Atti Accademia Nazionale dei Lincei

Classe Scienze Fisiche Matematiche Naturali

Rendiconti

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Atti della Accademia Nazionale dei Lincei. Classe di Scienze Fisiche, Matematiche e Naturali. Rendiconti, Serie 8, Vol. **79** (1985), n.6, p. 189–195.

Accademia Nazionale dei Lincei

<http://www.bdim.eu/item?id=RLINA_1985_8_79_6_189_0>

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Atti della Accademia Nazionale dei Lincei. Classe di Scienze Fisiche, Matematiche e Naturali. Rendiconti, Accademia Nazionale dei Lincei, 1985.

SEZIONE III

(Botanica, zoologia, fisiologia e patologia)

Genetica. — *Microevolutive aspects of the* SD (Segregation Distortion) *phenomenon in Sardinian natural populations of* Drosophila melanogaster (*). Nota di ROSADELE CICCHETTI (**), GABRIELLA ARGENTIN (***), ADOLFO RANDACCIO (**) e BENEDETTO NICOLETTI (***), presentata (****) dal Corrisp. G. CHIEFFI (*****).

RIASSUNTO. — È stata studiata la frequenza dei cromosomi SD (Segregation Distortion) nelle popolazioni sarde di *Drosophila melanogaster*, mai analizzate precedentemente; a questo scopo è stato esaminato un campione di 1.108 maschi, provenienti da cinque diverse popolazioni. Contemporaneamente si è saggiata l'eventuale presenza sui cromosomi III di geni modificatori e l'interazione tra questi modificatori e i cromosomi II SD.

I risultati ottenuti hanno permesso di accertare un'alta frequenza di cromosomi II SD e la presenza di numerosi cromosomi III « aumentatori », in grado di intensificare il potere di distorsione del fattore *Sd*.

I dati raccolti ribadiscono che il fenomeno SD è ubiquitario, perchè presente anche in Sardegna, dove appare notevolmente diffuso; essi confermano inoltre che la penetranza e l'espressività del sistema SD è il risultato dell'interazione di diversi fattori.

INTRODUCTION

The Segregation Distortion (SD) phenomenon was observed for the first time in 1959 in a natural population of *Drosophila melanogaster* [1]; it consists of a second chromosome altered recovery among the progeny of heterozygous males for a II SD chromosome and homologous one, sensitive to it. The distortion degree for the second chromosome segregation is measured by the ratio between SD progeny and total progeny, and is denoted by « *k* ».

The *Sd* factor, the main responsible for the Segregation Distortion, has been located in 1972 at the locus 52.9 [2] and [3] in the region between 37 D2-D7 and 38 A6-B2 of the salivary chromosomes.

Through some experiments of genetic recombination it has been possible to detect other loci interested in the SD phenomenon expression [3-6]. Among these loci [6], there is the *Responder* locus (*Rsp*), in two allelic forms: the " sen-

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(****) Nella seduta del 14 dicembre 1985.

(*****) Lavoro eseguito con contributo MPI 40%.

sitive" (Rsp^{sens}), responsible in *cis* for the sensitivity to the *Sd* factor action, and the "insensitive" (Rsp^{ins}), responsible for the insensitivity of the chromosome on which it is located.

Subsequently, data have been accumulated to clarify some basal mechanisms for the SD phenomenon. Studies on the SD/SD+ heterozygous males fertility permitted, in fact, the rejection of "functional pole" hypothesis [7], proposing that of "sperm disfunction" [8, 9]. Electron microscope observations, performed [10, 11] on testes of SD/SD+ heterozygous males and control males, are completely consistent with the data on their fertility.

Later, various models [12-15] have been proposed in order to explain the mechanism through which SD makes disfunctional the gametes carriers of SD+ chromosome; all the models foresee a two loci interaction: an acting locus and a responding one.

The SD phenomenon has been studied from the population point of view, by analyzing the relationships between the II chromosome *Sd* locus and the modifiers carried by others chromosomes [15-19], as well as their influence on the SD chromosomes frequencies evolution in different natural populations. In particular, on the wild third chromosomes, Segregation Distortion modifiers ("enhancers") have been demonstrated [20, 21] which increase k values, characteristic of each SD chromosome. The SD chromosomes' frequencies, which theoretically should completely replace in the natural population the SD+ chromosomes, remain at values only rarely exceeding 5%. Several analyses of natural populations allow us to conclude that the factors of male and/or female sterility, the lethal and suppressor genes, the presence of II SD+ chromosomes insensitive to the *Sd* factor action [24] play an important role in order to oppose the SD chromosome's diffusion.

Finally, it appears that in natural populations there are different interacting forces, peculiar to every population, some of them favouring and some contrasting the SD chromosomes diffusion in the populations.

Materials and methods

1. *Populations and stocks*:

Drosophila melanogaster individuals were caught in five different sites of Sardinia: Ittiri in the north-western area; Dorgali and Tortolì in the middle-eastern area; Villasor in the South and Arborea in the middle-western area; samples for three of these populations were caught in two different years. All samples were collected in cellars storing fermenting grapes.

To test the frequency of II SD chromosomes and III modifiers chromosomes we employed the "5" laboratory isogenic line:

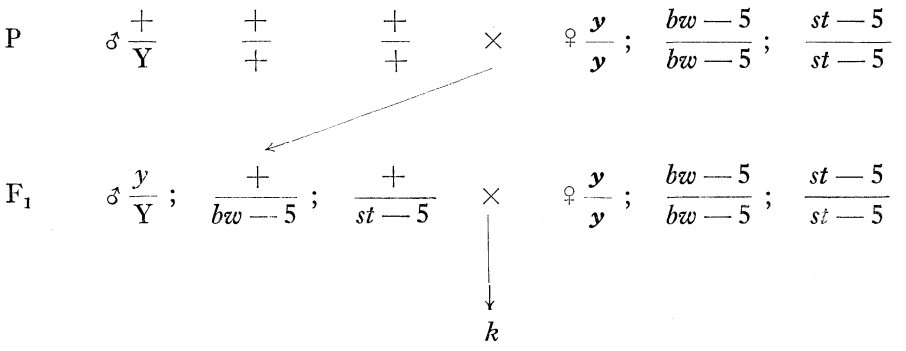
$$y/\mathrm{Y};\ bw-5/bw-5;\ st-5/st-5$$

isolated for its sensitivity to the *Sd* factor action.

An exhaustive description of this line markers is given in Lindsley and Grell, 1968 [26].

2. *Cross scheme*:

The stock y/Y; $bw-5/bw-5$; $st-5/st-5$ has been employed in the following cross, in order to estimate the II SD chromosomes frequency in wild males:



The distortion degree in II chromosomes segregation, has been measured by k values calculated—in the heterozygous males for an *Sd* factor and the *bw*-5 chromosome sensitive to it—as the ratio between SD progeny and total progeny. Usually, the k values fluctuate between 0.9 and 1.0; anyway, all the chromosomes exhibiting k values higher than 0.65 are considered as SD chromosomes (with more or less distortion power).

The F_2 heterozygous males for a II wild SD chromosome, bearing (y/Y; SD/*bw*-5; III/*st*-5) or not (y/Y; SD/*bw*-5; *st*-5/*st*-5) a III wild chromosome, have been crossed with the above described females.

On the basis of the k values calculated from the above cross, it has been possible to verify the eventual presence on the III chromosome of SD modifying factors.

Single males crosses were carried out at 24 °C $\pm$ 1 °C in vials containing standard food.

The k values were calculated twice: on the 14th and on 18th day from the cross date.

RESULTS AND DISCUSSION

The analysis of 1.108 males, coming from five Sardinian natural populations, heterozygotes for the II and III wild chromosome, made it possible to set the II SD chromosomes frequency and the III enhancer chromosomes presence.

The Table 1 shows the distribution of the SD chromosomes found in the different populations and their *k* values: it is evident that the SD chromosomes are present in all examined populations.

Their frequency (reported in Table 1, last column) is very high, exceeding 10%, except for the Villasor-81 and Tortolì-81 populations.

TABLE I

Frequency and features of the SD *chromosomes found in the* Drosophila melanogaster *natural Sardinian populations. The chromosomes have been grouped in three k series : low* (0.66-0.75), *medium* (0.76-0.85) *and high* (0.86-1.0) *distortion power.*

Populations	n° of examined chromosomes	n° of SD chromosomes with *k* values between:			Total SD chromosomes	SD chromosomes frequency (%)
		.66-.75	.76-.85	.86-1.0		
Arborea-81 . . .	186	13	10	0	23	12.4
Arborea-83 . . .	80	11	9	6	26	32.5
Dorgali-81 . . .	165	22	15	6	43	26.1
Dorgali-83 . . .	40	8	3	2	13	32.5
Ittiri-81	170	10	5	5	20	11.8
Ittiri-83	118	15	3	1	19	16.1
Tortolì-81 . . .	190	9	2	2	13	6.8
Villasor-81 . . .	159	7	1	1	9	5.6

Many studies (1,21-23) on American, Japanese, Australian and Italian *Drosophila melanogaster* populations, report more often an SD chromosome frequency of 5%, sometimes reaching 10% in the Italian populations. The 5% frequencies, mentioned in literature, are consistent with the theoretical ones obtained by Crow (24) through computer simulation of a theoretical population evolution.

According to Crow, in a population initially Sd^+ Rsp^{sens}, a competition arises among: 1. the already existing Sd^+ Rsp^{sens} chromosomes; 2. the *Sd* Rsp^{ins} chromosomes, later appeared in the population; 3. the Sd^+ Rsp^{ins} chromosomes, insensitive to the *Sd* factor and probably derived from the crossing-over between the first two chromosomes.

The competition model results in a spiral progress, foreseeing, in different moments, the SD or insensitive SD^+ chromosomes prevailing, until the achievement, after less and less extended cycles and after almost 1.000 generations, of the equilibrium, at 4–5% values for the SD chromosomes.

The model also implies some modifiers (" softeners ") which " soften " the SD phenomenon, like male and/or female sterility factors and " suppressor " genes associated with the *Sd* factor.

In consideration of the SD chromosomes high frequency in the examined Sardinian populations, we can suppose, referring to them, the absence of these " softeners " factors and/or of Sd^+ Rsp^{ins} chromosomes.

It should be stressed (Table I) that the SD chromosome frequencies are lower in the populations examined in 1981, than in those of 1983. In order to explain these results—and the SD chromosomes high frequencies—we can hypothesize that the SD chromosome has recently appeared in the Sardinian populations.

The samples collected in 1983 could then derive from populations in which (lowering the Sd^+ Rsp^{ins} chromosomes frequency because of their actual uselessness) the SD chromosomes are showing a further, high increase. On the contrary, the 1981 populations were probably in the Sd^+ Rsp^{ins} chromosome's highest frequency moment and of SD chromosome lowest frequency.

It could, therefore, be a matter of populations which have not yet achieved their equilibrium.

We also analyzed the progeny of SD chromosomes heterozygous males, crossed with *y*; *bw* — 5; *st* — 5 females, in order to point out the III modifier chromosome's eventual presence.

The SD chromosomes to be tested, in the presence or absence of the III wild chromosome (see M. and M.), were selected with a different distortion power: high, medium, low. We studied a total of 84 genotypes coming from the eight populations and distributed all over the three *k* values classes.

The comparison between the *k* values in presence and in absence of the III chromosome, clearly shows that in many cases the *k* values lowered signi-

TABLE II

Frequency of III chromosomes modifiers of the Segregation Distortion in the examined natural Sardinian populations of Drosophila melanogaster.

	Populations	n° examined III chromosomes	Distorted segregation		Modifiers III chromosomes frequency (%)
			Steady	Modified	
1981	Arborea	8	4	4	50.0
1981	Dorgali	20	8	12	40.0
1981	Ittiri	12	5	7	41.7
1981	Tortolì	6	—	6	0
1981	Villasor	8	3	5	37.5
1983	Arborea	14	6	8	42.9
1983	Dorgali	8	—	8	0
1983	Ittiri	8	2	6	25.0
	Total	84	28	56	

ficantly in the III chromosomes absence. This effect must, obviously, be attributed to the presence of III chromosomes carriers of factors able to modify, by increasing it, the *k* value.

Table II reports the " enhancer " chromosomes presence percentages in the different Sardinian populations.

In the Tortolì-81 and Dorgali-83 populations we found no enhancer factors; anyway, we cannot exclude that it happens for a genetic drift effect, on account of their frequency.

All these data, globally, encourage our conclusion that the tested Sardinian populations—as they show high SD chromosomes frequencies—are still far from equilibrium conditions.

From our observations as a whole it is, however, confirmed that in *Drosophila melanogaster* natural populations the SD chromosomes frequencies evolution and the equilibrium values achievement (about 5%), derive from the different contrasting forces' interaction.

Between them, there is the presence of III enhancer chromosomes which promote the *Sd* factors diffusion.

If these latter's frequency should appears as ubiquitous and with frequency values varying enough, the microevolutive aspects of the SD system, should be re-examined.

Acknowledgments. — The Authors thank Dr M.G. De Rossi for her help in analyzing the 1983 populations.

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