A DEVIANT SEX RATIO IN *DROSOPHILA MELANOGASTER*[1]

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Received December 31, 1968

A substantial number of cases of deviant sex ratio has been found in species or strains of various animals, that are probably due either to meiotic drive, as defined by SANDLER and NOVITSKI (1957), or to gametic selection or competition. Deviant sex ratios have been reported by GERSHENSON (1928), STURTEVANT and DOBZHANSKY (1936), MCILHENNY (1937), NOVITSKI (1947, 1953), MCWHIRTER (1956), WEIR (1958), NOVITSKI and HANKS (1961), STALKER (1961), WILKES (1964), YANDERS (1965), HANKS (1965), HICKEY and CRAIG (1966) and HANKS (1968).

HANKS (1965) showed that a strain of Oregon R consistently gave a grand mean of approximately 53.5%, that this was due to the genotype of the male parent, and that the time of action was probably prezygotic. It was suggested that the deviant sex ratio was due to preferential segregation of the *X* to the "functional" pole and the *Y* to the "non-functional" pole during spermatogenesis.

YANDERS (1965) found a significant trend in the sex ratio of an Oregon-R strain that was dependent upon the age of the male parent at the time of mating. The trend was towards a greater proportion of females with increasing age and it was suggested that the trend was due to preferential segregation of the *Y* chromosome to the "non-functional" pole.

The present analysis attempts to determine which chromosome is responsible for the deviant sex ratio in another strain, *bw st* F-7, and whether a zygotic or prezygotic phenomenon is involved.

MATERIALS AND METHODS

The strain of Drosophila analyzed, *bw*, brown, *st*, scarlet F-7, was originally used at Oak Ridge, Tennessee, but the original source is unknown. It has been kept by mass transfer since 1957. The *bw st* F-11 and *dp*, dumpy, e^{11}, ebony F-8 strains, which were used as controls, came from the stock center at Philadelphia. The Canton S and *y*, yellow B-32 strains came from E. NOVITSKI; the *se*, sepia D-4 strain from the stock center at California Institute of Technology, and the spa^{pol}, sparkling-poliert E-2 strain from B. HOCHMAN. All of these strains have been kept by mass transfer in half-pint bottles. All males to be tested were raised in incubators at 25 ± 1°C but were mated and the progeny reared at approximately 23° to 25°C. Extensive testing of males was done by mating to five females and subculturing twice allowing the flies to remain in a culture bottle for three days. Each culture was counted every 1 to 2 days to completion. Single males were used for all extensive tests. A minimum of 200 progeny were required before the test was included in the results, and this value was chosen before the experiments were performed. Several

[1] This investigation was supported by a grant from the Public Health Service (GM 13393-01).

males and females were mated in cultures begun to obtain males for extensive testing. Females used for testing purposes were raised in mass cultures. Flies were cultured on yeast, cornmeal, sugar, agar, and propionic acid food seeded with live baker's yeast. Zephiran (benzalkonium chloride) was used to control bacterial growth.

RESULTS AND DISCUSSION

The nature of the inheritance of the deviant sex ratio: Males from the *bw st* F-7 strain show a 53.3 grand mean percentage of females compared with 49.9 for *dp* e^{11} (F-8) control males (Table 1). The deviation persists (59.9 and 54.4) even when males were tested using different strains of females (Tables 1, 2). Sepia, sparkling-poliert, and yellow females all give deviant sex ratios when mated to *bw st* F-7 males. This consistently high percentage of females in progenies from *bw st* F-7 males suggests that the trait is a stable genetic characteristic.

Reciprocal matings were made between the *bw st* F-7 strain and a *y* strain (B-32) (Table 2). When *y* B-32 males are mated to *bw st* F-7 females, the mean percentage of females is 49.4, but when *bw st* F-7 males are mated to *y* B-32 females, the mean percentage of females is 54.6 (Table 2). Since the strain of the female parent does not account for the sex ratio deviation either in the data Table 1 or in the data from reciprocal matings, the largest component determining the excess of females is apparently determined by the genotype of the male parent. Any effect of the yellow phenotype on the male progeny produced when *y* B-32

TABLE 1

Sex ratios of bw st *F-7 and* dp e^{11} *F-8 control males*

5 *se* females × *bw st* F-7 male		5 *spa*^{pol} females × *bw st* F-7 male		5 *se* females × *dp* e^{11} F-8 male	
Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females
932	52.5	716	61.9	794	49.8
635	52.9	394	53.6	947	50.7
764	52.5	490	62.0	692	48.8
1100	55.0	486	59.0	822	48.8
1126	51.7	531	58.4	265	58.1
882	50.1	741	59.9	482	47.9
834	57.8	807	57.4	532	49.1
799	50.9	692	59.5	680	50.9
1089	51.2	475	64.4	461	45.8
1280	52.7	495	63.2	671	49.9
1257	50.5	635	61.3	726	47.5
513	50.3	511	61.1	467	52.2
857	57.6	494	52.0	820	49.3
697	52.1	525	68.0	...	...
1145	59.0	747	59.0	...	...
995	54.2	580	62.6	...	...
1082	51.8	362	61.6	...	...
1046	54.1	739	52.6	...	...
999	55.0	...	...	...	...
$\bar{X} = 53.3$		$\bar{X} = 59.9$		$\bar{X} = 49.9$	
$n = 19$		$n = 18$		$n = 13$	

TABLE 2

Reciprocal matings between the two strains, y *B-32 and* bw st *F-7*

y B-32 female × *bw st* F-7 male		*bw st* F-7 female × *y* B-32 male	
Total progeny	Percent females	Total progeny	Percent females
554	54.2	574	49.8
742	53.4	670	52.8
725	52.8	746	48.1
565	57.5	469	48.2
925	54.6	419	51.3
980	55.2	338	46.8
615	54.3	210	45.2
817	55.5	237	46.8
760	56.6	259	47.5
1043	53.9	382	52.4
891	56.1	792	53.8
781	52.5	310	48.4
1159	53.9	430	50.7
931	54.4	...	...
879	54.4	...	...
785	54.4	...	...
722	53.6	...	...
925	53.7	...	...
715	55.2	...	...
608	55.6	...	...
$\bar{X} = 54.6$		$\bar{X} = 49.4$	
$n = 20$		$n = 13$	

females were used is thought to be slight, because mating Canton-S males to *y* B-32 females gives a grand mean of 51.7% females in a total of 1883 progeny. In addition, Canton-S males characteristically give close to 51% females when mated to females from strains other than yellow (HANKS 1965 and unpublished).

Genetic analysis of the deviant sex ratio: Results from reciprocal matings and tests of F_1 males allow the exclusion of the *X*, the *Y*, and a simple dominant autosomal effect as the main cause. Reciprocal matings were made between *bw st* F-7 and a control strain, *dp* e^{11} F-8, and F_1 males were tested extensively (Tables 3, 4). In no case (47.0, 51.2, 48.4, 50.3, 49.7) did a grand mean show a significant excess of females. Further, these results exclude any effect of the *X* in causing the excess of females characteristic of *bw st* F-7 males. If the *X* were the cause of the deviant sex ratio, then the F_1 males resulting from a cross of *bw st* F-7 females to *dp* e^{11} males would have given grand means significantly higher than males from reciprocal matings. The *Y* chromosome is also excluded as a simple cause of the deviant sex ratio. If it were the cause, the F_1 males carrying the *Y* obtained from the *bw st* F-7 males would give a deviant sex ratio while those not carrying it would not give a deviant sex ratio. If there were a simple dominant autosomal effect, then the F_1 males would be expected to show at least an intermediate deviant sex ratio. They clearly do not.

The F_1 male progeny were backcrossed to females from the *bw st* F-7 strain

TABLE 3

Results of testing F_1 males from reciprocal matings of the dp e^{11} *F-8 strain and the* bw st *F-7 strain to 5* se *D-4 females*

bw st F-7 females × *dp* e^{11} males *se* females × F_1 male		*dp* e^{11} females × *bw st* F-7 males *se* females × F_1 male		*dp* e^{11} females × *bw st* F-7 males *se* females × F_1 male	
Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females
1170	54.0	826	51.0	1124	49.9
1619	50.4	597	50.4	906	51.6
1317	51.6	803	53.6	1218	49.7
1177	51.1	797	53.2	993	51.4
1449	49.8	1038	50.4	1382	47.9
1165	52.6	822	49.4	1256	52.8
1310	50.3	929	51.2	695	49.5
1331	50.2	894	51.1	1321	48.5
1081	52.8	863	48.3	1056	51.4
1330	49.9	683	52.6	1537	47.6
1450	52.5	895	50.2	1251	49.3
1382	52.0	1035	51.7	1177	48.6
1517	50.9	782	49.0	1503	48.5
1548	50.0	769	48.8	1405	49.1
1390	49.2	549	51.7	...	...
...	...	789	50.7	...	...
...	...	593	43.6	...	...
...	...	983	49.3	...	...
$\bar{X} = 51.2$		$\bar{X} = 50.3$		$\bar{X} = 49.7$	
$n = 15$		$n = 18$		$n = 14$	

and the four classes of backcross progeny were tested extensively (Table 5). In the first cross the wild-type males gave a 50.6% female grand mean; *bw*, 51.9%; *st*, 54.6%; and *bw st*, 55.2%. The KRUSKAL-WALLIS one-way analysis of variance (SIEGEL 1956) shows that the grand means of the four classes are not all the same ($R < .001$).

In the second cross it was found, as expected from the first cross, that the + and *bw* classes of males give grand means different from either the *st* or *bw st* classes. Grand means (Table 6) were: for +, 49.6%; *bw*, 51.3%; *st*, 54.3%; and *bw st*, 54.7%. All P values obtained using the Mann-Whitney U test (SIEGEL 1956) were $< .001$.

Similarly in the third cross grand means (Table 7) were: for +, 51.0%; *bw*, 50.9%; *st*, 52.7%; and *bw st*, 53.7%. All of the P values testing significance between + and *st* or *bw st* or *bw* and *st* or *bw st* were $< .05$. In all three experiments the *st* and *bw st* classes of males always gave grand mean sex ratios more deviant than the + and *bw* classes of males. If the deviant sex ratio were caused by a recessive gene on the second chromosome, one would expect the *bw* and *bw st* classes to give more deviant sex ratios than the + and *st*. classes. This is clearly not found, and thus a second chromosome recessive gene can be excluded.

If the deviant sex ratio resulted from a recessive gene on the fourth chromosome, approximately one-half of the backcross progeny would be heterozygous.

TABLE 4

Results of testing F_1 males from reciprocal matings of the dp e^{11} *F-8 strain and the* bw st *F-7 strain*

bw st F-7 females × *dp* e^{11} males 5 *se* females × F_1 males		*dp* e^{11} females × *bw st* males 5 *se* females × F_1 males	
Total progeny	Percent females	Total progeny	Percent females
1362	44.8	849	50.9
901	51.0	641	47.7
506	46.6	973	49.3
1066	48.2	970	48.9
949	47.7	882	49.4
764	48.4	383	47.8
703	46.9	677	46.5
1166	47.1	877	50.7
1088	48.6	757	48.5
1133	47.0	615	52.0
1135	47.4	756	43.7
854	47.2	813	48.5
766	39.8	852	49.9
421	45.4	543	44.9
1393	51.3	652	46.0
1163	50.6	579	45.6
509	47.3	559	48.3
640	45.2	778	52.1
847	42.1	761	48.2
$\bar{X} = 47.0$		$\bar{X} = 48.4$	
$n = 19$		$n = 19$	

One would then expect all classes to be intermediate. Clearly this is not found; thus a main effect of the fourth chromosome can be excluded.

The association of the deviant sex ratio with *st*, shows that genetic material in the third chromosome determines the deviant sex ratio. Results of tests with a control strain exclude the *st* gene itself as the cause of the deviant sex ratio. Males from a control *bw st* strain, F-11, were crossed to *dp* e^{11} F-8 females. Males from F-11 give a non-deviant sex ratio (51.9, Table 8). The grand mean of the F_1 was 50.9. The F_1 males were also backcrossed to *bw st* F-11 females and four classes of backcross progeny males were tested, giving + 50.0%; *bw*, 50.6%; *st*, 50.3%; and *bw st*, 51.1% (Table 9). These results indicate that the *st* phenotype itself does not give rise to the deviant sex ratio, but that other genetic material in the third chromosome of the *bw st* F-7 strain does so.

Zygotic mortality versus *a prezygotic phenomenon*: *A priori* one might expect that the deviation in favor of females found in the *bw st* F-7 strain would be due to the loss of more males than females during development. A close look at the data shows little evidence for this contention.

The most compelling evidence against the zygotic mortality cause of the deviant sex ratio is explained below. The data obtained by testing backcross progeny males extensively show that there is an association of the deviant sex ratio with

TABLE 5

Results of tests of backcross progeny males mated to 5 se D-4 females

P_1 *bw st* F-7 females × *dp* e^{11} males
bw st F-7 females × F_1 males

+ males		*bw* males		*st* males		*bw st* males	
Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females
1610	50.2	770	47.8	1227	55.8	1048	57.7
1201	51.0	1131	51.2	1140	55.5	877	53.0
1403	48.4	1185	53.8	1346	56.5	874	52.9
856	50.8	698	49.6	986	54.9	938	54.3
1148	48.3	698	53.6	1241	60.0	1290	56.1
959	49.0	1150	49.4	969	53.9	1214	56.1
775	51.5	1258	50.2	806	56.1	916	53.5
1219	51.9	958	51.2	950	53.7	1026	53.2
927	48.9	772	54.2	1198	57.0	1196	57.6
1207	51.4	1114	50.6	1125	54.2	874	54.7
1142	49.4	1176	51.7	1248	50.2	889	55.0
1151	51.2	967	53.7	1180	54.2	1040	55.5
1490	54.2	911	51.9	1626	53.4	771	55.4
1110	49.8	1000	53.5	888	51.0	795	55.7
1049	51.8	1021	53.3	1332	54.1	1179	52.9
1192	51.1	1286	52.6	811	53.9	928	57.0
...	...	1265	53.8	...	...	1431	58.7
...	...	891	52.8	...	...	807	53.4
...	...	...	...	...	...	1378	55.2
...	...	...	...	...	...	1271	55.2
$\bar{X} = 50.6$		$\bar{X} = 51.9$		$\bar{X} = 54.6$		$\bar{X} = 55.2$	
$n = 16$		$n = 18$		$n = 16$		$n = 20$	

KRUSKAL-WALLIS one-way analysis of variance shows result to be highly significant ($P < .001$).

males that contain both third chromosomes from the *bw st* F-7 strain. The deviant sex ratio is expressed in their progeny. Thus zygotic mortality would have to be due to a dominant gene with incomplete penetrance. One would predict that such a gene on the third chromosome would have an effect in the progeny whether transmitted by the male or female parent; however, in the reciprocal matings the *bw st* F-7 female parent does not produce deviant sex ratio progenies (Table 2, 49.4%).

One would also expect any dominant gene with incomplete penetrance to have an intermediate effect on the progenies from males that are heterozygous for the gene. F_1 males from a cross of *dp* e^{11} F-8 × *bw st* F-7 would be heterozygous. The grand means are 47.0, 51.2, 48.4, 50.3, and 49.7 (Tables 3 and 4). The + and *bw* backcross progeny males are also heterozygous for the third chromosome gene. The grand mean percentages of females for their progenies are 50.6, 51.9, 49.6, 51.3, 51.0, and 50.9 (Tables 5, 6, and 7). Clearly there is no intermediate effect. Since these results do not show the expectation provided under the zygotic mortality hypothesis, they tend to exclude this hypothesis.

TABLE 6

Results of backcross progeny males mated to 5 se D-4 females

P_1 *dp* e^{11} females × *bw st* F-7 males
bw st F-7 females × F_1 males

+ males		*bw* males		*st* males		*bw st* males	
Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females
1152	50.5	747	51.5	1248	53.0	747	52.7
1263	49.6	479	49.7	1159	55.6	955	55.1
1281	50.2	844	50.0	1098	51.5	1057	54.3
1239	48.9	1296	49.4	1024	54.0	1272	54.1
1060	47.6	1106	51.8	745	54.9	655	51.1
798	48.8	933	49.9	1054	55.5	292	50.7
921	51.8	1094	53.3	1127	54.7	807	53.2
1303	51.7	1287	52.1	742	55.5	847	57.3
941	47.4	1328	51.7	519	53.2	799	55.3
1349	50.0	958	52.6	823	57.7	791	55.5
984	48.1	987	49.6	1251	54.0	1007	54.1
1079	51.0	903	51.6	688	51.7	695	55.8
1032	49.3	1262	50.7	987	52.2	930	56.7
766	50.4	1150	53.0	1263	56.3	892	56.3
1386	49.9	784	49.0	530	55.3	864	55.9
542	48.0	827	55.0	1126	54.8	885	56.0
1221	50.5	1227	53.8	1144	53.1	838	56.8
813	48.1	1103	54.3	1226	55.1	559	54.2
773	51.7	1035	49.2	602	52.3	1106	54.4
1351	49.4	700	46.9	857	55.2	...	...
$\bar{X} = 49.6$		$\bar{X} = 51.3$		$\bar{X} = 54.3$		$\bar{X} = 54.7$.....	
$n = 20$		$n = 20$		$n = 20$		$n = 19$	

Tests of significance between + and *st* or *bw st* or between *bw* and *st* or *bw st* show $P < .001$ (one tailed test).

Since the above evidence is inferential only it was thought desirable to obtain direct evidence on the question. Egg counts and eclosion data ordinarily give only total mortality with no estimate of the magnitude or direction of differential mortality during development. This weakness can be largely overcome by using yellow as a larval character. Yellow (B-32) virgins were mass mated to *bw st* (F-7) males (experimental) and Canton-S males (control). The progeny were collected during the first larval instar and separated into male or female classes in separate vials (10 larvae per vial). After the larvae had pupated the number of adults was counted, classified, and divided into arbitrary sets of data containing 110 to 145 larvae per set. Results are given in Table 10. The mean difference in eclosion between males and females was calculated and showed 3.98% greater mortality for the females from the *bw st* F-7 cross and 3.56% greater mortality for the males for the Canton-S cross. Not only is there not a greater mortality of males but there is a greater mortality of females in *bw st* F-7 strain than in the control strain ($P < .002$). Clearly, this shows that male mortality in post-egg development does not account for the deviant sex ratio in the *bw st* F-7 strain.

TABLE 7

Results of backcross progeny males mated to 5 se *D-4 females*

P_1 *dp e*^{11} females × *bw st* F-7 males
bw st F-7 females × F_1 males

+ males		*bw* males		*st* males		*bw st* males	
Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females
1001	49.0	1307	50.0	556	48.9	872	51.4
1075	52.6	1049	52.0	661	54.6	684	55.7
1021	58.8	1354	51.5	877	51.7	816	52.7
1058	50.7	570	52.3	1046	51.6	671	53.8
932	50.0	909	48.7	942	54.7	791	55.8
442	48.2	1033	50.6	951	52.0	1069	54.0
763	49.2	1024	52.5	838	52.6	1033	52.8
1001	51.2	1206	52.3	871	52.9	725	53.2
692	52.3	1124	49.7	1269	50.4	781	53.1
1038	50.2	808	50.0	960	51.8	953	56.4
633	46.9	1284	53.2	1078	53.2	766	55.7
721	53.4	863	49.5	909	57.3	942	53.3
1087	52.2	785	50.1	1242	52.8	1021	53.1
898	50.7	1216	51.1	954	50.4	594	51.7
1025	49.7	1452	50.6	762	52.4	801	54.1
1032	51.2	1086	50.0	1444	52.5	1092	52.3
...	...	...	...	855	56.1	1026	54.3
$\bar{X} = 51.0$		$\bar{X} = 50.9$		$\bar{X} = 52.7$		$\bar{X} = 53.7$	
$n = 16$		$n = 16$		$n = 17$		$n = 17$	

Tests of significance between + and *st* or *bw st* or between *bw* and *st* or *bw st* show $P<.05$ in each case (one tailed test).

TABLE 8

Tests of bw st *F-11 and of* F_1 *males from a cross of* bw st *F-11 males to* dp e^{11} *F-8 females (Control)*

5 *se* females × *bw st* F-11 male		P_1 *dp e*^{11} females × *bw st* F-11 males 5 *se* females × F_1 male	
Total progeny	Percent females	Total progeny	Percent females
640	47.7	1117	51.8
557	50.6	1024	51.7
418	54.8	1093	52.8
441	52.8	1339	51.1
370	51.1	1461	49.4
367	57.5	1051	49.7
407	49.9	1264	50.9
491	52.6	1542	51.0
237	49.8	1146	49.5
...	...	1098	51.8
...	...	1351	50.4
$\bar{X} = 51.9$		$\bar{X} = 50.9$	
$n = 9$		$n = 11$	

TABLE 9

Results of testing backcross progeny males (Control)

dp e^{11} females × *bw st* F-11 males
bw st F-11 females × F_1 males

+ males		*bw* males		*st* males		*bw st* males	
Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females	Total progeny	Percent females
1433	49.1	1416	51.0	1282	51.2	440	52.0
1450	51.2	1330	54.4	1217	50.9	803	47.3
1395	50.7	848	50.4	1092	48.9	404	48.5
1200	52.3	1414	47.8	1137	48.6	420	53.6
1136	50.8	1057	49.0	743	49.7	371	45.0
913	47.3	1367	48.7	1371	52.2	661	51.0
718	51.1	1503	50.6	1223	50.5	971	53.1
988	46.6	1274	50.4	1647	50.7	927	50.0
866	43.6	967	51.2	1021	51.0	790	52.9
1585	53.8	1496	50.9	1218	52.9	600	52.5
1324	50.1	1533	52.8	1026	50.1	517	53.2
1263	49.2	1536	50.1	1276	50.4	935	50.0
1274	52.8	991	50.9	1420	48.0	424	58.5
1165	50.9	1212	51.1	1092	49.6	238	49.6
1459	50.5	1472	51.0	605	48.9	979	48.3
1513	50.4	1196	49.9	1348	52.1	436	52.1
1319	50.2	1218	50.2	1346	50.1	423	51.3
$\bar{X} = 50.0$		$\bar{X} = 50.6$		$\bar{X} = 50.3$		$\bar{X} = 51.1$	
$n = 17$		$n = 17$		$n = 17$		$n = 17$	

Other tests using a different stock show that the *total* mortality for the egg stage alone is approximately 4%; therefore, it is quite improbable that excess male mortality in the egg stage is the immediate cause of the deviant sex ratio. We conclude that very probably there is a gene which when homozygous has a prezygotic effect which causes the deviant sex ratio.

The sex ratio values obtained by testing F_1 males (Table 3) from reciprocal crosses are 51.2 and 50.3 while those obtained (Table 4) in a repeat experiment are 47.0 and 48.4, respectively. While the comparable values are indeed statistically significant ($P < .05$) we interpret them to be due to uncontrollable environmental variables since values of this magnitude are rarely seen. The Table 4 results were included to show the complete range of variability found.

HANKS (1965) provided evidence that the genotype of the male parent of a strain of Oregon-R was important in determining the sex ratio in the progeny, and that it most probably was a prezygotic phenomenon. It has been shown that the deviant sex ratio in the *bw st* F-7 strain is due to a gene or genes on the third chromosome. The bulk of the evidence favors a prezygotic explanation. Probably the best hypothesis that can be used to explain this prezygotic phenomenon is the existence of a "functional-non-functional" pole at anaphase I (PEACOCK and ERICKSON 1965), with preferential segregation.

I wish to thank MRS. HELEN WEBB for excellent technical assistance and ROBERT WILSON and

TABLE 10

Relative survival of males and females during post-egg development
y B-32 ♀♀ × *bw st* F-7 ♂♂ (Experimental)

Females			Males			
Larvae	Adults	Percent eclosing	Larvae	Adults	Percent eclosing	Eclosing difference percent
145	97	66.90	150	107	71.33	+4.43
119	98	82.35	121	102	84.30	+1.95
136	96	70.59	144	118	81.94	+11.35
130	104	80.00	130	109	83.85	+3.85
129	98	75.97	131	103	78.63	+2.66
140	102	72.86	140	104	74.29	+1.43
130	101	77.69	130	113	86.92	+9.23
130	103	79.23	130	99	76.15	−3.08
1,059	799	....	1,076	855	....	+31.82
						$\bar{X} = +3.98$

y B-32 ♀♀ × Canton S ♂♂ (Control)

Females			Males			
Larvae	Adults	Percent eclosing	Larvae	Adults	Percent eclosing	Eclosing difference percent
120	107	89.17	121	95	78.51	−10.66
126	106	84.13	128	108	84.38	+ 0.25
121	114	93.44	120	101	84.17	− 9.27
120	106	88.33	120	102	85.00	− 3.33
120	105	87.50	120	105	87.50	0.00
120	107	89.17	120	107	89.17	0.00
120	109	90.83	120	109	90.83	0.00
110	104	94.55	110	98	89.09	− 5.46
957	858	....	959	825	....	−28.72
						$\bar{X} = -3.56$

Test of significance shows $P < .002$.

AFTON ARP for assistance in conducting experiments. I wish also to thank ROBERT SEEGMILLER for perfoming statistical tests. Stocks were generously provided by DRS. B. HOCHMAN and E. NOVITSKI.

SUMMARY

The deviant sex ratio of strain *bw st* F-7 was analyzed. The main cause is a recessive gene or genes on the third chromosome which produces the deviant sex ratio only when present in the male parent. The evidence probably rules out zygotic mortality as the cause and thus a prezygotic phenomenon is suggested.

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