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Radiation Hazards
(Non-Ionizing Radiations - Biologic Effects and Safety Considerations)
Biological Effects, Critical Organs, Health Implications

EFFECTS ON THE EYE

[Z electromagnetic radiation at microwave frequencies]

by

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SUMMARY

Studies of the biological effects of electromagnetic radiation (EMR) exposure often cite lens opacification as a major threat to man. The purpose of this paper is to analyze, collectively, the EMR research studies on ocular effects and provide an overview of the practical aspects of this problem today. The principal conclusions from this effort are: (1) The acute thermal insult from high intensity EMR fields is cataractogenic if intraocular temperatures reach 45-55° C. (2) The EMR exposure threshold is about 100-150 mW/cm^2 applied for about 60-100 minutes. (3) There does not appear to be a cumulative effect from EMR exposures unless each single exposure is sufficient to produce some irreparable degree of injury to the eye.

INTRODUCTION

The fact that microwaves can produce lenticular opacities of the eye has been known for over two decades (1-6). In any general assessment of the biological effects of electromagnetic radiation fields, the eye is often identified as the principal organ of concern. However, the bulk of available experimental evidence (7-10) supports the position that electromagnetic (microwave/radiofrequency) radiation (EMR) exposures greater than 100 mW/cm^2 for periods longer than an hour are required to produce lens opacification. Validation and acceptance of this threshold value for EMR cataractogenesis have a profound effect on future research needs to assess the consequences of man's exposure in EMR fields.

EVIDENCE OF EMR CATARACTOGENESIS

Numerous experiments have been conducted to determine the EMR thresholds for production of lenticular opacities as a function of frequency, power density, and exposure time. Extensive reviews of such data are reported in the open literature (7, 8, 11). The following table provides a summary of research findings and conclusions of representative studies.

SUMMARY OF EXPERIMENTAL EVIDENCE OF EMR CATARACTOGENESIS

Refs	Investigator/ Author, Date	Freq. (MHz)	Animal	Exposure Profile	Results/Conclusions
2	Richardson A.W., 1948	2,400 10,000	Rabbits	100 W output, eyes 5 cm from source, temp at posterior pole of lens as high as 55° C.	32 of 54 irradiated eyes developed lenticular opacities. Concluded - thermal effect.
3	Osborne, S.L., 1948	2,450	Dogs	Eyes exposed to 350-450 mW/cm^2 for 20 min per exposure over 3-week period.	No evidence of damage to the eyes.
5, 6	Daily, L., 1950-1952	2,450	Dogs Rabbits	100 W output, intraocular temp rise 3° C in dogs. Repeated exposures with 2-5 inches space between "C" director and cornea for 10-30 minutes.	Anterior cortical cataracts developed in 24 hrs and regressed over 9 wks. Posterior cortical cataracts developed over 9 wks. Under same exposure conditions for rabbits, 7 of 17 albino and 3 of 17 pigmented rabbits developed cataracts.
				6-10 exposures of the eyes of dogs to 300 mW/cm^2 for 30 min.	Failed to show any ocular damage.

SUMMARY (Continued)

Refs	Investigator/ Author, Date	Freq. (MHz)	Animal	Exposure Profile	Results/Conclusions
4	Richardson A.W., 1951	10,000	Rabbits	34-67 W output, pulsed exposures, 3-5 min, at 5 cm distance.	16 of 21 rabbits developed opacities within 60 days.
12, 13	Williams, D.B., 1955	2,450	Rabbits	5 min at 590 mW/cm^2 to 90 min at 290 mW/cm^2. Intraocular temps, 49-53° C.	Cataracts developed over 1-14 day latent period. Threshold ~120 mW/cm^2.
8, 14	Addington, C.H., 1958-1959	200	Guinea pigs Dogs Mice	Free space exposures of 50 to 350 mW/cm^2, 60 min/day; 3, 5, or 7 day/wk or continuously for periods up to 45 wks. Average increase in rectal temp < 2° F.	No evidence of lens change could be found
8, 15	Cogan, D.G., 1958	400	Rabbits	60 mW/cm^2 within waveguide and in free space	No cataracts produced with whole body exposures near lethal levels.
7, 8	Carpenter, R.L., 1958-1960, 1968, 1972	2,450	Rabbits	50 mW/cm^2 to 120 mW/cm^2, 1 hr/day for 20 consecutive days. Continuous (CW) and Pulsed (P).	Cataract threshold ≃120 mW/cm^2 for cumulative exposures, 1-6 day latent period for appearance of cataracts. CW vs P exposures inconclusive.
23	Reider, D.R., 1971	20	Primates	Four rhesus monkeys exposed to pulsed fields 180 mW/cm^2 for 4 hours.	Eye examination 1, 4, & 7 days postexposure and weekly for 8 weeks revealed no ocular change.
16	Birenbaum, L., 1969	5,500 (800 - 6,300)	Rabbits	100 exposures to pulsed fields. 62 exposures to CW fields.	Thresholds for cataracts similar to Carpenter data (~120 mW/cm^2), latent period 4 days. No detectable difference between P and CW exposures. Effectiveness of radiation diminished with decreasing frequency.
8	Baillie, H.D., 1969	2,500	Dogs	5000 mW/cm^2 exposures under hypothermic conditions (cooled to 22° C).	Without cooling, immediate and delayed cataracts were produced. Under hypothermic conditions no cataracts were produced even with repeated exposures. Concluded - Cataract production is thermal effect.
7, 8	Michaelson, S.M., 1961-1974	2,800	Rabbits	Free space exposures 220-240 mW/cm^2 for one hour.	Produced rapid and complete opacification (also profound thermal effects were observed).
		2,800	Dogs	Pulsed exposures at 165 mW/cm^2 for 3 hrs in a single exposure or 6 hr/day for 3 wks.	Did not produce any lenticular changes for several years after irradiation.
		1,280	Dogs	Pulsed fields at 20, 50, or 100 mW/cm^2, 6 hr/day, 5 days/wk, 2-4 wks.	Periodic examination for 12 months after exposures did not reveal abnormalities of the lens or retina.

SUMMARY (Continued)

Refs	Investigator/ Author, Date	Freq. (MHz)	Animal	Exposure Profile	Results/Conclusions
7, 8	Michaelson, S.M., 1961-1974	24,000	Dogs	Pulsed fields, ~6 hr/day, 5 days/wk, or ~16 hr/day, 4 days/wk, for 20 months. (In these exposures the dogs were free to move around.)	No eye abnormalities in lens or retina.
		2,800	Dogs	Single or fractionated exposures to 350 mW/cm^2 for 20 min.	No permanent lenticular alterations.
				Single or fractionated exposures to 700 mW/cm^2 for 20 min.	Resulted in lens opacification.
9, 10	Guy, A.W., 1974 Kramar, P.O., 1975	2,450	Rabbits	Exposure levels from ~100 mW/cm^2 to 500 mW/cm^2 for 10-100 minutes.	Exposure threshold for cataract production was 150 mW/cm^2 for 100 min. Data suggest critical temperature for cataractogenesis is ~43° C.
		2,450	Rabbits	Exposed to same exposure levels under hypothermic conditions.	Concluded that single potentially cataractogenic exposures will not injure the eye under conditions of controlled general hypothermia. Conclusion - Heat alone is responsible for damage to the lens following single, high-level irradiation.
		2,450	Rabbits	Exposure levels - 100 mW/cm^2, 30 min/day, 4 days; 100 mW/cm^2, 60 min/day, 5-9 days; 100 mW/cm^2, 120 min/day, 8-9 days.	Periodic exams for six months after exposure revealed no ocular damage.
		918	Rabbits	Exposure 1400 mW/cm^2 for 30 min.	Concluded threshold for cataractogenesis is higher for this frequency when compared to 2450.
24	Williams, R.J., 1974	2,450 2,860	Rabbits	Multiple exposure CW and pulsed, 225 mW/cm^2, 20-30 min daily for up to 5 weeks.	Radiation did not appear to influence the normal cornea. No detectable effect.
21	Appleton, B. 1975	3,000	Rabbits	100 or 200 mW/cm^2 for 15 or 30 min.	Examination daily for 14 days, weekly for one month, and monthly for a year revealed no ocular changes.
				300, 400, or 500 mW/cm^2 for 15 min.	Acute ocular changes during exposure. Animal deaths occurred after 30 min @ 300 mW/cm^2 and 15 min @ 500 mW/cm^2. No lens changes or cataracts were noted at one year postexposure.

SUMMARY (Continued)

Refs	Investigator/ Author, Date	Freq. (MHz)	Animal	Exposure Profile	Results/Conclusions
26	Williams, R.J., 1975	2,450	Rabbits	250 mW/cm^2, 20 min/day, 5 day/wk for 6 wks. 165 mW/cm^2, 20 min - 2 times daily, 5 day/wk, for 3 wks.	Electron microscopy revealed prominent ultrastructural changes in one lens that had appeared normal by slit lamp biomicroscopy.

The fact that these studies were conducted over a span of 25 years poses some difficulty in comparing the research results and conclusions, particularly considering the lack of quantitative dosimetry in some of the earlier investigations. However, taken collectively, they reveal certain consistencies which must be considered in an analysis of EMR cataractogenesis. The acute thermal insult appears as a primary mechanism for producing eye trauma leading to lens opacities, but is effective only above some power density-time threshold. These studies indicate this threshold value is greater than 100 mW/cm^2 applied for more than an hour. Although most of the experimental work has been conducted using 2450 MHz radiation sources, the data suggest that lower frequencies require more intense radiation exposures to produce comparable lenticular damage. This is logical from the standpoint of thermal insult since EMR energy transfer to biological tissue is frequency dependent, with the higher frequencies producing the maximum energy density.

The studies conducted under hypothermic conditions provide remarkably strong evidence that heat alone is responsible for ocular lens damage following single high-intensity EMR exposures. Thus, time of exposure is also a critical parameter for lens injury.

In studies where the radiation was applied to the whole body of the animal, lethality often resulted. In a practical sense, this should reduce the concern for acute eye injuries from EMR exposures. Studies such as these cause questions to be raised concerning the selection of test subjects and extrapolation of the research findings to man. However, it is believed that the EMR exposures used in these experiments represent a worst case biological insult, i.e., these exposures were more traumatic to the animals than they would be to man.

Many different types of retrospective studies have been conducted in an attempt to gain useful data from actual or suspected exposure of human populations to EMR fields. One of the earliest of such studies was performed in 1943 (7, 8, 17) on 45 military radar operators. In 1958, another study of 335 microwave workers was reported (7, 8, 18). Neither survey revealed any significant findings. A more extensive but different type of study (7, 8, 19) of the records of 2,946 World War II and Korean veterans treated by the U.S. Veterans Administration Hospitals for cataracts compared to those of 2,164 veterans without cataracts was made to determine if the cataract incidence could be related to greater occupational risk (exposure to EMR). It was concluded that the group occupationally exposed or associated with microwaves exhibited no increased risks of cataracts. References 7 and 8 discuss, in detail, the major controversies concerning the interpretation and validity of a number of occupational surveys and individual case reports.

In spite of repeated attempts to analyze and apply data from retrospective studies, little has been gained from such efforts. However, the following general observations are worthy of consideration:

(1) Human data alone does not provide conclusive evidence that EMR produces cataracts in man.

(2) Some surveys may indicate statistically significant increases in lenticular defects in microwave workers, but none has shown any clinically significant defects in terms of decreased visual acuity, i.e., no apparent loss of functional vision.

(3) Case reports of diathermy treatment in the area of the eye using multiple exposures at power densities of 80-240 mW/cm^2 did not result in production of cataracts.

(4) The exposure levels with which clinically significant cataracts have been tenuously associated indicate the cataractogenic threshold is over 100 mW/cm^2 for man.

(5) Human populations, including groups that work with or near EMR emitters, are rarely subjected to fields having average power densities greater than about 1 mW/cm^2 and in most cases the fields are lower.

DISCUSSION

Interpretation and significance of controlled research studies and retrospective surveys of various population groups will be debated for many years to come. The controversies will include applicability of specific laboratory procedures used to administer and measure EMR fields and the tools and

techniques used to quantitate biological response. The current state-of-knowledge concerning EMR effects on the eye may be summarized briefly as follows:

The acute thermal insult resulting from EMR exposures is believed to be the predominant mechanism responsible for the production of lenticular opacities in the eye (7, 9). It appears that intraocular temperatures in the range of ~45-55° C must be reached before opacities develop. Thus, cumulative effects of EMR exposures would not be anticipated unless each single exposure exceeded the critical threshold level necessary to produce some degree of irreparable injury. Based on the experimental evidence summarized herein, the threshold level is greater than 100 mW/cm^2 applied for more than one hour. A latency period of several days is indicated for the development of cataracts. Additionally, Michaelson reports (7), "No one has yet been able to produce cataracts even by repetitive exposures when the power density is really below threshold." Appleton, who has been actively engaged in clinical surveys of numerous military population groups (20) and microwave research studies (21), further states: "1. Lens damage probably has not occurred in humans from cumulative exposure to low levels of microwave energy. 2. Lens damage probably could not occur in a human from acute exposure to microwave energy without associated severe facial burns." (22).

While the emphasis in past research studies and in this paper is on acute EMR cataractogenesis, future studies of the effect of EMR on the eye should consider more subtle indications of energy transfer, such as alterations in lens protein and/or ultrastructural changes, and any possible long-term adverse consequences (25, 26).

REFERENCES

1. Daily, L., K. G. Wakim, J. F. Herrick, and E. M. Parkhill. Effects of microwave diathermy on the eye. Am J Physiol 155:432, 1948 (Abstract).

2. Richardson, A. W., T. D. Duane, and H. M. Hines. Experimental lenticular opacities produced by microwave irradiation. Arch Phys Med 29:765, 1948.

3. Osborne, S. L. and J. N. Frederick. Microwave radiations. JAMA 137:1036-1404, 1948.

4. Richardson, A. W., T. D. Duane, and H. M. Hines. Experimental cataracts produced by 3-centimeter pulsed microwave irradiation. AMA Arch Ophthalmol 45:382-386, 1951.

5. Daily, L., K. G. Wakim, J. F. Herrick, and E. M. Parkhill. The effects of microwave diathermy on the eye: an experimental study. Am J Ophthalmol 33:1241-1254, 1950.

6. Daily, L., K. G. Wakim, J. F. Herrick, E. M. Parkhill, and W. L. Benedict. The effects of microwave diathermy on the eye of the rabbit. Am J. Ophthalmol 35:1001-1017, 1952.

7. Michaelson, S. M. Effects of exposures to microwaves: problems and perspectives. Environ Health Persp, Vol 8, pp 133-156, 1974.

8. Milroy, Wm. C. and S. M. Michaelson. Microwave cataractogenesis: a critical review of the literature. Aerosp Med, Vol 43, No. 1, Jan 72.

9. Guy, A. W., J. C. Lin, P. O. Kramar, and A. F. Emery. Quantitation of microwave radiation effects on the eyes of rabbits at 2450 MHz and 918 MHz. University of Washington Scientific Report No. 2, January 1974.

10. Kramar, P. O., A. F. Emery, A. W. Guy, and J. C. Lin. The ocular effects of microwaves on hypothermic rabbits: a study of microwave cataractogenic mechanisms. Ann NY Acad Sci, Vol 247, Feb 1975, pp 155-163.

11. Cleary, S. F. Biological effects of microwave and radiofrequency radiation. CRC Crit Rev in Environ Control, June 1970.

12. Williams, D. B., J. P. Monahan, Wm. J. Nicholson, and J. J. Aldrich. Biologic effects of studies on microwave radiation: time and power thresholds for the production of lens opacities by 12.3 cm microwaves. USAF School of Aviation Medicine Report 55-94, August 1955.

13. Williams, D. B., J. P. Monahan, Wm. J. Nicholson, and J. J. Aldrich. Biologic effects of studies on microwave radiation: time and power thresholds for the production of lens opacities by 12.3 cm microwaves. AMA Arch Ophthalmol 54:863-874, 1955.

14. Addington, C., F. Fischer, R. Neubauer, C. Osborn, Y. Sarkees, and G. Swartz. Review of work conducted by the University of Buffalo: studies on the biological effects of 200 Mc. Proceedings Second Ann Tri-Serv Conf on Biol Effects of Microwave Energy; RADC-TR-54, 1958, pp 189-201.

REFERENCES (Continued)

15. Cogan, D. G., S. J. Fricker, M. Lubin, D. D. Donaldson, and H. Hardy. Cataracts and ultra-high frequency radiation. AMA Arch Ind Health 18:299-302, 1958.

16. Birenbaum, L., G. M. Grosof, S. W. Rosenthal, and M. M. Zaret. Effect of microwaves on the eye. IEEE Trans BME-16:7-14, 1969.

17. Daily, L. A clinical study of the results of exposure of laboratory personnel to radar and high frequency radio. U.S. Navy Med Bull 41:1052-1056, 1943.

18. Barron, C. I. and A. A. Baraff. Medical considerations of exposure to microwaves (radar). JAMA 168:1194-1199, 1958.

19. Cleary, S. F., B. S. Pasternack, and G. W. Beebe. Cataract incidence in radar workers. Arch Environ Health 11:179-182, 1965.

20. Appleton, Budd. Results of clinical surveys for microwave ocular effects. U.S. Dept of Health, Education, and Welfare, Public Health Service report DHEW Pub. No. (FDA) 73-8031, BRH/DBE 73-3, Feb 1973.

21. Appleton, Budd, S. E. Hirsch, and P. V. K. Brown. Investigation of single-exposure microwave ocular effects at 3000 MHz. Ann NY Acad Sci, Vol 247, pp 125-134, Feb 1975.

22. Appleton, Budd. Commentary: Microwave Cataracts. JAMA Vol 229, No. 4, July 22, 1974.

23. Reider, D. R., D. L. Epstein, and J. H. Kirk. Aeromedical Review: possible cataractogenic effects of radiofrequency radiation. USAF School of Aerospace Medicine Review 3-71, August 1971.

24. Williams, R. J. and E. D. Finch. Examination of the cornea following exposure to microwave radiation. Aerosp Med pp 393-396, April 1974.

25. Weiter, J. J., E. D. Finch, W. Schultz, and V. Frattali. Ascorbic acid changes in cultured rabbit lenses after microwave irradiation. Ann NY Acad Sci, Vol 247, pp 175-181, Feb 1975.

26. Williams, R. J., A. McKee, and E. D. Finch. Ultrastructural changes in the rabbit lens induced by microwave radiation. Ann NY Acad Sci, Vol 247, pp 166-174, Feb 1975.