

THE INFLUENCE OF OXYGEN  
ON THE PRODUCTION OF DOMINANT LETHAL MUTATIONS  
IN DROSOPHILA VIRILIS OOCYTES

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J.R.D.

## PREFACE

Much research has been done and is continually being done in the many fields and areas of genetics. All of this work will certainly have its place in our understanding the broad spectrum of life and the factors affecting life. The research done in this thesis is intended to contribute in some small way to an understanding of these complexities.

The material in this thesis is presented in various sections along with appropriate figures and charts so that the reader may best follow the pattern of the research.

J.R.D.

## TABLE OF CONTENTS

	PAGE
INTRODUCTION . . . . .	1
METHODS AND MATERIALS. . . . .	14
RESULTS AND DISCUSSION . . . . .	20
Results. . . . .	20
Discussion . . . . .	23
SUMMARY. . . . .	26
BIBLIOGRAPHY . . . . .	28

## LIST OF TABLES

TABLE	PAGE
1. X-ray dose effect on <u>Drosophila virilis</u> Texmelucan oocytes irradiated in air. . . . .	33
2. X-ray dose effect on <u>Drosophila virilis</u> Texmelucan oocytes irradiated in oxygen . . . . .	34
3. The effect of irradiation on the percentage hatch of stage 14 <u>Drosophila virilis</u> oocytes. Females irradiated in 1 atm. of air . . . . .	35
4. The effect of irradiation on the percentage hatch of stage 14 <u>Drosophila virilis</u> oocytes. Females irradiated in 1 atm. of oxygen. . . . .	38

LIST OF FIGURES

FIGURE	PAGE
1. Dose response curve for stage 14 oocytes of <u>Drosophila virilis</u> Texmelucan . . . . .	32

## INTRODUCTION

After the demonstration that high energy radiation produced mutations (Muller 1927), many workers believed that ionizations produced by radiation always acted directly upon genetic material. Fricke (1934), however, postulated that ionizing radiation produced its chemical and biological, including mutagenic, influence through the formation of active oxygen-containing groups. It was suggested that an intermediary pathway from mutagenic agent to mutation existed because of the independent demonstrations that: (1) iron salts increased x-ray mutagenesis in Drosophila spermatozoa much more than could be accounted for by increased absorption of radiation (Rapoport 1943); (2) oxygen concentration present at the time of x-ray treatment of Vicia faba (the broad bean) greatly influenced the yield of chromosome aberrations (Thoday and Read 1947).

In Drosophila and Habrobracon, induction of the breakage-fusion-bridge cycle with consequent gene imbalance has been considered as the

primary source of dominant lethality (von Borstel and Pardue 1956). Habrobracon eggs irradiated during the first meiotic metaphase show terminal deletions which result in bridges in the second meiotic anaphase and bridge breaking during cleavage. They found the breakage-fusion-bridge cycle did not become established in eggs irradiated in the first meiotic prophase. The evidence they obtained suggested that most radiation-induced dominant lethal effects occur earlier in development than does death from aneuploidy in the progeny of nonirradiated triploids.

Drosophila embryos resulting from fertilization by sperm irradiated at about 50% lethal dose (2000r), like Habrobracon embryos from eggs irradiated at about 50% lethal dose (15,000r), died during early cleavage. When studying the nuclei of these early cleavage cells, von Borstel and Pardue (1956) found that they gave a negative reaction to the Faelgen test. The Faelgen reaction, a test for the presence of deoxyribonucleic acid in cells, will give

a deep purple color when positive and no color when negative. From this evidence they suggested that radiation-induced dominant lethal mutations are not necessarily due to chromosomal deficiencies but may be associated with the interference of DNA synthesis.

Patterson, et al. (1932) showed aging of virgin Drosophila melanogaster females resulted in an increase in frequency of radiation-induced dominant lethals among the first eggs laid. This observation was not understood until Parker (1955) made a comparison between radiation-induced dominant lethals and detachments of attached-X's in newly emerged adult Drosophila females and those held virgin for five to seven days before irradiation and mating. He found the increase in frequency of dominant lethals involves a shift from a "two-hit" to a "one-hit" type of survival curve. If the breakage in unaged oocytes is of single chromatids, two sister chromatids must be broken independently to give rise to an anaphase II bridge which might be a cause

of dominant lethal formation in irradiated oocytes. This gives rise to a two-hit curve. If the breakage in aged cells is primarily isochromatidal it should yield a one-hit survival curve. Parker also found that radiation-induced detachments of attached-X's is increased markedly by aging, and that these detachments show a linkage with ends of other chromosomes.

The combined effects of aging of *Drosophila* females with irradiation was not thoroughly understood until the complete cytological clarification of oogenesis by King, Rubinson, and Smith (1956), as follows:

The two ovaries of *Drosophila melanogaster* are each subdivided into an average of 12 egg tubes or ovarioles. The ovariole is differentiated into an anterior germarium and a series of egg chambers. At the apex of the germarium is a region containing 50 or so mitotically active cells. It is assumed that a germarial cell divides into two daughter cells. One of these cells repeats the process, while the other oogonium undergoes four consecutive, synchronous divisions to produce a cyst of 16 daughter cells.

The 16 daughter cells eventually form a spherical cyst, the germarial cyst, in the posterior region of the ovarium. It is incompletely covered by a layer of follicle cells and is often separated from the next closest cyst by an indentation in the ovarium and by a transverse layer of follicle cells. In the germarial cyst it is sometimes impossible to distinguish the daughter cells from one another, but usually the most posterior one becomes the oocyte. Once the cyst has been completely surrounded by a layer of follicle cells, it is pinched off from the ovarium and so becomes the first egg chamber.

King (et.al 1956) subdivided oogenesis into 14 stages with the germarial cyst and the first chamber as stages 1 and 2 respectively. In stage 1 the oocyte nucleus is characterized by a u-shaped accumulation of condensed chromatin. In stage 2 development the 15 nurse cells contain thread-like chromosomes, whereas the oocyte nucleus, located in the posterior periphery of the chamber, shows condensed chromatin. In stage 3 development the oocyte nucleus, which is still located in the posterior periphery

of the egg chamber, first shows the karyosome. The nurse cell nuclei are partially filled by fine chromatin strands and other material. At stage 4 the nurse cell nuclei contain several masses of material, some of which are connected together by fine strands. The cyst is still quite small and circular. Stage 5 chambers are characterized by a nonhomogeneous group of nurse cell nuclei, some of which look like those of stage 4 and some like those of stage 6. In stage 6 developing nurse cells are completely filled by chromosomal material. Karyosome and chromatic strands are distinct in the enlarging nucleus of the oocyte. The egg chamber is still spherical in stage 6. The stage 7 cysts are the first to show ellipsoidal shape. Here the nurse cells and the oocyte are more or less equal in size. Stage 8 is characterized by an oocyte which is significantly larger than the average nurse cell. At stage 9 development the oocyte makes up about one-third of the cyst volume. The nurse cell nuclei closest to the oocyte are somewhat larger than the rest, and the border cells make their appearance.

At stage 10 the oocyte makes up about one-half of the cyst volume. The nurse cells bordering the oocyte now show distinctly larger nuclei. Stage 11 chambers usually lack border cells. The oocyte makes up about three-fourths of the total volume of the chamber, and an epithelial wall completely separates the oocyte from the nurse cells for the first time. The oocyte has almost reached its maximum size in the stage 12 chamber. The nurse cell nuclei are crowded together at the anterior pole. The nuclei start to degenerate at stage 13. The micropile is completed, but the dorsal appendages are only partly grown. Stage 14 is the fully developed, ovarial, primary oocyte with completed dorsal appendages.

Between stages 1 and 14 the oocyte increases in volume by a factor of over 100,000 times. Under optimum conditions it probably takes 3 days for the completion of this process. During the first seven stages the oocyte and the nurse cells grow at identical rates, but, subsequently, yolk formation begins

and the oocyte grows faster. By stage 11 the oocyte contains more cytoplasm than do all the nurse cells put together, and by stage 12 the oocyte has almost reached its maximum volume. The oocyte nucleus increases in size from stages 2-10, and it subsequently remains fairly constant in volume until the nuclear membrane disappears late in stage 13. From stages 8 through 10 the nurse cells continue growing, but are outdistanced by the volume increase of the oocyte. The nurse cells begin to shrink during stages 11 and 12 and are subsequently resorbed.

Female flies which are between 0 and 4 hours old contain eggs which are up through stage 7 in development. Females which have been held virgin for 5 to 7 days usually contain at least one egg in each ovariole which is in stage 14 development. This study deals with radiation effects on stage 14 oocytes.

Giles and Riley (1950) demonstrated that for Tradescantia neither the presence nor the absence of oxygen before or after irradiation treatment affected the aberration frequency.

However, Abrahamson (1959) showed that the presence of oxygen at the time of x-irradiation of Drosophila produced considerably greater chromosomal damage, measured as half-translocations, than does the absence of oxygen. Half-translocations are so called (Herskowitz and Abrahamson 1956) because, of the four pieces produced by breaking two non-homologous chromosomes, only two pieces which join eucentrically are retained in the fertilized egg. The other centric piece is cast into a polar body joined or unjoined to the other acentric fragment.

The type of half-translocation studied by Abrahamson (1959) was one in which an attached-X chromosome was broken into two arms, only one of which was retained in the egg after joining eucentrically to a piece of another broken chromosome. The other reciprocal pieces were incorporated into a polar body.

It was observed by Abrahamson that above-normal concentrations of oxygen increase the x-radiation induced half-translocation rate significantly above the rate obtained in air. Furthermore, treatment with oxygen after irradiation had no detectable effect on the rearrangement frequency.

Oxygen at high tensions exhibits a toxic effect on many insects. It was observed by Williams and Beecher (1944) that oxygen reduced viability of adult Drosophila azteca. Oxygen was noted to have delayed embryonic development of Drosophila melanogaster by Glass and Plaine (1952). Oxygen tension was noted in this study to reduce the viability of Drosophila virilis Texmelucan.

Dickerman (1963) in working with the effects of various gases upon dominant lethal production in Drosophila virilis oocytes found that the greatest number of dominant lethal mutations were produced in oocytes from females irradiated in excess oxygen. The greatest amount of damage was noted in both stages 7 and 14 oocytes when ten atmospheres of oxygen were used. Fewer dominant lethal mutations were induced with one atmosphere of oxygen. Dickerman also found that more dominant lethals were produced when flies were irradiated in ten atmospheres of air than in one atmosphere. This was noted for both stage 7 and 14 oocytes. It was also noted that there was more damage in ten atmospheres of air than in one atmosphere of oxygen.

Dose fractionation studies of x-ray-induced dominant lethality (Parker 1959; Parker and Hammond 1958) in stage 7 and stage 14 oocytes of Drosophila melanogaster have demonstrated that a significant amount of x-irradiation damage can be repaired within 15 minutes in stage 7 oocytes, however, no repair in stage 14 oocytes was noted until after fertilization.

The high sensitivity and one-hit survival curve of stage 14 oocytes as contrasted with the low sensitivity and two-hit survival curve of stage 7 oocytes has been attributed to the difference in repair (Parker 1959; Parker and Hammond 1958; Abrahamson 1961). Primary breaks in the two types of cells may rejoin in the original manner, rejoin with an independently produced break, or may not be repaired. The first phenomenon leads to no observable effect, the second to a multi-hit dose response, while the third is a one-hit event. Parker (1963) states that rapid repair of damage in stage 7 oocytes brings about the first two events. He states that anaphase I, which may intervene

between breakage and repair in stage 14 cells, results in breaks separated at the time of repair in stage 14 oocytes so that there is a one-hit survival curve.

There appears to be a system (Rinehart 1964) for repair of x-ray damage in the stage 7 oocyte which is maintained at a functional level by an oxygen-requiring process. In the absence of oxygen, Rinehart observed that the repair system is reduced at a rate that can be measured at 25°C but not at 4°C. He also found that the administration of oxygen to cells whose repair system has been inactivated by oxygen deprivation results in regeneration of the system. Rinehart stated that this repair system appears to be inactive in stage 14 oocytes until after fertilization.

Parker and Hammond (1958) postulated that the relation between time and induction of primary lesions, chromosome movement, and repair may provide a partial explanation of the greatly increased sensitivity of stage 14 oocytes compared with stage 7 oocytes, where repair ordinarily occurs within 30 minutes of the time of

irradiation, and presumably before much chromosome movement.

Although the dose response curves in air of various species of Drosophila have been determined, the dose response curves in oxygen or other gases have not been ascertained. The purpose of this study is to determine the dose response curve of stage 14 oocytes irradiated in oxygen and compare it to the dose response curve of stage 14 oocytes irradiated in air.

## METHODS AND MATERIALS

Female flies for these experiments were obtained from a stock of Drosophila virilis Texmelucan. A culture of Drosophila virilis Texmelucan males and females was set up in half pint bottles. After one week the cultures were transferred to new media. The larvae in the old cultures were spread on new media in half pint bottles to eliminate crowding. Nine days from the date of spreading the adult flies began to emerge from the pupae. These cultures were then cleared of all emerged flies. The females to be used were collected over a period of four to six hours and were separated from the males during the following 24 hour period. They were then placed on yeasted media and allowed to mature as virgins for five days prior to irradiation. This procedure for obtaining virgin females to be irradiated and for control matings was followed throughout the experiment.

Hybrid males from a cross of Drosophila virilis Argentina males and Drosophila virilis

Brazil females were used. The hybrid males were cultured in the same manner as the Drosophila virilis Texmelucan females. The males were allowed to mature from seven to ten days prior to mating with the control or irradiated females.

In all of the experiments the flies were kept on banana media. The banana media consists of 2.5 liters of water, 50 g agar, 60 g brewers yeast, 670 g strained bananas, 135 ml malt syrup, 135 ml Karo syrup and 15 ml Tegosept-M solution. After being brought to boiling, the water is removed from the heat source and the agar is dissolved in it. The yeast is killed by adding ethyl alcohol and then is added to the agar solution. The remaining ingredients are added and the solution is boiled for at least five minutes.

The flies were mated on yeasted media during the first part of the experiment to enhance egg-laying. It was found that if both the males and virgin females were allowed to mature on yeasted media they did not have to be mated on yeasted media. In some of the

experiments bakers' black paste dye was added. This dark background facilitated egg-counting to a degree, but not enough to be continued in all experiments.

Drosophila virilis females contain 16-18 ovarioles per ovary or 32-36 ovarioles per female. In newly emerged females the most mature oocyte present in each ovariole is stage 7 (King et al. 1956). Female flies allowed to mature more than two days usually contain one stage 14 oocyte per ovariole. The Drosophila virilis virgin females were irradiated so that stage 14 oocytes were the most mature cells treated.

The females were x-rayed using a General Electric maximar 250III machine operated at 250 kv and 15 ma. with a .6 mm Sn plus a 1 mm Al plus a .25 mm Cu filter. Females to be irradiated in air were placed in a leucite container after being etherized. The females were never, however, under ether while being irradiated. The females irradiated in air and the females irradiated in oxygen were placed 20 cm from the x-ray source. The females were irradiated at room temperature

each time.

After irradiation the virgin females were mated individually with two hybrid males in 30 mm glass vials on banana media. The males and females were transferred to fresh media every twenty-four hours and the eggs laid during these periods were counted. The counts were generally continued for five days and only rarely for six days. Only egg samples of thirty-six or fewer eggs per female are included in this study. This was to assure the number of eggs sampled did not exceed the number of oocytes in stage 14 development during irradiation treatment.

Females irradiated in oxygen were placed in a leucite container and placed in a pressure chamber with a glass top. The chamber was constructed so that there were four hand regulated valves which could be used as inlets or outlets. A pressure gauge was attached to one of the valves so internal gas pressure could be measured at any time. After the females were placed in the chamber, oxygen was flushed through several times to remove all air in the chamber. The females were kept in

the chamber in one atmosphere of oxygen for 15 minutes prior to irradiation, during irradiation, and 15 minutes after irradiation. Females irradiated in oxygen are weakened and, therefore, were not mated for one day following irradiation.

Females for control matings were treated in the same manner as females irradiated in oxygen and in air, but were not subjected to any irradiation treatment.

Females were irradiated for time periods of .3 minutes, .6 minutes, 1 minute, 2 minutes, 2.5 minutes, 3 minutes and 4 minutes in air. Females were irradiated in oxygen for time periods of .6 minutes, 1 minute, 1.5 minutes, 2 minutes, and 2.5 minutes. The x-ray dose was then measured by a Victoreen-r meter and the dose found to be 182r per minute.

The percentage hatch was scored by comparing the number of eggs with the number of pupae that developed. Pupae are used because they can be counted quickly and easily. The percentage hatch was calculated for each day and for the total counting period. The percentage of non-hatch of irradiated eggs

was considered to be due to the induction of dominant lethal mutations. A small percentage of non-hatch is due to non-fertilization of the eggs, but it is assumed that this value is the same for both irradiated females and controls. The dose response curves due to irradiation in air and in oxygen were determined.

## RESULTS AND DISCUSSION

Preliminary tests were carried out to determine the percentage of dominant lethal mutations produced in stage 14 oocytes irradiated with 350r. This was done to obtain a basic dose response and also to determine the reliability of counting technique upon the media used. Tests were also run to determine the stage of maturity at which the oocytes should be irradiated to obtain the best repeatable results. Females irradiated on the sixth day after hatching yielded the most consistent results.

The dose response curves of stage 14 Drosophila virilis Texmelucan oocytes, irradiated in air and irradiated in oxygen, are graphically represented in Figure 1. The dose response curve in Drosophila melanogaster, due to dominant lethal production obtained by Parker (1959) was very similar to the dose response curve for stage 14 oocytes of Drosophila virilis Texmelucan irradiated in air in this study and also for the dose response curve

obtained by Dickerman (1963) when working with D. virilis stage 7 and stage 14 oocytes. The dose response curve obtained by Dickerman is steeper than the slope obtained in this study. This difference may be due to the difference in dose rate used. Dickerman irradiated with doses of 300r to 320r per minute, while the dose rate in this study was 182r per minute. Both studies show, however, a steady increase in dominant lethal production in stage 14 oocytes irradiated in air.

Table 1 shows the results of stage 14 oocytes irradiated in air with varying x-ray doses. It can be noted that oocytes irradiated for .6 minutes in air show a higher percentage hatch than oocytes irradiated for .3 minutes in air. The difference in percentage hatch of 2.35 percent is not great enough to affect the slope of the dose response curve.

Table 2 shows the results of stage 14 oocytes irradiated in oxygen at varying x-ray doses. Oxygen appears to increase the frequency of dominant lethal production in oocytes irradiated with increasing irradiation doses.

The effect of irradiation on the percentage hatch of oocytes irradiated in air is shown in Table 3. Data for irradiation time intervals of 3.0 minutes and 4.0 minutes (545r and 728r) and the corresponding control data were obtained from experiments conducted by Dickerman (1964). Table 4 shows the effect of irradiation on the percentage hatch of oocytes irradiated in oxygen. In four of the five groups irradiated in oxygen, eggs were not laid on all five days on which egg samples were scored.

It was observed in this study that oxygen had a toxic effect on Drosophila virilis Texmelucan females held under oxygen tension longer than 60 minutes. If left in oxygen for longer periods a large percentage of the females died and the remainder were considerably weakened. The number of eggs laid by females after treatment under oxygen tension for longer than 60 minutes was severely decreased. If eggs were deposited by these females, none were laid before the third day after mating. In general, there was also a reduction in the number of eggs laid by females irradiated in oxygen and held

under pressure for 15 minutes prior to, during, and 15 minutes after irradiation. This reduction was probably due to the weakening effect of oxygen pressure upon the females. The toxic effect of oxygen pressure was also observed by Williams and Beecher (1944) when working with Drosophila azteca.

It is known that there is a small percentage of non-hatch in all groups due to non-fertilization of eggs. Because this error is present in all groups and is so small, it is not calculated and corrected for in this study. It would not affect the results to any significant degree.

Ulrich (1958) demonstrated that for Drosophila the nucleus was the site of irradiation damage and that this damage was increased in the presence of oxygen. Abrahamson (1959) demonstrated that oxygen present in Drosophila oocytes at the time of irradiation produced a significantly higher frequency of half-translocations than that produced in air. In this study the increase in irradiation damage in the presence of oxygen was also observed, and was

found to be directly proportional to the level of irradiation. This linear response had not been demonstrated previously.

It was postulated that the dose response of oocytes irradiated in oxygen would parallel that of the oocytes irradiated in air (Dickerman, 1964). However, this study has demonstrated a divergence between the dose response curves of oxygen and air. (See Figure 1).

Very little work has been done on the actual effect of oxygen concentration at the time of irradiation. Oocytes irradiated in oxygen perhaps show a dose response which is due to direct and indirect damage to chromosomes. The direct damage is thought to be due to the penetration of the x-rays and the consequent fragmentation of the chromosomes. The indirect effect may result from damage caused by particles moving through or near the chromosomal material.

The presence of oxygen in the cell obviously must contribute to these indirect effects of irradiation. However, the divergence of the slopes of the dose response curves of oxygen

and air noted in this study cannot be explained simply in terms of increased oxygen concentration in the cell. The progressive increase of irradiation damage in oxygen over damage in air with equal doses and constant gaseous pressure may be due to cumulative indirect effects of oxygen.

## SUMMARY

The results of this study show there is a linear relationship between the production of dominant lethals in stage 14 oocytes irradiated in air and in oxygen. Oocytes irradiated in oxygen are much more sensitive to irradiation damage than oocytes irradiated in air at equal doses. The production of dominant lethals in oocytes irradiated in oxygen appears to increase with an increase in irradiation when compared with oocytes irradiated in air, for the number of dominant lethals produced in oocytes irradiated in oxygen for .6 minutes is about the same as oocytes irradiated in air for 1 minute, and the dominant lethals produced in oocytes irradiated in oxygen for 2.5 minutes is nearly equal to the amount produced in oocytes irradiated for 4 minutes in air.

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FIGURE 1

Dose response curve for stage 14 oocytes of Drosophila virilis Texmelucan. Oocytes irradiated in air are represented by  $\square$ . Oocytes irradiated in oxygen are represented by  $\triangle$ .

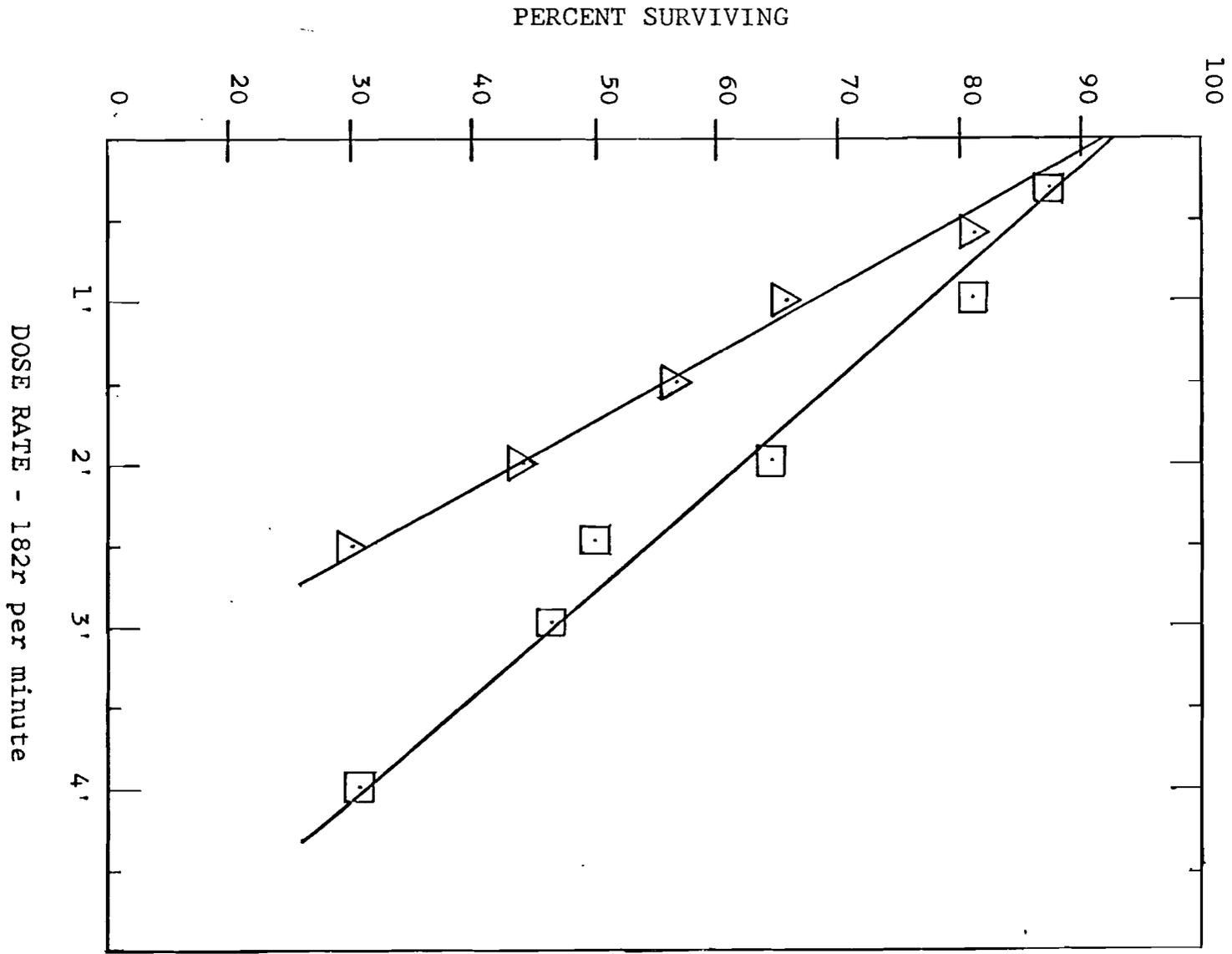


TABLE 1

X-ray dose effect on Drosophila virilis Texmelucan stage 14 oocytes irradiated in air.

<u>Dose</u>	<u>Time</u>	<u>Number of eggs</u>	<u>Number of pupae</u>	<u>% hatch</u>
54.6r	0.3 min	857	774	89.03%
109.2r	0.6 min	1327	1215	91.56%
182r	1.0 min	1477	1202	81.38%
364r	2.0 min	876	560	63.93%
455r	2.5 min	1281	644	50.27%
546r	3.0 min	2876	1306	45.41%
728r	4.0 min	2695	814	30.20%

TABLE 2

X-ray dose effect on Drosophila virilis Texmelucan stage 14 oocytes irradiated in oxygen.

<u>Dose</u>	<u>Time</u>	<u>Number of eggs</u>	<u>Number of pupae</u>	<u>% hatch</u>
109.2r	0.6 min	453	368	81.23%
182r	1.0 min	1157	765	66.12%
273r	1.5 min	277	160	57.76%
364r	2.0 min	831	363	43.68%
455r	2.5 min	296	90	30.40%

TABLE 3

The effect of irradiation on the percentage hatch of stage 14 *Drosophila virilis* oocytes. Females irradiated in 1 atmosphere of air. Controls not irradiated.

Dose	Time		Days					Total
			1	2	3	4	5	
54.6r	0.3 min	eggs	158	338	216	145		857
		pupae	142	298	201	133		774
		% hatch	89.86	88.16	93.06	91.72		89.03
Or		eggs	84	687	402	120	129	1422
		pupae	77	644	389	118	128	1356
		% hatch	91.66	93.74	96.76	98.33	99.22	95.36
109.2r	0.6 min	eggs		601	518	121	87	1327
		pupae		567	471	95	82	1215
		% hatch		94.34	90.92	78.42	94.25	91.56
Or		eggs	282	498	437	271	63	1551
		pupae	249	443	417	250	62	1421
		% hatch	88.30	88.95	95.42	92.25	98.41	91.62
182r	1.0 min	eggs	582	385	171	114	51	1477
		pupae	276	329	153	110	28	1202
		% hatch	59.70	85.46	89.47	96.49	90.32	81.3
Or		eggs	51	76	391	201	17	736
		pupae	50	70	371	192	16	699
		% hatch	98.04	92.10	94.88	95.52	94.12	94.9

TABLE 3 continued:

<u>Dose</u>	<u>Time</u>		1	2	<u>Days</u> <u>3</u>	4	5	<u>Total</u>
364r	2.0 min	eggs	525	317	34			876
		pupae	308	220	32			560
		% hatch	58.67	69.40	94.12			63.93
Or		eggs	6	29	208	163	119	527
		pupae	6	28	196	151	111	494
		% hatch	100.00	96.55	94.26	92.64	93.26	93.74
455r	2.5 min	eggs	914	250	89	13	15	1281
		pupae	456	121	45	11	11	644
		% hatch	49.89	48.40	50.56	84.61	76.79	50.27
Or		eggs	523	158	15			696
		pupae	496	147	15			658
		% hatch	94.84	93.04	100.00			94.54

TABLE 3 continued:

<u>Dose</u>	<u>Time</u>		1	2	<u>Days</u> 3	4	5	<u>Total</u>
546r	3.0 min	eggs						2876
		pupae						1306
		% hatch						45.41
Or		eggs						2361
		pupae						2276
		% hatch						96.39
728r	4.0 min	eggs						2695
		pupae						814
		% hatch						30.20
		eggs						2361
		pupae						2276
		% hatch						96.39

TABLE 4

The effect of irradiation on the percentage hatch of stage 14 *Drosophila virilis* oocytes. Females irradiated in 1 atmosphere of oxygen. Controls not irradiated.

<u>Dose</u>	<u>Time</u>		<u>Days</u>					<u>Total</u>
			1	2	3	4	5	
109.2r	0.6 min	eggs		120	151	111	71	453
		pupae		118	121	75	54	368
		% hatch		98.33	80.13	67.57	76.05	81.23
Or		eggs	282	498	437	271	63	1551
		pupae	249	443	417	250	62	1421
		% hatch	88.30	88.95	95.42	92.25	98.41	91.62
182r	1.0 min	eggs	638	265	195	32	27	1157
		pupae	410	187	135	19	14	765
		% hatch	64.20	70.56	69.23	59.37	51.85	66.12
Or		eggs	3	334	170	9		506
		pupae	3	307	150	9		469
		% hatch	100.00	91.91	88.23	100.00		92.69
273r	1.5 min	eggs		21	62	141	52	277
		pupae		10	33	81	36	160
		% hatch		47.62	53.23	57.45	69.23	57.76
Or		eggs	51	76	391	201	17	736
		pupae	50	70	371	192	16	699
		% hatch	98.04	92.10	94.88	95.52	94.12	94.97

TABLE 4 continued:

<u>Dose</u>	<u>Time</u>		<u>Days</u>					<u>Total</u>
			1	2	3	4	5	
364r	2.0 min	eggs	289		350	211		831
		pupae	124		159	98		363
		% hatch	42.90		45.43	44.45		43.68
Or		eggs	6	29	208	163	119	527
		pupae	6	28	196	151	111	494
		% hatch	100.00	96.55	94.26	92.64	93.26	93.74
455r	2.5 min	eggs	240	56				296
		pupae	70	20				90
		% hatch	29.16	35.63				30.40
Or		eggs	523	158	15			696
		pupae	496	147	15			658
		% hatch	94.84	93.04	100.00			94.54