Effects of Microwave Radiation on the Lens of the Eye (1981)

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EFFECTS OF MICROWAVE RADIATION ON THE LENS OF THE EYE

Working Group 35
Committee on Vision
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National Research Council

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BACKGROUND

Working Group 35 of the Committee on Vision was formed in response to concern expressed by representatives to the Committee on Vision from several federal agencies about workers who may be exposed to microwave radiation. The Committee was requested to review the scientific literature concerning ocular effects of microwave radiation and to comment on the adequacy of existing studies, particularly in regard to the potential for ocular damage from microwave exposure below the current standard. The Department of the Air Force also requested comment on the desirability of routine screening for ocular changes in all its personnel working around microwave devices.

Potential hazards of human exposure to microwave radiation are of considerable concern because large numbers of people in the military services and in industry work in the vicinity of microwave generators and also because people may be exposed to radiation from devices such as microwave ovens and diathermy devices used in physical therapy. A recent report (National Research Council [NRC] 1979) reviewed the diverse biological effects produced by microwave radiation and discussed the problems of assessing the potential for health hazards from low levels of exposure. Cataractogenesis is the most clearly documented irreversible effect of overexposure to microwave radiation, but the mechanisms underlying this effect are poorly understood.

The current standard for maximum permissible occupational exposure to microwave radiation, recommended by the American National Standards Institute (ANSI), is 10 mW/cm^2. (The relevant parts of the ANSI standard are reproduced as Appendix A.) This report surveys existing studies of cataract induction by microwave radiation exposure and discusses the implications of this literature for human exposure at levels below 10 mW/cm^2. The report also suggests the kinds of study still needed. The report does not comment on the appropriateness of the existing standard, however, because such comment would have gone beyond the scope of this study. Standards for whole-body exposure are based on considerations of all possible biological effects and also on policy issues such as degree of acceptable risk, margin of safety, and cost-benefit considerations. (See Assenheim et al. 1979 for discussion of criteria for exposure standards.) Nevertheless, this review should be useful to those with responsibility for safety standards, particularly in regard to eye protection.

NATURE OF MICROWAVE CATARACTOGENESIS

Absorption of microwave radiation by biological tissues increases molecular kinetic energy and thus causes an increase in tissue temperature, depending on the thermal dissipative capacity of the tissue and the intensity and duration of exposure. Temperature increases of sufficient magnitude and duration may produce tissue damage. In contrast with ionizing radiation, which produces irreversible biological effects by disruption of molecular bonds, low-intensity microwave radiation does not have sufficient energy for such disruptions. Among ocular tissues the lens appears to be the most sensitive to microwave radiation. Although other tissues can be affected by microwave radiation, this report considers only effects on the lens. At sufficiently high intensities, microwave radiation has been demonstrated to induce formation of cataracts in experimental animals. (Cleary 1980, reproduced as Appendix B, provides detailed discussion and literature citation.) The exact mechanisms of microwave cataractogenesis are not well understood. Microwave cataract induction apparently involves thermal mechanisms; however, evidence from animal studies indicates that it is not a nonspecific thermal effect but rather depends on other undetermined factors probably related to the mode of absorption of microwave radiation in the mammalian eye (Appendix B).

Pulse modulation has not been shown to produce ocular effects different from those of continuous wave (cw) microwave fields; thus, most experimental studies have employed cw fields, and safety considerations have been based on time-averaged intensity (Appendix B).

Studies of time-intensity relationships for acute exposures suggest that ocular effects of microwave radiation are threshold phenomena related to total absorbed energy, which is consistent with a thermal mechanism (Appendix B). For rabbits, with acute exposure there is an apparent temperature threshold of about 41°C for lens opacification, and the degree of the effect depends on how long the tissue temperature is elevated (Appendix B). For low-intensity fields, temperatures may not reach 41°C. For acute exposures, field intensities of greater than 100 mW/cm^2, lasting more than 1 hour, were required for lens opacification. Repeated exposures below the threshold for a single exposure have been shown to induce cataracts, but this has not been demonstrated below 100 mW/cm^2 under the limited number of exposure durations and fractionation regimes investigated to date.

ESTIMATING HUMAN THRESHOLDS

A number of individual cases of cataract in humans have been attributed to microwave exposure. Unfortunately, documentation of exposure history (field intensity, wavelength, duration, and geometry, etc.) has often been inadequate, but in all cases in which field intensity was known it was well in excess of 100 mW/cm^2 (Appendix B;

NRC 1979:72-74). The lack of adequate information has precluded establishing a threshold for induction of human cataracts; however, the available data suggest rough comparability of sensitivity in rabbits and humans (Appendix B).

A number of retrospective surveys of human workers who may have been exposed to microwave radiation have been conducted (Appendix B). Most of these studies have not revealed any evidence of cataract occurrence attributable to microwave exposure, but the data on exposure in such studies are too limited to make inferences about thresholds for effects in humans.

Even if good human exposure data were available, estimating thresholds would be difficult. Biological effects depend, among other things, on the amount of microwave energy absorbed, and absorption depends on radiation field wavelength and polarization, geometry of the exposed tissue, and proximity of nearby structures, in addition to field intensity (NRC 1979:45-54). Considerable differences have been noted among species. In general, extrapolation from one species to another is complicated both by differences in absorption and possible biological variations.

The current ANSI standard of 10 mW/cm^2 was established on the basis of thermal considerations. The basal metabolic rate for the human body is about 1 mW/g, and during exercise the body dissipates heat at rates in the 1-10 mW/g range. In the frequency range of 10-10,000 MHz, field intensities of more than 1 mW/cm^2 would be required for absorption of 1 mW/g of tissue (NRC 1979:43). Thus it was assumed that below about 10 mW/cm^2 the thermal effects of microwave radiation should be no greater than those experienced by body tissues during exercise.

On the basis of observations that some biological effects, particularly neuronal and behavioral responses, occur at low-field intensities, some eastern European countries have established guidelines for acceptable microwave exposure of less than 10 mW/cm^2. These effects, however, are generally transient and have not been shown to harm health (NRC 1979:6,62). At this time, the American National Standards Institute is considering lowering the 10 mW/cm^2 standard, particularly in frequency bands in which tissue absorption is high.

FINDINGS AND CONCLUSIONS

1. Existing evidence does not suggest that microwave fields of less than 10 mW/cm^2 can induce cataracts.

Fields of intensities greater than 10 mW/cm^2 are required to induce cataracts in animals. No evidence for cataract induction below 10 mW/cm^2 has been found in human studies, although the available

data are insufficient to establish human thresholds. The effects of long-term occupational exposure to low-level microwave radiation have not been studied adequately. Although there is currently no evidence that long-term human exposure to field intensities around 10 mW/cm^2 can induce cataracts, that possibility cannot be unequivocally excluded on the basis of existing knowledge.

2. Existing evidence does not suggest that routine screening for cataracts of all Air Force personnel working in the vicinity of microwave devices is justified at this time.

Clinical surveys of large numbers of personnel occupationally exposed to microwave fields have yielded no evidence of increases in ocular defects attributable to microwave exposure. Thus there is no evidential basis for recommending mass screening. If feasible, a well-designed prospective study might be useful, as discussed below.

3. More research is needed to characterize the effects of low-level microwave radiation, particularly for chronic exposure.

Laboratory research is required to elucidate the mechanisms of ocular damage from microwave exposure. The effects of microwave fields on biological molecules and cellular systems need to be understood better in order to clarify the mechanisms of organ damage. The possibility of delayed or late-occurring biological effects of both acute and chronic low-level exposure particularly needs to be investigated with experimental animal models.

Better dosimetry methods need to be developed for measuring fields and temperatures in tissue. Experimental data should include absorbed radiation dose as well as the incident exposure and information on wavelength, modulation, polarization, and anatomical configurations. Quantitative, objective indices of lens changes (e.g., by use of a nepthelometer) need to be developed, and findings should be expressed in terms commonly understood among the several technical disciplines concerned with microwave radiation exposure. The effects of varying the orientation of linearly polarized radiation with respect to the eye and surrounding structures need to be studied.

Experimental study of the heat transfer characteristics of the human eye and theoretical models are needed in order to develop better approaches to scaling data on microwave effects from animal eyes to human eyes.

Epidemiological studies, although difficult, are an important source of information about effects of microwave radiation on humans. There have been several retrospective studies of microwave effects, and they do not suggest increased risk of cataracts from occupational exposure to fields below 10 mW/cm^2. It is, however, difficult in such studies to obtain adequate records of individuals' radiation exposure histories, and finding appropriate control groups is

problematic. An additional source of data for retrospective study might be the scientists exposed to various levels of nonionizing radiation for considerable periods during World War II. Among the groups that might be studied are workers at the M.I.T. Radiation Laboratory (see Norman 1979), at the Army Signal Corps Laboratories at Fort Monmouth, and at industrial laboratories involved in the development of radar.

A well-designed prospective study, with careful matching of test and control groups, could provide valuable information about possible effects on humans of low-level microwave exposure. However, such a study, if adequately designed, would be expensive. Since the main source of subjects for such a study would presumably be workers whose microwave exposure would be limited to less than 10 mW/cm^2, the study designers would need to think carefully about the number of subjects that would be required to reveal any low-incidence effects.

In any kind of study of microwave effects on humans it is important to include information about exposure, including wavelength, polarization, modulation, etc. as well as field intensities, dosimetry, and anatomical configuration. Characteristics and configuration of nearby objects, such as spectacles and helmets and other reflecting surfaces, may also be important. The subjects' thermo-regulatory responses should also be considered.

4. A study of the use of microwave diathermy devices might be useful.

Apparatus for therapeutic heating by microwave irradiation has been readily available since shortly after World War II. It has been used both under medical supervision and in gymnasiums and training rooms for amateur and professional athletes without medical supervision. To produce an apparently beneficial effect, microwave energy that may be considerably greater than 10 mW/cm^2 may be applied locally to various parts of the body. Professional athletes may receive prolonged and frequent exposures over a period of years and thus might provide a useful population for study. It should be noted that diathermy exposure is local rather than whole-body, and that the eyes would be exposed only indirectly; furthermore, there are not likely to be good records of individuals' exposures. An investigation of the use of microwave diathermy devices might provide useful information about the effects of local applications of relatively high microwave fields on the human body. Since it is also not clear whether the possibility of deleterious effects of chronic use of microwave diathermy devices has been adequately examined, such a study might be useful for health and safety reasons.

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APPENDIX A

AMERICAN NATIONAL STANDARD C95.1-1974

The following excerpts from ANSI C95.1-1974, "Safety Level of Electromagnetic Radiation with Respect to Personnel," were approved by the American National Standards Institute on November 15, 1974, and published by the Institute of Electrical and Electronics Engineers.

SECTION 3. RECOMMENDATIONS (p.7)

For normal environmental conditions and for incident electromagnetic energy of frequencies from 10 MHz to 100 GHz, the cw radiation protection guide is 10 mW/cm^2 and the equivalent free-space electric and magnetic field strengths are approximately 200 V/m rms and 0.5 A/m rms, respectively. For modulated fields, the power density and the squares of the field strengths are averaged over any 0.1 hour period; that is, none of the following should be exceeded, as averaged over any 0.1 hour period:

Mean Squared Electric Field Strength	- 40 000 V^2/m^2
Mean Squared Magnetic Field Strength	- 0.25 A^2/m^2
Power Density	- 10 mW/cm^2
Energy Density	- 1 mWh/cm^2

SECTION 5. WHOLE BODY IRRADIATION AND PARTIAL BODY IRRADIATION (p.8)

These formulated recommendations pertain to both whole body irradiation and partial body irradiation. Partial body irradiation must be included since it has been shown that some parts of the human body (for example, the eyes or testicles) may be harmed if exposed to incident radiation levels significantly in excess of the recommended levels.

Microwave Cataractogenesis

STEPHEN F. CLEARY

Abstract—Experimental investigations of microwave cataractogenesis, as well as the application of theoretical methods, suggest the involvement of thermal damage. Time-intensity cataract thresholds for acute exposures of rabbits indicate dose reciprocity. The induction of lens opacification following repeated exposure at intensities below the threshold for single-dose exposures suggests a cumulative component of lens damage and the existence of repair mechanisms. Repair mechanisms are also indicated in experimental biochemical studies of microwave effects on rabbit lens epithelial cells with a 10–20-day cellular recovery period. Experimental studies have revealed a relationship between the site of ocular damage and radiation wavelength. Cataract induction has also been reported in humans accidentally overexposed to microwave radiation. Although dosimetric data is not adequate to specify exposure thresholds, acute lens opacification in humans appears to involve thermally induced lens damage that occurs at exposure intensities of 100 mW/cm^2 or greater. Epidemiological studies of workers have in some instances suggested that occupational microwave exposure may result in lens alterations but there is no evidence that such effects are associated with visual impairment or cataract formation.

Introduction

RESEARCH conducted during the past two decades has revealed that exposure of biological systems to microwave and radio-frequency (RF) radiation results in a wide variety of effects depending upon the field intensity, exposure duration, and other exposure parameters. Other than lethality due to acute overexposure, cataract induction is the only apparent irreversible effect of such exposure, a fact that has been known or expected since the early days of microwave bioeffect research. Cataractogenesis appears to be the only documented form of human morbidity known or suspected to result from microwave exposure. Microwave cataractogenesis, probably the most extensively investigated exposure effect, has been the subject of numerous experimental as well as epidemiological studies. In reviewing the biological effects of electromagnetic (EM) energy, it is thus natural to consider the topic of microwave cataractogenesis as a central issue and to examine in detail the adequacy of the data in this area since it should represent the most complete available information regarding the effect of EM radiation on mammalian systems. The purpose of this review is to describe the current state of knowledge in this area and to consider the adequacy of the data on microwave cataractogenesis.

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Experimental Microwave Cataractogenesis

Microwave radiation has been shown to induce damage in ocular tissues, the site of damage depending upon the wavelength of the radiation and the mode of exposure. The extent of damage is primarily dependent upon the radiation intensity or power density and total exposure duration. In the frequency range from 1 to 10 GHz the lens appears to be the ocular tissue of greatest sensitivity due to the absorption characteristics of the mammalian eye, the mitotic and metabolic status of lens fibers, and the relative avascularity of the lens which predisposes it to thermal damage. The lens fibers, which are formed from postmitotic cells of limited metabolic capacity, appear to be subject to a relatively greater extent of irreversible damage than most other tissues. The apparent sensitivity of lens tissue to microwave and other types of radiation, such as ionizing radiation, may be related to the fact that lens opacification is readily observable due to the transparency of the lens and other preretinal ocular structures. Opacification of lens fibers results from any physical, chemical or metabolic stress that alters the paracrystalline state of lens proteins, but the basic mechanisms are not well understood. Opacification sufficient to cause loss of visual function is generally referred to as a cataract.

Acute high-intensity microwave exposure of rabbits can cause immediate tearing, injection, pupillary constriction, and anterior chamber turbidity, the extent of which is dependent upon the field intensity and exposure duration [1]. The latency for the development of lens changes detectable by slit-lamp biomicroscopy is inversely related to the microwave intensity. Typically, following cataractogenic exposures to 2.45-GHz fields in the range of 100 to 300 mW/cm^2, lens changes are first seen 24 to 48 h postexposure, a significantly shorter latency than encountered in cataract induction following exposure to ionizing radiation. In the lower end of this intensity range, reversible minor degrees of change are induced consisting of a milky band in the posterior lens cortex immediately adjacent to the posterior capsule that extends to the equatorial zone, as well as a chain of vacuoles or small vesicles in the posterior suture region. Exposure to higher intensity fields results in more pronounced and permanent changes including denser banding, increased vacuolization, and well-defined circumscribed opacities in the posterior lens cortex

detectable with an ophthalmoscope [1]. Large vesicles may appear at the lens equator and in some instance posterior cortical opacities are encountered that involve the lens equator as well as the anterior subcapsular cortex. In general it has been found that microwave cataracts in the rabbit involve only the posterior lens cortex, except following very high intensity exposures where opacification extends throughout the entire lens substance [1].

The induction of cataracts in experimental animals, principally New Zealand white rabbits, has been described by a number of investigators using a variety of microwave exposure modalities [1]-[3]. Generally microwave field intensities necessary for cataract induction in the rabbit are such that acute whole body exposure would be lethal due to hyperthermia. Most cataract studies have, therefore, employed focused or near-zone fields which limit exposure to the head or eye. Localized thermal trauma is still of such a magnitude to necessitate the use of general or local anesthesia. Corneal irrigation with physiological saline solutions has also been used to prevent corneal damage due to tissue dehydration during microwave exposure. Anesthesia and corneal irrigation, as well as air temperature and humidity, may significantly affect the temperature of ocular structures [4]. If it is assumed that cataract induction is a thermal phenomenon, such factors complicate the interpretation and intercomparison of the results of experimental microwave cataract studies, and introduce uncertainty in the extrapolation to microwave cataract induction in humans. Applying the results of animal experimentation to cataract induction in human beings is also complicated due to size differences in animal and human heads and marked anatomical differences in the relative location of the eye in the skull. At a frequency of 2.45-GHz, Carpenter [3] has reported that the measured microwave intensity at the position of the head of a rabbit is reduced by 40 percent, due to field perturbation by the animal. A further reduction in field intensity of 40 percent was detected when the animal's ears were fastened against its back. Comparisons of cataract thresholds in rabbits and rhesus monkeys using a 2.45-GHz cavity-backed resonant slot radiator has been reported by Kramer *et al.* [5]. Cataracts were induced in rabbits exposed for 140 min to incident power levels of 180 mW/cm^2, whereas monkeys sustained facial burns but no lens damage at incident power levels of up to 500 mW/cm^2. The marked variation in cataract thresholds was attributed to differences in the anatomical configuration of the ocular structures [5].

The effect of radiation frequency, field polarization, and orientation have not been determined for microwave energy distribution in the eyes of human beings exposed to microwave fields. Since EM wave absorption in a lossy dielectric scatterer, such as the mammalian head, is a function if its shape and dimensions, the heating potential of a given frequency of microwave radiation differs significantly both with respect to location and magnitude for an experimental animal as compared to man at microwave frequencies greater than 500 MHz. It is not possible to experimentally determine microwave heating patterns in the human eye; consequently, theoretical methods must be used to simulate ocular tissue geometry and thermodynamics to estimate ocular heating patterns [6]. Theoretical determinations of microwave-induced heating patterns in human [6] and rabbit ocular structures [7], [8] have been attempted but limitations on computer methodology and/or mathematical complexities have limited the usefulness of such results in the comparison of human and rabbit ocular microwave heating patterns. Theoretical calculations of microwave heating patterns in the rabbit eye have, however, predicted frequency-dependent temperature distributions that are in agreement with experimental determinations [8]. Frequency-dependent microwave heating patterns in the rabbit are based upon resonances of the eye-scatterer and the head. At frequencies of less than approximately 1.5 GHz the dimensions of the eye and orbit are not large enough to result in field concentration effects. Peaking of microwave absorption in this frequency range would thus have to depend on whole-head resonance. At frequencies greater than 1.5 GHz the eye-scatterer can theoretically cause resonant absorption independent of field concentration effects of the whole head. Heating potential peaks may thus occur within rabbit ocular tissues at frequencies greater than 1.5 GHz [6]. The application of data derived from animal studies of microwave-induced ocular damage to the assessment of human cataract risks from microwave exposure must take into account the frequency-dependent resonance phenomena which are not presently well understood for the human eye.

The effects of acute exposure of the eyes of experimental animals, predominantly rabbits, are dependent upon microwave frequency, intensity, and exposure duration. Pulse-modulated microwave fields have not been shown to produce qualitatively or quantitatively different ocular effects compared to continuous wave (CW) fields of equal intensities, which suggests that such effects do not depend upon the magnitude of the instantaneous absorbed power or electric field strength [9]. Time-intensity relationships for the induction of cataracts in rabbits have been determined for microwave radiation frequencies of 2.45, 5.4, 5.5, 8.2, and 10 GHz [1], [10]-[13]. These experiments, conducted in the near field with the animals under anesthesia, employed various field measurement methods which preciudes a quantitative comparison of the data. In these acute exposure studies the minimum exposure time required to induce acute lens opacification (i.e., changes detectable within the first few days postexposure) was determined at a given microwave intensity. Although quantitative comparisons are not possible, in those studies where similar dosimetric methods were used there was a high degree of concordance in the time-intensity opacification relationships [1]. The hyperbolic form of the time-intensity relationships indicates dose reciprocity, suggesting that lens opacification in the rabbit, for single acute exposures, is a threshold phenomenon related to the total absorbed energy and indirectly suggestive of a thermal exposure effect.

The effects of multiple exposures to subthreshold intensities of 2.45-GHz microwaves repeated at regular intervals have also been investigated in an anechoic chamber in the near field of a dipole antenna [3]. Opacification was detected in one of eleven rabbits exposed 20 to 24 times for 1 h to an 80-mW/cm^2 field, whereas 4 opacities were produced in a group of 10 animals similarly exposed at 100 mW/cm^2 [3]. Repeated exposures at 120 mW/cm^2 resulted in opacities in 8 out of 10 rabbits, in spite of the fact that a single 1-h exposure at this intensity produced no opacities. Small central opacities were induced at 120 mW/cm^2 when the irradiation time was increased to 4 or 4.5 h [3]. The animals were not anesthetized except in the case of exposures lasting for up to 5.5 h. Carpenter *et al.* [3] indicate that there is uncertainty in the power densities in these experiments due to the measurement techniques used for the near-field microwave source. Subsequent revaluation indicate that the intensity was 180 mW/cm^2 rather than 120 mW/cm^2 for the 4- or 4.5-h exposures [52].

The majority of the investigations of experimental microwave cataractogenesis have involved single or multiple subthreshold exposures in the intensity range of 80-500 mW/cm^2. In a study of chronic low-intensity exposure effects, Ferri and Hagen [14] exposes 6 unanesthetized rabbits to 2.45-GHz-CW radiation in the far field at an intensity of 10 ± 3 mW/cm^2 for 8 h/day, 5 consecutive days a week, for

periods of from 8 to 17 weeks. Six months of observation did not reveal any abnormal ocular changes in the exposed animals [14].

Time-intensity relationships for cataract induction in the rabbit, as well as experimental studies of microwave-induced ocular heating, suggest the involvement of thermal damage to lens tissue. In the case of single acute near-field exposure of rabbits, there is an apparent temperature threshold of approximately 41°C for lens opacification, the effect being dependent upon the duration of the temperature elevation. Repeated exposures at intensities that apparently do not induce tissue temperature elevations of this magnitude have, however, been shown to induce opacification depending upon the duration and number of exposures, which suggests a component of microwave-induced lens damage dependent upon the cumulative time-temperature history of the exposed tissue [15]. Comparison of the effects of lens temperature elevation by other heating modalities indicates that microwave radiation induces a unique type of thermal stress in the mammalian lens.

The involvement of lens heating in cataractogenesis has been investigated by altering ocular temperatures by whole-body or localized hyper- or hypothermia. Whole-body hypothermia of dogs was used to limit the intra-ocular temperature rise during exposure to cataractogenic 2.5-GHz microwave fields. Cataracts were induced in normothermic eyes but not in the eyes of animals subjected to whole-body hypothermia at 22°C, leading the investigator to conclude that microwave cataractogenesis was directly or indirectly a thermal phenomenon that occurs when the intra-ocular temperature exceeds 43°C [16]. Similar conclusions were derived from studies of whole-body hypothermia of rabbits in which case 2.45-GHz microwave-induced intra-ocular heating was limited to less than 41°C and no lens opacification was detected [17]. The hypothesis that lens opacification was solely due to heating was tested by subjecting rabbits to localized or whole-body hyperthermia sufficient to raise the retrolental temperature to 42°C for a period of 30 min or more. No detectable lens opacification resulted from such treatment in spite of the fact that the intra-ocular temperature was similar to that known to produce cataracts from microwave exposure [18]. The effects of localized lens heating and the joint effect of localized heating and microwave exposure at subthreshold cataract intensities have also been investigated [19]. Localized heating of the rabbit eye at the same rate, to the same temperature, and for durations equal to those experienced during cataractogenic microwave exposures did not produce cataracts with one exception that was attributed to severe uveitis rather than a direct effect on the lens [19]. Cataracts were not detected during a 10-day postexposure period in 5 rabbit eyes heated to 49°C for 30 min [19]. In other experiments, rabbit body temperatures were increased by raising the environmental temperature of the ears in conjunction with microwave exposure at an intensity sufficient to increase the retrolental temperature to a level normally associated with cataract induction. In 3 of the 10 animals, opacities were induced in the posterior cortex of the lens, and in two instances in which lens opacification results, the microwave doses were 90 and 91 percent of the 40-min microwave cataractogenic dose. In the third case, the eye exposed to 76 percent of the cataractogenic dose developed opacities in the posterior subcapsular cortex of the lens 4 days postexposure [19]. The results of these experiments indicate that microwave cataractogenesis is not a nonspecific thermal effect, but is dependent upon other undetermined factors which appear to be related to the mode of absorption of microwave radiation in the mammalian eye.

Experimental studies of exposure to EM radiation in the frequency range of from 0.385 to 107 GHz have revealed qualitative frequency-dependent differences in the effects on ocular tissues. Of those frequencies that have been investigated to date, lens opacities have been induced by radiations in the range of from 0.8 to 10 GHz. The majority of the studies in this frequency range have been at 2.45 GHz [1], [3], [12], [13], [20]–[24], but opacities have been reported to result from exposure at 800 MHz [10]; 2.8 GHz [25]; 3 GHz [2], [26]; 4.2, 4.6, 5.2, 5.4, 5.5, and 6.3 GHz [10]; 5.5 GHz [9]; 8.23 and 9.37 GHz [27]; and 10 GHz [12], [27], [28].

Differences in the experimental procedures such as the mode of application of the microwave energy and the field intensity measurement techniques preclude a comparison of the relative cataractogenic potential of these radiation frequencies with the exception of the series of experiments reported by Birenbaum *et al.* [10], in which a dielectric waveguide insert adapter was used to apply various frequencies of pulse-modulated microwaves to the corneal surface of the eye. The field strength for the various frequencies in the range of from 4.2 to 6.3 GHz was held constant while exposure time was varied to establish cataract thresholds. An inverse relationship was found between the microwave frequency and the exposure time. As the radiation frequency was decreased, longer exposure times were required for lens opacification. An alternative interpretation of these data is that for increasing frequency in this range, the field intensity required to produce opacification decreases. In these experiments the opacities developed in the anterior cortex of the lens, in contrast to opacification of the posterior subcapsular lens cortex when the microwave energy was not applied directly to the corneal surface but instead across an air space, thus cataract thresholds may have been dependent upon the mode of application of the microwave field which may have affected the intra-ocular energy absorption patterns.

Hagen and Carpenter [29] determined the relative cataractogenic effects of 2.45 and 10-GHz microwave fields using dielectric lenses to concentrate the radiation in the eye region of rabbits. They concluded that when the rabbit eye is subjected to a single acute exposure, the cataractogenic potential decreases as the frequency is increased from 2.5 to 10 GHz [29]. Temperature measurements revealed that the aqueous humor of the anterior chamber (anterior to the lens) was heated to a greater extent at 10 GHz than at the lower frequency where the maximum temperature elevation occurred in the vitreous body posterior to the lens [29], results that agree qualitatively with theoretical predictions based upon the inverse relationship between microwave frequency and penetration depth in lossy dielectric material.

An inverse relationship between microwave frequency and penetration depth has been demonstrated in an experimental study of the ocular effects of 35- and 107-GHz microwaves on the rabbit eye [30]. The effects of acute exposure were limited to the corneal stroma, suggesting that maximum field absorption and heating occurred in the superficial regions of the eye. The 107-GHz radiation was more effective in producing immediate stromal damage to the cornea, but repair occurred within 24 h postexposure, in contrast to the effects of 35-GHz radiation which had greater persistence and were associated with significant degrees of epithelial damage [30]. Keratitis was induced at lower field intensities for both frequencies than levels that produced other ocular effects such as iritis or lens opacification [30]. The experimental methods employed in this investigation do not permit a direct comparison of microwave intensity thresholds for corneal damage with cataract thresholds at lower frequencies, but the intensities and exposure times appear to be of the same order of magnitude.

No ocular effects were detected in experiments conducted at 385 and 468 MHz [31] which is consistent with the hypothesis that ocular damage is dependent upon localized energy deposition which is determined by the physical characteristics (i.e., dimensions and shapes) of ocular tissues and the wavelength of the radiation.

The etiology of microwave cataracts has been studied biochemically. A 20-percent reduction in ascorbic acid concentrations was detected in rabbit lenses 6–18 h postexposure to microwave radiation [32]. The finding that cultured rabbit lenses lost ascorbic acid in an intensity-dependent fashion was interpreted as a thermally induced exposure effect [33]. Lenses maintained in culture media were exposed to either pulsed or CW *S* band radiation for 10 to 15 min at intensities of up to 200 mW/cm^2. Lens ascorbic acid content was measured 1–3 days postexposure. Control lenses exposed to the same time-temperature conditions as microwave irradiated lenses exhibited the same decrease in ascorbic acid as irradiated lenses. No difference was detected in ascorbic acid levels in lenses exposed to a given average microwave intensity by either pulse-modulated or CW radiation. The ascorbic acid concentration in the lens is thus suggested to be a sensitive index of lens injury, although the decrease may not be a primary event in microwave cataractogenesis [33].

The effects of single and multiple microwave exposures on ascorbic acid levels in the vitreous, lens, and aqueous humor, at postexposure times ranging from 5 min to 4 weeks have also been investigated [34]. Ascorbic acid concentrations were unaltered, with the exception of a 20-percent decrease 1 week postexposure, at which time lens opacification appeared [34]. Ascorbate levels in the vitreous humor were unchanged, but the levels in the aqueous humor decreased continuously for up to 1 week following acute exposures, followed by increasing levels which reached normal values 2 weeks postexposure. Ferri [35], in an attempt to relate biochemical alterations to the mechanism of lens opacification, studied the relative effects of microwave exposure and hyperthermia on ascorbic acid levels in the aqueous humor under *in vivo* and *in vitro* conditions. Heating of the aqueous humor without microwaves increased the ascorbate levels, in contrast to decreased levels which resulted from microwave exposure. The hypothesis that heat alone caused reduced levels of ascorbate which subsequently led to reductions in ascorbate in the lens was thus rejected and it was suggested that cataractogenesis is a secondary reaction and that the primary initiating or damaging effect of microwave exposure may not occur at the lens but at some other sensitive site which culminates in lens opacification [35].

Cataractogenic doses of 2.45-GHz CW-microwave radiation have also been shown to alter the mitotic activity of the lens epithelium of the rabbit [36]. By the use of autoradiography, Van Ummersen and Cogan detected two patterns of response as defined by the presence or absence of vesicular strings in the exposed lenses. When vesicular strings were not induced there was an initial pronounced suppression of mitotic activity followed by a gradual return to normal levels 20 days postexposure. In lenses with vesicular strings there was a precipitous transient increase in mitotic activity on the fourth to fifth day after exposure with a return to normal levels by 30 days postexposure. The vesicles, which apparently represented lens hydration, caused accelerated proliferation of the overlying epithelial cells. If equatorial vesicles did not develop, the mitotic activity of the lens epithelium was similar to that observed after exposure to ionizing radiation. The earliest effect of cataractogenic doses of microwaves on the rabbit lens epithelium was inhibition of DNA synthesis and mitosis 6- to 24-h postexposure. Forty-eight hours after exposure the level of DNA synthesis and mitotic activity showed some recovery of the epithelial cells. The first visually observable change in the lens was granular aggregates in the posterior lens cortex near the lens equator [36]. The results of this study suggest that microwave cataractogenesis involves alteration in lens epithelial cells. The transient nature of the effects may be interpreted as suggesting a recovery period of from 10 to 20 days during which time cellular damage is repaired. Transient cellular alteration following microwave exposure is consistent with experimental studies of cataract induction by repeated exposure of rabbits to subthreshold doses in which case cataract induction was found to depend upon the interval between exposures [12].

Electron microscopic analysis of lenses exposed to cataractogenic doses of *S*-band microwave radiation detected marked cellular deformation in the immediate subcapsular region of the lens. The cells were swollen and vacuolated, and many contained coarsely granular and clumped cytoplasm [37]. The only detectable abnormality in some cases involved the cell membrane of the interdigitating processes which was electron dense and irregular with some evidence of membrane reduplication and degeneration. The lenses of a group of rabbits exposed to 165 mW/cm^2, 20 min/day, twice daily, 5 day/week for a total of 36 exposures appeared normal by slit-lamp examination 5 days postexposure. However, morphological alterations were detectable by electron microscopy. The fibers of the posterior subcapsular cortex were enlarged and contained large intercellular clefts and the outermost cortical equatorial lens fibers were found to be extensively altered. In this region, cells containing several cysts or vacuoles and surrounded by thickened and fragmented membranes were also found, suggesting organelle destruction [37]. Cataractogenic microwave exposure thus results in extensive membrane damage in lens fibers, and less severe but detectable ultrastructural damage is induced by exposures that do not result in lens damage detectable by slit-lamp biomicroscopy.

Microwave Cataracts in Humans

Over 50 cases of human cataract induction has been attributed to microwave exposures, primarily encountered in occupational situations involving acute exposure to presumably relatively high intensity fields. Generally it has not been possible to obtain sufficient information to determine, with any degree of certainty, human cataract thresholds and the inadequacies in the data in some cases casts serious doubts about the validity of some of the reported incidents of microwave cataract induction.

Microwave cataractogenesis was first reported in 1952 but no details of the exposure were indicated [38]. Hirsch and Parker [39] reported bilateral cataract induction in a 32-year-old microwave worker who had been intermittently exposed during a period of one year to radiation in the frequency range originally reported to be from 0.2 to 3 GHz. Exposures of various durations to fields of 100 mW/cm^2 or greater led to the development of bilateral posterior subcapsular and nuclear cataracts in addition to the appearance of cellular debris in the aqueous and vitreous humors, vitreous opacities, and choroiditis in the left eye [39]. Upon subsequent reexamination, after the lens of the left eye had been surgically extracted, a persistent degree of uveitus and chorioretinitus was detected in the left eye and an apparently nonprogressive cataract in the lens of the right eye [40]. In this later report, the exposure frequencies were indicated to be in the range of from 4 to 5 GHz at microwave intensities of from 40 to 380 mW/cm^2, with possible near-field exposures at intensities as high as 1.16 W/cm^2. Exposures leading to cataract formation appeared to have occurred primarily during a three-day period immediately

preceding the onset of symptoms, suggesting a short latency for cataract induction in humans exposed to high intensity microwave fields [40].

Shimkhovich and Shilyaev reported the development of bilateral cataracts in a 1-month period in a 22-year-old technician exposed approximately five times to 3-GHz radiation at an estimated intensity of 300 mW/cm^2 for 3 min/exposure [41]. Zaret *et al.* [42] reported 42 cases of microwave cataracts resulting from occupational exposure. The posterior lens capsule was reported to be the initial site of lens pathology in some of the patients in this group. Microwave exposure parameters were available in only one case where the exposures were for durations of approximately 50 h/month over a 4-year period at intensities of less than 10 mW/cm^2 in most instances, but with 1 period of 6 months or more during which the average power density was approximately 1 W/cm^2 [42]. Cataract induction in a 50-year-old female which was attributed to intermittent exposure to radiation from a 2.45-GHz microwave oven has also been reported [43]. The exposures, which presumably occurred over a period of approximately 6 years prior to the detection of lens opacification, were from leakage radiation of approximately 1 mW/cm^2 during oven operation with levels of up to 90 mW/cm^2 when the oven door was opened [43]. The exposure conditions and clinical manifestations of this case have generated significant controversy concerning the involvement of microwave exposure. Bouchat [43], however, has indicated that the clinical observations reported by Zaret correspond to a case previously reported by Bouchat and Marsol, in which case the induction of capsular cataracts was attributed to microwave exposure of a 23-year-old microwave worker exposed over a period of 44 months [44].

Data on microwave-induced cataracts in humans resulting from acute high-intensity exposure suggest the mechanism of thermal damage. To the extent that such comparisons may be made, the estimated acute microwave cataract dose in humans is in reasonable agreement with data derived from experimental irradiations of rabbits, suggesting at least to a first approximation, similar sensitivity of rabbits and human beings to microwave cataract induction. It may further be conjectured that the intensity–exposure time relationships are qualitatively similar, indicating that a threshold intensity for human cataract induction exists for acute single microwave exposure. Repeated subthreshold exposure of rabbits results in lens opacification which is indicative of a cumulative effect upon lens tissue of undetermined time dependency. Consequently, it is not possible to specify an intensity–time threshold for cataract induction in human beings repeatedly exposed to microwaves [45].

The ocular effects of occupational microwave exposure have been the subject of a number of epidemiological studies. A retrospective study of minor or noncataractous lens changes in military and industrial microwave workers revealed an apparent exposure-related increased incidence as compared to an age-matched control group [46]. The most statistically significant finding was an increased incidence of minor types of defects in the posterior pole of the lenses of the microwave workers, which appeared to be related to job specialization. Individuals employed in research and development had a higher incidence of such changes than those in other microwave work categories. Although the incidence of noncataractous changes was correlated with several types of microwave exposure parameters, the most significant correlation of lens defects in both microwave workers and controls was with the age of the individual, such that a highly statistically significant age-dependent linear increase in the mean number of defects was found [46]. The results of this study were interpreted as suggesting that occupational microwave exposure increased the rate of aging of the lens, but since cataract incidence was not investigated, relationships were not drawn between the incidence of such lens defects and cataract induction [46].

The effect of employment in microwave occupations on cataract induction was investigated in a retrospective study of 3000 military personnel with cataracts and a control group of 2000 cataract-free military personnel. There was no evidence that military occupational microwave employment was related to an increased risk of cataract induction. These results were interpreted to suggest that if exposures are limited to less than 10 mW/cm^2 the risk of cataracts from military microwave exposure was not detectably increased [47].

There was no indication of lens damage attributable to occupational microwave exposure in a five-year survey of microwave workers in which the lenses of potentially exposed workers and controls were examined biomicroscopically for lens defects [48]. In another investigation, the incidence of lenticular opacities was determined in 841 microwave workers with potential occupational exposures of various durations. The microwave worker population was subdivided into two groups consisting of 507 individuals potentially exposed to mean microwave intensities in the range of from 0.2 to 6 mW/cm^2, and a group of 334 workers potentially exposed to mean intensities of less than 0.2 mW/cm^2 [49]. Intergroup analyses of the incidence of lenticular opacities, as well as intragroup comparisons based on age and duration of microwave employment, indicated that the incidence of lenticular opacities was statistically significantly correlated with age, but not with exposure level or duration of microwave employment [49].

Double-blind ophthalmological examinations of 447 military microwave workers and 340 controls with no known history of occupational microwave exposure were conducted by Shacklett *et al.* [50] to determine the relative incidence of lens opacities, lens vacuoles, and posterior subcapsular iridescence; lens changes that were considered to indicate early stages of cataract formation. No statistically significant differences were detected in the incidence of any of the types of lens changes between the microwave workers and controls [50]. Ocular examinations of 705 microwave workers revealed no lenticular or retinal defects that could be attributed to microwave exposure in the most recently reported study of the effects of occupational microwave exposure on the eye [51].

The results of epidemiological studies of the relationship of occupational microwave exposure to ocular changes do not provide evidence of deleterious effects. If it is assumed that human exposures in such environments are limited to less than 10 mW/cm^2, this intensity may be suggested as a practical limit for ocular damage from intermittent exposure to microwave fields. The inherent limitations on the interpretation of data of this type, namely the inadequacies in exposure histories and biasing effects such as the removal from employment of any individuals who have, in fact, experienced ocular pathology, preclude the conclusion that long-term occupational exposure of humans to microwave intensities in the range of 10 mW/cm^2 does not result in lens changes. In spite of the inherent limitations in experimental data, the apparent involvement of thermal mechanisms in microwave cataractogenesis supports the hypothesis that repeated exposures in excess of 10 mW/cm^2 would be necessary for cataract induction in human beings. It is somewhat surprising and unfortunate that more definitive data on microwave cataractogenesis is not available in view of the extent of the investigations in this area. The problem of interspecies extrapolation of bioeffects data is well examplified by microwave cataractogenesis which appears to be a much more well defined, and quantifiable endpoint than most reported biological effects of microwave exposure.

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This report reviews scientific literature concerning the effects of microwave radiation on the lens of the eye, particularly in regard to potential for cataractogenesis at low exposure levels. The partially-understood biophysical mechanism of microwave cataractogenesis is briefly discussed. No evidence was found for cataract induction by microwave fields of less than 10 mW/cm^2; however the possibility could not be excluded unequivocally on the basis of existing knowledge. Suggestions are made for the kinds of data that should be collected in any future laboratory research and epidemiological studies on this topic.

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