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# The sensitivity of $SD^+$ chromosomes decreases over the generations in heterozygosis with SD chromosomes in Drosophila melanogaster males

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# **Genetica.** — *The sensitivity of* SD$^+$ *chromosomes decreases over the generations in heterozygosis with* SD *chromosomes in* Drosophila melanogaster *males* (*). Nota di GIOVANNI TRIPPA e ROSADELE CICCHETTI (**), presentata (***) dal Socio G. MONTALENTI.

RIASSUNTO. — Il grado di sensibilità media di una serie di cromosomi secondi non-*SD*, sensibili all'azione disfunzionante dei fattori *Sd*, diminuisce col numero di generazioni trascorse in eterozigosi con il cromosoma $SD^{R-1}$. Questo fenomeno si manifesta nel corso di alcune generazioni. La diminuzione complessiva del grado di sensibilità dei cromosomi $SD^+$ è dovuta sia alla comparsa nelle generazioni di un maggior numero di maschi con valori più bassi di segregazione distorta, sia al fatto che nell'ambito di alcune « famiglie » alcuni individui di una serie di fratelli presentano nel susseguirsi delle generazioni un crescente grado di insensibilità alla distorsione. Parallelamente molti di questi maschi mostrano un accentuato grado di sterilità.

Le numerose analogie tra la magnificazione di *bobbed* (Ritossa, 1976), e la diminuzione della distorsione della segregazione in maschi $SD/SD^+$ rendono suggestiva l'ipotesi che un meccanismo simile alla magnificazione dei geni per l'rDNA sia alla base della aumentata insensibilità dei cromosomi $SD^+$ ad *SD*.

## INTRODUCTION

$SD/SD^+$ males of *Drosophila melanogaster*—where *SD* is a second segregation distorter [1] chromosome and $SD^+$ a suitable second chromosome sensitive to the action of *SD*—produce almost exclusively *SD* progeny due to dysfunction of $SD^+$ gametes [2–3]. Despite the fact that numerous aspects of the segregation distortion phenomenon have been clarified [4], the mode of action of the *Sd* factors is still a matter of speculation [4–6].

The following circumstantial evidence has recently drawn our attention to the sensitivity of the second $SD^+$ chromosomes to dysfunction by *SD*: 1) the identification and localization of the two main elements of the *SD* system: the *Sd* locus (segregation distorter) and *Rsp* (Responder) [1, 5–8], 2) the fact that the degree of segregation distortion, measured by *k* (= *SD* individuals/total progeny) at $F_1$ of $SD/SD^+$ males, varies from 0.5 to 1.0 depending on the $SD$-$SD^+$ combinations [9, 10]; moreover, *Sd* genes of different origin can complement for male fertility in $SD_i/SD_j$ combinations, suggesting that there are different *Sd* and/or *Rsp* alleles ([5, 8, 11, 12], and finally 3) the

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difficulty in explaining the rapid appearance of non-*SD* second chromosomes insensitive to *SD* in laboratory populations containing only $Sd\ Rsp^{\mathrm{ins}}$ and $Sd^{+}\ Rsp^{\mathrm{sens}}$ chromosomes [13].

We therefore believe that studies on the sensitivity of second $SD^{+}$ chromosomes sensitive to *SD* contribute useful information on the mode of action underlying the segregation distortion phenomenon.

In the present paper we report that in the interval of just six generations the mean degree of sensitivity (*k*) of a series of second chromosomes sensitive to *SD* varies from 0.96 (high sensitivity) to 0.75 (moderate sensitivity). Still more important is the fact that in each generation only some of a series of males show this decrease in *k* and if some $SD^{+}$ chromosomes are followed over the generations, these show greater insensitivity values than the mean.

## MATERIALS AND METHODS

*Chromosomes used*:

1. $SD^{R-1} = Sd^{R-1}\ Rsp^{\mathrm{ins}}\ bw^{R-1}$, isolated from a natural population collected near Rome [14, 15]. Besides the factors $SD^{R-1}$ and $Rsp^{\mathrm{ins}}$ it carries a *bw* (*brown*: dark red eyes) isoallele which in the genotype $bw^{R-1}/bw$; $st/st$ determines a characteristic eye colour phenotype. The $\bar{k}$ value of $Sd^{R-1}/bw\text{–}5$ males is 0.97.

2. A series of second chromosomes marked with *bw–5* sensitive to *SD* derived from the isogenic line *y* ; *bw–5* ; *st–5*.

*Cross scheme*:

This is shown in Fig. 1.

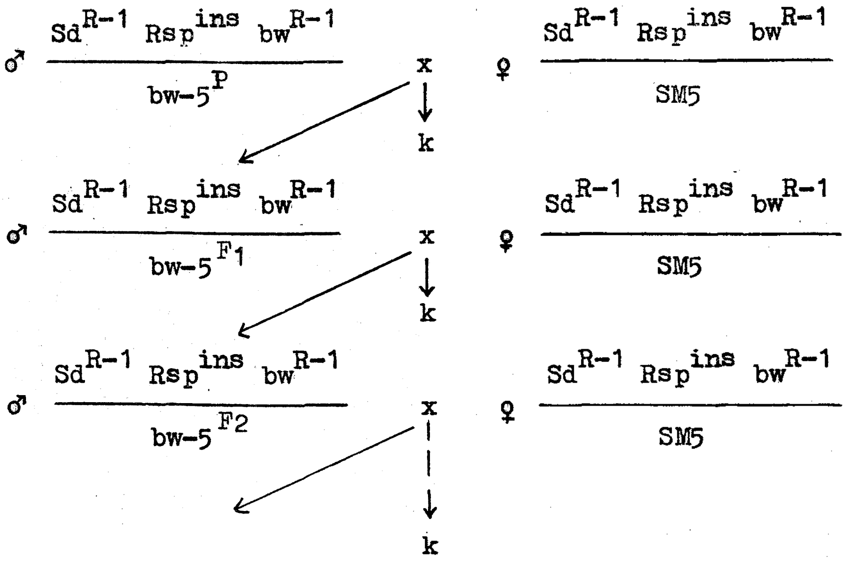

Fig. 1. – Cross scheme for testing the sensitivity (*k*) of *bw–5* chromosomes to $Sd^{R-1}$ gene in consecutive generations.

*Sensitivity of* bw–5 *chromosomes to the* $\mathrm{SD}^{\mathrm{R-1}}$ *chromosome.*

The degree of sensitivity is obtained with the following formula:

$$k = \frac{\textit{Curly}\ \text{individuals} - \textit{SD}/\textit{bw}\ \text{individuals}}{\textit{Curly}\ \text{individuals}}\,. \tag{1}$$

It is not possible to calculate the *k* value in the usual way in this type of cross because: 1. the $SD^{\mathrm{R-1}}/SD^{\mathrm{R-1}}$ individuals appear with a lesser frequency than expected and 2. the two classes *SD/Cy* and *bw/Cy* cannot be distinguished from each other. A series of observations permitted us to apply formula (1). In fact: *a*) the two classes *bw/Cy* and $bw/SD^{\mathrm{R-1}}$ have an equivalent viability and *b*) the segregation of the chromosomes $SD^{\mathrm{R-1}}$ and *Cy* in *SD/Cy* females is normal. It must however be noted that the decrease in *k* values observed over the generations cannot be explained merely by the non occurrence of these two events, unless it is admitted that one or the other or both together always vary in the same direction.

51 $Sd^{\mathrm{R-1}}\,Rsp^{\mathrm{ins}}\,bw^{\mathrm{R-1}}/bw\text{-}5;\ st\text{-}5/st\text{-}5$ males were crossed with females of the strain $Sd^{\mathrm{R-1}}\,Rsp^{\mathrm{ins}}\,bw^{\mathrm{R-1}}/SM5;\ st\text{-}5/st\text{-}5$ according to the scheme in Fig. 1 to recover at each generation *bw*-5 chromosomes which had escaped the $SD^{\mathrm{R-1}}$ action. In each generation, after being kept for 3 or 4 days with two females, the males were transferred twice to obtain the greatest number of $SD^{\mathrm{R-1}}/bw\text{-}5$ males and total progeny. All $SD^{\mathrm{R-1}}/bw\text{-}5$ males born in each of the six generations studied were crossed, independently of the *k* values of their fathers.

The experiments were carried out at 24 °C ± 1 °C in vials containing standard food.

## RESULTS AND DISCUSSION

The pattern of the *k* values of $SD^{\mathrm{R-1}}/bw\text{-}5$ males in the six generations examined is shown in Fig. 2. This figure shows that the decrease in *k* from 0.96 (high sensitivity) to about 0.70 (moderate sensitivity) occurs over a few generations and can only depend on the appearance of an increased degree of insensitivity of *bw-5* chromosomes. The occurrence of the $SD^{+}$ chromosomes insensitive to *SD* in laboratory populations [13, 16] has been explained by the formation of recombinant $Sd^{+}\,Rsp^{\mathrm{ins}}$ chromosomes by double heterozygous $Sd\ Rsp^{\mathrm{ins}}/Sd^{+}Rsp^{\mathrm{sens}}$ females [16]. Our cross scheme permits us to exclude this possibility since the *bw–5* chromosome is always transmitted from male to male. On the other hand the existence of second chromosomes giving rise to rare phenomena of recombination in *Drosophila melanogaster* males which are recovered with both a lower [17] and a higher frequency (*SD*) than expected (M.M. Green, personal comunication) has been known from some time. Also this possibility can be excluded in our experiments since the presence of $bw^{\mathrm{R-1}}$ on the $SD^{\mathrm{R-1}}$ chromosome would have led after recombination to the occurrence of both sterile $Sd^{\mathrm{R-1}}\,bw^{\mathrm{R-1}}/Sd^{\mathrm{R-1}}\,bw\text{-}5;$

*st-5/st-5* males (with characteristic eye colour, see Materials and Methods) and $Sd^{R-1}\ bw^{R-1}/Sd^{+}\ bw^{R-1}$; *st*-5/*st*-5 males (with *st* eye colour) genotypically classifiable as $Sd^{R-1}\ bw^{R-1}/Sd^{R-1}\ bw^{R-1}$; *st*-5/*st*-5 which, expected to be sterile, should have been fertile. Instead 241 males with the *st* phenotype, tested for fertility, were all sterile.

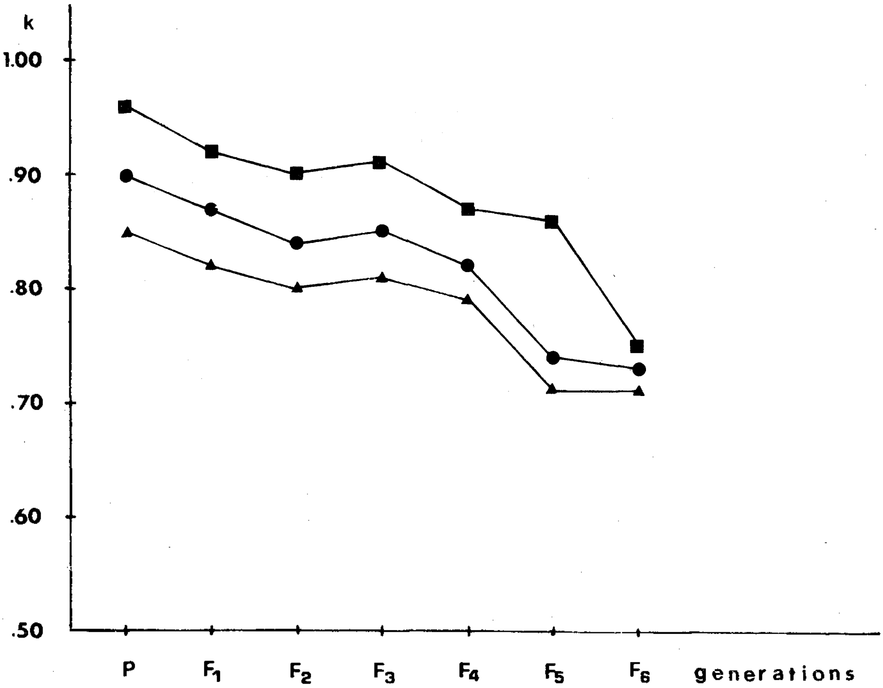

Fig. 2. – Decrease in sensitivity ($k$) of second chromosomes *bw–5* to $Sd^{R-1}$ gene after having been in heterozygosis with the $SD^{R-1}$ chromosome for a different number of generations.

■ = mean $k$ values ($\bar{k}$); ● = $\bar{k}$ values excluding those between 1.00 and 0.95;
▲ = $\bar{k}$ values excluding also $k$ values between 0.94 and 0.90.

Examining the segregation ratio in more detail we were able to observe that: 1) only some males in each generation show this increased insensitivity of the *bw-5* chromosome to $SD^{R-1}$, 2) on the whole the number of males in which the $k$ value is notably lowered increases over the generations, and 3) within some "families" where this phenomenon has arisen it is not present in all males, despite the fact that some of them show an insensitivity value greater than the mean.

It is interesting to note, moreover, that in consecutive generations and with the occurrence of a more widespread and increased insensitivity of the *bw-5* chromosomes, there is an increasing degree of sterility in $Sd^{R-1}\ Rsp^{ins}\ bw^{R-1}/bw\text{-}5$ males. Thus, considering that the degree of sterility of the $SD/SD^{+}$ heterozygotes is related to the $k$ values [3], paradoxically, instead of recovering part of their fertility these males seem to lose the advantage which should derive from a greater insensitivity to *SD* of *SD*$^+$ chromosomes, unless there is an insensitivity recovery threshold of *SD*$^+$ chromosomes.

Some models have been proposed to interpret the *SD* phenomenon, above all on the basis of analogies with the behaviour of the $sc^4$–$sc^8$ deficiencies [18] and with the intracistronic complementation of enzyme activity [5]. These are still the subject of discussion.

Despite the fact that the available data do not permit a precise hypothesis on the mechanisms underlying the observations reported, the remarkable similarity between the series of events leading to the magnification of the *bobbed* phenotype [19] and the processes which in an *SD*/*SD*$^+$ male lead to a decreased sensitivity to *SD* of the *SD*$^+$ chromosomes, makes the hypothesis that a mechanism analogous to the magnification of genes for rDNA is responsible for the decrease in distorted segregation, a very attractive one, especially taking into account the fact that *Sd* could actually be the locus of the sperm histones [20, 21].

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