Heredity (1982), **49** (2), 243–246 0018-067X/82/04780243$02.00

NOTES AND COMMENTS

SOMATIC EFFECTS OF HYBRID DYSGENESIS IN *DROSOPHILA MELANOGASTER*

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Received 28.xii.81

ALL of the aberrations which have been associated with the phenomenon of hybrid dysgenesis in *Drosophila melanogaster* are discovered in the phenotypic composition of the progeny of hybrids rather than in somatic effects on the hybrids themselves (Thompson, Woodruff and Schaefer, 1978). The apparent restriction of dysgenic effects to the germ line may be a feature of the functional biology of dysgenesis or it may be that somatic dysgenic effects have hitherto been overlooked in spite of their occurrence; recombination being the only effect which has been actively investigated in somatic tissues (Thompson *et al.*, 1978). This note reports experiments studying F1 phenotypic abnormalities, which might be due to dominant mutation, male mating competitiveness and developmental rate in dysgenic and non-dysgenic hybrids.

Strains which interact to produce dysgenesis are labelled as M (for maternal) and P (for paternal) according to whether they contribute as males or females to crosses producing dysgenesis. The M strains used were Canton S (CS), a strain heterozygous for SM1 and In(1) bw^{v1} (*Cy*) and marker strains carrying *al cn bw*; *e* (*al*) or *B*. The P strains were Harwich (H, Kidwell *et al.*, 1977) and Para Wirra (PW, Colgan and Angus, 1978). Females are written first in all crosses. Stocks were maintained at 25°C on standard medium unless otherwise indicated.

Somatic mutation was scored by inspection of the morphology of both wings of flies from mass cultures in attempts to detect phenocopies of *Curly* with curved wings. Only those individuals with fully extended wings were scored. Pure crosses of M strains gave 4293 progeny of which three had one or both wings curved. There were three flies with curved wings in 7819 hybrid progeny of M strains. None of 6594 P×M hybrids examined had a curved wing. Amongst 18577 M×P hybrids, five had curved wings. All aberrant flies were crossed to M flies but all were sterile or produced wild-type progeny. These results show that there is no general enhancement of somatic mutation by hybrid dysgenesis. But mutation in the germ line of dysgenic hybrids is concentrated at "hot spots" (Berg *et al.*, 1980). Somatic mutation might be similarly concentrated and, by chance, the "hot spots" might be absent from loci (*Cy*, *Coi*, *Cd*, *Cu*, *U*, *Tu*, *Tul*, *Cu*-3, etc.) where mutation might have been detected in this experiment.

The mating success of hybrid males was scored by direct observation of female-choice experiments in vials containing three pairs of males of the two hybrid types. One CS female was introduced to a mating vial which was observed for one hour if mating did not occur. One hour later, a second

TABLE 1

The numbers of matings by various types of hybrid males

Rearing Temp.	Male type	M×P clipped	P×M clipped	Total
18°C	CS×H	56	41	97
	H×CS	50	34	84
25°C	CS×H	36	34	70
	H×CS	44	58	102
29°C	CS×H	69	103	172
	H×CS	205	152	357
29°C	CS×PW	13	11	24
	PW×CS	33	22	55

female was introduced and the observation was repeated. Up to three introductions were made to each vial. Females used in the 25°C series were aged 2–5 days and those used in the 18°C and 29°C series 7–12 days. Males were identified by wing clipping. All flies were allowed one day to recover from clipping and etherisation. The results of the experiment are summarised in table 1. In the 18°C series there was no apparent difference in overall mating competitiveness ($\chi^2, P > 0{\cdot}25$) and wing clipping did not seem to affect success ($\chi^2, P > 0{\cdot}75$), although H×CS males may be more successful at later introductions of females (χ^2, $0{\cdot}25 > P > 0{\cdot}1$). In the 25°C series, there was a significantly higher proportion of matings by H×CS males ($P < 0{\cdot}025$). Wing clipping ($P > 0{\cdot}25$) and female introduction ($P > 0{\cdot}5$) did not affect mating success. In the 29°C series there was, overall, a highly significant excess of matings by H×CS males ($P < 0{\cdot}001$). There was an association of wing clipping and mating success ($P < 0{\cdot}001$) which can be interpreted to mean that clipping reduces the advantage of H×CS males when these compete with unclipped CS×H males and exacerbates the disadvantage of marked CS×H males. There was no association of mating success with female introduction. Mating success at the various rearing temperatures is not directly comparable since the 25°C series was performed separately. Nevertheless there is a tendency to an increase in H×CS success with an increase in rearing temperature. This increase is highly significant ($P < 0{\cdot}001$) between the 18°C and 29°C-reared males (which in the experimental design were the progeny of the same parents). The increase is similar to the increase in intensity of germ line dysgenic effects with rearing temperatures (Kidwell *et al.*, 1977) and may be interpreted as evidence that the reduced mating competitiveness of CS×H males is due to hybrid dysgenesis. The success of PW×CS males in competition with CS×PW males (table 1) may indicate that a reduction in mating competitiveness usually accrues to dysgenic hybrids.

The effect of dysgenesis on developmental rate was examined by scoring the time of emergence of progeny (of four or five pre-mated females) produced in a one-day period of oviposition. Only one cross (e.g., CS×H or CS×CS) was set up in each experimental culture. In one experiment, CS×H and H×CS crosses were set up at 18°C. One week later the same parents were used in a second round of oviposition at 29°C. The results are summarised in table 2. The hybrid males do not differ in emergence patterns when reared at 18°C. But a Mann–Whitney one-tail test of 18°C-reared females is significant ($P \simeq 0{\cdot}015$) implying earlier emergence of CS×H females. Both male and female CS×H hybrids emerged earlier than H×CS

TABLE 2

The emergence of CS × H and H × CS hybrids by specified times after commencement of experiments

		18°C Reared					29°C Reared				
Hybrid	Sex	378*	381	384	402	Total†	185	188	191	209	Total
CS × H	♂	5	14	10	185	352	29	23	28	128	407
	♀	56	85	32	176	407	78	38	36	110	474
H × CS	♂	12	34	26	259	567	16	26	46	235	656
	♀	89	113	38	268	645	26	42	58	235	728

* Hours after commencement of experiments.
† Includes flies emerging after last specified time.

hybrids when reared at 29°C (χ^2, $P < 0{\cdot}005$ for males and $P < 0{\cdot}001$ for females). The disparity in emergence times of the hybrids seems to be more marked at the higher rearing temperature. The point is difficult to test in general but can be illustrated amongst the earliest emerging flies. More CS × H males have eclosed at 29°C when 8 per cent of H × CS flies have emerged than have eclosed at 18°C when 13 per cent of H × CS flies have done so (contingency χ^2 on CS × H data, $P < 0{\cdot}05$). More CS × H females reared at 29°C have emerged by the time 10 per cent of H × CS females have eclosed than have emerged at 18°C when 14 per cent of H × CS females have done so (CS × H data, $P < 0{\cdot}001$). So a parallel with the temperature effects of germ line dysgenesis suggests that developmental rate may also be subject to dysgenic alteration. In each of two confirmatory experiments both male and female CS × H hybrids developed significantly more rapidly at 29°C than H × CS individuals of the same sex. This was also true for *Cy* × H hybrids in comparison to H × *Cy* hybrids and for comparisons of the progeny of H and CS males crossed to CS or *B* females, although the difference between emergence patterns of *B* × H and *B* × CS males was not quite significant ($0{\cdot}1 > P > 0{\cdot}05$). Further both sexes amongst *Cy* × PW hybrids emerged earlier than their counterparts in PW × *Cy* hybrids. However, both sexes of PW × CS hybrids developed more rapidly than CS × PW hybrids. Thus, whilst the results for all crosses involving Harwich are in accordance with the hypothesis that dysgenesis increases the rate of development of affected flies, the results for crosses using Para Wirra show that such an increase is not a necessary concomitant of dysgenesis.

In summary, although there is no evidence from the present experiments that dysgenesis increases somatic mutation, both male mating competitiveness and the developmental rate of hybrids may be affected by dysgenesis. These latter two properties are general aspects of the phenotype and need not, necessarily, be direct effects of dysgenesis on the soma. Both could be secondary effects of the gonadal atrophy characteristic of the syndrome (Angus and Raisbeck, 1979; Schaefer *et al.*, 1979).

Acknowledgements.—I wish to thank Audrey Graham, Maree MacDonald and John Sved for assistance. The research was supported by the Australian Research Grants Committee and the Australian Institute of Nuclear Science.

References

ANGUS, D. S., AND RAISBECK, J. A. 1979. A transmissible factor involved in hybrid sterility in *Drosophila melanogaster*. *Genetica*, *50*, 81–87.

BERG, R., ENGELS, W. R., AND KREBER, R. A. 1980. Site specific X-chromosome rearrangements from hybrid dysgenesis in *Drosophila melanogaster*. *Science*, *207*, 606–611.

COLGAN, D. J., AND ANGUS, D. S. 1978. Bisexual hybrid sterility in *Drosophila melanogaster*. *Genetics*, *89*, 5–14.

KIDWELL, M. G., KIDWELL, J. F., AND SVED, J. A. 1977. Hybrid dysgenesis in *Drosophila melanogaster*: a syndrome of aberrant traits including mutations, sterility and male recombination. *Genetics*, *86*, 813–833.

SCHAEFER, R. E., KIDWELL, M. G., AND FAUSTO-STERLING, A. 1979. Hybrid dysgenesis in *Drosophila melanogaster*: morphological and cytological studies of ovarian dysgenesis. *Genetics*, *92*, 1141–1152.

THOMPSON, J. N. JR., WOODRUFF, R. C., AND SCHAEFER, G. B. 1978. An assay of somatic recombination in male recombination lines of *Drosophila melanogaster*. *Genetica*, *49*, 77–80.