Paper

ACUTE OCULAR INJURIES CAUSED BY 60-GHZ MILLIMETER-WAVE EXPOSURE

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Abstract—**The goal of this study was to examine the clinical course of 60-GHz millimeter-wave induced damages to the rabbit eye and to report experimental conditions that allow reproducible induction of these injuries. The eyes of pigmented rabbits (total number was 40) were irradiated with 60-GHz millimeter-waves using either a horn antenna or one of two lens antennas (6 and 9 mm diameter; φ6, φ9) Morphological changes were assessed by slit-lamp microscopy. Additional assessments included corneal fluorescein staining, iris fluorescein angiography, and lens epithelium light microscopy. Under the standardized eye-antenna positioning, the three antennas caused varying damages to the eyelids or eyeglobes. The most reproducible injuries without concurrent eyelid edema and corneal desiccation were achieved using the φ6 lens antenna: irradiation for 6 min led to an elevation of the corneal surface temperature (reaching 54.2 ± 0.9°C) plus corneal edema and epithelial cell loss. Furthermore, mitotic cells appeared in the pupillary area of the lens epithelium. Anterior uveitis also occurred resulting in acute miosis (from 6.6 ± 1.4 to 2.2 ± 1.4 mm), an increase in flares (from 6.7 ± 0.9 to 334.3 ± 130.8 photons per second), and iris vasodilation or vessel leakage. These findings indicate that the three types of millimeter-wave antennas can cause thermal injuries of varying types and levels. The thermal effects induced by millimeter-waves can apparently penetrate below the surface of the eye.**
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Key words: exposure, occupational; laboratory animals; microwaves; radiation, nonionizing

INTRODUCTION

With recent advances in electromagnetic field (EMF) technology, EMFs with super and extremely high frequencies, including millimeter-waves (MMWs), are likely come into wide-spread use in daily life, with applications ranging from wireless transmission of voice and data to intelligent transport systems such as automobile collision radar. The rapid development of these new technologies is likely to increase public concerns about possible health effects of exposures to very high frequency EMFs, including MMWs.

Safety guidelines on EMF exposures published by various organizations, including the International Commission on Non Ionizing Radiation Protection (ICNIRP 1998) and the International Committee on Electromagnetic Safety (ICES) of the Institute of Electrical and Electronics Engineers (IEEE 2005), cover the frequency range of MMWs, or up to 300 GHz. These guidelines are based on established scientific evidence and are mainly concerned with thermal effects. Few studies, however, address the rationale for the exposure limits at MMW frequencies. The limit values for MMW exposures were derived primarily from extrapolations of experimental data for frequencies up to several GHz, or from the effects of infrared radiation. To confirm the scientific basis for the exposure limits in the MMW region, it is necessary to obtain data derived directly from experiments using MMWs.

Of the human tissues, the eye is considered most vulnerable to MMW-induced injuries as it is located on the surface of body where most energy of MMWs is dissipated. While previous studies have investigated the ocular effects of EMF exposures, there is little specific information on the effects of MMWs. For example, exposure to MMWs can have varying effects on the cornea (Rosenthal et al. 1976; Kues et al. 1999; Chalfin et al. 2002). However, since damage to the corneal epithelium can also be caused by simple desiccation (due to mechanical prevention of blinking; Fujihara et al. 1995),

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some part of the changes may not have been caused by MMW irradiation itself but corneal desiccation.

We have developed an acute ocular injury model and used this model to examine the clinical course of MMW-induced damages. We also determined the experimental conditions leading to reproducible induction of ocular injuries caused by exposure to MMWs.

MATERIALS AND METHODS

Animals

Specific pathogen-free (SPF) male pigmented Dutch-belted rabbits (body weight, 1.6–2.2 kg; age, 12–14 wk; total $N = 40$) were purchased from Sankyo Lab Service Co., Inc. (Toyama, Japan). They were kept in SPF cages with unrestricted access to sterilized food and ultraviolet-irradiated water.

Before the study, the eyes of each rabbit were examined using an SL-130 slit-lamp microscope (ZEISS, Tokyo, Japan) to ensure absence of abnormalities in the anterior segment. Each eye was recorded photographically and the images used as the baseline.

All animal experiments were conducted in accordance with the animal study guidelines established by Kanazawa Medical University and the Association for Research in Vision and Ophthalmology (ARVO) Statement for the Use of Animals in Ophthalmic and Vision Research.

Exposure apparatus and conditions

Each rabbit was immobilized using a Lucite animal holder (specially constructed for the present study). General anesthesia was induced by intramuscular injection of a mixed solution containing 2 mg kg^{-1} xylazine and 20 mg kg^{-1} ketamine. The upper and lower eyelids were retracted with surgical tape to keep the eyes open, and 0.4% oxybuprocaine topical anesthetic was administered to each eye immediately prior to MMW exposure.

Since anesthesia suppressed blinking, to minimize corneal desiccation and the resulting epithelial cell damage, 2% polyvinyl alcohol (My Tear; Senju Pharmaceutical Co., Ltd., Osaka, Japan) eye drops were administered as needed.

All rabbits received topical ofloxacin (Tarivid; Santen Pharmaceutical Co., Ltd., Osaka, Japan) ointment to prevent secondary infection after MMW exposure and or ocular examination.

Temperature and humidity during MMW exposure were maintained at 20–25°C and 33–43%, respectively, through air-conditioning.

Part 1: Differences in ocular injuries caused by three different types of antennas

To ensure maximal ocular damage that could be caused by exposure at 3 W, i.e., with the maximal output of the 3-W transmitter (QBY-603400, Quinstar Technology, Inc., California), a conical horn antenna of diameter 15.5 mm (QWH-VCRROO, Quinstar Technology, Inc., Fig. 1a) was placed 10 mm (half value width, 15 mm diameter) from the corneal apex, and MMW at 475 mW cm^{-2} was delivered to each eye for 30 min.

In addition, using two different lens antennas (specially constructed for the present study), one with a focal distance of 150 mm [half value width at focal point:

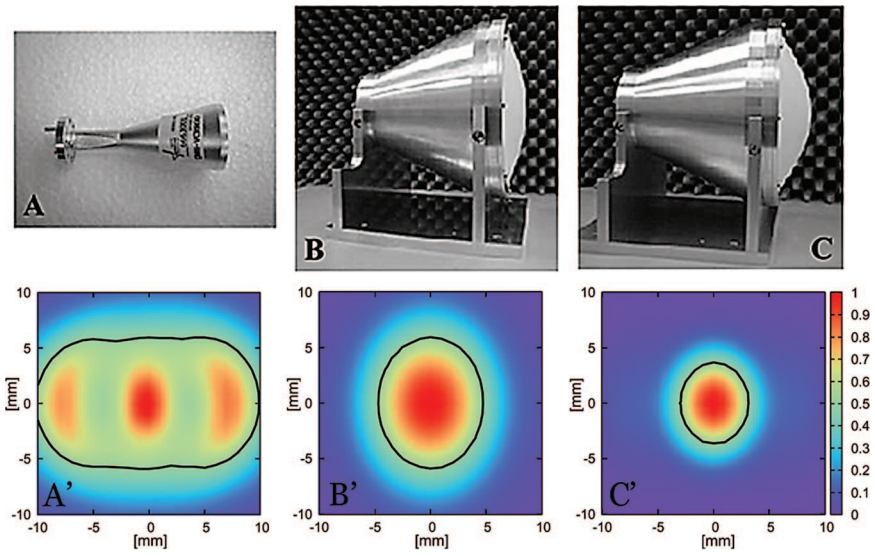

Fig. 1. Three different antennas. (A) Horn antenna, (B) φ9 lens antenna with a 250 mm focal point (half value width at focal point, approximately 9 mm diameter, and (C) φ6 lens antenna with a 150 mm focal point (half value width at focal point, approximately 6 mm diameter). A′, B′, C′ indicate the power density distribution of these antennas. The major-minor axis lines denote the 50% area of maximum power density.

approximately 6 mm diameter (the φ6 lens antenna), Fig. 1c] and the other a focal distance of 250 mm [half value width at focal point: approximately 9 mm diameter (the φ9 lens antenna), Fig. 1b], rabbit eyes were exposed at 1,898 mW cm^{-2} for 6 min.

MMWs irradiated one eye of each rabbit. Each group comprised 6–10 rabbits. The extent or severity of anterior uveitis (iritis and cyclitis) was quantified based on changes in the pupil diameter. In addition, to assess corneal epithelial damage, the corneas were stained with fluorescein strips wetted with normal saline immediately after exposure with follow-ups after 1 and 3 d.

The eye position relative to the antenna was a very important factor when inducing ocular damages. If the exposure areas also encompassed the eyelids, an acute swelling of the eyelids during exposure tended to prevent MMWs from entering the eyeglobe, thereby affecting the final outcome. A standardized exposure position of the eye was therefore established as follows:

1. The distance from the antenna surface to the exposure point of the eye was accurately determined using two laser pointers;
2. The apex of the cornea of each rabbit was positioned at the exposure point established in (1); and
3. With thermography (Neo Thermo TVS-700, Nippon Avionics Co., Ltd., Tokyo, Japan), the center of exposure was set at the center of the pupillary area.

Thermography was used for centering the exposure positioning and recording ocular surface temperature immediately before beginning exposure and 30 s before the end of the exposure.

Part 2: Acute ocular injury by 60-GHz MMWs using the φ6 lens antenna

Using the standardized experimental setup as in Part 1 above, MMWs with a power density of 1,898 mW cm^{-2} were transmitted to the rabbit eye for 6 min using the φ6 lens antenna (a focal distance: 150 mm), and the extent of ocular damages assessed. Thermography was also used to record ocular surface temperatures. This experimental group was comprised of 8 rabbits.

Flare in the aqueous humor, or the scattering light by small particles, which indicates ocular inflammation (Sawa et al. 1988), was measured using a FC-2000 laser flare cell meter (Kowa, Tokyo, Japan).

To determine whether MMW exposure could cause ocular barrier breakdown or iris vessel dilatation, fluorescein angiography was performed using another set of 6 rabbits. Immediately after a 1,898 mW cm^{-2} exposure, 1 mL of 10% fluorescein sodium was injected into the ear vein. Iris vessels were examined using a slit-lamp microscope equipped with a fluorescence excitation filter (blue filter) and an excitation light cut filter (yellow filter).

To observe damages to the crystalline lens, lenses from enucleated eyes were fixed with acetic acid (100%) and ethanol (100%) mixed solution (volume per volume = 1:3) and flat preparations of the epithelium obtained. Lens epithelial cells were examined by light microscopy with Mayer's hematoxylin staining.

Data analysis

Wherever appropriate the data were analyzed using Student's two-tailed t test, and a p-value <0.05 was considered statistically significant.

RESULTS

The type and the extent of ocular injuries induced by the three different kinds of antennas varied greatly and the results are summarized in Table 1.

In all eyes, reduction of the pupil size and increase of the flare value were both seen immediately after the MMW irradiation. However, individual values in the horn antenna group and the φ9 lens antenna group varied greatly. For example, even though the flare value was elevated to 220–522 photons per second in 2 out of 4 rabbits in the horn antenna group and to 27–86 photons per second in 1 out of 4 rabbits in the φ9 lens antenna group, the rest showed little elevation. The p-values were therefore 0.15 and 0.11, respectively, in each group. Pupil sizes also varied in the same manner.

On the other hand, the results of the φ6 lens antenna showed a high degree of reproducibility. Both miosis (pupil contraction) and flares were seen in the exposed eyes, and the changes were highly significant (with $p = 0.0001$, $p = 0.015$, respectively).

Horn antenna: 475 mW cm^{-2}, 30 min exposure; the φ9 lens antenna: 1,898 mW cm^{-2}, 5 min exposure; and the φ6 lens antenna: 1,898 mW cm^{-2}, 6 min exposure. Values shown are mean ± SD, or number of positive per all examined rabbits.

The horn antenna. Fig. 2 illustrates the typical ocular damages and the subsequent clinical courses. The maximum corneal surface temperature was 45°C (average temperature: 44.2°C, $N = 4$). Pupil diameter changed from 7.1 mm (Fig. 2a) before the exposure to 4.1 mm (Fig. 2b) after. This result indicates severe anterior uveitis (Duke-Elder 1966). Fluorescein staining of the cornea immediately after the exposure revealed slight epithelial cell damage that was, however, also present in the control eyes. This change could be attributed to desiccation (Fig. 2b) owing to the relatively long exposure time (30 min). It

Table 1. Ocular injures caused by 60-GHz MMWs using different antennas.

Antenna	Examination items	PRE[a]	CTL[b]	EXPO[c]
A. Horn antenna fluence: 1,350 J cm^{-2}	Pupil size (mm)	7.2 ± 0.6	6.7 ± 0.6	3.5 ± 1.2
	Flare (photon per second)	6.1 ± 4.5	6.6 ± 2.7	250.7 ± 220.7
	Epithelial fluorescein staining + (immediately after)	1 out of 4	4 out of 4	4 out of 4
	Corneal edema + (immediately after)	0 out of 4	0 out of 4	4 out of 4
B. ϕ9 lens antenna fluence: 900 J cm^{-2}	Pupil size (mm)	4.9 ± 0.5	4.7 ± 0.6	2.2 ± 1.4
	Flare (photon per second)	6.7 ± 0.9	4.5 ± 1.2	174.7 ± 130.0
	Epithelial fluorescein staining + (immediately after)	1 out of 3	1 out of 3	0 out of 3
	Corneal edema + (immediately after)	0 out of 3	0 out of 3	0 out of 3
	Iris vasodilation (immediately after)	0 out of 3	0 out of 3	3 out of 3
	Corneal edema + (1 d after EXPO)	0 out of 3	0 out of 3	2 out of 2
C. ϕ6 lens antenna fluence: 1,080 cm^{-2}	Pupil size (mm)	6.6 ± 1.4	6.4 ± 1.2	1.9 ± 0.3[d]
	Flare (photon per second)	5.8 ± 3.2	9.9 ± 5.9	334.3 ± 130.8[d]
	Epithelial fluorescein staining + (immediately after)	0 out of 8	1 out of 3	2 out of 3
	Corneal edema + (immediately after)	0 out of 8	0 out of 2	1 out of 3
	Iris vasodilation (immediately after)	0 out of 8	0 out of 8	8 out of 6
	Corneal edema + (1 d after EXPO)	0 out of 8	0 out of 8	8 out of 8

[a] PRE: Pre-exposed eye.
[b] CTL: Non-exposed contralateral eye.
[c] EXPO: Exposed eye.
[d] CTL vs. EXPO $p < 0.05$.

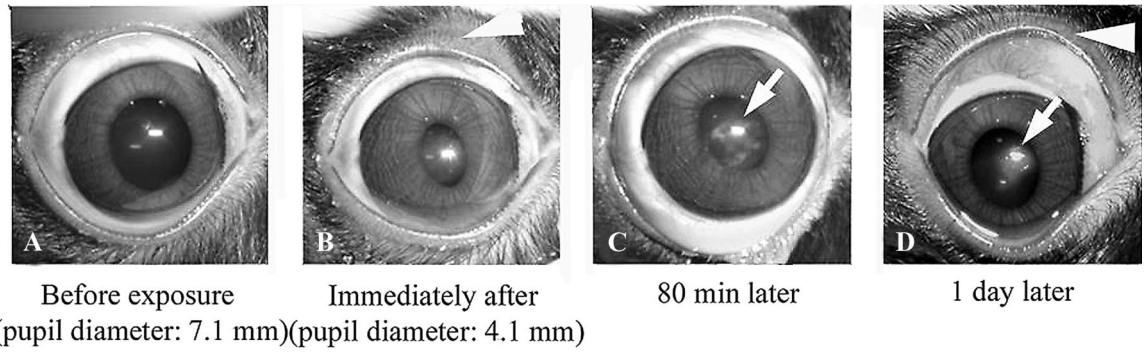

Fig. 2. Ocular damage and clinical course after exposure to 60 GHz 475 mW cm^{-2} for 30 min with a horn antenna. (A) Shows a normal eye before exposure. (B) Shows eyelid edema (arrow head) appearing during irradiation. Dry spots on the cornea and miosis occurred immediately after exposure. Corneal opacity (C, arrow) began to form within 80 min, which further progressed 1 day later (D, arrow).

should be noted that an entirely different type of corneal stromal damage was detected 80 min post-irradiation (Fig. 2c; indicated by arrow). This damage was most likely MMW-induced (see Discussion). And 1 d after the exposure, eyelid edema (Fig. 2d; arrow head) and corneal opacity (Fig. 2d; arrow) both further progressed.

In fact, hot or burn spots appeared in the eyelids immediately after irradiation (Fig. 2b, indicated by arrow head). Therefore, MMW exposure with the horn antenna affected not only the eyeglobe, but also the adjacent skin areas including the eyelids. When the eyelids became acutely edematous, there was a narrowing of the palpebral fissure. As a result, intraocular damages were minimized. This explained why some rabbits showed lower flare value change.

The φ9 lens antenna. The severity of inflammatory response in rabbits irradiated at 1,898 mW cm^{-2} for 5 min is shown in Fig. 3. Corneal surface temperature reached 45–48°C ($N = 3$). Similar to horn antenna irradiation, eyelids exposed to MMWs using the φ9 lens antenna showed resulting eyelid edema (Fig. 3a) and narrowing of the palpebral fissure. The pupil size remained unchanged (i.e., no intraocular inflammation was induced) and the flare value changed in only 1 of 4 rabbits (Table 1). Again, the partial lid closure due to edema allowed insufficient electromagnetic radiation to reach and enter the eyeglobe. When MMW-induced eyelid edema occurred, the surgical tape could no longer keep the eyelid open. Only when the eyeglobe was exposed sufficiently did the pupil size decrease and the flare value increase (to over

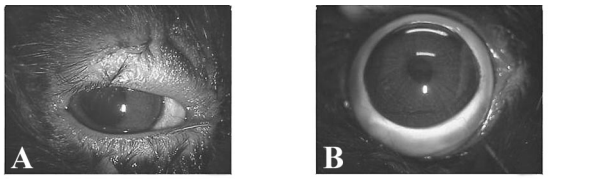

Fig. 3. Ocular conditions after MMW irradiation at 60 GHz 1,898 mW cm^{-2} for 5 min using the φ9 lens antenna with a focal distance of 250 mm. (A) Eyelids exposed to MMWs, showing narrowing of the palpebral fissure; (B) MMW irradiation to the eyeglobe (no exposure to eyelids) was possible in the absence of lid edema.

200 photons s^{-1} in 2 of 3 rabbits). Since individual rabbits responded differently, the φ9 lens antenna could not reproducibly induce intraocular inflammation.

The φ6 lens antenna. Acute intraocular injury was consistently induced (in 8 out of 8 rabbits) using the φ6 lens antenna with a focal distance of 150 mm. There were no hot spots on or around the eyelids, hence no lid edema or closure.

Fig. 4 records the course of ocular damages caused by MMW exposure at 1,898 mW cm^{-2}. The corneal temperature rose rapidly immediately after the start of exposure, reaching a plateau within 1 min. Maximum corneal surface temperature reached 54.2 ± 0.9°C (N = 8). And immediately after irradiation, miosis (pupil contraction; 8 out of 8 rabbits, MMW-exposed eyes: 1.74 ± 1.02 mm pupil diameter, non-exposed control eyes: 5.17 ± 1.08 mm, $p < 0.001$) and iris vasodilation (6 out of 8 rabbits) were both noted. Starting 1 day after irradiation, corneal opacity (8 out of 8 rabbits), corneal epithelial damage (8 out of 8 rabbits), and ciliary injection (6 out of 8 rabbits) were confirmed. Starting 3 d after the exposure, corneal opacity (corneal stromal edema) and ciliary injection peaked, although these symptoms gradually improved. However, corneal opacity progressed to corneal scarring by 7 wk after exposure. Corneal epithelial cell damage peaked 1 d after the exposure, and mild damage persisted for up to 8 d post-exposure followed by total resolution. While slight variations were observed, these findings were common to all 8 rabbits in this experimental group.

Fig. 5 shows the results of angiography of iris vessels. In unexposed control eyes, only iris vessels appeared filled, with no fluorescein leakage into the anterior chamber. However, in the irradiated eyes, leakage of the fluorescein was clearly seen that originated from areas with vasodilation.

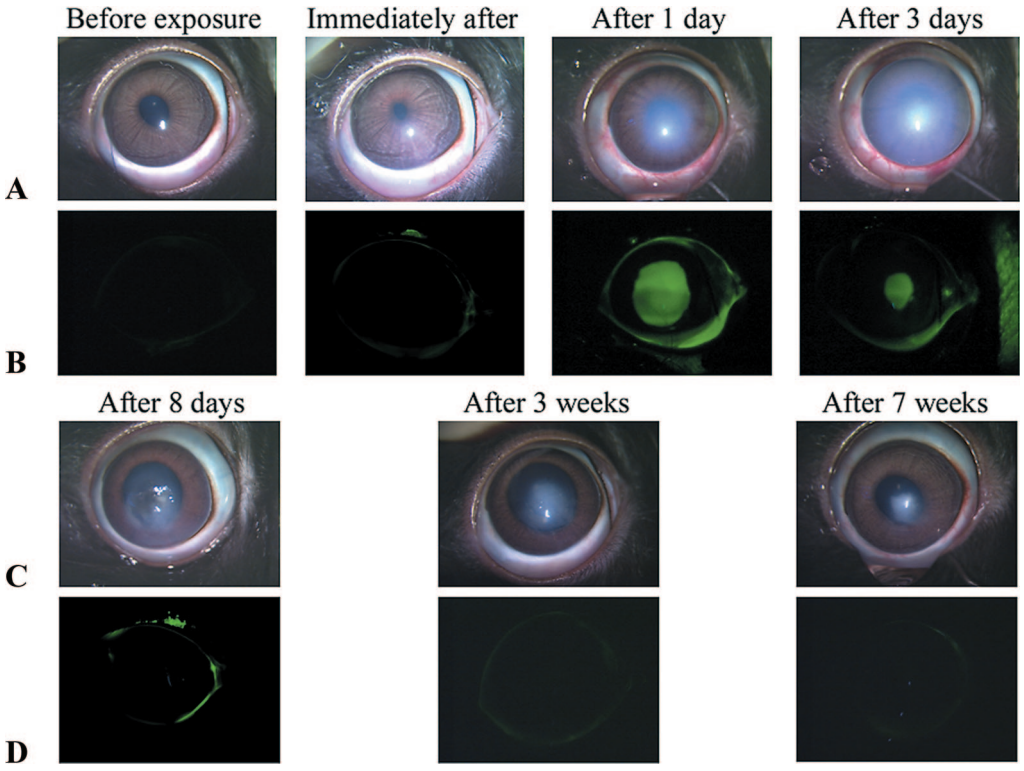

Fig. 4. Clinical course of ocular damages after MMW exposure at 60 GHz 1,898 mW cm^{-2} for 6 min using the φ6 lens antenna with a focal distance of 150 mm. (A) and (C) Anterior segment images, (B) and (D): Fluorescein-stained images.

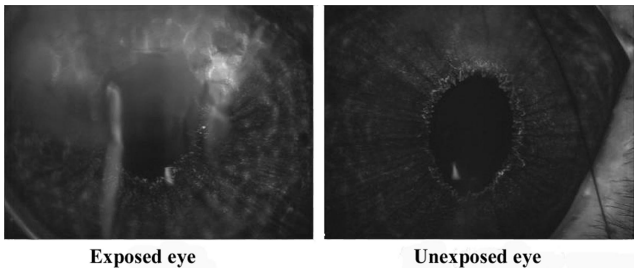

Fig. 5. Iris angiography. In the control eyes, only iris vessels were filled with no leakage of fluorescein into the anterior chamber. In MMW-exposed eyes, leakage of the fluorescein was seen that originated from the areas with vasodilation.

Flat preparations from lens epithelium 3 d after MMW irradiation exhibited mitotic cells within the pupillary area (Fig. 6). No mitotic cells were observed in the non-exposed contralateral eyes. These cells are normally absent from central lens epithelium as cells in this area do not proliferate (Paterson and Delamere 1992). On the other hand, injured or dead cells are usually eliminated through phagocytosis by normal cells (Shui et al. 2000). And to fill in the vacant space, an adjacent normal cell would begin to divide. The presence of the mitotic cells therefore indicates repairing of lens damages that, in the present case, occurred 3 days after the MMW irradiation.

DISCUSSION

Several previous studies have examined ocular injuries caused by MMW exposure and the results ranged from no damage to only mild corneal change. For example, 60-GHz MMWs at 10 mW cm^{-2} did not seem to cause any ocular damages (Kues et al. 1999); 35-GHz MMWs at 2 W cm^{-2} for 1.5–5 s induced corneal damages that healed within 24 h (Chalfin et al. 2002); and 35- or 107-GHz MMWs at 50 mW cm^{-2} for 15–80 min induced both epithelial damage and stromal edema in the cornea—although they were transient and both began to recover on the next day (Rosenthal et al. 1976). None of the above was designed to investigate ocular injuries caused by high-intensity MMW exposure. And none followed the clinical course of the injuries. Therefore, a comparison between the results of these studies and ours is not feasible.

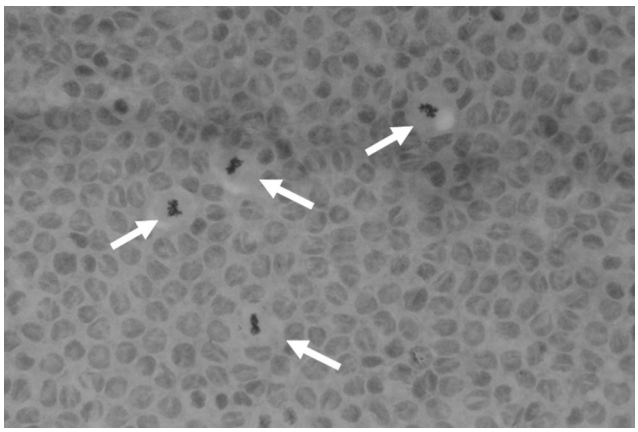

Fig. 6. Lens epithelium from the pupillary area 3 d after MMW exposure. Arrows indicate mitotic cells, suggestive of injury to these areas. Magnification, 400×.

In fact, the types of antennas can also dictate the entire experimental outcome. Previous studies have employed either a circular horn antenna (Rosenthal et al. 1976; Kues et al. 1999) or an open-ended waveguide (Chalfin et al. 2002). In our study, a circular horn antenna and two lens antennas (the φ6 and the φ9 mm) were used. Khizhnyak and Ziskin (1994) mentioned that the horn antenna, depending its size and shape, gave non-uniform heating patterns, and local specific absorption rates (SAR) could exceed the average by a factor of over 10. From our studies, the reason for the inconsistent induction of intraocular damages with either the horn antenna or the φ9 mm lens antenna became clear. Konno et al. (2005), who have previously measured the distribution of power density of the horn and lens antennas used in our study, have reported that the half value width 60 mm from the antenna (with an irradiated elliptical area of 17.7 mm in major axis and 16.1 mm in minor axis) covered the entire upper and lower eyelids of the rabbits (anatomical dimensions: major axis: 16.2 $\pm$ 0.7 mm; minor axis: 9.2 $\pm$ 1.6 mm; $N = 10$). In contrast, with the φ9 mm lens antenna the half value widths were major axis: 9.7 mm and minor axis: 9.5 mm. This explains why sometimes intraocular damages with the φ9 mm lens antenna could be induced, while other times only eyelid edema with little or no intraocular changes. And the degree of edema is further complicated by the individual rabbit's own physiologic response. As the edematous lids began to encroach into the corneal area, the amount of MMWs entering the eye was no longer uniform (Fig. 3). We have observed the same phenomenon when measuring intraocular temperatures following infrared exposure (Kojima et al. 2002). We therefore decided to use a lens antenna that could limit the area of MMW exposure such as that of the φ6 mm lens antenna, together with an XYZ stage to accurately center the exposure at the apex of the cornea (i.e., the center of the pupillary area). Under this condition, maximum output can induce acute injuries or changes that include miosis, iris vasodilation, corneal opacity, corneal epithelial damage, and ciliary injection.

In the present study, transient corneal epithelial damage was seen in both exposed and unexposed contralateral (control) eyes after a long 30-min exposure at 475 mW cm^{-2} with the horn antenna.

Inhibition of normal rabbit blinking has been found to lead to corneal epithelial damage due to corneal desiccation (Fujihara et al. 1995). In order to avoid damage caused by corneal desiccation, we used 2% polyvinyl alcohol to inhibit desiccation although this solution might be expected to change the absorption characteristics of the MMWs.

In contrast to the other two types of lens antenna, the φ6 mm lens antenna allows delivery of stronger MMWs directly to the center of the pupillary area without causing eyelid edema. This enabled us to shorten the exposure time (e.g., 6 min), and reduce the incidence of transient cornea epithelial injury caused by desiccation as in the case of the horn antenna. Therefore, only the type of corneal epithelial damage that occurred at the site of exposure and persisted for more than 1 d could be attributed to MMW exposure as that with the φ6 mm lens antenna.

CONCLUSION

Our study has shown that both the horn and the φ9 mm lens antennas can cause thermal burns to the eyelids that eventually affected the degree of intraocular injury. On the other hand, ocular damages can be induced with a high degree of reproducibility by using the φ6 mm lens antenna. The severity, clinical course, and the healing process can all be quantitatively analyzed. And with the φ6 mm lens antenna, iris vasodilation was also seen. The vessels dilated immediately after a 6-min exposure at 1,898 mW cm^{-2} in all 8 rabbits (Fig. 5) suggesting that MMW-generated heat traveled to the iris, possibly via the circulating aqueous humor. The mechanisms involved in the above phenomenon are not yet completely understood at present.

Future investigation may include the analysis of irradiation parameters, exposure areas and duration, and the relationship of heat generated by MMWs and absorption by ocular tissues.

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