实验生物学论文选集

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内 容 简 介

本文集收集了李汝祺先生从1927—1966年四十年来在国内、外杂志上发表过的学术性论文和综述性论著，外文部分三十篇、中文部分十篇，共四十篇（并附中文或外文摘要）。其中包括细胞遗传学、胚胎学和实验生物学三大部分。现将它们汇集成册，重新发表。

可供细胞学、胚胎学、生态学、遗传学等有关科研人员和大专院校师生参考。

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自　　序

这本论文选集可以说是我个人教和学零星不全的札记。这里有些文章是我的导师毕瑞吉斯博士（Dr. C. B. Bridges）和我一起发表的，也有一些是我和我的同学、研究生在他们毕业论文的基础上一同写的。这些文章的署名一向是实际进行工作者列在前头，我个人的名字附在后头。这不是什么虚伪谦让而是实事求是。因为事实是在和同学一起研究问题的时候，我所得的收获远比我交给他们的多得多。

这里有一部分论文的内容已被国内外杂志和书刊所引用，还有一部分原来就在国外杂志上发表的。但这些都不能说明这些论文的水平。论文水平高低的标准应该是在本学科里有无新的贡献。如果用这一标准衡量这本选集的论文，除少数几篇同我的导师和两、三个研究生合作的以外，其余的大多是低水平的。不过在这些水平不高的论文中，我曾提出一些问题，而这些问题至今还没得到人们的注意。

这本选集原可以叫做实验动物学论文选集，但里面却夹杂着短短一篇有关玉簪花染色体的论文。玉簪花是植物，那末只好叫它为实验生物学论文选集了。对实验两字有人可能会有意见，因为选集中有些文章的内容是形态描述，用现代的眼光看似乎不应列入实验之类，但是大多数的工作还是有些实验性质的，所以才采用了实验两字。

选集作者从1926年回国后，即从事教学工作，并一开始就意识到要不断提高教学质量，就必须结合自己所教的课程做一些科学研究工作。我所教的课目较多，但大多集中在细胞学、胚胎学及遗传学范围以内。所以除果蝇的突变种外，其余的都是就地取材：如遗传学使用了瓢虫、胚胎学使用了北方狭口蛙、细胞学使用了中国马蛔虫。这些材料在全国都是取之不尽，用之不竭的。关于同学的论文工作，在选题上我一向先同他们进行协商，往往就在这些材料上提出自己的问题及解决问题的方法。许多的论文题目都不是我给他们出的，只是后来在贯彻高等教育六十条的1961—1965年这五、六年间，我们才系统地进行一些摇蚊唾腺染色体和X射线对小鼠卵巢发育影响的工作，并通过同学和研究生们的努力收集一定数量的资料。可惜的是这些资料，有些已写成文章；有的尚在计划处理中，“文化大革命”初期绝大部分都丢失了。从七十年代起，为了适应当时的科研项目必须与工、农、医实践相结合，我又和两位同事进行了一些肿瘤细胞超微结构变化的研究，并由我执笔写了四篇论文，在≪动物学报≫和≪北京大学学报≫上发表。但是，它们都属于同外单位协作项目并以集体名义发表的，所以都未被收入选集。

最后，我想在此向许多的友好同志表示我的衷心感谢。没有这些火热心肠的同志们的鼓励和大力帮助，我是不会有这样大的勇气和决心来把这些陈旧的写作公布于世，来暴露自己的不学无术。对我来说，惟一的安慰是由此可以得到同行们的批评指正，使我能够纠正过去的错误，学到一些更正确的东西。

李汝祺

1979年2月于北京大学生物系

目　　录

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THE EFFECT OF CHROMOSOME ABERRATIONS ON DEVELOPMENT IN *DROSOPHILA MELANOGASTER*[1)2)]

Ju-chi Li

(Columbia University, New York)

INTRODUCTION

In the course of the breeding work on *Drosophila* in the Columbia University Laboratory, a number of distinct somatic types have arisen that have been interpreted as due to deficiencies, duplications or translocations of parts of chromosomes, and to non-disjunction, resulting in the addition or loss of a whole chromosome. Some of these changes have been shown to be lethal in varying degrees, destroying individuals at specific stages of development. Such lethal effects are recognized mainly from distorted ratios among the surviving adults; and so far little effort has been made to determine the precise stage at which death takes place.

In the present work, the developmental stages of these flies are followed by the use of a new technique, by which eggs, larvae, and pupae could be handled in large numbers. As a detailed description of these methods is given in the appendix, only the most important features of the technique need be brought out here. The stocks used for analysis were first purified by selection and inbreeding with the purpose of making them homozygous. Each test was accompanied by control matings. Both the experimental and the control series were treated alike under identical environmental conditions. Matings were made in 4×1 inch vials; and the flies were raised in a constant temperature incubator (24°C) on standardized food. Eggs were isolated and counted every twenty-four hours. After hatching, a second egg count was taken to determine the number of eggs that hatched into larvae and also the number that died in the egg stage. The counts of the pupae followed; and finally, the adults that emerged from the pupa cases were classified to ascertain which, if any, of the expected classes were missing.

EXPERIMENTAL RESULTS

Notch deficiencies

The first "Notch" mutation was found in 1914 by Dexter (1914) who showed that the character was sex-linked, dominant in the female and lethal in the male. Bridges soon afterward found a second "Notch" and located it at 1.5 units to the right of the white locus (Morgan and Bridges 1916). Later several other Notches were found by Morgan, Bridges, Muller and Gowen. One, Notch 6, found by Bridges, showed a new

1) Received May 27, 1926.

2) Reprinted from *Genetics* **12**: 1—58, J. 1927.

characteristic for Notch, namely, "Pseudo-dominance" for facet (Metz and Bridges 1917). When the Notch 6 female was crossed to facet males, all of the Notch 6 daughters were at the same time facet. This similarity to the behavior of the forked in the forked-Bar deficiency (Bridges 1917) and vermilion in vermilion deficiency (Bridges 1919) suggested that Notch 6 is a deficiency that includes the locus for the facet character. Notch 8, found by Mohr in 1918, has been the object of extensive study (Mohr 1919, 1923). Notch 8 showed all the phenomena encountered in the previous Notches, but differed from them in involving a longer section. Thus, Notch 8 gave pseudo-dominance and exaggeration with facet, with white and its nine allelomorphs, located 1.5 units to the left of facet and with Abnormal, located 1.5 units to the right of facet. This interval of approximately 3 units is the minimum measure of the length of the deficient region. Very extensive linkage tests showed that crossing over was entirely eliminated from the deficient region. Up to the year 1924, twenty-five Notches had been discovered, four of which (N8, N9, N18, and N21) have been shown to be extensive enough to cover the locus for white.

Notch 8 of Mohr, Notch 18 of Bridges and two others, Notch 11 and Notch 19 (both found by Bridges), have been used by the author in making a comparative study of the differences in the length of the deficient regions. From linkage results with four mutant characters, namely, scute, broad, echinus and ruby (*sc br ec rb*) and later with five characters, namely, scute, broad, apricot, echinus and ruby (*sc br* w^a *ec rb*) through eight successive generations, the conclusion was reacred that the deficiency of Notch 18 is slightly greater than that of Notch 8 and also that in Notch 18 the adjacent regions are reduced somewhat in the amount of crossing over. The extent of deficiency of Notch 19 is probably less than that of Notch 11 and in both Notch 19 and Notch 11 is much less than in Notch 18 and Notch 8. (Data unpublished.)

Notch male: In carrying out the above mentioned linkage experiments the stocks of Notches (N8, N11, N18, and N19) were all crossed to the same stock (*sc br ec rb*) for eight generations, and they were then considered satisfactory material for an analysis of the fate of the Notch males, since in a large proportion of their autosomal genes they had presumably become homozygous and homogeneous. A forked stock (pedigree No. 15138) closely inbred for at least eighteen generations was obtained through the kindness of Doctor *Siurtevant*. For experimental matings, males from the forked stock were crossed to Notch females from the separate Notch stocks. Not-Notch *sc br* w^a *ec rb* sisters were mated to forked males and formed the control of the experiments. As will be seen in the data, only two of the four Notches were used in the experiment: Notch 8 represents the type of the longer affected region and Notch 19, the shorter (tables 1 to 4).

As shown in the above tables, the percentage of zygotes that died in larval and pupal stages is about the same in the experiments and in their respective controls. We do find, however, a great difference between the experimental and control series in the number of eggs that died, a difference amounting to approximately 25 percent of the total number of eggs. Since no Notch male appeared in the experiments, and since the sex ratio in the experiments was 2 females to 1 male and 1 to 1 in the controls, it is evident that the quarter of the zygotes that died as eggs were males that received the deficient X chromosome from the mother, namely, the Notch males.

Between Notch 8 and Notch 19, there was probably no difference in respect to the fate of the Notch males. A slightly higher general mortality (presumably in both males

Table 1 Notch 8/ *sc br w^a ec rb* ♀ × forked ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+♀	+♂	N♀
56	18	32.1	38	10	17.8	28	0	0.0	9	9	10
162	59	36.4	103	19	11.7	84	1	0.6	32	17	34
64	15	23.4	49	9	14.0	40	0	0.0	11	17	12
77	21	27.3	56	10	13.0	46	0	0.0	16	15	15
191[1)]	50	26.2	141	19	9.9	122	1	0.5	36	42	43
354	112	31.6	242	42	11.9	200	3	0.8	67	62	68
274	85	31.0	189	31	11.3	158	2	0.7	57	48	51
476	137	28.8	339	37	7.8	302	8	1.7	103	82	109
1654	497	30.0	1157	177	10.7	980	15	0.9	331	292	342

1) When an experiment has been repeated, the results of the second experiment have been tabled with those of the first. The spacing between lines, as shown in this table and similar ones that follow, is meant to separate two such results.

Table 2 Notch 8 control, *sc br w^a ec rb* ♀ × forked ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+♀	+♂	N♀
137	14	10.2	123	15	10.9	108	2	1.6	50	56	...
189	25	13.2	164	12	6.3	152	2	1.1	73	77	...
182	13	7.1	169	9	4.9	160	1	0.5	81	78	...
207	6	2.9	201	5	2.4	196	1	0.5	109	86	...
157	2	1.3	155	5	3.2	150	1	0.6	71	78	...
152	1	0.7	151	22	14.5	129	3	2.0	68	58	...
1024	61	5.9	963	68	6.6	895	10	1.0	452	433	...

Table 3 Notch 19/*sc br w^a ec rb* ♀ × forked ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+♀	+♂	N♀
104	33	31.8	71	9	8.6	62	1	1.0	21	17	23
128	42	33.8	86	18	14.0	68	1	0.8	22	22	23
28	9	32.0	19	6	21.4	13	1	3.6	5	3	4
133	42	31.6	91	32	24.0	59	1	0.8	29	19	19
83	29	35.0	54	9	10.8	45	1	1.2	17	14	13
87	51	58.5	36	10	11.5	26	1	1.1	9	6	10
128	32	25.0	96	5	3.9	91	0	0.0	29	29	33
172	122	71.0	50	8	4.6	42	0	0.0	8	14	20
863	360	41.7	503	97	11.2	406	6	0.7	131	124	145

and females, both Notch and not-Notch) in some of the matings in which Notch 19 was used may be accounted for as due to other modifiers in the stock. This is borne out by the fact that in the control matings of the Notch 19 experiment the same high mortality is present.

The result of the experiments on Notches is summarized in table 5 as follows:

Table 4 Notch 19 control, *sc br w^e ec rb* ♀×forked ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+♀	+♂	N♀
95	5	5.3	90	9	9.5	81	7	7.4	39	35	...
42	1	2.4	41	7	16.7	34	1	2.4	18	15	...
117	5	4.3	112	9	7.7	103	4	3.4	43	56	...
196	21	10.7	175	14	7.1	161	1	0.5	92	68	...
110	19	17.3	91	18	16.4	73	0	0.0	37	36	...
175	27	15.4	148	42	24.0	106	1	0.6	60	45	...
735	78	10.6	657	99	13.5	558	14	1.9	289	255	...

Table 5 Summary of the Notch experiments

CROSSES	EGGS	DISTRIBUTION OF DEATHS				ADULTS			REMARKS
		Eggs	Larvae	Pupae	Total	+♀	+♂	N♀	
N8 ♀×f♂	1654	30.0	10.7	0.9	41.6	331	292	342	Notch males
N19 ♀×f♂	863	41.7	11.2	0.7	53.6	131	124	145	died as eggs
Totals	2517	34.1	10.9	0.8	45.8	462	416	487	
N8 control	1024	5.9	6.6	1.0	13.5	452	433	...	Controls
N19 control	7∞5	10.6	13.5	1.9	26.0	289	255	...	
Totals	1759	7.9	9.5	1.3	18.7	741	688	...	

Autosomal deficiencies

Most of the sectional deficiency cases have been found in the X chromosome and are accompanied by a 2 : 1 sex ratio, for a male that receives a deficient X chromosome does not survive, as was illustrated in the Notch deficiencies. There is nothing in the Y chromosome that counterbalances the deleterious effect of these deficiencies, or, in other words, the Y chromosome contains no gene that replaces the losses incurred in the X chromosome.

Deficiencies present in the autosomes would be easily overlooked, unless the sections lost were so extensive that dominant character changes were produced, or unless such deficiencies were inbred, in which case a lethal effect would be manifest in homozygous condition. Two autosomal deficiencies that give dominant character changes have been found by Bridges and are here used to test the fate of the homozygous deficient types.

Plexate: Like Notch, Plexate is a mutation that affects the wings, causing a slight plexus condition of the veins. A single Plexate female was found by Bridges (June 16, 1922) in a cross of attached X female and normal male (data unpublished). The Plexate character proved to be dominant in outcrosses and to be lethal when homozygous, that is, when one Plexate is mated to another, the expected homozygous Plexate class is missing, giving only heterozygous Plexates and normal sibs in a 2:1 ratio. The viability of the mutant in heterozygous condition is excellent. By linkage tests, the gene for Plexate was found to be situated in the right end of the second chromosome. When Bridges

Table 6 Sooty Plexate ♀×sooty Plexate ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		Px	
									♀	♂	♀	♂
302	85	28.1	217	54	17.9	163	1	0.3	25	26	63	48
387	115	29.8	272	56	14.5	216	4	1.0	31	47	78	56
123	35	28.4	88	6	4.9	82	0	0.0	17	14	26	25
198	59	28.9	139	34	17.2	105	3	1.5	19	15	38	30
263	76	28.9	187	37	14.6	150	4	1.5	26	20	46	54
210	55	26.2	155	27	12.9	128	4	1.9	19	26	40	39
258	68	26.4	190	28	10.8	162	2	0.8	21	28	59	52
1741	493	28.3	1248	242	13.9	1006	18	1.1	158	176	350	304

Table 7 Sooty Plexate ♀×sooty ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		Px	
									♀	♂	♀	♂
341	18	5.3	323	45	13.2	278	2	0.6	72	76	69	59
314	38	12.1	276	47	15.0	229	3	0.9	64	57	53	52
237	27	11.4	210	34	14.3	176	0	0.0	47	39	38	52
166	19	11.5	147	25	15.0	122	0	0.0	33	29	33	27
1058	102	9.6	956	151	14.3	805	5	0.5	206	201	193	190

Table 8 Sooty ♀×Sooty Plexate ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		Px	
									♀	♂	♀	♂
296	8	2.7	288	22	7.4	266	2	0.7	74	65	69	56
220	5	2.3	215	14	6.4	201	0	0.0	57	45	48	51
288	6	2.1	282	37	12.8	245	2	0.7	56	55	68	64
307	11	3.6	296	33	10.7	263	2	0.7	56	67	69	69
1111	30	2.7	1081	106	9.5	975	6	0.5	243	232	254	240

Table 9 Sooty Not-Plexate ♀×Sooty ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		Px	
									♀	♂	♀	♂
302	23	7.6	279	33	10.9	246	1	0.3	123	122	···	···
340	16	4.7	324	39	11.5	285	0	0.0	152	133	···	···
296	7	2.4	289	40	13.5	249	4	1.4	126	119	···	···
938	46	4.9	892	112	11.9	780	5	0.5	401	374	···	···

crossed Plexate to speck, all the F_1 Plexates were exaggerated and gave pseudo-dominance for speck. The same relation held for balloon. A region from speck to balloon (0.5 ± unit) therefore gives the minimum measure of the deficient section.

In order to compare this case of autosomal deficiency with that of Notch, a test of the homozygous and heterozygous Plexates was made. The Plexate stock was first rear-

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ranged by outcrossing Plexate males to sooty females of the inbred sooty stock (See Appendix p. 76) for two successive generations. The F_2 sooty Plexate females were then backcrossed to the sooty stock, in order to substitute by crossing over a part of the second chromosome. Two more backcrosses with the females were thus made before the following analyses were carried out: The experimental series consisted of three types of matings, namely, Plexate by Plexate, Plexate ♀ by sooty ♂ and sooty ♀ by Plexate ♂; the controls were reciprocal crosses between not-Plexate sibs and pure sooty flies (tables 6, 7).

Between the controls and the sooty ♀ by Plexate ♂ series (tables 8, 9, 10), no significant difference could be found in the number of deaths in any of the three developmental stages. It is evident, therefore, that heterozygous Plexates all came through under the experimental conditions. Comparison of the reciprocal crosses of the experimental series (compare table 7 with table 8) shows that when Plexate females were used, more of the flies died in both egg (6.9 percent) and larval stages (4.8 percent). This additional mortality was apparently due to the "maternal effect" of the Plexate females.

In the Plexate by Plexate series (table 6), the percentages of mortality occurring in larval and pupal stages were not appreciably different from those in the controls. But in the egg stage, there were approximately 25 percent more of the zygotes that died in the experiment than in the controls. The conclusion seems logical that the homozygous Plexate flies died as eggs.

Table 10 Sooty ♀×Sooty Not-Plexate ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		Px	
									♀	♂	♀	♂
301	10	3.3	291	27	9.0	264	0	0.0	133	131	···	···
280	8	2.9	272	23	8.2	249	2	0.7	135	112	···	···
581	18	3.1	563	50	8.6	513	2	0.3	268	243	···	···

When such unhatched eggs were examined under high power, it was found that the embryos had reached a distinctly advanced stage in their development. They showed well developed pharyngeal apparatus and setae on the transverse body ridges. Even the tracheal system was more or less complete. These eggs invariably turned reddish brown and disintegrated. Since the egg mortality in the present case was only slightly complicated by maternal effect, and since practically 90 percent of the unhatched eggs reached this specific and characteristic stage of development, we can hardly avoid attributing this specific lethal effect to the homozygous deficiency.

It will be noted, in passing, that the results shown in the tables of the present experiment are remarkably consistent in all the pairs tested as compared with those of the Notch experiments. This contrast is due to the difference in the time of which these tests were made, that is, the Plexate experiment was carried out at the very last stage of the present work, whereas the Notch work was done more than a year earlier. In the meantime, the technique of handling the material had been improving. Furthermore, in the case of the Notches, two or even three pairs were put into each vial, and not a single vial was discarded from the final counts, although in some vials all the females had died before the fifth day of the experiment.

Table 11 Sooty Minute-1 ♀×sooty Minute-1 ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		M-1	
									♀	♂	♀	♂
205	71	34.6	134	17	8.3	117	8	3.9	26	19	33	31
262	82	31.3	180	44	17.5	130	0	0.0	27	22	37	50
337	115	34.1	222	63	18.7	159	9	2.2	25	30	54	41
122	42	34.4	80	20	16.4	60	1	0.8	10	9	12	28
167	51	30.6	116	21	12.6	95	3	1.8	23	12	31	26
139	48	34.5	91	22	15.8	69	4	2.9	9	12	17	27
177	64	36.2	113	11	6.2	102	4	2.3	21	15	29	33
1409	473	33.6	936	198	14.1	738	29	2.1	141	119	213	236

Table 12 Sooty Minute-1 ♀×Sooty ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		M-1	
									♀	♂	♀	♂
243	29	11.9	214	38	15.6	176	7	2.8	40	50	36	43
212	37	17.4	175	42	19.8	133	4	1.9	32	28	35	34
230	34	14.8	196	45	19.6	151	1	0.4	36	35	33	46
130	15	11.5	115	15	11.5	100	2	1.5	35	23	18	22
1029	145	14.1	884	173	16.8	711	16	1.5	175	171	164	185

Table 13 Sooty ♀×sooty Minute-1 ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		M-1	
									♀	♂	♀	♂
264	7	2.7	257	9	3.4	248	4	1.5	68	63	66	47
311	8	2.6	303	22	7.1	281	5	1.6	66	70	82	58
245	5	2.0	240	16	6.5	224	4	1.6	57	49	54	60
280	13	4.6	267	32	11.4	235	7	2.5	61	61	51	55
1100	33	3.0	1067	79	7.2	988	20	1.8	252	243	253	220

Table 14 Sooty not-M-1 ♀×sooty ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		M-1	
									♀	♂	♀	♂
280	11	3.9	269	12	4.3	257	4	1.4	131	122	...	...
351	7	2.0	344	38	10.8	306	3	0.9	149	154	...	...
236	5	2.1	231	8	3.4	223	0	0.0	125	98	...	...
867	23	2.7	844	58	6.7	786	7	0.8	405	374	...	...

Minute-1: "Minutes" are mutations characterized by tinier and slenderer bristles, smaller size of body, late hatching and usually poorer viability than the normal flies. Minutes are dominants that are lethal when homozygous. A brief account of these mutants has been published by Morgan, Bridges and Sturtevant in the Year Books of the Carnegie Institution from 1923 to 1925. Minute-k, Minute-n and Minute-o were shown

Table 15 Sooty ♀×sooty not-M-l ♂

EGGS	EGGS DIED	PER-CENT	LARVAE	LARVAE DIED	PER-CENT	PUPAE	PUPAE DIED	PER-CENT	+		M-l	
									♀	♂	♀	♂
171	3	1.8	168	4	2.3	164	0	0.0	84	80	...	...
273	7	2.6	266	26	9.5	240	3	1.1	111	120	...	...
444	10	2.3	434	30	6.8	404	3	0.7	195	206	...	...

to be sexlinked, dominant in the female and lethal in the male and are possibly deficiencies similar to the Notches. Among the autosomal Minutes may be mentioned Minute-h, Minute-i, and Minute-j, located in the third chromosome, Minute-l, Minute-m and Minute-p in the second chromosome. The supposition that many Minutes are probably deficiencies is strengthened by the demonstrative work with Minute-l.

Among the offspring of a cross between a triploid female and a diploid male, Bridges found one Minute female (July 7, 1923, result unpublished). He named the mutant Minute-l. Like all other Minutes, it is a dominant character and lethal when homozygous. It gave a reduction of crossing over values in the right end of the second chromosome. Its locus was found to be in the neighborhood of plexus. When it was crossed to plexus, it gave exaggerated Minute-l and plexus compounds. It also showed pseudo-dominance with arc, plexus and arc being about one unit apart. No crossing over has so far been observed between arc and plexus in the presence of Minute-l. Thus, this particular case fulfills the expectations for a typical sectional deficiency.

The analysis of Minute-l, as to the fate of homozygous and heterozygous Minutes, was made simultaneously with the Plexate case. Precisely the same procedure was followed in both cases. The sooty Minutes were finally used in tre tests appearing in tables 11 to 15, and the crosses between not-Minute sibs with the pure sooty formed the controls.

The results in tables 13, 14 and 15 show that the percentage of mortality in all three developmental stages was practically the same in the sooty ♀ by Minute-l ♂ series as in the controls. The heterozygous Minutes appear, therefore, to have practically all survived under the experimental conditions. A small number (about 1 percent) of the heterozygous Minutes died in an advanced pupal stage, and these when examined were found to be more often females than males. Under the ordinary breeding conditions, Minute-l is generally only about 60—70 percent as viable as normals. The improvement of the viability of the Minute-l flies in the present experiment may be attributable to a greater proportional amount of food, less larval crowding and to other more favorable environmental conditions of the vial method as contrasted to the usual milk bottle method of culture. Heterozygous Minutes, however, emerged later than their normal sibs.

When the Minute-l by Minute-l series is compared with the controls (tables 11 to 14) no significant difference in percentages of deaths in either the larval or the pupal stage is found. There was, however, an excess of approximately 25 percent of the zygotes that died in the egg stage in the experiment as compared with the deaths of eggs in the controls. The quarter of the total number of flies that died as eggs were presumably homozygous Minutes.

In all cases where the Minute-l females were used in the crosses (tables 11 and 12), there was a remarkably higher percentage of egg and larva mortality than in cases where

the sooty females were used. This is an example of maternal effect. The Minute-1 females regularly lay abnormal eggs, comparable to those observed in singed females (Mohr 1922). The eggs vary in size and shape through a wide range, but they do not approach those of the normal at one extreme, nor are they quite like the eggs of singed at the other. They are, as a rule, smaller than normal in size and more oval in shape, with a lesser degree of concavity on the dorsal side. The "floats" are more or less normal, but sometimes appear thinner and more delicate. By far the most interesting feature of these eggs is the character of the yolk. Instead of being opaque white as in normal eggs, the yolk is nearly transparent with finer glassy granules. The developing embryo inside of the egg is often visible. Most of the eggs on the extreme abnormal side failed to hatch or else hatched as undersized larvae. The percentages of such extremely abnormal eggs and of mortality resulting from maternal effect were very close.

Sectional duplications

The first "duplication" case in *Drosophila* was reported by Bridges (1919). It was interpreted as having arisen by a section of the X chromosome, including the loci for vermilion and sable, becoming detached from its normal location in the middle of the chromosome and jointed on to the "zero" end of its mate. This interpretation is challenged by the recent work of Bonnier (1926), who suggests that this so-called duplication is due to gene mutation, that is, to a recessive suppressor similar to others known (tan suppressor), rather than to a dislocation of allelomorphic genes. On the other hand, Pale translocation seems to be well established as a clear-cut case involving duplication.

Pale translocation: In preparing some stocks of sex-linked characters for work with lethal tumors, Bridges found that in one culture there were flies present with a light yellow eye color in addition to the expected eosin flies. These "Pale" flies were subsequently shown to be a specific dilution of eosin. Later, through extensive crosses, Bridges was led to the conclusion that the peculiar phenomenon encountered could be explained as due to the transference of a piece from the right end of the second chromosome to an attachment near the right end of the third chromosome, a rare case of autosomal translocation (Morgan and Bridges 1925).

Through study of the dominance relations of genes, it has been shown that for most mutant characters one dominant gene is the suppressor of two recessives. Hence the wild-type genes in the duplicating section act as dominant suppressors of all the mutants that lie to the right of arc in the second chromosome, including plexus, lethal-IIa, brown, blistered, purploid, morula, speck and balloon, amounting to seven units on the map. This is an accurate measure of the duplicating fragment. A further proof is found in the fact that a second chromosome which has lost its right end cannot undergo crossing over in that region with the normal homologous chromosome.

A cytological analysis seemed to show that the shorter pair of V's has one arm abnormally short (the right end of the second chromosome). It has not yet been established, however, that the third chromosome is increased by a corresponding section, (Morgan, Bridges and Sturtevant 1925).

This stock was examined by the author in the latter part of 1924. At first two recessive characters, plexus and speck, were used as an index of the different kinds of combinations of the sectional deficiencies and sectional duplications. Unfortunately, the

plexus speck chromosome was found to contain a certain recessive modifier that killed off a very high proportion of the zygotes at an early stage. About 75 percent of the eggs failed to hatch in the control series consisting of plexus speck by plexus speck matings. After many fruitless attempts to eliminate this modifier, the stock was finally abandoned; instead of using plexus and speck as an index, only plexus, of different origin, was used in the following work.

The locus of plexus is in that part of the second chromosome that has been transferred to the third chromosome. In the diagram of the relations in the Pale stock (figure 1) the section of the second chromosome (broad line) joined to the third chromosome (thin line) near its end carried a wildtype dominant allelomorph of plexus. Each of the normal second chromosomes of this stock (full length broad line) carries a recessive plexus gene (locus indicated by an arrow head). The second chromosome from which the end has been removed is indicated by the shorter broad line with the dotted end.

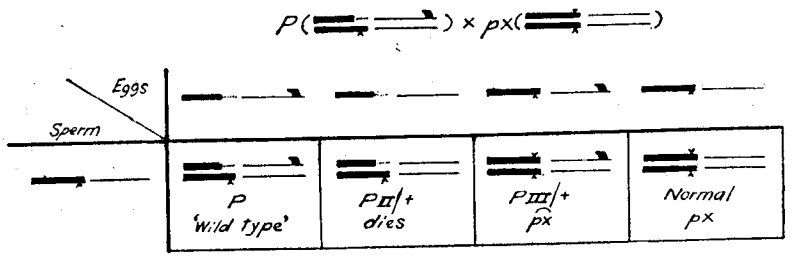

Figure 1

As seen in figure 1, there are three classes of adults in the Pale stock, namely, (1) the Pale translocation (symbol: P), (2) the heterozygous duplication (symbol: $P_{III}/+$) and (3) the normal. In the absence of speck and eosin, the task of differentiating these classes depends on the accurate separation of (1) the heterozygous plexus representing the Pale from (2) the semi-plexus (symbol: s-px) representing the heterozygous duplication, in which the homozygous plexus is partly suppressed by an allelomorphic wild-type gene, and (2) semi-plexus from (3) the homozygous plexus representing the normal condition of the chromosomes. While the heterozygous plexus is phenotypically a wild type and easily distinguished, the separation of semi-plexus from homozygous plexus is by no means easy, as there is a considerable variation in the expression of the plexus character in homozygous condition.

A careful comparative study of a homozygous plexus stock with that in which 40—50 percent of the flies were expected to be of the semi-plexus type led to the discovery of a reliable criterion. In the first place, the degree of the plexus condition in the semi-plexus type is generally considerably less than in the homozygous type, but an infallible character is found in the differences in the second posterior cell of the wing. In the homozygous plexus, no matter how near it may approach otherwise in appearance to the semi-plexus condition, the posterior crossvein invariably branches out into the posterior cell, whereas in semi-plexus the cell is clean like that of the normal wings. This was made certain by testing many of these supposed semi-plexus individuals, and in every case their offspring gave the expected results.

With the Pale, the semi-plexus and homozygous plexus types differentiated and the stock purified by an adequate amount of inbreeding and selection, experiments were

planned to test the fate of the different aberrations. This was done by mating these three classes of flies in all possible combinations. Six series of crosses were thus made, three of which were with reciprocal crosses:

1. Homozygous plexus by homozygous plexus of course forms the control of the experiments, all of the flies in this series have normal chromosomes and are expected to reach the adult stage. The proportions of deaths of the homozygous plexus class that occur in this control may therefore be expected to occur in each of the other experiments in addition to the deaths due to the specific chromosome aberrations.

2. A cross of homozygous plexus with semi-plexus gives in one-half of their progeny heterozygous duplication on the third chromosome ($P_{III}/+$) which, according to Bridges, is only 80 percent as viable as the normal under ordinary culture conditions.

3. When semi-plexus is mated to semi-plexus, one-quarter of the offspring receives both of the duplication-bearing third chromosomes (symbol: P_{III}/P_{III}). These flies die. The ratio of adults expected from such a cross, therefore, is approximately two semi-plexus to one homozygous plexus.

4. In the homozygous plexus by Pale cross (see figure 1), one-quarter of the progeny contains both the deficient second chromosome and the duplication-bearing third chromosome and reconstitutes the Pale class. Another quarter receives the deficient second chromosome. The heterozygous condition for Pale deficiency (symbol: $P_{II}/+$) is lethal. A third quarter receives the duplication-bearing third chromosome and forms the semi-plexus class. Finally, the fourth quarter, having both the normal second and the normal third chromosomes, gives the homozygous plexus class with normal chromosomes and viability.

5. A more complicated situation is to be found in the cross between semi-plexus and Pale. The number and kind of zygotes produced by such a cross are shown in figure 2.

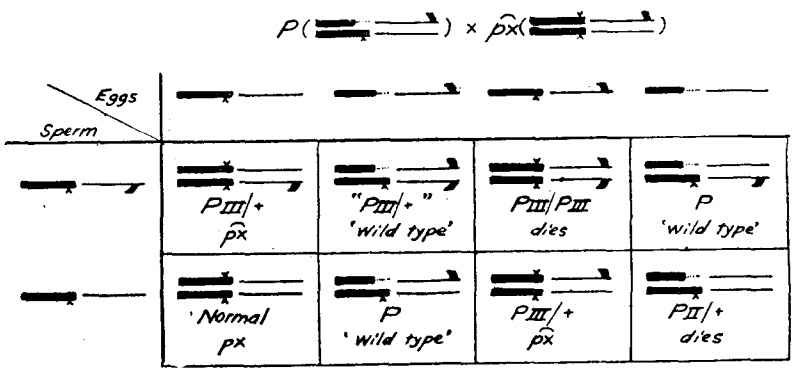

Figure 2

As seen in the above diagram, two of the eight combinations are fatal, namely, homozygous duplication of the third chromosome (P_{III}/P_{III}), and the heterozygous deficiency of the second chromosome ($P_{II}/+$), amounting together to 25 percent of the total number of zygotes. The remaining three-quarters, that survive, consist of three classes, the heterozygous plexus, semi-plexus and homozygous plexus in a zygotic ratio of 3:2:1. Of the heterozygous plexus class, two-thirds are Pales of the usual type and one-third is of the constitution in which a homozygous duplication is partly balanced by a hetero-

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