GENETICS OF PARTHENOGENESIS IN *DROSOPHILA MELANOGASTER*. I. THE MODES OF DIPLOIDIZATION IN THE GYNOGENESIS INDUCED BY A MALE-STERILE MUTANT, *ms(3)K81*

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ABSTRACT

Sperm that are produced by males homozygous for *ms(3)K81*, a male sterile mutant of *Drosophila melanogaster*, are defective in syngamy but are capable of activating eggs to develop gynogenetically. The activated eggs usually produce haploid embryos, but a small fraction (10^{-4}–10^{-5}) of them give rise to diploid impaternate adults. To know the cytological mechanisms by which these impaternates restore diploidy, the genotypes of impaternate progeny obtained from females doubly heterozygous for visible markers were examined. The results show that, as generally found among parthenogenetic Drosophila, diploidy is restored after completing meiosis either by pronuclear fusion or by gamete duplication (doubling of a haploid cleavage nucleus). The fusion of two nonsister nuclei following meiosis II (central fusion) was indicated to be a predominant mode of diploidization in this species. Two meiotic mutants, *mei-9* and *mei-S332*, which are known to greatly increase meiotic nondisjunction, did not cause an increased incidence of impaternates. This seems to exclude the possibility that some impaternates might have been derived from diploid egg nuclei produced through nondisjunction.

AFTER examining more than half-a-million unfertilized eggs of *Drosophila melanogaster*, HARRISON D. STALKER concluded, "These tests with *D. melanogaster* seem to indicate that . . . parthenogenesis in this species does not constitute a serious complication in genetic experimentation" (STALKER 1954). An absence of parthenogenesis in *D. melanogaster* has been propitious for a vast area of genetics. However, the genetics of parthenogenesis is still poorly understood. Much controversy on the evolutionary origin of parthenogenetic organisms seems to have been evoked by our lack of knowledge about the genetic mechanisms controlling parthenogenesis (for recent reviews, see CUELLAR 1974, 1977; MAYNARD SMITH 1978; SUOMALAINEN, SAURA and LOKKI 1976; TEMPLETON 1983; WHITE 1978).

Recently, a unique male sterile mutant, *ms(3)K81*, that induces gynogenesis was discovered in a natural population of *D. melanogaster* (FUYAMA 1984).

Males homozygous for *ms(3)K81* produce motile sperm and transmit them at copulation. These sperm can activate eggs to initiate development, but they never participate in syngamy. The eggs thus activated usually produce haploid embryos that die before hatching. Occasionally, they develop normally to give rise to impaternate adults. These impaternates are invariably diploid. This shows that the unfertilized egg of *D. melanogaster* has a capacity to restore diploidy, which is a prerequisite to automictic parthenogenesis (TEMPLETON 1983). The absence of parthenogenesis in this species, as well as in many other obligatorily bisexual species of Drosophila, may be understandable if we assume that, in these species, stimulation by sperm is indispensable to egg activation. If this should be the case, the above system would provide us a very useful tool with which to study the genetics of parthenogenesis. However, because the rate of the occurrence of the gynogenetic progeny is very low (around 10^{-5}; FUYAMA 1984) and because gynogenesis is quite uncommon in insects (ENGELMANN 1970), there exists a possibility that the impaternates might be produced by those mechanisms that are peculiar to the insemination by *ms(3)K81* sperm and/or are not involved in usual parthenogenesis. It is desirable, therefore, to determine how eggs can restore the diploidy in this system. The present paper will show that the mechanisms of diploidization in the gynogenesis induced by *ms(3)K81* are indistinguishable from those prevailing among automictic thelytokous parthenogens.

MATERIALS AND METHODS

Strains: *ms(3)K81* (map position: *3*-91.3) has been described (FUYAMA 1984). The cytological position of this mutant was localized to 97D3-5 by the use of the deficiencies around the rough locus, which were kindly supplied by D. R. KANKEL (Y. FUYAMA, unpublished results).

Descriptions for two meiotic mutants, *mei-9* and *mei-S332* are given in BAKER and HALL (1976), and for other marker strains used, see LINDSLEY and GRELL (1968).

Flies were reared on a cornmeal-yeast-glucose medium at 25°.

Tests for the modes of diploidization: The protocol described by CARSON (1973) was employed to study the mode of diploidization in the eggs activated by the *ms(3)K81* sperm; the genotypes of impaternate progeny of females heterozygous for two linked recessive markers were determined by a progeny test. Fifty virgin females heterozygous for given markers were mated with 25–30 homozygous *ms(3)K81* males in a culture vial (3 × 10.5 cm); they were transferred to a new vial every 4 to 5 days, allowing them to oviposit for 16 days. Because of very low productivity of parthenogenetic progeny (at most a few larvae per vial), overcrowding did not occur, although oviposition might have been depressed to some degree due to limited space. Female impaternate offspring were individually crossed to males homozygous for the markers, and the resulting progeny were examined to determine the genotype of each impaternate.

Three experiments were performed, each employing a different combination of markers:

Experiment 1: F_1 females produced from reciprocal crosses between *cn bw* (*cinnabar, 2*-57.5; *brown, 2*-104.5) and a wild-type strain were used as the mothers.

Experiment 2: Females carrying two sex-linked recessive markers, *white-apricot* (*1*-1.5) and *miniature* (*1*-36.1), in repulsion (w^a +/+ *m*) were used.

Experiment 3: Females were used which were doubly heterozygous for all the three major chromosomes produced by crossing a *cv f* (*crossveinless, 1*-13.7; *forked, 1*-56.7) stock and a *cn bw; ri e* (*radius incompletus, 3*-47.0; *ebony, 3*-70.7) stock.

TABLE 1

Productivity of impaternate progeny by the females heterozygous for recessive markers

Genotype of mother	No. of mothers tested	No. of impaternates produced			Impaternates/ mother
		Female	Male	Gynander	
cn bw/+ +	5000	66	7	0	0.0146
w^a +/+ *m*	8500	65	4	0	0.0081
+/*cv f;cn bw*/+;*ri e*/+	11550	50	5	1	0.0048

Impaternate productivity of meiotic mutants: To test for the possibility that the gynogenetic progeny might be derived from diploid gametes produced through meiotic nondisjunction, two meiotic mutants, *mei-9*^{a} and *mei-S332*, were tested for their productivity of impaternate progeny. These mutants are known to greatly increase the frequency of nondisjunction at meiosis I or II, respectively (BAKER and HALL 1976).

RESULTS AND DISCUSSION

Impaternate productivity of doubly heterozygous females

Table 1 summarizes the number of impaternate progeny obtained from females doubly heterozygous for recessive markers. The impaternate productivity varied somewhat among the genotypes of the mothers, ranging from 0.005 to 0.015 per mother. These rates are in general agreement with those previously obtained with different strains (FUYAMA 1984). The results seem to suggest that most strains of *D. melanogaster* are capable of producing gynogenetic progeny by mating with the *ms(3)K81* males. The rate shown above is probably an underestimate, because the parents were still fecund at the end of the egg-laying period and because their oviposition may have been suppressed due to crowding. Taking these and an estimate of fecundity of this species into consideration, the probability of an egg giving rise to an impaternate adult is likely to fall within the range from 10^{-4} to 10^{-5}. This rate appears to be consistent with those that have been reported for several species of Drosophila in which sporadic parthenogenesis (tychoparthenogenesis) is known to occur: In *D. mercatorum*, CARSON (1967) reported 5×10^{-5} as the average of ten laboratory strains. Almost the same rate, 4×10^{-5}, was found in a natural population of the same species (TEMPLETON, CARSON and SING 1976). STALKER (1954) examined a total of 28 Drosophila species for their parthenogenesis ability; among them, three, *D. parthenogenetica, D. polymorpha* and *D. affinis* produced adult progeny by parthenogenesis at rates varying from 8×10^{-4} to 5×10^{-5}.

Another point worth mentioning is the frequency of the male progeny; about 8% of total parthenogenetic progeny produced in the present experiments were sterile (most likely *XO*) males. This rate seems to be exceptionally high as compared with the previous findings in other parthenogenetic Drosophila: In *D. parthenogenetica*, 1.5% of the progeny of diploid virgin females were male (STALKER 1954). A similar frequency was reported for *D. mercatorum* (CARSON 1962). In *D. ananassae* and *D. pallidosa, XO* males are much less frequent (0.5–0.2%; FUTCH 1973, 1979). STALKER (1954) considered that the

XO male was produced through nondisjunction at the second meiotic division, followed by the fusion of two central polar nuclei. The proportion of the males obtained in the present experiments, however, seems too high to be explained by this mechanism. Perhaps, an *X* chromosome is lost during postmeiotic cleavage division, as suggested for *D. mercatorum* (TEMPLETON 1983).

Triploid females constituted about one-quarter of the parthenogenetic progeny of *D. parthenogenetica* (STALKER 1954), but neither triploid females nor intersexes were found among the impaternates of *D. melanogaster.*

The modes of diploidization

All parthenogenesis so far known in Drosophila is thelytokous (female producing) and automictic; *i.e.* diploid chromosome number is restored after the completion of meiosis. Diploidy is restored either by the fusion of two or more pronuclei (pronuclear fusion) or by the duplication of a pronucleus (gamete duplication). Cytological mechanisms of diploidization are quite variable among species. *D. mangabeirai,* the only one species known to reproduce exclusively by parthenogenesis, restores diploidy by the fusion of two nonsister pronuclei following meiosis II (central fusion; MURDY and CARSON 1959). In a tychoparthenogenetic species, *D. parthenogenetica*, central fusion and the fusion of two sister pronuclei produced at meiosis II (terminal fusion) occur at almost equal frequency. In addition, the fusion including three egg pronuclei is very common in this species, which gives rise to triploid progeny (STALKER 1954). The mechanism of restoration in *D. mercatorum* is very different from those found in the above two species; in >90% of eggs, diploidy is restored after meiosis by gamete duplication (or by the fusion of identical haploid cleavage nuclei), and in the remainder, restoration occurs by terminal fusion (CARSON 1973; TEMPLETON and ROTHMAN 1973). As a result, most of the parthenogenetic progeny of this species are completely homozygous. A mixture of gamete duplication and terminal fusion is also found in *D. ananassae* and *D. pallidosa* (FUTCH 1973).

Genotypes of the impaternates produced by doubly heterozygous females: For the purpose of knowing the cytological mechanisms of diploidization in the gynogenesis of *D. melanogaster,* the genetic constitutions of the impaternate progeny that were produced by the female doubly heterozygous for recessive mutants were examined. When the mother carries two recessive markers in coupling (*a b*/+ +), those impaternates homozygous for either marker and homozygous for the + allele of the other (*a* +/*a* + or + *b*/+ *b*) must be produced by gamete duplication, except in the case of four-strand double crossovers. On the other hand, heterozygotes arise exclusively by the fusion of either central or terminal pronuclei. The other genotypes (*a b*/*a b* and + +/+ +) can be produced by both gamete duplication and fusion (CARSON 1973).

Table 2 lists the genotypes of the impaternate female progeny which were produced by the *cn bw*/+ + mothers and which, themselves, produced sufficient numbers of offspring in the progeny test. Among 59 progeny whose genotypes were determined, 50 (84.7%) were judged to be produced by pronuclear fusion. Two progeny (3.4%) had the genotypes (+ *bw*/+ *bw*) likely to be explained

TABLE 2

Genotypes of impaternates produced by *cn bw*/+ + females

Mode of diploidization	Genotype	No. of impaternates	%
Duplication	*cn* +/*cn* +	0	0.0
	+ *bw*/+ *bw*	2	3.4
Duplication or fusion	+ +/+ +	3	5.1
	cn bw/*cn bw*	4	6.8
Fusion	*cn bw*/+ + or *cn* +/+ *bw*	37	62.7
	+ +/*cn* +	9	15.3
	+ +/+ *bw*	0	0.0
	cn bw/*cn* +	1	1.7
	cn bw/+ *bw*	3	5.1
Total		59	100.0

TABLE 3

Genotypes of impaternates produced by w^a +/+ *m* females

Mode of diploidization	Genotype	No. of impaternates	%
Duplication	+ +/+ +	3	5.5
	w^a *m*/w^a *m*	7	12.9
Duplication or fusion	w^a +/w^a +	6	11.1
	+ *m*/+ *m*	7	13.0
Fusion	w^a +/+ *m* or + +/w^a *m*	16	29.6
	+ +/w^a +	1	1.9
	+ +/+ *m*	5	9.3
	w^a +/w^a *m*	3	5.6
	+ *m*/w^a *m*	6	11.1
Total		54	100.0

by gamete duplication. However, the two marker loci are located so far apart (47 map units) that there is a possibility that these two impaternates resulted from four-strand double crossovers followed by terminal fusion. If this is the case, we would expect many more single crossover types; however, only one possible individual (*cn bw*/*cn* +) was actually found. Therefore, the frequency of terminal fusion, if any, seems to be very low.

The results obtained with experiment 2 (Table 3) differ considerably from those of experiment 1; 18.4% (10 of 54) of impaternate female progeny had the genotypes which are primarily produced by gamete duplication and only 57.4% (31 of 54) were the heterozygotes resulting from pronuclear fusion.

The genetic constitution of 48 female impaternates that were obtained from the mothers whose three major chromosomes were doubly marked are summarized in Table 4. These impaternates were classified in 19 types according to their genetic constitutions. Two individuals belonging to type 1 and type 2

TABLE 4

Genotypes of impaternates produced by + +/*cv f;cn bw*/+ +; *ri e*/+ + females

Type	Genotype of impaternates						No. produced
	cv	*f*	*cn*	*bw*	*ri*	*e*	
Duplication							
1	++	++	*mm*	*mm*	*mm*	++	1
2	*mm*	*mm*	++	*mm*	++	++	1
Fusion							
3	+*m*	+*m*	+*m*	+*m*	+*m*	+*m*	16
4	++	+*m*	+*m*	+*m*	+*m*	+*m*	5
5	+*m*	+*m*	+*m*	++	+*m*	+*m*	3
6	*mm*	+*m*	+*m*	++	+*m*	+*m*	3
7	++	++	+*m*	+*m*	+*m*	+*m*	2
8	+*m*	+*m*	*mm*	+*m*	+*m*	+*m*	2
9	+*m*	+*m*	+*m*	+*m*	+*m*	*mm*	2
10	+*m*	+*m*	+*m*	*mm*	+*m*	+*m*	2
11	+*m*	+*m*	*mm*	*mm*	+*m*	+*m*	2
12	+*m*	*mm*	+*m*	+*m*	+*m*	++	2
13	++	+*m*	+*m*	+*m*	++	++	1
14	++	+*m*	+*m*	*mm*	+*m*	+*m*	1
15	+*m*	+*m*	++	++	+*m*	+*m*	1
16	*mm*	+*m*	+*m*	++	+*m*	++	1
17	*mm*	+*m*	+*m*	++	+*m*	+*m*	1
18	*mm*	+*m*	+*m*	+*m*	+*m*	+*m*	1
19	*mm*	*mm*	+*m*	+*m*	+*m*	+*m*	1
Homozygotes	18	7	7	16	3	8	
Heterozygotes	30	41	41	32	45	40	
% Heterozygotes	63	85	85	67	94	83	

++ and *mm*, respectively, indicate homozygote for wild-type allele and mutant allele; +*m* indicates heterozygote.

had the second or third chromosome constitutions that are characteristic of gamete duplication. Indeed, these individuals were completely homozygous for all six marker loci; thus, there is little doubt that they were produced by gamete duplication. The remaining 46 impaternates were unambiguously determined to be restored by pronuclear fusion, because they were heterozygous for at least three marker loci. A closer examination of the genotypes indicates that central fusion is a predominant mode of diploidization: All but one of 46 fusion-type impaternates were heterozygous for *ri*, which can seldom occur by terminal fusion because of the close linkage of this locus to its centromere. Moreover, the proportion of heterozygotes for each marker locus decreases as the locus becomes more distant from the centromere, but just the reverse is expected by terminal fusion (TEMPLETON 1983). What is difficult to explain by central fusion is the frequent occurrence of homozygotes for the *cn* locus; these constituted 11% (5 of 46) of fusion-type progeny. On the assumption of central fusion, these progeny require a crossing over between *cn* and the centromere. Considering the close proximity of this locus to the centromere

(less than 2.5 map units), the observed frequency of recombinants seems too high. The possibility of terminal fusion is unlikely judging from the genotypes of the other chromosomes. There is, as yet, no satisfactory explanation for this phenomenon other than a possible experimental error. It is known that the centromeric region shows more variation in crossing over than do distal parts of the chromosomes (MATHER 1936). Some subtle factors might have led to an increased crossing over between *cn* and the centromere in these strains.

Frequency of each mode of diploidization: A shortcoming of the experimental procedure described above is that, as shown in the first two experiments, some genotypes may arise from either duplication or fusion. CARSON (1973) estimated the rate of fusion and duplication in the parthenogenetic strains of *D. mercatorum* by assuming an equal number of complementary genotypes being produced. A small number of impaternate progeny obtained in the present experiments, however, discouraged us from employing this method to estimate the relative frequency of each mode of restoration. Instead, a different approach was used.

The method used here is an application of the method originally developed by NACE, RICHARDS and ASHER (1970) for constructing linkage maps in amphibians by the use of artificial gynogenesis. The original model assumed only terminal fusion as the mechanism of diploidization; thereafter, the model was extended by ASHER (1970) and TEMPLETON and ROTHMAN (1973) to include the mixture of duplication and the two kinds of fusion (also see, TEMPLETON 1983).

Let E_1, E_2 and E_3 be the probability of eggs developing by terminal fusion, central fusion and gamete duplication, respectively; $E_1 + E_2 + E_3 = 1$. Then, the probability of a heterozygous female producing a heterozygous parthenogenetic progeny (K) is given as follows:

$$K = E_1 y + E_2 (1 - y/2), \tag{1}$$

where y = probability of recombination between the locus and its centromere (not equal to recombination frequency).

The relationship between y and the map distance in units of 10^{-2} from the centromere (x) is given by a set of parametric equations with a parameter t (uncorrected map distance):

$$\begin{aligned} y &= \frac{2}{3}\frac{(e^{3kt} - 1)(e^{2t} - 1)}{e^{(2+k)t}(e^{2kt} - 1)} \\ x &= \frac{kt(1 - e^{-2t})}{(1 - e^{-2kt})}, \end{aligned} \tag{2}$$

where k = coefficient of coincidence (c.c.).

Since map distance (x) is well established for many loci in *D. melanogaster*, we can estimate the probability of recombination (y) for a respective locus if an appropriate value of c.c. is given. The proportion of heterozygous progeny (K) can be obtained experimentally. Putting these values into equation (1), we have a linear equation relating E_1 and E_2. When female parents are heterozygous for two markers, we can get an estimate for E_1 and E_2 by solving the

TABLE 5

Frequency of each mode of diploidization estimated for the progeny from *cn bw*/+ + females

c.c.[a] assumed		Terminal fusion	Central fusion	Gamete duplication
cn	*bw*			
0.6	1.0	0.026	0.850	0.124
0.2	0.8	0.055	0.849	0.096
0.1	1.0	0.025	0.850	0.124

[a] Coefficient of coincidence; for details see text.

TABLE 6

Frequency of each mode of diploidization estimated for the progeny from *w*[a] +/+ *m* females

c.c. assumed		Terminal fusion	Central fusion	Gamete duplication
w[a]	*m*			
1.0	1.0	0.131	0.493	0.376
1.0	0.8	0.113	0.508	0.379
0.8	0.6	0.122	0.508	0.370

TABLE 7

Frequency of each mode of diploidization estimated for the progeny from + +/*cv f*;*cn bw*/+ +;*ri e*/+ + females

c.c. assumed for *ri*	Terminal fusion	Central fusion	Gamete duplication
0.6	−0.032	0.928	0.104
0.4	−0.027	0.928	0.099
0.2	−0.022	0.928	0.093
0.1	−0.019	0.928	0.090

two linear equations simultaneously. When the number of markers is more than two, the least square estimates can be obtained.

In practice, the map position of each marker locus was adopted from LINDSLEY and GRELL (1968), and that of the centromere was assumed to be 65.0, 55.0 and 48.0 for the *X*, the second and the third chromosomes, respectively. Unfortunately, no *a priori* value for the coefficient of coincidence is available. Therefore, in the case of experiments 1 and 2, the probability of recombination (y) was calculated for various trial values of c.c.; in experiment 3, the maximum value of c.c. (1.0) was assigned to the *cv* locus, and a minimum trial value was assigned to *ri*; then, c.c. values for the other loci were obtained, to a first approximation, on the assumption that c.c. is proportional to the map distance.

The frequencies of terminal fusion, central fusion and gamete duplication thus estimated are shown in Tables 5, 6 and 7. These estimates seem to be in

TABLE 8

Productivity of impaternate progeny by meiotic mutants

Genotype of mother	No. of mothers tested	No. of impaternates produced		Impaternates/ mother
		Female	Male	
y mei-9^a	600	2	1	0.005
y mei-9^a/FM7c	500	4	0	0.008
y; cn mei-S332	600	1	4	0.008
y; cn mei-S332/SM1	600	0	0	0.0

general agreement with those inferred from the genotypes of impaternate progeny: About 85% of impaternates produced by the females heterozygous for *cn bw* restored their diploidy by central fusion, and around 10% restored it by gamete duplication; the ratio of terminal fusion is, if any, very low (Table 5). The proportion of central fusion is even larger in the progeny obtained in experiment 3, and in this case, terminal fusion is probably absent (Table 7). In contrast, the estimates from experiment 2 suggest that more than one-third of the impaternates resulted from gamete duplication and about 10% from terminal fusion (Table 6).

The differences found between experiment 2 and the other two experiments may be explained by the differences in the frequency of recessive lethal genes between the *X* chromosome and the autosomes. However, such differences can hardly be attributed to lethal genes alone unless the frequency of lethals on the autosomes is extremely high. The relative frequency of each mode of diploidization probably varies with strains, suggesting that a different set of genes is involved in the respective mechanism.

Impaternate productivity of meiotic mutants

From the above results, it appears that the restoration of diploidy in the gynogenesis of *D. melanogaster* is predominantly caused by the fusion of two egg pronuclei. However, it should be noted that pronuclear fusion is a genetic equivalent to the suppression of either meiosis I (central fusion) or meiosis II (terminal fusion). Although a regular occurrence of recombinant types among the impaternates produced in the above experiments suggests that meiosis has proceeded normally, one may imagine that some impaternates might have derived from those diploid gametes which were resulted from nondisjunction at meiosis. The very low incidence of impaternates and high frequency of *XO* males seem to support this idea. If such were the case, those meiotic mutants which are known to cause meiotic nondisjunction at high frequencies will lead to an increased production of impaternates.

Two meiotic mutants, *mei-9^a* and *mei-S332*, that are known to greatly increase the frequency of nondisjunction at meiosis I or meiosis II, respectively (BAKER and HALL 1976), were tested for their ability to produce gynogenetic progeny. As shown in Table 8, the females homozygous for either of the mutants did not bring about any appreciable increase in the number of prog-

eny as compared with respective heterozygotes. The results indicate that the occurrence of a diploid egg nucleus is not sufficient to produce diploid parthenogenetic progeny. Perhaps nuclear fusion is a prerequisite to an embryo developing normally.

In the course of these experiments, a culture of *y mei-9/FM7c* produced an exceptionally large number of impaternate progeny. These progeny were omitted from the data shown in Table 8 because later investigations have shown that factors not located on the *X* chromosome were responsible for their occurrence. From these incidental impaternates, a strain that can reproduce gynogenetically was successfully established. Each female of this strain produces approximately 15 impaternate progeny, hence the strain can be maintained without difficulty. Detailed accounts of this strain will be published elsewhere.

General considerations

Since an extensive survey carried out by STALKER (1954) for the capacity of parthenogenesis in Drosophila, the conviction that most species of this genus are preadapted to parthenogenesis has been widely accepted (*e.g.*, TEMPLETON 1983). The present study has shown that *D. melanogaster* is not an exception; meiosis proceeds normally without fertilization and egg pronuclei or cleavage nuclei fuse to restore diploidy, which satisfy the conditions necessary for automictic parthenogenesis (TEMPLETON 1983). Why, then, have we been convinced that the virgin females of *D. melanogaster* will never produce progeny? A plausible answer to this question may be that unfertilized eggs of this species, even if restored to diploidy, do not initiate development unless stimulated by sperm. It should be noted that the stimulation provided by the *ms(3)K81* sperm is, on the other hand, sufficient to bring about haploid embryos (FUYAMA 1984). Parthenogenetic species usually possess means of egg activation that do not depend on sperm entry (WENT 1982), but this is probably not true for obligatory bisexual species such as *D. melanogaster*. When such a bisexual species evolves to a parthenogen, in addition to the capacity for restoration, the acquisition of the mechanism of autonomous egg activation may be an essential step. A lack of autonomous egg activation in *D. melanogaster* is, however, a property extremely convenient for studying the genetic mechanisms of parthenogenesis, for it allows us to conduct conventional genetic experimentation.

The mechanism by which the eggs of *D. melanogaster* can restore diploidy has been shown to be a mixture of pronuclear fusion and gamete duplication. This seems to be the rule rather than the exception so far as the parthenogenesis of Drosophila is concerned (TEMPLETON 1983). Although the number of strains examined in the present study is insufficient to draw a general conclusion, central fusion was indicated to be a predominant mode of restoration in *D. melanogaster*. This is in contrast to the case of *D. mercatorum*, in which more than 90% of parthenogenetic progeny restore diploidy by gamete duplication (CARSON 1973; TEMPLETON and ROTHMAN 1973). Such diversity among species presumably is caused by the differences in the frequency of the genes controlling the respective modes of diploidization. We may expect to obtain, by means of artificial selection or by induction of mutation, those

parthenogenetic strains of *D. melanogaster* each of which is endowed with a particular mechanism of parthenogenesis.

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