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Further Analysis of Hybrid Dysgenesis in *Drosophila melanogaster*

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Abstract

A systematic survey of the sterility of second-generation females produced by backcrossing hybrids of dysgenically interacting strains of *D. melanogaster* is reported. The effects of F_1 rearing temperature and chromosomal constitution on this sterility were explicable in terms of the regulation of a transposable, inserted genetic element called the P factor which is known to be associated with hybrid dysgenesis. No applied radiation or chemical treatment induced dysgenic aberrations where they would not normally be found but, where they were found, EDTA slightly increased and ultraviolet radiation reduced their intensity. The results of previous temperature-shift experiments have implicated different periods during development as having the most effect on female fertility. The present studies show little effect of interstrain variation on this critical period. Critical periods are identified for male sterility and recombination. Transmission ratio distortion has been shown to occur only in one of the reciprocal-cross hybrids (M × P) of parental strains causing dysgenesis. The degree of distortion in such crosses is shown here to vary between M strains crossed to the same P strain. Few data are available for the second (P × M) cross. The data reported here confirm that whatever distortion occurs in this cross is slight.

Introduction

The term 'hybrid dysgenesis' designates an association of germ-line aberrations in the hybrids of certain strains of *Drosophila melanogaster* (Kidwell *et al.* 1977). Two types of dysgenesis have been described. The I–R type is characterized by the embryonic death of eggs laid by female progeny of an R (for reactive) strain female and an I (for inducer) strain male (Bregliano *et al.* 1980). The P–M type is characterized by aberrations, including hybrid sterility, male recombination, transmission ratio distortion and increased mutation rates, in the progeny of an M (for maternal) strain female and a P (for paternal) strain male (Kidwell *et al.* 1977).

The hypothesis that dysgenesis is caused by genetic elements which are inserted in the chromosomes of one of the strains interacting to produce the phenomenon has been suggested by a number of authors (Minamori 1971; Green 1977; Slatko 1978). Indeed, it has recently been shown that P–M dysgenesis is associated with a DNA sequence called the P factor which is found in multiple copies in the chromosomes of P strains but which is absent from the chromosomes of M strains (Bingham *et al.*, personal communication). The nature of this association remains obscure. But features of the breeding properties of dysgenesis suggest affinities with some known types of chromosomally insertable elements such as eukaryotic RNA and DNA tumour viruses (Watson 1976; Stanier *et al.* 1977; Berns and Hauswirth 1979) and the *copia*-like elements of *D. melanogaster* (Potter *et al.* 1979; Ilyin *et al.*

1980; Flavell and Ish-Horowicz 1981). It has been suggested (Engels 1979*a*) that dysgenesis is akin to the zygotic induction of phage λ in *E. coli*. To make this analogy explicit it can be hypothesized that copies of the P factor are maintained in integrated form in P-strain chromosomes by a cytoplasmically circulating repressor produced by the factor itself. Little or no repressor is transmitted through the cytoplasm of the sperm so that there is an initial lack of the substance in M × P hybrids, allowing the dysgenic expression of the P factor. This expression might or might not require excision of the factor from the chromosomes. The factor may have a free cytoplasmic state in which it is capable of dysgenic expression and which is transmissible through the cytoplasm of the egg. There may be a cytoplasmic state of the factor in which it is repressed in the manner of the P1 group of phages in *E. coli* and of phage λ in certain mutants of this bacterium (Stanier *et al.* 1977).

Although a number of studies have been made of the breeding properties of hybrid dysgenesis, knowledge of these properties remains incomplete. In this paper are reported the results of a series of experiments which are aimed at extending the characterization of dysgenic properties. These results will be discussed in relation to extension of the basic induction hypothesis outlined above.

No systematic study has yet been made of the sterility of female progeny of P × M and M × P hybrids in the various possible backcrosses. In this paper, the sterility of progeny of backcrosses of these hybrids to M and P stocks has been studied in relation to the F_1 rearing temperature and to the exact chromosome constitution (established by marked inversions) of the tested females.

Various physical treatments have been shown to have effects on characters of *D. melanogaster* now known to be subject to dysgenic aberrations (Levine 1955; Colwell and Burdick 1959; Suzuki 1965; Sankaranarayanan and Sobels 1976; Shukla and Auerbach 1979). It is of interest to see if there is an interaction of such treatments and hybrid dysgenesis. In particular, attempts to induce the dysgenic expression of the P factor in P and P × M flies are relevant to induction hypotheses because such treatments as ultraviolet irradiation and mitomycin C are known to induce the lytic cycle of phage λ in *E. coli* (Hayes 1968; Stent and Calendar 1978).

In general, high rearing temperatures give higher intensities of dysgenic effects than low rearing temperatures. This property has been used to identify periods during the development of females which may be important in the causation of sterility. Engels and Preston (1979) nominated a period in the first day after hatching. Studies by Angus and Raisbeck (1979), Kidwell and Novy (1979) and Eggleston and Kearsey (1980) suggested that later stages are more important. Interstrain differences, particularly in the P factor complement, might be responsible for the variation in these results. This possibility has been investigated using P strains of diverse geographic origin. The periods of most effect on dysgenic male sterility and recombination are here determined for the first time.

The remaining experiments comprise studies of transmission ratio distortion. Such distortion has not been found in P × M hybrids but the data (Engels 1979*b*) for testing this point are few. Further data have here been collected in an effort to reveal slight transmission distortion in this type of hybrid. Also, the same type of P strain males have been crossed to females from different M strains to ascertain whether there is an interaction of strains which affects the intensity of distortion since it may be that distortion is not simply an intrinsic property of the P factor carrying chromosomes.

Materials and Methods

Stocks and Culturing and Scoring Methods

Genetic symbols in the following list of stocks of *Drosophila melanogaster* are explained in Lindsley and Grell (1968):

M stocks:

al = *al cn bw*; *e* (Sved 1976).

cn,e = *cn*; *e* (Sved 1976).

CS = Canton S, a wild-type stock provided by Dr M. G. Kidwell (Kidwell *et al.* 1977).

H-41 = *In*(1)sc^{s11} sc^{8R+3} w^a *B*; *In*(2LR) SM1, al^2 *Cy* cn^2 sp^2/*In*(2LR) bw^{v1}, ds^{33k} dp^{ov} bw^{v1}; *In*(3LR) Ubx^{130}, Ubx^{130} e^s/*In*(3LR) *C*, *Sb*; spa^{pol}, provided by Dr R. Frankham, School of Biological Sciences, Macquarie University.

P stocks:

H = Harwich, a U.S.A. wild-type stock provided by Dr Kidwell (Kidwell *et al.* 1977).

PW = Para Wirra 85, an Australian wild-type stock provided by Dr D. S. Angus (Colgan and Angus 1978).

Cultures were maintained in 190-ml milk bottles or 2·5 by 10 cm shell vials on standard semolina–treacle medium. All stocks were maintained at 25±2°C unless otherwise indicated. Female fertility was scored as in Sved (1976) except that those females laying less than 10 eggs in 5 days were considered sterile. Female fertility may also be assayed by dissection (Schaefer *et al.* 1979). Here females are considered fertile if, after ageing, they are found to have at least one apparently normal ovariole (in either ovary). Male fertility was tested by examining vials set up with one male and two virgin females for offspring after 13 days at 25°C. Any male not producing progeny by this time was considered sterile. For male recombination and transmission distortion experiments, counts were made of all progeny emerging within 13 days at 25°C.

Experimental Procedures

Second-generation female fertility

All eight possible second-generation backcrosses of hybrids of CS and H parents were made at 29°C using as parents CS, H and hybrid flies reared at 18°C (for one series) or 25°C (for a second series). The fertility of female progeny of the backcrosses was scored. Two series of crosses to CS and H flies were made using hybrids of H and H-41. The genotypes of female progeny in these series were scored and their fertility assessed. In one series, the parents of the second generation were reared at 18°C and in the other they were reared at 25°C.

Physical treatments

EDTA (0·005 M) and magnesium chloride (0·04 M) were added to ordinary culture medium for larval development of the treated flies. Mitomycin C (2 mM) was administered by the procedure of Shukla and Auerbach (1979). Gamma irradiation of adults (30 Gy) was carried out 1–3 days after eclosion. Ultraviolet irradiation of bottle cultures where larvae were beginning pre-pupation was carried out from above, after removal of the bungs, for 16 h using a 15-W General Electric lamp at a distance of 30 cm (approx. 20×10^{-7} J mm^{-2} s^{-1}). The first 2-day batch of flies emerging from these cultures (and controls) were used in the experiments.

Critical periods for dysgenic effects

Parental crosses were set up in vials at 29°C (18°C for controls) after mating for 3 days at 25°C. Oviposition was allowed for 2 h. At various times thereafter, vials were shifted to 18°C for completion of development. The fertility of hybrid females was tested. Hybrid *al*×H males were backcrossed to *al* females to allow scoring of sterility, recombination and transmission distortion.

Transmission ratio distortion

Females from the *cn,e* and H-41 stocks were crossed to H males from the one set of culture bottles at 18 and 29°C. The male hybrids were crossed at 25°C to the marker stock if the markers

were recessive (*cn,e*) and to a wild-type female if the markers were dominant (H-41). Transmission ratio distortion was determined from counts of the genotypes of progeny of these crosses. A second design was used in crosses of H females to *al* males. The same parents were used to set up crosses at 18°C (7 days of oviposition) and, later, at 25 or 29°C (5 days of oviposition). The hybrid males were backcrossed to *al* females at 25°C and their progeny were scored.

Table 1. Female sterility in second-generation progeny of parental crosses of M and P strains

Female type[A]	CS and H stocks; F_1 reared at 25°C		CS and H stocks; F_1 reared at 18°C		H-41 and H stocks; F_1 reared at 25°C		H-41 and H stocks; F_1 reared at 18°C	
	Scored	% sterile	Scored	% sterile	Scored	% sterile	Scored	% sterile
M × (M × P)	76	33	24	42	31	29	70	57
M × (P × M)	62	95	25	72	94	91	81	89
P × (M × P)	61	7	25	0	67	4	70	1
P × (P × M)	62	0	25	0	68	0	79	3
(M × P) × M	30	47	31	84	—[B]		87	89
(P × M) × M	59	0	19	32	156	30	80	30
(M × P) × P	62	100	18	95	—[B]		91	99
(P × M) × P	61	2	23	26	93	79	100	58

[A] M strains are CS and H-41 whilst H is the P strain. Females are written first in all crosses. The brackets enclose crosses producing the F_1 hybrids.
[B] (H-41 × H) females reared at 25°C proved to be sterile.

Results

Second-generation Female Sterility

The collected results of tests of female sterility are given in Table 1. Seven points emerge from an examination of these data:

1. The frequency of sterility is higher, in general, in the progeny of the crosses of H-41 and H flies than in the progeny of CS and H flies.
2. Crosses of both types of hybrid males to P females produce fertile progeny in all crosses.
3. In all series crosses of P × M males to M females produce more sterility in progeny than crosses of M × P males.
4. There is, generally, a higher proportion of sterile progeny when the F_1 of crosses of CS and H flies are reared at the lower temperature. The absence of data (due to hybrid sterility) for H-41 × H females reared at 25°C obscures this trend, if it exists, in crosses of H-41 and H.
5. M × P female hybrids produce a much higher proportion of sterile daughters than do P × M females.
6. In the two series using H-41 flies, (P × M) × P females are more often sterile than (P × M) × M flies.
7. The rate of sterility in (P × M) × M and (P × M) × P females is higher than in the P × M females (Kidwell *et al.* 1977; Colgan and Sved, unpublished data) in all crosses except those of the (H × CS) × CS and (H × CS) × H females reared at 25°C. The degree of sterility of these types of second-generation females is directly related to the number of Harwich chromosomes they possess. Amongst the (H × H-41) × CS females, 0/17 of the series reared at 25°C (and 4/15 of the series reared at 18°C) without Harwich chromosomes are sterile, as are 11/63 (15/25) with one Harwich chromosome,

31/60 (27/41) with two and 4/16 (4/14) with three. In the series of (H × H-41) × H females reared at 25°C, 3/6 (5/9) with three Harwich chromosomes are sterile, as are 21/28 (17/36) with four, 33/40 (27/35) with five and 17/19 (10/19) with six.

Physical Treatments

The effects of all five physical treatments on male recombination and female sterility were tested on treated and untreated series comprising M × M, P × M and M × P crosses.* The interaction of dysgenesis and mitomycin C on the induction of translocations was investigated by the methods of Shukla and Auerbach (1979). Only EDTA and ultraviolet treatments had any effect. EDTA marginally increased recombination: 35 recombinants (occurring in at least 25 separate events) were found in 1983 progeny of treated *al* × H males and 19 (>16 events) in 2556 progeny of untreated males. This chemical exacerbates female sterility: 97 of 115 treated *al* × H females were sterile but only 48 of 113 untreated females were sterile. EDTA had no effect on fertility or recombination in reciprocal H × *al* hybrids. The effects of ultraviolet irradiation are variable but are usually meliorative. There were 24 recombinants (>12 events) in 674 progeny of untreated *al* × H males and 21 (>7 events) in 2145 progeny of treated males. This data set is collected from two experiments. In the first there was no effect of irradiation. In the second, 2 recombinants (2 events) were found in 1523 progeny of treated males and 11 (>7 events) amongst 371 controls. In tests of female sterility, 62 of 209 untreated females were fertile whilst 102 of 244 treated females were fertile. Ultraviolet irradiation had no significant effect on fertility or recombination in the reciprocal H × *al* hybrids.

Critical Periods for Dysgenic Effects

The results of sterility and male recombination tests performed during this experiment are given in Table 2. There was, as expected, a general increase in the degree of female sterility as more of the rearing time was spent at 29°C. Only 1 of 128 females spending 6 h or less at 29°C was sterile. All females from PW crosses spending more than 2 days at 29°C were sterile. Most females from H crosses reared at 29°C for more than 2 days were sterile but some were fertile even though they were wholly reared at the high temperature. The changes between the extremes of sterility occurred between 6 h and 2 days for CS × PW females and between 6 h and 3 days for the other hybrids. There was little evidence that crosses differed in this period since the earlier attainment of full sterility in CS × PW flies may be due to chance effects in the small sample available for this cross in the 1–2-day period. Nevertheless, the variation in previous reports of the critical period for female sterility may yet be due to interstrain variation, if a more intense dysgenic interaction than we have found can be cited in explanation of the shorter and earlier transition period found by Engels and Preston (1979).

The data for *al* × H male sterility followed the pattern of an increase in sterility with an increase in rearing time at 29°C. The change between initial and final levels

* This data has been lodged as an Accessory Publication with the Editor-in-Chief, Editorial and Publications Service, CSIRO, 314 Albert Street, East Melbourne, Vic. 3002. Copies may be obtained on request.

Table 2. Summary of sterility and male recombination in temperature-shift experiments

Days at 29°C[A]	CS × PW females		*al* × PW females		CS × H females		*al* × H females		*al* × H males sterility		*al* × H males recombination	
	Scored	% sterile	Scored	% sterile	Scored	% sterile	Scored	% sterile	Scored	% sterile	Counted	% recom.
0	14	7	15	0	14	0	34	0	12	9	858	0·8
¼	4	0	5	0	11	0	31	0	10	0	0	
½	24	46	10	90	19	32	5	20	2	100	0	
1	5	20	4	75	25	16	97	38	23	0	1891	0·6
2	4	100	22	62	32	78	111	61	39	0	2337	0·5
3	12	100	9	100	14	93	54	93	138	5	7294	1·8
4	11	100	4	100	37	97	52	100	75	17	3433	2·9
5	25	100	10	100	28	89	61	95	23	70	317	4·1
6	11	100			26	100	54	100	31	77	182	4·4
>6	18	100	20	100	20	90	58	97	33	67	357	1·4

[A] Flies were reared at 29°C for this period of time before being shifted to 18°C.

Table 3. Numbers of second-generation flies of various genotypes in transmission ratio distortion experiments

F_1 males	F_1 rearing temp. (°C)	II—wild-type[A]				II—marked			
		III—wild-type		III—marked		III—wild-type		III—marked	
		Males	Females	Males	Females	Males	Females	Males	Females
cn,e × H	18	328	356	345	342	327	367	290	320
	29	292	289	327	379	288	241	292	331
H × *al*	18	34	34	29	35	15	11	11	8
	25	101	94	105	56	28	41	28	25
H × *al*	18[B]	85	75	75	97	63	72	68	71
	29	600	598	650	662	519	543	470	482

[A] Column headings indicate whether flies in the subdivision are wild-type or marked for the chromosome.
[B] With two recombinants.

of sterility occurred in flies raised at 29°C for between 3 and 5 days, with the change between days 4 and 5 being particularly marked. In elapsed time, the period causing most change in fertility differed between females and males.

The level of male recombination tended to increase with increases in rearing time at 29°C although there was a decline in the category spending most time at this temperature. For the first 2 days recombination remained near the level found in controls raised at 18°C. There was a marked increase during each of the next 2 days. The onset of these increases was earlier than the large increases in male sterility.

Transmission Ratio Distortion Experiments

The results of transmission ratio distortion experiments are shown in Table 3. Deviations from 1 : 1 transmission ratios may be due to viability effects as well as to dysgenically induced distortions. Increases in rearing temperature increase the intensity of most dysgenic effects (Kidwell *et al.* 1977; Engels 1979*b*). Dysgenically induced transmission distortion is in favour of M chromosomes (Kidwell *et al.* 1977), so it will be assumed here that differences in ratios are evidence of dysgenic distortion if there are fewer P chromosomes at the higher temperature. The first two lines of the table detail distortion in *cn,e* × H males. Contingency χ^2 analysis of these data showed that there was an increase in the proportion of ebony flies at the higher temperature ($P \ll 0{\cdot}001$) whilst there was no significant difference in the proportion of second or sex chromosome types. Hence transmission distortion may be restricted to certain pairs of chromosomes in dysgenic hybrids.

The effect of transmission distortion on different members of homologous pairs was investigated in a second experiment. All four male (major) chromosomal types from the H-41 × H cross were examined. The scoring of bw^{v1} in heterozygous Bar females is difficult so no classification of this character was attempted in this class. For analysis, data of all genotypic classes containing a specified marker chromosome were pooled and the totals subjected to χ^2 testing. There were no significant differences in the proportions of the *Cy*, *Sb* or *Ubx* chromosomes at the two temperatures. The result for the bw^{v1} chromosome in males was near significance ($0{\cdot}06 > P > 0{\cdot}05$) and there were fewer males at 29°C ($P < 0{\cdot}05$). There may be an interaction of the bw^{v1} and Harwich chromosomes in transmission ratio distortion. The results of subsequent investigations of this point were, however, so heterogeneous that no conclusions could be reached.

Transmission ratio distortion was investigated in P × M hybrid males. No significant changes occur in the frequency of any of the three major Harwich chromosomes in the data given in Table 3 for the 29°C series. A repeat of this experiment comparing distortion at 18 and 29°C was hampered by the low sample sizes at 29°C (2246 flies emerged from the 18°C cultures but only 146 from the 29°C cultures) and is not detailed in Table 3. Nevertheless, there was no appreciable change in frequency in any pair of chromosomes. Unfortunately, the data could not be pooled because of heterogeneity in the 18°C results of the two repetitions. The comparison of hybrids reared at 18 and 25°C showed no significant differences.

Discussion

The results of tests of the fertility of second-generation females illustrate and extend the basic induction hypothesis of P factor regulation. Transmission of the

factor through the egg cytoplasm is suggested by the findings that there is more sterility among progeny of M × P females (with free cytoplasmic P factor under the hypothesis) than among those of P × M females which are not expected, under the hypothesis, to carry free cytoplasmic P factor. The magnitude of this effect is too large to be explained by chromosomal contamination which rarely causes total sterility in the P–M system of dysgenesis (Sved and Colgan, unpublished data). Further, most (H-41 × H) × CS females reared at 18°C without Harwich chromosomes are sterile. Crosses of hybrid males to P females produce fertile progeny so that whatever cytoplasmic P factor is transmitted through the sperm is usually inactivated in the F_2. Again, Sved (1976) and Engels (1979*a*) found that less than 5% of M × (M × P) females without P chromosomes were sterile. The higher rate of sterility in the progeny of P × M males might be due to the presence of the Harwich X chromosome in these males or to individual variations in dysgenic responses. Such variations might also explain some of the effects of F_1 rearing temperature on F_2 sterility. Some P strain flies will carry more copies of the P factor or more easily expressed copies so that higher proportions of their M × P progeny will be sterile. M × P hybrids reared at low temperatures and P × M hybrids are not affected by sterility so that all can contribute to the second generation. Progeny of these hybrids would be more often sterile because the rearing conditions of the F_2 allow the expression of their dysgenic potential.

The fact that some progeny of P × M females are sterile is interesting in view of the absence of sterility amongst these females themselves. The relation between the frequency of this sterility and the number of Harwich chromosomes suggests that this expression of dysgenesis is due to events during F_2 development rather than to the transmission of free P factor through the cytoplasm of the eggs of P × M females. Nevertheless, some (H × H-41) × CS females reared at 18°C without Harwich chromosomes (other than the small fourth chromosome) were sterile. Perhaps some free P factor is formed during P × M development and can be transmitted through the egg. It might then be enquired as to why free P factor is not produced in pure-strain P females in such a quantity as to affect their progeny since it has, for instance, been shown that mutation rates are higher in P × M hybrids than in pure-strain P flies (Simmons *et al.* 1980). It can be speculated that the kinetics of repressor production and P factor regulation are upset in P × M hybrids. In particular, the eggs of such females may be deficient in repressor. If this substance is not produced until later in the flies development (as suggested by the very fact of the occurrence of dysgenesis in M × P flies) then this deficiency would be exacerbated by the presence of more Harwich chromosomes (with their P factors) in the F_2.

The induction of the lytic cycle of phage λ in *E. coli* by such agents as mitomycin C and ultraviolet irradiation is mediated by the host *recA*$^+$ gene product (Witkin 1976). Despite the restricted nature of this phenomenon, it is perhaps surprising, under the induction hypothesis, that no physical treatment here reported or the tetracycline treatment of Colgan and Angus (1978) has proved capable of increasing dysgenic aberrations in the P × M cross. This finding and the curious amelioration of dysgenic effects by ultraviolet irradiation of M × P flies should be explained in complete descriptions of the regulation of the P factor.

There is a difference in the 'critical periods' for the increases of hybrid sterility in the two sexes in elapsed developmental time. It might be argued that dysgenesis affects different stages in the development of the sexes. For instance, a significant

proportion of females spending 12 h or less at 29°C is sterile. Few males which spend less than 3 days at 29°C are sterile. The temperature-shift experiments do not, however, prove this contention. There are more cells in the male germ line than in the female so that the chance of a male germ-line cell escaping dysgenic effects is higher than the chance of a female germ-line cell, other factors being equal, simply because dysgenic expression is a stochastic, and not a deterministic, process. Males must be reared for a longer period at the higher temperatures which increase dysgenic expression in order that they are rendered sterile. The true critical period for the expression of dysgenic sterility remains obscure although the present experiments do show that the end of this period is not earlier than 2 days in females or 4 days in males because rearing temperature has an effect on the level of sterility even after flies have spent these times at 29°C.

Transmission ratio distortion might be explained in terms of meiotic chromosome breakage. In the present experiments using *cn,e* × H males, distortion is found for the third chromosome pair but not for the second. At the time of these experiments, female sterility in the Harwich stock was mainly associated with the third chromosome (Sved and Colgan, unpublished data). Chromosome breakage might, then, be restricted to those chromosomes carrying the P factor and might not affect all P strain chromosomes carried by an individual. No distortion was found for the third chromosome in the experiments using H-41 × H males although these were set up at the same time and using males from the same culture bottles as those involving *cn,e* × H males. The lack of distortion hints that there is a possibility of an interaction between the M and P stocks in this regard so that the degree of transmission distortion in a cross is not simply an intrinsic property of the P chromosomes.

Many aspects of the regulation of the P factor await explanation. Firstly, the temperature dependence of dysgenic effects must be explained by a quantitative relation of the kinetics of interaction of the P factor and its putative repressor. An understanding of these kinetics might also allow an insight into the cause of the findings that mutation rates are higher in P × M hybrids than in P strain flies (Simmons *et al.* 1980) and that the progeny of P × M females are more often sterile than those of P flies. Secondly, there is a surprising lack of dysgenic effects which can be shown to operate exclusively in the somatic tissues of the F_1 hybrid. Dysgenesis does have such somatic effects as increasing rates of development and decreasing mating ability, but these are general and are probably secondary manifestations of the gonadal atrophy associated with P–M dysgenesis (Colgan, unpublished data). As yet, the limitations of dysgenic effects to the germ line are wholly unexplained. Thirdly, the physical basis of dysgenic aberrations requires elucidation. Sterility may be due to the chromosome breakage reported by Henderson *et al.* (1978), a process analogous to bacterial cell lysis or to a defect to date unspecified. Mutation and transmission distortion might be explained, respectively, as the results of insertions of (fragments of) the P factor and chromosome breakage. But the physical basis of dysgenically induced recombination is very puzzling. Sved (1978) has shown that such recombination produces chromosomes that, being viable as homozygotes, cannot carry the large deletions which random breakage and reunion of chromosomes would be expected to generate. Further, if dysgenesis is caused by a transposable element the possibility might be considered that it can be horizontally transmitted by feeding or injection. Such transmission of hybrid

sterility may well occur (Colgan and Angus 1978; Angus and Raisbeck 1979; Colgan, unpublished data). But male recombination is not induced in the progeny of flies treated by such methods (Sved *et al.* 1978).

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