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INCIDENCE OF LENS OPACITIES IN MICE EXPOSED TO X RAYS AND THERMAL NEUTRONS

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HEALTH AND BIOLOGY

ABSTRACT

The incidence of lens opacities in Swiss mice exposed to 250-KVP X rays and to the mixed radiation of the thermal column of the Los Alamos homogeneous reactor (under conditions described in LA-1408) has been determined at various times after radiation exposure. When an RBE of one was assumed for the gamma contaminant in the thermal column flux, and the RBE for thermal neutrons was calculated from the data collected 30 weeks after radiation exposure, the RBE of thermal neutrons was estimated to be approximately 15, and 1.6×10^9 n/cm^2 was equivalent to one rem. The RBE of thermal neutrons calculated on the basis of the production of lens opacities was approximately nine times the RBE calculated on the basis of the production of lethality at 30 days. The RBE of thermal neutrons based on 30 day lethality was not significantly different from the RBE based on lethality at 30 weeks.

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INCIDENCE OF LENS OPACITIES IN MICE EXPOSED TO X RAYS AND THERMAL NEUTRONS

1. INTRODUCTION

The cataractogenic effect of X rays (1-9), gamma rays (1-4) and neutrons of various energies (9-13) in human beings and experimental animals has been described in great detail. It is generally believed that neutrons are more effective in cataract production than are X rays or gamma rays although considerable uncertainty exists as to the absolute value for biological effectiveness.

A study of the cataractogenic effect of thermal neutrons by Christenberry and Furth (9) indicated that thermal neutrons were at least twice as effective as X rays in the production of lens opacities in mice. Because of the high gamma contaminant in their neutron source, and the relatively few animals and dosages employed, it is difficult to determine from their data any absolute value for relative biological effectiveness of thermal neutrons in the production of lens opacities.

The present study was undertaken to determine the relative effectiveness of a single acute exposure to thermal neutrons and 250-KVP X rays using the production of lens opacities in mice as the biological response. The present report is based on preliminary data obtained in this study.

2. RANDOMIZATION AND HANDLING OF MICE

The mice used in this study were white Swiss females obtained from

Brennan, Harris, Carter, and Langham (14) at the termination of their study of the 30-day lethality produced by thermal neutrons and X rays. Six hundred and twenty three mice surviving various doses of thermal column and X radiation and 216 unirradiated control mice of the same age were available for observation. The animals were marked individually and randomly distributed 10 or 11 to a cage according to a system of random numbers. All the mice received water and Purina laboratory chow ad libitum. At a later date additional mice exposed to thermal column gamma radiation alone were randomized in with the mice mentioned above.

3. RADIATION EXPOSURE TECHNIQUES

The methods of exposure and measurements of radiation dose have been described in detail by Brennan et al (14). The number of mice surviving for 30 days after irradiation (the time at which observations were begun in this study) and the radiation dosages employed are listed in Table 1. The thermal column flux to which the animals were exposed consisted of a high flux of thermal neutrons contaminated with gamma rays having an average effective energy of about 4 Mev. The dose of thermal column exposure is given in rem[(a)] based on the 30-day lethality studies of Brennan et al (14). Their studies showed that one rem of thermal column exposure consisted of 1.04×10^{10} n/cm^2

(a) One rem is that dose of any ionizing radiation which produces a relevant biological effect equal to that produced by one roentgen of high voltage X radiation, other exposure conditions being equal.

plus 0.39 r of gamma contaminant. In this report thermal column exposure or exposure to the thermal column flux signifies exposure to the mixed radiation.

4. METHOD OF EXAMINING MICE

Up to the present, experimental investigations of the cataractogenic effects of radiation have not resulted in a generally accepted definition of cataracts in experimental animals. General practice has been to apply the term "cataract" to abnormal opacities of the lens which may be seen with the slit lamp or the opthalmoscope following radiation exposure. Many of these abnormalities are extremely minute and may even show regression and disappearance on subsequent examinations. The lack of a quantitative description of the degree of lens abnormality constituting a true cataract in experimental animals makes the evaluation and comparison of the results reported by various investigators extremely difficult and complicates the extrapolation of results from experimental animals to man. In this report all lens abnormalities characteristic of radiation damage and visible with the opthalmoscope are referred to as "lens opacities," and the first examination at which an opacity which subsequently persisted was seen was taken as the end point of the latent period for its production. We propose that the term "cataract" be applied only to those lens opacities which on subsequent examination show progression to the point of involving the entire lens.

The eyes of all mice were examined at two to three week intervals beginning 30 days after exposure. Atropine sulfate was dropped into the

eyes and the eyes examined in a darkened room with the opthalmoscope. To avoid bias in the data all mice were observed as unknowns; i.e., the examiner made the observation without any knowledge of the exposure history of the animals. At each examination the location and approximate size of each opacity was sketched. Many of the mice of the Swiss strain normally show a small congenital defect at the posterior suture line of the lens. These congenital opacities were recorded and were discounted in interpreting the occurrence of new opacities following radiation exposure.

Undoubtedly many of the defects observed in the present study were not sufficient to impair vision. Whether or not they will progress to mature cataracts remains to be seen and will be considered in the final report.

5. INCIDENCE OF LENS OPACITIES

5.1 Mice Exposed to X Rays

Observations of the incidence of lens opacities in mice have been made for 30 weeks following the time of radiation. Table 2 shows the incidence of subsequently persisting opacities at various times after X-ray exposure. X-ray dosages of 200 or more roentgens produced opacities in 100 per cent of the mice in 30 weeks. At lower doses the incidence at 30 weeks decreased with decreasing doses. The data also show that increasing the dose decreases the latent period for the production of lens opacities.

5.2 Mice Exposed to Thermal Column Radiation

As in the case of mice exposed to X rays, mice exposed in the thermal column (thermal neutrons plus gamma contaminant) were observed for 30 weeks following exposure. The incidence of subsequently persisting lens opacities occurring at various times for each dose level are tabulated in Table 3. These data show that doses of 51.6 rem and higher produced 100 per cent opacities at 30 weeks. In general, at doses below 51.6 rem the incidence of opacities at 30 weeks decreased with decreasing doses. It is again apparent that increasing the dose decreases the latent period for opacity production.

5.3 Mice Exposed to ∽ 4 Mev Gamma Rays of the Thermal Column

Mice exposed in the thermal column were exposed not only to thermal neutrons but to a certain amount of gamma rays as well. It was shown by Brennan et al (14) that the gamma contaminant (effective energy ∽4 Mev) was equivalent to 0.21 rem/sec of thermal column exposure and the thermal neutron output was equivalent to 1.04 rem/sec. As noted previously these values for rem are based on 30-day lethality studies. A second source of gamma contamination was produced by the mice themselves becoming activated and serving as gamma sources. The gamma contaminant from this source amounted to approximately 0.12 rem/sec. The percentages of the total rem delivered in the thermal column contributed by each of these three sources of radiation were

Thermal neutrons - 76 per cent

Thermal column gamma contaminant - 15 per cent

Intermouse gamma contaminant - 9 per cent

Since it is not known whether the RBE for the gamma contaminants is the same for the production of lens opacities as for the production of 30-day lethality it is necessary to assess an RBE for these contaminants in order to determine in absolute terms the RBE of thermal neutrons. For this reason mice were exposed to dosages of thermal column gamma rays relatively free from thermal neutrons and were observed for lens opacities. The method of obtaining this gamma flux has been described in detail by Brennan et al (14). Observations of these mice have not been continued for a sufficient period to assess the relative effectiveness of the gamma radiation for the production of lens opacities. Therefore, it is not possible at present to subtract the gamma component of thermal column flux and to establish absolutely either latent period for production of opacities or the dose required to produce 50 per cent opacities at 30 weeks. Calculations of the RBE of thermal neutrons in the present study are made by assuming that the relative effectiveness of the gamma contaminant for producing lens opacities is the same as that for the production of acute lethality. It should be pointed out that this assumption probably tends to maximize the RBE for thermal neutrons.

6. INCIDENCE OF TRANSIENT LENS OPACITIES

In certain of the mice irradiated with low doses of either X rays or thermal column radiation minimal lens opacities appeared beginning at 14 weeks, persisted for two to four subsequent examinations, and then disappeared. Whether they will recur at some later time remains to be

determined. The incidence of these transient opacities at various dosage levels is tabulated in Table 4. These data show that transient opacities were found only in animals exposed to low doses of radiation. It is also apparent that they occur at much lower exposures to thermal column flux than to X rays.

There was no evidence found in the present study that there is a greater degree of recovery from lens damage following X rays than following neutron exposures. Animals exposed to low doses of both types of radiation showed some transient opacities (Table 4). It is possible that the reason other investigators have assumed that a difference in recovery exists is that they were comparing relatively high effective doses of neutrons with relatively low doses of X rays. Because of the high value for the RBE of thermal neutrons it is possible that sufficiently high exposures were given to prevent the occurrence of transient opacities in the case of neutrons but not in the case of X rays.

7. RELATIVE BIOLOGICAL EQUIVALENCE OF THERMAL COLUMN FLUX AND 250-KVP X RAYS

7.1 Validity of Method of Comparison

7.1.1 End Point Used

It should be reemphasized that in the present study the incidence of lens opacities rather than mature cataracts is being utilized as the basis of comparison. In many cases the degree of damage was slight and undoubtedly did not interfere with vision. All lens abnormalities characteristic of radiation damage and visible with the opthalmoscope

were recorded, and the first examination at which an opacity which subsequently persisted was seen was taken as the end point of the latent period for its production. Only those opacities that persisted after four subsequent examinations were used for the calculation of RBE.

In considering the validity of the method of comparison, the reliability with which the end point (lens opacity) was determined should be reviewed. It is believed that the end point was determined with considerable accuracy for the following reasons: (a) the mice were examined as "unknowns" at each opthalmoscopic examination and there was a high degree of internal consistency among examinations on individual mice, (b) on several occasions the mice were examined by a second investigator and there was good agreement between the results reported by the two independent observers.

7.1.2 Appearance of Opacities in Controls

The mice used in this study (female white Swiss) showed a high incidence of congenital opacities located at the posterior pole of the lens. The presence of these defects did not introduce error in the present study since they were recognized early and allowed for in tabulation of the results. Spontaneous opacities indistinguishable from those induced by radiation occurred in 4.2 per cent of the control mice. This relatively low spontaneous incidence was not taken into account in calculating latent periods for production of opacities. The spontaneous incidence was allowed for, however, in calculating the dose required to produce 50 per cent opacities at 30 weeks and the calculations were

weighted accordingly.

7.1.3 Gamma Contaminant in Thermal Neutron Flux

Although the gamma contaminant in the thermal column flux is relatively small it must be considered in determining the RBE of thermal neutrons in producing lens opacities. Brennan et al (14) found that thermal column gamma rays and the gamma rays from the activated mice dosing one another contributed about 24 per cent of the total effective radiation dose. Since it appears very likely that the RBE of thermal neutrons for producing lens damage is considerably higher than for the production of acute lethality the per cent of the effective dose contributed by the gamma contamination is probably considerably less than 24 per cent. Studies of the RBE of the pile gamma rays in producing lens opacities are now in progress. The results of the studies will be given in the final report. In the meantime it is possible to calculate a range of values for the RBE of thermal neutrons free from gamma contaminant by making certain reasonable assumptions as to the relative effectiveness of this type of radiation in producing lens opacities.

7.2 RBE of Thermal Column Exposure for the Production of Lens Opacities During a 30-Week Latent Period

Table 5 summarizes the incidence of lens opacities 30 weeks after exposure to X rays and thermal column flux. These data are shown graphically in Fig. 1 in which per cent incidence is plotted against exposure time in seconds. The results were subjected to probit transformation and the probit regression lines are shown in Fig. 2. The equations for the

two regression lines are

Thermal column exposure $Y = 3.359 + 2.175X$

250-KVP X-ray exposure $Y = 1.417 + 2.175X$

where Y is the probit of the per cent incidence of opacities and X is the log of the duration of exposure.

By substituting Y = 5 (50 per cent incidence of opacities) into these equations the dose required to produce a 50 per cent incidence of opacities may be calculated. The values obtained are

Thermal column exposure - 4.7 seconds

250-KVP X-ray exposure - 44.4 seconds

In previous sections of this report the dose of thermal column flux has been expressed in rem based on the lethality studies reported by Brennan et al (14). It is, of course, desirable to express rem on the basis of effectiveness in producing lens opacities. Using the values given above, however, it is possible to tentatively express thermal column exposure in rem based on effectiveness in producing lens changes. These data show that 4.7 seconds of pile exposure is equivalent to 44.4 seconds of X-ray exposure in producing lens opacities in 50 per cent of exposed animals 30 weeks after exposure. Since X rays were delivered at the rate of 0.993 r/sec then

4.7 sec pile exposure = 44.09 r X rays and

1 sec pile exposure delivers $\frac{44.09}{4.7} = 9.38$ rem.

To determine the RBE of thermal column exposure it is necessary to divide the value for rem by the number of rep delivered in the same

time interval. According to Brennan et al (14), one second of thermal column exposure delivers 1.16 rep of mixed radiation. Dividing rem by rep (definition of RBE) the value for the RBE of thermal column exposure becomes $\frac{9.38}{1.16} = 8$.

7.3 RBE of Thermal Neutrons for the Production of Lens Opacities During a 30-Week Latent Period

To calculate the RBE of thermal neutrons alone it is necessary to subtract the contribution of the thermal column and intermouse gamma rays from the values obtained for the mixed radiation. Such calculations require assessment of the RBE of the gamma contaminants in producing lens opacities. If it is assumed that their RBE for the production of lens opacities is equal to that based on 30-day lethality, then

1 sec pile exposure	= 9.38 rem/sec total
4 Mev gamma contaminant	= 0.21 rem/sec*
Intermouse gamma	= 0.12 rem/sec*
Total contribution of thermal neutrons alone	= 9.05 rem/sec.

If one second of thermal column exposure delivers 0.61 rep/sec of thermal neutrons (14), then the RBE of thermal neutrons is $\frac{\text{rem/sec}}{\text{rep/sec}} = \frac{9.05}{0.61} = 14.8$. The neutron flux was determined as 1.42×10^{10} n/cm^2/sec, therefore 1 rem for the production of lens opacities is equal to $\frac{1.42 \times 10^{10}}{9.05} = 1.57 \times 10^9$ thermal neutrons/cm^2.

In the above calculations the RBE of the $\sim$4 Mev gamma contaminant is considered to be 0.53 and that of the intermouse gamma contaminant

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* Taken from data of Brennan, Harris, Carter, and Langham (14).

is considered to be 0.8. These values were determined in the 30-day lethality studies (14). If it is assumed that both gamma contaminants are equally as effective as X rays (RBE = 1) in producing lens opacities then the calculations become

1 sec thermal column exposure	= 9.38 rem/sec total
4 Mev gamma contaminant	= 0.40 rem/sec
Intermouse gamma	= 0.15 rem/sec
Total contribution of thermal neutron dose	= 8.83 rem/sec.

The RBE of thermal neutrons in this case becomes $\frac{8.83}{0.61} = 14.5$ and 1 rem of thermal neutrons = $\frac{1.42 \times 10^{10}}{8.83} = 1.61 \times 10^{9}$ n/cm^2.

From the above calculations it can be seen that widely varying assumptions as to the RBE of the gamma contaminants in producing lens opacities alters the value of the RBE for thermal neutrons very little. It is tentatively concluded, therefore, that the RBE of thermal neutrons for the production of lens opacities in mice is about 15, and that one rem of thermal neutrons (based on ability to induce lens opacities) is about 1.6×10^{9} n/cm^2. These values may be compared with an RBE of 1.7 and a value of 1.37×10^{10} n/cm^2/rem on the basis of the dose required to produce lethality of 50 per cent of the exposed animals in 30 days.

Since 30 weeks was used as the latent period for the development of lens opacities, lethality values at the same time limit should be used for comparison of the two biological effects. A consideration of the LD-50 doses of X rays and thermal neutron irradiation at 30 weeks indicates the following: (a) the absolute LD-50/30 week dose in each

case is lower than the 30-day LD-50 dose; (b) 1.25 x 10^{10} n/cm^2 are equivalent to 1 rem of X rays (based on lethality at 30 weeks) compared to 1.37 x 10^{10} n/cm^2 using lethality at 30 days. However, the variance of the result has not been exhaustively investigated and the values may not be significantly different. Using the value for lethality at 30 weeks for comparison would lower the relative RBE of thermal neutrons for opacity production by about 10 per cent. A more complete discussion of the lethality and longevity data will appear in a final report.

8. SUMMARY AND CONCLUSIONS

White Swiss mice exposed to single doses of 250-KVP X rays and to a flux of thermal neutrons plus gamma rays in the thermal column of the Los Alamos homogeneous reactor have been observed for 30 weeks and the incidence and latent period of radiation-induced lens opacities determined as a function of radiation dosage. X-ray dosages of 200 roentgens or more produced opacities in 100 per cent of the animals. At lower doses the incidence at 30 weeks decreased with decreasing radiation. The observations showed also that increasing the dose decreased the latent period for the production of lens changes. Thermal flux exposures of 52 rem (based on 30-day lethality observations) and higher produced 100 per cent opacities in 30 weeks. Below this dose the incidence decreased with decreasing doses. Again the higher dosages produced the shorter latent period for opacity production.

At the lower doses transient lens opacities were observed both for X rays and thermal column exposure. There was no evidence found in the

present study that there was a greater degree of recovery from lens damage following X-ray exposure than following exposure to the radiation of the thermal column.

By making certain assumptions regarding the relative biological effectiveness of the gamma contaminant of the thermal column it was possible to calculate the relative biological effectiveness of thermal column flux and of thermal neutrons with regard to the production of lens changes. The RBE of thermal neutrons following a latent period of 30 weeks was approximately 15, and 1.6×10^9 n/cm^2 was equivalent to 1 rem. When the LD-50/30 was used as the biological indicator the RBE was 1.7, and 1.37×10^{10} n/cm^2 was equivalent to 1 rem. These data indicate that the relative biological effectiveness of thermal neutrons with regard to the production of lens abnormalities 30 weeks after exposure is approximately 7 times the RBE determined from the LD-50/30. The RBE for thermal neutrons in producing lethality in mice was only slightly greater at 30 weeks than at 30 days.

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Table 1--Doses of 250-KVP X Rays and Thermal Column Radiation Received by Swiss Mice and 30-Day Survival

Dose, 250-KVP X Rays (r)	No. Mice Alive at 30 Days	Dose, Thermal Column Radiation (rem*)	No. Mice Alive at 30 Days
12.5	25	3.2	25
25	25	6.4	25
50	25	12.8	25
100	25	25.7	25
200	44	51.6	25
300	45	102.4	25
400	41	196.6	45
450	14	307.1	43
500	18	409.5	41
550	12	443.6	34
600	1	477.8	56
650	1	511.9	12
		563.7	5

* Value for rem based on 30-day lethality studies of Brennan et al (14)

1 rem = 1.04×10^{10} n/cm^2 plus 0.39 r of gamma rays.

Table 2--Incidence of Lens Opacities in Mice at Various Times After 250-KVP X Radiation

Dose X Rays (r) \ Time (wks) →	Per Cent Incidence of Lens Opacities at Various Times After Radiation											
	6	8	11	14	17	18	19	22	24	25	28	30
12.5	--	0	--	0	8	10	--	--	--	--	--	12
25	--	0	--	0	--	--	--	--	0	10	10	10
50	--	0	--	0	33	--	50	50	--	59	--	59
100	--	0	--	8	25	--	60	71	78	--	--	83
200	--	0	--	3	46	--	90	100	--	--	--	100
300	--	0	--	5	72	88	100	--	--	--	--	100
400	--	0	--	36	74	74	100	--	--	--	--	100
450	--	0	--	50	86	--	100	--	--	--	--	100
500	13	26	44	79	100	--	--	--	--	--	--	100

Table 3--Incidence of Lens Opacities in Mice at Various Times After Exposure to Thermal Column Radiation

Dose (rem[a]) / Time (wks) →	Per Cent Incidence of Lens Opacities at Various Times											
	6	8	11	14	17	18	19	22	24	25	28	30
3.2	0	0	--	4	--	18	--	25	--	--	--	25
6.4	0	0	--	4	29	--	43	50	--	67	--	79
12.8	0	0	--	9	17	--	26	31	--	--	--	43
25.7	0	0	--	21	46	--	73	92	--	--	--	92
51.6	0	0	--	22	42	--	60	92	--	--	--	100
102.4	0	0	--	36	82	90	100	--	--	--	--	100
196.6	0	0	--	27	80	91		100	--	--	--	100
307.1	0	--	--	63	94	100	--	--	--	--	--	100
409.5	0	0	3	58	87	100	--	--	--	--	--	100
443.6	13	45	100	--	--	--	--	--	--	--	--	100
477.8	9	44	100	--	--	--	--	--	--	--	--	100

[a] Value for rem based on 30-day lethality studies of Brennan et al (14).

Table 4--Incidence of Transient Lens Opacities in Mice Exposed to 250-KVP X Rays and Thermal Column Radiation

Dose, X Rays (r)	Incidence of Transient Opacities (Per Cent)	Dose, Thermal Column Radiation (rem)	Incidence of Transient Opacities
12.5	0	3.2	28
25	15	6.4	8
50	35	12.8	4
100	9	25.7[a]	0
200[a]	0		

[a] No transient opacities were found at doses higher than those shown in this table.

Table 5--Incidence of Lens Opacities in Mice at 30 Weeks After Exposure to Thermal Column and 250-KVP X Radiation

Length of X-ray Exposure (sec)[a]	Incidence of Lens Opacities (per cent)	Length of Thermal Column Exposure (sec)	Incidence of Lens Opacities
12.59	12.0	2.36	20.0
25.18	10.0	4.68	79.2
50.35	49.1	9.36	43.5
100.70	82.6	18.8	92.3
201.41	100	37.8	75.0
302.11	100	75	100
402.82	97.3	144	100
453.17	100	225	100
503.52	100	300	100
553.88	100	325	100
604.23	100	350	100
654.58	100	375	100
		413	100

[a] X-rays were delivered at the rate of 0.993 r/sec.

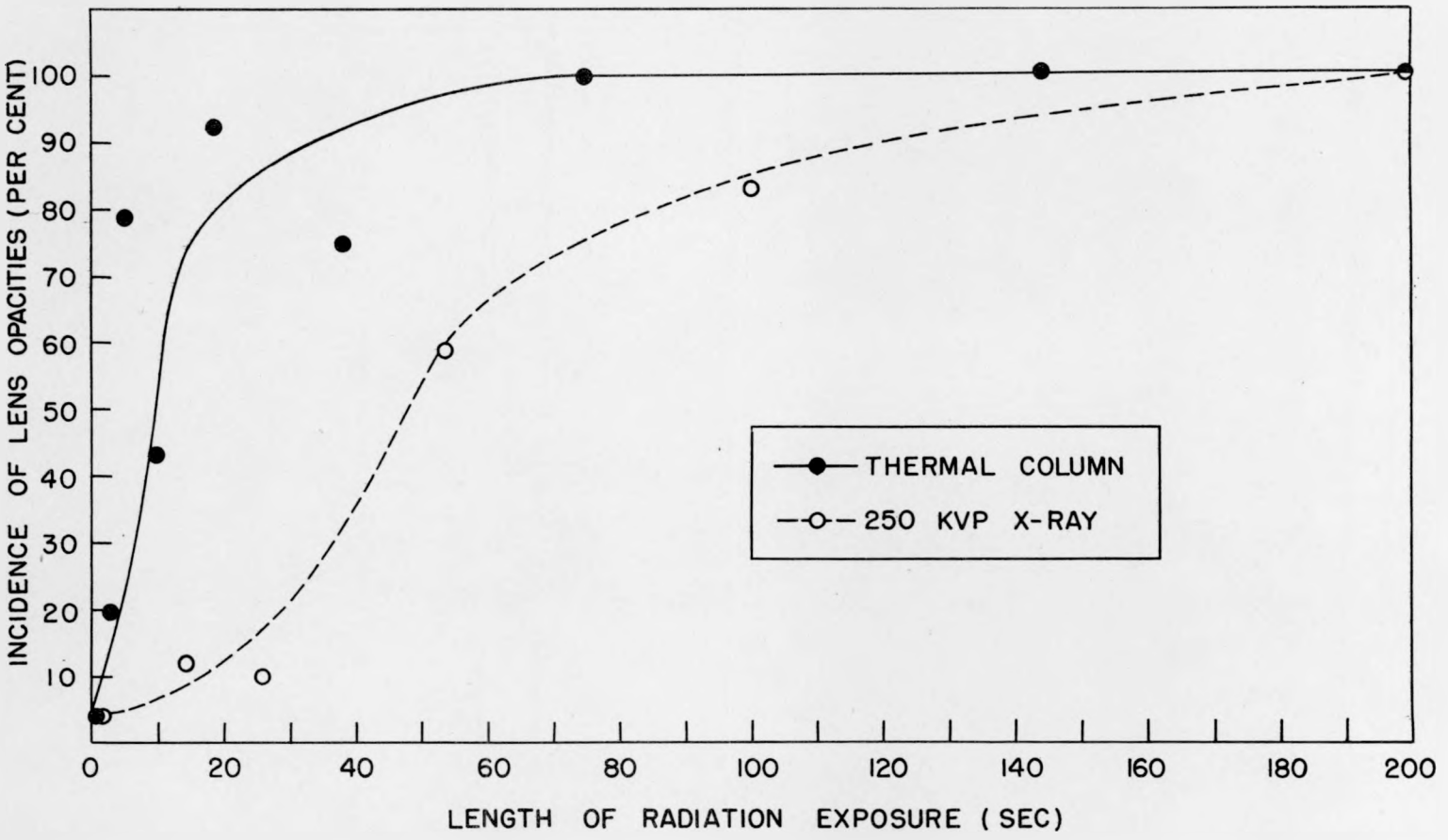

Fig. 1--Incidence of Lens Opacities in Mice at 30 Weeks after Various Lengths of Exposure to Thermal Column and X Radiation.

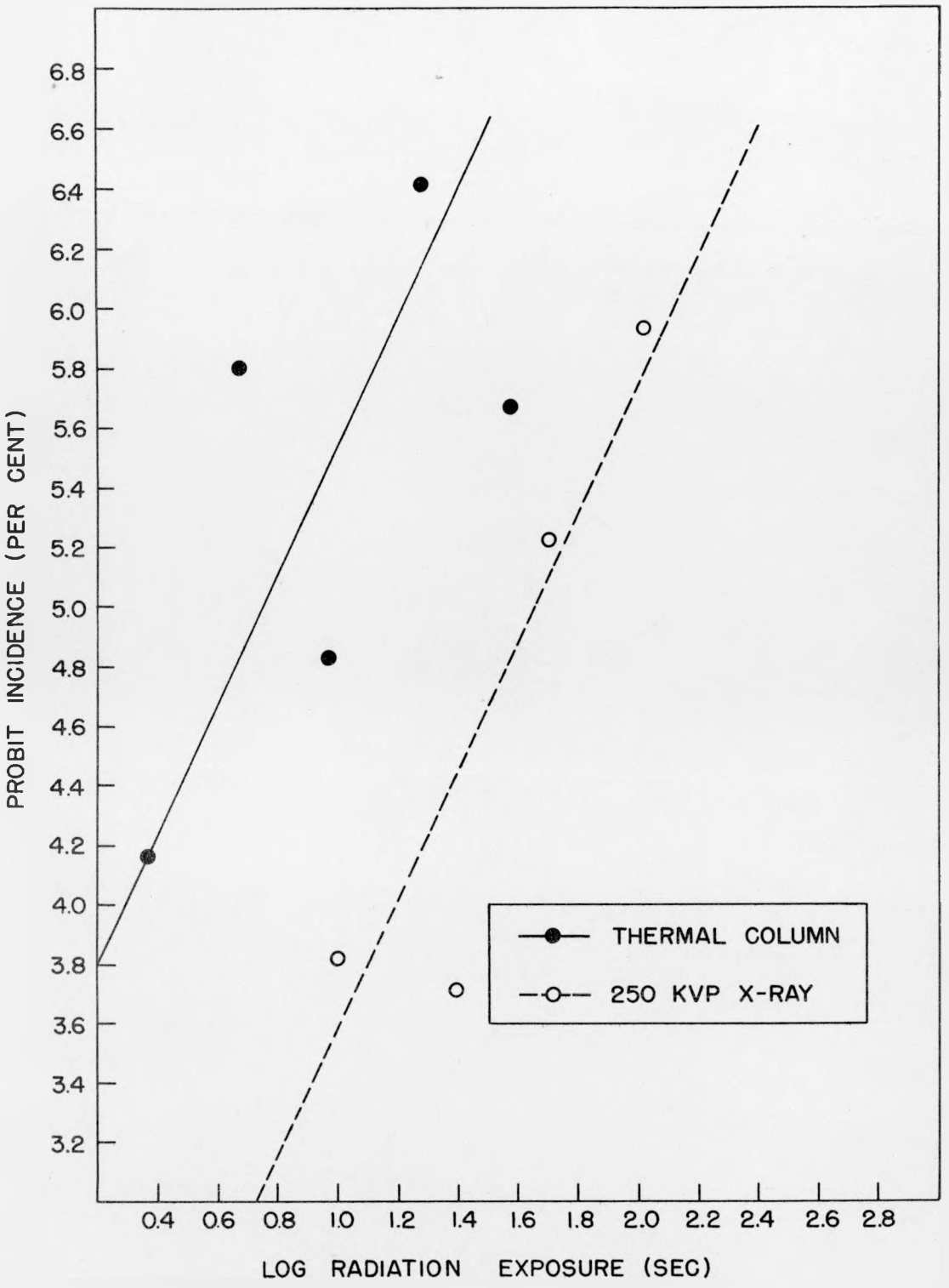

Fig. 2--Regression Lines for Probit of Per Cent Incidence of Lens Opacities Against Time of Exposure to Thermal Column and 250-KVP X Radiation Following Latent Period of 30 Weeks.