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# Ocular Effects of Radiofrequency Energy

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Radiofrequency (RF) energy has been reported to cause a variety of ocular effects, primarily cataracts but also effects on the retina, cornea, and other ocular systems. Cataracts have been observed in experimental animals when one eye was exposed to a localized, very high RF field and the other eye was the unexposed control. The results show that 2450 MHz exposures for ≥30 min at power densities causing extremely high dose rates (≥150 W/kg) and temperatures (≥41°C) in or near the lens caused cataracts in the rabbit eye. However, cataracts were not observed in the monkey eye exposed to similar exposure conditions, reflecting the different patterns of energy absorption (SAR, specific absorption rate) distribution, due to their different facial structure. Since the monkey head is similar in structure to the human head, the nonhuman primate study showed that the incident power density levels causing cataracts in rabbits and other laboratory animals cannot be directly extrapolated to primates, including human beings. It is reasonable to assume that an SAR that would induce temperatures ≥41°C in or near the lens in the human eye would produce cataracts by the same mechanism (heating) that caused cataracts in the rabbit lens; however, such an exposure would greatly exceed the currently allowable limits for human exposure and would be expected to cause unacceptable effects in other parts of the eye and face. Other ocular effects including corneal lesions, retinal effects, and changes in vascular permeability, have been observed after localized exposure of the eye of laboratory animals to both continuous wave (CW) and pulsed wave (PW) exposures, but the inconsistencies in these results, the failure to independently confirm corneal lesions after CW exposure, the failure to independently confirm retinal effects after PW exposure, and the absence of functional changes in vision are reasons why these ocular effects are not useful in defining an adverse effect level for RF exposure. While cataracts develop after localized exposure of the eye at SARs ≥ 150 W/kg, whole body exposure at much lower levels (14–42 W/kg) is lethal to rabbits. Two studies reported cataracts in this animal after 30 daily exposures at SARs at the upper end of the lethal range, e.g., 38–42 W/kg; however, long term exposure of rabbits (23 h/day, 6 months) at 1.5 W/kg (17 W/kg in the rabbit head) did not cause cataracts or other ocular effects. A long term (1–4 years) investigation of monkeys exposed at high SARs (20 and 40 W/kg to the monkey face) found no cataracts or other ocular effects or change in visual capability. The results of these long term studies support the conclusion that clinically significant ocular effects, including cataracts, have not been confirmed in human populations exposed for long periods of time to low level RF energy. The results of four recent human studies show that there is no clear evidence of an association between RF exposure and ocular cancer. Bioelectromagnetics Supplement 6:S148–S161, 2003. 

**Key words: cataracts; microwaves; human studies; cornea; retina**

## INTRODUCTION

Radiofrequency (RF) energy has been reported to cause a variety of ocular effects, primarily cataracts in the lens, but also effects on the retina, cornea, and other ocular systems. The literature describing ocular effects is discussed below and summarized in four tables (Tables 1–4).

## CATARACTS AND RELATED STUDIES (NEAR FIELD EXPOSURE)

Table 1 is a summary of studies describing cataracts and related effects after localized exposure of the eye or head of laboratory animals; these studies provided information on power density and/or specific absorption rate (SAR). Cataracts have been observed in some experimental animals when one eye was exposed to a very high RF field and the other eye was the unexposed control. Two early cataract studies by Daily et al. [1950a] and Baille [1970] used the dog as the experimental animal but the animal model most often used was the albino rabbit. In many studies, the microwave frequency was 2450 MHz as shown in Table 1.

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**TABLE 1. Cataracts and Related Studies (Near Field Exposure)**

| Effects | Species | Frequency (MHz) | Power density ($mW/cm^2$) | Exposure (days × min) | SAR (W/kg) | Reference |
|---|---|---|---|---|---|---|
| Cataract | Dog | 2500 | 5000 | 1 × 1 | Not determined | Baille [1970] |
| No cataract at 10 weeks after single exposure | Sheep | 64 | — | 1 × 40 | 4 | Barber et al. [1990] |
| Cataract and transient effects (acute inflammatory reactions of cornea, iris, and/or ciliary body) | Rabbit | 5500 (CW and PW) | 470–785[a] | 1 × 2–100 | 300–500[b] | Birenbaum et al. [1969a] |
| Cataract and iritis | Rabbit | 800 (CW) | 785[a] | 1 × 25 | 500[b] | Birenbaum et al. [1969b] |
| | | 4200 (PW) | 785[a] | 1 × 17 | 500[b] | |
| | | 4600 (PW) | 785[a] | 1 × 15 | 500[b] | |
| | | 5200 (PW) | 500–785[a] | 1 × 5–12 | 350–500[b] | |
| | | 5400 (CW and PW) | 500–785[a] | 1 × 3–4 | 300–500[b] | |
| | | 5500 (CW and PW) | 500–785[a] | 1 × 2–3 | 300–500[b] | |
| | | 6300 (PW) | 785[a] | 1 × 5 | 500[b] | |
| Cataract and transient effects (swelling and chemosis of bulbar and palpebral conjunctivae, papillary constriction, hyperemia of iris and limbal vessels, and vitreous floaters and filaments) | Rabbit | 2450 (CW) | 180<br>120–180 | 1 × 240<br>20 × 60 | Not determined | Carpenter [1979] |
| No cataract | Rabbit | 2450 (CW) | 75 | 20 × 60 | Not determined | Carpenter [1979] |
| Cataract and transient effects (pupillary constriction and anterior chamber turbidity) | Rabbit | 2450 (CW) | 150 | 1 × 100 | 138[c] | Guy et al. [1975] |
| Cataract | Rabbit | 2450 (1000 Hz mod) | 285 | 1 × 30 | 15.3 (head) | Foster et al. [1986] |
| Cataract and transient effects (pupillary constriction, hyperemia of bulbar and palpebral conjunctivae) | Rabbit | 2450 (CW) | 295 | 1 × 30 | Not determined | Hagan and Carpenter [1976] |
| | | 10000 (CW) | 375 | 1 × 30 | | |
| Cataract and transient effects (constricted pupil, dilated conjunctival and iris vessels, turbid anterior chamber) | Rabbit | 2450 (CW) | 180 | 1 × 140 | 100[c] | Kramar et al. [1978] |
| 2nd to 3rd degree nasal burns; no ocular effects | Monkey | 2450 (CW) | 300 | 1 × 22 | 115[c] | Kramar et al. [1978] |
| No ocular effects | Monkey | 9310 (PW) | 150 (av) | 30–40 × 294 − 665[d] | 20 (see below: McAfee et al., 1983) | McAfee et al. [1979] |
| No cataracts; no effects on cornea, aqueous and vitreous humors or retina; and no loss of visual capability 4 years after 9310 MHz exposure and 1 year after 2450 MHz exposure | Monkey | 9310 (PW)<br>9310 (PW)<br>2450 (CW) | 150 (av)<br>300 (av) | 408–946 min over 34 months<br>275–594 min over 34 months<br>549–750 min over 4 months | 20<br>40<br>20 | McAfee et al. [1983] |
| No cataract but inflammation of cornea (keratitis) | Rabbit | 35000<br>107000 | 40<br>40 | 1 × 60<br>1 × 60 | >175[e]<br>>238[e] | Rosenthal et al. [1976] |

Adapted from Elder [1984]. Studies included in this table had power density and/or SAR data.
[a]Estimate of average power density calculated by dividing the microwave power by the irradiated area (d = 1.27 cm) of the eye.
[b]Estimate based on the assumption that all incident power was absorbed in the eye (2 g).
[c]Maximum SAR in the eye.
[d]Total exposure time in minutes for the entire 30–40 day experimental period.
[e]Estimated SAR values for the cornea.

Exposure of the rabbit eye to 2450 MHz resulted in a direct relationship between maximal RF energy absorption and maximal temperature in the vitreous body at a point midway between the posterior surface of the lens and the retinal surface [Guy et al., 1975; Kramar et al., 1975]. The locus of peak energy absorption and peak temperature correlates well with the observation of irreversible changes (cataracts) in the posterior cortical lens after a latent period of a few days if the RF exposure was at or above threshold levels, e.g., 150 W/kg for 30 min and retrolental temperature above 41 °C [Carpenter et al., 1960; Carpenter, 1979; Guy et al., 1975; Kramar et al., 1975]. Thresholds as a function of time ($\leq$100 min), power density, and SAR for induction of cataracts in rabbits by single exposure to near field 2450 MHz fields are shown in figures in Guy et al. [1975] and Kramar et al. [1975]; these figures show excellent agreement between threshold data published by Guy's laboratory and Carpenter's laboratory.

The results described above led to the conclusion that microwave induced cataracts are due to an elevation in temperature. This conclusion was addressed by several investigations designed to examine directly the relation between temperature increase and cataract formation. Two of these experiments were not successful in causing cataracts in rabbits by heating the eye with nonmicrowave techniques to temperatures known to be associated with microwave induced cataracts [Kramar et al., 1976; Carpenter et al., 1977]. The time temperature profile of microwave energy absorption in the eye is difficult to duplicate with nonmicrowave techniques and is probably responsible for cataracts not being observed as expected in these two studies.

Other experimental approaches, such as those by Baille [1970] and Kramar et al. [1975], did provide evidence that RF heating was important in cataract formation. Baille [1970] reported that cataracts did not develop when the eye was irradiated under hypothermic conditions. Five years later, Kramar et al. [1975] published a pivotal paper containing convincing, analytical data demonstrating the importance of temperature in the formation of microwave cataracts. This study showed that retrolental temperatures above 41 °C were necessary for cataract formation because no cataracts formed in the rabbit eye exposed to cataractogenic levels of RF energy when the animal's body was submerged in cold water and the microwave induced intraocular temperature was limited to <41 °C.

The data of Kramar et al. [1975] provide strong evidence that microwave cataracts are caused by the elevated temperature resulting from absorption of RF energy in the eye. In general, cataractogenic exposure levels were so thermally stressful that localized exposure of the eye (and head) caused the whole body (rectal) temperature of rabbits to increase by 1.2–2.7 °C [Carpenter et al., 1975, 1977; Foster et al., 1986] and whole body exposure at these levels produced extreme body temperatures resulting in death of the animal [Appleton et al., 1975a; Carpenter et al., 1975].

Because lens opacities are permanent changes, most of the early literature on ocular effects of RF energy emphasized cataracts, but these reports also described a variety of important transient ocular responses in animals receiving a cataractogenic exposure (see Table 1). The transient effects described by Carpenter [1979; p. 1050] which varied in severity according to the RF energy and duration of exposure, included "swelling and chemosis of bulbar and palpebral conjunctivae, pupillary constriction, hyperemia of iris and limbal vessels, and vitreous floaters and filaments." Birenbaum et al. [1969b] reported that iritis (inflammation of iris) was almost always coupled with lens opacities.

As shown in Table 1, cataracts have been produced in the rabbit eye by radiofrequencies ranging from 0.8 to 10 GHz. Two comparisons can be drawn from these data to demonstrate the frequency dependence of cataractogenesis in this animal. First, Guy et al. [1974] provided data showing why 2.45 GHz is more effective than 0.918 GHz. At 2.45 GHz, peak energy absorption occurred near the lens; at 0.918 GHz, peak energy absorption occurred deeper in the head resulting in a higher threshold for lens opacities than at 2.45 GHz. Second, Hagan and Carpenter [1976] compared the relative effects at 2.45 and 10 GHz and found that the cataractogenic potential was greater at the lower frequency, a result consistent with peak energy absorption at the higher frequency occurring in eye tissues near the surface of the eye and not in or near the lens. At higher frequencies of 35 and 107 GHz that did not cause opacities in the lens [Rosenthal et al., 1976], effects were observed in other tissues of the rabbit eye, e.g., inflammation of the cornea. The frequency dependent distribution of RF energy observed in the rabbit eye demonstrates that higher frequencies have greater potential for effects on the structures near the outer surface of the eye and lower potential for effects within the eye, such as lens opacities.

A study in nonhuman primates demonstrated that incident power density at the thresholds for cataracts in rabbits did not produce cataracts in the monkey eye, but caused undesirable effects such as facial burns [Kramar et al., 1978]. The SAR threshold for cataracts in the monkey eye, however, might be the same as the SAR threshold for cataracts in rabbit eyes, but the rabbit and monkey experiments showed clearly that the same incident power density exposure did not produce

similar effects on the face and eye of these two experimental animals. The dissimilar effects reflect the different patterns of 2450 MHz energy absorption in the monkey and rabbit heads due to their different facial structure. Rabbit eyes protrude in comparison to monkey eyes that are more embedded in the eye sockets. The results of the nonhuman primate study showed that cataractogenic power density levels in rabbits and dogs should not be directly extrapolated to primates, including human beings. As discussed below, the modeling study of Taflove and Brodwin [1975] can be used to estimate exposure conditions which could cause cataracts in the human eye. It is important to assess other effects on the human eye and face caused by such an exposure.

McAfee et al. [1983] exposed monkeys without restraint or anesthesia by training animals to expose their face and eyes to microwave energy at two different frequencies while receiving a food reward for rapid lever pressing. Monkeys received 15 min of exposure during behavioral work sessions lasting an average of 25 min. Total exposure time over a 4 month period to 2450 MHz (20 W/kg in head) ranged from 549 to 750 min. Total exposure time over 34 months to 9310 MHz (PW) ranged from 408 to 946 min (20 W/kg in head) and 275–594 min (40 W/kg in head). Eye examinations performed at approximately 6 month intervals over 1 year after the 2450 MHz exposures and 4 years after 9310 MHz exposure revealed no ocular effects on visual capability, lens, cornea, retina, and aqueous and vitreous humors.

At the same average power, continuous wave (CW) and pulse wave (PW) exposures at 5.5 GHz were shown to be equally effective in producing cataracts in rabbits [Birenbaum et al., 1969a]. These authors concluded that the average, not the peak, rate of energy absorption (SAR) determined whether lens opacities occurred, which is consistent with a thermal mechanism. As mentioned above, Kramar et al. [1975] showed that the RF exposure must produce a temperature exceeding 41 °C in or near the lens to cause cataracts.

The question of cumulative effects, i.e., effects which increase in significance in the course of repeated exposures, is of special concern in assessing the biological effects of RF energy. Cataract formation had been reported in dogs exposed repeatedly to subthreshold levels by Baille [1970] but this paper did not provide SAR data and few animals were used. Because of these experimental deficiencies, the results in Carpenter [1979] described below are most often cited as evidence for cumulative effects from RF exposure. Carpenter observed cataracts in rabbits following repeated daily exposure (1 h/day for 13–20 days) of the eye to RF energy at subthreshold levels of approximately 75% of the cataract threshold (150 W/kg) for a single exposure. The subthreshold levels were very high, and these levels, as well as the threshold levels, have been shown to be incompatible with survival if given to a rabbit as a whole body exposure. Appleton et al. [1975a], for example, observed that unanesthesized rabbits became heat stressed and struggled out of the field during a 15 min whole body exposure at 14 W/kg; thus, repeated exposures to subthreshold levels as high as 75% of the cataractogenic dose rate (150 W/kg) would be highly unlikely. The cumulative exposure conditions described by Carpenter [1979] are applicable to localized exposure of the eye of restrained animals only and cannot be extrapolated to unrestrained, whole body exposure situations because whole body exposure at such levels are lethal to the animal [Appleton et al., 1975a; Carpenter et al., 1975].

One study investigated cataract formation and temperature levels in anesthesized sheep exposed in an magnetic resonance imaging (MRI) whole body coil or head coil [Barber et al., 1990]. Ten weeks after exposure for 40 min at 4 W/kg, the sheep were found to be in good health with no cataracts. Exposure of the head at this SAR for 75 min caused the temperature of the eye to increase about 1.5 °C; a whole body exposure at 4 W/kg for more than 82 min caused deep body temperature to rise in excess of 2 °C.

Biochemical changes have been reported in lenses exposed to RF energy. Kinoshita et al. [1966] found that ascorbic acid levels decreased in lenses at 18 h, but not at 6 h, after a cataractogenic exposure of the rabbit eye; this decrease in ascorbic acid preceded cataract formation. In an in vitro study, Weiter et al. [1975] exposed cultured rabbit lenses to 2450 MHz (CW) and reported that the ascorbic acid concentration in the lens decreased as the microwave power was increased. No differences were found, however, between exposed and control lenses subjected to identical time temperature conditions. Weiter et al. [1975] also compared the effects of CW and pulsed fields at the same average power on cultured lenses and found no difference in ascorbic acid concentrations. These authors concluded that the decrease in ascorbic acid concentration is a direct thermal effect of microwave exposure. In other work, Van Ummersen and Cogan [1976] found a marked, but transient, suppression of DNA synthesis and mitosis in the epithelium of the rabbit lens from eyes given a cataractogenic exposure to RF energy. It is reasonable to assume that these biochemical changes are thermal effects since cataractogenic exposure levels increase the temperature in the lens.

Using microscopy techniques, Stewart-DeHaan et al. [1983; 1985] and Creighton et al. [1987] examined

rat lenses exposed in vitro to 918 MHz fields [CW and PW (24 kW peak power, 10 μs pulse width)] and found that pulsed fields produced greater changes. A 20 min PW exposure caused effects at an average SAR as low as 5.75 W/kg, but an estimate of the extremely high peak field is $3 \times 10^5$ W/kg. The usefulness of these in vitro results for defining an adverse effect level has not been established because the effects have not been independently replicated and threshold values for similar effects in live animals, if they occur, are not known.

The statement that the eye cannot effectively dissipate heat due to limited blood vascular systems is frequently mentioned, but Carpenter et al. [1977] took exception to this statement based on the following simple experiment. ''If the temperature at the posterior pole of the lens in an anesthetized rabbit is measured prior to and during microwave irradiation, it may be found to rise perhaps 5 °C in the course of a 15 min exposure. If a lethal dose of anesthetic is then injected intravenously, the heart will stop beating, whereupon the intraocular temperature will rapidly rise another 10 °C, thus indicating that the vascular system is capable of handling at least two-thirds of the thermal stress which radiation imposes on the eye'' [Carpenter et al., 1977, p. 354]. A similar observation is described in earlier work by Daily et al. [1950a, see p. 1245], who concluded that blood circulation in tissues adjacent to the avascular aqueous and vitreous humors was the explanation for the rapid rate of cooling of these humors following exposure of the eye to microwave energy.

The summary of this section is combined with the summary of the next section (see below).

## OCULAR EFFECTS (FAR FIELD EXPOSURE)

Table 2 is a summary of studies of ocular effects involving whole body exposure of the rabbit. Cataracts were observed in two studies [Williams et al., 1975; Hirsch et al., 1977] but only after 30 daily exposures at intense RF fields at or very near lethal levels (~40 W/kg). In an earlier study that exposed animals to the same conditions as described in Hirsch et al. [1977], Appleton et al. [1975a] described ocular and other effects in anesthesized animals exposed once to SARs up to 70 W/kg. The authors concluded that, ''It is noteworthy that one year after a single microwave exposure sufficient in intensity to cause both thermal cutaneous and acute gross ocular effects, no lens changes or cataracts were observed'' [Appleton et al., 1975a, p. 131]. It should be noted that effects on the animals were more severe than described in the quotation because rabbits were reported to die during a 30 min exposure at 42 W/kg [Appleton et al., 1975a]. These authors also observed that awake, unrestrained animals would not tolerate cataractogenic levels of RF energy because unanesthesized rabbits became heat stressed and struggled out of the field during a 15 min, whole body, exposure at 14 W/kg, an SAR well below the cataractogenic level. With the same exposure system, anesthetized rabbits given 30 daily exposures for the same duration (15 min/day) and SAR (14 W/kg) did not develop ocular effects (iritis or cataracts), but one of the five animals died after 22 exposures [Hirsch et al., 1977].

The anesthetic (diallyl-barbituric acid) used by Daily et al. [1950b] was found to have a profound effect on cataract development in the rabbit. These authors reported that cataracts were produced in anesthetized animals following 2450 MHz exposure under identical conditions that did not produce cataracts in unanesthetized rabbits. They concluded that the eye temperature in anesthetized animals was higher than the eye temperature in unanesthetized rabbits following RF exposure under identical conditions.

Guy et al. [1980] chose a whole body SAR of 1.5 W/kg so unanesthesized rabbits would survive long term exposure (23 h/day for 6 months at 2450 MHz); no cataracts or other ocular effects were found even though the peak SAR in the head was 17 W/kg. Chou et al. [1982] compared the effects of CW and pulsed 2450 MHz fields on rabbits exposed for 2 h/day for 3 months (SAR = 1.64 W/kg in head). Neither the CW nor PW exposure caused a significant change in any health measure including cataracts, body weight, hematology, blood chemistry, and pathology of major organs. In 1983, Chou et al. reported no effect on cataract formation, blood measures, and pathology in rabbits exposed 7 h/day, 5 day/week for 13 weeks at two exposure levels (0.55 and 5.5 W/kg in the head). Although cataracts have been observed in animals exposed to life threatening RF fields, the work of Guy et al. [1980] and Chou et al. [1982, 1983] showed that long term, low level RF exposures did not cause cataracts.

### Summary of Cataract Studies (Far and Near Field Exposure)

Far field exposure studies show that cataracts do not form in rabbit eyes unless intense RF fields at or near lethal levels are applied. Near field exposure studies show that RF exposure causes cataracts in rabbits when the lens of the eye is exposed to very high levels (≥150 W/kg) for sufficient times (≥30 min) that result in temperatures ≥41 °C in or near the lens. The formation of cataracts in the rabbit eye is a consequence of intense, localized exposure of the eye and the structure of the rabbit head because exposure conditions that

**TABLE 2. Ocular Effects (Far Field Exposure)**

| Effects | Species | Frequency (MHz) | Power density ($mW/cm^2$) | Exposure (days × min) | SAR (W/kg) | Reference |
|---|---|---|---|---|---|---|
| No ocular effects, including no lenticular changes | Rabbit | 3000 (CW) | 100, 200 | 1 × 15, 30 | 14[a], 28[a] | Appleton et al. [1975a] |
| Acute ocular changes, e.g., hyperemia of lids and conjunctiva, meiosis, anterior chamber flare, engorgement of iris vessels, and periorbital cutaneous burns; no lenticular changes | | | 300, 400, 500 | 1 × 15 | 42[a], 56[a], 70[a] | |
| Death | | | 300 | 1 × 30 | 42[a] | |
| Death | | | 500 | 1 × 15 | 70[a] | |
| No cataracts; no effect on blood measures and pathology | Rabbit | 2450 (CW and PW) | 1.5 | 1 × 120 (×3 months) | 1.6 (head) | Chou et al. [1982] |
| No cataracts; no effect on body mass, blood measures, and pathology | Rabbit | 2450 (CW) | 0.5, 5 | 5 × 420 (×13 weeks) | 0.55, 5.5 (head) | Chou et al. [1983] |
| No cataracts | Rabbit | 385 (CW) | 60 | 10 × 15 | 48[a] | Cogan et al. [1958] |
| | | 385 (CW) | 30 | 10 × 90 | 24[a] | |
| | | 468 (CW) | 60[b] | 10 × 20 | 8.1 | |
| No cataracts | Rabbit | 2450 (CW) | 10 | 5 × 480 (×8–17 weeks) | 1.5[a] | Ferri and Hagan [1976] |
| No ocular effects | Rabbit | 2450 (CW) | 10 | 180 × 1380 | 17[c] | Guy et al. [1980] |
| No ocular effects | Rabbit | 3000 (CW) | 50 | 30 × 15 | 7[a] | Hirsch et al. [1977] |
| No ocular effects (but one of five rabbits died in each group) | | | 100, 200 | | 14[a], 28[a] | |
| Iritis, cataracts (but one of five rabbits died) | | | 300 | | 42[a] | |
| No cataracts (electron microscopic changes reported in one lens) | Rabbit | 2450 | 165 | 36 × 40 | 25[a] | Williams et al. [1975] |
| Lens opacities | | | 250 | 30 × 20 | 38[a] | |

Adapted from Elder [1984].
[a]Estimated average whole-body SAR values (Durney et al., 1978, Figure 31).
[b]Waveguide average whole-body exposure.
[c]Maximal SAR in head.

**TABLE 3. Corneal, Retinal, and Other Ocular Effects (Near Field Exposure)**

| Effects | Species | Frequency (MHz) | Power density ($mW/cm^2$) | Exposure duration | SAR (W/kg) | Reference |
|---|---|---|---|---|---|---|
| No corneal endothelial abnormalities, cataracts, or effects on retina or vitreous humor (corneal effects due to CW exposure in Kues et al. [1985] not confirmed) | Monkey − anesthesia | 2450 (CW) | 15.9–43 | 240 min | 4.1–11 (cornea) | Kamimura et al. [1994] |
| No effect | Monkey + anesthesia | 2450 (CW) | 20 (cornea) | 240 min | 5.2 (cornea) | Kues et al. [1985] |
| Transient corneal endothelial abnormalities | | " | 30 (cornea) | 240 min | 7.8 (cornea) | |
| No effect | Monkey + anesthesia | 2450 (PW) 10 μs, 100 pps | 5 (cornea) | 240 min once a week for 2 weeks | 1.3 (cornea) | |
| Transient corneal endothelial abnormalities | | " | 10 (cornea) | 240 min once a week for 2–11 weeks | 2.6 (cornea) | |
| Transient corneal endothelial abnormalities | | " | 15 (cornea) | 240 min | 3.9 (cornea) | |
| Transient corneal endothelial abnormalities and increased vascular permeability of iris (ophthalmic drug pretreatment) | Monkey + anesthesia | 2450 (PW) 10 μs, 100 pps | 1 | 240 min daily for 3 days | 0.26 | Kues et al. [1992] |
| No effects on cornea, iris, or lens | Rabbit and monkey + anesthesia | 60000 (CW) | 10 (cornea) | 8 h or five daily 4 h exposures | Not determined | Kues et al. [1999] |
| No corneal abnormalities detected by same procedure used in Kues et al. [1985, 1992] but corneal effects detected at high magnification (TEM); no overall change in iris vascular permeability; retinal degeneration reported | Monkey − anesthesia | 1250 (PW) 0.5 or 10 μs, 1 or 16 pps, 1 MW peak power | | 240 min daily for 3 days × 3 weeks | 3.5–5.0 (retina) | Kues and Monahan [1993] |
| No retinal effects (retinal degeneration in Kues and Monahan [1993] not confirmed) | Monkey − anesthesia | 1250 (PW), 1.04 MW peak power, 5.59 μs pulse width at 0, 0.59, 1.18, and 2.79 pps | | 240 min daily for 3 days × 3 weeks | 4.3, 8.4, and 20.2 (retina) | Lu et al. [2000] |
| Enhanced cone photoreceptor response to light flash (authors did not consider this change an adverse effect) | | | | | 8.4 and 20.2 (retina) | |
| Degenerative retinal changes observed by electron microscopy; no lens opacities and no effect on blood–brain barrier or blood–retina barrier permeability | Rabbit | 3100 (PW), 1.4 μs pulse length, 300 pps | 55 | 1–1.5 h once or 1 h/day, 3 day/week up to 53 exposure hours over 3 months | 30 W/kg at retina (est.); "...40 °C near the retina 3 min after irradiation" | Paulsson et al. [1979] |

**TABLE 4. Ocular Effects, Including Cancer, in Human Beings**

| Effects | Comments | Frequency (MHz) | Power density ($mW/cm^2$) | Reference |
|---|---|---|---|---|
| No difference in presence or absence of opacities, vacuoles, and posterior subcapsular iridescence | Clinical survey of 91 US Army Signal Corps personnel (135 controls) | Not specified | Not specified | Appleton and McCrossan [1972] |
| No difference in presence or absence of opacities, vacuoles, and posterior subcapsular iridescence | Clinical survey of 1542 US Army personnel (801 controls) | Not specified | Not specified | Appleton et al. [1975b] |
| Lens opacities with no decrease in vision and retinal lesions (two cases with decrease in vision) [retinal effects not confirmed by Hathaway et al., 1977] | 68 Radar/microwave workers (30 controls) in electronics industry | Not specified | Not specified | Aurell and Tengroth [1973] |
| No significant risk of cataracts | Case-control study of 2946 US military veterans (2164 controls) | Not specified | Job classification used as indicator of exposure | Cleary et al. [1965] |
| More subclinical lens changes than controls, but no cataracts and no decrease in visual acuity | 736 Microwave workers (559 controls) | Not specified | Not specified | Cleary and Pasternak [1966] |
| No lenticular or retinal defects [retinal effects reported by Aurell and Tengroth, 1973 not confirmed] | 705 Microwave workers (US Army) | Not specified | Majority not exposed $>0.1$ $mW/cm^2$ | Hathaway et al. [1977] |
| No statistically significant increase in posterior subcapsular cataracts (PSCs) in individuals but significant increase in PSCs in eyes | 53 Radiolinemen; 39 controls | 558 kHz–527 MHz | 0.08–3956 $mW/cm^2$ | Hollows and Douglas [1984] |
| Lens opacities but no impairment in visual acuity | 102 Microwave workers employed for $>4$ years (100 controls); subset of above 400 workers | 600–10,700 | Not specified | Majewska [1968] |
| No difference in lens anomalies. The subsets of controls and ''exposed'' groups having a familial history of eye problems showed a higher percent of lens changes in exposed personnel but no statistical test applied | 377 US military personnel involved in operation or maintenance of radar equipment (320 controls) | Not specified | Not specified | Odland [1973] |
| No statistically significant changes in lens opacities, lens vacuoles, and posterior subcapsular iridescence | 447 Military microwave workers; 340 controls | Not specified | Not specified | Shacklett et al. [1975] |
| No differences in lens opacities | 507 Radar workers (334 controls) | Not specified | 0.2–6 $mW/cm^2$ (workers); $<0.2$ $mW/cm^2$ (controls) | Siekierzynski et al. [1974] |
| Cancer studies | | | | |
| Twofold increase in uveal melanoma | Microwave/radar workers (21 patients; 22 controls) | Not specified | Not specified | Holly et al. [1996] |
| No association between cancer, including eye cancer, and mobile phone use | 420095 mobile phone subscribers in Denmark (1982–1996) | Not specified | Not specified | Johansen et al. [2001] |
| No increase in uveal melanoma incidence rate during 1943–1996 in Denmark | 493 cases | Not specified | Not specified | Johansen et al. [2002] |
| Increased uveal melanoma<br>(1) Odds ratio = 3.0<br>(2) Odds ratio = 4.2 | <br>(1) 16 cases<br>(2) 6 cases | <br>(1) Any kind of RF exposure<br>(2) ''Probable/certain mobile phone exposure'' | Not specified | Stang et al. [2001] |

cause cataracts in rabbits do not induce cataracts in nonhuman primates with facial structures similar to those of human beings. RF exposure conditions that would cause cataracts in the human eye have not been defined but the high levels required would be expected to produce unacceptable effects on other parts of the eye and face prior to cataract formation [COMAR, 2002].

## CORNEAL, RETINAL, AND OTHER OCULAR EFFECTS (NEAR FIELD EXPOSURE)

These studies are summarized in Table 3. Single or repeated exposure over 5 days of the rabbit and monkey eye to 60 GHz fields at 10 $mW/cm^2$ did not cause cellular damage in the cornea, affect vascular permeability in the iris, or induce lens opacities as determined by a variety of techniques including histopathology [Kues et al., 1999]. Other studies by Kues et al. at lower frequencies, however, described effects on the cornea, iris, and retina; some of these effects were reported to occur at an SAR as low as 0.26 W/kg. Kues et al. [1985] reported that both CW and pulsed 2450 MHz exposures of the eye of anesthesized monkeys caused corneal lesions; the effective SAR for CW exposures was about twice the effective SAR of 2.6 W/kg for pulsed fields. Kues et al. [1992] reported that the effective SAR for pulsed exposures was ten times lower (0.26 W/kg) for corneal lesions and for increased vascular permeability of the iris if the eye had been treated with ophthalmic drugs for glaucoma. The effects on the cornea and iris vasculature were transitory and resolved over a period of 1 week.

In unanesthesized monkeys exposed at a different frequency (1250 MHz, pulsed) to an SAR of 3.5–5 W/kg in the retina, Kues and Monahan [1993] failed to find corneal lesions with the same procedure during the same time period (i.e., 24 h postexposure) as described in their 1985 paper; however, corneal effects were observed in five of six animals with a different, high magnification technique (transmission electron microscopy). In their 1993 report, other measurements on seven monkeys showed that the iris vascular permeability was increased in two animals, decreased in two animals, and was unchanged in three subjects. Therefore there was no overall change in the blood–aqueous barrier that includes the iris vasculature that was reported to have increased permeability in their earlier study [Kues et al., 1992]. Electroretinography was used to detect functional changes (electrical) in retinal response to light and three of five animals had more than marginal change to retinal cone cells. In all three studies by Kues et al. [1985, 1992] and Kues and Monahan [1993], the pulsed exposures produced peak field strengths ranging from 5 to 15 $kW/cm^2$ (see Table 1 in Lu et al., 2000). The studies by Kues et al. are summarized in Table 3 along with summaries of the studies described below that attempted to reproduce some of Kues' results.

Independent studies have failed to confirm the results of Kues et al. showing: (a) corneal lesions after CW exposure and (b) retinal degeneration after pulsed exposure. Kamimura et al. [1994] found no corneal effects as well as no effects on the lens, vitreous humor, and retina in the eyes of unanesthesized monkeys exposed to 2450 MHz CW fields of up to twice the intensity in Kues et al. [1985] and concluded that the effects reported by Kues et al. may have been due to use of anesthesia. In addition, the retinal degeneration reported by Kues and Monahan [1993] was not confirmed by Lu et al. [2000] who exposed the monkey face to the same pulsed frequency (1250 MHz) at 4.3, 8.4, and 20.2 W/kg. Lu et al. stated that one of the procedures (fluorophotometry) and the repetitive use of ketamine for physical restraint in the Kues et al. studies may have predisposed the monkeys to retinal injuries. This conclusion was based on the observation that, regardless of the RF exposure used in the experiments by Kues et al., retinal histopathology was within normal limits in monkey eyes not subjected to fluorophotometry whereas retinal injury was observed in five monkeys (including one sham) in which fluorophotometry was used.

The functional significance of the reported ocular effects has been studied in tests of contrast sensitivity that are used as a measure of basic visual spatial resolution. In two experiments, visual contrast sensitivity in monkeys was not altered by exposures at 5.6 GHz (pulsed, 1–6 W/kg whole body average SAR) or at 1.3 GHz (pulsed, 1 W/kg whole body average SAR, 4 W/kg at the eye). These studies by D'Andrea are summarized in Figure 1 in Lu et al. [2000], a paper coauthored by D'Andrea, but have not been published in detail because verification of the peak field strengths is needed (J. D'Andrea, personal communication, June 23, 2001). As described above, McAfee et al. [1983] observed no effect on visual capability in monkeys that received repeated high intensity exposure to the face at 2.45 and 9.31 (0.5 μs pulse, 40 kW peak power) GHz.

Electron microscopy revealed degenerative retinal changes in rabbits exposed to pulsed 3100 MHz radiation at 55 $mW/cm^2$ (30 W/kg at retina estimated) that resulted in a temperature increase to 40 °C near the retina 3 min after exposure [Paulsson et al., 1979]. Although thermal effects in the retina were observed at this intense exposure, no lens opacities and no effect on blood–brain barrier permeability or retinal vascular permeability were found.

### Summary of Corneal, Retinal, and Other Ocular Effects (Near Field Exposure)

Kues and Monahan [1993] did not observe corneal effects similar to those reported at 2.6 W/kg in their earlier studies and this failure raises a question about the repeatability of the effects reported at 0.26 W/kg when glaucoma drugs were used [Kues et al., 1992]. In an independent attempt to confirm corneal effects in monkeys, Kamimura et al. [1994] exposed unanesthesized primates and failed to replicate the corneal effects reported by Kues et al. [1985] in anesthesized monkeys exposed to CW fields. In other work by Kues and Monahan [1993], as explained above, the permeability change of the eye vasculature is not consistent with their 1992 results. The effects on the retina reported by Kues et al. were not confirmed in an independent study by Lu et al. [2000]. In addition, Lu et al. [2000] contains a summary of D'Andrea's unpublished results showing no functional changes in vision in monkeys exposed to conditions similar to those causing ocular effects in the studies by Kues et al. McAfee et al. [1983] also found no effect of PW and CW microwave radiation on visual capability in monkeys.

It is important to note that the ocular effects reported by Kues et al. were found with pulsed exposures with peak field strengths of 5–15 kW/cm$^2$. The fast rise time of the RF fields employed by Kues et al., however, is not an explanation for (a) the positive effects from Kues' laboratory and (b) failure of Lu et al. [2000] to confirm some of these reported ocular effects because Kues et al. [1985] reported that CW exposures, in which issues related to rise times do not exist, caused similar ocular effects as PW exposures.

## OCULAR EFFECTS IN HUMAN BEINGS

The reports of RF-induced cataracts in laboratory animals and case reports of cataracts in microwave workers led to studies of ocular effects in human populations (see Table 4). Cleary and Pasternak [1966] found more subclinical lens changes in a group of 736 microwave workers than in 559 controls, but no cataracts or decrease in visual acuity were noted. Majewska [1968] observed more lens opacities in 200 Polish microwave workers than in a control group of equal size but these opacities did not impair visual acuity, thus there was no clinical significance. In this same study, a subgroup of 102 workers with over 4 years of exposure was examined for effects of long term exposure and, compared to 100 controls, opacities increased out of proportion to the age of the workers.

In a study said to be the first to report retinal lesions, Aurell and Tengroth [1973] found more retinal lesions and lens opacities in 68 workers (30 controls) who tested and measured radar/microwave fields or who worked in experimental laboratories in an electronic industry. The lens opacities were not associated with loss of vision but the retinal lesions were reported to decrease vision in two subjects; however, the retinal effects were not confirmed by Hathaway et al. [1977] (see below).

In Odland [1973], subsets of the exposed and control groups having a familial history of eye problems showed a higher percent of lens changes in exposed personnel but no statistical tests were done. Odland [1973] and Aurell and Tengroth [1973] did not perform statistical tests or provide exposure information. Hollows and Douglas [1984] found no statistically significant increase in posterior subcapsular cataracts (PSCs) in individuals but reported a significant increase in PSCs based on total number of eyes.

The results of these five studies, some without statistical evaluation and most with little or no exposure data, fail to demonstrate clinically significant ocular effects in human populations exposed to RF energy. This conclusion is supported by the results of a number of studies described below reporting no ocular effects in human populations.

Cleary et al. [1965] analyzed groups of U.S. military veterans sufficient to detect a twofold increase in relative risk of cataracts but found no significant effect in 2946 exposed subjects compared to 2164 control subjects. Other studies reporting no ocular effects in human populations exposed to RF energy include Appleton and McCrossan [1972]; Siekierzynski et al. [1974]; Appleton et al. [1975b]; Shacklett et al. [1975]; and Hathaway et al. [1977]. Siekierzynski et al. [1974], for example, found no lens opacities in a comparison of Polish workers ($n = 334$) exposed to $<0.2$ mW/cm$^2$ compared to workers ($n = 507$) exposed to 0.2–0.6 mW/cm$^2$; Shacklett et al. [1975] found no opacities or other ocular effects in 477 U.S. military and civilian personnel compared to 340 controls.

The analysis of lens abnormalities (opacities, vacuoles, and posterior subcapsular iridescence) in 91 U.S. Army Signal Corps personnel by Appleton and McCrossan (1972) showed no differences compared to 135 controls, but there is no evidence of statistical analysis (see below). Later, in a paper by Appleton et al. [1975b], the 1972 paper is called a preliminary report and the 1975 paper describes a much larger study group. No differences were found in lens abnormalities in 1542 U.S. Army personnel described as ''microwave workers'' compared to 801 controls who had not worked with or near microwave equipment. As in their earlier paper, no evidence of a statistical analysis is provided in their 1975 paper.

The data of Appleton and McCrossan [1972] was analyzed by Frey [1985] who came to the conclusion that there was a statistically significant increase in lens abnormalities in the exposed group. Frey's analysis was examined by Wike and Martin [1985] who concluded that (1) ''Frey's improper analysis of the data has led to an erroneous conclusion'' and (2) the occurrence of opacities is significantly associated with age of the subjects and not with groups in both Appleton and McCrossan [1972] and Appleton et al. [1975b]. In sum, independent statistical analysis has confirmed the results of studies of U.S. military personnel showing an association between age and lens opacities but showing no association between RF exposure and ocular effects.

The study by Hathaway et al. [1977] resulted from the continuation of the medical surveillance program of U.S. Army microwave workers described in Appleton and McCrossan [1972] and Appleton et al. [1975b]. No statistical difference was found in occurrence of lenticular defects related to use of microwave equipment or length of time using such devices. The estimated RF exposure was low, since the majority of the 705 microwave workers were not exposed to $>0.1$ mW/cm$^2$. In addition, the results in Hathaway et al. regarding retinal effects did not confirm the retinal effects reported by Aurell and Tengroth [1973], that is, the incidence of retinal effects were similar to that of the controls and not the exposed subjects in Aurell and Tengroth [1973]. Based on these results, Hathaway et al. concluded that periodic medical surveillance as then conducted on microwave workers was nonproductive and suggested that medical surveillance be limited to preassignment, postaccident exposure, and termination eye examinations. The results in Hathaway et al. [1977] corroborate the absence of ocular effects in RF exposed populations reported by Cleary et al. [1965]; Appleton and McCrossan [1972]; Appleton et al. [1975b]; Shacklett et al. [1975]; and Siekierzynski et al. [1974].

Cataracts due to RF exposure have not been reported in the human studies discussed above, but one may assume that an RF exposure that would induce temperature levels $\geq 41$ °C in or near the lens in the human eye would produce cataracts by the same mechanism that causes cataracts in the rabbit lens [see Kramar et al., 1975]. Two relevant modeling studies of the human eye by Hirata et al. [2000, 2002] showed that 5 mW/cm$^2$, the maximum permissible exposure limit for 1.5–100 GHz in controlled environments [FCC, 1996], caused a temperature change in the lens less than 0.3 °C at frequencies from 0.6 to 6 GHz. This small temperature change is overestimated because the eye model was thermally isolated from the head and did not incorporate blood flow. In another modeling study, Athey [1989] calculated an increase of 1.6 °C in the vitreous humor of the human eye following a 1 h exposure of the head in an MRI system (average SAR in head = 3.2 W/kg).

The modeling study of Taflove and Brodwin [1975] can be used to estimate exposure conditions which could cause cataracts in the human eye. Their calculations showed that thermal equilibrium was achieved in 10 min with an incident power density of 100 mW/cm$^2$ (1500 MHz) and, at the center of the eyeball model, resulted in a temperature of 40.4 °C and a peak SAR of about 110 W/kg. These results suggest that power densities greater than 100 mW/cm$^2$ at 1500 MHz could cause peak SARs ($\geq$150 W/kg) and temperatures ($\geq$41 °C) in the human lens that are known to produce cataracts in rabbit eyes. Such exposures would significantly heat the corneal surface. The data of Taflove and Brodwin [1975] showed that the 100 mW/cm$^2$ exposure would increase the surface temperature of the model to 38.5 °C, well above the average corneal temperature of 32.7 °C measured in human subjects by Shellock and Crues [1988]. The results of these modeling studies demonstrate that RF exposures greatly in excess of currently allowable limits would be required to produce cataracts in human beings.

The report by Shellock and Crues [1988] mentioned in the above paragraph was an experimental MRI study involving exposure of the head at 64 MHz (peak SAR range = 2.5–3.1 W/kg). An exposure of at least 15 min caused the temperature of the cornea to increase 0–1.8 °C (average = 0.5 °C); the highest corneal temperature after exposure was 34.4 °C.

Four studies addressed eye cancer in human populations exposed to RF energy. Two of these studies reported an association between RF exposure and uveal melanoma, a cancer of the pigmented vascular tissue in the eye including the iris. Holly et al. [1996] found a twofold increased risk of uveal melanoma in workers exposed to RF fields. In a case-control study, Stang et al. [2001] reported a threefold increase for exposure to radio sets and a fourfold increase in mobile phone users; however, the authors concluded that, ''Several methodologic limitations prevent our results from providing clear evidence on the hypothesized association.'' In an attempt to confirm these observations, Johansen et al. [2002] contrasted the incidence rate of this rare cancer with the number of mobile phone subscribers in Denmark. No increasing trend in the incidence rate of ocular malignant melanoma was found; this result is in sharp contrast to the exponentially increasing number of mobile phone subscribers. In an earlier work, Johansen et al. [2001] found no association between mobile phone use and eye and brain cancer, leukemia,

and more than 20 other cancers in a cohort study of 420 000 users of mobile phones. The results of these four studies show that there is no clear evidence of an association between RF exposure and ocular cancer; these four studies are summarized at the bottom of Table 4.

In another study, based on photosensitivity and other measures, Lim et al. [1993] described an ocular effect (abnormal cone function) in a man accidentally exposed twice for 15 min to 6000 MHz radiation while inspecting a satellite transmitter. The exposures were sufficiently intense to cause facial erythema (eyelid burns), bilateral foreign body sensation, and blurred vision. These observations support the conclusion stated above that the high exposure levels required to produce cataracts in the human eye would cause undesirable effects on other parts of the eye and face.

### Summary of Human Ocular Studies

There are a few studies in Table 4 describing effects on the lens, mostly subclinical effects, and there is one unconfirmed report of retinal effects. These results show that clinically significant ocular effects, including cataracts, have not been confirmed in human populations exposed for long periods of time to low level RF fields. Based on the results of four studies summarized in Table 4, an association between RF exposure and ocular cancer has not been established.

## CONCLUSIONS

Whole body (far field) exposure studies show that cataracts do not form in rabbit eyes unless intense RF fields at or near lethal levels are applied. In localized (near field) studies, threshold exposure conditions for cataracts ($\geq$150 W/kg for $\geq$30 min) have been determined that are associated with temperatures $>$41 °C in or near the lens of the rabbit eye; however, cataracts were not observed in the monkey eye exposed to similar high intensity fields. This difference reflects the different patterns of RF energy absorption in the rabbit and monkey heads due to their different facial structure. Since the monkey head is similar in structure to the human head, the results of the nonhuman primate study showed that cataractogenic exposure levels in rabbits and other laboratory animals should not be directly extrapolated to primates, including human beings. It is reasonable to assume that an RF exposure which would induce temperatures $\geq$41 °C in or near the lens in the human eye would produce cataracts by the same mechanism (heating) that caused cataracts in the rabbit lens; however, such an exposure would greatly exceed the currently allowable limits for human exposure and would be expected to cause unacceptable effects in other parts of the eye and face.

Other ocular effects including corneal lesions, retinal effects, and changes in vascular permeability, were reported by Kues' laboratory following both CW and PW exposures, but the inconsistencies in Kues' results, the failure to independently confirm corneal lesions after CW exposure, the failure to independently confirm retinal effects after PW exposure, and the absence of functional changes in vision are reasons why the ocular effects reported by Kues et al. are not useful in defining the adverse effect level for RF exposure.

Studies with rabbits [Guy et al., 1980] and monkeys [McAfee et al., 1983] show that long term exposure to RF energy did not cause cataracts or other ocular effects at high SARs (17 W/kg in the rabbit head and 20 and 40 W/kg in the monkey face). These results support the conclusion that clinically significant ocular effects, including cataracts, have not been confirmed in human populations exposed for long periods of time to low level RF fields. The results of four other recent human studies show no clear evidence of an association between RF exposure and ocular cancer.

## REFERENCES


Appleton B, McCrossan GC. 1972. Microwave lens effects in humans. Arch Ophthal 88:259–262.

Appleton B, Hirsch SE, Brown PVK. 1975a. Investigation of single exposure microwave ocular effects at 3000 MHz. Ann NY Acad Sci 247:125–134.

Appleton B, Hirsch S, Kinion RO, Soles M, McCrossan GC, Neidlinger RM. 1975b. Microwave lens effects in humans. II. Results of five-year study. Arch Ophthalmol 93:257–258.

Athey TW. 1989. A model of the temperature rise in the head due to magnetic resonance imaging procedures. Magn Reson Med 9:177–184.

Aurell E, Tengroth B. 1973. Lenticular and retinal changes secondary to microwave exposure. Acta Ophthalmologica 51:764–771.

Baille HD. 1970. Thermal and nonthermal cataractogenesis by microwaves. In: Cleary SF, editor. Biological effects and health implications of microwave radiation. Rockville, MD: U.S. Department of Health, Education, and Welfare, Public Health Service, Bureau of Radiological Health (BRH/DBE 70-2). NTIS PB 193 898. pp 59–65.

Barber BJ, Schaefer DJ, Gordon CJ, Zawieja DC, Hecker J. 1990. Thermal effects of MR imaging: Worst-case studies on sheep. Am J Roentgenol 155:1105–1110.

Birenbaum L, Grosof GM, Rosenthal SW, Zaret MM. 1969a. Effect of microwaves on the eye. IEEE Trans Biomed Eng BME-16:7–14.

Birenbaum L, Kaplan IT, Metlay W, Rosenthal SW, Schmidt H, Zaret MM. 1969b. Effect of microwaves on the rabbit eye. J Microwave Power 4:232–243.

Carpenter RL. 1979. Ocular effects of microwave radiation. Bull NY Acad Med 55:1048–1057.

Carpenter RL, Biddle DK, Van Ummersen CA. 1960. Opacities in the lens of the eye experimentally induced by exposure to microwave radiation. IRE Trans Med Electron 7:152–157.

Carpenter RL, Hagan GJ, Ferri ES. 1975. Use of a dielectric lens for experimental microwave irradiation of the eye. Ann NY Acad Sci 247:142–154.

Carpenter RL, Hagan GJ, Donovan GL. 1977. Are microwave cataracts thermally caused? In: Hazzard DG, editor. Symposium on biological effects and measurement of radio frequency/microwaves. Rockville, MD: HEW Publication (FDA) 77-8026. pp 352–379.

Chou C-K, Guy AW, McDougall JA, Han L-F. 1982. Effects of continuous and pulsed chronic microwave exposure on rabbits. Radio Sci 17(Number 5S):185S–193S.

Chou C-K, Guy AW, Borneman LE, Kunz LL, Kramar P. 1983. Chronic exposure of rabbits to 0.5 and 5 mW/cm$^2$ CW microwave radiation. Bioelectromagnetics 4:63–77.

Cleary SF, Pasternak BS. 1966. Lenticular changes in microwave workers: A statistical study. Arch Environ Health 12:23–29.

Cleary SF, Pasternak BS, Beebe GW. 1965. Cataract incidence in radar workers. Arch Environ Health 11:179–182.

Cogan DG, Fricker SJ, Lubin M, Donaldson DD, Hardy H. 1958. Cataracts and ultra-high-frequency radiation. AMA Arch Ind Health 18:299–302.

COMAR. 2002. Medical aspects of radiofrequency radiation overexposure. IEEE Committee on Man and Radiation (COMAR). Health Phys 82:387–391.

Creighton MO, Larsen LE, Stewart-DeHaan PJ, Jacobi JH, Sanwal M, Baskerville JC, Bassen HE, Brown DO, Trevithick JR. 1987. In vitro studies of microwave-induced cataract. II. Comparison of damage observed for continuous wave and pulsed microwaves. Exp Eye Res 45:357–373.

Daily L Jr, Wakim KG, Herrick JF, Parkhill EM, Benedict WL. 1950a. The effects of microwave diathermy on the eye. Am J Opthalmol 33:1241–1254.

Daily L Jr, Wakim KG, Herrick JF, Parkhill EM, Benedict WL. 1950b. The effects of microwave diathermy on the eye of the rabbit. Am J Opthalmol 35:1001–1017.

Durney CH, Johnson CC, Barber PW, Massoudi H, Iskander MF, Lords JL, Ryser DK, Allen SJ, Mitchell JC. 1978. Radiofrequency radiation dosimetry handbook. 2nd edition. Report SAM-TR-78-22. Brooks Air Force Base, TX: USAF School of Aerospace Medicine. 141 p.

Elder JA. 1984. Special senses. In: Elder JA, Cahill DF, editors. Biological effects of radiofrequency radiation. Washington, DC 20460: Environmental Protection Agency (EPA-600/8-83-026F). pp 5-64–5-78. (available from National Technical Information Service, 5285 Port Royal Road, Springfield, VA 22161, Report #PB-85-120-848).

FCC. 1996. Guidelines for evaluating the environmental effects of radiofrequency radiation (FCC 96-326). Washington, DC 20554: Federal Communications Commission (FCC).

Ferri ES, Hagan GJ. 1976. Chronic, low-level exposure of rabbits to microwaves. In: Johnson CC, Shore ML, editors. Biological effects of electromagnetic waves. Vol. 1. Rockville, MD: HEW Publication (FDA) 77-8010. pp 129–142.

Foster MR, Ferri ES, Hagan GJ. 1986. Dosimetric study of microwave cataractogenesis. Bioelectromagnetics 7:129–140.

Frey AH. 1985. Data analysis reveals significant microwave-induced eye damage in humans. J Microwave Power 20: 53–55.

Guy AW, Lin JC, Kramar PO, Emery AF. 1974. Quantification of microwave radiation effects on the eyes of rabbits at 2450 and 918 MHz. Report to Office of Naval Research (Contract No. N00014-67-A-0103-0025) (available from National Technical Information Service, 5285 Port Royal Road, Springfield, VA 22161, Report #AD-A007-521, 39 pages).

Guy AW, Lin JC, Kramar PO, Emery AF. 1975. Effect of 2450-MHz radiation on the rabbit eye. IEEE Trans Microwave Theory Tech MTT-23:492–498.

Guy AW, Kramar PO, Harris CA, Chou CK. 1980. Long-term 2450-MHz CW microwave irradiation of rabbits: Methodology and evaluation of ocular and physiologic effects. J Microw Power 15:37–44.

Hagan GJ, Carpenter RL. 1976. Relative cataractogenic potencies of two microwave frequencies (2.45 and 10 GHz). In: Johnson CC, Shore ML, editors. Biological effects of electromagnetic waves. Vol. 1. Rockville, MD: HEW Publication (FDA) 77-8010. pp 143–155.

Hathaway JA, Stern N, Soles EM, Leighton E. 1977. Ocular medical surveillance on microwave and laser workers. J Occup Med 19:683–688.

Hirata A, Matsuyama S, Shiozawa T. 2000. Temperature rises in the human eye exposed to EM waves in the frequency range 0.6–6 GHz. IEEE Trans Electromagn Compatibility 42: 386–393.

Hirata A, Watanabe H, Shiozawa T. 2002. SAR and temperature increase in the human eye induced by obliquely incident plane wave. IEEE Trans Electromagn Compatibility 44:592–594.

Hirsch SE, Appleton B, Fine BS, Brown PVK. 1977. Effects of repeated microwave irradiations to the albino rabbit eye. Invest Ophthalmol Vis Sci 16:315–319.

Hollows FC, Douglas JB. 1984. Microwave cataract in radiolinemen and controls. Lancet 2(8399):406–407.

Holly EA, Aston DA, Ahn DK, Smith AH. 1996. Intraocular melanoma linked to occupations and chemical exposures. Epidemiology 7:55–61.

Johansen C, Boice JD Jr, McLaughlin JK, Olsen JH. 2001. Cellular telephones and cancer—a nationwide cohort study in Denmark. J Natl Cancer Inst 93:203–207.

Johansen C, Boice JD Jr, McLaughlin JK, Christensen HC, Olsen JH. 2002. Mobile phones and malignant melanoma of the eye. Br J Cancer 86:348–349.

Kamimura Y, Saito K, Saiga T, Amemiya Y. 1994. Effect of 2.45 GHz microwave irradiation on monkey eyes. IEICE Trans Commun E77-B:762–765.

Kinoshita JH, Merola LO, Dikmak E, Carpenter RL. 1966. Biochemical changes in microwave cataracts. Doc Ophthalmol 20:91–103.

Kramar PO, Harris C, Guy AW, Emery AF. 1975. The ocular effects of microwaves on hypothermic rabbits: A study of microwave cataractogenic mechanisms. Ann NY Acad Sci 247: 155–165.

Kramar PO, Harris C, Guy AW, Emery AF. 1976. Mechanism of microwave cataractogenesis in rabbits. In: Johnson CC, Shore ML, editors. Biological effects of electromagnetic waves. Vol. 1. Rockville, MD: HEW Publication (FDA) 77-8010. pp 49–60.

Kramar P, Harris C, Emery AF, Guy AW. 1978. Acute microwave irradiation and cataract formation in rabbits and monkeys. J Microw Power 13:239–249.

Kues HA, Monahan JC. 1993. High-peak-power microwaves: Health hazard assessment. Final report submitted to the U.S. Army Medical Research and Development Command, December 1993, 100 pages (MIPR No. 92MM2600).

Kues HA, Hirst LW, Lutty GA, D'Anna SA, Dunkelberger GR. 1985. Effects of 2.45-GHz microwaves on primate corneal endothelium. Bioelectromagnetics 6:177–188.

Kues HA, Monahan JC, D'Anna SA, McLeod DS, Lutty GA, Koslov S. 1992. Increased sensitivity of the non-human primate eye to microwave radiation following ophthalmic drug treatment. Bioelectromagnetics 13:379–393.

Kues HA, D'Anna SA, Osiander R, Green WR, Monahan JC. 1999. Absence of ocular effects after either single or repeated exposure to 10 mW/cm$^2$ from a 60 GHz source. Bioelectromagnetics 20:463–473.

Lim JI, Fine SL, Kues HA, Johnson MA. 1993. Visual abnormalities associated with high-energy microwave exposure. Retina 13: 230–233.

Lu S-T, Mathur SP, Stuck B, Zwick H, D'Andrea JA, Ziriax JM, Merritt JH, Lutty G, McLeod DS, Johnson M. 2000. Effects of high peak power microwaves on the retina of the Rhesus monkey. Bioelectromagnetics 21:439–454.

Majewska K. 1968. Investigations on the effect of microwaves on the eye. Pol Med J 7:989–994.

McAfee RD, Longacre A Jr, Bishop RR, Elder ST, May JG, Holland MG, Gordon R. 1979. Absence of ocular pathology after repeated exposure of unanesthetized monkeys to 9.3-GHz microwaves. J Microw Power 14:41–44.

McAfee RD, Ortiz-Lugo R, Bishop R, Gordon R. 1983. Absence of deleterious effects of chronic microwave radiation on the eyes of Rhesus monkeys. Ophthalmology 90:1243–1245.

Odland LT. 1973. Radio-frequency energy: A hazard to workers? Ind Med Surg 42:23–26.

Paulsson L-E, Hamnerium Y, Hansson H-A, Sjostrand J. 1979. Retinal damage experimentally induced by microwave radiation at 55 mW/cm$^2$. Acta Ophthalmologica 57:183–197.

Rosenthal SW, Birenbaum L, Kaplan IT, Metlay W, Snyder WZ, Zaret MM. 1976. Effects of 35 and 107 GHz CW microwaves on the rabbit eye. In: Johnson CC, Shore ML, editors. Biological effects of electromagnetic waves. Vol. 1. Rockville, MD: HEW Publication (FDA) 77-8010. pp 110–128.

Shacklett DE, Tredici TJ, Epstein DL. 1975. Evaluation of possible microwave-induced lens changes in the United States Air Force. Aviat Space Environ Med 46:1403–1406.

Shellock FG, Crues JV. 1988. Corneal temperature changes induced by high-field-strength MR imaging with a head coil. Radiology 167:809–811.

Siekierzynski M, Czerski P, Gidynski A, Zydecki S, Czarnecki C, Dziuk E, Jedrezejczak W. 1974. Health surveillance of personnel occupationally exposed to microwaves. III. Lens translucency. Aerospace Med 45:1146–1148.

Stang A, Anastassiou G, Ahrens W, Bromen K, Bornfeld N, Jockel KH. 2001. The possible role of radiofrequency radiation in the development of uveal melanoma. Epidemiology 12:7–12.

Stewart-DeHaan PJ, Creighton MO, Larsen LE, Jacobi JH, Ross WM, Sanwal M, Guo TC, Guo WW, Trevithick JR. 1983. In vitro studies of microwave-induced cataract: Separation of field and heating effects. Exp Eye Res 36:75–90.

Stewart-DeHaan PJ, Creighton MO, Larsen LE, Jacobi JH, Sanwal M, Baskerville JC, Trevithick JR. 1985. In vitro studies of microwave-induced cataract: Reciprocity between exposure duration and dose rate for pulsed microwaves. Exp Eye Res 40:1–13.

Taflove A, Brodwin ME. 1975. Computation of the electromagnetic fields and induced temperatures within a model of the microwave-irradiated human eye. IEEE Trans Microwave Theory Tech MTT-23:888–896.

Van Ummersen CA, Cogan FC. 1976. Effects of microwave radiation on the lens epithelium in the rabbit eye. AMA Arch Ophthalmol 94:828–834.

Weiter JJ, Finch ED, Schultz W, Frattali V. 1975. Ascorbic acid changes in cultured rabbit lenses after microwave irradiation. Ann NY Acad Sci 247:175–181.

Wike EL, Martin EJ. 1985. Comments on Frey's ''Data analysis reveals significant microwave-induced eye damage in humans.'' J Microwave Power 20:181–184.

Williams RJ, McKee A, Finch ED. 1975. Ultrastructural changes in the rabbit lens induced by microwave radiation. Ann NY Acad Sci 247:166–174.