height and weight of the body. We have taken the final cell size of 1.31 cm (rather than 0.5 in.) to obtain the total height and body weight of 176.85 cm and 70 kg, respectively.

The electrical properties measured for the various tissues at both LF and UHF frequencies are shown in [Table A-1](#).

Finite-Difference Time-Domain Method

The finite-difference time-domain (FDTD) method was proposed by Yee²⁸ and developed by Umashankar and Taflove,²⁹ Holland,³⁰ and Kunz and Lee.³¹ It has been extended for calculations of the distribution of electromagnetic (EM) fields in a human model for incident plane waves,^{1,2,32-34} for pulsed exposures,³⁵ and for exposures in near fields.^{36,37} In the method, described in detail elsewhere,^{1,28,29,32} the coupled Maxwell's equations in the differential form are solved for various cubic subvolumes (cells) of the model and its surroundings in a time-stepped manner. To ensure stability of the solution, the time step δt is taken to be $\Delta/2v$ where Δ is cell size and v is maximum velocity of the EM wave encountered anywhere in the modeled space. For our calculations, $v = c$, is the velocity of EM waves in air. Because we have taken $\Delta = 1.31$ cm, the time step $\delta t = 0.02183$ ns.

TABLE A-1. Tissue Properties for Human Model^a

Tissue Type	Mass Density, ρ , kg/m ³	174.625 kHz σ , S/m	225 MHz σ , S/m ϵ_r	250 MHz σ , S/m ϵ_r	300 MHz σ , S/m ϵ_r	350 MHz σ , S/m ϵ_r
Air	1.2	0.0	0.0 1.0	0.0 1.0	0.0 1.0	0.0 1.0
Muscle	1,050	0.40	1.22 57	1.18 58	1.05 59	1.03 61
Fat, bone	1,020	0.05	0.075 6.6	0.08 7.2	0.09 8.3	0.10 9.5
Blood	1,000	0.68	1.16 65	1.17 65	1.19 65	1.20 65
Intestine	1,000	0.30	0.61 29	0.62 29	0.64 27	0.66 26
Cartilage	1,000	0.05	0.075 6.6	0.08 7.3	0.09 8.4	0.10 9.5
Liver	1,030	0.20	0.73 55	0.75 54	0.78 52	0.82 50
Kidney	1,020	0.34	1.09 64	1.11 62	1.14 58	1.16 53
Pancreas	1,030	0.45	1.09 67	1.11 64	1.14 58	1.16 53
Spleen	1,030	0.45	0.88 94	0.89 93	0.89 92	0.90 90
Lung ^b	330	0.05	0.21 12	0.22 12	0.22 12	0.22 12
Heart	1,030	0.40	0.87 59	0.90 58	0.90 57	1.0 56
Nerve, brain	1,050	0.18	0.60 60	0.61 58	0.61 55	0.62 51
Skin	1,000	0.34	0.79 56	0.78 54	0.77 51	0.76 48
Eye	1,000	0.45	1.85 80	1.80 78	1.70 74	1.60 70

Source: Johnson and Guy;²⁶ Stuchly and Stuchly.²⁷

^a σ , electrical conductivity; ϵ_r , dielectric constant (= relative permittivity).

^bWe have used 33% lung tissue and 67% air for calculating the electrical properties of the lung.

Both sinusoidal and prescribed time-varying incident fields can be used with the FDTD method. For sinusoidally varying fields, the solution is completed when a sinusoidal steady-state behavior is observed for each. For lossy biological bodies, this typically takes a stepped time on the order of 3 to 4 time periods of oscillation.

The method is relatively straightforward to use at UHF frequencies^{2, 34} because three to four periods of oscillation are not an inordinate lengths of time to cover with iterations of time step $\delta t = 0.02183$ ns. For calculations of mass-normalized rates of energy absorption (specific absorption rates, SARs), both maximum and minimum values of components E_x , E_y , and E_z in time are obtained for each cell. The SAR for the (i, j, k) cell in the body can then be calculated from

$$SAR(i,j,k) = \sum_{x,y,z} \frac{\sigma(i,j,k)[E_{x,max}(i,j,k) - E_{x,min}(i,j,k)]^2}{8\rho(i,j,k)} \quad (A-1)$$

where $\sigma(i, j, k)$ and $\rho(i, j, k)$ are the volume-averaged electrical conductivity and mass density for cell (i, j, k). From the individual SARs thus calculated, one can obtain the section-averaged SARs for each of the sections of the body that are 1.31 cm from each other and can obtain the whole-body-averaged SAR.

At low frequencies, use of the FDTD method is not as straightforward: a horrendous number of iterations would be needed to cover three to four periods of oscillation for converged results. A scaling procedure was recently developed that recognizes the quasistatic nature of coupling at lower frequencies, as previously pointed out by Kaune and Gillis³⁸ and Guy et al.³⁹ According to a logic similar to that of those authors, the fields outside the body depend not on the internal tissue properties, but only on the shape of the body, as long as the quasistatic approximation is valid; that is, the size of the body is smaller by a factor of 10 or more than the wavelength, and $|\sigma + j\omega\epsilon| \gg \omega\epsilon_0$, where σ and ϵ are the conductivity and the permittivity of the tissues, respectively, $\omega = 2\pi f$ is the radian frequency, and ϵ_0 is the permittivity of the free space outside the body. Under those conditions, the fields in air are normal to the body surface and the internal tissue fields are given from the boundary conditions in terms of the fields outside:

$$j\omega\epsilon_0 \hat{n} \cdot E_{air} = (\sigma + j\omega\epsilon) \hat{n} \cdot E_{tissue} \quad (A-2)$$

A higher quasistatic frequency f' may therefore be used for irradiation of the model, and the internal fields E' thus calculated may be scaled back to the frequency f of interest, e.g., 174.625 kHz. From Equation A-2, we can write

$$E_{\text{tissue}}(f) = \frac{\omega}{\omega'} \frac{(\sigma' + j\omega'\epsilon')}{(\sigma + j\omega\epsilon)} E'_{\text{tissue}}(f') \approx \frac{f\sigma'}{f'\sigma} E_{\text{tissue}}(f') \quad (\text{A-3})$$

assuming that $\sigma + j\omega\epsilon \approx \sigma$ at both f' and f , which is a close approximation.

For our calculations, we have used a full-scale anatomically based model of the human body and a frequency f' of 10 MHz to reduce the computation time by orders of magnitude. Because in the FDTD method one needs to calculate in the time domain until convergence is obtained (typically three to four periods), frequency scaling to 10 MHz for f' reduces the needed number of iterations by almost a factor of 60. At the higher irradiation frequency σ' , we have taken $\sigma' = \sigma$, i.e., conductivities of the various tissues at 174.625 kHz (see Table A-1). Furthermore, we have taken the incident E field $E_i(f) = fE_i(f)/f'$ to obtain $E_{\text{tissue}}(f)$ at, say, $E_i(f) = 50$ V/m. The incident B field $H_i(f)$ has similarly been taken to be considerably lower, $fH_i(f)/f'$, in recognition that currents induced are proportional to the frequency of the incident fields.

Quantities of interest for the LF band are the internal total electric fields and current densities for the various regions of the body. We have used the calculated internal fields to obtain the total electric field E_T (i, j, k) and current density J_T (i, j, k) for (i, j, k) cell in the body with the following equations:

$$E_T(i,j,k) = \left[\sum_{x,y,z} \frac{1}{8} [E_{x,\max}(i,j,k) - E_{x,\min}(i,j,k)]^2 \right]^{1/2} \quad (\text{A-4})$$

$$J_T(i,j,k) = \sigma(i,j,k)E_T(i,j,k) \quad (\text{A-5})$$

REFERENCES

1. Sullivan, D. M., D.T. Borup, and O. P. Gandhi. 1987. Use of the finite-difference time-domain method in calculating EM absorption in human tissues. *IEEE Trans. Biomedical Engineering BME* 34:148-57.
2. Sullivan, D. M., O. P. Gandhi, and A. Taflove. 1988. Use of the finite-difference time-domain method in calculating EM absorption in man models. *IEEE Trans. Biomedical Engineering BME* 35:179-186.
3. Chatterjee, I., M. J. Hagmann, O. P. Gandhi. 1981. An Empirical Relationship for Electromagnetic Absorption in Man for Near-Field Exposure Conditions. *IEEE Trans. on Microwave Theory and Techniques MTT-29*:1235-1238.
4. Gandhi, O. P., J. Y. Chen, and A. Riazi. 1986. Currents induced in a human being for plane-wave exposure conditions 0-50 MHz and for RF sealers. *IEEE Trans. Biomedical Engineering BME* 33:757-767.
5. Institute for Electric and Electronic Engineers Standards Coordinating Committee (SCC) 28, IEEE C95.1, 1991. IEEE Standard for Safety Levels with Respect to Human Exposure to Radiofrequency Electromagnetic Fields, 3 kHz to 300 GHz. Approved September 26, 1991.
6. Taylor, L. S. 1981. The mechanisms of athermal microwave biological effects. *Bioelectromagnetics* 2:259-267.
7. Postow, E., and M. L. Swicord. 1986. Modulated fields and "window" effects. Pp. 425-460 in *Handbook of Biological Effects of Electromagnetic Fields*, C. Polk and E. Postow, eds. Boca Raton, FL: CRC Press.
8. Tenforde, T. S., and T. F. Budinger. 1986. Biological effects and physical safety aspects of NMR imaging and in vivo spectroscopy. Pp. 493-548 in *NMR in Medicine: Instrumentation and Clinical Applications*, S. R. Thomas, and R. L. Dixon, eds. Medical Physics Monograph No. 14. New York, NY: American Association of Physicists in Medicine.
9. Michaelson, S. M., and J. C. Lin. 1987. *Biological Effects and Health Implications of Radiofrequency Radiation*. New York, NY: Plenum Press.
10. Blackman, C.F. 1990. ELF effects on calcium homeostasis. Pp. 187-208 in *Extremely Low Frequency Electromagnetic Fields: The Question of Cancer*, B. W. Wilson, R. G. Stevens and L. E. Anderson, eds. Columbus, OH: Battelle Press.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

11. Merritt, J. H., W. W. Shelton, and A. F. Chamness. 1982. Attempts to alter $^{45}\text{Ca}^{2+}$ binding to brain tissue with pulse-modulated microwave energy. *Bioelectromagnetics* 3:475-478.
12. Adey, W.R. 1981. Tissue interactions with nonionizing electromagnetic fields. *Physiol. Rev.* 61:435-514.
13. Tenforde, T. S., and W. T. Kaune. 1987. Interaction of extremely-low-frequency electric and magnetic fields with humans. *Health Phys.* 53:585-606.
14. Tenforde, T. S. 1991. Biological interactions of extremely-low-frequency electric and magnetic fields. *Bioelectrochem. Bioenerget.* 25:1-17 [*J. Electroanalyt. Chem.* 320:1-17].
15. Tenforde, T. S. 1992. Biological interactions and potential health effects of extremely-low-frequency magnetic fields from power lines and other common sources. *Annu. Rev. Publ. Health* 13:173-196.
16. Bernhardt, J. 1979. The direct influence of electromagnetic fields on nerve and muscle cells of man within the frequency range of 1 Hz to 30 MHz. *Radiat. Environ. Biophys.* 16:309-323.
17. Cole, K. S. 1968. *Membranes, ions, and impulses*. Berkeley, CA: University of California Press.
18. Foster, K. R., and H. P. Schwan. 1986. Dielectric properties of tissues. Pp. 27-96 in *Handbook of Biological Effects of Electromagnetic Fields*, C. Polk and E. Postow, eds. Boca Raton, FL: CRC Press.
19. Pickard, W. F., and F. J. Rosenbaum. 1978. Biological effects of microwaves at the membrane level: Two possible athermal electrophysiological mechanisms and a proposed experimental test. *Math. Biosci.* 39: 235-253.
20. Montaigne, K., and W. F. Pickard. 1984. Offset of the vacuolar potential of Characean cells in response to electromagnetic radiation over the range 250 Hz-250 kHz. *Bioelectromagnetics* 5:31-38.
21. Gandhi, O. P., and I. Chatterjee. 1982. Radio-frequency hazards in the VLF to MF band. *Proc. IEEE* 70:1462-1464.
22. Guy, A. W., and C.-K. Chou. 1985. Very low frequency hazard study—Part I. *Bioelectromagnetics Research Laboratory*, University of Washington, Seattle.

23. Gandhi, O. P., I. Chatterjee, D. Wu, J. A. D'Andrea, and K. Sakamoto. 1985. Very low frequency hazard study. USAFSAM-TR-84, USAF School of Aerospace Medicine, Brooks AFB, Texas.

24. Chatterjee, I., D. Wu, and O. P. Gandhi. 1986. Human body impedance and threshold currents for perception and pain for contact hazard analysis in the VLF-MF band. *IEEE Trans. Biomedical Engineering BME* 33:486-494.

25. Eycleshymer, A. C., and D. M. Schoemaker. 1970. *A Cross-Section Anatomy*. New York, NY: Appleton-Century-Crofts.

26. Johnson, C. C., and A. W. Guy. 1972. Nonionizing electromagnetic wave effects in biological materials and systems. *Proc. IEEE* 60:692-718.

27. Stuchly, M. A., and S. S. Stuchly. 1980. Dielectric properties of biological substances—Tabulated. *J. Microwave Power* 15:19-26.

28. Yee, K. S. 1966. Numerical solution of initial boundary value problems involving Maxwell's equations of isotropic media. *IEEE Trans. Antennas and Propagation AP* 14:302-307.

29. Umashankar, K., and A. Taflove. 1982. A novel method to analyze electromagnetic scattering of complex objects. *IEEE Trans. Electromagnetic Compatibility EMC* 24:397-405.

30. Holland, R. 1977. THREDE: A free-field EMP coupling and scattering code. *IEEE Trans. Nuclear Science NS* 24:2416-2421.

31. Kunz, K. S., and K. M. Lee. 1978. A three-dimensional finite-difference solution of the external response of an aircraft to a complex transient EM environment: Part 1-The method and its implementation. *IEEE Trans. Electromagnetic Compatibility EMC* 20:328-332.

32. Spiegel, R. J. 1984. A review of numerical models for predicting the energy deposition and resultant thermal response of humans exposed to electromagnetic fields. *IEEE Trans. Microwave Theory and Tech MTT* 32:730-746.

33. Spiegel, R. J., M. B. E. Fatmi, and K. S. Kunz. 1985. Application of a finite-difference technique to the human radio-frequency dosimetry problem. *J. Microwave Power* 20:241-254.

34. Chen, J. Y., and O. P. Gandhi. 1989. RF currents induced in an anatomically-based model of a human for plane-wave exposures (20-100 MHz). *Health Phys.* 57:89-98.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

COUPLING OF GWEN ELECTROMAGNETIC FIELDS TO THE HUMAN BODY	44
35. Chen, J. Y., and O. P. Gandhi. 1991. Currents induced in an anatomically-based model of a human for exposure to vertically polarized electromagnetic pulse. <i>IEEE Trans. Microwave Theory and Tech</i> MTT 39:31-39.	
36. Wang, C. Q., and O. P. Gandhi. 1989. Numerical simulation of annular phased arrays for anatomically based models using the FDTD method. <i>IEEE Trans. Microwave Theory and Tech</i> MTT 37:118-126.	
37. Chen, J. Y., and O. P. Gandhi. 1989. Electromagnetic deposition in an anatomically based model of man for leakage fields of a parallel-plate dielectric heater. <i>IEEE Trans. Microwave Theory and Tech</i> MTT 37:174-180.	
38. Kaune, W. T., and M. F. Gillis. 1981. General properties of the interaction between animals and ELF electric fields. <i>Bioelectromagnetics</i> 2:1-11.	
39. Guy, A. W., S. Davidow, G. Y. Yang, and C. K. Chou. 1982. Determination of electric current distributions in animals and humans exposed to a uniform 60-Hz high intensity electric field. <i>Bioelectromagnetics</i> 3:47-71.	

4

Perception and Behavioral Effects of Electromagnetic Fields

Some animals respond to extremely low levels of electromagnetic fields (EMF), usually at frequencies ranging from DC to extremely-low-frequency (ELF) and usually with specialized receptors.¹ Although the responses have been described and can be demonstrated at will, the mechanisms are not understood. Perceptual and behavioral responses to very low levels of EMFs at low frequency and above have not been reported in humans, and there are no mechanisms at DC or ELF that might imply as yet unreported responses, although they cannot be excluded. There is a considerable literature on perception and behavioral responses to stimulation at magnitudes consistent with direct electrical stimulation of nervous tissues by induced currents in the tissue, but they are limited to the frequency ranges that stimulate excitable membranes.

In the radiofrequency (RF) range of concern for assessment of GWEN sites, there have been many reports of sensory perception. The responses may be organized in the following categories:

- Stimulation of nervous structures by electric and magnetic fields and associated currents in the body. Above a threshold that is frequency-dependent, these currents are perceived as a painful stimulus that increases with current intensity.
Electroencephalographic (EEG) activity in cats and rabbits has been reported to be altered by exposure to amplitude-modulated RF. Exposure to 147-MHz fields, amplitude-modulated between 1 and 25 Hz, altered the ability of cats to produce selected EEG rhythms. Changes in EEG frequency spectrum were also observed in rabbits chronically exposed to 1-10 MHz fields that were amplitude-modulated at 14-16 Hz.² Other studies have shown small changes in EEG patterns, particularly desynchronization, in rats and rabbits after exposure to 12.95-GHz field at 1 W/kg.^{3,4} Some later studies failed to find an effect. The lowest levels used, in the above studies are 10,000 times that which would be encountered near GWEN installations.
- Shocks and burns. When the human body is in an EMF of suitable frequency and intensity and it makes contact with a conducting body in the same field, an electrical current is produced that can cause perceptible electrical shock, muscular contractions, burns, and possible death.
- Heating. If enough RF power is absorbed in human tissue, especially skin, it can raise the tissue temperature and cause a sensation of warming that will be due to thermal stimulation of temperature receptors. Thermal perception of absorbed RF energy is frequency-dependent: the threshold energy decreases as the frequency increases.⁵ There is a delay in the perception of warmth after the start of irradiation; the delay may vary from 5 sec or more at GHz frequencies to as little as 1 sec for

infrared radiation.⁶ Justesen et al.⁷ compared thermal perception in human volunteers who were irradiated on the forearm in a 100-cm² area with far-infrared radiation or 2.45-GHz microwave radiation. The thresholds of perception for a 10-sec exposure were 1.7 mW/cm² for far infrared radiation and 26.7 mW/cm² for microwave radiation.

- Auditory perception. A special effect has been reported in which microwave RF emitted in the form of very short pulses (1-20 μ sec) is perceived by humans and animals as clicks or other sounds. This perception could well result when thermal absorption leads to thermoelastic expansion of tissues and fluids in the head and is sensed by auditory receptors. If the energy flux in the pulse exceeds about 40 μ J/cm², delivered in a few microseconds, auditory perceptions occur.⁸

The auditory perception of pulsed microwave fields was first reported in 1947 and has been studied extensively. Frey⁹ reported on controlled experimental exposures at frequencies of 0.2-8.9 GHz and pulse widths of 1-1,000 μ sec. He found that, depending on the characteristics of the field, sensations were perceived as buzzing, ticking, hissing, or knocking sounds. Sound was perceived at all frequencies up to 8.9 GHz. Guy et al.¹⁰ demonstrated that the threshold for auditory perception was four times higher at 3.75 kHz in subjects with neurosensory deficits compared with normal subjects, thus indicating that the effect was in the acoustic elements involved in hearing.

- Behavioral changes. Epidemiologic studies of groups of people occupationally or environmentally exposed to electromagnetic fields in the RF and ELF range have yielded perceptual and behavioral responses, including fatigue, difficulty in concentrating, and increased frequency of headaches. A number of researchers have used disruption of behavior patterns, such as work stoppage, to study the effect of RF fields on animals, including rodents¹¹⁻¹³ and monkeys.¹⁴ Several carrier frequencies, field zones, and modulation characteristics were used. A relatively narrow range of threshold of specific absorption rates (SARs), about 4-9 W/kg, was found. Lebovitz¹⁵ examined the effect of repeated exposures to a pulsed 1,300-MHz field on behavioral performance in rats. He exposed animals to SARs of 1.6, 3.6, or 6.7 W/kg for 3 h/day, 5 d/wk for 6-9 wk and found that rates of lever-pressing for food were slightly reduced at the highest SAR. However, the ability of the rats to discriminate improved as a positive function of SAR when lever-pressing was not reinforced by the presence of food. DeLorge¹⁶ used a different experimental paradigm and showed a disruption in performance of rats at an SAR of 2.5 W/kg when they were exposed to a pulsed 1,300-MHz field. Behavioral studies performed by Hjeresen et al.¹⁷ showed that rats placed in a shuttlebox tended to remain in the side shielded from pulsed RF fields. Because the rats also tended to avoid pulsed sound waves, the investigators suggested that the rats' avoidance of the RF fields might be related to the hearing of the pulsed fields. The response to a pulsed field is stronger than that to a continuous field. For example, Carroll et al.¹⁸ found that rats exposed to an intense field that was not pulse-modulated did not readily learn to escape from it. It appears that hearing the pulses is a more effective cue for escaping than is the warming that results from a continuous field.

Studies in Eastern Europe have investigated populations exposed to EMFs ranging from 50 Hz to microwave frequencies.¹⁹ Complaints included irritability, lethargy, insomnia, impotence, headaches, loss of memory, and inability to concentrate. The syndrome was identified as neurasthenia or "microwave sickness." Energy magnitudes associated with the syndrome have been reported for a few microwatts to a few thousand microwatts per square centimeter. An epidemiologic study of the personnel in the American embassy in Moscow found an excess of the same neurasthenic symptoms, but the symptoms were not correlated with measured individual exposures.²⁰

Eastern European investigators have also reported on rats and rabbits exposed for one to several hours a day over periods of weeks or months. Power densities ranging from 0.6 to 30,000 $\mu\text{W}/\text{cm}^2$ were reported to alter conditional reflexes²¹ and decrease latency of audiogenic seizures.²² Attempts to confirm the findings were made by several investigators; some effects of exposure to RF fields were found, but most were at higher field intensities, and in general the results did not support the findings from Eastern Europe.

Studies with ELF fields have suggested that behavior can be influenced by exposure to either magnetic or electric fields. Persinger²³ reported that prenatal exposure of rats to 0.5-Hz, 0.05-to 3-mT fields, resulted in changes in juvenile or adult rats' emotionality and ability to perform a conditioned-suppression test. Frey²⁴ found that prenatal exposure of rats to a 60-Hz field at 3.5 kV/m caused changes in open-field activity.

McGivern et al.²⁵ studied male rats that had been exposed prenatally on days 15-21 of gestation. Exposures were for 15 min twice a day, to a 15-Hz, 800- μT pulsed magnetic field. Exposed animals showed a significant reduction in scent marking, compared with sham-exposed or caged controls. Exposed males had larger seminal vesicles, prostates, and epididymides than did control males.

Transient neurobehavioral changes in rats exposed prenatally and postnatally to a 60-Hz electric field (65-kV/m effective field) were reported by Sikov et al.²⁶ Exposed animals showed significantly more motility than did controls. Development of righting reflex and negative geotropism was also delayed in exposed rats; the percentage of pups that failed to show these behaviors was increased on day 14 of postnatal life, but not on day 21.

Lovely et al.²⁷ exposed gravid Sprague-Dawley rats to a 60-kV/m, 60-Hz electric field and then tested the offspring at the age of 90 days in three tasks: shuttlebox avoidance, a residential maze, and a preference-avoidance test. No differences were noted between exposed and sham-exposed animals.

Changes in learning have been reported in rats exposed to a combination of 60-Hz electric fields (30 kV/m and 0.1 mT) and magnetic fields (10 kV/m and 0.033 mT) throughout gestation and during the first 8 days of postnatal life.²⁸ Both acquisition and extinction of a schedule-controlled response were affected in the exposed animals. In contrast, other studies failed to find an effect of exposure to ELF fields on behavior.^{29,30}

Exposure to 60-Hz electric fields (30 or 60 kV/m) has been reported to affect the social behavior of baboons.^{31,32} The investigators used a number of measures of social behavior, but found that passive affinity, tension, and stereotypy performance were significantly increased in exposed groups. The authors suggested that the changes might indicate a stress response to the fields.

Although there is evidence that exposure of experimental animals to electric or magnetic fields can influence neurobehavioral function, there is a paucity of direct observations at the 175-kHz frequency and at the ultra-high frequencies of 200400 MHz used in the GWEN system. Moreover, magnitudes of the exposure usually required to produce an effect are substantially higher than those likely to be encountered as a result of operation of the GWEN system. It therefore seems unlikely that electromagnetic fields from GWEN will affect neurosensory or neurobehavioral function in persons living around GWEN sites.

REFERENCES

1. Kalmijn, A. J. 1978. Experimental evidence of geomagnetic orientation in elasmobranch fishes. Pp. 347-353 in *Animal Migration, Navigation and Homing*, K. Schmidt-Konig, and W. K. Keeton, eds. New York: Springer Verlag.
2. Takeshima, S., B. Onaral, and H. P. Schwan. 1979. Effects of modulated RF energy on the EEG of mammalian brains: effects of acute and chronic irradiations. *Radiat. Environ. Biophys.* 16:15-27.
3. Baranski, S., and Z. Edelwejn. 1975. Experimental morphologic and electroencephalographic studies of microwave effects on the nervous system. *Ann. N.Y. Acad. Sci.* 277:109-116.
4. Servantie, B., A.M. Servantie, and J. Etienne. 1975. Synchronization of cortical neurons by a pulsed microwave field as evidenced by spectral analysis of electrocorticograms from the white rat. *Ann. N.Y. Acad. Sci.* 247:82-86.
5. Hendler, F., J. D. Hardy, and D. Murgatroyd. 1963. Skin temperature and temperature sensation produced by infrared and microwave irradiation. *Biology and Medicine Vol. 3, Temperature: its measurement and control in Science and Industry*, C. M. Hertzfeld, ed. New York: Reinhold Publishing Corp.
6. Eijkman, E., and A. J. H. Vendrik. 1961. Dynamic behavior of the warmth sense organ. *J. Exp. Psychol.* 62:403-408.
7. Justesen, D. R., E. R. Adair, J. C. Stevens, and V. Bruce-Wolfe. 1982. A comparative study of human sensory thresholds: 2450-MHz microwaves vs. far-infrared radiation. *Bioelectromagnetics* 3:117-125.
8. Lin, J. C. 1990. Auditory perception of pulsed microwave radiation. Pp. 277-318 in *Biological Effects and Medical Applications of Electromagnetic Energy*, O. P. Ghandi, ed. Englewood Cliffs, New Jersey: Prentice Hall.
9. Frey, A. H. 1961. Auditory system response to modulated electromagnetic energy. *J. Appl. Phys.* 17:689-692.
10. Guy, A. W., C.-K. Chou, J. C. Lin, and D. Christensen. 1975. Microwave induced acoustic effects in mammalian auditory systems and physical materials. *Ann. N.Y. Acad. Sci.* 247:194-218.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

11. Justesen, D. R., and N. W. King. 1970. Behavioral effects of low level microwave irradiation in the closed space situation. Pp. 154-179 in *Biological Effects and Health Implications of Microwave Radiation*, S. F. Cleary, ed. HEW Publication BRH/DBE 70-2.
12. Lin, J.C. 1977. On microwave-induced hearing sensation. *IEEE Trans. Microwave Theory Tech.* 25:605-613.
13. D'Andrea, J. A., O. P. Gandhi, and J. L. Lords. 1977. Behavioral and thermal effects of microwave radiation at resonant and nonresonant wavelengths. *Radio Sci.* 12:251-256.
14. DeLorge, J. O. 1979. Operant behavior and rectal temperature of squirrel monkeys during 2.45 GHz microwave irradiation. *Radio Sci.* 12:217-225.
15. Lebovitz, R. M. 1981. Prolonged microwave irradiation of rats: effects on concurrent operant behavior. *Bioelectromagnetics* 2:169-185.
16. DeLorge, J. O. 1983. The thermal basis for disruption of operant behavior by microwaves in three species. Pp. 379-399 in *Microwaves and Theroregulation*, E. R. Adair, ed. Academic Press: New York.
17. Hjeresen, D. L., S. R. Doctor, and R. L. Sheldon. 1979. Pp. 194-214 in *Proceedings of the Symposium on Electromagnetic Fields in Biological Systems*, S. S. Stuchly, ed. International Microwave Power Institute, Edmonton, Canada.
18. Carroll, D. R., D. M. Levinson, D. R. Justesen, and R. L. Clarke. 1980. Failure of rats to escape from a potentially lethal microwave field. *Bioelectromagnetics* 1:101-115.
19. Sadchikova, M. W. 1974. Clinical manifestations of reactions to microwave irradiation in various occupational groups. Pp. 261-268 in *Biological Effects and Health Hazards of Microwave Radiation*, P. Czerski, K. Ostraowski, C. Silverman, M. L. Shore, M. J. Suess, and B. Waldeskog, eds. Warsaw: Polish Medical Publishers.
20. Lilienfeld, A.M., J. Tonascia, S. Tonascia, C. H. Libauer, G. M. Cauthen, J. A. Markowitz, and S. Weida. 1978. Foreign Service Health Status Study-Evaluation of the Health Status of Foreign Service and Other Employees from Selected Eastern European Posts. Final Report Contract 6025-619073. Washington, D.C.: Department of State.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

21. Lobanova, E. A. 1974. The use of conditional reflexes to study microwave effects on the central nervous system. Pp. 109-118 in *Biologic Effects and Health Hazards of Microwave Radiation*, P. Czerski et al., eds. Warsaw: Polish Medical Publishers.

22. Stverak, I., K. Marha, and G. Pafkova. 1974. Some effects of various pulsed fields on animals with audiogenic epilepsy. 1974. Pp. 141-144 in *Biologic Effects and Health Hazards of Microwave Radiation*, P. Czerski, K. Ostrowski, C. Silverman, J. L. Shore, M. J. Suess, and B. Waldeskog, eds. Warsaw: Polish Medical Publishers.

23. Persinger, M. A. 1969. Open field behavior in rats exposed prenatally to a low intensity-low frequency, rotating magnetic field. *Developmental Psychobiol.* 2:168-171.

24. Frey, A. H. 1982. Neural and behavioral consequences of prenatal exposure to 3.5 kV/m 60 Hz fields. Abstr. 4th Annual Meeting Bioelectromagnetics Society, Los Angeles, California.

25. McGivern, R. F., R. Z. Sokol, and W. R. Adey. 1990. Prenatal exposure to low-frequency electromagnetic field demasculinizes adult scent marking behavior and increases accessory sex organ weights in rats. *Teratology* 41:1-8.

26. Sikov, M. R., L. D. Montgomery, L. G. Smith, and R. D. Phillips. 1984. Studies on prenatal and postnatal development in rats exposed to 60-Hz electric fields. *Bioelectromagnetics* 5:101-112.

27. Lovely, R. H., J. A. Creim, and R. D. Phillips. 1984a. Adult behavioral effects of prenatal and early postnatal exposure to 60-Hz electric fields in rats. In *Interaction of Electromagnetic Fields with Biological System*. Twenty-first General Assembly of the International Union of Radio Science (URSI), August 27-30, 1984, Florence, Italy (abstract).

28. Salzinger, K., S. Freimark, M. McCullough, D. Phillips, and L. Birenbaum. 1990. Altered operant behavior of adult rats after perinatal exposure to a 60-Hz electromagnetic field. *Bioelectromagnetics* 11:105-116.

29. Lovely, R. H., J. A. Creim, and R. D. Phillips. 1984. Effects of prenatal exposure to 60-Hz electric fields on open field and maze performance of F-2 generation Hanford Miniature swine. Pg. 10 in *Sixth Annual Scientific Session of the Bioelectromagnetics Society*, July 15-19, Atlanta, Georgia (abstract).

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

30. Durfee, W. K., P. R. Plante, P. Martin, S. Muthukrishnan, and C. Polk. 1976. Exposure of domestic fowl to ELF electric and magnetic fields. In *Biological Effects of Electromagnetic Waves*, C. C. Johnson, and M. L. Shore (eds). Selected papers of the USNC/URSI Annual Meeting, Boulder, CO, October 20-23, 1975. Vol. 1. Washington, D.C.: U.S. Government Printing Office.

31. Easley, S. P., A. M. Coelho, Jr., and W. R. Rogers. 1991. Effects of exposure to a 60 kV/m, 60-Hz electric field on the social behavior of baboons. *Bioelectromagnetics* 12:361-375.

32. Coelho, A. M., Jr., G. P. Easley, and W. R. Rogers. 1991. Effects of exposure to a 60 kV/m, 60-Hz electric field on the social behavior of baboons. *Bioelectromagnetics* 12:361-375.

5

Effects of Electromagnetic Fields on Development

Developing organisms are highly sensitive to physical and chemical agents. For example, the teratologic effects of embryonic or fetal exposures to ionizing radiation that do not produce oven damage to adults are well documented.¹ Developing organisms are therefore often used as indicators of biologic effects produced by exposure to various agents.

Various studies have examined the effects of electromagnetic fields (EMFs) on developing organisms, but most have been conducted under conditions different from those associated with operation of the GWEN system. Many of the studies have used nonmammals, including fish, drosophila, and chickens. For example, Zimmerman et al.² reported that exposure of fertilized sea urchin eggs to a 60-Hz, 0.1-mT field for 24 h delayed development by about 1 h; no other developmental abnormalities were found. Cameron et al.³ found developmental delays in fish embryos, but no gross abnormalities; several possible mechanisms for the developmental delays were postulated including changes in transcription, alterations in mass migration of cells, and changes in cellular free calcium—but additional data are needed to evaluate the suggestions.

Delgado and co-workers⁴⁻⁷ aroused a great deal of interest among investigators working with EMF when they exposed fertilized chicken eggs to pulsed magnetic fields with repetition frequencies of 10, 100, and 1,000 Hz at intensities of 0.12, 1.2, and 12 μ T. They found dramatic effects of the exposures; exposure to the 100-Hz 1.2- μ T field produced the greatest effects. There was a generalized inhibition of development. Brain vesicles, the auditory pit, the neural tube, the foregut, heart vessels, and somites were all affected; the cephalic nervous system was the most sensitive, and the heart the least. They also compared various pulse shapes and modalities for effects on development. They used pulses of 0.4, 1.0, 10.4, 13.9, and 104 μ T with rise and fall times of 100 μ sec, a declining plateau, and long-duration postpulse negative amplitude. Exposure at 0.4 μ T resulted in a slight decrease in incidence of developmental abnormalities. Exposures at 1.0 and 13.9 μ T produced a 50-77% incidence of abnormalities. The 13.9- μ T group had an increased incidence of heart and vessel abnormalities and a delay in development. Heart and vessel abnormalities were also found in the 1.0 μ T group.

Other groups of eggs were exposed to a square-wave pulse of 0.4 μ T with a 2- μ sec rise and fall time; this exposure regimen resulted in an 83% incidence of abnormal embryos, compared with 35% in the controls. Another exposure regimen used a generally square wave of 1.0 μ T with rise and fall times originally reported as 42 μ sec, but later corrected to 1.7 μ sec; this resulted in a 71% incidence of abnormalities vs. a 17% incidence in the controls. The use of the 0.4- μ T pulse produced increases in

abnormalities in all five embryonic systems examined—the cephalic nervous system, truncal nervous system, heart, vessels, and somites. Particularly obvious were the short truncal nervous systems with large open folds and abnormal torsion of the embryos. In addition, the embryos exposed to $0.4 \mu\text{T}$ were more advanced than the normal controls or the controls with abnormalities. The advance in development was estimated to be 8-9 h. In contrast, embryos exposed to $1.0 \mu\text{T}$ showed a delay in development of approximately 10 h. Of some concern in the latter study is the apparent lack of double-blind evaluation of the embryos. The noncontinuity or lack of a dose-response relation is also of concern, despite the general consideration of "windows" in the EMF area.

Leal et al.⁶ analyzed their data on chick development relative to the intensity of the earth's horizontal magnetic field. They suggested that the incidence of abnormalities in their control animals increased as the field increased and resulted in a lower apparent excess of abnormalities in exposed animals. The roughly even split in their data from 13 experiments between those which showed an increased incidence of abnormalities after exposure to pulsed magnetic fields and those which showed a decreased incidence did not give credibility to their suggestion. Chacon et al.⁷ extended the studies by exposing fertilized eggs to a bipolar pulse with a $1\text{-}\mu\text{T}$ amplitude, $2\text{-}\mu\text{sec}$ rise and fall times, $500\text{-}\mu\text{sec}$ width, and 30-Hz repetition rate. Eggs were placed with their narrow ends toward the west. Examination of embryos was blind. The incidence of abnormal embryos was not significantly affected by EMF exposure, although the incidence of malformed or underdeveloped optic vesicles appeared to be higher in exposed embryos. The proportion of nondeveloping eggs was larger in the exposed group.

Maffeo et al.⁸ attempted to repeat the experiments of Delgado et al. by exposing fertilized chicken eggs to 1.2- or $12\text{-}\mu\text{T}$ pulsed fields at 100 or 1,000 Hz for 48 h. No differences were found between sham-exposed and exposed eggs. In some replicates, controls (not sham-exposed) had a higher incidence of abnormalities than did the sham-exposed or exposed eggs. There seemed to be a high degree of variability among replicates.

Maffeo et al.⁹ attempted to repeat the work of Ubeda et al. on effects of pulse shape on chick-embryo development. They used a $1\text{-}\mu\text{T}$ field with a $42\text{-}\mu\text{sec}$ rise and fall time and 0-5 msec pulse duration. The repetition rate was 100 Hz. Positive controls were x-irradiated with 1,552 rads. Exposure to EMF did not alter the incidence of abnormalities. Exposure to x rays consistently produced abnormalities.

Juutilainen and colleagues also studied the effects of magnetic fields on chick-embryo development.¹⁰⁻¹² Juutilainen et al.¹⁰ compared 100-Hz sinusoidal, square (unipolar and bipolar), and pulsed waveforms at field strengths of 0.1-80 A/m ($0.12\text{-}96 \mu\text{T}$). The incidence of abnormal embryos was increased by exposure to all waveforms at or above 1 A/m except the unipolar square wave. The anterior part of the neural tube was particularly sensitive, but in the most severe cases the whole

embryo was malformed. Development may have been delayed by the 0.1 A/m unipolar waveform, but not by other waveforms or at other field strengths. There was no evidence of an intensity window for production of abnormalities, but the data suggested a threshold at about 1 A/m. There also were no differences between effects produced by the sinusoidal, square, and pulsed fields. Although the unipolar and bipolar waveforms generate identical electric fields, no abnormalities were found with the unipolar waveform; this suggests that it might not be induced fields that are responsible for the effects.

Juutilainen and Saali¹¹ used a sinusoidal field with frequencies of 1, 10, 16.7, 30, 1,000, 10,000, and 100,000 Hz to study the effects of frequency on development. Field strengths of 0.1, 1, 10, and 100 A/m were used. The incidence of abnormalities increased in groups exposed to fields of 1 A/m or greater at 16.7-100,000 Hz. There did not appear to be a dose-response relation above 1 A/m level, nor did there appear to be a window above 16.7 Hz. None of the fields affected the stage of development.

Juutilainen et al.¹² further examined the role of field strength on chick development by exposing embryos to a 50-Hz field of varied magnetic intensity. Again, field strengths below 1 A/m did not affect the incidence of abnormalities. However, all field strengths of 1 A/m and above increased the incidence of abnormalities from approximately 16% to 29-36%. The stage of development was unaffected. In general, the affected animals in this and other studies by the Juutilainen group had neural tube defects, half of which were classified as mild and half as severe.

The difference in results obtained by various investigators led to a comparative study among six laboratories. The study, dubbed "Operation Henhouse," was funded by the U.S. Office of Naval Research, the Ontario Ministry of Labor, the Spanish Ministry of Health, the Swedish National Institute of Occupational Health, the U.S. Food and Drug Administration, and the U.S. Environmental Protection Agency.¹³ The study involved laboratories in Canada, Spain, Sweden, and the United States. It was designed so that each laboratory would use identical equipment and protocols. They used an unipolar, pulsed magnetic field (500 μ sec pulse duration, 100 pulses/sec, 1- μ T peak flux density, and 2- μ sec rise and fall time). The field was applied for 48 h, and then the eggs were evaluated for development, structure, and stage of maturity. Of the six laboratories, two had significantly higher incidences of abnormalities in the exposed group. Combining all the data from all six laboratories led to an overall increase in the incidence of abnormalities that was statistically significant. Other measures did not differ between exposed and control groups.

Martin¹⁴ had reported an increased incidence of malformations in chick development when eggs were exposed to an unipolar, 60-Hz, 1 μ T magnetic field. In a later experiment, however, Martin found no differences in incidences of malformations or death between exposed and control eggs when 60-Hz, 3- μ T unipolar, bipolar, or split-sine waves were used.

The response of developing mammal to EMF exposure has been less intensively examined than that of other animals. However, a few studies with mice and rats have examined the effect of electric or magnetic fields on the incidence of fetal abnormalities. Marino et al.¹⁵ reported decreased weight gains in mice prenatally and postnatally exposed to vertical (15-kV/m) or horizontal (10-kV/m) electric fields. The watering tubes were not grounded, so it is likely that microshocks influenced the growth of the mice.

Seto et al.¹⁶ examined the effects of a 60-Hz, 80-kV/m electric field on the fertility, fecundity, nurturing, survival, and sex ratio of rat offspring. There were no significant differences in any of those measures between exposed and sham-exposed animals through three generations of study. A third filial generation of females were bred and either exposed or sham-exposed until days 16-20 of gestation. They were then killed, and their offspring examined for teratologic changes. No malformations were found in either group, and sex ratios were the same in both groups.

In contrast with the results of Seto et al.,¹⁶ results of another study by the same group¹⁷ suggested that prenatal development in the rat was inhibited by exposure to a 60-Hz, 80-kV/m electric field. Earflap separation and eye-opening were delayed in exposed offspring. Time to vaginal opening was shortened by electric field exposure, and prenatally exposed males exhibited significantly fewer intromissions and ejaculations than did sham-exposed animals. The latter findings suggest that prenatal exposure to electric fields might have affected sexual differentiation.

Portet and Cabanes¹⁸ exposed rabbits to a 50-Hz electric field (50-kV/m) for 16 h/d in the last 2 wk of gestation and for 6 wk after birth. No differences between controls and exposed were found in serum concentrations of glucose, triglycerides, or cholesterol. Plasma concentrations of thyroid, pituitary, and adrenal hormones were unaffected by exposure to the electric field. Adrenal cortisol was significantly lower in the exposed group, but, corticosterone was unaffected. Growth and development were not affected.

Frolen et al.¹⁹ studied the effects of a pulsed magnetic field on prenatal CBA mice; there were 154 control litters with 1,113 live fetuses and 211 exposed litters with 1,530 live fetuses. They characterized their fields as saw-toothed with a mean peak strength of 15 μ T. The pulses had a frequency of 20 kHz with 45 μ sec rise and 5- μ sec fall times. Fetal mortality was increased in the exposed group, but the incidence of abnormalities did not differ between the two groups. The incidence of early resorptions was increased in the exposed group.

Stuchly et al.²⁰ exposed female rats to a saw-tooth magnetic field similar to that produced by video display terminals for 2 wk before breeding and throughout pregnancy (7 h/d). The intensities were 5.7, 23, and 66 μ T peak to peak. The duration of each

cycle was 56 μ sec, and the fall time was 12 μ sec. Exposure to the magnetic fields had no effect on incidence of malformations or resorptions.

Two large studies performed at the Battelle Pacific Northwest Laboratories have examined the effect of prenatal exposure to electric or magnetic fields on in utero development of the rat. The first study²¹ used 60-Hz electric fields of 0, 10, 65, and 130 kV/m. Animals were exposed for 19 h/d throughout gestation, and exposed pups were allowed to be born in the field. The number of pups and pup mortality were not affected by exposure. At weaning, two F1 females per litter were randomly selected and maintained under their own exposure conditions until 11 wk of age. They were then mated to unexposed males and continued in the field until day 20 of gestation. They were euthanatized, and the pups were examined for developmental abnormalities. There was no evidence of teratologic effects or other developmental abnormalities.

In the second Battelle study,²² female rats were mated and sperm-positive animals were randomly distributed among three groups. Exposures were to 0.09- μ T (sham-exposed), 0.61- μ T (low-exposure), or 1,000- μ T (high-exposure) 60-Hz horizontal magnetic fields for 20 h/d from mating until day 20 of gestation. Replicate experiments were performed to provide approximately 180 litters per exposure group. A total of 7,903 fetuses were evaluated. There was no difference in incidence of malformations between exposed and control groups.

Sikov and coworkers²³ exposed Hanford miniature swine to a 60-Hz, 30 kV/m electric field for 20 h/d, 7 d/wk and compared their reproductive performance with that of sham-exposed animals. There was no effect of E-field exposure on the incidence of abnormalities in the first litters produced by the F₀ females, although there was a suggestion that the exposed offspring fared slightly better than the controls. However, subsequent breeding of F₀ females to produce an F_{1b} generation or of F_{1a} to produce an F₂ generation resulted in a significantly increased incidence of malformations in the exposed animals. A subsequent breeding of F₁ females to produce an F_{2b} generation resulted in no differences in incidences of malformations between exposed and sham-exposed animals. The reasons for these inconsistencies were discussed, but a definite explanation was not given.

Wertheimer and Leeper²⁴ reported that women exposed to increased 60-Hz electromagnetic fields, either from sleeping on waterbeds or under electric blankets or from exposure to ceiling-cable heat, had a greater incidence of spontaneous abortions than women who did not use waterbeds or electric blankets or who had other forms of heating in their homes. In the case of the ceiling heat, no differences were found until the data were stratified on the basis of seasonal exposures; that is, the rate of abortions went up when the amount of heating increased (expressed as percent change from previous month). However, the correlation was very weak when the actual numbers of heating degree days were used for comparison.

The use of visual display terminals (VDTs), which produced EMFs of 3-30 kHz, has been suggested to affect pregnancy outcome adversely. Interest in the possible relationship between VDT use and pregnancy outcome was stimulated in part by a report of a high percentage of spontaneous abortions among women working in the Dallas computer center of Sears, Roebuck and Company (NIOSH Report EPI-80-113-2, Atlanta, GA, 1981). Abortions reportedly occurred after conceptions that took place between May 1979 and June 1980. That observation was followed by a similar report of abortions in workers in Southern Bell's central computer facility in Atlanta (HETA 83-329-1498, Cincinnati, OH, 1984). The two clusters of high rates of spontaneous abortions were investigated by the National Institute for Occupational Safety and Health (NIOSH), and a number of factors were evaluated for their possible contribution to the increased rate of spontaneous abortion. The investigations at each site concluded that there was no correlation between VDT use and spontaneous abortion. No other factor could be positively identified as contributing to the problem, the NIOSH investigators concluded that chance occurrence was the most reasonable explanation in both situations. Goldhaber et al.²⁵ used data from the Kaiser Permanente system and found that women in some job classifications who used VDTs more than 20 h/wk had a higher incidence of abortions.

Several other studies have failed to find evidence of a relationship between VDT use and fetal loss. Kurppa et al.²⁶ performed a case-control study with data in the Finnish National Registry of Congenital Malformations. They found no association between VDT use and birth defects. McDonald et al.²⁷ examined data on a large number of pregnancies in Montreal hospitals for the period 1982-1984 with interview techniques; they found no association of birth defects or spontaneous abortions with VDT use. Ericson and Kallen²⁸ used the Swedish Registry of Congenital Malformations and identified three cohorts of women who had high, medium, and low probability of using video display equipment during 1980-1981. Their analysis did not identify significant differences in incidences of malformations or perinatal deaths among the three cohorts, although there was a weak trend for more spontaneous abortions in the group with highest VDT exposure. However, comparison of pregnancy outcomes during 1980-1981 with data from 1976-1977 did not show any consistent pattern, despite the large increase in computerization that had occurred in the workplace. The same investigators²⁹ performed a case-control study with data from their cohort study. They found a weak association between VDT use and birth defects, but found that the association was not significant when stress and smoking were taken into account. Bryant and Love,³⁰ in a case-control study involving 334 cases of spontaneous abortion, found no evidence that VDT use influenced the rate of abortion. Studies on a large population of telephone workers (Schnorr et al.³¹) provided strong evidence that exposure to VDTs during pregnancy had no effect on the pregnancy outcome.

Some studies performed to examine the developmental effects of RF and microwave radiation have been reviewed by Lary and Conover³² and O'Connor.³³ Most of the early experimental studies were performed at 2,450 MHz, the operating

frequency of many microwave ovens, and at levels that induced some heating. Lary et al.³⁴⁻³⁷ have explored the relationship between RF exposures and developmental changes with different frequencies and magnitudes of exposure. They established that abnormalities in development are related to maternal hyperthermia and not to some direct effect of RF radiation on the embryo or fetus. Rugh and McManaway³⁸ and Schmidt et al.³⁹ also provided evidence that developmental abnormalities did not result from the direct action of RF radiation. Evidence from the studies of Lary and colleagues and others generally indicates that maternal colonic temperatures need to reach 41°C or higher before mortality and malformations are increased. However, some studies⁴⁰⁻⁴² have found increased prenatal mortality and decreased fetal body weight at maternal colonic temperatures as low as 39.5°C, and others^{43, 44} found no effects at 40.3-40.6°C. The data of Lary et al.³⁶ indicate that the duration of temperature increase in the dam influences the effects. Moreover, the sensitivity of the embryo to various noxious agents varies widely with the stage of gestation. Such factors might help to explain the different observations. Developmental abnormalities found after RF irradiation were usually those associated with the head and included micrognathia, agnathia, microtia, anotia, exencephaly, encephalocele, and facial aplasia. The results indicate that the malformations resulted from increased heat load and that prolongation of increased temperatures exacerbated the effects. The results are in general agreement with those of other studies of the effects of hyperthermia on mammalian development.

In most of the studies just cited, the frequencies used were substantially different from those produced by the GWEN system. VDTs emit fields with frequencies up to about 30 kHz, but that still is far below the GWEN frequencies. Juutilainen et al.¹⁰ used fields up to 100 kHz, thus coming closest to the GWEN frequency; moreover, the actual fields they used were at least in the same range as would be expected from GWEN. Their data indicated that malformations in chick embryos could be produced by exposure to a magnetic field with a threshold of approximately $1 \text{ A/m} = 1.2 \mu\text{T} = 12 \text{ mG}$, which is about 20-times higher than the field at the GWEN perimeter fence. It is not clear that the chick-embryo system is indicative of what happens in mammals, but there are no studies of the effects of GWEN-frequency fields on mammalian development. The studies of Seto et al.,¹⁶ Portet and Cabanes,¹⁸ and Rommereim et al.²¹ with ELF electric fields have provided convincing evidence that exposure to these fields during gestation does not produce teratological changes. It is well documented that higher-frequency (radiofrequency, RF) fields are capable of inducing profound developmental changes. However, the evidence is very convincing that such effects are related to tissue heating by EMF fields.

Results of some of the studies with chick embryos suggest that exposure to low-intensity magnetic fields can cause developmental abnormalities, but there is not much evidence that these results are relevant to GWEN fields or to mammals. Examination of the literature does not reveal any studies performed with developing mammalian systems under conditions similar to those to be encountered with GWEN. However,

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

data obtained from studies with RF fields suggest that reproducible developmental effects are produced only when maternal body temperature is increased. That will not happen in people around GWEN sites.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

REFERENCES

1. Brent, R. 1980. Radiation teratogenesis. *Teratology* 21:281-298.
2. Zimmerman, S., A. M. Zimmerman, W. D. Winters, and I. L. Cameron. 1990. Influence of 60-Hz magnetic fields on sea urchin development. *Bioelectromagnetics* 11:37-45.
3. Cameron, I. L., K. E. Hunter, and W. D. Winters. 1985. Retardation of embryogenesis by extremely low frequency 60-Hz electromagnetic fields. *Physiol. Chem. Phys. Med. NMR* 17:135-138.
4. Delgado, J. M. R., J. Leal, J. L. Monteagudo, and M. G. Gracia. 1982. Embryological changes induced by weak, extremely low frequency electromagnetic fields. *J. Anat.* 134:533-551.
5. Ubeda, A., J. Leal, M. A. Trillo, M. A. Jimenez, and J. M. R. Delgado. 1983. Pulse shape of magnetic fields influences chick embryogenesis. *J. Anat.* 137:513-536.
6. Leal, J., K. Shamsaifar, M. A. Trillo, A. Ubeda, V. Abaira, and L. Chacon. 1989. Embryonic development and weak changes of the geomagnetic field. *J. Bioelectricity* 7:141-153.
7. Chacon, L., M. A. Trillo, A. Ubeda, and J. Leal. 1990. A 30-Hz pulsed magnetic field can stop early embryonic development. *J. Bioelectricity* 9:61-66.
8. Maffeo, S., M. W. Miller, and E. L. Carstensen. 1984. Lack of effect of weak low frequency electromagnetic fields on chick embryogenesis. *J. Anat.* 139:613-618.
9. Maffeo, S., A. A. Brayman, M. W. Miller, E. L. Carstensen, V. Ciaravino, and C. Cox. 1988. Weak low frequency electromagnetic fields and chick embryogenesis: failure to reproduce positive findings. *J. Anat.* 157:101-104.
10. Juutilainen, J., M. Harri, K. Saali, and T. Lahtinen. 1986. Effects of 100-Hz magnetic fields with various waveforms on the development of chick embryos. *Radiat. Environ. Biophys.* 25:65-74.
11. Juutilainen, J., and K. Saali. 1986.. Development of chick embryos in 1 Hz to 100 kHz magnetic fields. *Radiat. Environ. Biophys.* 25:135-140.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

12. Juutilainen, J., E. Laara, and K. Saali. 1987. Relationship between field strength and abnormal development in chick embryos exposed to 50 Hz magnetic fields. *Int. J. Radiat. Biol.* 52:787-793.

13. Berman, E., L. Chacon, D. House, B. A. Koch, W. E. Koch, J. Leal, S. Lovtrup, E. Mantiply, A. H. Martin, G. I. Martucci, K. H. Mild, J. C. Monahan, M. Sandstrom, K. Shamsaifar, R. Tell, M. A. Trillo, A. Ubeda, and P. Wagner. 1990. Development of chicken embryos in a pulsed magnetic field. *Bioelectromagnetics* 11:169-187.

14. Martin, A. H. 1992. Development of chicken embryos following exposure to 60-Hz magnetic fields with differing waveforms. *Bioelectromagnetics* 13:223-230.

15. Marino, A. A., R. O. Becker, and B. Ullrich. 1976. The effect of continuous exposure to low frequency electric fields on three generations of mice: a pilot study. *Experientia* 32:565-566.

16. Seto, Y. J., D. Majeau-Chargois, J. R. Lyman grover, W. P. Dunlap, E. F. Walker, and S. T. Hsieh. 1984. Investigation of fertility and in utero effects in rats chronically exposed to a high-intensity 60-Hz electric field. *IEEE Trans. Biomed. Eng.* BME-31:693-701.

17. Burack, G. D., Y. J. Seto, S. T. Hsieh, and J. L. Dunlap. 1984. The effects of prenatal exposure to a 60-Hz high-intensity electric field on postnatal development and sexual differentiation. *J. Bioelectricity* 3:451-467.

18. Portet, R., and J. Cabanes. 1988. Development of young rats and rabbits exposed to a strong electric field. *Bioelectromagnetics* 9:95-104.

19. Frolen, H., B. M. Svendenstäl, P. Bierke, and H. Fellner-Feldegg. 1987. Repetition of a study of the effect of pulsed magnetic fields on the development of fetuses in mice. Swedish University of Agricultural Sciences, Uppsala. Project SSI 346,86. Final Report, June 1987.

20. Stuchly, M. A., J. Ruddick, D. Villeneuve, K. Robinson, B. Reed, D. W. Lecuyer, K. Tan, and J. Wong. 1988. Teratological assessment of exposure to time-varying magnetic fields. *Teratology* 38:461-466.

21. Rommereim, D. N., R. L. Rommereim, M. R. Sikov, R. L. Buschbom, and L. E. Anderson. 1990. Reproduction, growth, and development of rats during chronic exposure to multiple field strengths of 60-Hz electric fields. *Fund. Appl. Toxicol.* 14:608-621.

22. Rommereim, D. N., R. L. Rommereim, D. L. Miller, R. L. Buschbom, and L. E. Anderson. 1993. Developmental toxicology evaluation of 60-Hz horizontal magnetic fields in rats. *Current Topics in Occupational Health Fund* (in press).
23. Sikov, M. R., J. L. Beamer, D. N. Rommereim, R. L. Buschbom, W. T. Kaune, and R. D. Phillips. 1987. Evaluations of reproduction and development in Hanford miniature swine exposed to 60-Hz electric fields. Pp. 379-393 in *Interaction of Biological Systems with Static and ELF Electric and Magnetic Fields*. eds. L.E. Anderson, B. J. Kelman, and R. J. Weigel. Pacific Northwest Laboratory, Richland, WA.
24. Wertheimer, N., and E. Leeper. 1989. Fetal loss associated with two seasonal sources of electromagnetic field exposure. *Am. J. Epidemiol.* 129:220-224.
25. Goldhaber, M. K., M. R. Polen, and R. A. Hiatt. 1988. The risk of miscarriage and birth defects among women who use visual display terminals during pregnancy. *Am. J. Indust. Med.* 13:695-706.
26. Kurppa, K., P. C. Holmberg, K. Rantala, T. Nurminen, and L. Saxen. 1985. Birth defects and exposure to video display terminals during pregnancy. *Scand. J. Work Environ. Health* 11:353-356.
27. McDonald, A. D., N. M. Cherry, C. Delorme, and J. C. McDonald. 1986. Visual display units and pregnancy: evidence from the Montreal survey. *J. Occup. Med.* 28:1226-1231.
28. Ericson, A., and B. Kallen. 1986a. An epidemiological study of work with video screens and pregnancy outcome. I. A registry study. *Am. J. Indust. Med.* 9:447-457.
29. Ericson, A., and B. Kallen. 1986b. An epidemiological study of work with video screens and pregnancy outcome: II. A case-control study. *Am. J. Indust. Med.* 9:459-475.
30. Bryant, H. E., and E. J. Love. 1989. Video display terminal use and spontaneous abortion risk. *Int. J. Epidemiol.* 18:132-138.
31. Schnorr, T. M., B. A. Grajewski, R. W. Hornung, M. J. Thun, G. M. Egeland, W. E. Murray, D. L. Conover, and W. E. Halperin. 1991. Video display terminals and the risk of spontaneous abortion. *N. Engl. J. Med.* 324:727-733.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

32. Lary, J. M., and D. L. Conover. 1987. Teratogenic effects of radiofrequency radiation. *IEEE Eng. Med. Biol. Mag.* Vol. 6 (March):42-46.

33. O'Connor, M. E. 1990. Teratogenesis: nonionizing electromagnetic fields. Pp. 358-372 in *Biological Effects and Medical Applications of Electromagnetic Energy*, O. P. Gandhi, ed. Englewood Cliffs, NJ: Prentice Hall.

34. Lary, J. M., D. L. Conover, E. D. Foley, and P. L. Hanser. 1982. Teratogenic effects of 27.12 MHz radiofrequency radiation in rats. *Teratology* 26:299-309.

35. Lary, J. M., D. L. Conover, P. H. Johnson, and J. R. Burg. 1983. Teratogenicity of 27.12-MHz radiation in rats is related to duration hyperthermic exposure. *Bioelectromagnetics* 4:249-255.

36. Lary, J. M., D. L. Conover, and P. H. Johnson. 1983. Absence of embryotoxic effects from low-level (nonthermal) exposure of rats to 100 MHz radiofrequency radiation. *Scand. J. Work Environ. Health* 9:120-127.

37. Lary, J. M., D. L. Conover, P. H. Johnson, and R. W. Hornung. 1986. Dose-response relationship between body temperature and birth defects in radiofrequency-irradiated rats. *Bioelectromagnetics* 7:141-149.

38. Rugh, R., and M. McManaway. 1976. Anesthesia as an effective agent against the production of congenital anomalies in mouse fetuses exposed to electromagnetic radiation. *J. Exp. Zool.* 197:363-368.

39. Schmidt, R. E., J. H. Merritt, and K. H. Hardy. 1984. In utero exposure to low-level microwaves does not affect rat foetal development. *Int. J. Radiat. Biol.* 46:383-386.

40. Michaelson, S. M., R. Guillet, and F. W. Hegeness. 1978. Influence of microwave exposure on functional maturation of the rat. Pp. 300-316 in *Developmental Toxicology of Energy-related Pollutants*, D. D. Mahlum, M. R. Sikov, P. L. Hackett, F. D. Andrew, eds. DoE Symposium Series 47, Washington, D.C.

41. Nawrot, P. S., D. I. McRee, and R. W. Staples. 1981. Effects of 2.45 GHz CW microwave radiation on embryofetal development in mice. *Teratology* 24:303-314.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

EFFECTS OF ELECTROMAGNETIC FIELDS ON DEVELOPMENT	65
42. Berman, E., H. B. Carter, and D. House. 1982. Observations of Syrian hamster fetuses after exposure to 2450-MHz microwaves. <i>J. Microwave Power</i> 17:107-112.	
43. Chernovetz, M., D. Justensen, N. King, et al. 1975. Teratology, survival, and reversal learning after fetal irradiation of mice by 2450-MHz microwave energy. <i>J. Microwave Power</i> 10:391-409.	
44. Berman, E., H. B. Carter, and D. House. 1981. Observations of rat fetuses after irradiation with 2450-MHz (CW) microwaves. <i>J. Microwave Power</i> 16:9-13.	

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

6

Effects of Electromagnetic Fields on Organs and Tissues

INTRODUCTION

A large body of literature exists on the response of tissues to electromagnetic fields, primarily in the extremely-low-frequency (ELF) and microwave-frequency ranges. In general, the reported effects of radiofrequency (RF) radiation on tissue and organ systems have been attributed to thermal interactions, although the existence of nonthermal effects at low field intensities is still a subject of active investigation. This chapter summarizes reported RF effects on major physiological systems and provides estimates of the threshold specific absorption rates (SARs) required to produce such effects. Organ and tissue responses to ELF fields and attempts to characterize field thresholds are also summarized. The relevance of these findings to the possible association of health effects with exposure to RF fields from GWEN antennas is assessed.

NERVOUS SYSTEM

The effects of radiation on nervous tissues have been a subject of active investigation since changes in animal behavior and nerve electrical properties were first reported in the Soviet Union during the 1950s and 1960s.¹ RF radiation is reported to affect isolated nerve preparations, the central nervous system, brain chemistry and histology, and the blood-brain barrier.

In studies with in vitro nerve preparations, changes have been observed in the firing rates of Aplysia neurons and in the refractory period of isolated frog sciatic nerves exposed to 2.45-GHz microwaves at SAR values exceeding 5 W/kg.^{2,3,4} Those effects were very likely associated with heating of the nerve preparations, in that much higher SAR values have not been found to produce changes in the electrical properties of isolated nerves when the temperature was controlled.^{5,6} Studies on isolated heart preparations have provided evidence of bradycardia as a result of exposure to RF radiation at nonthermal power densities,⁷ although some of the reported effects might have been artifacts caused by currents induced in the recording electrodes or by nonphysiological conditions in the bathing medium.^{8,9,10} Several groups of investigators have reported that nonthermal levels of RF fields can alter Ca^{2+} binding to the surfaces of nerve cells in isolated brain hemispheres and neuroblastoma cells cultured in vitro (reviewed by the World Health Organization¹¹ and in Chapters 3 and 7 of this report). That phenomenon, however, is observed only when the RF field is amplitude-modulated at extremely low frequencies, the maximum effect occurs at a modulation frequency of 16 Hz. A similar effect has recently been reported in isolated frog hearts.¹² The importance of changes in Ca^{2+} binding on the functional properties of nerve cells has

not been established, and there is no clear evidence that the reported effect of low-intensity, amplitude-modulated RF fields poses a substantial health risk.

Results of *in vivo* studies of both pulsed and continuous-wave (CW) RF fields on brain electrical activity have indicated that transient effects can occur at SAR values exceeding 1 W/kg.^{13, 14} Evidence has been presented that cholinergic activity of brain tissue is influenced by RF fields at SAR values as low as 0.45 W/kg.¹⁵ Exposure to nonthermal RF radiation has been reported to influence the electroencephalograms (EEGs) of cats when the field was amplitude-modulated at frequencies less than 25 Hz, which is the range of naturally occurring EEG frequencies.¹⁶ The rate of Ca^{2+} exchange from cat brain tissue *in vivo* was observed to change in response to similar irradiation conditions.¹⁷ Comparable effects on Ca^{2+} binding were not observed in rat cerebral tissue exposed to RF radiation,¹⁸ although the fields used were pulsed at EEG frequencies, rather than amplitude-modulated. As noted above, the physiological significance of small shifts in Ca^{2+} binding at nerve cell surfaces is unclear.

A wide variety of changes in brain chemistry and structure have been reported after exposure of animals to high-intensity RF fields.¹⁹ The changes include decreased concentrations of epinephrine, norepinephrine, dopamine, and 5-hydroxytryptamine; changes in axonal structure; a decreased number of Purkinje cells; and structural alterations in the hypothalamic region. Those effects have generally been associated with RF intensities that produced substantial local heating in the brain.

Extensive studies have been carried out to detect possible effects of RF radiation on the integrity of the blood-brain barrier.^{20, 21} Although several reports have suggested that nonthermal RF radiation can influence the permeability of the blood-brain barrier, most of the experimental findings indicate that such effects result from local heating in the head in response to SAR values in excess of 2 W/kg. Changes in cerebral blood flow rate, rather than direct changes in permeability to tracer molecules, might also be incorrectly interpreted as changes in the properties of the blood-brain barrier.

Effects of pulsed and sinusoidal ELF fields on the electrical activity of the nervous system have also been studied extensively.^{22, 23} In general, only high-intensity sinusoidal electric fields or rapidly pulsed magnetic fields induce sufficient current density in tissue (around 0.1-1.0 A/m² or higher) to alter neuronal excitability and synaptic transmission or to produce neuromuscular stimulation. Somewhat lower thresholds have been observed for the induction of visual phosphenes (discussed in the next section) and for influencing the electrical activity of *Aplysia* pacemaker neurons when the frequency of the applied field matched the endogenous neuronal firing rate.²⁴ Those effects, however, have been observed only with ELF frequencies and would not be expected to occur at the higher frequencies associated with GWEN transmitters. Recent studies with human volunteers exposed to 60-Hz electric and magnetic fields with intensities comparable with those of high-voltage power lines have shown no

consistent effects on the EEG.²⁵ Minor changes were observed in reaction time and heart rate, but the variations were within normal ranges.

VISUAL SYSTEM

Cataract development as a result of exposure of the eye to high-intensity RF radiation has been studied for more than 30 years. Extensive experiments have been carried out with rabbits to determine the dependence of cataractogenesis on the frequency and intensity of RF fields and on exposure time.²⁶⁻²⁸ In general, the lowest thresholds for cataract induction have been observed with near-field exposure at 1-10 GHz, and a power density greater than 100 mW/cm² applied for at least an hour is required. Most of the evidence indicates that the mechanism of injury leading to lens opacity is thermal, and pulsed and CW microwave fields appear to have similar thresholds for producing cataracts.¹⁹ Multiple subthreshold exposures do not lead to cataracts if the time between exposures is long enough to permit the tissue to return to its normal temperature.¹ At frequencies where the wavelength of the RF field is not well matched to the dimensions of the eye, cataracts are not produced even at extremely high power densities approaching the lethal levels. Although it is difficult to extrapolate results from laboratory animals to humans, the threshold power density required to produce cataracts is expected to be similar in rabbits and humans because of the structural similarities and comparable dimensions of the eyes in these species.

Results of recent studies with monkeys have indicated that vascular leakage can increase at relatively low power densities of pulsed 2.45-GHz radiation when the eye is pretreated with timolol maleate, which decreases intraocular pressure by reducing the production of aqueous humor. A power density as low as 1 mW/cm², corresponding to an intraocular SAR of 0.26 W/kg, has been observed to produce the effect.²⁹ Those findings might have implications for RF ocular damage in humans being treated with timolol maleate for glaucoma. However, the threshold power density at 2.45 GHz is still well in excess of the intensity of the RF fields produced by GWEN antennae in areas accessible to the general public.

A visual phenomenon associated with exposure to ELF fields that has been studied for nearly a century is the induction of a flickering illumination known as phosphenes. Time-varying magnetic fields with either pulsed or sinusoidal waveforms and frequencies below 100 Hz have been shown to produce phosphenes when the time rate of change of the field exceeds 1.3 T/sec.³⁰ The frequency most likely to produce magnetophosphenes with sinusoidal fields is 20 Hz; the threshold flux density for eliciting the visual effect is 8 mT.³¹ A similar frequency dependence has been observed for electrophosphenes produced by placing electrodes in contact with the forehead near the eyes.³² The locus of the effect is in the retina, and available evidence suggests that induced currents in the retina elicit visual responses similar to those resulting from photic stimulation.³⁰ Changes in visually evoked potential (VEP) have also been

reported in response to ELF magnetic fields with flux densities that are 5-10 times greater than those required to produce phosphenes.³ Because phosphenes and VEP alterations are observed only with fields below 100 Hz, such phenomena are not expected to occur in response to the low-frequency fields associated with GWEN antenna.

ENDOCRINE SYSTEM

Many studies with rodents and monkeys have demonstrated that exposure to thermogenic levels of RF radiation produces endocrine alterations, the most consistent change being an increase in plasma corticosterone.^{1, 34} SAR values in excess of 3 W/kg produce an increase in plasma corticosterone in rats that depends on secretion of adrenocorticotrophic hormones by the pituitary.³⁵ Decreased thyroid hormone levels have also been observed in response to thermogenic levels of RF radiation, and this response has been associated with an inhibition of thyrotropin secretion by the pituitary.^{36, 37} In general, the alteration of hormone concentrations is reversible after termination of the RF exposure. Those findings indicate that RF heating alters the complex interactions of the hypothalamic, pituitary, adrenal, and thyroid systems that are important in the maintenance of homeostasis.¹⁹ It is noteworthy that a 2-yr exposure of rats to nonthermogenic pulsed 2.45-GHz microwaves did not produce detectable endocrine alterations.³⁷

Studies on the possible endocrine effects of ELF electric and magnetic fields have yielded inconsistent results.²² Increases, decreases, and no change in plasma steroid hormones have been reported. Results of studies with dogs and rats suggest that the threshold 60-Hz electric field required to produce changes in blood concentrations of corticosterone or testosterone is in excess of 10 kV/m.³⁸⁻⁴⁰ Results of experiments with monkeys exposed to 60-Hz electric and magnetic fields at intensities typical of those in the vicinity of high-voltage transmission lines indicated that a decrease in neurotransmitter concentrations occurs during chronic exposure.⁴¹ However, there were no other observations of behavioral or physiological changes in the exposed animals.

The most widely studied effect of ELF fields on the endocrine system is an apparent depression in the nocturnal rise of pineal melatonin.⁴² Changes in pineal melatonin have reportedly occurred after 2-3 wk of exposure to electric fields with intensities exceeding 1.7 kV/m in air. The effect is reversible, with a return of nocturnal pineal melatonin to control values within 3 d of termination of exposure. A similar effect on pineal melatonin has been observed after exposure of rodents to a 0.05 mT static magnetic field that was continuously switched on and off in 5-min cycles for 1 h beginning 3.5 h after the onset of darkness.⁴³ Interest in this phenomenon has centered around the effects of melatonin on cell proliferation and its possible carcinostatic effects.⁴⁴⁻⁴⁶ A major problem in the interpretation of the results of studies

is lack of quantitative information on the threshold fields required to alter melatonin concentration. It is also unclear whether ELF fields directly alter pinealocyte functions or whether the reported alteration in pineal melatonin production is secondary to effects of the fields on the nervous system. Further studies are needed to assess the possible influence of field-induced changes in pineal melatonin on physiological regulation and the risk of endocrine-dependent cancers. It is not now possible to extrapolate the available information obtained with 60-Hz fields or intermittent DC magnetic fields to the possible effects of fields with higher frequencies.

IMMUNE SYSTEM

Effects of RF-field exposure on cellular components of the immune system have been reported with both in vitro and in vivo test systems.¹ Lymphoblast transformation and changes in responsiveness to mitogens have been reported, although the effects observed in different laboratories have been quite variable. It appears from available information that the threshold SAR for altering lymphocyte responses to mitogens exceeds 4 W/kg with both pulsed and CW microwaves.⁴⁷⁻⁴⁹ Thermogenic levels of exposure have been found to decrease natural killer cell activity and to activate macrophages.^{50, 52} The changes observed in components of the immune system at RF power densities that produce tissue heating are consistent with the expected effects of increased release of steroid hormones into the circulation.^{1, 52, 53} In one study involving a 2-yr exposure of rats to a nonthermal level of pulsed 2.45-GHz microwaves (SAR, 0.4 W/kg), no significant irreversible changes were found in the concentrations of lymphocytes or their responses to mitogen stimulation.

Numerous studies have been carried out to determine the effects of ELF electric and magnetic fields on components of the immune system. In general, sinusoidal ELF fields have been found to have no significant effects on immune competence after in vivo exposure of laboratory animals.^{22, 25} However, reduced mitogen responses and decreased target-cell toxicity have been reported for lymphocytes exposed in vitro to pulsed magnetic fields or 60-Hz amplitude-modulated RF fields.⁵⁵⁻⁵⁷ These effects might have resulted from the relatively high current densities induced in the cell suspensions. In a study involving 60-Hz electric and magnetic fields with a sinusoidal waveform and intensities comparable with those of the fields near high-voltage power lines, no effects were observed on the immunologic functions of peripheral human and canine lymphocytes obtained from donors that either were normal or had been challenged with specific antigens.⁵⁸

HEMATOLOGIC AND CARDIOVASCULAR SYSTEMS

Several studies have been performed to assess the effects of both thermogenic and nonthermogenic levels of RF radiation on blood chemistry and blood-cell counts.

Most were conducted with 2.45-GHz microwaves, and the results indicate that power densities producing an average whole-body SAR of less than 2.5 W/kg do not produce significant alterations in hematologic indexes.⁵⁹ In one chronic-exposure study with rabbits subjected to 2.45-GHz microwaves 23 h/d for 180 d, a small decrease was observed in eosinophil count, serum albumin concentration, and calcium concentration.⁶⁰ None of the other 38 blood characteristics measured in the study were found to change in response to chronic RF exposure.

At high levels of RF exposures of mice leading to rectal temperature increases of 2–4°C, decreased lymphocyte counts and increased neutrophil counts were observed.⁶¹ It was suggested that the release of adrenal steroids into the blood as a result of heat stress could have produced the changes in blood-cell counts.^{52, 53} Thermogenic levels of pulsed or CW microwaves might also affect the cellular composition and proliferative capacity of bone-marrow cells.^{62, 63} Both bradycardia and tachycardia have been observed in laboratory animals exposed to thermogenic levels of RF radiation with SAR values in excess of 2.5 W/kg.^{64, 65} In general, the effects on cardiac dynamics were transient and consistent with effects expected from body heating.

As reviewed by several authors,^{22, 54, 66} the threshold ELF fields for producing significant cardiovascular and hematologic alterations are high. For example, cardiovascular indexes were not affected by exposure to 60-Hz, 100-kV/m fields.⁶⁷ Acute human exposures to ELF electric fields up to 200 kV/m and magnetic fields up to 5 mT have also failed to show any consistent hematologic or cardiovascular effects.^{68, 69}

ANIMAL CARCINOGENESIS

A few studies have investigated the carcinogenic potential of microwave radiation in whole animals. Male Swiss albino mice were exposed to a radar transmitter with 9.27-GHz frequency modulated with 2- μ sec pulses at a pulse repetition frequency of 500/sec for 59 wk, 5 d/wk and 4.5 min/d.⁷⁰ The power used (1 kW/m²) caused a temperature rise of 3.3°C. There was no difference between exposed and control animals in a number of characteristics examined, including body weight, red-cell and white-cell counts, and body temperature. Testicular degeneration occurred in 23 of 57 (40%) of treated animals and in 3 of 37 (8.1%) of the controls. Monocytic or lymphatic organ tumors or myeloid leukemia was seen in 21 of 60 (35%) treated and 4 of 40 (10%) control animals. However, that increase was seen in the animals killed at 16 mo—I mo after cessation of treatment—but not at 19 mo. In addition, there has been considerable criticism of the experimental methods (e.g., definition of leukosis as an increase in circulating leukocytes, which could have been due to infection) and statistical analysis.^{71, 72} Thus, the study is of questionable use for supporting an increase in cancer risk. The incident power density was approximately 100 times greater than the highest relevant GWEN levels.

Female RFM mice were exposed to 0.8-GHz microwave radiation for 2 h/d, 5 d/wk for 35 wk at 430 W/m.⁷³ Red and white cell counts, hemoglobin, hematocrit, activity, body weight, and survival were measured, but no histopathologic examinations, were carried out. The only statistically significant finding was an increase in body weight of animals older than 86 wk in exposed mice over control animals.

A University of Washington study on male Sprague-Dawley rats was designed to simulate the maximum absorbed power (0.4 W/kg) of 0.45-GHz radiation.⁷⁴ The frequency was chosen as typical of a midrange radar system. Rats were exposed at 2.45 GHz, because it yielded a ratio of wavelength to maximum body dimension similar to that of children exposed at 0.45 GHz. Benign pheochromocytoma of the adrenal medulla was the only lesion with a statistically significant increase in incidence. However, that incidence was not higher than that seen in control rats of other colonies. The time for appearance of first tumor was also shorter in treated animals (457 d) than in control animals (540 d). When all malignant tumors observed at all sites are combined, there is a statistically significant increase in incidence in the exposed animals. That holds true for carcinomas, but not for sarcomas.

An effect on the process of carcinogenesis has been reported in several studies that used injected tumor cells or animals treated with low doses of a known carcinogen. The effects of 2.45-GHz microwave radiation at 50 and 150 W/m² in an anechoic chamber was determined.⁷⁵ Lung sarcoma cells were injected intravenously into Balb/C mice, and the lung-cancer colonies were counted after 1, 2, and 3 mo of treatment. After 3 mo, the numbers of lung nodules were 3.6 ± 2.2 , 7.7 ± 2.0 , 6.1 ± 8 , and 10.8 ± 2.1 in control animals, chronically stressed animals, animals exposed at 50 W/m², and animals exposed at 150 W/m², respectively. The time to appearance of spontaneous breast tumors in 50% of C3H/HeA mice decreased in animals treated with chronic stress, 50 W/m², and 150 W/m² (255, 261, and 219 d, respectively, compared with 322 d in controls). The time of appearance of skin tumors induced by benzo[a]pyrene was also shortened when irradiation for 1 or 3 mo preceded carcinogen application or when radiation and carcinogen were given at the same time.⁷⁵ Again, stress closely duplicated the effect of 50-W/m² radiation, and, although the results of 150 W/m² were significantly greater than those of stress or 50 W/m², thermal effects might be responsible for the results at 150 W/m². The power density of 50 W/m² is approximately 7 times the low-frequency power density at the GWEN site boundary.

Negative and beneficial results of exposure have also been reported. The effect of continuous or pulsed waves of 2.45 GHz (10 W/m²; SAR, 1.2 W/kg) was studied in black C57/6J mice with B16 melanoma.⁷⁶ No significant effects on tumor development or survival times were observed. Beneficial effects of induced hyperthermia have been observed after treatment with microwave radiation.^{77, 78} Lung sarcoma cells injected into Balb/C mice demonstrated temporary regression after exposure to 2.45-GHz radiation. After radiation exposure was stopped, tumor volumes increased and lung metastases exceeded those in untreated animals.⁷⁷ Sarcoma cells were implanted on

postpartum day 16 into CFW mice that had been irradiated in utero with 2.45-GHz microwaves (35 W/kg) during days 11-14 of gestation; the mice were then subjected to additional exposure to microwave radiation. Fetal exposure to radiation that increased the dam's colonic temperatures by an average of 2.2°C decreased tumor incidence (13%, with 46% in controls). Both tumor-bearing and tumor-free animals that were irradiated as fetuses lived longer, on the average, than controls. Enhanced immunocompetence related to increases in temperature was suggested as an explanation.

In summary, several studies have provided some evidence of possible carcinogenic potential, and others have shown no effect. Consistent reproducible studies demonstrating a dose-response relation in animals are lacking, and the interpretation of several studies is complicated by thermally induced stress. All studies were conducted with electromagnetic fields larger than the relevant GWEN fields.

Considerable interest has arisen concerning a possible role of electromagnetic fields as cocarcinogens or cancer promoters. Results of cellular studies that support such speculation are discussed in [Chapter 7](#). Studies by McLean et al.⁷⁹ have begun to explore directly the possible role of 60-Hz magnetic fields as cancer promoters. In their studies, cancers in SENCAR mice—known for their sensitivity to the promoting effects of 12-O-tetradecanoylphorbol-13-acetate (TPA)—were initiated with dimethylbenzanthracene and then promoted with TPA, a 2-mT magnetic field, or a combination of TPA and a magnetic field. Tumor development was then observed for 20 wk. There was no significant difference in tumor development between those promoted with TPA alone and those promoted with TPA and a magnetic field. However, recent results of another experiment by the same group of investigators suggest that a 60-Hz magnetic field acts as a co-promoter. Additional studies are required to substantiate those findings. Beniashvili et al.⁸⁰ treated rats with nitrosomethyl urea and then exposed them to either a 0.2 G static or 50-Hz magnetic field for either 0.5 or 3 h/d for up to two years. They found an increased incidence and number of mammary tumors in the groups exposed to the magnetic fields, with the ac field being more active than the static one. They also reported an increased tumor response in rats exposed to the 50-Hz field alone.

CONCLUSIONS

Very few studies have been performed on the responses of organs and tissues to electromagnetic fields in the low-frequency or ultra-high-frequency ranges used by GWEN transmitters, but it is possible to conclude from the analysis presented here that effects of RF radiation are unlikely to occur at the power densities and absorbed energies associated with GWEN fields. The possible existence of nonthermal effects—such as the reported effects of low-intensity, amplitude-modulated RF fields on Ca^{2+} binding in nerve tissue—do not alter that conclusion, in that the waveforms and

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

frequency spectra of these fields are different from those of the GWEN fields. Physiologic effects of ELF fields—with frequencies less than one five-hundredth those of GWEN fields—are generally associated with high field intensities and large induced-current densities in tissue. Some physiologic effects of ELF fields that might result from low induced current in tissue, such as alterations in pineal melatonin concentration, have not been shown to pose a direct risk to human health. In sum, studies on physiologic effects of ELF fields have yielded little evidence that exposure to the low-frequency fields from GWEN antennae in areas of most likely public access would represent a health risk.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

REFERENCES

1. Michaelson, S. M. 1986. Interaction of unmodulated radiofrequency fields with living matter: Experimental results. Pp. 339-423 in Handbook of Biological Effects of Electromagnetic Fields, C. Polk, and E. Postow, eds. Boca Raton, FL.
2. McRee, D. I., and H. Wachtel. 1980. The effects of microwave radiation on the vitality of isolated frog sciatic nerves. Radiat. Res. 82:536-546.
3. McRee, D. I., and H. Wachtel. 1982. Pulse microwave effects on nerve vitality. Radiat. Res. 91:212-218.
4. Wachtel, H., R. Seaman, and W. Joines. 1975. Effects of low-intensity microwaves on isolated neurons. Ann. NY Acad. Sci. 247:46-62.
5. Chou, C.-K., and A. W. Guy. 1973. Effect of 2450 MHz microwave fields on peripheral nerves. Pp. 318-310 in Digest of Technical Papers. IEEE International Microwave Symposium (Boulder, CO, June 1973).
6. Courtney, K. R., J. C. Lin, A. W. Guy, and C.-K. Chou. 1975. Microwave effect on rabbit superior cervical ganglion. IEEE Trans. Microwave Theory Tech. MTT-23: 809-813.
7. Liddle, C. G., and C. F. Blackman. 1984. Endocrine, physiological and biochemical effects. Pp. 5.79-5.93 in Biological Effects of Radiofrequency Radiation, J. A. Elder, and D. F. Cahill, eds. U.S.E.P.A. Report No. EPA-600/8-83-026F. Research Triangle Park, NC: Health Effects Research Laboratory, United States Environmental Protection Agency.
8. Chapman, R. M., and C. A. Cain. 1975. Absence of heart rate effects in isolated frog heart with pulse modulated microwave energy. J. Microwave Power 13:411-419.
9. Yee, K.-C., C.-K. Chou, and A. W. Guy. 1984. Effect of microwave radiation on the beating rate of isolated frog hearts. Bioelectromagnetics 5:263-270.
10. Yee, K.-C., C.-K. Chou, A. W. Guy. 1988. Influence of microwaves on the beating rate of isolated rat hearts. Bioelectromagnetics 9: 175-181.
11. World Health Organization Environmental Health Criteria. 1991. Electromagnetic Fields (300 Hz-300 GHz). Geneva, Switzerland: WHO.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

12. Schwartz, J.-L., D. E. House, and G. A. R. Mealing. 1990. Exposure of frog hearts to CW or amplitude-modulated VHF fields: Selective efflux of calcium ions at 16 Hz. *Bioelectromagnetics* 11:349-358.

13. Johnson, C. C., and A. W. Guy. 1972. Nonionizing electromagnetic wave effects in biological materials and systems. *Proc. IEEE* 60:692-718.

14. Servantie, A.M., and J. Etienne. 1975. Synchronization of cortical neurons by a pulsed microwave field as evidenced by spectral analysis of electrocorticograms from the white rat. *Ann. NY Acad. Sci.* 247:82-86.

15. Lai, H., M. A. Carino, A. Horita, and A. W. Guy. 1989. Low-level microwave irradiation and central cholinergic activity: A dose-response study. *Bioelectromagnetics* 10:203-208.

16. Bawin, S. M., R. J. Gavalas-Medici, W. R. Adey. 1973. Effects of modulated very high frequency fields on specific brain rhythms in cats. *Brain Res.* 58:365-384.

17. Adey, W. R., S. M. Bawin, and A. F. Lawrence. 1982. Effects of weak amplitude-modulated microwave fields on calcium efflux from awake cat cerebral cortex. *Bioelectromagnetics* 3:295-307.

18. Merritt, J. H., W. W. Shelton, and A. F. Chamness. 1982. Attempts to alter $^{45}\text{Ca}^{2+}$ binding to brain tissue with pulse-modulated microwave energy. *Bioelectromagnetics* 3:475-478.

19. Michaelson, S. M., and J. C. Lin. 1987. *Biological Effects and Health Implications of Radiofrequency Radiation*. New York, NY: Plenum Press.

20. Justesen, D. R. 1980. Microwave irradiation and the blood-brain barrier. *Proc. IEEE* 68:60-67.

21. Williams, W. M., S. T. Lu, M. Del Cerro, and S. M. Michaelson. 1984. Effect of 2450 MHz microwave energy on the blood-brain barrier to hydrophilic tracers. *Brain Res. Rev.* 7:191-212.

22. Anderson, L. E., and R. D. Phillips. 1985. Biological effects of electric fields: An overview. Pp. 345-378 in *Biological Effects and Dosimetry of Static and ELF Electromagnetic Fields*, M. Grandolfo, S. M. Michaelson, and A. Rindi, eds. New York, NY: Plenum Press.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

23. Tenforde, T. S., and T. F. Budinger. 1986. Biological effects and physical safety aspects of NMR imaging and in vivo spectroscopy. Pp. 493-548 in *NMR in Medicine: Instrumentation and Clinical Applications*, S. R. Thomas, and R. L. Dixon, eds. Medical Physics Monograph No. 14. New York, NY: American Association of Physicists in Medicine.

24. Wachtel, H. 1978. Firing-pattern changes and transmembrane currents produced by extremely low frequency fields in pacemaker neurons. Pp. 132-146 in *Biological Effects of Extremely Low Frequency Electromagnetic Fields*, R. D. Phillips, M. F. Gillis, W. T. Kaune, and D. D. Mahlum, eds. U.S. Department of Energy Technical Information Center Report No. CONF-781016. Springfield, VA: National Technical Information Service.

25. Graham, C., M. R. Cook, and H. D. Cohen. 1990. Immunological and Biochemical Effects of 60-Hz Electric and Magnetic Fields in Humans. Midwest Research Institute Final Report for Contract No. DE-FCO1-84-CE76246 (Order No. DE90006671). Oak Ridge, TN: U.S. Department of Energy, Office of Scientific and Technical Information.

26. Carpenter, R. L., and C. A. Van Ummerson. 1968. The action of microwave power on the eye. *J. Microwave Power* 3:3-19.

27. Guy, A. W., J. C. Lin, P. O. Kramar, and A. F. Emery. 1975. Effect of 2450 MHz radiation on the rabbit eye. *IEEE Trans. Microwave Theory Tech.* MTT-23:492-498.

28. Kramar, P., C. Harris, A. F. Emery, A. W. Guy. 1978. Acute microwave irradiation and cataract formation in rabbits and monkeys. *J. Microwave Power* 13:239-249.

29. Monohan, J. C., H. A. Kues, D. S. McLeod, S. A. D'Anna, and G. A. Luty. 1988. Lowering of microwave exposure threshold for induction of primate ocular effects by timolol maleate. Bioelectromagnetics Society Tenth Annual Meeting, Abstract B-07-1, p. 48 (Stamford, CT, June 1988).

30. Tenforde, T. S. 1990. Biological effects of extremely low frequency magnetic fields. Pp. 291-315 in *Extremely Low Frequency Electromagnetic Fields: The Question of Cancer*, B. W. Wilson, R. G. Stevens, and L. E. Anderson, eds. Columbus, OH: Battelle Press.

31. Lovsund, P., P. A. Oberg, S. E. G. Nilsson, and T. Reuter. 1980. Magnetophosphenes: A quantitative analysis of thresholds. *Med. Biol. Eng. Comput.* 18:326-334.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

32. Lovsund, P., P. A. Oberg, and S. E. G. Nilsson. 1980. Magneto- and electrophosphores: A comparative study. *Med. Biol. Eng. Comput.* 18: 758-764.

33. Silny, J. 1986. The influence threshold of a time-varying magnetic field in the human organism. Pp. 105-112 in *Biological Effects of Static and Extremely Low Frequency Magnetic Fields*, J. H. Bernhardt, ed. Munich, Germany: MMV Medizin Verlag.

34. Lotz, W. G., S. M. Michaelson. 1978. Temperature and corticosterone relationships in microwave-exposed rats. *J. Appl. Physiol. Resp. Envir. Exercise Physiol.* 47:438-445.

35. Lotz, W. G., and S. M. Michaelson. 1979. Effects of hypophysectomy and dexamethasone on rat adrenal response to microwaves. *J. Appl. Physiol. Resp. Envir. Exercise Physiol.* 47:1284-1288.

36. Lu, S.-T., W. G. Lotz, and S. M. Michaelson. 1980a. Advances in microwave-induced neuroendocrine effects: The concept of stress. *Proc. IEEE* 68:73-77.

37. Lu, S.-T., W. G. Lotz, and S. M. Michaelson. 1980b. Delineating acute neuroendocrine responses in microwave-exposed rats. *J. Appl. Physiol.: Resp. Environ. Exercise Physiol.* 48:927-932.

38. Gann, D. J. 1976. Final Report. Electric Power Research Institute Contract No. RP98-02. Baltimore, MD: Johns Hopkins University.

39. Free, M. J., W. T. Kaune, R. D. Phillips, and H. C. Cheng. 1981. Endocrinological effects of strong 60-Hz electric fields on rats. *Bioelectromagnetics* 2:105-121.

40. Lymanogrover, J. R., E. Kekn, and Y. J. Seto. 1983. 60-Hz electric field alters the steroidogenic response of rat adrenal tissue in vitro. *Life Sci.* 32:691-696.

41. Wolpaw, J. R., R. F. Seegal, R. I. Dowman, and S. Satya-Murti. 1987. Chronic Effects of 60-Hz Electric and Magnetic Fields on Primate Central System Function. Technical report prepared for the New York State Power Lines Project. Albany, NY: Wadsworth Laboratories (E297), Empire State Plaza.

42. Wilson, B. W., L. E. Anderson, D. I. Hilton, and R. D. Phillips. 1981. Chronic exposure to 60-Hz electric fields: Effects on pineal function in

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

the rat. *Bioelectromagnetics* 2: 371-380. [Erratum: *Bioelectromagnetics* 4:293 (1983)].

43. Lerchl, A., K. O. Nonaka, K. A. Stokkan, and R. J. Reiter. 1990. Marked rapid alterations in nocturnal pineal serotonin metabolism in mice and rats exposed to weak intermittent magnetic fields. *Biochem. Biophys. Res. Comm.* 169:102-108.

44. Tamarkin, L., C. J. Baird, and O. F. Almeida. 1985. Melatonin: A coordinating signal for mammalian reproduction. *Science* 227:714-720.

45. Blask, D., and S. Hill. 1986. Effects of melatonin on cancer: Studies on MCF-7 human breast cancer cells in culture. *J. Neural Transm. (Suppl.)* 21:433-449.

46. Stevens, R. G. 1987. Electric power use and breast cancer: A hypothesis. *Am. J. Epidemiol.* 125:556-561.

47. Roberts, N. J. Jr., S.-T. Lu, and S. M. Michaelson. 1983. Human leukocyte radiation. *Science* 220:318-320.

48. Roberts, N. J. Jr., S. M. Michaelson, and S.-T. Lu. 1984. Exposure of human mononuclear leukocytes to microwave energy pulse modulated at 16 or 60 Hz. *IEEE Trans. Microwave Theory Tech.* MTT-32: 803-807.

49. Smialowicz, R. J., M. M. Riddle, P. L. Brugnolotti, J. M. Sperrazza, and J. B. Kinn. 1979. Evaluation of lymphocyte function in mice exposed to 2450 MHz (CW) microwaves. Pp. 122-152 in *Electromagnetic Fields in Biological Systems*, S. S. Stuchly, ed. Edmonton, Canada: International Microwave Power Institute.

50. Yang, H. K., C. A. Cain, J. Lockwood, and W. A. F. Tompkins. 1983. Effects of microwave exposure on the hamster immune system. I. Natural killer cell activity. *Bioelectromagnetics* 4:123-139.

51. Rama Rao, G., C. A. Cain, J. Lockwood, and W. A. F. Tompkins. 1983. Effects of microwave exposure on the hamster immune system. II. Peritoneal macrophage function. *Bioelectromagnetics* 4:141-155.

52. Liburdy, R. P. 1979. Radiofrequency radiation alters the immune system: Modification of T-and B-lymphocyte levels and cell-mediated immunocompetence by hyperthermic radiation. *Radiat. Res.* 77:34-46.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

53. Liburdy, R. P. 1980. Radiofrequency radiation alters the immune system. II. Modulation of in vivo lymphocyte circulation. *Radiat. Res.* 83:63-73.

54. Smialowicz, R. J. 1985. Hematologic and immunologic effects of extremely low frequency electromagnetic fields. Pp. 203-225 in *Biological and Human Health Effects of Extremely Low Frequency Electromagnetic Fields*. Arlington, VA: American Institute of Biological Sciences.

55. Conti, P., G. E. Gigante, M. G. Cifone, E. Alese, G. Ianni, M. Reale, and P. U. Angeletti. 1983. Reduced mitogenic stimulation of human lymphocytes by extremely low frequency electromagnetic fields. *Fed. Europ. Biochem. Soc. (FEBS)* 162:156-160.

56. Conti, P., G. E. Gigante, M. G. Cifone, E. Alese, C. Fieschi, M. Bologna, and P. U. Angeletti. 1986. Mitogen dose-dependent effect of weak pulsed electromagnetic field on lymphocyte blastogenesis. *Fed. Europ. Biochem. Soc. (FEBS)* 199:130-134.

57. Lyle, D. B., R. D. Ayotte, A. R. Sheppard, and W. R. Adey. 1988. Suppression of T-lymphocyte cytotoxicity following exposure to 60-Hz sinusoidal electric fields. *Bioelectromagnetics* 9:303-313.

58. Winters, W. D. 1987. Biological Functions of Immunologically Reactive Human and Canine Cells Influenced by In Vitro Exposure to 60-Hz Electric and Magnetic Fields. Technical report prepared for the New York State Power Lines Project. Albany, NY: Wadsworth Laboratories (E-297), Empire State Plaza.

59. Smialowicz, R. J. 1984. Hematologic and immunologic effects. Pp. 5.13-5.28 in *Biological Effects of Radiofrequency Radiation*, J. A. Elder, and D. F. Cahill, eds. U.S. Environmental Protection Agency Report No. EPA-600/8-83-026F. Health Effects Research Laboratory, Research Triangle Park, NC: United States Environmental Protection Agency.

60. McRee, D. I., M. J. Galvin, and C. L. Mitchell. 1988. Microwave effects on the cardiovascular system: A model for studying the responsivity of the automatic nervous system to microwaves. Pp. 153-177 in *Electromagnetic Fields and Neurobehavioral Function*, M. E. O'Connor, and R. H. Lovely, eds. New York, NY: Alan R. Liss.

61. Liburdy, R. P. 1977. Effects of radiofrequency radiation on inflammation. *Radio Sci.* 12:179-183.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

62. Huang, A. T.-F., N. G. Mold. 1980. Immunologic and hematopoietic alterations by 2450-MHz electromagnetic radiation. *Bioelectromagnetics* 1:77-87.

63. Ragan, H. A., R. D. Phillips, R. L. Buschbom, R. H. Busch, and J. E. Morris. 1983. Hematologic and immunologic effects of pulsed microwaves in mice. *Bioelectromagnetics* 4:383-396.

64. Phillips, R. D., E. L. Hunt, R. D. Castro, and N. W. King. 1975. Thermoregulatory, metabolic and cardiovascular responses of rats to microwaves. *J. Appl. Physiol.* 38:630-635.

65. Chou, C.-K., K.-C. Yee, and A. W. Guy. 1980. Microwave radiation and heart-beat rate of rabbits. *J. Microwave Power* 15:87-93.

66. Tenforde, T. S. 1986. Interaction of ELF magnetic fields with living matter. Pp. 197-225 in *Handbook of Biological Effects of Electromagnetic Fields*, C. Polk, and E. Postow, eds. Boca Raton, FL: CRC Press.

67. Hilton, D. I., and R. D. Phillips. 1981. Growth and metabolism of rodents exposed to 60-Hz electric fields. *Bioelectromagnetics* 2:381-390.

68. Hauf, R. 1985. Hematological and biochemical effects of ELF fields in man: Laboratory experiments. Pp. 525-537 in *Biological Effects and Dosimetry of Static and ELF Electromagnetic Fields*, M. Grandolfo, S. M. Michaelson, and A. Rindi, eds. New York, NY: Plenum Press.

69. Sander, R., J. Brinkmann, B. Kuhne. 1982. Laboratory studies on animals and human beings exposed to 50 Hz electric and magnetic fields. In *Proc. Int. Conf. on Large High Voltage Electr. Syst.*, Paper No. 36-01 (Paris, France, Sept. 1982).

70. Prausnitz, S., C. Susskind, and P. O. Vogelhut. 1962. Effects of chronic microwave irradiation on mice. *I.R.E. Trans. Biomed. Electron.* 9:104-108.

71. Heynick, L. N. 1987. Critique of the literature on bioeffects of radiofrequency radiation: a comprehensive review pertinent to Air Force operations. USAFSAM-TR-87-3.

72. Roberts, N. J., Jr., and S. M. Michaelson. 1983. Microwaves and neoplasia in mice: Analysis of a reported risk. *Health Phys.* 44:430-433.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

73. Spalding, J. F., R. W. Freyman, and L. M. Holland. 1971. Effects of 800-MHz electromagnetic radiation on body weight, activity, hematopoiesis and life span in mice. *Health Phys.* 20:421-424.

74. Kunz, L. L., R. B. Johnson, D. Thompson, J. Crowley, C.-K. Chou, and A. W. Guy. 1985. Effects of long-term low-level longevity, cause of death and histopathological findings, University of Washington. USAMSAM-TR-85-11-8.

75. Szmigielski, S., A. Szudznski, A. Pietraszek, M. Bielec, M. Janiak, and J. K. Wrembel. 1982. Accelerated development of spontaneous and benzopyrene-induced skin cancer in mice exposed to 2,450-MHz microwave radiation. *Bioelectromagnetics* 3:179-191.

76. Santini, R., P. Hosni, P. Deschaux, and H. Pacheco. 1988. B16 melanoma development in black mice exposed to low-level microwave radiation. *Bioelectromagnetics* 9:105-107.

77. Roszkowski, M., J. K. Wrembel, K. Roszkowski, M. Janiak, and S. Szmigielski. 1980. Does whole-body hyperthermia therapy involve participation of the immune system? *Natl. J. Cancer* 25:289-292.

78. Preskorn, S. H., W. D. Edwards, and D. R. Justesen. 1978. Retarded tumor growth and greater longevity in mice after fetal irradiation by 2,450-MHz microwaves. *J. Surgical. Oncol.* 10:483-492.

79. McLean, J.R.N., M.A. Stuchly, R.E.J. Mitchell, D. Wilkinson, H. Yang, M. Goddard, D.W. Lecuyer, M. Schunk, E. Callary, and D. Morrison. 1991. Cancer promotion in a mouse-skin model by a 60-Hz magnetic field: II. Tumor development and immune response. *Bioelectromagnetics* 12:273-287.

80. Beniashvili, D. Sh., V. G. Bilanishvili, M. Z. Menabde. 1991. Low-frequency electromagnetic radiation enhances the induction of rat mammary tumors by nitrosomethyl urea. *Cancer Letters* 61:75-79.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

7

In Vitro Cellular and Subcellular End Points

As in the case of the other biologic end points reviewed here, there are few reports of in vitro end points related to electromagnetic fields (EMFs) with the same combination of frequencies and waveforms as encountered in GWEN. Therefore, a general overview of results of irradiation at frequencies up to 10 GHz must be considered, but with some attention to extension of results to possible GWEN-related health effects. According to the data presented in Chapter 3 and some additional plane-wave approximations, relevant exposure and dose metrics predicted for GWEN site boundaries are listed in Table 7-1:

TABLE 7-1 GWEN Exposure and Dose Metrics at Site Boundary

Exposure or Dose Metric	Low Frequency	Ultra-High Frequency
Maximum incident power density, W/m ²	7 ^a	0.01
Maximum electric field in human, V/m	2.2	1 ^a
Maximum magnetic induction in human, μ T	0.07	0.007 ^a
Maximum specific absorption rate (SAR), W/kg	0.001 ^a	0.0005
Maximum induced current density, A/m ²	0.7	1 ^a

^a Calculated from plane-wave approximation.

A practical problem in the literature is that typically only one or a few dose metrics associated with biologic exposures are given, other measures being omitted inadvertently, for convenience, or in the belief that they are not biologically relevant. The omitted dose metric is often large; it is then difficult to compare exposure conditions associated with reported bioeffects in relation to the GWEN levels shown in Table 7-1.

Three papers by the Brighton¹⁻³ group report effects of small in vitro exposures to 60-kHz continuous-wave electric fields, somewhat close to GWEN low-frequencies; these studies are described later. Otherwise, a review of the available in vitro data makes it readily apparent that almost all experiments at frequencies above GWEN

frequencies involve exposures at appreciably larger levels than shown in [Table 7-1](#), whereas some experiments at extremely low frequency (ELF) involve exposures at or below the levels shown in [Table 7-1](#). With very limited data at low-or ultra-high-frequencies, results with low-level ELF fields might seem applicable to the GWEN situation, but there are several pitfalls, as follows:

- At ELF, the intracellular electric fields and current densities are usually very small, because of poor coupling with external fields. Thus, an external field of hundreds of kilowatts per meter would be required to produce the internal fields listed in [Table 7-1](#).
- On the basis of such endpoints as field perception threshold, which have been studied over a wide range of frequencies, biological sensitivity at GWEN frequencies would be expected to be much less (lower by a factor as large as 100) than at ELF. On the basis of biologically equivalent field levels, the numbers in [Table 7-1](#) would be scaled down by the appropriate factor; unfortunately, this factor is unknown and speculative for many endpoints.
- There might be qualitative differences in biological response between ELF and low-frequency (LF) or ultra-high frequency (UHF).
- SAR is not considered to be an appropriate dose metric at ELF and is almost never reported in ELF studies.

Although population exposure to GWEN fields will produce no detectable tissue heating, most of the in vitro literature (excluding ELF exposure) deals with irradiation at SAR values higher than in the GWEN setting. That raises the question of extrapolation to low EMF levels and the specter of thermal artifacts. Some of the earlier reports of EMF biologic effects did not adequately describe temperature measurement and regulation and must be discounted. However, the temperature of cells exposed in vitro can be readily controlled by controlling the temperature of the surrounding culture medium, as reported by numerous investigators. This conclusion follows from the fact that it is impossible in aqueous media to maintain appreciable constant temperature gradients across cellular dimensions,⁴ and, except for extremely high-power radiation in submicrosecond pulses, transitory heating from pulsed irradiation is prevented by the brief (much less than 1 μ sec) thermal relaxation time for cells in aqueous media.⁵ Thus, reports with adequate descriptions of temperature measurements of an irradiated bulk cellular medium are acceptable on the grounds of thermometry, but extrapolation to GWEN of results obtained at higher field levels, different frequencies, and different waveforms remains open to question.

Comprehensive and recent reviews of the in vitro literature are available.⁶⁻¹² Up to a frequency of 10 GHz, most reported biological effects are in the categories shown in Table 7-2 and are considered separately in pages to follow (mutagenic effects, chromosomal aberrations, and neoplastic transformation). Analysis and numerous references through 1988 for the topics in the table are available in the cited reviews.

The amount of recent published research on EMF-induced cellular and subcellular effects appears to be small relative to the growing general concern for possible EMF hazards. Livingston et al.¹³ recently published the results of a battery of cytogenetic and cytotoxic tests that used human lymphocytes and Chinese hamster ovary fibroblasts exposed to 60-Hz electric current densities ranging from 0.03 to 30 A/m² and a magnetic field of 220/ μ T; the results were negative. Cleary et al.^{14,15} reported positive results of continuous 27- and 2,450-MHz radiation in inducing cell proliferation with SAR values more than 10,000 times those of GWEN. Another pair of papers^{16,17} presented negative results of searches to demonstrate an ion cyclotron resonance effect. However, Ross¹⁸ reported that fibroblast proliferation changed after exposure to mixed static and sinusoidally time-varying magnetic fields in the gauss range, and interpreted these results as consistent with a calcium ion cyclotron resonance mechanism. Lyle et al.¹⁹ reported that exposure to combined static and time-varying magnetic fields changed calcium uptake in normal and leukemic lymphocytes—further support of the idea of ion cyclotron resonance. Two studies with 60-Hz, 430-V/m electric-field exposure (more than 100 times the magnitude of GWEN fields) investigated changes in growth and hydrogen ion excretion in plant-root preparations.^{20,21}

Microwave-induced changes in channel formation in an artificial lipid bilayer system have been reported,²² but at field amplitudes and SAR values 100,000 times those relevant to GWEN. At the macromolecular level, alterations in ATPase activity after irradiation with microwaves (SAR, 20 W/kg) have recently been described.²³ Effects of exposure on DNA repair were recently investigated. Treatment of cells with 5 Gy of ⁶⁰Co radiation was followed by exposure to 60-Hz fields (magnetic field of 1 mT, electric field of 1 or 20 V/m, or combined fields of 0.2 V/m and 0.05 mT, 6 V/m and 0.6 mT, or 20 V/m and 1 mT) or sham exposure.²⁴ None of the exposures affected the repair of single-strand breaks. At the other extreme of amplitude, Fitzsimmons et al.²⁵ elicited bone tissue proliferation and mitogenic activity with an ELF electric field of approximately 10⁻⁵ V/m. Finally, three reports by Cossarizza et al.²⁶⁻²⁸ describe increases in cell proliferation and interleukin-2 activity and decreases in DNA repair in human lymphocytes exposed to pulsed fields similar to those used clinically for low-level electric facilitation of bone repair.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 7-2 Categories of In Vitro Experiments

Cellular and Subcellular System	Investigated End Points
Macromolecular preparations	Electromagnetic absorption in DNA, enzyme activity.
Prokaryotes	Growth rate, metabolism, enzyme activity.
Membrane	Cation flux, membrane fluidity, protein shedding, electrophoretic mobility.
Hematopoietic and Immunological	Cell growth, mitogenic response, lymphoblastoid transformation, T-lymphocyte activity, lymphocyte capping, protein synthesis, macrophage activity, levels of DNA and RNA.
Neurons	Firing rate, membrane potential, metabolic rate.
Salivary gland cells	RNA transcription, polypeptide synthesis.
Bone cells	Protein and DNA synthesis, Ca ²⁺ and cyclic AMP concentrations, collagenproduction, alkaline phosphatase and mitogen activity, cell proliferation.
Biochemistry	Changes in Ca ²⁺ efflux, ornithine decarboxylase, PKC.

Several groups of results will be considered in more detail below, selected according to the following criteria:

- Some of the reported dose metrics are near or below maximum GWEN values.

- The results come from a reasonably large and consistent body of evidence.
- The end points have perceived relevance to health effects.

BONE HEALING

In connection with in vivo experiments to stimulate bone healing in test animals, Brighton et al.¹⁻³ measured in vitro responses of cultured bovine chondrocytes and rat bone cells to 60-kHz continuous sine-wave electric fields administered by capacitive coupling for various durations between 5 min and 48 h. In some conditions, modest but statistically significant field-associated changes in isotope uptake and in cyclic AMP (cAMP) concentration were observed; they depended on such factors as field amplitude and serum concentration of the culture medium. Those results are of interest in relation to GWEN, because the induced voltages and currents that yielded positive results ranged down to less than 1 V/m—lower than the highest levels of GWEN LF fields shown in Table 7-1.

As reviewed by Bassett^{29, 30} and Tenforde,¹⁰ the use of small electromagnetic pulses to treat bone fractures and pseudoarthroses has developed into a refined clinical approach based on the delivery of well-defined electric fields to bone. That used to be accomplished via implantation of electrodes to impose the specified electric field; but pulsed external coils are now used to induce electric currents in bone by generating rapidly time-varying magnetic fields. Large magnetic fields (up to 400/ μ T) are also produced, but these are ignored and presumably are considered to be without biomedical significance, in light of the historical and theoretical explanation of the therapeutic action as a purely bioelectrical phenomenon. On these grounds, the relevant dose and exposure metrics are usually small, with current densities of 2-20 mA/m² (Tenforde)¹⁰ and induced electric fields near 0.2 V/m. Those values are lower than the maximum values at GWEN site boundaries (Table 7-1), even if we apply a bioeffective scaling factor that reduces the listed LF and UHF current densities and voltages. A typical electric-field waveform used for bone-fracture treatment is shown in Figure 7-1. Similar fields have been used in in vitro investigations with bone-cell cultures,^{31, 32} other cell types, including human lymphocytes,^{27, 28} and tumor cells.³³ Similar pulsed fields have also been found to alter RNA transcription.³⁴

MUTAGENIC EFFECTS

Variations in reported frequency, intensity, field orientation, period of exposure, and whether the field is continuous or pulsed make comparisons of data difficult. However, results of at least 20 studies of induction of gene mutations in bacteria, yeast, plants, insects, and mammalian cells in culture have been negative.³⁵ For example, a

recent study of forward mutations at the thymidine kinase locus in L5178 mouse leukemic cells showed no statistically significant increase in induced mutation frequency due to exposure to 2.45-GHz pulsed waves (500 or 600 w).³⁶ Several reports of positive results have, however, appeared. A single positive study in salmonella treated with 2.45-GHz fields showed increased induction of mutations, probably because of heating.³⁷ Studies in plants have also suggested induction of embryolethal and morphological mutants treated with 200-MHz radiowaves.³⁸ The most reasonable conclusion to draw from the full array of studies is that direct DNA damage does not occur.

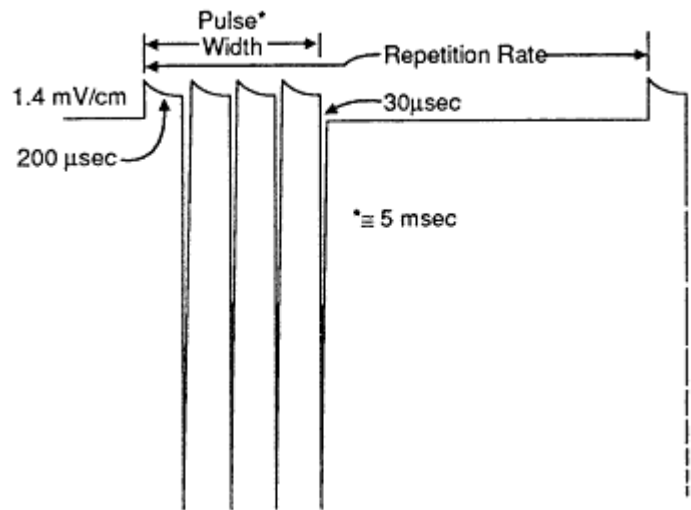


Figure 7-1.
Electric-field waveform used in bone-healing therapy.
Source: C.A.L. Bassett, 1978, ref. 29.

CYTOGENETIC EFFECTS

Studies of chromosomal changes in mammalian cells, although producing predominantly negative results, have sometimes produced conflicting results. Two studies have indicated chromosomal breaks after exposure to 2.45-GHz microwaves.^{39, 40} In one study, incident power varied between 25 and 200 W; 39 positive results were seen only when assays were performed without temperature control. In the other study, exposure of two kangaroo-rat cell lines maintained at 37°C with exposure to 2.45-GHz radiation demonstrated increases in chromosome aberrations in one line.⁴⁰ No increase in sister chromatid exchange (SCE) was seen after treatment of Chinese hamster ovary cells with 2.45 GHz.⁴¹

Most studies of chromosomal aberrations induced in mice and hamsters with in vivo exposure, especially those at extremely low frequencies, have had negative results. For example, a recent study of 60-Hz fields with current densities of $3\text{--}3,000/\mu\text{A}/\text{cm}^2$ and a fixed magnetic field of 0.22 mT produced negative results.¹³ However, significant increases were found in chromosomal aberrations in Ehrlich ascites tumor cells exposed in the peritoneal cavities of mice for 14 d to horizontal electric fields of 80-160 V/cm (60 Hz), compared with controls.⁴² Vertical electric fields produced no difference from controls, and no effect was seen with a 4-15 wk exposure.⁴³

Female CD-1 mice treated with 2.45-GHz microwaves at $200\text{ W}/\text{m}^2$ did not differ from controls in SCE frequency in bone marrow cells.⁴⁴ Other studies in mice also failed to demonstrate significant differences in chromosomal aberrations in spermatocytes⁴⁵ or bone marrow⁴⁶ after treatment with 2.45-GHz microwaves; but positive results have been reported in rat bone marrow⁴⁷ and Chinese hamster corneal epithelium.⁴⁸

Studies on human cells exposed in vitro to low frequencies have also produced conflicting results. No increase in chromosomal breaks, SCE, or chromosomal aberrations were seen in several studies.^{13, 49-52} For example, one study used 60-Hz fields with variable electric current densities of $3\text{--}3,000/\mu\text{A}/\text{cm}^2$ and a fixed magnetic field of 0.22mT.¹³ No changes in SCE, micronuclei, clonogenicity, or cell-cycle kinetics were observed. No increase in micronuclei after exposure to pulsed magnetic fields (50 Hz, 2.5 mT) was observed in human lymphocytes;⁵³ nor did the exposure influence the extent of micronuclei induction by mitomycin C. However, some studies have reported positive results. No increase in aberrations were observed in human lymphocytes following exposure of whole blood to a 50-Hz electric field, but exposure to 10-/μsec long spark-discharge pulses did cause a significant increase in chromosomal breaks.⁵⁴ A recent study of pulsing 50-Hz fields (1.05 mT) demonstrated a suppression of mitotic activity and higher incidence of chromosomal aberrations after 24, 48, and 72 h of treatment.⁵⁵ A reduction in the cell proliferation index and an increase in SCE rate were seen only after 72 h of exposure.

Treatment of human lymphocytes with radiofrequencies has produced conflicting results, one study showing increases in chromosomal stickiness, breaks, and dicentrics,⁵⁶ and another, using a magnetic resonance imaging device, showing no increase in chromosomal aberrations or SCE.⁵⁷

In summary, studies of chromosomal effects of EMFs have produced conflicting results. Most studies at extremely low frequencies have been negative, although exceptions can be found. At radiofrequencies, some positive studies of chromosomal aberrations have been reported, but studies of SCE have been predominantly negative.

CELL TRANSFORMATION

Exposure to 2.45-GHz radiation at 120 pulses/sec followed by treatment with the tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) induced the transformation of mouse C3H/10T1/2 cells.⁵⁸ TPA treatment of cells previously irradiated with microwaves and x rays led to a statistically significant 3.5-to 1.6-fold increase in transformation, compared with the transformation frequency of cells previously treated with x rays alone at 1.5 and 4.5 Gy, respectively. A later study⁵⁹ showed that treatment with microwaves followed by TPA significantly increased the transformation frequency. Additional studies showed a dose-response relationship between field exposures (SAR values of 0.1, 1, and 4.4 W/kg) and transformation frequency.⁶⁰ EMFs alone had no effect on cell survival or induction of transformation. Transformation by fields plus TPA was as great as that produced by 1.5 Gy of x rays. The frequency of transformation was additive when doses of x rays were given as a cocarcinogen.

In contrast with most other in vitro studies on the genotoxicity of EMFs, these results suggest that EMFs may act as an initiator. A long-standing criticism of the 10T1/2 system is that it uses cells that are immortalized, and might already have genetic changes. Hence, it is not strictly comparable to an animal model of initiation and promotion.

EFFECTS ON TRANSCRIPTION

Salivary gland chromosomes of the dipteran *Sciara coprophilia* have been used to study effects of weak pulsed EMFs on transcription.³⁴ Normal transcription patterns during cell differentiation are known from transcription autoradiography and can be correlated with chromosomal structure by banding and puff formation in the four polytene chromosomes. Transcription was measured in three ways: nascent RNA chains attached to specific chromosomal regions were identified with conventional autoradiography after incorporation of [³H] uridine; regions sensitive to DNase I, and therefore transcriptionally active, were examined with nick translation directly on the DNA of the cytological preparations; and RNAs of various size classes were isolated with sucrose density-gradient centrifugation. In the presence of a repetitive single pulse signal (single 380- μ sec pulses used clinically to yield 0.15 V/m in bone, repeated at 72 Hz), marked and specific increases in RNA transcription in most of the bands and interbands of the chromosomes were seen at 15 and 45 min. At 30 min, transcription was approximately equal to control values. With the repetitive pulse train signal (5-msec pulse trains of 200- μ sec pulses repeated at 15 Hz), increases in RNA transcription were seen only after 45 min of exposure and were lower than with the single pulse. Sucrose gradient analysis of RNA indicated a 4-fold increase in total RNA and an 11-fold increase in mRNA with the single pulse.

Those results were later extended to show that sinusoidal fields with the same frequency as the repetition frequency of the fast single-pulse signals also induce increases in transcriptional activity.⁶¹ Sine waves of 72, 222, or 4,400 Hz (measured magnetic fields of 1.15, 0.37, or 0.018 mT) were used. At higher frequencies, the same general pattern of induced transcription was seen, but it was not as dramatic as that observed at 72 Hz. Analysis of the grain-count distribution over the X chromosome of *Sciara* showed augmented uptake of [³H] uridine into RNA after short exposures of the cells to three signals: single repetitive pulse at 72 Hz (15 mV and 3.5 mT), repetitive pulse bursts at 15 Hz (14.5 mV and 1.9 mT), and sine waves at 72 Hz (0.8 mV and 1.1 mT).⁶² The quoted voltages refer to coil-probe readings with calculated electric fields in cortical bone not exceeding 0.15 V/m. The electric fields were induced by magnetic fields at the quoted strengths. Transcription was augmented in previously active loci, as well as in chromosomal regions that were not detectably active in control cells.

Polypeptide synthesis after exposure to fields of various waveforms and frequencies was studied with two-dimensional gel electrophoresis.⁶³ The polypeptide patterns were qualitatively and quantitatively different for each type of exposure and different from those of control and heat-shocked cells. Individual proteins were not identified.

The same group of investigators has demonstrated increases in selected RNA transcripts in cultured human cells (HL60).⁶⁴ RNA with homology to β -actin, histone H2B, and v-myc increased 20 min after exposure to five ELF electromagnetic signals. Continuous sine waves at 60 and 72 Hz (coil-probe reading, 0.8 mV; 1.1 mT) produced the greatest increase, followed by the pulse train (burst-repetition rate, 15 Hz) and a single pulse (72 Hz, 15 mV, 3.5 mT). A followup study of sinusoidal fields at 15-150 Hz (0.5 mV, 0.2-2.3 mT) also demonstrated an increase in c-myc and histone H2B transcripts.⁶⁵ The maximum increase (4-fold over control) occurred at 45 Hz.

Another group has also investigated changes in transcription by measuring [³H]uridine incorporation into HL60 cells after exposure to a sine-wave ELF field at 60 Hz.⁶⁶ Cells were exposed to a uniform magnetic field, usually at 1 mT, with different samples simultaneously exposed to induced electric fields of 0-4.5 mV/m. There was no effect in cells exposed to a null electric field, so the magnetic field was claimed to play no role in the transcriptional changes. A transient increase in transcription rates with a maximum of 50 or 60% enhancement was seen after 30-120 min of exposure to very small electric fields; the rate declined to basal values by 18 h.

A mathematical linear, multistep reaction model has been developed to demonstrate that a transient response to fields can lead to apparent amplitude windows.⁶⁷ The model assumes that switching on the fields causes a sudden increase in an intermediate reaction rate in the synthesis of RNA and that the change in reaction rate increases with field strength.

One study in bacteria demonstrated effects on the *lac* operon in *E. coli*.⁶⁸ The β -galactosidase gene is under control of the *lac* operon and normally is repressed. Changes in repressor protein lead to changes in the rate of synthesis of β -galactosidase. Bacteria exposed to a 50-Hz square-wave magnetic field of 0-0.7 mT displayed a complex response with increasing field strength. A decrease was seen at fields up to 0.3 mT, then an increase up to 0.54 mT, then another decrease at field levels greater than 0.56 mT. Results also depended on cell concentration. Strengths of induced electric fields were not reported.

Those studies demonstrate biological effects, but not health consequences. Changes in transcription can result from normal stimulation of cell growth. In most of the studies, the electric-field strength was lower than the corresponding GWEN values (Table 7-1), but the magnetic fields were much larger.

TUMOR PROMOTION

The preponderance of negative over positive results with respect to direct genotoxic or clastogenic effect of EMFs has led many investigators to believe that these fields can be carcinogenic only in the sense of tumor promotion. The idea of two-stage carcinogenesis and tumor promotion was developed originally in animal models, and it assumes that a subthreshold exposure to a carcinogen (initiation) is followed by continuous application of a nongenotoxic irritant (promotion). Although tumor promoters are not carcinogenic themselves and are not genotoxic agents, they have a proliferative effect that allows the expansion and selection of initiated (genetically altered) cells.^{69, 70} It is now accepted that many promoters act at the level of membrane signal transduction, altering the normal homeostatic mechanisms of the cell. It is also accepted that, to be called a tumor promoter, a substance must induce proliferation in the target cell population.⁷¹

Three lines of evidence have been used to support a putative promotional effect of EMFs on tumor formation: field-induced changes in Ca^{2+} , induction of ornithine decarboxylase, and alterations in cellular kinases. Because the reported evidence involves relatively small fields with respect to GWEN and has generated concern for hazards, a critique of methods and interpretations is appropriate.

Calcium Homeostasis

It is generally accepted that Ca^{2+} plays a major role in signal-transduction mechanisms in the cell membrane. Increases in free Ca^{2+} in the cytoplasm have been shown after exposure of cell cultures to epidermal growth factor (EGF),⁷² and it is known that calcium-dependent processes, such as the generation of cAMP, alter the binding of some hormones to their receptors.⁷³ From the point of view of promotion,

the most relevant aspect of calcium-dependent processes is the phospholipase C/protein kinase C (PKC) cascade.⁷⁴ The activation of phospholipase C results in an increase in cytoplasmic free Ca^{2+} as a result of the release of inositol phosphate from the cell membrane. PKC is activated by diacylglycerol (also released by the action of phospholipase C) and calcium. We know that TPA, the classical skin-tumor promoter, binds and activates PKC, and it is believed that tumor promotion is mediated through PKC activation. It has been speculated that modulation of intracellular Ca^{2+} by EMF has an effect on signal transduction mechanisms; therefore, this modulation could be a mechanism of tumor promotion.

Since the early work of Bawin et al.,^{75, 76} at least two laboratories have published reports showing that EMFs affect the efflux of Ca^{2+} from brain tissue. The basic assay used in most of the studies^{76, 77} is carried out on entire forebrain removed from the animal and pre-incubated in a physiologic solution containing radioactive calcium. After being washed in a nonradioactive solution to eliminate loosely associated radioactive calcium, the tissues are exposed to the EMF, and the radioactive calcium released by the tissue is determined. Bawin et al.⁷⁵ showed a modestly increased efflux of calcium after exposure to 147-MHz RF fields modulated between 6 and 25 Hz, but not the unmodulated carrier wave (147 MHz) alone. The incident power density was 10-20 W/m². The resulting tissue electric field of 1-10 V/m is too low to directly influence Ca^{2+} binding by a thermal mechanism.⁷⁸ The maximum effect occurred at a modulation frequency of 16 Hz. The same laboratory reported an apparently contradictory study in which a decrease in Ca^{2+} efflux, rather than an increase, occurred when electric fields without an RF carrier were used; this study also contradicted the study of Blackman et al.,⁷⁷ which showed that fields without an RF carrier produce an increase in Ca^{2+} efflux.

The work of Bawin et al.^{75, 76} and the data of Blackman et al.^{77, 79} indicate that the effect of EMFs on Ca^{2+} efflux occurs only at specific amplitudes and frequencies. Unlike most chemical-or radiation-induced biological effects in which a dose-response relation is normally shown (i.e., increasing dose results in an increase in effect until the effect eventually reaches a plateau), EMFs produced a maximum effect at specific windows (e.g., 5-10 V/m in air and 30-60 V/m in air), with insignificant trends at lower or higher intensities. Frequency windows were also described. RF waves appear not to affect Ca^{2+} efflux, except when they are modulated between 6 and 25 Hz. Therefore, the essential component seems to be an ELF field centered around 16 Hz. (Effects have also been reported at 45, 60, 75, 90, and 105 Hz, and it has been suggested that the results are related to the fact that these frequencies are multiples or harmonics, of 15 Hz.) The electric field in cell-culture media or tissue was much less than 1 V/m in the ELF experiments.

The effect of Ca^{2+} efflux seems to be largely restricted to neural tissue. In the Bawin and Adey study,⁷⁶ no effect was observed in muscle subjected to the same experimental conditions as the brain.^{73, 79} However, a positive effect was obtained by

Lin-Liu and Adey⁸⁰ in isolated synaptosomes, and this further suggests the tissue specificity of the phenomenon. In contrast, the positive results with efflux in bone tissue exposed in vitro to small electromagnetic pulses must be recalled.^{31, 32}

More recent studies showed similar results in tissue-culture systems. Dutta et al.⁸¹ reported enhanced efflux of ions in cultured neuroblastoma cells exposed to 915 or 147 MHz,⁸² both modulated at 16 Hz. SAR values in those experiments were as low as 0.0005 W/Kg; positive results were reported at 0.05 and 0.005 W/Kg.

Two major subjects warrant discussion regarding these studies: the experimental procedure itself and the possible relevance of the findings to tumor promotion. The studies have been criticized for their experimental model.⁸³ Almost all have been performed with the experimental approach described by Bawin and Adey in 1976,⁷⁶ which uses isolated whole-brain hemispheres. It has been shown that electrical activity disappears within minutes of tissue removal from the animal and that the amount of available O₂ is drastically reduced in the first 10 min. Although Blackman et al.^{77, 79} have argued that the tissue is metabolically active, it is difficult to assess the extent to which residual metabolic activity is comparable with physiological cell metabolism and the extent to which normal cell structures are preserved. It is well known that Ca²⁺ movements take place and are part of the physiopathology of necrosis caused by anoxia. As to the relevance of the studies for tumor promotion, although they might explain some of the behavioral or neurophysiological effects of EMFs, the implications for tumor promotion are speculative. As discussed earlier, it is accepted that tumor promoters induce proliferation in the target tissue. However, in the experiments on modulation of Ca²⁺ efflux, it has not been shown that the effects are related to perturbation in any homeostatic mechanism. Furthermore, no experimental evidence links the changes in Ca²⁺ movement with cell proliferation. In summary, although a link between EMFs and signal transduction by alteration of Ca²⁺ levels cannot be ruled out, it is premature to infer that such mechanisms are involved in tumor promotion.

Ornithine decarboxylase

Ornithine decarboxylase (ODC) is the rate-limiting enzyme in the polyamine biosynthetic pathway. Although the function of polyamines has not been completely elucidated, it is known that they play a role in cell proliferation. Induction of proliferation in quiescent cell populations by hormones, growth factors, or tumor promoters is normally preceded by ODC induction and increases in cellular polyamines.⁸⁴ The induction of ODC by most tumor promoters has been considered a necessary, although not sufficient, step in tumor promotion, so ODC has been used as an early indicator of tumor promotional effects.^{69, 70} Some studies have suggested that EMFs can induce ODC in tissue-culture cells. Byus et al.⁸⁵ studied the effect of small 60-Hz electric fields on ODC activities of three established cell lines (human CEM lymphoma cells, mouse P3 myeloma cells, and Reuber H35 hepatoma cells).

Cells were exposed for 1 h to an electric field of 1 V/m. The EMF produced a transient increase in ODC activity in all cell lines, from about 40% in Reuber cells to about 400% in CEM cells. The peak of activity was different in the three cell types, but all the cells returned to normal values 3 h after exposure. In the same study, Reuber H35 cells were exposed to fields of varied strength (0.01-1 V/m), and an effect on ODC was seen only immediately after the termination of exposure and only at the highest and lowest exposures. Exposures for longer periods either had no effect or else decreased ODC activity, depending on the duration of exposure.

In a second study,⁸⁶ the same group exposed three cell lines (Reuber H35, CHO, and 294T melanoma) to a 16-Hz-modulated microwave field (450 MHz, 10 W/m²). The cultures were measured during the logarithmic phase growth, as opposed to the quiescent phase in the previous study. ODC activity increased significantly (by up to 50%) in all three lines. The effect of different modulation frequencies on ODC activity was also investigated in Reuber H35 hepatoma cells. The maximum effect was produced with modulation at 16 Hz; frequencies of 60 and 100 Hz failed to produce any effect. Interaction of EMFs and TPA was another subject of the study. The experiments suggested a synergistic effect of TPA and EMFs on ODC induction.

Although those authors have shown that ELF electric fields and 16-Hz amplitude-modulated RF fields have some biological effect on ODC activity, they have not shown ODC induction by these fields. Enzymatic activity can be regulated at transcriptional, translational, and post-translational levels. The modest reported changes in enzymatic activity (compared with those seen with growth factors and tumor promoters) and the kinetics of the effect of EMFs (very different from the kinetics of induction by tumor promoters) argue against the possibility that these changes represent true enzymatic induction. No known studies have attempted to show at what degree of cellular metabolic regulation the fields interact. All these studies were performed in transformed cell lines, and there are no reports of similar EMF effects on animals or even on nontransformed cells in culture. Finally, in all cases where tumor promoters have been shown to induce ODC, the induction of the enzyme has been coupled with induction of cell proliferation; this has not been the case in the studies on EMF effects.

Protein Kinase C

In a single study published in 1984, Byus et al.⁸⁷ studied the effect of 450-MHz microwaves modulated at 3-100 Hz on protein kinases in cultures of human tonsil lymphocytes. The incident power density was 10 W/m². The RF carrier field did not produce any effect on cAMP-dependent kinase activity, but a decrease of about 50% in the cAMP-independent kinase activity, PKC, was reported for fields modulated at 16 Hz. The decrease in enzymatic activity was transient, and activity returned to normal within 1 h, even in the presence of continued field exposure. Other modulation

frequencies produced less marked results, and unmodulated 450-MHz fields did not produce any detectable changes in PKC.

That study has been considered of special interest, because PKC has been shown to mediate many of the actions of TPA and other tumor promoters (e.g., teleocidine). The putative modulation (down-regulation) of a protein kinase by EMFs has been used to support a putative tumor-promotional activity of EMFs. However, although the study was reported 8 yr ago, there has been no confirmation that the protein kinase involved was actually PKC. It is surprising that, in spite of the fact that PKC modulation by EMFs would constitute a strong argument in favor of tumor promotion, no attempt has been made to use more novel approaches to confirm the results independently.

CONCLUSIONS

There are no data on cellular and subcellular end points after exposure to EMFs matching those of GWEN in frequency, waveform, or duty cycle. We are forced to state, in the absence of reports on cellular response to fields even remotely resembling GWEN fields, that firm conclusions cannot be drawn. In a search for a match of experimental exposures with maximum exposures expected at a GWEN station site boundary, we found some experiments that yielded positive results. However, the exposures used are much higher than expected population exposures associated with GWEN fields, and the experimental end points involved have no demonstrated connection with health risk. Furthermore, the extremely low duty cycle of GWEN must be acknowledged, and the precautions regarding interfrequency comparisons must be applied.

An interesting perspective for risk assessment is provided by the observation of beneficial results of low-level magnetic pulses in bone healing. Those pulses are regarded by practitioners and patients as therapeutic, and the possible adverse effects on exposed tissues are not considered to be important in clinical practice.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

REFERENCES

1. Brighton, C. T., A. S. Unger, and J. L. Stambough. 1984. In vivo growth of bovine articular cartilage chondrocytes in various capacitively coupled electrical fields. *J. of Ortho. Res.* 2:15-22.
2. Brighton, C. T., and P. F. Townsend. 1988. Increased cAMP production after short-term capacitively coupled stimulation in bovine growth plate chondrocytes. *J. of Ortho. Res.* 6:552-558.
3. Brighton, C. T., and W. P. McCluskey. 1988. Response of cultured bone cells to a capacitively coupled electric field: inhibition of cAMP response to parathyroid hormone. *J. of Ortho. Res.* 6:567-571.
4. Love, L. A., and F. W. Kremkau. 1980. Intracellular temperature distribution produced by ultrasound. *J. Acoust. Soc. Am.* 67:1045-1051.
5. Friend, A. W., Jr., S. L. Gartner, K. R. Foster, and H. Howe, Jr. 1981. The effects of high power microwave pulses on red blood cells and the relationship to transmembrane thermal gradients. *IEEE Trans. Microwave Theory and Techniques* MTT-29:1271-1276.
6. Cleary, S. F. 1989. Biological effects of radiofrequency radiation: an overview. Pp. 59-79 in *Electromagnetic Biointeraction. Mechanisms, Safety Standards, Protection Guides*, G. Franceschetti, O. P. Gandhi, and M. Grandolfo, eds. New York and London: Plenum Press.
7. Cleary, S. F. 1990. Cellular effects of radiofrequency electromagnetic fields. Pp. 339-366 in *Biological Effects Medical Applications of Electromagnetic Energy*, O. P. Gandhi, ed. Englewood Cliffs, NJ: Prentice Hall.
8. Anderson, L. E. 1990. Biological effects of extremely low frequency and 60 Hz fields. Chapter 9 in *Biological Effects and Medical Appliances of Electromagnetic Energy*, O.P. Gandhi, ed. Englewood Cliffs, NJ: Prentice Hall.
9. Miller, M. W. 1986. Extremely low frequency (ELF) electrical fields: experimental work on biological effects. Pp. 139-168 in *CRC Handbook of Biological Effects of Electromagnetic Fields*, C. Polk and E. Postow, eds. Boca Raton, FL: CRC Press.
10. Tenforde, T. S. 1986. Interaction of ELF magnetic fields with living matter. Pp. 197-225 in *CRC Handbook of Biological Effects of Electromagnetic Fields*, C. Polk and E. Postow, eds. Boca Raton, FL: CRC Press.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

11. Michaelson, S. M. 1986. Interaction of nonmodulated fields with living matter. Pp. 339-423 in CRC Handbook of Biological Effects of Electromagnetic Fields, C. Polk and E. Postow, eds. Boca Raton, FL: CRC Press.
12. Postow, E., and M. L. Swicord. Modulated fields and "window" effects. 1986. Pp. 425-460 in CRC Handbook of Biological Effects of Electromagnetic Fields, C. Polk and E. Postow, eds. Boca Raton, FL: CRC Press.
13. Livingston, G. K., K. L. Witt, O. P. Gandhi, I. Chatterjee, and J. L. Roti Roti. 1991. Reproductive integrity of mammalian cells exposed to power frequency electromagnetic fields. Environ. Mol. Mutag. 17:49-58.
14. Cleary, S. F., L.-M. Liu, and R. E. Merchant. 1990. In vitro lymphocyte proliferation induced by radio-frequency electromagnetic radiation under isothermal conditions. Bioelectromagnetics 11:47-56.
15. Cleary, S. F., L.-M. Liu, and R. E. Merchant. 1990. Glioma proliferation modulated in vitro by isothermal radiofrequency radiation exposure. Radiat. Res. 121:38-45.
16. Liboff, A. R., and W. C. Parkinson. 1991. Search for ion-cyclotron resonance in an Na⁺-transport system. Bioelectromagnetics 12:77-84.
17. Parkinson, W. C., and C. T. Hanks. 1989. Search for cyclotron resonance in cells in vitro. Bioelectromagnetics 10:129-146.
18. Ross, S. M. 1990. Combined DC and ELF magnetic fields can alter cell proliferation. Bioelectromagnetics 11:27-36.
19. Lyle, D.B., X. Wang, R. D. Ayotte, A. R. Sheppard, and W. R. Adey. 1991. Calcium uptake by normal and leukemic T-lymphocytes exposed to low-frequency magnetic fields. Bioelectromagnetics 12:145-156.
20. Azadniv, M., M. W. Miller, A. A. Brayman, and C. Cox. 1990. Repetitive pulsed-train "off" duration mitigates reductions in root growth rates of *Pisum sativum* L. induced by 60-Hz electric field. Radiat. Res. 124:62-65.
21. Brayman, A. A., and M. W. Miller. 1990. 60-Hz electric field exposure inhibits net apparent H⁺-ion excretion from excised roots of *Zea mays* L. Radiat. Res. 123:22-31.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

22. Sandblom, J., and S. Theander. 1991. The effect of microwave radiation on the stability and formation of gramicidin-A channels in lipid bilayer membranes. *Bioelectromagnetics* 12:9-20.
23. Brown, H. D., and S. K. Chattopadhyay. 1991. Ouabain inhibition of kidney ATPase by 9.14 GHz radiation. *Bioelectromagnetics* 12:137-144.
24. Frazier, M. E., J. A. Reese, J. E. Morris, R. F. Jostes, and D. L. Miller. 1990. Exposure of mammalian cells to 60-Hz magnetic or electric fields: analysis of DNA repair of induced, single-strand breaks. *Bioelectromagnetics* 11(3):229-234.
25. Fitzsimmons, R. J., J. R. Farley, W. R. Adey, and D. J. Baylink. 1989. Frequency dependence of increased cell proliferation in vitro in exposures to a low-amplitude, low-frequency electric field: evidence for dependence on increased mitogen activity released into culture medium. *J. Cellular Physiology* 139:586-591.
26. Cossarizza, A., D. Monti, F. Bersani, R. Paganelli, G. Montagnani, R. Cadossi, M. Cantini, and C. Franceschi. 1989. Extremely low frequency pulsed electromagnetic fields increase interleukin-2 (IL-2) utilization and IL-2 receptor expression in mitogen-stimulated human lymphocytes from old subjects. *FEBS* 248:141-144.
27. Cossarizza, A., D. Monti, F. Bersani, R. Cadossi, M. Cantini, and C. Franceschi. 1989. Extremely low frequency pulsed electromagnetic fields increase cell proliferation in lymphocytes from young and aged persons. *Biochem. Biophys. Res. Comm.* 160:692-698.
28. Cossarizza, A., D. Monti, P. Sola, G. Moschini, R. Cadossi, F. Bersani, and C. Franceschi. 1989. DNA repair after gamma irradiation in lymphocytes exposed to low-frequency pulsed electromagnetic fields. *Radiat. Res.* 118:161-168.
29. Bassett, C. A. L. 1978. Pulsing electromagnetic fields: a new approach to surgical problems. Pp. 255-306 in *Metabolic Surgery*, H. Buchwald and R. L. Varco, eds. New York: Grune and Stratton.
30. Bassett, C. A. L., S. N. Mitchell, and S. R. Gaston. 1982. Pulsing electromagnetic field treatment in ununited fractures and failed arthroses. *JAMA* 247:623-626.
31. Colacicco, G., and A. A. Pilla. 1983. Electromagnetic modulation of biological processes: bicarbonate effect and mechanistic considerations in the

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Ca²⁺ uptake by embryonal chick tibia in vitro. *Z. Naturforsch.* [C] 38:465-467.

32. Colacicco, G., and A. A. Pilla. 1984. Electromagnetic modulation of biological processes: influence of culture media and significance of methodology in the Ca-uptake by embryonal chick tibia in vitro. *Calcified Tissue Int.* 36:167-174.

33. Colacicco, G., and A. A. Pilla. 1983. Electromagnetic modulation of biological processes: ATPase function and DNA production by Raji cancer cells in vitro. *Z. Naturforsch.* [C] 38:468-470.

34. Goodman, R., C. A. Bassett, and A. S. Henderson. 1983. Pulsing electromagnetic fields induce cellular transcription. *Science* 220:1283-1285.

35. Brown, H. D., and S. K. Chattopadhyay. 1988. Electromagnetic-field exposure and cancer. *Cancer Biochem. Biophys.* 9:295-342.

36. Meltz, M. L., P. Eagan, and D. N. Erwin. 1990. Proflavin and microwave radiation: Absence of a mutagenic interaction. *Bioelectromagnetics* 11:149-158.

37. Blevins, R. D., R. C. Crenshaw, A. E. Hougland, and C. E. Clark. 1980. The effects of microwave radiation and heat on specific mutants of *Salmonella typhimurium* LT2. *Radiat. Res.* 82:511-517.

38. Harte, C. 1973. Mutations in *Oenothera hookeri* after prolonged influence of radiowaves during one vegetation period. *Theor. Appl. Genet.* 43:6-12.

39. Alam, M. T., N. Barthakur, N. G. Lambert, and S. S. Kasatiya. 1978. Cytological effects of microwave radiation in Chinese hamster cells in vitro. *Can. J. Genet. Cytol.* 20:23-30.

40. Yao, K. T. S., and M. M. Jiles. 1970. Effects of 2450 MHz microwave radiation on cultivated kangaroo-rat cells. Pp. 123-133 in *Biological Effects and Microwave Radiation: Symposium Proceedings*, S.F. Cleary, ed. Public Health Service Publication No. BRH/DBE 70-2. Government Printing Office: Washington, D.C.

41. Ciaravino, M., M. L. Meltz, and D. N. Erwin. 1987. Effects of radiofrequency radiation and simultaneous exposure with mitomycin C on the frequency of sister chromatid exchanges in Chinese hamster ovary cells. *Environ. Mutagen.* 9:393-399.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

IN VITRO CELLULAR AND SUBCELLULAR END POINTS	103
42. Mitchell, J. A., A. A. Marino, T. J. Berger, and R. O. Becker. 1978. Effect of electrostatic fields on the chromosomes of Ehrlich ascites tumor cells exposed in vivo. <i>Physiol. Chem. Phys.</i> 10:79-85.	
43. Marino, A. A., T. J. Berger, J. T. Mitchell, B. A. Duhacek, and R. O. Becker. 1974. Electric field effects in selected biologic systems. <i>Ann. N.Y. Acad. Sci.</i> 238:436-443.	
44. McRee, D. I., G. MacNichols, and G. K. Livingston. 1981. Incidence of sister chromatid exchange in bone marrow cells of the mouse following microwave exposure. <i>Radiat. Res.</i> 85:340-348.	
45. Beechey, C. V., D. Brooker, C. I. Kowalczyk, R. D. Saunders, and A. G. Searle. 1986. Cytogenetic effects of microwave irradiation on male germ cells of the mouse. <i>Int. J. Radiat. Biol.</i> 50:909-918.	
46. Ramaiya, L. K., M. D. Pomerantseva, G. A. Vilкина, and V. S. Tikhonchuk. 1980. Study of the action of UHF microwaves on mammalian germ and somatic cells. <i>Cytol. Genet.</i> 14:1-5.	
47. Kapustin, A. A., M. I. Rudnev, G. I. Leonskaya, and G. K. Konobeeva. 1976. Cytogenetic effects of a varying UHF electromagnetic field. <i>Cytol. Genet.</i> 10:16-18.	
48. Yao, K. T. S. 1982. Cytogenetic consequences of microwave irradiation on mammalian cells incubated in vitro. <i>J. Hered.</i> 73:133-138.	
49. Cohen, M. M., A. Jybsjam, J. A. Asterborski, D. McCulloch, and D. A. Paskewitz. 1986. Effect of low-level 60-Hz electromagnetic fields on human lymphoid cells: I. Mitotic lymphocytes. <i>Bioelectromagnetics</i> 7:415-424.	
50. Cohen, M. M., A. Kunska, J. A. Astemborski, and D. McCulloch. 1986. The effect of low-level 60-Hz electromagnetic fields on human lymphoid cells: II. Sister chromatid exchanges in peripheral lymphocytes and lymphoblastoid cell lines. <i>Mutat. Res.</i> 172:177-184.	
51. Rosenthal, M., and G. Obe. 1989. Effects of 50-Hz electromagnetic fields on proliferation and on chromosomal alterations in human peripheral lymphocytes untreated or pretreated with chemical mutagens. <i>Mutat. Res.</i> 210:329-335.	

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

52. Garcia-Sagredo, J. M., L. A. Parada, and J. L. Monteagudo. 1990. Effect on SCE in human chromosomes in vitro of low-level pulsed magnetic field. *Environ. and Mol. Mutag.* 16:185-188.

53. Scarfi, M. R., F. Bersane, A. Cossarizza, D. Monti, G. Castellani, R. Cadossi, G. Franceschetti, and C. Franceschi. 1991. Spontaneous and mitomycin C-induced micronuclei in human lymphocytes exposed to extremely low frequency pulsed fields. *Biochem. Biophys. Res. Commun.* 176:194-200.

54. Nordenson, I., K. Hansson-Mild, S. Nordstrom, A. Sweins, and E. Birke. 1984. Clastogenic effects in human lymphocytes of power frequency electric fields: In vivo and in vitro studies. *Radiat. Environ. Biophys.* 23:191-201.

55. Khalil, A. M., and W. Qassem. 1991. Cytogenetic effects of pulsing electromagnetic fields on human lymphocytes in vitro: chromosome aberration, sister chromatid exchanges and cell kinetics. *Mutat. Res.* 274:141-146.

56. Stodolnik-Baranska, W. 1984. The effects of microwaves on human lymphocyte cultures. *Biologic effects and health hazards of microwave radiation. Proc. Int. Symp. Warsaw, 1973. Pol. Med. Publishers.* Pp. 189-195.

57. Wolff, S., T. L. James, G. B. Young, A. R. Margulis, J. Bodycote, and V. Afzal. 1985. Magnetic resonance imaging: Absence of in vitro cytogenetic damage. *Radiology* 155:163-165.

58. Balcer-Kubiczek, E. K., and G. H. Harrison. 1985. Evidence for microwave carcinogenesis in vitro. *Carc.* 6(6):859-864.

59. Balcer-Kubiczek, E. K. and G. H. Harrison. 1989. Induction of Neoplastic transformation in C3H/10T1/2 cells by 2.45 GHz microwaves and phorbol ester. *Radiat. Res.* 117:351-537.

60. Balcer-Kubiczek, E. K., and G. H. Harrison. 1991. Neoplastic transformation of C3H/10T1/2 cells following exposure to 120-Hz modulated 2.5 GHz microwaves and phorbol ester tumor promoter. *Radiat. Res.* 126:65-72.

61. Goodman, R., and A. S. Henderson. 1986. Sine waves enhance cellular transcription. *Bioelectromagnetics* 7:23-29.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

62. Goodman, R., J. Abbott, and A. S. Henderson. 1987. Transcriptional patterns in the X chromosome of *Sciara coprophilia* following exposure to magnetic fields. *Bioelectromagnetics* 8: 1-7.

63. Goodman, R., and A. S. Henderson. 1988. Exposure of salivary gland cells to low-frequency electromagnetic fields alters polypeptide synthesis. *Proc. Natl. Acad. Sci. (USA)* 85:3928-3932.

64. Goodman, R., L. X. Wei, J. C. Xu, and A. Henderson. 1989. Exposure of human cells to low-frequency electromagnetic fields results in quantitative changes in transcripts. *Biochem. Biophys. Acta* 1009:216-220.

65. Wei, L. X., R. Goodman, and A. Henderson. 1990. Changes in levels of c-myc and histone H2B following exposure of cells to low-frequency sinusoidal electromagnetic fields: evidence for a window effect. *Bioelectromagnetics* 11: 269-272.

66. Greene, J. J., W. J. Skowronski, J. M. Mullins and R. M. Nardone. 1991. Delineation of electric and magnetic field effects of extremely low frequency electromagnetic radiation on transcription. *Biochem. Biophys. Res. Commun.* 174: 742-749.

67. Litovitz, T. A., C. J. Montrose, R. Goodman, and E. C. Elson. Amplitude windows and transiently augmented transcription from exposure to electromagnetic fields. *Bioelectromagnetics* 11: 297-312.

68. Aarholt, E., E. A. Flinn, and C. W. Smith. 1982. Magnetic fields affect the Lac operon system. *Phys. Med. Biol.* 27(4):606-610.

69. Yuspa, S. H. 1986. Cutaneous chemical carcinogenesis. *J. Am. Acad. Dermatol.* 15:1031-1044.

70. Slaga, T. J. 1989. Cellular and molecular mechanisms involved in multistage skin carcinogenesis. Pp. 1-18 in *Skin Tumor: Experimental and Clinical Aspects*, C. J. Conti, T. J. Slaga, and A. J. P. Klein-Szanto, eds. New York: Raven Press.

71. Weinstein, I. B. 1988. The origins of human cancer: Molecular mechanisms of carcinogenesis and their implications for cancer prevention and treatment. Twenty-seventh G. H. A. Clowes Award Lecture. *Cancer Res.* 48:4135-4143.

72. Grynkiewicz, G., M. Poenie, and R. Y. Tsien. 1985. A new generation of Ca²⁺ indicators with greatly improved fluorescence properties. *J. Biol Chem.* 260:3440-3450.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

73. Adey, W. R. 1990. Electromagnetic fields, cell membrane amplification, and cancer promotion. Pp. 211-249 in *Extremely Low Frequency Electromagnetic Fields: The Question of Cancer*, B. W. Wilson, R. G. Stevens, and L. E. Anderson, eds. Columbus, OH: Battelle Press.

74. Nishizuka, Y. 1984. The role of protein kinase C in cell surface signal transduction and tumor promotion. *Nature* 308:693-698.

75. Bawin, S. M., L. K. Kaczmarek, and W. R. Adey. 1975. Effects of modulated VHF fields on the central nervous system. *Ann. N.Y. Acad. Sci.* 274:74-81.

76. Bawin, S. M., and W. R. Adey. 1976. Sensitivity of calcium binding in cerebral tissue to weak environmental electric fields oscillating at low frequency. *Proc. Natl. Acad. Sci. (USA)* 73:1999-2003.

77. Blackman, C. F., S. G. Genane, L. S. Kinney, W. T. Jones, and D. E. House. 1982. Effects of ELF fields on calcium-ion efflux from brain tissue in vitro. *Radiat. Res.* 92:510-520.

78. Tenforde, T. S. 1980. Thermal aspects of electromagnetic field interactions with bound calcium ions at the nerve cell surface. *J. Theoretical Biol.* 83:517-521.

79. Blackman, C. F. 1990. ELF effects on calcium homeostasis. Pp. 187-208 in *Extremely Low Frequency Magnetic Fields: The Question of Cancer*, B. W. Wilson, R. G. Stevens, and L. E. Anderson, eds. Columbus, OH: Battelle Press.

80. Lin-Liu, S., and W. R. Adey. 1982. Low frequency amplitude modulated microwave fields change calcium efflux rates from synaptosomes. *Bioelectromagnetics* 3:309-322.

81. Dutta, S. K., A. Subramoniam, B. Ghosh, and R. Parshad. 1984. Microwave radiation-induced calcium ion efflux from human neuroblastoma cells in culture. *Bioelectromagnetics* 5:71-78.

82. Dutta, S. K., B. Ghosh, and C. F. Blackman. 1989. Radiofrequency radiation-induced calcium ion-efflux enhancement from human and other neuroblastoma cells in culture. *Bioelectromagnetics* 19(2):197-202.

83. Myers, R. D., and D. H. Ross. 1981, Radiation and brain calcium. A review and critique. *Neurosci. Biobehav. Rev.* 5:503-543.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

84. Pegg, A. E., and H. G. Williams-Ashman. 1981. Pp. 3-42 in *Polyamines in Biology and Medicine*. D. A. Morris and L. J. Marton, eds. New York: Marcel Dekker.

85. Byus, C. V., S. E. Pieper, and W. R. Adey. 1987. The effects of low-energy 60 Hz environmental electromagnetic fields upon the growth-related enzyme ornithine decarboxylase. *Carcinogenesis* 8:1385-1389.

86. Byus, C. V., K. Kartun, S. Pieper, and W. R. Adey. 1988. Increased ornithine decarboxylase activity in cultured cells exposed to low energy modulated microwave fields and phorbol ester tumor promoters. *Cancer Res.* 48:4222-4226.

87. Byus, C. V., R. L. Lundak, R. M. Fletcher, and W. R. Adey. 1984. Alterations in protein kinase activity following exposure of cultured human lymphocytes to modulated microwave fields. *Bioelectromagnetics* 5:341-351.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

8

Human Laboratory and Clinical Evidence of Effects of Electromagnetic Fields

The acute effects of electric and magnetic fields and currents on human volunteers have been extensively studied in the laboratory. End points that have been examined include perception,¹⁻⁷ behavior,⁸⁻¹¹ nervous system function,^{12,13} endocrine function,¹⁴ blood composition,¹⁵ mood,^{16,17} and bone growth.¹⁸⁻²⁴ Except for studies of the perception of contact currents, however, there have been no laboratory studies of the effects of low-frequency (LF) fields or currents on humans. Most of the laboratory evidence involves exposures at extremely low frequency (ELF), with fewer studies being reported at microwave frequencies. The relevance of this research to the possible effects of GWEN fields is therefore uncertain (see section on Frequency Extrapolation). A body of clinical research on the effects of continuous-wave (CW) LF fields on bone stimulation might be of greater relevance to GWEN exposures. This chapter briefly describes the human laboratory and clinical evidence. More detailed reviews are available elsewhere.²⁵⁻²⁹

CUTANEOUS PERCEPTION

Cutaneous perception is one of the best-studied of the effects of electromagnetic fields (EMFs) on humans. At extremely low frequencies, one perceives the presence of electric fields through stimulation of vibration or pressure sensors. Studies of electric-field perception at 50-60 Hz have yielded thresholds of 2-30 kV/m, depending on the subject, posture, and humidity.^{6,7,30} The threshold for cutaneous perception of ELF magnetic fields is unknown, but Graham et al.⁶ report that seated subjects were unable to detect the presence of 60-Hz magnetic fields up to 32 A/m ($= 400 \text{ mG} = 40/\mu\text{T}$).

The detection of LF currents flowing through the skin is reviewed in [Chapter 3](#). Such currents can arise from contacts with ungrounded metal structures near GWEN relay nodes (RNs). Briefly, the sensation of LF currents passed through either a fingertip or a gripped contact is one of warmth, with perception thresholds of 20-300 mA, depending on the subject and type of contact.^{4,5} [Chapter 3](#) also shows that currents arising from contact with vehicles, fences, and other large conducting objects that might be in the vicinity of a GWEN RN are below the threshold of perception for any type of contact. Cutaneous-perception thresholds for incident EMFs at ultra-high frequencies (UHF) comparable to the GWEN transmitters (225-400 MHz) have not been measured, but, because of differences in penetration depth, are expected to be somewhat above the 15-to 44 mW/cm² threshold measured by Justesen for 2,450-MHz fields applied to the forearm.¹¹ That is roughly 10,000 times the UHF power density at the boundary of GWEN RNs.

PHOSPHENES

ELF currents passing through the retina cause the perception of images called phosphenes. The effect is noted only in the ELF band and can be induced by either direct coupling or induction. Thresholds for magnetically-induced phosphenes range from 8 mT at 20 Hz to 20 mT at 60 Hz.^{12, 31, 33} The minimum retinal current density required to produce the effect is less than 1 $\mu\text{A}/\text{cm}^2$ at 20 Hz. Although GWEN LF and UHF fields can induce current densities up to 1 $\mu\text{A}/\text{cm}^2$, GWEN frequencies are too high for phosphenes to be observed.

PACEMAKER INTERFERENCE

Many older models of cardiac pacemakers are susceptible to electromagnetic interference. The threshold of susceptibility depends on the device design and on the frequency and amplitude of the interference. Measurements with a variety of pacemakers available around 1975, for instance, showed interference thresholds as low as 6 V/m for pulsed fields at 450 MHz.³⁴ Newer pacemakers incorporate rejection circuitry that makes them immune to RF interference. Because there is a finite lifetime for pacemakers, most of the older models have been replaced by the new types.

MICROWAVE AUDITORY EFFECT

Humans exposed to rectangular microwave pulses of microsecond duration perceive a sound if the energy flux of the pulse is high enough (about 40 $\mu\text{J}/\text{cm}^2$).¹³ The phenomenon has been shown to arise from an acoustic wave generated in the head by the small but rapid thermoelastic expansion of tissue caused by microwave-energy absorption. The energy flux from GWEN LF or UHF emissions is much too small to elicit this auditory effect.

CIRCADIAN RHYTHMS

Wever³⁵⁻³⁷ used a specially constructed living environment with no time cues to demonstrate that human circadian periods are lengthened by an average of 20 min when the living space is shielded against natural electric and magnetic fields. Introduction of a weak 2.5-V/m, 10-Hz square-wave electric field into the shielded environment was found to shorten circadian periods by 1.3 h relative to the field-free condition. The same square-wave field was also found to weakly entrain free-running circadian rhythms. Sinusoidal electric-field exposures of the same frequency and intensity were not as effective as the square-wave fields. The experiments have not been repeated elsewhere.

BRAIN-EVOKED POTENTIALS

In an extensive series of experiments, Graham et al.¹⁴ repeatedly observed an influence of combined 60-Hz electric and magnetic fields on the late components of the auditory-evoked potential. The effects were observed for both continuous and intermittent exposures and were more pronounced at combined fields of 9 kV/m and 20 μ T than at higher or lower combined-field strengths. Because only late components are affected, Graham and colleagues conclude that the effect is on the cognitive process of stimulus evaluation and not on overall neural conduction velocity.

Alterations in visually-evoked potentials (VEPs) were also reported by Silny³⁸ to occur in response to sinusoidal 50-Hz magnetic fields with flux densities exceeding 5 mT. The change in VEP was characterized by a reversal of polarity and a decrease in the amplitude of the three major evoked potentials. The effects were observed within 3 min after onset of the magnetic-field exposure, and the VEP returned to normal by approximately 30-70 min after termination of the exposure.

HEART RATE

Lowering of heart rate has been often, but not universally, observed in experiments involving human exposure to ELF electric fields, magnetic fields, and injected currents.¹⁴ Table 8-1 lists relevant findings. The studies reporting heart-rate decreases generally characterize them as temporary, noncumulative, and within the range of normal physiologic variation. Extrapolation of the findings to GWEN LF and UHF exposures is difficult. The current density induced by LF exposures at the GWEN site boundary is hundreds of times larger than the ELF current densities associated with changes in heart rate (0.1 μ A/cm²). As the discussion in Chapter 3 points out, however, the sensitivity of muscle and nerve tissue at LF and UHF is likely to be much lower than that at ELF. Although experiments on the effects of exposure to radiofrequency (RF) fields on human heart rate are lacking, evidence from animal studies shows no acute effects of RF irradiation on heart rate in situ²⁶ (also see Chapter 6). Long-term effects of small RF exposure on cardiovascular function remain largely unstudied.

REACTION TIME

The effects of ELF electric fields on human reaction time in response to auditory or visual stimuli have been extensively studied, but the results have been inconsistent.^{14, 27} Increased auditory or visual reaction times have been noted at thresholds ranging from 1-2 V/m in a 3-Hz field⁸ to 10 kV/m in a 50-Hz field.⁴⁵ Decreased visual reaction times were found by Hauf⁴⁶ in 50-Hz fields of 1 and 15 kV/m, but were not verified by later experiments in the same laboratory.^{41, 47}

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 8-1 Effects of ELF Fields and Currents on Human Heart Rate

Exposure	Effect on heart rate	Reference
Occupational exposure in 50-Hz switchyards	Decrease	39
50-Hz electric field, 4 kV/m	Decrease	40
50-Hz injected current, 2 mA	Decrease	41
50-Hz electric field, 10-15 kV/m	Decrease	42
Combined 60-Hz electric and magnetic fields up to 15 kV/m and 300 mG	Decrease	14
50-Hz electric field up to 20 kV/m	Not significant	43, 46
Occupational exposure from high voltage transmission lines	Not significant	44
50-Hz injected current, 0.5 mA	Not significant	10

Gamberale and colleagues⁴⁴ found that a normal increase in reaction time throughout the day was smaller for high-voltage linemen when they were working on energized lines than when they were working on de-energized lines.

In an extensive series of experiments involving exposures to 60-Hz electric fields up to 12 kV/m and 60-Hz magnetic fields up to 30 μ T (300 mG), Graham et al. found no consistent effect of field exposures on reaction times.¹⁴

MOOD AND COGNITIVE FUNCTION

Stollery¹⁶ measured the acute effects of 50-Hz current on mood and verbal reasoning. Subjects were exposed or sham-exposed to a 36-kV/m field for 5.5 h on each of 2 days. Five tests of mood and verbal reasoning were administered throughout the experiment. Exposure-related effects were noted on the second day in stress level, as measured by a mood-adjective checklist, and in response times in tests of syntactic reasoning. In a second series of experiments involving an identical exposure regimen, Stollery measured the effects of 50-Hz current on vigilance and concentration.⁹ No effects on those end points were observed, and the suggestion of exposure-induced stress raised by the earlier study was not confirmed.

In their investigation of high-voltage linemen doing simulated line inspections, Gamberale and colleagues found no effect on wakefulness or stress, as measured by a mood-adjective questionnaire.⁴⁴ They also observed no effects on vigilance, perceptual speed, or short-term memory.

BLOOD COMPOSITION

Human laboratory studies examining field effects on blood composition are limited to the ELF range. Sander and colleagues⁴⁸ exposed volunteers to 50-Hz electric fields of 10-20 kV/m for 6-22 h/d for a week and found no effects of exposure on the concentrations of 33 constituents of blood. Gamberale⁴⁴ found no effects on serum concentrations of seven hormones in linemen performing simulated inspections of a 400-kV transmission line. In an experiment involving 11 subjects, Beischer, et al.¹⁵ found increases in serum triglycerides among subjects exposed to a 1-G (100- μ T) 45-Hz magnetic field. In contrast, Rupilius⁴⁷ found no difference in triglyceride concentrations between a control group and subjects exposed for 3 h to combined 3-G (300- μ T) and 20-kV/m 50-Hz fields.

BONE REPAIR AND GROWTH STIMULATION

Induced or injected currents have been widely used to treat recalcitrant nonunions of fractured bone by stimulating bone growth. Sinusoidal, DC, and pulsed currents have each produced some measure of success.^{24, 49} Brighton et al. have observed enhancements in bone-fracture repair with 60-kHz CW currents.¹⁹⁻²² The threshold electric field in bone tissue^{19, 50} needed to produce the 60-kHz effects ranges from 5 to 20 mV/cm, corresponding to a range of induced current of 2.5-10 μ A/cm². That is comparable with the average LF electric field and current density induced in bone tissue of a person standing at the boundary of a GWEN RN site. There are few data on the frequency dependence of the bone-healing effects reported by Brighton and colleagues. One study of healing of rabbit fibula showed significant field-induced

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

healing at 60 kHz, but not at 10 kHz or 250 kHz with induced currents of the same intensity.²⁰ Limited evidence of the role of duty cycle in the bone-healing effect at 60 kHz shows no clear pattern. Field-induced enhancements in thymidine incorporation in vitro were found at duty cycles as short as 0.25%²² and 0.5%,²³ but disuse osteoporosis in vivo was reversed only by duty cycles greater than about 10%.²¹

Although studies in different biological systems have shown that 60-kHz tissue fields around 2 V/m can evoke measurable biological effects, there is little evidence with which to judge the strength of such effects at GWEN LF frequencies and duty cycles. If GWEN LF fields can induce biological responses in bone tissue, evidence is too scant to conclude whether the responses would be beneficial or harmful.

CONCLUSIONS

As with the animal and in vitro evidence reviewed by the committee, few human laboratory data are directly relevant to GWEN-related exposures. However, the many laboratory studies of human exposure to ELF fields have not identified any deleterious acute effects, although data on chronic exposure are lacking. Studies demonstrating bone growth induced by kilohertz fields are interesting, but there is no evidence of effects of such exposures on healthy tissues.

REFERENCES

1. Tucker, R. D., and O. H. Schmitt. 1978. Tests for human perception of 60 Hz moderate strength magnetic fields. *IEEE Transactions on Biomedical Engineering*, BME-25(6):509-518.
2. Dalziel, C. F., and T. H. Mansfield. 1950. Effect of frequency on perception currents. *AIEE Transactions*, Vol 68, Part II: 1162-1168.
3. Dalziel, C. F. 1954. The threshold of perception currents. *AIEE Transactions on Power Apparatus and Systems*, 73:990-996.
4. Chatterjee, I., D. Wu, and O. P. Gandhi. 1986. Human body impedance and threshold currents for perception and pain for contact hazard analysis in the VLF-MF band. *IEEE Transactions on Biomedical Engineering*, 33(5):486-494.
5. Gandhi, O. P., I. Chatterjee, D. Wu, J. A. D'Andrea, and K. Sakamoto. 1985. Very low frequency study. ASAFSAM-TR-84. Brooks Air Force Base, Texas: USAF School of Aerospace Medicine.
6. Graham, C., M. R. Cook, and H. D. Cohen. 1983. Human perception of 60-Hz electric and magnetic fields. *Bioelectromagnetics Society 5th Annual Meeting (Abstract 0-11)*, Boulder, CO, June 12-17, 1983.
7. Kato, M., S. Ohta, K. Shimizu, Y. Tsuchida, and G. Matsumoto. 1989. Detection threshold of 50 Hz electric fields by human subjects. *Bioelectromagnetics* 10(3):319-327.
8. Konig, H. L. 1974. Behavioral changes in human subjects associated with ELF electric fields. Pp. 81-99 in *ELF and VLF Electromagnetic Field Effects*, M. A. Persinger, ed. New York, NY: Plenum Press.
9. Stollery, B. T. 1987. Effects of 50-Hz electric currents on vigilance and concentration. *Br. J. Ind. Med.* 44:111-118.
10. Stollery, B. T., D. E. Broadbent, W. R. Lee, J. C. Male, W. T. Norris, and J. A. Bonnell. 1989. Psychological functioning during human exposure to 50-Hz electric currents. Report to the Central Electricity Generating Board, Leatherhead, Surrey, U.K.
11. Justesen, D. R. 1988. Microwave and infrared radiations as sensory, motivational, and reinforcing stimuli. Pp. 235-264 in *Electromagnetic*

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Fields and Neurobehavioral Function, M.D. O'Connor and R. H. Lovely, eds. New York, NY: Alan R. Liss, Inc.

12. Carstensen, E. L., A. Buettner, W. L. Genberg, and M. W. Miller. 1985. Sensitivity of the human eye to power frequency electric fields. *IEEE Transactions on Biomedical Engineering*, BME-32:561-565.

13. Lin, J.C. 1990. Auditory perception of pulsed microwave radiation. Pp. 277-318 in *Biological Effects and Medical Applications of Electromagnetic Energy*, O. P. Gandhi, ed. Englewood Cliffs, NJ: Prentice Hall.

14. Graham, C., M. R. Cook, and H. D. Cohen. 1990. Immunological and biochemical effects of 60 Hz electric and magnetic fields in humans. Final report prepared by Midwest Research Institute for the U.S. Department of Energy, Report No. DE90006671. Springfield, VA: National Technical Information Service.

15. Beischer, D., J. Grissett, and R. Mitchell. 1973. Exposure of man to magnetic fields alternating at extremely low frequency. Report NAMRL-1180, Naval Aerospace Medical Research Laboratory, NTIS.

16. Stollery, B. T. 1986. Effects of 50-Hz electric currents on mood and verbal reasoning. *Br. J. Ind. Med.* 43:339-349.

17. Stollery, B. T. 1987. Human exposure to 50-Hz currents. Pp. 445-454 in *Interaction of Biological Systems with Static and ELF Electric and Magnetic Fields*, L. E. Anderson, B. J. Kelman, and R. J. Weigel, eds. Twenty-third Hanford Life Sciences Symposium. Report No. CONF-841041. Richland, WA: Battelle Pacific Northwest Laboratories.

18. Bassett, C. A. L., S. N. Mitchell, and S. R. Gaston. 1985. Pulsing electromagnetic field treatment in ununited fractures and failed arthrodeses. *J. Am. Med. Assoc.* 247(5):623-628.

19. Brighton, C. T., and S. R. Pollack. 1985. Treatment of recalcitrant nonunion with a capacitively coupled electrical field. *J. Bone and Joint Sur.* 67-A(4):577-585.

20. Brighton, C. T., W. J. Hozack, M.D. Brager, R. E. Windsor, S. R. Pollack, E. J. Vreslovic, and J. E. Kotwick. 1985. Fracture healing in the rabbit fibula when subjected to various capacitively coupled electrical fields. *J. Orthop. Res.* 3:331-340.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

21. Brighton, C. T., G. T. Tadduni, and S. R. Pollack. 1985. Treatment of sciatic denervation disuse osteoporosis in the rat tibia with capacitively coupled electrical stimulation. *J. Bone and Joint Sur.* 67-A:1022-1028.
22. Brighton, C. T., L. Jensen, S. R. Pollack, B. S. Tolin, and C. C. Clark. 1989. Proliferative and synthetic response of bovine growth plate chondrocytes to various capacitively coupled electrical fields. *J. Orthop. Res.* 7:759-765.
23. Kuhlman, J. R., and C. T. Brighton. 1991. Stimulation of bovine growth plate chondrocytes with pulsed capacitively coupled electric fields. Pp. 153-158 in *Electromagnetics in Biology and Medicine*, C. T. Brighton and S. R. Pollack, eds. San Francisco, CA: San Francisco Press.
24. Swicord, M. L. 1989. Overview of the literature on electrical bone repair and growth stimulation. HHS Publ. No. (FDA) 90-8277. Washington, D.C.: U.S. Department of Health and Human Services.
25. Michaelson, S. M. 1979. Human responses to power-frequency exposures. Pp. 1-20 in *Biological Effects of Extremely Low Frequency Electromagnetic Fields*, R. D. Phillips, M. F. Gillis, W. T. Kaune, and D. D. Mahlum, eds. U.S. Department of Energy, NTIS CONF-781016.
26. National Council on Radiation Protection and Measurements (NCRP). 1986. *Biological Effects and Exposure Criteria for Radiofrequency Electromagnetic Fields*. NCRP Report No. 86. Bethesda, MD: National Council on Radiation Protection Measurements.
27. Stollery, B. T. 1992. Electrical fields. Pp. 211-236 in *Handbook of Human Performance*, D. Jones and A. Smith, eds. London, Academic Press.
28. Environmental Protection Agency. 1983. Biological effects of radiofrequency radiation. D. F. Cahill, and J. A. Elder, eds. EPA 600/8-83-026F, PB85 120848. NTIS PB85 120848. Springfield, VA: National Technical Information Service.
29. World Health Organization. 1987. *Environmental Health Criteria 69: Magnetic Fields*. Geneva, Switzerland: WHO.
30. Deno, D. W., and L. E. Zaffanella. 1982. Field effects of overhead transmission lines and stations. Chapter 8 in *Transmission Line Reference Book*, Publication Number EL-2500. Palo Alto, CA: Electric Power Research Institute .

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

31. Lovsund, P., P. A. Oberg, and S. E.G. Nilsson. 1979. Influence on vision of extremely low frequency electromagnetic fields, *Acta Ophthalmol.* 57:812-821.
32. Lovsund, P., P. A. Oberg, S. E.G. Nilsson, and T. Reuter. 1980. Magnetophosphenes: a quantitative analysis of thresholds. *Med. Biol. Eng. Comput.* 18:326-334.
33. Lovsund, P., P. A. Oberg, and S. E. G. Nilsson. 1980. Magneto-and electrophosphenes: a comparative study. *Med. Biol. Eng. Comput.* 18:758-764.
34. Mitchell, J. C., and W. D. Hurt. 1976. The biological significance of radiofrequency radiation emission on cardiac pacemaker performance. Technical Report 76-4, U.S. Air Force School of Aerospace Medicine.
35. Wever, R. 1973. Human circadian rhythms under the influence of weak electric fields and the different aspects of these studies. *Intl. J. Biometeorology*, 17:227-232.
36. Wever, R. 1974. ELF effects on human circadian rhythms. Pp. 101-144 in *ELF and VLF Electromagnetic Field Effects*, M. Persinger, ed. New York, NY: Plenum Press.
37. Wever, R. 1977. Effects of low-level, low frequency fields on human circadian rhythms. *Neuroscience Research Progress Bulletin*, 15 (1):39-45.
38. Silny, J. 1986. The influence threshold of the time-varying magnetic field in the human organism. Pp. 105-112 in *Biological Effects of Static and Extremely Low Frequency Magnetic Fields*. Munich, Germany: MMV Medizin Verlag.
39. Sazanova, T. E. 1976. Physiological effects of work in the vicinity of 400-500 kV outdoor installations. Publication of the Institute of Labor Protection of the All-Union Central Council of Trade Unions, No. 46, 34-49, Moscow.
40. Waibil, R. 1975. Objective proof of the influence of 50 Hz electric fields on man. Third International Colloquium of ISSA for Prevention of Occupational Risks Due to Electricity, Marbella, Spain. Cited in E. L. Carstensen, *Biological Effects of Transmission Line Fields*. New York, NY: Elsevier.

41. Eisemann, U. B. 1976. Investigation on the long-term effect of small alternating current of 50 Hz on the human being. M.D. Dissertation, Albert-Ludwigs University, Germany.

42. Kalyada, T. V., T. I. Krivova, V. N. Nikitina, Y. A. Morozov, N. V. Revnova, and G. G. Shaposhnik. 1985. Biological action of a 50 Hz electric field on the human body. Pp. 317-332 in Proceedings of the U.S.-USSR Workshop in Physical Factors—Microwave and Low Frequency Fields, National Institute of Environmental Health Sciences, May 1985.

43. Hauf, R. 1974. Effect of 50 Hz alternating fields on man. Sonderdruck 26:318-320.

44. Gamberale, F., B. A. Olson, P. Eneroth, T. Lindh, and A. Wennberg. 1989. Acute effects of ELF electromagnetic fields: a field study of linemen working with 400-kV power lines. Br. J. Indust. Med. 46:729-737.

45. Teresiak, Z., and M. Szuba. 1989. Harmful influence of electric field on human organism and methods of protection in high current engineering objects. Sixth International Symposium on High Voltage Engineering, Mississippi State University, New Orleans, LA, August 28-September 1, 1989.

46. Hauf, R. 1976. Influence of alternating electric and magnetic fields on human beings. Revue Generale de l'Electricite, Vol. 85, Special Issue on Research on the Biological Effects of Electric and Magnetic Fields. Pp. 31-49.

47. Rupilius, J.P. 1976. Research on the action of electric and magnetic 50 Hz alternating field on man. M.D. Thesis, Albert-Ludwigs University, Germany: J. Krause Publisher, Freiburg am Breisgau.

48. Sander, R., J. Brinkmann, and B. Kuhne. 1982. Laboratory studies on animals and human beings exposed to 50-Hz electric and magnetic fields. Paper 36-01, International Conference on Large High Voltage Electric Systems, 1-9 September, 1981, Paris.

49. Brighton, C. T. 1991. Advanced clinical applications of electromagnetic field effects: bone and cartilage. Pp. 293-308 in Electromagnetics in Biology and Medicine, C. T. Brighton and S. R. Pollack, eds. San Francisco, CA: San Francisco Press.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

50. Pollack, S. R. 1991. Dosimetry in electromagnetic field stimulation. Report prepared for the National Institutes of Health. Department of Bioengineering: University of Pennsylvania.

9

Epidemiological Research Relevant to Identification of Health Hazards Associated with GWEN Fields

An extensive, but fragmented, literature deals with the potential health effects of extremely-low-frequency (ELF) magnetic fields (50-60 Hz) encountered in occupational and residential settings. Investigators have been following at least three different avenues of inquiry: occupational hazards, general environmental hazards, and cancer etiology. Research in all those fields has been hampered by technical difficulties in measuring actual exposure to ELF fields. Because of the difficulty in measuring exposure to ELF fields, most investigators have had to study ecological associations of magnetic fields and disease rather than exposure of individuals with and without disease to magnetic fields (case-comparison studies) or disease incidence among individuals with smaller and larger exposures (cohort studies). Another set of measurement problems, those related to morbidity, has led to serious concern about the influence of ascertainment bias on the results of the few studies that looked at diseases other than cancer. Most of those studies have tended to concentrate on cancer end points in population-based registries and control subjects. The entire health-effects literature was reviewed by Coleman and Beral¹ and Gordon et al.² and expanded and updated by Tenforde,³ Theriault,⁴ and Poole and Trichopoulos.⁵ This committee endorses the general conclusions of the three 1991 reports, and this chapter summarizes the conclusions and highlights the results of key investigations. It does not provide a comprehensive literature review.

STUDIES OF GENERAL ENVIRONMENTAL EXPOSURE

Most of these studies measured residential exposure in one of two ways: by direct measurement of magnetic fields in houses or multiple-family dwellings or by use of wire codes, with electric-current configurations (high-current or low-current) as indirect measurements of magnetic-field exposure.

The archetypal residential-exposure study was published by Wertheimer and Leeper.⁶ It used a case-control design and measurements of wiring configuration to consider 344 cancer deaths of newborn to 18-yr-old children recorded in Denver, Colorado, between 1950 and 1973. The dead children were compared with 344 living children of the same ages. The configuration of the wiring around the home of each dead or matched child was coded "high current" or "low current" according to the types of neighboring electrical distribution lines. For example, a home less than 40 m from large-gauge primary wires was coded as "high current". A major flaw in the study was that wiring configurations of households were not blindly classified.

The address recorded as the place of death was the residence of each dead (case) and matched living (control) child 2 yr before the diagnosis of each dead child. A total of 491 case and 472 control addresses were coded. The homes of 182 of 309 dead children were classified as having high-current configurations, compared with 103 of 369 homes of living children—a relative frequency of 2.11. The relative frequency proved to be rather consistent and significantly different from unity for the results of a number of analyses of subsets of the data, e.g., for birth and death addresses of case and control children 0-5 yr old, for those 6-18 yr old, for children of all ages who had leukemia and their controls, and for children who had nervous system tumors and their controls.

That startling result led others to attempt to replicate the findings. One of the first groups, Fulton et al.⁷ in Rhode Island, used 119 cases, defined as leukemia patients with onset before age 20 and year of onset between 1964 and 1978. Controls were matched, two to one, by birth year. An attempt was made to use the same scheme as Wertheimer and Leeper⁶ to code the electrical distribution-line configuration for the residences of all cases and the birth residences of all controls. Fulton et al. coded 198 case and 225 control residences and found no association between leukemia risk and high-current wiring configuration. Wertheimer and Leeper⁸ later reevaluated the data of Fulton et al. and suggested that the coding scheme was not exactly comparable with their own and that electrical distribution systems are different in Rhode Island and Colorado. Moreover, Wertheimer and Leeper asserted that, if the analysis was restricted to data on children 8 yr old and older, marginally significant increases in risk could be seen.

Shortly thereafter, a Swedish study by Tomenius⁹ made more direct measure-of exposure and coding wiring configurations. Tomenius studied 716 incident cases among people 0-18 yr old, born in Stockholm county, and diagnosed there during 1958-1973. Each case was matched to a control by age, sex, and church district of birth. Birth and diagnosis addresses were determined for cases and controls (for a control, the address when the matching case was diagnosed). A total of 1,129 case and 969 control dwellings were visited by the investigators. "Visible electrical structures" were recorded at each address, and magnetic field strengths were measured. A statistically significant odds ratio (3.7; $P < 0.05$) for nervous system tumors was observed when dwellings with measured field strengths greater than $0.3\mu\text{T}$ were compared with those with field strengths less than $0.3\mu\text{T}$. However, a decreased leukemia risk of 0.3 bordered on significance. There also appeared to be a generally increased risk of cancer (odds ratio, 2.1) for dwellings within 150 m of 200-kV transmission lines. Because many of the dwellings near 200-kV lines were also above $0.3\mu\text{T}$, those results are not wholly independent.

Savitz et al.^{10, 11} conducted a study on childhood cancer in the Denver, CO, area in which the residence at the time of diagnosis was measured for the strength of the electric and magnetic fields and the current configuration of the surrounding electrical

distribution wires was coded using the method of Wertheimer and Leeper.⁶ Whenever possible, prior residences were also measured and coded. In contrast with the earlier study,⁶ the assessment and classification of homes were blind. The Savitz et al.¹¹ study included 356 children aged 0-14 yr who were residents of the 1970 Denver Standard Metropolitan Statistical Area and had a cancer diagnosis during 1976-1983. Controls, selected with random-digit dialing, were similar to the cancer children in age, sex, and geographic area of residence. Residential histories were obtained during interviews. Odds ratios of between 1.17 (appliances on) and 1.49 (appliances off) were obtained for homes with measured 60-Hz magnetic fields of greater than 0.25 μ T. Associations with wire configurations were much higher. The odds ratio was 2.2 for "very high" current configuration, compared with buried cable, when the residence at the time of diagnosis was considered and rose to 5.22 when the same comparison was made for the residences occupied by cases 2 yr before diagnosis. The last result was statistically significant.

London et al.¹² conducted a case-control study of electric and magnetic fields and childhood cancer in Los Angeles. The basic population consisted of 232 cancer children, drawn from the Los Angeles County Cancer Surveillance tumor registry, and 232 controls, some drawn from neighbors of the cancer children and some from random-digit dialing surveys. Attempts were made to match cases and controls by age, sex, and ethnicity, but this was not always possible; in the case of random-digit dialing, no match could be obtained in all cases. When a match could not be obtained readily, matching criteria were relaxed first on ethnicity, then on sex, and last on age. Efforts were made to obtain spot and 24-h readings of electric and magnetic fields in the residences of cases and controls and to characterize the configuration of distribution wiring around the residences with the coding scheme of Wertheimer and Leeper.¹³ Odds-ratio analyses of 24-h magnetic fields (162 cases and 143 controls) and spot measurements of magnetic fields (140 cases and 109 controls) showed no association of leukemia risk with increased magnetic or electric field strength. The one significant odds ratio (2.15) was observed in a comparison of underground and very-low-current wiring-configuration residences (31 cases and 38 controls) with very-high-current configuration residences (42 cases and 24 controls) on the Wertheimer-Leeper coding scheme. The authors suggested that the results support an association between leukemia risk and wiring codes, but not with either electric or magnetic fields. Their discussion reflects the uncertainty of whether lack of association with magnetic fields is a result of deficiencies in field measurements (measuring the wrong thing) or the observed association of risk with wiring codes is the product of some unidentified confounder.

Wertheimer and Leeper¹³ also conducted the first of a relatively small series of studies on residential fields and adult cancer. They used a case-control design and four series of cases. The first two series of cases were all people who died of cancer in 1967-1975 in the towns of Longmont and Boulder, Colorado (plus a few cancer survivors living in these towns found through the Colorado cancer registry). The other

two series, in Denver and the Denver suburbs, included both dead and surviving cancer patients. A control matched by age, sex, and year of death was selected for each dead patient; and a living neighborhood control for each survivor. Analysis was restricted to subjects whose history could be traced for at least 4 yr before diagnosis. It included 194 cases and controls in Longmont, 321 in Boulder, 255 in Denver suburbs, and 409 in Denver. The electric-wiring configuration was coded for the residence in which the subject had spent most of the 3-10 yr before diagnosis. Control addresses for Longmont and Boulder were chosen in the same manner for the same 3-10 yr. The addresses chosen by random-digit dialing to recruit the Denver controls were used for measurement purposes. As in the authors' earlier study, there was no blinded coding and classification of homes. Comparison of very-high-current configuration with low-current configuration in all four series showed odds ratios of 1.5-2. The analysis suggested that at least three of the comparisons were statistically significant (the authors used a "C" statistic, based on a sign test, which is less powerful than the more conventional chi-squared test).

Another study of acute nonlymphocytic leukemia was conducted by Severson et al.¹⁴ in western Washington. Wiring configurations and direct measurements (short-term and 24 h) of 60-Hz electric and magnetic fields were recorded. The case series consisted of people aged 20-79 yr old, diagnosed with acute nonlymphocytic leukemia during 1981-1984 in King, Pierce, or Snohomish County. The control group, matched by sex and age, was selected from the same three-county area with random-digit dialing. Approximately one control per case was selected. Subjects were interviewed for a complete residential history. All addresses occupied by cases during the 15 yr before diagnosis and on a randomly assigned reference date by control were coded for wiring configuration, and the 60-Hz electric and magnetic fields were measured for the current residence at the time the interview occurred (or for the last residence of dead subjects). A total of 114 cases and 133 controls were interviewed. An analysis of the data showed no association of leukemia risk with wire code (odds ratios, 0.60-1.36). A similar analysis based on measured fields also showed no significant association with leukemia risk. The latter result was criticized by Wertheimer and Leeper¹⁵ on the grounds that measurements were made during only 39% of case interviews and 70% of control interviews. They also asserted that the lack of association with wiring configuration was due to a high degree of nondifferential misclassification of exposure.

McDowall¹⁶ reported the results of a population-based cohort study in the United Kingdom. The cohort was identified with maps of East Anglia and data from the 1971 British Census to enumerate the population living in the vicinity of "electrical installations." A total of 2,839 dwellings inhabited by 7,920 people were identified, and this cohort was traced through 1983. A total of 7,631 people were successfully traced, of whom 814 had died during 1971-1983. The overall standard mortality ratio (SMR) was 89, which reflected a low death rate from circulatory disease. Only one cancer SMR—for lung cancer in women—was found to be significantly increased. The observation that leukemia in women and leukemia in both sexes had above-average

SMRs among people living within 15 m of an electrical installation was interesting, but the results were not statistically significant.

In another United Kingdom study, Youngson et al.¹⁷ estimated exposure to the field strengths due to maximum load currents from overhead power lines. They correlated the estimates with measurements of magnetic fields made on the premises of home addresses of a sample of people in their study living close to power lines. For analysis, they used estimates of power flowing through the lines during the 5 yr before diagnosis of their cases. Of 1,511 people 15 yr old or older who were diagnosed with hematological malignancy between January 1, 1983, and December 31, 1985, 1,491 resided within the North West Regional Hospital Authority boundaries of England and Wales. Matching controls (by sex, age, and year of diagnosis) were randomly selected from computer lists of all inpatient discharges in the same area. Matched-pair analysis, with conditional logistic regression, showed no statistically significant associations of disease with either magnetic-field exposures or distance of homes from power lines. However, as in many other studies, the odds ratios for subgroups of cases (versus their controls) living within 30 m of overhead transmission lines verge on statistical significance. Of more importance for the present report is the finding of a nonsignificant trend with distance from the power lines, which suggested an increased risk—a relative risk of about 1.3—associated with larger magnetic fields or with proximity to overhead power lines. However, on the basis of both direct and indirect measurements of exposure, the investigators stated that their study provided no consistent evidence that distance from power lines correlates accurately with direct measurements of magnetic-field exposure.

Those selected studies have been described in enough detail to emphasize the overwhelming dilemma faced by investigators who want to answer the question, "Is any health effect associated with exposure to power-frequency magnetic fields?" Conceptually, direct measurements of electric and magnetic fields in homes must be better assays of the exposure of people living in those homes than are territorial distribution wire codes. Furthermore, 24-h measurements must be better than spot measurements of direct exposure. On balance, the more direct and the better the measurement of exposure to magnetic fields, the smaller the likelihood of detecting increased risks of leukemia in either children or adults.

Studies that included all cancer deaths, rather than just leukemia in adults, have had to deal with the additional bias of the types of death involved, because leukemia incidence is overwhelmed by the incidences of the most common and most lethal cancers, such as those of the lung and colon. In this context, the results of the relationship (or lack of relationship) of cancer deaths with measured field exposures are even more difficult to interpret.

In contrast, classification of residential addresses according to wire-code configurations—the least direct and most tenuous measurement of a person's field

exposure—suggests that children with leukemia are more likely (perhaps twice as likely) to be found in homes coded as having high versus low current configurations.

There are at least two possible interpretations of these dichotomous results. Either wire code is a better indicator of long-term exposure to magnetic fields than is more direct measurement of those fields, or wire code is a surrogate of some household exposure other than exposure to the ambient 60-Hz magnetic field. The wire code could also be a surrogate of demographic and lifestyle characteristics of residents in the coded households that are associated with higher risks of leukemia. The former might well be a function of the age of the buildings.

This report addresses a much more limited question: "Do the studies provide any evidence that people living close to GWEN sites will have an increased risk of developing leukemia, other cancers, and other diseases?" The reported studies do not provide any evidence that any measurable risk is likely to be due to GWEN-related exposure.

OCCUPATIONAL STUDIES

Occupational studies have used job codes and titles as indicators of worksite exposure to magnetic fields. The general conclusions of a current comprehensive review of studies of occupational exposures by the National Institute for Occupational Safety and Health¹⁸ are endorsed by this committee, including its lack of confidence in occupational codes as adequate indicators of exposure. Because of that lack of confidence and because the results of occupational studies are less relevant than residential studies to the general environmental setting that is central to the present report, those studies are described here in much less detail.

Milham^{19, 20} using proportionate mortality ratio (PMR) inventories, showed excess leukemia associated with the occupation "electrical worker" and excess brain cancer with the occupation "electrician." Case-control studies have been conducted of brain cancer²¹⁻²³ and leukemia;^{24, 25} they typically report increased odds ratios—1.5 to 3—for such job titles as "electrician" or "welder." Cohort studies have also been carried out on workers in the electrical industry;²⁶⁻²⁸ these studies have led to reports of excess cancers in many anatomic sites, from melanoma to male breast cancer.

The epidemiological results have generated a number of useful hypotheses; e.g., if ELF magnetic fields interfere with melatonin metabolism, excess breast cancer²⁹ might be expected. However, the literature is of limited value for risk analysis. First, there have been inconsistent increases in cancers of different sites, even though, as in the residential studies, brain cancer and leukemia appear to be possible candidates. More important, the excesses detected are related to occupation, not to exposure. That is, electricians might be at excess risk for brain cancer, but this does not implicate ELF

magnetic-field exposure as the causative factor. However, the hypotheses raised by the studies should be considered further, because some measurements of worker exposure suggest increased exposure to ELF magnetic fields, but the measurements have not yet been made in the context of epidemiological research and cannot be related to specific disease risks.

EPIDEMIOLOGIC STUDIES OF HEALTH EFFECTS OF MICROWAVE EXPOSURE

Another relevant data source, epidemiologic studies of populations exposed to radar and microwave, is rather limited. Two populations, U.S. naval personnel exposed to radar and American embassy personnel in Moscow, dominate the literature. The first group of studies, reviewed by Tenforde and Budinger,³⁰ consist of cohort studies of high-exposure and low-exposure groups. One such study³¹ followed a high-exposure group of 20,109 Korean War veterans involved in radar repair, compared with a similar group of 20,781 veterans involved in radar operation and considered to have received low exposure. No excesses of either cancer or cataracts (hypothesized to be related to occupational exposure to microwaves) were seen in the high-exposure cohort. Another study of occupational exposures to radar showed a similar lack of effect.³²

Szmigielski et al.³³ reported a significant elevation of cancer incidence rates among Polish military personnel exposed to radar relative to nonexposed personnel. An elevated cancer rate was observed both for all cancers combined and for specific cancers of the lymphatic and hematopoietic system. Cancer rates were reported as incidence rate per 100,000 per year for the period 1971-1980, and no data were presented on the actual numbers of cases in the exposed and nonexposed groups. The design of this epidemiological study and the methods used for data analysis were not adequately described to permit a judgment on the reliability of the results.

The Moscow embassy studies involved followup of 1,827 persons who had served in the embassy from 1953 to 1976, during which period the embassy was exposed to radiofrequency (RF) power densities of as much as 15 $\mu\text{W}/\text{cm}^2$ for 18 h/d. The morbidity and mortality experience of this group was compared with that of a group of 2,561 persons who had served as employees at eight unirradiated East European embassies or consulates during the same period. A variety of health end points were compared, but no significant exposure-related effects were observed.³⁴

Another negative study of cancer associated with microwave radiation was conducted by Selvin et al.³⁵ It examined whether cases of bone, brain, and lymphatic cancer in persons less than 20 yr old in San Francisco from 1973 to 1986 might be clustered around a large microwave-transmission tower. The study was based on 30 cases of bone cancer, 27 cases of brain cancer, and 26 cases of lymphatic cancer. Clustering was shown for lymphatic cancer, but not centered near the tower. No

significant clustering was shown for the other two cancers. Thus, the study was negative.

Because the studies in question are of rather low power and lack precise measurements of exposure and because radar and microwave radiation have frequencies very different from those of GWEN fields, the findings do not offer much insight as to whether the GWEN system poses any risks to public health.

RADIO BROADCAST STATIONS

One study that has direct relevance to RF exposures near GWEN frequencies was performed in the workforce of the Meadowlands Sports Complex (MSC) in New Jersey.³⁶ This study was prompted by a cancer cluster that occurred on the New York Giants football team between 1980 and 1987. Because there were three AM broadcasting stations located at the MSC and five more within 2-3 miles, measurements of electric field strengths were made at MSC; it was found that they were in the top 0.1% of field strengths measured in urban areas in the United States. The root summed square for all AM signals ranged from 0.013-9.63 V/m.

The study involved 147 cancer cases that occurred in the Meadowlands workforce during 1978-1987. The expected numbers of deaths from various types of cancer were calculated with New Jersey mortality data. Two significant proportionate cancer incidence ratios (PCIRs), based on one case each, were observed for male breast cancer and nasopharyngeal cancer. Significantly increased PCIRs were not observed for either brain cancer or leukemia. Although this result does not rule out odds ratios of 2 or less for these cancers, the study had sufficient power to detect odds ratios of 5 or more with a probability of about 90%.

A proportionate-mortality analysis based on 225 deaths and 65 cancer deaths was performed in the study. The overall proportionate mortality ratio (PMR) for all cancers was almost exactly equal to 1. Because all cancer PMRs as small as 1.5 would likely have been detected, that result strongly suggests that no large general cancer increase was present. The generally negative findings of the study support the view that exposure to high AM radio-broadcast field strengths does not result in large excess cancer risks.

Another study on cancer incidence in relation to RF exposure from radio and television broadcast stations was conducted in the state of Hawaii using data from the Hawaii Tumor Registry.³⁷ Cancer incidence rates were examined for nine census tracts with broadcast towers (in Honolulu and Waikiki) and for two tracts without towers (more centrally located on the island of Oahu). The standardized incidence ratio for cancer was significantly elevated for both males and females living in tracts with broadcast towers relative to the general population of Hawaii. No significant elevation

in cancer incidence was observed for the two populations living in census tracts without towers. This study was very weak due to the inability to simultaneously adjust the data for race, age and sex (due to the small sample sizes), or to account for differences in expected cancer rates in rural areas (without towers) relative to urban areas (with towers). In addition, no exposure data were presented and the RF exposure consists of a mixture of AM and FM signals. As a consequence, the results of this study cannot be regarded as conclusive.

AMATEUR RADIO OPERATORS

Milham³⁸⁻⁴⁰ reported that amateur radio operators in the states of Washington and California have a significantly elevated risk of mortality from acute myeloid leukemia, multiple myeloma and other neoplasms of the lymphoid tissues. Mortality data were obtained from the death certificates of members of the American Radio Relay League whose deaths were reported in the League's magazine. Comparison data were based on the 1976 report of U.S. age-specific white male death frequencies. The overall death rate of the amateur radio operators was significantly lower than that of the general population for all causes of mortality and for all classes of cancer combined. Because Milham's study provided no estimate of exposure to either RF or ELF fields among the amateur radio operators, the results cannot be considered directly relevant to GWEN fields.

CONCLUSIONS

Reports of occupational studies have generated useful hypotheses, but so far none has been helpful in specifically testing hypotheses or quantifying risk. It is even harder to conclude how much has been learned about hypothesized risks from the studies of residential exposure. Certainly, the two Wertheimer and Leeper studies^{6, 13} suggest that a consistent hazard is associated with exposure to "high-current" configuration wiring codes. However, it is not clear that high-current configuration wiring codes are closely associated with magnetic-field exposure. Poole and Trichopoulos,⁵ citing Kaune⁴¹ and Barnes,⁴² point out that the observed correlation coefficient between magnetic field and wiring code rarely exceeds 0.6 and is more often in the range of 0.4-0.5. It could be argued that nondifferential misclassification, which reduces the apparent strength of associations between diseases and their causes, is masking a true causal link between magnetic-field strength and leukemia.⁵ Results of studies that have measured both field strength and wiring codes^{10, 14} do not support the idea that magnetic fields are a clear-cut cause of leukemia, inasmuch as associations of wiring codes with disease are stronger than those for 60-Hz magnetic fields. Youngson et al.¹⁷ strengthened those observations and emphasized that any association between distance from power lines and hematological cancer is likely to be very small and that

even large observational epidemiological studies will probably not prove that it does or does not exist.

That does not mean that further efforts should not be made to learn why high-current electrical wiring configuration might be an indicator of increased disease risk. Poole and Trichopoulos⁵ have suggested that poor households are harder to recruit as controls when random-digit dialing is used for this purpose. Those households might be more often associated with high-current configuration (inner-city) wiring. According to their hypothesis, the increased odds ratio would indicate a deficit of high-current configuration wiring among the controls, not an excess among the cases.

A parallel argument might be made to explain the results of the Wertheimer and Leeper⁶ study. Cases were selected from death certificates, and controls were selected from birth certificates. If poor children with leukemia are more likely to die than the more affluent, an excess of high-current wiring configuration associated with death certificates could reflect that difference. A dramatic change in survival of leukemia victims was in fact occurring at the time of their study, in which 48% of the childhood cancers were leukemias. Death certificates used in the study were collected in 1950-1973, a period when death rates for childhood-leukemia death rates fell by about 33%.⁴³ If richer children got the best treatment first, the hypothesis would be strengthened. The investigators in both studies (Savitz;¹⁰ Wertheimer and Leeper⁶) considered and dismissed confounding by socioeconomic factors as a possible explanation of their findings, but the possibility of confounding factors probably deserves further consideration.

The only two residential studies that showed systematic internal consistency (Wertheimer and Leeper)^{6,13} were the most likely to be influenced by procedural bias. Coding of wiring configurations was not blind to the identity of case and control subjects, and the choice of control groups was rather arbitrary (two control definitions were used in the first study, three in the second).

With respect to its relevance to the potential health effects of GWEN fields, the literature provides no consistent or even suggestive evidence that household measurements of magnetic-field exposures are associated with increases in rates of hematological or other cancers in children or adults. Whether distance of residences from power lines is a surrogate measure of exposure to other cancer-causing agents is an unanswered question of public-health importance. But, the answer is unlikely to provide any new information that could be used to assess the potential risk associated with GWEN electromagnetic fields.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

REFERENCES

1. Coleman, M., and V. Beral. 1988. A review of epidemiological studies of the health effects of living near or working with electricity generation and transmission equipment. *Int. J. Epid.* 17(1):1-13.
2. Gordon, I., M. Motika, and T. Nolan. 1990. Epidemiological studies of cancer and powerline frequency electromagnetic fields: a meta analysis. Report 242. The University of Melbourne Statistical Consulting Center, Melbourne, Australia.
3. Tenforde, T. S. 1992. Biological interactions and potential health effects of extremely low-frequency magnetic fields. *Ann. Rev. Public Health* 13:173-196.
4. Theriault, G. 1991. Health effects of electromagnetic radiation on workers: Epidemiological studies. Pp. 93-124 in *Proceedings of the Scientific Workshop on the Health Effects of Electric and Magnetic Fields in Workers*. P. J. Bierbaum and J. M. Peters, eds. Publ. No. 91-111. Cincinnati, OH: U.S. Department of Health and Human Services.
5. Poole, C., and D. Trichopoulos. 1991. Perspective: Extremely low-frequency electric and magnetic fields and cancer. *Cancer Causes and Control* 2:267-276.
6. Wertheimer, N., and E. Leeper. 1979. Electrical wiring configurations and childhood cancer. *Am. J. Epidemiol.* 109:273-284.
7. Fulton, J.P., S. Cobb, L. Preble, L. Leone, and E. Forman. 1980. Electrical wiring configuration and childhood leukemia in Rhode Island. *Am. J. Epidemiol.* 111:292-296.
8. Wertheimer, N., and E. Leeper. 1980. Electrical wiring configurations and childhood leukemia in Rhode Island. *Am. J. Epidemiol.* 111:461-462.
9. Tomenius, L. 1986. 50-Hz electromagnetic environment and the incidence of childhood tumors in Stockholm County. *Bioelectromagnetics* 7:191-207.
10. Savitz, D. A. 1987. Case-control study of childhood cancer and residential exposure to electric and magnetic fields. Contractor's Final Report, New York State Power Lines Project, Contract 218217, March 30, 1987.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

11. Savitz, D. A., H. Wachtel, F. A. Barnes, E. M. John, and J. G. Tvrdik. 1988. Case-control study of childhood cancer and exposure to 60-Hz magnetic fields. *Am. J. Epidemiol.* 128(1):21-38.

12. London, S. J., D. C. Thomas, J. D. Bowman, E. Sobel, T.-C. Cheng, and J. M. Peters. 1991. Exposure to residential electric and magnetic fields and risk of childhood leukemia. *Am. J. Epidemiol.* 134:923-937.

13. Wertheimer, N., and E. Leeper. 1982. Adult cancer related to electrical wires near the home. *Int. J. Epidemiol.* 11:345-355.

14. Severson, R. K., et al. 1988. Acute nonlymphocyte leukemia and residential exposure to power frequency magnetic fields. *Am. J. Epidemiol.* 128(1):10-20.

15. Wertheimer, N., and E. Leeper. 1989. Fetal loss associated with two seasonal sources of electromagnetic field exposure. *Am. J. Epidemiol.* 129(1):220-224.

16. McDowall, M. N. 1986. Mortality of persons resident in the vicinity of electricity transmission facilities. *Br. J. Cancer* 53:271-279.

17. Youngson, J. H. A. M., A. D. Clayden, A. Myers, and R. A. Cartwright. 1991. A case/control study of adult hematological malignancies in relation to overhead powerlines. *Br. J. Cancer* 63:977-985.

18. National Institute for Occupational Safety and Health. 1991. In Proceedings of the Scientific Workshop on the Health Effects of Electric and Magnetic Fields on Workers, P. J. Bierbaum, and J. M. Peters, eds. Publication No. 91-111. Ohio: U.S. Department of Health and Human Services.

19. Milham, S., Jr. 1982. Mortality from leukemia in workers exposed to electrical and magnetic fields. (Letter to the Editor). *N. Engl. J. Med.* 307(4):249.

20. Milham, S., Jr. 1985. Mortality in workers exposed to electromagnetic fields. *Environ. Health Perspec.* 62:297-300.

21. Lin, R. S., P. C. Dischinger, J. Conde, and K. P. Farrell. 1985. Occupational exposure to electromagnetic fields and the occurrence of brain tumors. *J. Occup. Med.* 27:413-419.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

22. Speers, M. A., J. G. Dobbins, and V. S. Miller. 1988. Occupational exposures and brain cancer mortality: a preliminary study of East Texas residents. *Am. J. Ind. Med.* 13:629-638.

23. Savitz, D. A., and L. Feingold. 1989. Association of childhood cancer with residential traffic density. *Scan. J. Work Environ. Health* 15:360-3.

24. Stern, F. B., R. A. Waxweiler, J. J. Beaumont, S. T. Lee, R. A. Rinsky, R. D. Zumwalde, W. E. Halperin, P. J. Bierbaum, P. J. Landrigan, and W. E. Murray, Jr. 1986. A case-control study of leukemia at a naval nuclear shipyard. *Am. J. Epidemiol.* 123:980-992.

25. Flodin, U., M. Fredriksson, Om Axelsson, B. Persson, and L. Hardell. 1986. Background radiation, electrical work, and some other exposures associated with acute myeloid leukemia in a case-referent study. *Arch. Environ. Health* 41:77-84.

26. Olin, R., D. Vägerö, and A. Ahlbom. 1985. Mortality experience of electrical engineers. *Br. J. Ind. Med.* 42:211-2.

27. Matanoski, G. M., E. A. Elliott, and P. N. Breyse. 1991. Electromagnetic field exposure and male breast cancer. *The Lancet*: 337:737.

28. Vägerö, D., A. J. Swerdlow, and V. Beral. 1990. Occupational and malignant melanoma: a study based on cancer registration data in England, Wales and in Sweden. *Brit. J. Ind. Med.* 47:317-325.

29. Stevens, R. G., B. W. Wilson, and L. E. Anderson. 1990. The question of cancer. Pp. 369-370 in *Extremely Low Frequency Electromagnetic Fields: The Question of Cancer*, B. W. Wilson, R. G. Stevens, and L. E. Anderson, eds. Columbus, OH: Battelle Press.

30. Tenforde, T. S., and Budinger. 1986. Biological effects and physical safety aspects of NMR imaging and in vivo spectroscopy. Pp. 493-548 in *NMR in Medicine: Instrumentation and Clinical Applications*, S. R. Thomas, and R. L. Dixon, eds. New York, NY: American Association of Physicists in Medicine.

31. Robinette, C. D., C. Silverman, and S. Jablon. 1980. Effects upon health of occupational exposure to microwave radiation (radar). *Am. J. Epidemiol.* 112:39-53.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

32. Barron, C. I., and A. A. Baraff. 1958. Medical considerations of exposure to microwaves (radar). *JAMA* 168 (No. 9):1194-1199.

33. Szmigielski, S., M. Blelec, S. Lipski, and G. Sokolska. 1988. Immunologic and cancer-related aspects of exposure to low-level microwave and radiofrequency fields. Pp. 861-925 in *Modern Bioelectricity*, ed. A. A. Marino, New York: Marcel-Dekker.

34. Lilienfeld, A., J. Tonascia, S. Tonascia, C. Libauer, G. Cauthen, J. Markowitz, and S. Weida. 1978. Evaluation of health status of foreign service and other employees from selected Eastern European posts (unpublished).

35. Selvin, S., J. Schulman, and D. W. Merrill. 1992. Distance and risk measures for the analysis of spatial data: a study of three childhood cancers. *Soc. Sci. Med.* 34:769-777.

36. Kraut, W. T., E. Chan, P. J. Liroy, F. B. Cohen, B. D. Goldstein, and P. J. Landrigan. 1991. Epidemiologic investigation of a cancer cluster in professional football players. *Environ. Res.* 56:131-143.

37. Hawaii Department of Health Environmental Epidemiology Program. 1986. "Cancer incidence in census tracts with broadcasting towers in Honolulu, Hawaii." Report to the City Council, City and County of Honolulu, Hawaii.

38. Milham, S., Jr. 1985. Silent keys: Leukemia mortality in amateur radio operators. *Lancet* 1:812.

39. Milham, S., Jr. 1988. Increased mortality in amateur radio operators due to lymphatic and hematopoietic malignancies. *Am. J. Epidemiol.* 127:50-54.

40. Milham, S., Jr. 1988. Mortality by license class in amateur radio operators. *Am. J. Epidemiol.* 128:1175-1176.

41. Kaune, W. T., R. G. Stevens, N. J. Callahan, R. K. Severson, and D. B. Thomas. 1987. Residential magnetic and electric fields. *Bioelectromagnetics* 8:315-335.

42. Barnes, F., H. Wachtel, D. Savitz, and J. Fuller. 1989. Use of wiring configuration and wiring codes for estimating externally generated electric and magnetic fields. *Bioelectromagnetics* 10:13-21.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

43. Miller, R. W. 1979. Epidemiology of leukemia. Pp. 37-41 in Modern Trends in Human Leukemia, Vol. III, R. Neth, R. C. Gallo, P. H. Hotschneider, and K. Manweller, eds. New York, NY: Springer Verlag.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

10

Standards and Guidelines for Exposure to Radiofrequency and Extremely-Low-Frequency Electromagnetic Fields

Many countries and several international organizations have adopted guidelines or standards for exposures to electromagnetic fields (EMFs) in occupational environments and for exposures of general (nonoccupational) populations. Such guidelines and standards are based on evaluation of the established scientific literature, including an evaluation of the consistency of the findings that have been reported. Preliminary data or unproven hypotheses are often difficult to convert into recommended limits, and their use could result in the need for frequent changes in limits as research develops. Guidelines are usually based on effects observed in organisms, and laboratory effects are often difficult to interpret for deriving limits for protection of human beings. Many guidelines consider exposures of occupational groups and the general population separately, in view of their differences in length of exposure and distribution of states of health and physiological reserve capacities.

Standards and guidelines are based on dosimetric considerations combined with absorption characteristics of the body at different frequencies of EMFs. The basic premise is that any adverse effects are proportional to the rate of radiofrequency (RF) energy absorbed, and the specific absorption rate (SAR) is a common measure for characterizing the prescribed limits. Several authors have reviewed the different RF standards and guidelines.¹⁻³

Earlier standards and guidelines addressed limited frequency ranges, such as 300-300,000 MHz, but more recent standards and guidelines include the lower frequencies, down to 300 kHz or even 10 kHz. There are several standards and guidelines for electric-power frequencies of 50 and 60 Hz in the extremely-low-frequency (ELF) range.⁴⁻⁶

Many standards and guidelines are based on a conclusion from the relevant literature that, at a threshold RF exposure of about 4 W/kg, animals will show a change in their behavior when exposure conditions are well controlled. That threshold is reduced to 1 W/kg at high ambient temperatures. Most standards require that exposures be kept at or below one-tenth of the observed threshold SAR (0.4 W/kg). The recommended limits for the general population are reduced by a safety factor of 5 to an SAR of 0.08 W/kg, to allow for a greater range of physiological reserve capacity. For different frequency ranges, the recommended limits for unperturbed field strength and equivalent plane-wave power density are then adjusted for the different absorption characteristics of the human body. In the lower frequency ranges, further reductions in field strengths might be recommended to reduce the danger of RF burns from direct contact with metal objects.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

Some countries in Eastern Europe have adopted standards based on medical evaluations of workers in RF fields and on epidemiologic data on larger populations working or living in RF environments. That has resulted in the adoption of low limits that incorporate substantial safety factors.

Standards abstracted from several sources are summarized in Tables 10-1 and 10-2. Table 10-1 contains occupational exposure guidelines; Table 10-2 contains guidelines for the general public. In both tables, the guidelines are presented to represent frequencies as close as possible to those used by the GWEN system.

The electric and magnetic fields induced in the human body by low-frequency and ultra-high-frequency radiation from the GWEN transmitters at locations outside the perimeter fence (see Chapter 3) are less than the accepted limits for general-public exposures stated in all the standards or guidelines, except those promulgated in the former Soviet Union.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 10-1 OCCUPATIONAL EXPOSURE STANDARDS OR GUIDELINES FOR SEVERAL COUNTRIES OR AGENCIES

Country or Agency	Frequency(MHz)	Electric Field(V/m)	Magnetic Field (A/m)	Power Density (W/m ²)
International Radiation Protection Association	0.1-1.0	614	1.6/f*	—
	> 1.0-10	614/f	1.6/f	—
Australia	0.3-9.5	194	0.51	100
	>9.5-30	1,841/f	4.89/f	95/f
Austria	0.01-0.03	1,500	350	—
	0.03-2.0	1,500	7.06/f ^{1.113}	—
Canada (proposed)	0.01-1.2	600	4	—
	1.2-3.0	600	4.8/f	—
Germany	0.01-0.03	1,500	350	—
	0.03-2.0	1,500	7.5/f	—
Italy	0.3-3.0	194	2	—
	0.3-3.0 (peak)	300	4	—
	3.0-1500	61	0.16	10
United Kingdom	0.05-0.3	2,000	5/f	—
	0.3-10	600/f	5/f	—
United States (ANSI)	0.3-3.0	614	1.58	1,000
	3.0-30.0	1,892/f	4.74/f	9,000/f ²
IEEE for controlled environments	0.003-0.1	614	163	—
	0.1-3.0	614	16.3/f	—
United States (ACGIH, 1991)	0.01-3.0	614	1.63	1,000
	3.0-30	1842/f	4.89/f	9,000/f ²
Former Soviet Union	0.06-1.5	50	5	—
	1.5-3.0	50	—	—

* f = frequency in MHz

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 10-2 GENERAL PUBLIC EXPOSURE STANDARDS OR GUIDELINES FOR SEVERAL COUNTRIES OR AGENCIES

Country or Agency	Frequency (MHz)	Electric Field (V/m)	Magnetic Field (A/m)	Power Density (W/m ²)
International Radiation Protection Association	0.1-1.0	87	0.23/f ^{0.5} *	—
	> 1.0-10	87/f ^{0.5}	0.23/f ^{0.5}	—
Austria	0.01-0.03	670	157	—
	0.03-2.0	670	3.16/f ^{1.113}	—
Canada (proposed)	0.01-1.2	280	1.8	—
	1.2-3.0	280	2.1/f	—
Germany	0.01-0.03 0.03-2.0	1,500 1,500	350 7.5/f	—
Italy	0.3-3.0	60	0.2	—
	3.0-1500	20	0.05	1
United Kingdom	0.05-0.365	800	2 x 10 ⁶ /f	—
	0.365-0.475	800	5.5	—
United States (ANSI)	0.3-3.0	632	1.58	1,000
	3.0-30.0	1,897/f	4.74/f	9,000/f ²
Former Soviet Union	0.03-0.3	25	—	—
	0.3-3.0	15	—	—

* f = frequency in MHz

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

REFERENCES

1. Grandolfo, M., and K. H. Mild. 1989. Worldwide public and occupational radiofrequency and microwave protection guides. Pp. 99-134 in *Electromagnetic Biointeraction, Mechanisms, Safety Standards, Protection Guides*, G. Franceschetti, O. P. Gandhi, and M. Grandolfo, eds. New York, NY: Plenum Press.
2. Repacholi, M. H. 1990. Radiofrequency field exposure standards: Current limits and the relevant bioeffects data. Pp. 9-27 in *Biological Effects and Medical Applications of Electromagnetic Fields*, O. P. Gandhi, ed. Englewood Cliffs, NJ: Prentice Hall.
3. Szmigielski, S., and T. Obara. 1989. The rationale for the Eastern European radiofrequency and microwave protection guides. Pp. 135-151 in *Electromagnetic Biointeraction, Mechanisms, Safety Standards, Protection Guides*. G. Franceschetti, O. P. Gandhi, and M. Grandolfo, eds. New York, NY: Plenum Press.
4. American Conference of Government and Industrial Hygienists. 1990-1991. Threshold limit values and biological exposure indices. Am. Conf. Gov. Ind. Hygienists, Cincinnati, Ohio.
5. International Radiation Protection Association. 1990. Interim guidelines on limits of exposure to 50/60 Hz electric and magnetic fields. *Health Phys.* 58:113-122.
6. Grandolfo, M., and P. Vecchia. 1988. Standards on extremely low-frequency (ELF) electric and magnetic fields and their rationales. In *Non-Ionizing Radiations: Physical Characteristics, Biological Effects and Health Hazard Assessment*, M. H. Repacholi, ed. Proc. Int. Non-Ionizing Radiation Workshop, Melbourne, Australia, April 5-8, 1988.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

11

Risk Analysis and Management

This chapter summarizes information on population exposure to GWEN fields, compares GWEN exposures to existing standards and to exposures from other sources of nonionizing fields, draws on existing epidemiological evidence to derive upper bounds on GWEN-related risks, describes likely public concerns regarding risks associated with GWEN emissions, and suggests alternatives for addressing those concerns.

RISK ASSESSMENT

Scientific tools are too blunt to measure directly the human health risks associated with low-level exposures to most environmental agents.¹ Estimates of such risks are garnered instead by extrapolation both from studies at high exposure levels and from experiments with laboratory animals, cells, and tissues.

Risk assessment is an analytical tool for characterizing potential health risks to individuals or populations by combining existing epidemiologic and laboratory data on the potency of an agent with estimates of expected levels of human exposure. A National Research Council committee² has identified four elements of a complete risk assessment: hazard identification, exposure assessment, exposure-response evaluation, and risk characterization. Hazard identification involves reaching scientific consensus on whether exposure to an agent can cause an increase in the incidence of some deleterious health condition. Exposure assessment is the characterization of the spatial and temporal patterns of exposure to the agent. Exposure response evaluation involves gleaned from epidemiologic studies and experiments on laboratory animals a functional relation between the level of exposure and the magnitude of the induced health hazard. Risk characterization combines estimates of exposure and estimates of effect per unit of exposure from the two preceding stages to derive quantitative statements about human health risk.

Each stage of this risk assessment process involves uncertainties. Agents shown to be hazardous to some animals might not be hazardous to humans. Agents that induce deleterious effects at high exposure levels might be harmless or even beneficial at very low exposure levels of exposure. Exposure assessments are hampered by limitations in data collection. Finite resources limit the number of exposure conditions and health endpoints for which health responses can be evaluated. Because these and other uncertainties in the risk assessment process can be large, it is important that uncertainty be explicitly incorporated in any risk characterization.

Uncertainties in risk assessment are particularly large in the case of exposures to subthermal levels of nonionizing radiation because of the combination of perplexing epidemiological and laboratory findings, the lack of a testable hypothesis for health effects,

and difficulties in reproducing effects in separate laboratories. The problem of assessing risk associated with nonionizing fields is further complicated by evidence that effects might not be monotonically related to field strength. The laboratory literature includes many examples that suggest that effects appear only within particular windows of frequency or field intensity.³⁻⁵ This evidence greatly enlarges the range of exposure conditions that an exposure-response evaluation must consider to assure completeness. Finally, GWEN LF frequencies are 3,000 times higher than those associated with the ELF epidemiological studies reviewed in [Chapter 9](#). Risk extrapolations to GWEN LF exposures based on ELF epidemiologic results are, therefore, very sensitive to the choice of exposure measure. Because risk extrapolations with such large uncertainties are of little use in a policy context, they have not been pursued in this study.

In summary, the application of traditional tools of risk assessment in the GWEN context are frustrated by (1) uncertainties over what, if any, human health end points are affected by exposure to GWEN fields, (2) a lack of evidence of a relationship between increasing risk and increasing levels of some measure of field exposure (i.e., a dose-response relationship) for nonionizing fields of any frequency, and (3) the uncertainty of the basis for extrapolating effects from one exposure regime (frequency, field strength, etc.) to another.

Although it is premature to apply the traditional risk assessment process to GWEN exposure, a variety of analyses can provide valuable perspective on possible risks from GWEN emissions. In the remainder of this chapter, we summarize information on population exposure to GWEN fields, compare GWEN exposures to existing standards and to exposures to nonionizing fields from other sources of nonionizing fields, and draw on existing negative epidemiological evidence at or near GWEN frequencies to derive upper bounds on GWEN-related risks. We also discuss some important risk-management issues including likely public concerns regarding risks associated with GWEN emissions and ways to address those concerns.

GENERAL DESCRIPTION OF GWEN FIELDS

GWEN relay nodes (RNs) broadcast both low-frequency (LF) signals (150-175 kHz) and ultra-high-frequency (UHF) signals (225-400 MHz). GWEN's fixed input/output (I/O) terminals produce only UHF signals. GWEN LF and UHF transmitters are both frequency-modulated and transmit only periodically according to well-defined communication protocols. System design and protocol are such that time-averaged duty cycles for any LF transmitter can be no less than 0.0014 and no more than 0.27. At full RN implementation, typical LF duty cycles are expected to be about 0.014. Ground-to-air UHF transmitters would be constrained by design and protocol to time-averaged duty cycles of 0.019-0.27. Ground-to-ground UHF transmitters for the 16 RNs serving I/O nodes have a fixed duty cycle of 0.40. Unless otherwise noted, LF and UHF exposures reported below represent peak fields, not time-averaged fields.

GWEN LF broadcasts are made from a 299-ft vertical top-loaded monopole antenna and are isotropic in azimuth. The LF radiated power during LF broadcasts is nominally 2,000 W, but could be as high as 3,200 W, depending on the needs of the site. The intensity of the ground-level LF fields produced by a GWEN LF transmitter is shown in [Figure 2-4](#).

There are two types of GWEN UHF transmitters. UHF transmitters at most GWEN RNs are designed for airborne communication. They broadcast in a pattern that is omnidirectional in azimuth, but somewhat directional in elevation, with the strongest fields occurring in a shallow 8° cone extending outward and upward from the antenna position some 10 m above the ground. The radiated power from the UHF antenna is typically 50 W, but can be as high as 79 W with a duty cycle less than 0.27. The field profile of the antenna, in the cone of peak field strength with an assumed radiated power of 79 W is shown in [Figure 2-5](#). Field strengths at ground level are somewhat smaller than shown in [Figure 2-5](#), primarily because ground-level fields are strongly affected by ground reflections. For RNs constructed to date, UHF-transmitting antennae are outside the 4-ft fence that surrounds the LF antenna, a few tens of meters from the site boundary.

Twelve GWEN RNs are equipped with a ground-to-ground UHF-transmitting antenna that is directional in both azimuth and elevation. This antenna is mounted on top of a pole 20-150 ft high with its main lobe of 9-dB gain directed horizontally. The radiated power from the antenna is typically 50 W with a 0.4 duty cycle and a narrow beam.

In characterizing GWEN emissions in those two bands, it is important to distinguish between the far field, where the ratio of electric-field intensity to magnetic-field intensity is constant, and the near field, where the electric-and magnetic-field components of an electromagnetic wave are independent of one another. For GWEN transmissions, the near field extends to a couple of kilometers for LF transmissions and to several meters for UHF transmissions. Thus, much of the population exposure of interest occurs in the near field of the LF antenna and in the far field of the UHF antenna. Population exposures to UHF fields can be completely specified by the UHF electric field, by the magnetic field, or by power density alone. Descriptions of most LF exposures, however, need to include both the electric and magnetic fields.

COUPLING OF ELECTROMAGNETIC FIELDS TO HUMAN BODY

To the extent that nonionizing electric and magnetic fields have biological effects, the effects will depend on the fields induced in the body. In general, fields produced in the body are not the same as fields that one would measure in air at the same location if the body were absent. For that reason, nonionizing electromagnetic-field (EMF) exposures are usually estimated in two parts. First, the "incident" fields are calculated or measured. These are the electric and magnetic fields that are produced in the absence of distorting influences, such as a human body. Second, various dosimetric principles are used to relate incident fields to fields that are induced in the body.⁶ The relationship between incident and internal

fields depends on frequency, orientation of the body relative to the field, body size, and the impedance of the body to ground.

Comparisons of the intensity of exposure produced by various sources must account for any differences in those factors. For instance, UHF fields couple much more strongly to the body than do LF fields. A vertically polarized electric field at ultrahigh frequencies induces an internal electric field in an ungrounded standing adult that is 300 times that induced by an LF electric field of the same incident strength and polarization.⁶ In many areas outside the boundaries of GWEN sites, LF-field strengths at ground level can be expected to exceed UHF-field strengths by less than a factor of 300, so that the internal electric fields from a GWEN site will be predominantly from the UHF component.

The incident LF and UHF fields of the GWEN system induce electric fields, magnetic fields, and currents in the bodies of exposed people. The strength of this coupling is described in [Chapter 3](#) for a "worst case," defined as a grounded person standing near the 4-ft perimeter fence of a GWEN site. Dosimetric calculations show that body currents induced by electric-field coupling greatly exceed those induced by magnetic-field coupling for GWEN exposures. Later analyses of LF exposure and risk in this chapter therefore focus on the electric field.

SHIELDING BY BUILDINGS

Buildings and trees attenuate the electric-field component of LF fields and both the electric-and magnetic-field components of UHF fields. Measurements suggest that houses shield LF electric fields by 12-30 dB (75-97%), UHF electric fields by 0-20 dB (0-90%), and UHF magnetic fields by 0-10 dB (0-68%).⁷ People spend much of their day indoors, so the shielding can have a significant influence on exposure from sources outside the home.

POPULATION DISTRIBUTION AROUND GWEN SITES

If there is a public-health impact of exposure to GWEN fields, it will be proportional to the number of people exposed. The population density around candidate locations at 40 RN sites was estimated by surveying U.S. Geological Survey topographical maps of the areas.⁸ Survey locations were chosen randomly from the list of candidate locations for each site, unless a specific location had already been specified. Structures not specifically identified as schools, churches, or factories were counted as homes. Structures were counted in each of six contiguous concentric regions around the site, with outer radii of 200, 300, 500, 1,000, 2,000, and 4,000 m. The distance from the center of each site to the nearest house was noted. The raw data are listed in [Table 2-3](#) and presented in [Figures 11-1, 11-2, and 11-3](#). The data show that the density of houses within several kilometers of GWEN RN sites is typically about one house/km². That is sparse, compared with the average of 11 households/km² for the conterminous 48 states as a whole, and is about the same as the

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

average house density in Montana and the Dakotas. None of the 40 sites examined had a housing density exceeding 10/km². The typical distance from site center to the nearest house is 330 m. Only a few sites have houses within 150 m.

Several sources of bias are possible in these data. First, counting all structures as houses except those known to be something else will tend to bias housing densities upward. In contrast, the topographical maps used for this study had an average age of 15 yr (standard deviation, 8.7 yr); given general trends of increasing population density, this will tend to bias estimated housing densities downward. Finally, we do not know the extent to which the 40 RN sites examined are representative of the entire GWEN system.

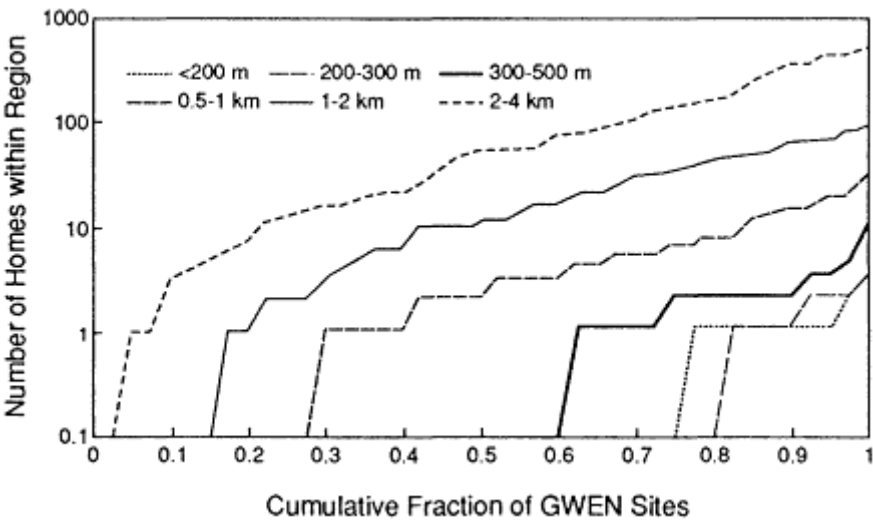


Figure 11-1.
Cumulative distribution of number of homes in six contiguous concentric regions around sample of 40 RN sites.

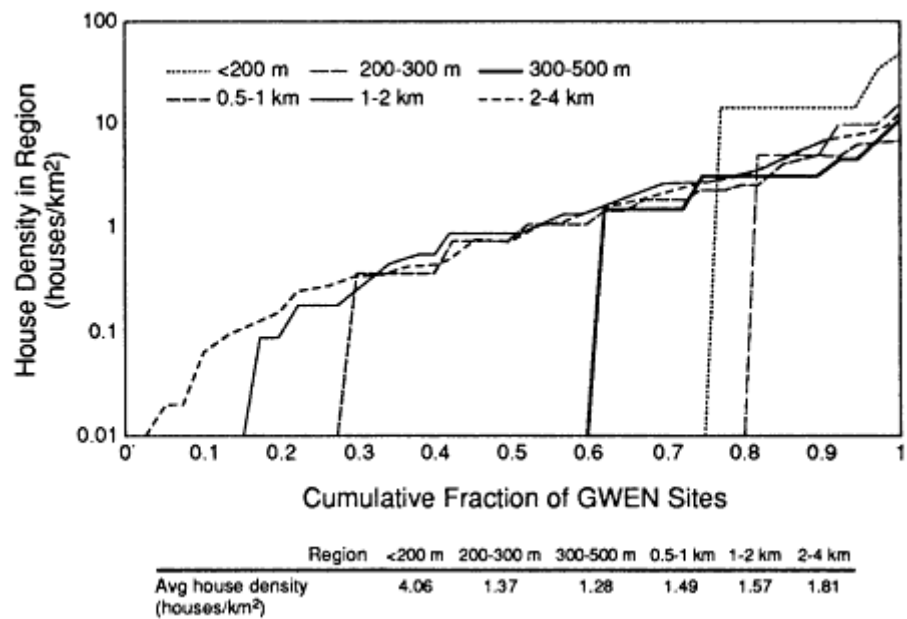


Figure 11-2.
Cumulative distribution of home density in six contiguous concentric regions around sample of 40 RN sites.

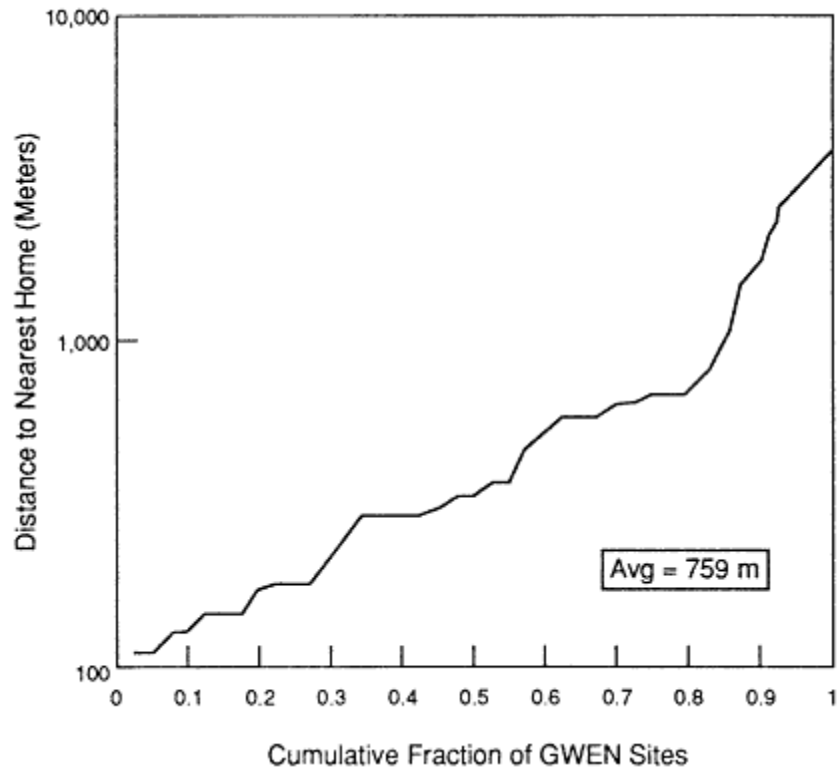


Figure 11-3.
Cumulative distribution of distance of center of 40 RN sites to nearest home.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

LF AND UHF EXPOSURES OF POPULATION AROUND SITES

Rough estimates of the numbers of people living in homes exposed to GWEN fields above various intensities can be derived from the population and field-strength data described above. Estimates are presented in Table 11-1. Estimates for RN sites are derived by assuming all structures identified in the survey of topographical maps are occupied at the national average rate of 2.6 persons/household.⁹ Estimates for the total GWEN system assume that existing thin-line connectivity capability (TLCC) relay nodes are similar to GWEN RNs. UHF exposures are assumed to occur at antenna height (10 m). At full RN implementation, time-averaged field strengths will be about 0.014 of peak values for LF and 0.019-0.027 of peak values for UHF.

TABLE 11-1. Approximate Number of People Living in Homes Exposed to GWEN Fields of Various Peak Intensities.

Peak Field Strength	40 RN Sites	Total GWEN System
0.02 V/m < UHF < 0.05 V/m	17,000	40,000
0.05 V/m < UHF < 0.1 V/m	2,000	4,700
0.1 V/m < UHF < 0.2 V/m	420	1,000
0.2 V/m < UHF < 0.5 V/m	80	200
0.5 V/m < UHF	0	0
0.1 V/m < LF < 0.2 V/m	11,000	28,000
0.2 V/m < LF < 0.5 V/m	3,300	7,800
0.5 V/m < LF < 1.0 V/m	400	1,000
1.0 V/m < LF < 2.0 V/m	80	200
2.0 V/m < LF	30	80
5 µG < LF < 10 µG	6,600	15,000
10 µG < LF < 20 µG	1,300	3,100
20 µG < LF < 50 µG	300	800
50 µG < LF < 100 µG	50	110
100 µG < LF < 200 µG	15	40
200 µG < LF	25	55

The persons exposed to the strongest GWEN fields continuously are, of course, those who live closest to the sites. Someone living at a site boundary would be exposed to GWEN LF fields at ground level of up to 40 V/m and 0.7 mG.

Plans for GWEN RNs call for 10-m-high UHF antennae to be near the corners of GWEN sites. A nearby house could theoretically be as close as 30 m from the base of the UHF antenna. That would expose residents on the second floor of a two-story house to power densities up to 1 $\mu\text{W}/\text{cm}^2$. Exposures at ground level would be somewhat smaller.

EXPOSURE COMPARISONS WITH EXISTING STANDARDS

National and international scientific bodies have developed exposure guidelines for radiofrequencies (RFs), including the LF and UHF bands; these are reviewed in [Chapter 10](#). The strongest fields that might be encountered at the boundary of a GWEN facility are LF fields of 40 V/m and 0.7 mG and UHF fields with power densities of 1 $\mu\text{W}/\text{cm}^2$. Those are well below all existing guidelines for LF magnetic fields and UHF power densities. Although peak LF electric-field exposures at the fenceline are close to both International Radiation Protection Association and USSR standards, time-averaged exposures are well below the guidelines.

EXPOSURE COMPARISONS WITH OTHER SOURCES

The most prominent sources of population exposure to RF radiation at or near GWEN frequencies are UHF television (470-806 MHz) and AM radio (535-1610 kHz) broadcasts. Environmental Protection Agency (EPA) measurements of RF radiation in 15 U.S. cities¹⁰ enable comparisons of GWEN exposures to exposures from those broadcast sources. [Figure 11-4](#) shows the numbers of people exposed above a given field level for both the 15-city urban population of the EPA study and populations near GWEN facilities. On the basis of the time-averaged electric field induced in body tissues, LF exposures of someone living at the boundary of a GWEN RN are comparable with the median exposure to AM-broadcast radiation in urban areas.

A second source of LF frequencies is the LORAN navigation system. The LORAN system consists of 20 transmitters along U.S. coastlines broadcasting pulse trains on a carrier wave of 150-170 kHz. LORAN stations are generally in rural areas and operate at peak powers of 275-1,600 kW, with a typical time-averaged emission of roughly 15 kW. By comparison, GWEN LF transmissions have a peak power of 2.0-3.2 kW, with typical time-averaged emissions of 30 W.

In countries other than the United States, a substantial source of exposure to LF fields is AM-radio broadcasts. These are made at frequencies of 150-175 kHz in France, Germany, Mongolia, Morocco, Turkey, and the USSR at 150-2,000 kW.¹¹

[Table 11-2](#) compares GWEN and other LF and UHF sources on the basis of broadcast power and number of emitters in the United States. GWEN sites are fewer, lower in power, and in more sparsely populated areas than many other broadcast sources at comparable

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

frequencies. The data in [Table 11-2](#) and [Figure 11-4](#) show that population exposures to GWEN fields are modest compared with those associated with broadcast sources in nearby bands.

TABLE 11-2. RF Power and Frequencies of GWEN and Other Sources of Radiofrequency Radiation

Source	Frequency, MHz	Range of RF Power Across Sources, kW	Time-averaged RF Power of Typical Source, kW	No. U.S. sites
GWEN LF	0.150-0.175	2-3	0.028	127
LORAN LF	0.150-0.170	275-1600	15	15
AM radio	0.525-1.610	0.25-50	5	4972
GWEN UHF	225-400	0.020	0.00066	139 ^a
Cellular-phone base station	800-900	0.040-4	0.4	< 5,000
UHF TV	470-806	5-5,000	350	769

^a Does not include 34 airborne I/O terminals.
Source: See references 10 and 12-14.

Comparisons can also be made between exposures to GWEN fields and exposures to less similar sources of nonionizing fields. [Figure 11-5](#) compares exposures at the perimeter of a GWEN site with other exposure situations involving sources in the extremely-low-frequency (ELF), very-high-frequency (VHF), and superhigh-frequency (SHF) bands. The situations include living next to a 500-kV transmission line, standing beneath a neighborhood distribution line, living in an urban area served by FM radio, using a cordless telephone, and standing 1 m away from a microwave oven. Comparisons are based on four measures of exposure: time-and body-averaged magnetic flux density, time-and body-averaged power absorption, instantaneous peak power absorption in any tissue, and fraction of time exposed. The data in [Figure 11-5](#) demonstrate how sensitive exposure comparisons are to the choice of exposure measure. For sources that vary widely in frequency, for instance, comparisons based on power absorption are very different from those based on incident field strength, because power absorption is proportional to the square of the frequency.

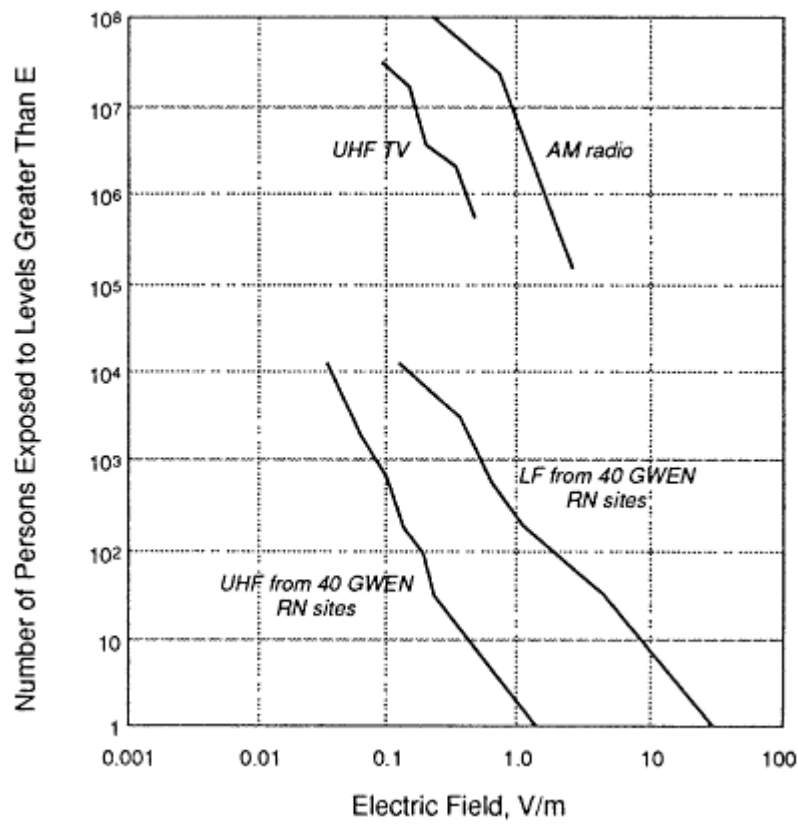


Figure 11-4. Cumulative distributions of people exposed to GWEN and commercial broadcast fields above given electric-field strength. Broadcast exposures are derived by combining census data with EPA survey of RF-field exposures in urban areas.^{9, 10} GWEN data are taken from Table 11-1. Curves for GWEN system assume 100% duty cycle. Time averaging based on expected GWEN duty cycles would shift GWEN curves to left by factors of 70 and 50 for LF and UHF, respectively.

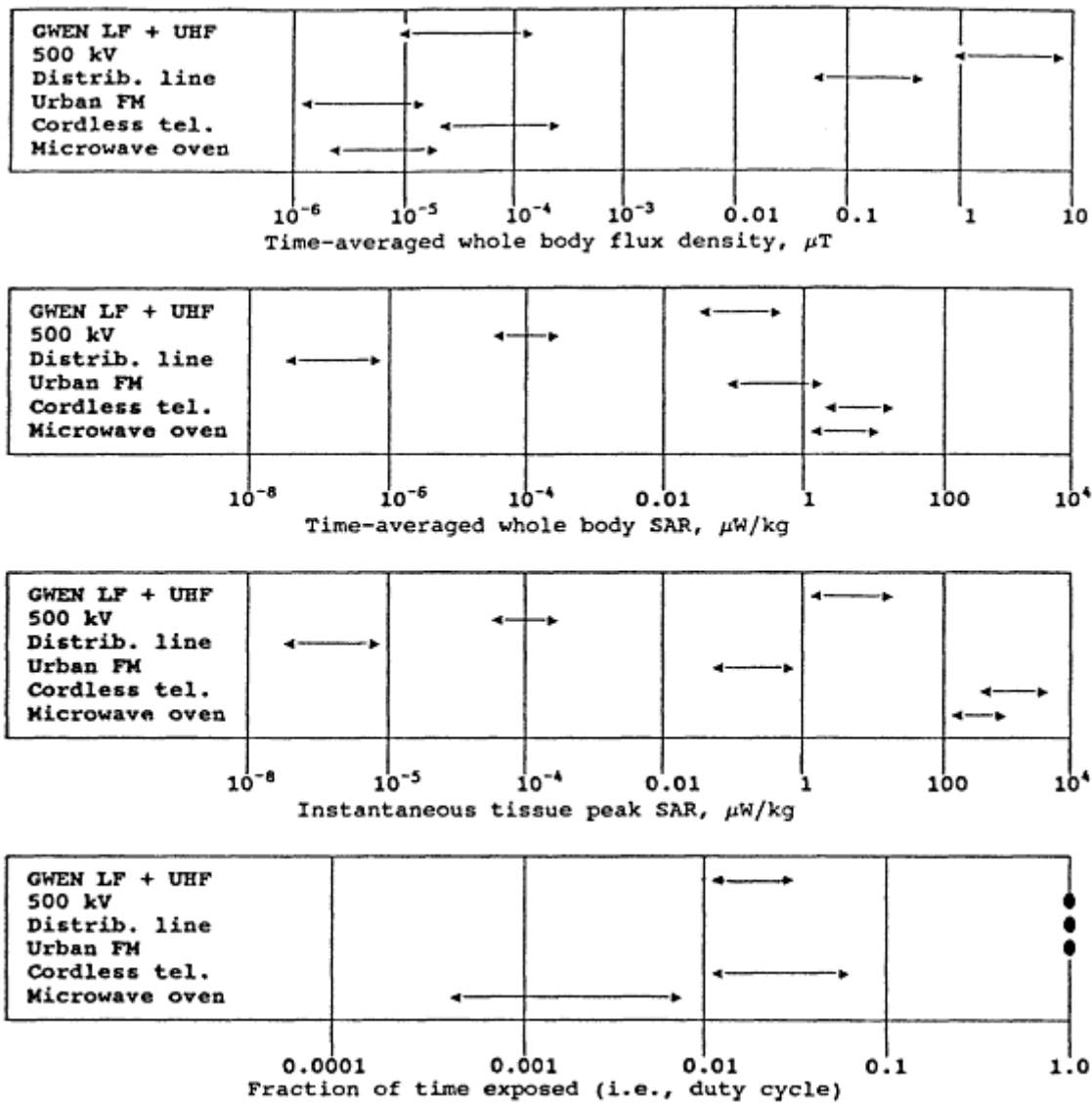


Figure 11-5. Comparison of exposures to GWEN fields at site boundary with five common exposure situations, based on four alternative exposure measures. Situations include living on edge of 500-kV right-of-way, standing beneath neighborhood distribution line, being exposed to ambient FM-broadcast radiation in urban areas, using cordless phone, and using microwave oven. SAR, specific absorption rate.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

BOUNDING GWEN RISKS

Although information to perform a traditional assessment of GWEN risks is lacking, gross upper bounds on possible GWEN risks can be identified by using negative epidemiological evidence related to exposures from existing RF broadcast facilities. These bounding arguments, developed in detail below, derive from the following observations:

- Changes in cancer morbidity and mortality over the periods of most rapid growth in MF, VHF, and UHF broadcasting have been no greater than a few chances in 100,000 per person per year.
- Local public health surveillance has identified no increase in diseases or deaths attributable to any particular RF broadcast facility.
- An epidemiological study of 7,889 workers in the New Jersey Meadowlands Sports Complex found no increase in the incidence of malignancies or deaths after exposure for up to 11 yr to broadcast radiation from several nearby AM antennae.

The upper bounds on GWEN risks derived below are based on indirect evidence and so represent the largest risk that cannot be ruled out by available evidence. The probability is small that GWEN risks could be as large as any of the bounds that fall out of these indirect arguments, as discussed in a later section of this chapter. In the committee's collective judgment, the probability that the true GWEN risk is essentially zero is far greater than the probability that it exceeds any of the upper-bound risk estimates derived in this report.

HISTORICAL GROWTH IN BROADCAST ACTIVITY

One indirect measure of the possible contribution of RF exposures to cancer is the correlation between time series of cancer morbidity and mortality and the number of active radio and TV stations. [Figure 11-6](#) depicts the growth in the number of AM-, FM-, and TV-broadcast stations licensed by the Federal Communications Commission (FCC) over the period 1940-1990. All three media have displayed large and steady growth during those years.

Cancer types that have been statistically associated with EMF exposure in some epidemiological studies are leukemias, nervous system tumors, non-Hodgkins lymphomas, and breast cancers (see [Chapter 9](#)). The 30-yr trend in the age-adjusted death rate from those diseases is shown in [Table 11-3](#). Time-series data on cancer incidence are more limited than death-rate data. [Table 11-4](#) shows changes in age

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

adjusted incidence for the same cancers over a 14-yr period beginning in 1973, the first year for which such data were collected by the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program.

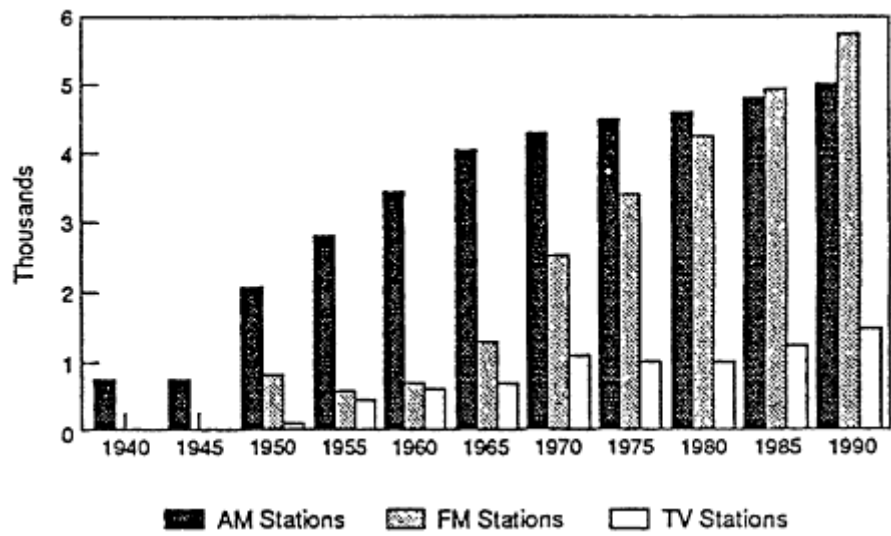


Figure 11-6.
Growth of licensed broadcast stations (based on FCC records).

Interpretation of the cancer data in Tables 11-3 and 11-4 is complicated by changes in the efficacy of cancer diagnosis, reporting, and treatment over the years as well as by changes in the risk of accident and disease from other causes. These data indicate that the combined effect of all influences on recorded death and incidence rates for these cancers is generally no more than a few chances per 100,000 per year. The potential contribution of EMF effects alone is unlikely to be significantly greater than this, because that would require that all other influences on recorded death and incidence rates combine roughly to obscure any EMF effects.

A person living adjacent to a GWEN RN in an LF field of 40 V/m with a duty cycle of 0.014 and a coupling efficiency that is 22% of that at AM frequencies would be exposed to a time-averaged electric field that is about half (0.12 V/m) that induced by the median AM broadcast exposure in the United States (0.28 V/m). Thus, the upper bound on broadcast risks for GWEN would not be expected to exceed those associated with the median AM broadcast exposure in the United States.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

TABLE 11-3. 30-Year Trends in Age-Adjusted Death Rates per 100,000 Population for Cancers that have been Associated with Electromagnetic-Field Exposure.

		Age-Adjusted Death Rate per 100,000 Population		
Cancer	Sex	1955-1957	1985-1987	Change, %
Breast	Male	0.3	0.27	-11
	Female	26.3	27.2	+ 3
Brain	Male	4.0	4.9	+ 22
	Female	2.7	3.3	+ 22
Leukemia	Male	8.5	8.2	-4
	Female	5.6	4.9	-12
Non-Hodgkin's lymphoma	Male	4.7	7.1	+ 51
	Female	3.0	4.8	+ 60

Source: Cancer Facts and Figures - 1991, American Cancer Society, Atlanta, GA, 1991.

TABLE 11-4. 14-Year Trends in Age-Adjusted Incidence per 100,000 Population for Cancers Possibly Associated with Electromagnetic-Field Exposure.

		Age-Adjusted Incidence Rate per 100,000 Population		
Cancer		1973	1986	Change, %
Breast		44.8	57.6	+ 29
Brain and nervous system		5.0	6.1	+ 22
Leukemia		10.5	9.3	-11
Non-Hodgkin's lymphoma		8.5	12.6	+ 48

Source: Cancer Statistics Review 1973-1986, National Cancer Institute, May 1989.

PUBLIC HEALTH SURVEILLANCE AROUND BROADCAST FACILITIES

Powerful commercial and navigation broadcast stations have been operating for many years at or near GWEN LF and UHF frequencies both in the United States and elsewhere^{11, 12} (see [Table 11-2](#)). Yet public health monitoring of spatial patterns of disease has identified no correlation of regional morbidity or mortality with distance from a broadcast antenna. That no such evidence has emerged suggests that, whatever the public health risks related to broadcast exposure might be, they are smaller than the detection threshold of the health surveillance apparatus. This observation can be used to place an upper bound on the possible health impacts of the GWEN system.

LF Broadcasting in Europe. Inquiries were made on behalf of the committee to the operators of several LF-Broadcast facilities in Europe. The results, shown in [Table 11-5](#), suggest that any possible health effects of LF fields are not large enough to have been spontaneously noticed in populations of workers and residents near the facilities. On a time-averaged basis, the electric fields to which the European broadcast facilities expose populations are up to 10 times stronger than the LF electric field at the boundary of a GWEN RN site.

LORAN Stations. The 15 LORAN-C navigation transmitters in the United States operate at LF frequencies and in a pulsed mode similar to that of GWEN LF transmitters.¹³ LORAN stations tend to be in areas of low to moderate population density, and there is no evidence of excess cancer risk associated with the fields from these facilities.

MEADOWLANDS SPORTS COMPLEX

The Meadowlands Sports Complex in East Rutherford, NJ, is very near a number of AM radio broadcast antennae.¹⁴ A recent study by Kraut and colleagues of cancer incidence and cancer deaths in a population of almost 8,000 workers at the sports complex found no significant differences in cancer risk between worker and reference populations over the period 1978-1987.¹⁵ Electric fields measured at outdoor locations across the complex ranged from 1 to 16 V/m, with typical values of 3-10 V/m.¹⁶ Indoors, electric fields were much smaller, typically less than 0.3 V/m. No significant differences in cancer risk were noted between populations of 2,500 outdoor workers and 5,500 indoor workers employed over that limit the relevance of this study to short-latency cancers such as leukemia. The average employment duration for the Meadowlands cohort was roughly 5 yr.

Only two leukemia cases and no leukemia details were identified in the entire 8,000 worker cohort. Both leukemia diagnoses were among indoor workers (Personal communication with Dr. Allen Kraut, University of Manitoba, Winnipeg, October 1, 1992). Among outdoor workers, 1.11 leukemia cases would have been expected in a

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

cohort of that size. The corresponding upper bound on the 95% confidence interval for proportionate incidence ratio for outdoor workers is about 3.3. This upper bound corresponds to an excess morbidity of roughly 3 chances in 10,000 per person per year. Since the 5 yr survival rate for adult leukemias is only about 30%, these morbidity bounds represent an upper bound on mortality risk of roughly 2×10^{-4} per person per year.

TABLE 11-5. Results of Inquiries to LF-Broadcast Facilities in Europe.

Location	Power kW	Frequency kHz	Period of operation	Population	Electric Field exposure V/m
Beidweiler, Luxembourg	10,000	234	1972-1991	300 villagers	18
Junglinster, Luxembourg	200-1,200	234	1932-1972	1,500 villagers	7-16
Allouis, France	2,000	164	1961-1991	station up to 1,000 workers	
Oslo, Norway	200	216	1954-1991	nearest residents in scattered farmhouses	< 16
Tromso, Norway	10	154	1926-1991	airport town population	3

BOUNDS ON EXCESS POPULATION RISK FROM GWEN FIELDS

The upper bound on risk of excess cancer mortality associated with operation of GWEN LF and UHF transmitters can be estimated from four factors: (1) information on health surveillance of census tracts surrounding AM broadcast facilities; (2) relative exposures to RF fields of populations near AM broadcast stations and GWEN facilities; (3) population density around GWEN facilities; and (4) the magnitude of GWEN fields as a function of distance from the transmitter. In this calculation, it is assumed that any elevation in health risk is associated with absorbed energy from GWEN fields, and hence

is proportional to the square of the electric field intensity in air. The effects of the calculation of choosing an alternative exposure metric, namely, the electric field in air, is also discussed.

In our approach, an upper bound estimate of the health risks associated with GWEN LF radiation is computed from information on the absence of public health effects associated with exposure to AM broadcast fields. Public health statistics are regularly reviewed at the level of the census tract, which can be smaller than a city block in urban areas and larger than a county in sparsely populated areas. On the average, there are about 70 census tracts for every AM radio station in the United States with an urban location. The spatial resolution of health surveillance is therefore fine enough in many areas to detect any strong risk gradients around AM broadcast facilities. Health surveillance studies to date have not indicated the existence of risk gradients around broadcast facilities for the cancers which previous epidemiological studies have associated with EMF exposure. These cancers include leukemias, non-Hodgkins lymphomas, nervous system tumors, and breast cancers, which collectively impose an average background mortality risk of 3×10^{-4} per person per year in the United States. For purposes of risk estimation, let k be the smallest excess relative risk that would be detected by the health surveillance system and consider a continuously broadcasting AM station that creates a spatially-averaged electric field E_{am} in an adjacent census tract. If R_o is the background risk of cancer death in the region, then the largest EMF effect that could go unnoticed in the census tract is a risk of kR_o . That no such effects have so far been established has implications for EMF risks from GWEN facilities that can be quantified by considering the relative exposures of populations around AM broadcast stations and GWEN RNs.

If we assume that the risk is proportional to RF energy absorbed in tissue (and hence proportional to the square of the electric field intensity), then the largest excess risk, R_g , from exposure to peak GWEN LF fields of strength E_g that is consistent with the lack of evidence for populations living near AM broadcast facilities is:

$$R_g(r) < kR_o \gamma^2 \delta E_g(r)^2 / E_{am}^2 \quad (11-1)$$

where r is distance from a GWEN LF antenna in km, γ is the ratio of electric field coupling to the body at GWEN LF frequencies compared to that at AM frequencies, and δ is the duty cycle of GWEN LF emissions. The AM broadcasting is assumed to be continuous. This expression does not account for electromagnetic shielding from houses or other objects, nor does it account for time spent away from home (i.e., when exposures may not occur). We assume that these two factors have equal influence on risk from commercial AM broadcast fields and GWEN fields, and therefore cancel out of Equation (11-1).

To first order, the average electric field (V/m) in a census tract of radius C directly adjacent to an AM antenna of power P (watts) with a surrounding exclusion zone of radius ϵ (meters) is:

$$E_{am} = \sigma P^{1/2} / (C + \epsilon) \quad (11-2)$$

where σ is a factor in the range 4-10 ohm^{1/2} that depends on ground conductivity, antenna height, and wavelength. Combining Equations (11-1) and (11-2) yields Equation (11-3) for the largest estimate of cancer death from exposure to GWEN fields that cannot be ruled out from health surveillance data:

$$R_g(r) < k R_o \gamma^2 \delta E_g(r)^2 (C + \epsilon)^2 / (\sigma^2 P) \quad (11-3)$$

If ρ is the average population density in the region in persons/km², then the upper bound on excess population risk for a single GWEN facility for those living between r_1 and r_2 kilometers from the antenna is:

$$\text{Cases per year} < \int_{r_1}^{r_2} 2 \pi \rho R_g(r) r dr \quad (11-4)$$

Equation (11-4) was evaluated using typical values of all parameters to estimate the total population risk from a single GWEN facility out to a distance of 10 km. Beyond this distance, GWEN fields are negligible relative to typical AM broadcast exposures in urban areas. The values assigned to the parameters in Equations (11-3) and (11-4) are as follows:

- $k = 1$. This value of k assumes that a doubling of EMF-related cancer mortality in census tracts around a typical AM broadcast antenna would have been detected, at least at one site.
- $R_o = 3 \times 10^{-4}$ per person per years This risk estimate includes only those cancers that the epidemiological literature has suggested might be associated with EMFs, namely, leukemias, non-Hodgkins lymphomas, nervous tissue tumors, and breast cancers.
- $\gamma = 0.22$. This value is the ratio of electric field coupling of GWEN LF fields to that of AM broadcast fields at the center frequency of each band.
- $\delta = 0.014$. This value is the best estimate for the typical duty cycle of GWEN LF emissions at full peacetime implementation.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

- Out to a distance of 0.3 km from a GWEN LF antenna, the electric field beyond the boundary fence (at 0.1 km) is approximately:

$$E_g(r) = 0.04/r^3 \quad (0.1 \text{ km} < r < 0.3 \text{ km}) \quad (11-5)$$

where $E_g(r)$ is the electric field intensity in V/m and r is the ground-level distance from the antenna in kilometers. Beyond 0.3 km from the LF antenna, the electric field is approximately:

$$E_g(r) = 0.5/r \quad (0.3 \text{ km} < r < 10 \text{ km}) \quad (11-6)$$

- $C = 150 \text{ m}$. This value of the census tract radius is typical of larger cities.
- $\epsilon = 100 \text{ m}$. The exclusion zone is assumed to be as large as a typical AM antenna height.
- $\sigma = 7 \text{ ohm}^{1/2}$, an average value for this parameter.
- $P = 10,000 \text{ watts}$, the power of a typical AM station.
- $\rho = 5 \text{ persons/km}^2$. Data from the survey of aerial photographs of prospective sites for GWEN RNs indicate that the average density of homes is less than 2 houses/km². Assuming the national average of 2.6 persons per household, the average population density around GWEN RNs would be about 5 persons/km².

Using these values in Equations (11-3) and (11-4) yields an upper bound estimate of population risk for a single GWEN site of 3.6×10^{-6} excess cancer cases per year. During a 70-yr lifetime, the upper bound risk estimate would be 2.5×10^{-4} cases per site. For the full complement of 125 GWEN sites, the total upper bound estimate on the number of possible excess cancer cases is 0.03 over a 70-yr lifetime. It should be noted that the expected lifetime of the GWEN system is only 15 yrs, so that this upper bound risk estimate is probably high by a factor of about 5.

A completely analogous set of calculations can be made using the electric field as an exposure metric. In this case, Equation (11-3) becomes

$$R_g(r) < kR_o \gamma \delta E_g(r) (C + \epsilon)/(\sigma P^{1/2}) \quad (11-7)$$

Combining Equations (11-4) and (11-7) and using the same parameter values as those listed above yields an upper bound estimate for population risk at a single GWEN site of 5.4×10^{-5} cases per year. For a 70-yr lifetime and the full complement of 125 GWEN sites, the total upper bound estimate on the number of possible excess cancer cases is 0.5.

Similar arguments can be made for the UHF emissions from the GWEN system. Again, less than one excess cancer death is predicted for the entire population living in the proximity of GWEN UHF transmitters for 70 yrs. The estimated maximum increase in cancer risk associated with GWEN fields is therefore well below the detection capability of the epidemiological survey system.

LIMITATIONS OF GWEN RISK ASSESSMENT

The approach taken in estimating cancer risk from exposure to GWEN fields was based on a bounding argument using negative data from public exposure to fields of similar frequency emitted by broadcast stations. In this approach the committee has estimated the magnitude of risks from GWEN fields in a manner that is consistent with the observation that existing RF broadcast facilities have had no apparent effect on the cancer risk of surrounding populations. Because of the lack of positive data on cancer risk and the resulting uncertainties in the parameters used for our calculation, the true risk is likely to be smaller than the upper bound on risk derived by the approach taken here. Thus, while the committee has provided an upper bound estimate of the health risks from GWEN fields, it should be recognized that this value is likely to be an overestimate of the true risk and should be used in a policy sense only to exclude actions that might be warranted by risks greater than the upper bound estimated here.

RISK PERCEPTION

Science can provide evidence, albeit uncertain, on how large the potential risks related to GWEN facilities might be. But the acceptability of those risks is a value judgment, not a technical issue. Evidence from social psychology shows that the depth of public concern about a real or potential public-health risk depends on many factors other than morbidity and mortality. They include the plausibility, familiarity, and fear of the hazard; the population distribution of risks and benefits of the technology in question; the fairness of the process by which the risk is imposed; and the credibility of the group charged with managing the risk.¹⁷

A number of nonrisk factors might pertain to the perception of risks associated with the GWEN system:

- Recent studies of the public perception of the risks associated with nonionizing fields show that the lay public believes that health risks related to such fields are plausible.¹⁸

- Studies of the qualitative attributes of the risk related to ELF fields from highvoltage transmission lines show that, although people rank transmission lines as among the least risky of 16 common known or potential hazards, they nonetheless view transmission-line risks as less familiar and more dreaded than many other risks^{19, 20} and therefore perceive a greater need for regulation. If GWEN risks are similarly perceived, the public might view them as less acceptable than commonly accepted hazards that have higher actual risks.
- We do not know whether nonthermal levels of nonionizing radiation present a significant risk. If they do, however, it seems likely that the public-health impact of the GWEN system will be negligible, compared with that of UHF-TV and AM-radio broadcasts. But, because the acceptability of risks depends, in part, on the distribution of benefits from an activity,²¹ GWEN risks might be judged less acceptable than those related to broadcast sources. That is because urban populations exposed to UHF and AM broadcast signals derive direct benefits (news and entertainment) from those activities, whereas the risks associated with the GWEN system are encountered by the few dozen households in the vicinity of each site and the benefits of GWEN are both indirect and spread over the entire U.S. population.
- Data from polls conducted over the last 25 yr have shown a steady decline in the trust that the public places in government and in risk management institutions.^{22, 23} This lack of trust is likely to affect the public's perception of the safety of the GWEN system.

EXPOSURE REDUCTION

Available evidence precludes assurances that exposures to GWEN emissions are without risk. In fact, no amount of scientific research can guarantee such assurances for any agent. The goals of risk management are to identify rationales and fair processes for balancing residual risk against the costs of mitigative actions for reducing that risk.

Options for Reducing Exposures to GWEN Fields. A number of actions might be taken to reduce population exposures to GWEN LF and UHF emissions. They include choosing sites for the RNs that avoid people, placing the UHF transmitting antenna in each site so as to minimize UHF exposures of those living nearby, placing the UHF transmitting antennae on higher poles, increasing the size of each GWEN RN site to reduce field strengths at the site boundary, and reducing the number of RNs in the final GWEN system. Each of those has costs-some major, some minor.

Implications for Other Broadcast Activities. Because their risks and benefits are distributed differently, the public acceptability of risks from GWEN facilities is likely to be smaller than that from radio and television broadcasting. Taking steps to mitigate exposures from GWEN facilities need not imply, therefore, that mitigation should be applied to radio and television broadcasting as well.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

RESEARCH NEEDS

Given the extent of population exposure to broadcast sources in the United States, the lack of epidemiological data relevant to the question of RF exposure and human cancer is surprising. The economic costs surrounding delays in the siting and construction of GWEN and other new broadcast sources are difficult to quantify, but they undoubtedly far exceed the few millions of dollars per year that the United States currently spends on RF biological effects research. Additional federally supported RF-effects research would not only further our scientific understanding of the interactions of EMFs with biological systems, but also provide a basis on which the public could generate informed opinions concerning possible health risks associated with RF emitters.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

REFERENCES

1. Hattis, D., and D. Kennedy. 1986. Assessing risks for health hazards: an imperfect science. *Technology Review*, Vol. 89. Pp. 60-71.
2. National Research Council, Committee on the Institutional Means for Assessment of Risks to Public Health . 1983. *Risk Assessment in the Federal Government: Managing the Process*. Washington, D.C.: National Academy Press.
3. Bawin, S. M., and W. R. Adey. 1976. Sensitivity of calcium binding in cerebral tissue to weak environmental electric fields oscillating at low frequency. *Proc. Natl. Acad. Sci. (USA)* 73:1999-2003.
4. Blackman, C. F., et al. 1979. Induction of calcium-ion efflux from brain tissue by radio-frequency radiation: effects of modulation frequency and field strength. *Radio Science* 14(6S):93-98.
5. Schwartz, J., D. E. House, and G. A. R. Mealing. 1990. Exposure of frog hearts to CW or amplitude modulated VHF fields: selective efflux of calcium ions at 16 Hz. *Bioelectromagnetics* 11:349-358.
6. Durney, C. H., H. Massoudi, and M. F. Iskander. 1986. *Radiofrequency radiation dosimetry handbook*. Report USAFSAM-TR-85-73. University of Utah for the United States Air Force School of Aerospace Medicine.
7. Smith, A. A. 1978. Attenuation of electric and magnetic fields by buildings. *IEEE Transactions on Electromagnetic Compatibility* EMC-20:411-418.
8. United States Air Force (USAF). 1991. Procedures and data for the survey of housing density around GWEN sites are available from Lt. Col. Stephen T. Martin, Headquarters, Electronic Systems Division, Hanscom Air Force Base, Massachusetts.
9. United States Bureau of the Census. 1990. *Statistical Abstract of the United States: 110th edition*. Washington, D.C.
10. Hankin, N. N. 1986. The radiofrequency radiation environment: environmental exposure levels and RF radiation emitting sources. Report EPA 520/1-85-014, July. Washington, D.C.: U.S. Environmental Protection Agency.
11. World Radio and TV Handbook. 1987.

About this PDF file: This new digital representation of the original work has been recomposed from XML files created from the original paper book, not from the original typesetting files. Page breaks are true to the original; line lengths, word breaks, heading styles, and other typesetting-specific formatting, however, cannot be retained, and some typographic errors may have been accidentally inserted. Please use the print version of this publication as the authoritative version for attribution.

12. Taishoff, S. 1990. The Broadcasting Yearbook, S. Taishoff, ed. New York, NY: Broadcasting Publications Inc.

13. United States Coast Guard. 1981. Specification of the Transmitted Loran-C Signal. COMDTINST M16562. July 4.

14. United States Federal Communications Commission. 1991. Personal communication with M. Ferrante, Mobile Services Division, December 13.

15. Kraut, A., E. Chan, P. J. Liroy, F. B. Cohen, B. D. Goldstein, and P. J. Landrigan. 1991. Epidemiologic investigations of a cancer cluster in professional football players. *Environ. Res.* 56:131-143.

16. Tell, R. A., and W. Van Pelt. 1988. An investigation of radiofrequency field strengths in the vicinity of the meadowlands sports complex, East Rutherford, NJ. Prepared for the NJ Sports and Exposition Authority, July 29. Las Vegas, NV: R. E. Tell Associates.

17. Hohenemser, C., and R.E. Kasperon. 1982. Risk in the Technological Society. Boulder, CO: Westview Press.

18. Morgan, M. G., H. K. Florig, I. Nair, C. Cortes, K. Marsh, and K. Pavlosky. 1990. Lay understanding of low-frequency electric and magnetic fields. *Bioelectromagnetics* 11(4):313-335.

19. Slovic, P., B. Fischhoff, and S. Lichtenstein. 1980. Facts and fears: understanding perceived risk. Pp. 181-216 in *Societal Risk Assessment: How Safe is Safe Enough?* R. C. Schwing and W. A. Albers, Jr., eds. New York, NY: Plenum Press.

20. Morgan, M. G., P. Slovic, I. Nair, D. Geisler, D. MacGregor, B. Fischhoff, D. Lincoln, and K. Florig. 1985. Powerline frequency electric and magnetic fields: a pilot study of risk perception. *Risk Analysis* 5(2):139-149.

21. Slovic, P. 1987. Perception of risk. *Science* 236:280-285.

22. Lipset, S. M., and W. Schneider. 1983. The decline of confidence in American in American institutions. *Political Science Quarterly* 98 (3): 379-402.

23. Renn, D., and D. Levine. 1991. Credibility and trust in risk communication. Pp. 175-218 in *Communicating Risks to the Public*, R. E. Kasperon and P. J. M. Stallen, eds. Boston, MA: Kluwer Academic Publishers.