A factor on a wild third chromosome (III\textsuperscript{Ra})
that modifies the Segregation Distortion phenomenon in
\textit{Drosophila melanogaster}

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SUMMARY

III\textsuperscript{Ra} is a genetic modifier of Segregation Distortion (SD) in \textit{Drosophila melanogaster}, which was discovered in the same natural population from Ranna (Sicily) that carried SD\textsuperscript{Ra}. It is located at 49.7 ± 0.8 on chromosome III. III\textsuperscript{Ra} was found to have a dominant effect on segregation distortion which varied with the origin of the SD chromosome tested. Thus it enhanced the level of distortion caused by 14 SD chromosomes from seven natural populations in Southern Italy and Sicily, but decreased the level of distortion caused by SD\textsuperscript{11}\textsuperscript{1}, a chromosome from a natural population near Rome. Moreover, III\textsuperscript{Ra} determined or enhanced the distorting effect of SD\textsuperscript{Ra} in males heterozygous for SD\textsuperscript{Ra} and various SD\textsuperscript{+} wild chromosomes differently sensitive to SD\textsuperscript{Ra}. The frequency of chromosomes having an effect like III\textsuperscript{Ra} chromosome was very high (around 70\%) in samples from two natural populations of Southern Italy tested—those of Ranna and Corato. No effects of III\textsuperscript{Ra} other than its ability to modify SD have been detected.

1. INTRODUCTION

Examples of meiotic drive, as defined by Zimmering, Sandler & Nicoletti (1970), are found in maize, wheat, rye, mice, cattle and man. Factors which modify meiotic drive are also known, e.g. enhancing and suppressing elements in \textit{Drosophila} (see Zimmering et al. 1970; Peacock & Miklos, 1973), so that some cases of meiotic drive appear to be the result of the action of complex genetic systems rather than a single gene.

The Segregation Distortion phenomenon in \textit{Drosophila melanogaster} (Sandler et al. 1959) was recently analysed by Miklos (1972a, b) and Peacock & Miklos (1973), who showed that the system consists essentially of the SD gene, acting in the male line only. The effect of the SD factor (mapped on the second chromosome at locus 52.9 ± 1, Tanzarella et al. 1972) is to cause the degeneration of SD\textsuperscript{+} gametes in heterozygous SD/SD\textsuperscript{+} males (Nicoletti, 1968; Tokuyasu, Peacock & Hardy, 1972) and consequently the preferential recovery of gametes carrying the SD chromosome (Nicoletti, Trippa & De Marco, 1967; Hartl, Hiraizumi & Crow, 1967). The existence of other factors which influence the Segregation Distorter system, such as St(SD) = Stabilizer of SD and Ac(SD) = Activator of SD, was
proposed by Sandler & Hiraizumi (1960a, b) on the basis of the analysis of their data on distortion in terms of $k$ values ($k = \frac{SD}{SD + SD^+}$), the proportion of $SD$ individuals recovered among the progeny of $SD/SD^+$ heterozygous males). These authors interpreted the Stabilizer as a gene which controls the degree of mutability of the $SD$ locus and the Activator as a gene which controls the mechanism causing the $SD$ gene to operate. Data collected by Sandler & Hiraizumi (1960b) and Hiraizumi & Nakazima (1967) have recently been brilliantly re-analysed by Miklos (1972a, b) and Peacock & Miklos (1973), in terms of standard deviation units in addition to $k$ values. This has permitted interpretation of the data in terms of the presence or absence of simple modifiers of $SD$ action rather than of $SD$ Activator or Stabilizer effects, as defined above.

It has also been shown that Segregation Distortion can be modified by various factors: for instance, temperature (Mange, 1968; Hihara, 1971) and irradiation (Murnik, 1971) among environmental factors; and ageing of the $SD/SD^+$ male (Sandler & Hiraizumi, 1961; Hiraizumi & Watanabe, 1969; Nicoletti & Micheli, 1970) among physiological factors.

The following genetic factors affect Segregation Distortion:

1. The genetic factor responsible for differences in sensitivity of $SD^+$ second chromosomes to the action of $SD$; it has been mapped at or close to the $SD^+$ locus. According to Trippa & Loverre (1972) this factor is the $SD^+$ locus itself (as a series of different $SD^+$ alleles) whereas Sandler & Carpenter (1972) consider it more likely to be a different locus.

2. A series of modifiers of distortion located on the second chromosome between $cn^+$ and $c^+$, and between $c^+$ and $px^+$ (Miklos, 1972a).

3. A series of suppressor genes which greatly reduce the action of $SD$ designated $Su(SD) = \text{Suppressor of } SD$, identified on different chromosomes as follows: (a) on the proximal region of a laboratory $X$ chromosome (Sandler, 1962); (b) on a standard Muller-5 $X$ chromosome (Sandler & Rosenfeld, 1962); (c) on a laboratory $FM6 X$ chromosome (Nicoletti & Trippa, 1967); (d) on $X$ chromosomes from natural populations collected in Odate, Japan (Hiraizumi & Kataoka, 1965; Kataoka, 1967); (e) on second chromosomes from a natural population collected near Madison, Wisconsin (Hiraizumi, Sandler & Crow, 1960); (f) on $X$ and second chromosomes from a natural population collected in Madison, Wisconsin (Hartl, 1970).

All the genetic factors described so far are elements that modify the action of $SD$ and are located on either the $X$ or the second chromosome. This paper describes a factor located on a wild third chromosome which is able to modify the level of segregation distortion in males heterozygous for different $SD$ chromosomes and different second $SD^+$ chromosomes. Data are also supplied on the frequency of analogous third chromosomes modifying the $SD$ effect in two Italian natural populations.
2. MATERIALS AND METHODS

The following $SD$ second chromosomes were used:

(1) $SD^{Ra}$, isolated from the natural population obtained from Ranna, Sicily (Trippa et al. 1972). This chromosome contains a paracentric inversion on the left arm, a recessive gene $fs$ ($2^{TL}$) responsible for sterility in the female line only, located at locus 89-7 (Loverre et al. 1972), and a recessive mutation at locus $bw$ or a small deficiency at the same locus, not recognizable in the salivary chromosomes analysis, which when heterozygous with $bw$ exhibits a characteristic eye colour phenotype. The chromosome is homozygous viable.

(2) $SD^{R-1}$, isolated from a natural population from Rome (Nicoletti & Trippa, 1967; Sandler et al. 1968). This chromosome does not appear to be associated either with inversions detectable by salivary chromosome analysis or with recessive lethals. The $k$ analysis of several recombinants between $SD^{R-1}$ and a second multimarked chromosome did not reveal the presence of any kind of Stabilizer or Activator factors. The $k$ value for $SD^{R-1}/bw-5$ males is $0.988 \pm 0.002$.

(3) A series of 14 $SD$ chromosomes isolated from samples of 7 natural populations of Southern Italy (Trippa et al. 1972) and designated: Ca 148, Ca 230 (Castellaneta); Ra 88, Ra 126 (Ranna); Sa 262 (Sambiase); Pe 6, Pe 122 (Pedalino); Ar 249, Ar 38 (Archi); Ot 1 (Otranto); Co 17, Co 64, Co 100, Co IV (Corato).

The following lines and chromosomes were used for the construction of suitable genotypes, for mapping the modifying gene and for calculating the $k$ values in the progeny of $SD/SD^+$ males (for a more detailed description of the mutant genes and balancer chromosomes, see Lindsley & Grell, 1968):

(1) A series of eight second chromosomes derived from a sample of 291 $SD^+$ chromosomes from the Ranna and Corato populations, which were maintained in stocks either as homozygotes or heterozygous with $SM5$. These second chromosomes had previously been shown (Trippa et al. 1973) to be moderately sensitive ($k = 0.63$) to the distorting action of two $SD$ chromosomes derived by recombination from $SD^{R-1}$ (referred as $SD ern L Pin$ and $SD L$, whose $k$ in males heterozygous for sensitive chromosomes was, respectively, 0.95 and 0.90).

(2) Chromosome $III^{Ra}$: derived from the Ranna natural population isolated from the same male that carried $SD^{Ra}$. This chromosome segregated normally in $III^{Ra}/st-5$ heterozygous males ($k = 0.540 \pm 0.007$).

(3) Fifty-seven third chromosomes derived from samples of the same (geographic) populations of Ranna (47 chromosomes) and Corato (10 chromosomes), collected one year after the isolation of the $SD$ and $III^{Ra}$ chromosomes (October 1972) and balanced against $TM2$.

(4) $y; bw-5; st-5$: an isogenic line selected from a $bw; st$ stock for high sensitivity to $SD^{R-1}$ chromosome, by using the $y; SM5; TM2/T(2; 3) S 9, bw e$ strain. The sensitivity of this line to the $SD^{R-1}$ chromosome does not depend on the third chromosome because $SD^{R-1}/bw-5; st-5/st-5, SD^{R-1}/bw-5; +/st-5$ and $SD^{R-1}/bw-5; +/+ $ males yield $k$ values of $0.988 \pm 0.002$, $0.994 \pm 0.001$ and $0.984 \pm 0.003$, respectively.
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(5) $y; bw-7; st-7$: an isogenic line isolated as above from the same $bw; st$ stock and selected for average sensitivity to the $SD^{-1}$ chromosome ($SD^{-1}/bw-7; st-7/st-7$ males show $k = 0.710 \pm 0.007$ and $SD^{-1}/bw-7; +/+$ males yield $k = 0.716 \pm 0.013$).

(6) $SM5$: a balancer for chromosome 2; homozygous lethal.

(7) $TM2$: a balancer for chromosome 3; homozygous lethal.

(8) $GlSb/TM2$: balanced lethals stock where the $Gl Sb$ chromosome carries the same modifying factor present on the $III^{Ra}$ chromosome.

In all crosses for calculating $k$ values, females of the $y; bw-5; st-5$ stock were used. In all experiments matings were set up with one male and two virgin females in single vials with standard corn-meal-agar medium. The $k$ values were calculated within ten days after eclosion. All work was performed under standard conditions at $24 \pm 1 \, ^\circ C$.

3. RESULTS AND DISCUSSION

(i) Recovery and characterization of the $SD^{Ra}$ and $III^{Ra}$ chromosomes

During a search performed to assess the frequency of $SD$ chromosomes in natural populations of Drosophila melanogaster from Southern Italy (Trippa et al. 1972), a second and a third chromosome were isolated by crossing a wild-type male from the Ranna population with females of the $y; bw-5; st-5$ stock.

The $k$ values exhibited by $F_2$ males of $+/bw-5; +/st-5$ and $+/bw-5; st-5/st-5$ genetic composition were then measured. Their relative distribution is reported in Fig. 1, which shows that the wild second chromosome behaves as an $SD$ chromosome, altering the segregation of the two second chromosome homologues. This second chromosome is designated $SD^{Ra}$ (for description, see Materials and Methods). The data also show that there exists a significant difference ($P < 0.001$) in the $k$ values of the males of the two genotypes and that this difference stems from the presence of the wild third chromosome designated $III^{Ra}$, which enhances the distortion caused by $SD^{Ra}$. In a series of parallel crosses it has been ascertained that $III^{Ra}$ has an equal enhancing effect when heterozygous and homozygous, indicating that the factor(s) present on the $III^{Ra}$ chromosome is dominant.

(ii) Genetic localization of the Segregation Distortion modifier

To localize the modifier on $III^{Ra}$, $bw^+/bw-5; Gl Sb/III^{Ra}$ females were crossed with $SD^{Ra}/bw-5; st-5/st-5$ males. Since all the $SD^{Ra}$ male progeny carrying a third recombinant chromosome (about 100 males) yielded a $k$ value of 1.00, it was concluded that the $Gl Sb$ chromosome was carrying the same modifier as the $III^{Ra}$ chromosome. This was confirmed by testing $SD^{Ra}/bw-5; Gl Sb/st-5$ males, which yielded a $k$ value of 1.00; and on the basis of these results, $bw^+/bw-5; Gl st^+ Sb/Gl^+ st-5 Sb^+$ females were crossed with $SD^{Ra}/bw-5; Gl^+ st-5 Sb^+/Gl^+ st-5 Sb^+$ males to localize the modifier on the $Gl Sb$ chromosome. It was then possible to map the modifier factor by using the $k$ values shown by the various types of $SD^{Ra}/bw-5; third recombinant chromosome/st-5$ males.
Fig. 1. Distribution of $k$ values exhibited by $SD^{Ra}/bw-5; III^{Ra}/st-5$ (A) and $SD^{Ra}/bw-5; st-5/st-5$ (B) males. Each square represents the $k$ value shown by one male.

Table 1. Genetic localization of the $III^{Ra}$ modifier

| Types of crossover | $k \pm S.E.$ | $k \pm S.E.$ |
|--------------------|--------------|--------------|
|                    | (modifier absent) | (modifier present) |
| Gl st Sb$^+$      | 0.76 ± 0.01  | 1.00 ± 0.00  |
| Gl$^+$ st$^+$ Sb  | 1            | 12           |
| Gl st$^+$ Sb$^+$  | 6            | 13           |
| Gl$^+$ st Sb      | 35           | 26           |

(Distribution of the modifier among $F_1$ males of $SD^{Ra}/bw-5; different crossover/st-5$ genotypes. The parental cross was $bw^+/bw-5; Gl st^+ Sb$ (carrying the modifier)/$Gl^+ st-5 Sb^+$ females with $SD^{Ra}/bw-5; Gl^+ st-5 Sb^+/Gl^+ st-5 Sb^+$ males.)
From a first analysis of the figures given in Table 1 it can be established that the modifier lies in the region between st and Sb. Moreover, since out of 80 crossovers between st and Sb, 32 were crossovers between st and the modifier and 48 between the modifier and the Sb locus, it was possible to locate the modifier at about 32 eightieths of the distance between the two markers (14.2 map units) from st. Using the standard gene locations (Lindsley & Grell, 1968) of st (44-0) and Sb (58-2), the locus of the modifier can be calculated at 49.7 ± 0.8.

(iii) The III Ra factor as modifier of the distortion caused by SD chromosomes

An experiment was performed to ascertain the ability of the III Ra factor to modify the amount of distortion exhibited by the various SD chromosomes listed under Materials and Methods. The k value of SD/bw-5; III Ra/st-5 males was thus measured.

Table 2. Reversal of the modifier effect of the III Ra factor depending upon the source of the SD chromosomes

| Chromosome II tested | III Ra absent* (k ± S.E.) | III Ra present† (k ± S.E.) |
|----------------------|---------------------------|---------------------------|
| SD from South Italy   |                           |                           |
| Ra-88                | 0.70 ± 0.02               | 0.996 ± 0.002             |
| Ra-127               | 0.72 ± 0.03               | 0.969 ± 0.013             |
| Ca-148               | 0.73 ± 0.02               | 1.000 ± 0.000             |
| Ca-230               | 0.76 ± 0.02               | 0.999 ± 0.000             |
| Ot-1                 | 0.76 ± 0.03               | 0.994 ± 0.002             |
| Co-17                | 0.81 ± 0.02               | 1.000 ± 0.000             |
| Co-64                | 0.69 ± 0.01               | 1.000 ± 0.000             |
| Co-100               | 0.65 ± 0.01               | 1.000 ± 0.000             |
| Co-IV                | 0.69 ± 0.02               | 1.000 ± 0.000             |
| Ar-38                | 0.67 ± 0.03               | 1.000 ± 0.000             |
| Ar-249               | 0.74 ± 0.03               | 1.000 ± 0.000             |
| Pe-6                 | 0.76 ± 0.01               | 1.000 ± 0.000             |
| Pe-122               | 0.73 ± 0.01               | 0.998 ± 0.000             |
| Sa-262               | 0.75 ± 0.04               | 1.000 ± 0.000             |
| SD from Rome         |                           |                           |
| R-1                  | 0.99 ± 0.00               | 0.878 ± 0.020             |
| SD+ chromosomes      |                           |                           |
| Sa-235               | 0.57 ± 0.03               | 0.535 ± 0.013             |
| SM5                  | 0.45 ± 0.02               | 0.466 ± 0.011             |

Every k estimate was obtained from about 24* or 12† males, respectively. Each male was tested by examining a progeny of approximately 100 individuals. After having ascertained that the n k's of different males of the same group were homogeneous, the k was calculated as their arithmetical mean and its S.E. by the following formula: \( \sqrt{\frac{n}{n(n-1)}} \sum_{i=1}^{n} (k_i - \bar{k})^2 \) calculated. Two control crosses were made: (1) to evaluate the amount of distortion caused by the SD chromosomes in the absence of the III Ra chromosome; the k value yielded by SD/bw-5; st-5/st-5 males was thus measured; (2) to ascertain whether the III Ra factor modified the segregation of second chromosomes only when an SD chromosome was present in the male genotype, while not interfering...
A modifier of SD

In the segregation of two SD+ chromosomes. For this purpose, either a second chromosome extracted from the natural population of Sambiase, Sa-235, or the SM5 balancer chromosome was used. The k values of Sa-235/bw-5; IIIRa/st-5 and SM5/bw-5; IIIRa/st-5 males were then measured.

Table 2 shows the amount of segregation distortion exhibited by males carrying different SD or SD+ chromosomes when the IIIRa factor is present in their genotype and when it is absent. SD/bw-5; st-5/st-5 males yield k values varying between 0.65 and 0.81, SDR-1/bw-5; st-5/st-5 males, by contrast, exhibit a k value of 0.99. When IIIRa is also present the k exhibited by all the Southern Italian SD-bearing males increases to values between 0.99 and 1.00, while that shown by SD-1 males drops to 0.88 ± 0.02. Hence, from its behaviour in the laboratory, it would seem that the IIIRa factor should be regarded as a modifier of the Segregation Distortion phenomenon rather than specifically as an enhancer or a suppressor. Table 2 also shows that IIIRa did not alter the segregation of either the Sa-235 or the SM5 chromosome in comparison with a bw-5 chromosome, indicating that it does not affect the segregation of second chromosomes when neither carries SD.

(iv) The modification of SD factors induced by IIIRa is not heritable

Sandler & Rosenfeld (1962) demonstrated that the X-chromosome balancer, Muller-5, has one or more modifiers (suppressors) of Segregation Distortion and that the inhibiting action of the modifier remains heritable for at least two generations after the Muller-5 chromosome has been removed. These authors believed it reasonable to suppose that modification of SD activity involves changes at the SD locus itself and not in the products of SD activity.

To test whether the modifying action of IIIRa is heritable the distortion in the progeny of SD/bw-5; st-5/st-5 males, derived from SD/bw-5; IIIRa/st-5 males,

Table 3. Testing the heritability of the modifying effect of IIIRa on SD. k values shown by SD/bw-5; st-5/st-5 males and SD/bw-5; IIIRa/st-5 parental males and SD/bw-5; st-5/st-5 F1 males

| Types of males | With no IIIRa chromosome with no IIIRa chromosome and derived from parents Carrying IIIRa chromosome and derived from parents With no IIIRa chromosome but derived from parents |
|----------------|---------------------------------|---------------------------------|---------------------------------|
| SD chromosomes| With no IIIRa chromosome and derived from parents | Carrying IIIRa chromosome and derived from parents | With no IIIRa chromosome but derived from parents |
| examined      | (k ± s.e.)                        | (k ± s.e.)                       | (k ± s.e.)                       |
| Ra            | 0.72 ± 0.01                       | 1.00 ± 0.00                      | 0.70 ± 0.02                      |
| Ra-88         | 0.70 ± 0.02                       | 1.00 ± 0.00                      | 0.64 ± 0.02                      |
| Ca-230        | 0.76 ± 0.02                       | 1.00 ± 0.00                      | 0.69 ± 0.01                      |
| Ot-1          | 0.76 ± 0.03                       | 0.99 ± 0.00                      | 0.67 ± 0.04                      |
| Co-17         | 0.81 ± 0.02                       | 1.00 ± 0.00                      | 0.80 ± 0.02                      |
| Co-64         | 0.69 ± 0.01                       | 1.00 ± 0.00                      | 0.60 ± 0.01                      |
| Pe-122        | 0.73 ± 0.01                       | 1.00 ± 0.00                      | 0.75 ± 0.02                      |

For each SD chromosome the k values in columns two and four are not statistically different. The s.e.s were calculated as in Table 2.

* About eight males, each with a progeny of approximately 100 individuals, were tested.
was measured (Table 3). It appears that the fact of being derived from a male parent carrying \textit{III}^{Ra} does not alter the segregation distortion caused by any of the seven natural \textit{SD} chromosomes examined. Thus \textit{III}^{Ra} does not induce any hereditary modification of the \textit{SD} gene.

**(v)** \textit{Action of III}^{Ra} \textit{in males heterozygous for SD and second chromosomes moderately sensitive to SD}

A series of experiments was performed to check whether \textit{III}^{Ra} was capable of modifying the segregation distortion even when the \textit{SD}^{Ra} chromosome was heterozygous with second chromosomes of different sensitivity to \textit{SD}^{Ra}. These second chromosomes were derived from Ranna and Corato populations and the

Table 4. \textit{The dependence of the segregation ratio observed upon the \textit{SD}^{+} and \textit{SD} chromosomes, the presence of the \textit{III}^{Ra} modifier, and the provenience of all these chromosomes relative to each other}

| Chromosomes from nature | SD\textit{Ra}/SD\textit{+}; III\textit{Ra}/st-5 | SD\textit{Ra}/SD\textit{+}; III\textit{Ra}/st-5 |
|-------------------------|---------------------------------|---------------------------------|
|                         | \textit{SD}^{Ra-1}/\textit{SD}^{+} | \textit{SD}^{Ra-1}/\textit{SD}^{+} |
|                         | \textit{k} | No. of males | No. of males | \textit{k} | Total progeny | Total progeny | \textit{k} ± s.e. | Total progeny | Total progeny | \textit{k} ± s.e. |
| Ra-39                   | 0.67      | 4            | 434          | 0.54 ± 0.05 | 8            | 885          | 0.74 ± 0.04 |
| Ra-67                   | 0.65      | 12           | 825          | 0.50 ± 0.02 | 19           | 1878         | 0.61 ± 0.02 |
| Ra-64                   | 0.62      | 8            | 718          | 0.52 ± 0.01 | 7            | 522          | 0.63 ± 0.02 |
| Ra-144                  | 0.69      | 20           | 1986         | 0.58 ± 0.01 | 12           | 1350         | 0.75 ± 0.02 |
| Co-5                    | 0.68      | 7            | 865          | 0.76 ± 0.03 | 10           | 1503         | 0.97 ± 0.01 |
| Co-11                   | 0.60      | 7            | 497          | 0.78 ± 0.02 | 7            | 427          | 0.94 ± 0.03 |
| Co-23                   | 0.60      | 8            | 832          | 0.88 ± 0.04 | 8            | 680          | 1.00 ± 0.00 |
| Co-50                   | 0.60      | 12           | 1476         | 0.64 ± 0.03 | 18           | 2163         | 0.92 ± 0.02 |

Chromosomes from the laboratory

| bw-7                     | 0.71      | 30           | 2414         | 0.77 ± 0.01 | 26           | 3117         | 1.00 ± 0.00 |

For each \textit{SD}^{+} chromosome the \textit{k} values in columns five and eight are statistically different (P < 0.01). The s.e.s were calculated as explained in Table 2.

For each \textit{SD}^{+} chromosome the \textit{k} values in columns five and eight are statistically different (P < 0.01). The s.e.s were calculated as explained in Table 2.

Laboratory strain \textit{bw-7}, and were all selected on the basis of their moderate response to \textit{SD}^{Ra-1} (column 2, Table 4). Table 4 gives the average \textit{k} values exhibited by males heterozygous for the \textit{SD}^{Ra} chromosome and, in turn, these second chromosomes, with and without the \textit{III}^{Ra} modifier. The results shown in Table 4 allow the following observations to be made: (a) the \textit{SD}^{Ra-1} and \textit{SD}^{Ra} factors show different distorting effects when heterozygous with the same second \textit{SD}^{+} chromosomes (columns 2 and 5), (b) the two groups of second \textit{SD}^{+} chromosomes from Ranna and Corato respond differently to the same \textit{SD}^{Ra} chromosome (column 5), (c) the \textit{III}^{Ra} chromosome induces or enhances segregation distortion when in-
sensitive second chromosomes (Ranna, $k = 0.54$) are present and also when rather sensitive second chromosomes (Corato and bw-7, $k = 0.77$) are present in heterozygosis with $SD^{Ra}$. The increase in $k$ values in both cases is the same, being about 0.8 (the ratio between the mean $k$'s obtained without and with $III^{Ra}$).

Considering (a) and (b) it may be concluded that the best hypothesis to explain the occurrence of the Segregation Distortion phenomenon is one which involves the interaction of $SD$ and $SD^+$ factors (as suggested in an earlier paper, Trippa et al. 1974). Thus the sensitivity of the $SD^+$ chromosome is not a peculiar property of itself. The classification of a second $SD^+$ chromosome as more or less sensitive would, in fact, depend on the type of $SD$ chromosome with which it is paired in the male. In particular, as far as the sensitivity of $SD^+$ wild chromosomes examined is concerned, the presence of $SD^+$ Ranna chromosomes in $SD^{Ra}/SD^+$ males causes a reduction in the amount of distortion to quite normal values compared with the distortion in $SD^{-1}/SD^+$ males (from $k = 0.66$ to $k = 0.54$), while $SD^+$ Corato and bw-7 chromosomes in $SD^{Ra}/SD^+$ males exhibit an increase in the segregation distortion (from $k = 0.64$ to $k = 0.77$; columns 2 and 5). Thus the $SD^+$ Ranna chromosomes appear to be insensitive to an $SD$ chromosome derived from the same population ($SD^{Ra}$), while those from Corato and the bw-7 line are as sensitive to an $SD$ chromosome derived from another population, as to $SD^{Ra}$.

Finally, as far as the explanation of the findings reported in (c) is concerned, two different models may equally well account for them: (1) $III^{Ra}$ factor may increase the distorting action of $SD^{Ra}$; (2) $III^{Ra}$ may increase the sensitivity of the second $SD^+$ chromosomes. The results do not enable us to choose between the two models.

(vi) **Diffusion of third chromosomes carrying modifiers of the $SD$ phenomenon similar to $III^{Ra}$ in the natural populations of Ranna and Corato**

To estimate the frequency in nature of third chromosomes with behaviour similar to that of the $III^{Ra}$ chromosome, 57 third chromosomes of the two populations of Ranna and Corato were tested. The amount of distortion shown by $SD^{Ra}/bw-5; III^+/st-5$ males was calculated. Four males for each wild third chromosome sampled were tested in individual matings.

The distribution of $k$ values is given in Fig. 2. Of the sample of 47 third Ranna chromosomes, 31 (i.e. 66%) have a $k$ value greater than 0.90 with a mean $k = 0.99$; the remaining 16 chromosomes (34%) have a $k$ value between 0.66 and 0.85 with a mean $k = 0.78$. Of the ten Corato chromosomes, seven have a $k$ value higher than 0.90 with a mean $k = 0.98$, while three have a $k$ between 0.75 and 0.85 with a mean $k = 0.80$. The frequency of third $SD$-modifier chromosomes (of the kind that enhances the amount of distortion caused by $SD^{Ra}$) thus seems very high among third chromosomes of the Ranna and Corato populations. Around 70% of the third chromosomes in the samples of the two natural populations tested show the ability to modify the $k$ value from 0.72 (which represents the ‘normal’ distortion value of the $SD^{Ra}$ factor alone) to about 1.00.
Fig. 2. Distribution of $k$ values of $SD^{Ra}/bw-5$ males carrying, in turn, each one of the 47 $III^+$ chromosomes of the Ranna population (◇) or one of the ten $III^+$ chromosomes of the Corato population (□). Each square represents the $k$ value exhibited by four males of identical genotype.

4. CONCLUSIONS

The $III^{Ra}$ factor is the first gene modifying Segregation Distortion found on the third chromosome. We consider it as a modifier since with the $SD$ chromosomes derived from Southern Italian populations it acts as a $k$ enhancer but with the $SD^{R-1}$ chromosome it behaves like a $k$ reducer (see Table 2).

Recently the Segregation Distortion phenomenon has been clarified somewhat. Nicoletti (1968), by analysing testes of $SD/SD^+$ males with the electron microscope, found several cysts with degenerating spermatids that could account for the abnormal recovery of $SD^+$ sperms. Tokuyasu et al. (1972) by the same kind of analysis, were able to show the dysfunction of one half of the spermatozoa (probably the $SD^+$) together with the failure of the spermatid individualization phase during spermiogenesis. However, the precise mechanism through which this dysfunction occurs is still unknown (Peacock & Miklos, 1973). This fact makes
it even more difficult to interpret how the $III^{Ra}$ factor intervenes in such a puzzling mechanism.

As regards the sensitivity of $SD^+$ chromosomes to $SD$, for the first time it has been possible to measure the amount of distortion exhibited by males carrying an original $SD$ chromosome ($SD^{Ra}$) and original $SD^+$ second chromosomes extracted from the same natural population (Ranna). Previously it had always been necessary to use a marker either on the $SD$ or on the $SD^+$ chromosome and in so doing crossovers were always obtained, thus jeopardizing in some way the $SD$-$SD^+$ system that was operating in nature. If it is considered, too, that some males tested also carried the $III^{Ra}$ chromosome, it can be said that for these males it has been possible to re-establish in the laboratory to a great extent the genetic composition of the males of the natural population of Ranna.

The data of Table 4 reveal that the second $SD^+$ Ranna chromosomes are virtually all insensitive to $D^{Ra}$, while the $SD^+$ Corato chromosomes appear to be quite sensitive. This result thus seems to confirm the hypothesis put forward by Hirai-zumi et al. (1960) on the basis of data obtained from laboratory populations, of a spontaneous occurrence and successive diffusion of second $SD^+$ chromosomes insensitive to the $SD$ factors present in the same population. Hence the different levels of sensitivity of the $SD^+$ chromosomes in the populations would appear to depend closely on the presence of the $SD$ factors and the distorting power of these factors and the time-length of their interaction with the $SD^+$ chromosomes. The average level of sensitivity to $SD^{Ra}$ of the $SD^+$ chromosomes from the Corato population may be interpreted as indirect evidence of the unique nature of the $SD$-$SD^+$ system as regards the occurrence of insensitivity in the $SD^+$ chromosomes.

The occurrence, in the Ranna population, of $SD^+$ chromosomes insensitive to the $SD^{Ra}$ action, could be interpreted as a force that would rapidly lead to the disappearance of the $SD$ phenomenon. On the other hand, the simultaneous presence in the Ranna population of enhancer chromosomes of the $III^{Ra}$ type with the observed high value (see Fig. 2) must be considered as a counteracting factor in favour of $SD^{Ra}$.

This series of observations enables us to interpret the $SD$ phenomenon in the populations as the outcome of the interaction of opposite selective forces that result in maintaining the frequency of $SD$ genes at the generally observed values of 0.01-0.10 (Sandler, Hirai-zumi & Sandler, 1959; Hirai-zumi et al. 1960; Mange, 1961; Greenberg, 1962; Hirai-zumi & Nakazima, 1965; Watanabe, 1967; Trippa et al. 1972). Particularly in the Ranna population, the maintenance of $SD$ chromosomes might be due to an equilibrium among factors that tend to spread the $SD$ chromosomes in the population (the $SD$ factors themselves and the $III^{Ra}$ enhancer) and factors that, on the other hand, tend to limit this spread: the low distortion power of the $SD$ chromosomes (Trippa et al. 1973), the presence of insensitive $SD^+$ chromosomes, the complete sterility of $SD^{Ra}$/$SD^{Ra}$ males and females, and the semisterility of $SD/SD^+$ males.

Nothing is yet known as to the biological significance of keeping $SD$ chromosomes at the frequencies observed in natural populations. It is, however, known
that with the mechanism of the preferential segregation of a chromosome it is possible to diffuse clusters of genes rapidly in populations, thus accelerating changes of gene frequency.

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