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Reduced fertility in 'SD' males and its bearing on Segregation Distortion in Drosophila melanogaster

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Citogenetica. — *Reduced fertility in* 'SD' *males and its bearing on Segregation Distortion in* Drosophila melanogaster (*). Nota di BENEDETTO NICOLETTI (**), GIOVANNI TRIPPA (**) e ANTONIO DE MARCO (**), presentata (***) dal Socio G. MONTALENTI.

RIASSUNTO. — Maschi di *Drosophila melanogaster*, eterozigoti per un secondo cromosoma portatore di un fattore 'SD', producono circa metà della progenie rispetto a maschi eterozigoti per un cromosoma normale. Questa osservazione rende poco plausibile il meccanismo proposto da Peacock ed Erickson secondo cui la 'Segregazione Distorta' avverrebbe per una migrazione preferenziale del cromosoma 'SD' al 'polo funzionale' dello spermatocita I. La ridotta fertilità dei maschi SD sembra invece dimostrare la mancata funzione o formazione di spermatozoi portatori del cromosoma omologo di SD, avvalorando un'ipotesi già prospettata da Sandler e coll.

INTRODUCTION.

The Segregation Distortion phenomenon, described for the first time by Sandler and coll. (1959) in *Drosophila melanogaster*, consists in the production of an excess of SD progeny by males heterozygous for a second chromosome carrying the SD factor.

Depending on the stocks, the proportion of SD individuals in the progeny may reach and even exceed 99 %. Although Sandler and coll. carried out an accurate analysis of the Segregation Distortion phenomenon, a precise model to explain its mechanism is not yet available.

Originally Sandler et al. postulated a mechanism implying the induction of breaks by the SD factor, on the homologous chromosome (SD^+), with ensuing loss of the latter.

Cytological observations (Peacock and Erickson, 1965; Nicoletti e Trippa, 1967) were not able at that time, however, to confirm such a mechanism. An alternative explanation was then suggested by Peacock and Erickson (1965). According to these authors, the SD chromosome preferentially segregates towards the so called 'functional pole', present in all (that is, with and without SD) spermatocytes I of the *D.m.* males. The SD^+-bearing chromosome, was postuleted to move to the 'non functional pole' and for this reason is not recovered in the progeny.

This hypothesis implies that both +/+ males and males heterozygous for SD should yield the same number of functional spermatozoa, and therefore, the same number of progeny.

The results of the experiments reported in this paper, however, allow us to establish that SD/+ males produce half the progeny that +/+ males do.

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(***) Nella seduta del 14 novembre 1967.

Our data, therefore, are not in agreement with the expectation of the hypothesis suggested by Peacock and Erickson; hence, the mechanism proposed by those authors for the SD phenomenon is rendered rather unlikely. These data, however, do not give us any information on the question of functional and non functional poles in spermatocytes I of *D.m.* generally.

Materials and methods.

Drosophila melanogaster males with a second chromosome carrying the SD factor (SD–72 , SD^{NH}–2 , SD^{R-1}) and with a normal second chromosome for control, have been constructed and their fertility measured. All the males were heterozygous for a second chromosome marked by the recessive gene *bw*. These were otherwise coisogenic owing to a long series of backcrosses to *bw*/*bw* females. The males from the three different SD stocks, which have been under study in our laboratory for several years, have always shown a rather constant and high *k* value ($\sim$ 1) (*k* = SD flies/total flies from a backcross).

A summary of the various characteristics of the different SD stocks is shown in Table I.

Table I.

Characteristics of the SD *factors used in our crosses.*

	SD^{NH}–2	SD–72	SD^{R-1}
Chromosome	II	II	II
Localization	centr. region	centr. region	centr. region
Recessive lethals associated	+	+	—
Inversions associated	+	+	—
Segregation in ♂♂	distorted	distorted	distorted
Segregation in ♀♀	normal	normal	normal
k values (average)	.99	.95	.95

The stocks SD–72 and SD^{NH}–2 were kindly provided respectively by L. Sandler, Seattle, and by R.E. Denell Austin. SD^{R-1} was found in a natural population near Rome.

20 single males at the average age of 24 h for each cross were put in vials on standard cornmeal-agar food with three virgin *bw*/*bw* females.

Every three days the males were transferred to new vials with three new virgin females. This procedure was repeated 12 times. The three females were left in the original vials and their progeny scored 17 days after the beginning of eclosion.

The cultures were kept in a controlled temperature incubator at 24° C ± 1.

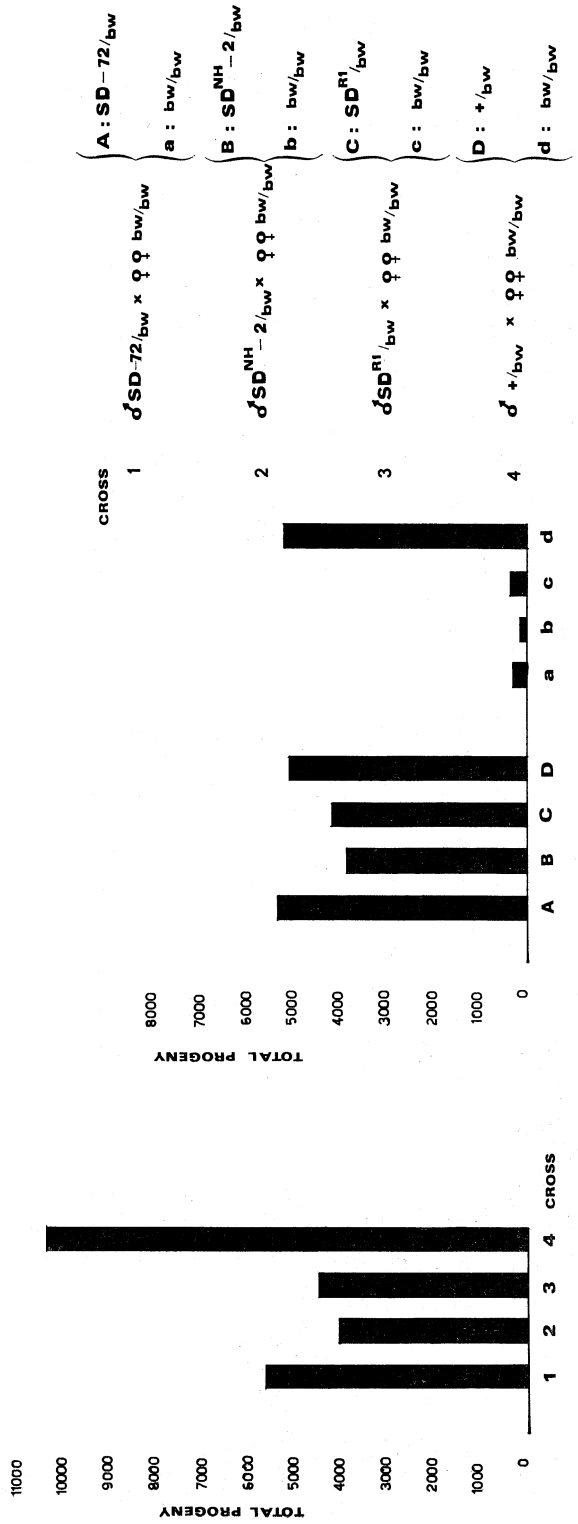

Fig. 1. – Total progeny of 12 fertile males for each cross.

RESULTS.

A summary of the results of the total progeny from the 12 of the 20 males tested, that were still alive at the 12th transfer, is reported in fig. 1. It is evident that the progeny yielded by the cross of $+/bw$ ♂♂ × bw/bw ♀♀ (10,374) is almost twice that yielded by the crosses SD/bw ♂♂ × bw/bw ♀♀ (SD–72 : 5,658; SD^{NH}–2 : 4,054; SD^{R-1} : 4,590).

Moreover, looking at the progeny classified according to the phenotype, + or bw (on the right in fig. 1), one sees a ratio of 1 : 1 for the cross $+/bw$ ♂♂ × bw/bw ♀♀, while the ratios for the backcrosses involving SD/bw males are quite close to 1 : 0. Thus the reduced fertility of the SD heterozygous males seems to be related to the absence of the class carrying the SD^+ chromosome, marked by bw.

The analysis of variance for the data regarding the $+/bw$ and SD/bw progeny (respectively produced by the 12 control $+/bw$ males and by the 36—three different SD have been considered—SD/bw males) reported in Table II shows no significant differences in the progeny number from the four crosses.

TABLE II.

Analysis of variance of SD/bw *and* $+/bw$ *progeny.*

Source of variation	Sum of squares	D.F.	Mean square	F
Between crosses	88,480	3	29,493	1,6
Within crosses	802,410	44	18,326	
Total	890,890	47		

This further strengthens the conclusions that in the SD males the reduced fertility is due to the failure of the production or functioning of the sperm carrying the SD^+ chromosome, since the SD sperm behaves similarly to those + in the control.

Fig. 3 shows the results of the progeny number of each males ranked in order of magnitude (12 in total) for the SD stocks (SD–72, SD^{NH}–2; SD^{R-1}) compared with the control males. The variability in the progeny yield by male is evident for the different crosses. It is clear, however, that the ratio near 2 : 1 for the progeny of the $+/bw$ compared to that of the SD/bw males is similar for all three SD lines considered.

The total progeny yielded by the 12 ♂♂ considered for each cross during the entire period in which they were tested are shown in fig. 3. For each cross, the progeny have been classified according the phenotype (+ or SD/bw and bw/bw).

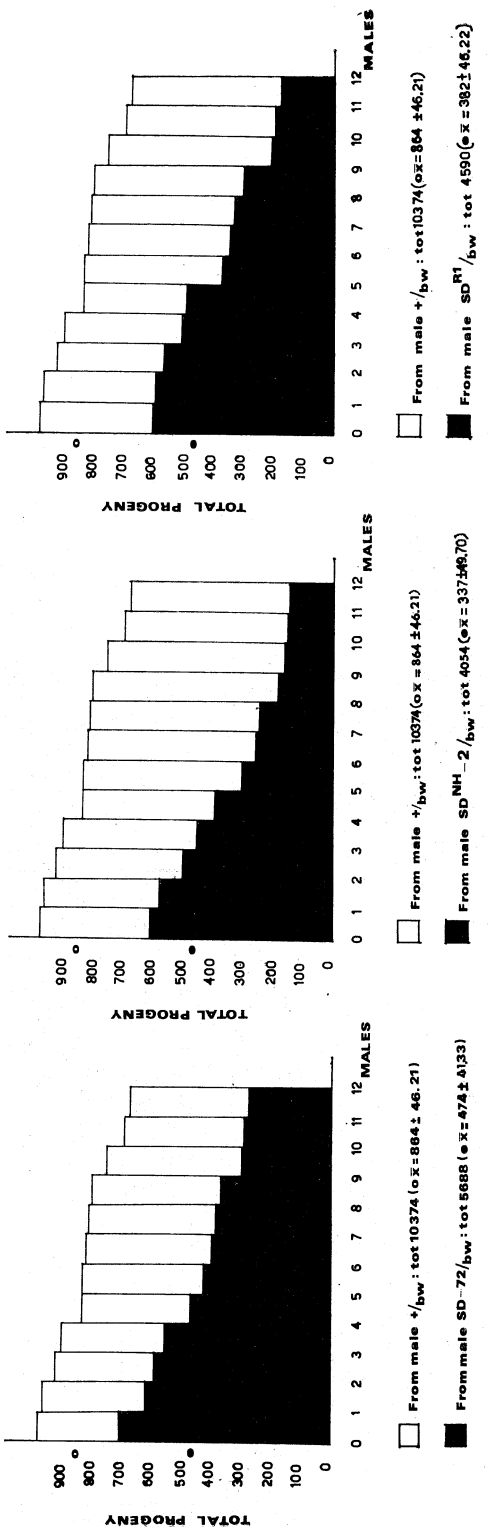

Fig. 2. – Ranked comparisons between the total progeny for each of the 12 males considered for the SD's and the control.

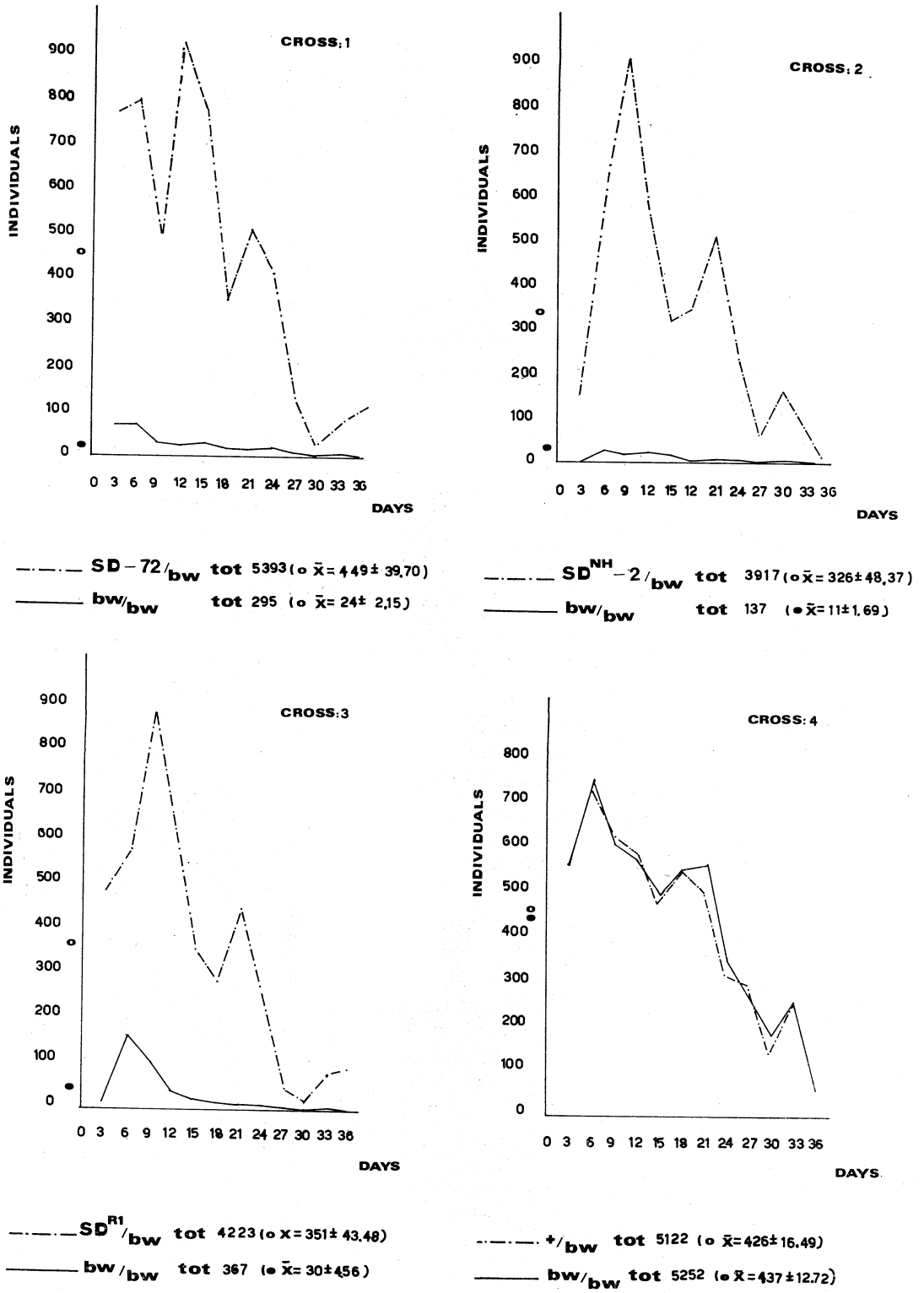

Fig. 3. – Total progeny and segregation from 12 males for each cross versus time.

For the SD crosses, the progeny are pratically all SD/*bw*, while in the control there is an equal number of the +/*bw* and *bw*/*bw* individuals.

In the four distributions, moreover, it is possible to note three distinct peaks about 10 days apart; this time corresponds quite well to the duration of spermiogenesis.

In fact, waves in sperm production have been already postulated by Bairati (1967) in *Drosophila*, with a time interval between subsequent waves similar to that, autoradiographically determined, necessary for an entire cycle of spermatogenesis (Chandley and Bateman, 1962; Olivieri and Olivieri, 1965).

Since the intervals between the peaks are similar for the control males and for those of the different crosses, this gives further support to the results of the analysis of variance: spermiogenesis for sperms carrying the SD chromosome in the SD/*bw* males or + chromosome in the +/*bw* males occurs similarly as far as time and quantity are concerned, even if the SD/*bw* males have a significantly reduced fertility.

If we consider now the *k* values for the total progeny (*k* = SD or + individuals/total individuals, from a backcross) for the 4 crosses, they showed distortion (i.e. *k* almost 1) for the progeny of the SD/*bw* males, while for the control males (+/*bw*) the *k* values in the progeny were completely normal ($k \sim 0.50$). The pattern of the *k* values as a function of time, see fig. 4, is similar for the three SD stocks considered and there are no significant variations with the age of the males tested. This observation does not agree with the results of Sandler and coll. (1961) who reported an inverse relation between *k* values and age of the SD males: we have not yet considered the possible reasons for this difference.

CONCLUSIONS.

From these results it is possible to draw some clear conclusions:

1) SD/*bw* males show a reduction in fertility of about 50 % when compared to +/*bw* males with a similar genetic background.

2) The reduction in fertility for the SD/*bw* males seems to have a direct relation to the lack of formation or lack of function of the sperm carrying the SD^+-bearing homologous chromosome. The characteristics of the production of SD spermatozoa by SD/*bw* ♂♂ and of spermatozoa by +/*bw* males give quite similar results, both in regard to their number and in the time required for their formation.

3) No indication was found of a relation between the age of the SD males and their *k* values.

These results enable us to disprove the model suggested by Peacock and Erickson to explain the Segregation Distortion phenomenon, since their model implies that the distortion in SD males is brought about only through a preferential movement of the SD chromosomes to the functional pole: this does not imply any reduction in the total number of spermatozoa derived from the functional pole.

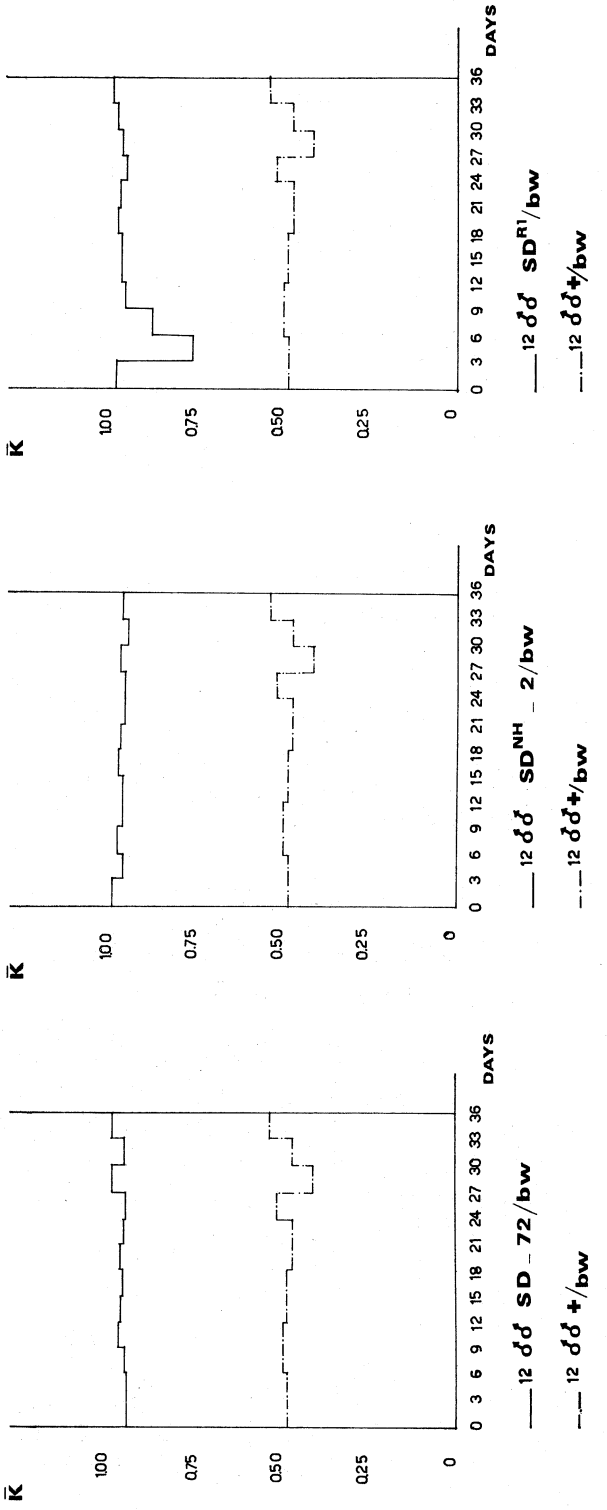

Fig. 4. – Average $\bar{k}$ versus time in the progeny of 12 fertile males for each cross.

The ascertainment that SD/*bw* ♂♂ produce only half the progeny that +/*bw* males do, suggests that the reduction in fertility of SD/*bw* ♂♂ could be explained as a lack in production or in function of sperm carrying the *bw* chromosome.

It is conforting to report that very similar results, with similar conclusions about the Peacock and Erickson model for SD, have been indipendently reached by Hartl, Hiraizumi and Crow (in press) in Madison, Wisconsin, USA.

It appears, therefore, that the mechanism determining Segregation Distortion in SD males must be related to the failure in the formation or in the function of the sperms carrying in the SD^+ chromosome (as suggested originally by Sandler and coll., 1959).

Some preliminary observations at the Electron Microscope (Nicoletti, 1967; Bertolini e Nicoletti, 1967) showing, in the testis of SD/*bw* ♂♂, degeneration for entire cists or for several elements in them, could represent the morphological basis for the failure of SD^+ sperms to function.

Nevertheless, at present, nothing is known about the precise mechanism of the non-formation, or non-function or degeneration of the SD^+-bearing (*bw*) gametes.

The evidence reported of a reduction in the reproductive fitness for the SD heterozygous males has its importance, moreover, on the evolution of the gene frequencies and SD factors in natural populations of *Drosophila*. The reduced fertility of the SD males should play an important role as a compensatory mechanism of the rapid spread of the SD factor in *Drosophila* populations.

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