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NOTES AND COMMENTS

THE *I-R* SYSTEM OF HYBRID DYSGENESIS IN *DROSOPHILA MELANOGASTER*: INFLUENCE ON *SF* FEMALES STERILITY OF THEIR INDUCER AND REACTIVE PATERNAL CHROMOSOMES

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SUMMARY

A specific kind of sterile F_1 female, denoted *SF*, arises when females from strains known as reactive are crossed with males from the complementary class of strains (inducer). It has been shown that this sterility results from the interaction between the maternal reactive cytoplasm and any one of the paternal inducer chromosomes. This interaction yields other dysgenic traits including non-disjunction and mutations.

In this note, the abilities of paternal gametes containing various combinations of inducer and reactive chromosomes to give more or less sterile *SF* females when fertilising standard reactive oocytes were compared. Although they did not cause *SF* sterility, reactive chromosomes, when present in sperm containing at least one inducer chromosome, were found to influence the intensity of sterility: variations of *SF* sterility were observed between *SF* females which differed only by one paternally inherited reactive chromosome.

Reactive chromosomes are known to control the cytoplasmic state of reactive females. The present results suggest that this chromosomal control also takes place in *SF* females.

1. INTRODUCTION

A SPECIFIC kind of sterile female, denoted *SF*, arises when females from *Drosophila melanogaster* strains known as reactive are crossed with males from the complementary class of strains (inducer). The genetics of this inducer-reactive interaction, which causes several other dysgenic traits in hybrids, was recently reviewed by Bregliano *et al.* (1980).

The sterility results from diminished egg hatchability and is characterised by physiological features such as reduction of sterility with ageing of the females. Even when assessed on a sample of the first-laid eggs, the fertility of *SF* females varies within broad limits, according to the choice of both reactive and inducer parental strains (Bucheton *et al.*, 1976). Among reactive as well as inducer strains there is considerable variation of efficiency in producing *SF* females with more or less reduced fertility. The efficiency of a reactive female is mainly transmitted to its daughters by the maternal cytoplasm.

The sterility of an *SF* female is the outcome of an interaction between this reactive cytoplasm and a paternal chromosome-linked factor, termed the *I* factor. When reactive females are crossed with heterozygous males bearing chromosomes from both inducer and reactive origins, *SF* sterility is only observed among the daughters which have inherited at least one inducer originating chromosome. Such a chromosome, able to induce the *SF*

sterility, is considered as carrying the *I* factor and is called an inducer chromosome (i^+). Any one of the three major chromosomes and even the small fourth may be an i^+ chromosome. The other chromosomes of inducer origin which, like reactive chromosomes, are not able to give rise to *SF* females when introduced by a paternal gamete into a reactive oocyte are called non-inducer chromosomes (i^0). They may co-exist in inducer strains with homologous i^+ chromosomes.

Assuming additivity of individual i^+ chromosomes efficiencies, the variations in inducer efficiency observed between inducer strains (Bucheton *et al.*, 1976) might simply be explained by variations of the relative proportions of i^+ and i^0 chromosomes in these strains: the more i^+ chromosomes a paternal gamete contained, the stronger would be its inducer efficiency. However, fertility was found not to be more reduced by sperm bearing two i^+ chromosomes than by that bearing only one (Pélisson, 1975). A similar result was reported by Kearsey *et al.* (1977), in a study which most probably dealt with the same kind of sterility. On the contrary, Engels (1979) recently showed that chromosomes of the *P* strains cause hybrid sterility nearly independently of each other. Although both *I-R* and *P-M* hybrid dysgenesis systems have been shown to be causally independent (Kidwell, 1979), the known genetical characteristics of the *P-M* system (Engels, 1979) have closely resembled those of the *I-R* system.

In this note, results are presented which show the non-additive effect of individual i^+ chromosome efficiencies. Additional results are provided which might explain and minimise the discrepancy with the *P-M* system: the inducer efficiency of a paternal gamete is not only controlled by its i^+ but also by its reactive chromosomes.

2. Materials and methods

All flies were bred in vials, at 20°C. All crosses were mass crosses involving about 10 flies of each sex. Chromosomes originating from an inducer or a reactive strain are respectively indicated by the indices -(*i*) and -(*r*).

(i) *Reactive marked stocks*

Cy/Pm; *H/Sb* comes from the Valencia University (Spain) and is listed in *Dros. Inf. Serv.*, *48*, 12.

The three following reactive strains derived by selection from our own original reactive strains: LH_{23} from the *LH* strain (genotype: *M-5/M-5*; *Cy/Pm*; *Sb/H*); seF_8 and e_{st28} from *se* and *e* strains, respectively.

(ii) *Inducer wild-type stocks*

L.M. is a laboratory stock kindly supplied by Dr M. Tichomirova from the University of Leningrad (U.S.S.R.).

Beaune, Hyères and Ménétréol originate from a small number of flies caught in different parts of France in 1972, 1969 and 1972 respectively.

(iii) *Fertility measurements*

Eggs from sets of about 20 2-day-old non-virgin females were collected on food stained with some carbon black during 48 hours. Thirty to forty

hours later, the percentage of hatched eggs was determined on a sample of about 400 eggs.

3. Results and discussion

(i) *Inducer efficiency of the various gametes produced by* i/r *heterozygous males*

M-5-(r); Cy-(r)/+-(i); Sb-(r)/+-(i) heterozygous males were obtained from a cross between males of the Beaune inducer strain and females of the LH_{23} reactive strain. They were mated with females of the e_{st28} reactive strain. Owing to the dominant morphological markers, four phenotypic classes of daughters, corresponding to the various paternal gametes, could be selected and tested for hatchability as described in Materials and Methods. The experiments as well as the control, which started with reactive seF_8 instead of inducer Beaune males, were repeated four times. In table 1 are

Table 1

Hatching percentages of eggs laid by sets of females according to their paternal genotype. A, B, C and D are the four phenotypic classes of females carrying the indicated paternal major autosomes. The paternal X *chromosome, not represented, is in each case the* M-5-(r) *chromosome from the* LH_{23} *strain.* Cy-(r) *and* Sb-(r) *are respectively the chromosomes* 2 *and* 3 *from the* LH_{23} *strain. The* +-(i) *and* +-(r) *autosomes come respectively from the Beaune and* seF_8 *strains. Paternal chromosomes* 4 *are not represented; they are all of reactive origin except for statistically half of A, B, C and D females which have inherited a* +-(i) *Beaune chromosome* 4.

Expt. no.	1	2	3	4	Mean ± S.E.
A: *Cy-(r); Sb-(r)*	78	84	75	74	77·6 ± 2·2
B: *Cy-(r); +-(i)*	29	26	37	34	31·7 ± 2·5
C: *+-(i); Sb-(r)*	11	13	30	17	17·7 ± 4·3
D: *+-(i); +-(i)*	33	40	34	43	37·3 ± 2·2
Control no.	1	2	3	4	Mean ± S.E.
cA: *Cy-(r); Sb-(r)*	79	78	76	77	77·6 ± 0·5
cB: *Cy-(r); se-(r)*	77	78	87	77	79·5 ± 2·3
cC: *+-(r); Sb-(r)*	86	85	89	84	85·8 ± 1·0
cD: *+-(r); se-(r)*	91	83	95	83	88·0 ± 2·8

given the hatchabilities of females which have inherited the various combinations of Beaune major autosomes.

Females of class A, statistically half of which carry the chromosome *4* of Beaune, are normally fertile, taking into account the fertility decrease caused by dominant markers in control cA females; Beaune chromosomes *4* are therefore non-inducer. On the contrary, B and C females are significantly less fertile than control cB and cC females; the third and second Beaune chromosomes they have respectively inherited may therefore be considered as inducer chromosomes (i^+).

Taking into account the fertility decrease caused by the *Cy* marker, B and D females, although differing in the number of i^+ chromosomes, have roughly similar fertilities. Moreover, C females which have only inherited i^+ chromosome *2*, are obviously less fertile than D females with both i^+ autosomes.

Similar observations can be made concerning three other experiments (Pélisson, 1975) involving chromosomes *2* and *3* from the Ménétréol, Hyères and L.M. strains. In no case has the D paternal gamete stronger inducer efficiency than those of B and C gametes. More surprising is the fact that, as in the case of C and D classes of table 1, gametes carrying a single i^+

chromosome often have stronger inducer efficiency than that of the gametes to which the other i^+ chromosome has been added.

These curious results might be explained by negative interactions between the efficiencies of i^+ heterologous chromosomes. An alternative hypothesis would involve the reactive chromosomes: in the case of table 1, some action of the *Sb* LH_{23} chromosome, enhancing *SF* sterility, might result in lower hatchability from C than from D females. The following experiment provides evidence for the latter hypothesis.

(ii) *Influence on* SF *females sterility of their reactive paternal chromosomes*

Using the +/+; *Cy*/*Pm*; *Sb*/*H* Valencia reactive strain instead of the LH_{23}, the same kind of matings as those described above were set up. The purpose was to compare the inducer efficiencies of paternal genotypes which

TABLE 2

Hatching percentages of eggs laid by sets of females according to their paternal genotype. The paternal X *chromosome, not represented here, is in each case the* +-(*r*) *chromosome from the Valencia strain.* +-(i) *and* +-(r) *chromosomes* 2 *come respectively from the Beaune and* seF_8 *strains.* Sb-(r) *and* H-(r) *are the homologous third chromosomes from the Valencia strain. Paternal chromosomes* 4 *are not represented; they are either reactive or non-inducer Beaune chromosomes.*

Expt. no.	1	2	3	4	5	6	7	Mean ± S.E.
+-(*i*); *Sb*-(*r*)	40	63	67	45	57	71	47	56·0 ± 4·5
+-(*i*); *H*-(*r*)	14	32	29	36	23	55	17	29·9 ± 5·3
Control no.	1	2	3	4	5	6	7	Mean ± S.E.
+-(*r*); *Sb*-(*r*)	96	98	96	92	92	92	97	94·9 ± 1·0
+-(*r*); *H*-(*r*)	98	95	97	99	98	97	97	97·2 ± 0·5

only differ in their reactive chromosome *3*. Therefore, not only +-(*r*); *Cy*-(*r*)/+-(*i*); *Sb*-(*r*)/+-(*i*) heterozygous males but also their +-(*r*); *Cy*-(*r*)/+-(*i*); *H*-(*r*)/+-(*i*) brothers were mated with e_{st28} reactive females. Among the female progeny of both crosses only one phenotypic class, from +-(*r*); +-(*i*); *Sb*-(*r*) and +-(*r*); +-(*i*); *H*-(*r*) paternal gametes respectively, was tested for hatchability. The experiments as well as the control, which started with reactive seF_8 instead of inducer Beaune males, were repeated seven times.

Hatchabilities from both classes of females are presented in table 2. As expected, females which have inherited the i^+ Beaune chromosome *2* are *SF* females with a lower hatchability than the corresponding control females bearing only reactive chromosomes. However, *H SF* females are significantly less fertile than *Sb* females. This difference, which is no longer detectable when females are old and fertile (results not shown) actually corresponds to a more intense *SF* sterility in *H* than in *Sb* females. Since *H* and *Sb* gametes statistically carry the same *X*, second and fourth chromosomes, the increase in inducer efficiency must be attributed to the replacement of the *Sb* by the *H* Valencia reactive chromosome.

In an experiment not reported here, *Sb* chromosomes from both Valencia and LH_{23} reactive strains were similarly compared and gametes carrying the LH_{23} *Sb* chromosome were found to have a stronger inducer efficiency than those with the Valencia *Sb* chromosome.

Present knowledge of the hereditary transmission of the reactive efficiency (also called reactivity level) suggest a reasonable explanation of the last

result. Bucheton and Picard (1978) have shown that the reactivity level of any reactive female, although mainly controlled by its maternal gamete, is also influenced by its paternally inherited chromosomes: when introduced in a foreign reactive oocyte any major reactive chromosome seems able to change the maternal reactivity level to some extent. Recently, Picard (1978) reported that chromosomal control of reactivity level might not only occur in reactive females but also in the daughters of *SF* females: to be sterile, they need to inherit reactive chromosomes able to maintain their cytoplasm in a reactive state. The second result presented in this Note agrees with this hypothesis in the sense that it suggests a change in the reactivity level in the *SF* females themselves: their sterility was influenced by the ability of the various third reactive paternal chromosomes assayed to maintain a more or less reactive cytoplasm. The same effect is expected for maternally inherited reactive chromosomes. This was in fact observed by Bucheton and Picard (1978, fig. 2). These facts lead to the conclusion that the inducer efficiency of a paternal gamete must no longer be only attributed to the characteristics of its i^+ chromosomes but also to the ability of its reactive chromosomes, if they have any, to maintain reactivity in the *SF* female. This must be kept in mind especially when the two following points are considered: Differences in inducer efficiency between sperm with a single i^+ chromosome will only show differences between these chromosomes if they are in the same reactive chromosomal background, that is only if they are homologous chromosomes. Since this was the case when i^+X chromosomes from the Otanu strain were compared (Pélisson, 1978), the conclusions concerning their differences in inducer efficiency still remain valid. For the same reason, no conclusions can be drawn from the comparison of inducer efficiencies of sperm bearing single or several i^+ chromosomes. The above results show that the inducer efficiency of sperm is never stronger with two i^+ chromosomes than with only one of them. However, this does not necessarily mean that the effects of individual i^+ chromosomes are not at all synergistic. They possibly might be so, if one assumes that when an i^+ chromosome is substituted, the same sterility is obtained because of the reactivity level increase brought about by the additional reactive chromosome. It must be emphasised that this compensation could not occur if the intensity of sterility was determined early in the *SF* female development. In this case, an independent action of i^+ chromosomes would have been found, similar to that reported by Engels (1979) with the *P-M* system. In fact, *I-R* and *P-M* hybrids have very different critical developmental stages: while the *P-M* sterility is due to a failure in the early development of the germ line (Schaefer *et al.*, 1979), all is likely to be normal in *SF* females until late oogenesis (Bregliano *et al.*, 1980). This trivial difference might possibly explain the discrepancy between Engels' results and those presented in this paper.

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