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TEMPERATURE-DEPENDENT GONADAL HYBRID DYSGENESIS IN *Drosophila willistoni*

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ABSTRACT

Temperature-dependent gonadal dysgenesis was shown to occur in the progeny of both inter- and intrastrain crosses involving two populations of *Drosophila willistoni*, one of which was an old laboratory stock, and the other, freshly collected from a natural population. We propose that the phenomenon observed was caused by the mobilization of transposable elements, as occurs in several other *Drosophila* species.

INTRODUCTION

In *Drosophila melanogaster*, *P* element transposition leads to a syndrome of aberrant genetic traits known as P-M hybrid dysgenesis (Kidwell *et al.*, 1977; Bingham *et al.*, 1982; Sved, 1987). Early investigations identified two alternative states, or cytotypes, which are determined by a combination of chromosomal and cytoplasmic factors that allow or repress transposition (Engels and Preston, 1979; Engels, 1983). The M cytotype is permissive and is characteristic of *D. melanogaster* strains that are completely devoid of *P* elements, i.e., M strains. The P cytotype is non-permissive, and is characteristic of P strains, i.e., those carrying many copies of autonomous *P* elements. More recent studies have revealed considerable variation in the inheritance of these regulatory states (Kidwell, 1985; Simmons *et al.*, 1987, 1990). Since these states are tissue-specific, they are confined to the germline of the progeny produced by certain interstrain crosses. Dysgenic traits include temperature-sensitive sterility and reduced viability and fecundity, which in turn lead to a reduction in organism fitness.

In addition to the P-M system, other families of transposal elements are capable of promoting hybrid dysgenesis. Of these, I-R (Bucheton *et al.*, 1976, 1984, 1986) and H-E (Blackman *et al.*, 1987; Yannopoulos *et al.*, 1987) have been particularly well studied. Lozovskaya *et al.* (1990) also demonstrated the occurrence of hybrid dysgenesis syndrome in *D. virilis* when the male parent came from a long-established laboratory strain. This hybrid dysgenesis system was attributed to the *Ulysses* transposable element and, more recently, to at least four transposable elements (Petrov *et al.*, 1995). A similar situation, characterized by gonadal dysgenesis and the presence of chromosomal aberrations, was also recently reported by Torti *et al.* (1994) for the medfly *Ceratitis capitata*.

Drosophila willistoni is a Neotropical species considered a paradigm for evolutionary studies (Dobzhansky and Powell, 1975; Ehrman and Powell, 1982; Cordeiro and Winge, 1995). Over the last decade, we have studied this species using genetic, ecological and evolutionary approaches (Valente and Araújo, 1985, 1986a,b, 1991; Valente *et al.*, 1993).

In an effort to identify the causal agents of the reduced viability of hybrid strains produced by crossing laboratory and natural populations of *D. willistoni*, and to analyze chromosomal inversions, we examined a series of traits that resembled previously studied gonadal dysgenesis syndromes (Kidwell *et al.*, 1977; Blackman *et al.*, 1987; Lozovskaya *et al.*, 1990). In this paper, we report the occurrence and some characteristics of a dysgenesis-like phenomenon in *D. willistoni*.

MATERIAL AND METHODS

Fly stocks and culture conditions

Two stocks of *D. willistoni* (WIP-11A and 17A2) were used. 17A2 was a wild-derived *D. willistoni* stock that had been in the laboratory since 1991 and was established from a natural population collected in a wilderness area (Eldorado do Sul, southern Brazil, 30°05'S-51°39'W). WIP-11A was a subculture of an old laboratory stock (WIP-4), originally collected in the "caatinga" desert near the city of Salvador (12°54'S-38°19'W), in northeastern Brazil and had been kept in the laboratory for 30 years. All cultures were maintained by mass matings on standard *Drosophila* culture media (Marques *et al.*, 1966).

Gonadal sterility tests

Gonadal sterility was scored in the F1 progeny of inter- and intrastrain crosses involving WIP-11A and 17A2 flies. Each cross was performed by mass mating 30 virgin

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females with 30 virgin males at 18°, 25° and 29 ± 1°C. At the onset of eclosion, F1 progeny was transferred to fresh culture vials and aged for five days at 25 ± 1°C. F1 males and females were then dissected in a drop of Ephrussi and Beadle (1936) saline solution. The frequency of gonadal sterility was calculated by dividing the number of dysgenic gonads by the total number of gonads.

Chi-square test was performed to assess the statistical significance of the differences among the crosses, sexes and temperatures, relative to the number of dysgenic gonads observed in the F1.

RESULTS

An elevation in environment temperature during mating increased the levels of gonadal dysgenesis in *D. willistoni* hybrid progenies. Dissection of the reproductive tract of both male and female F1 progeny from the four possible crosses involving WIP-11A and 17A2 revealed gonadal underdevelopment in several cases (Figure 1 and Table I). Although not shown, unilaterally dysgenic gonads were also observed.

In addition to complete ovarian reduction in the F1 progeny, some intermediate forms consisting of partially reduced ovaries containing only one or a few ovarioles were also detected. However, these intermediate phenotypes were excluded from the calculations of ovarian dysgenesis so as to avoid superestimation of the phenomenon as a result of the possible loss of parts of these soft, small structures during handling. Significant gonadal dysgenesis occurred at 29°C in all the crosses performed, with exception of the WIP-11A intrastrain cross. In this case, the levels of gonadal dysgenesis were trivial, occurring at nearly equal frequencies at all three temperatures at which the flies were mated and the F1 progeny developed. The statistical significance of the differences in gonadal dysgenesis levels is summarized in Table II.

The percentages of F1 dysgenic gonads were considerably higher in the second and third crosses (Table I) at 29°C. At 18° and 25°C, the levels of sterility in these crosses were very similar to those observed in the WIP-11A intrastrain cross. Considering all crosses, males and females were not equally affected. At 29°C, male descendants of the three last crosses showed similar proportions

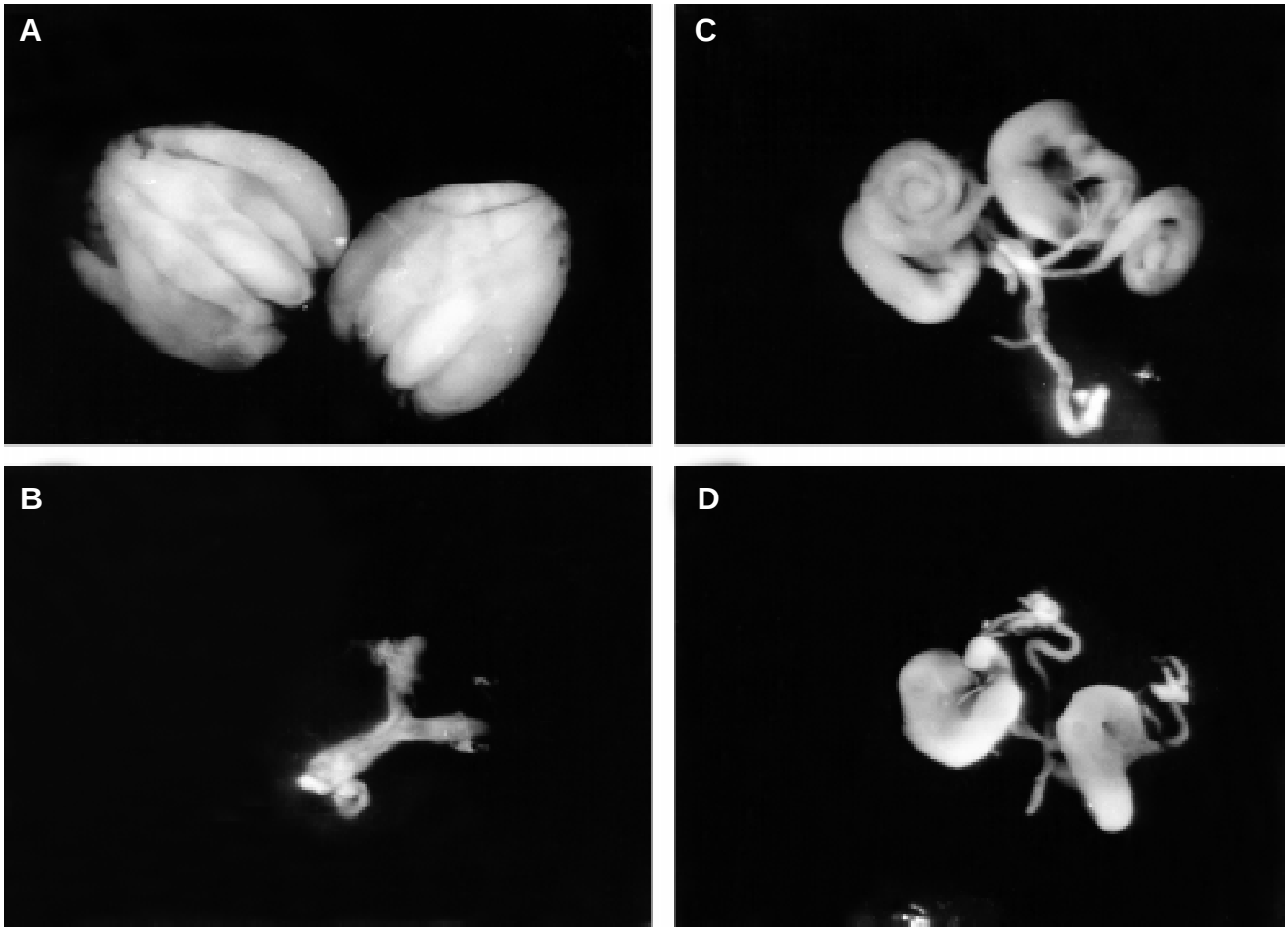

Figure 1 - Extreme phenotypes suggestive of gonadal dysgenesis in *D. willistoni*. A) Normal ovaries; (B) Bilaterally dysgenic ovaries; (C) Normal testes; (D) Bilaterally dysgenic testes.

Table I - Gonadal dysgenesis (GD) sterility in female and male F1 progeny of *D. willistoni* at different temperatures.

Cross type	Temperature	F1 Females		F1 Males	
	^{0}C	% GD	N	% GD	N
WIP-11A females x	18	2.2	184	1.7	174
WIP-11A males	25	1.1	174	1.2	160
	29	3.4	178	2.4	126
WIP-11A females x	18	0.9	216	2.8	216
17A2 males	25	0.0	180	2.4	168
	29	26.1	180	20.6	102
17A2 females x	18	0.0	190	2.0	196
WIP-11A males	25	1.2	168	0.0	156
	29	18.8	170	14.0	100
17A2 females x	18	2.2	180	2.5	162
17A2 males	25	5.5	182	1.7	172
	29	16.3	172	13.1	122

N = Number of ovaries (females) or testes (males) analyzed.

Table II - Contingency table for testing the effects of female and male gonadal dysgenesis (GD) at 29ºC.

Cross type	F1 Females		F1 Males	
	GD	NG	GD	NG
WIP-11A females x WIP-11A males	6*	172	3*	123
WIP-11A females x 17A2 males	47**	133	21**	81
17A2 females x WIP-11A males	32	138	14	86
17A2 females x 17A2 males	28	144	16	106

$\chi^2 = 35.567$, P = 0.001 (significant heterogeneity among females of the four crosses). $\chi^2 = 18.687$, P = 0.001 (significant heterogeneity among males of the four crosses). * Residual analysis indicating a lack of GD. ** Residual analysis indicating an excess of GD. $\chi^2 = 5.651$, P = 0.059 (nonsignificant variation in female GD among the last three crosses). $\chi^2 = 2.670$, P = 0.263 (nonsignificant variation in male GD among the last three crosses). GD = number of dysgenic gonads, GN = number of normal gonads.

of gonadal dysgenesis, whereas the females tended to have slightly higher sterility rates compared with males in the second and fourth crosses.

These results (Table I) suggest that gonadal sterility in *D. willistoni* is a temperature-dependent phenomenon, but different from the *P*-induced response in *D. melanogaster*. However, temperature alone did not induce the gonadal alterations observed since WIP-11A flies had similar levels of dysgenesis at all developmental temperatures and F1 female descendants of the cross of 17A2 males and females reared at 25°C also had an elevated frequency of dysgenic gonads. Thus, *D. willistoni* hybrid sterility is not independent of the cross direction (Table I).

In addition to gonadal dysgenesis, certain altered phenotypes were also observed in the offspring of WIP-11A and 17A2 crosses kept at 29ºC. These were similar to those of certain *P*-induced *D. melanogaster* mutants described by Lindsley and Zimm (1990). In our case, the low viability of the individuals affected meant that it was impossible to maintain them long enough to carry out crosses to differentiate these phenotypes from microorganism-promoted aberrant phenotypes and/or heat-induced phenocopies. However, we think that such phenotypes deserve attention because of their similarity to *P*-induced mutants. Specifically, they were similar to i) *Beadex*, based on their diminished 5th wing vein and/or the presence of "bubbles" or excisions at their edges (Figure 2B,C,E), ii) *held-up*, based on the vertical position of the wing insertion (Figure 2I), iii) *Notch*, based on shrinkage of the 5th wing vein or on the enlarged veins, with deltae in the extremities, and/or in the serrated edges. Sparse thoracic hairs with irregular distribution (Figure 3B), the presence of an intermediate thoracic furrow (Figure 3D) and some changes in the color, size, shape and arrangement of the eye ommatidia were also observed (Figure 4B,C and D), and iv) *extra-eye*, based on the duplication of head structures, that in extreme cases seemed to form supernumerary eyes (Figure 3D).

DISCUSSION

This study is the first report of gonadal dysgenesis in *D. willistoni*, although no causal relationship between the observed gonadal dysgenesis and transposon-homologous sequence was established. The alterations observed in the reproductive tract of *D. willistoni* and the external phenotypes were morphologically similar to those of *P*-induced traits in *D. melanogaster.*

The *P*-related gonadal dysgenesis in *D. melanogaster* and the phenomenon observed in *D. willistoni* were also similar in that both were triggered by high temperatures, although this temperature was higher in *D. willistoni* (>25ºC) than in *D. melanogaster* (23ºC) (Kidwell, 1983). While this phenomenon may have been caused by transposable elements, it may also be related to the hotter habitat (hot, humid Neotropical forests) of *D. willistoni*, in which case the higher triggering temperature may be a species adaptation to avoid high gonadal sterility rates. Females and males were both affected, although females tended to have slightly higher sterility rates at 29ºC.

Contrary to *P*-hybrid dysgenesis in *D. melanogaster,* the percentage of gonadal dysgenesis varied in the progeny of WIP-11A females x 17A2 males, 17A2 females x 17A2 males and 17A2 females x WIP-11A males. In the first two crosses, the male parents were from the same population (17A2) and would therefore be expected to contribute approximately to the same conditions for establishing gonadal dysgenesis.

Both *D. willistoni* strains studied have DNA sequences homologous to complete *P*-elements in *D. melanogaster*, as

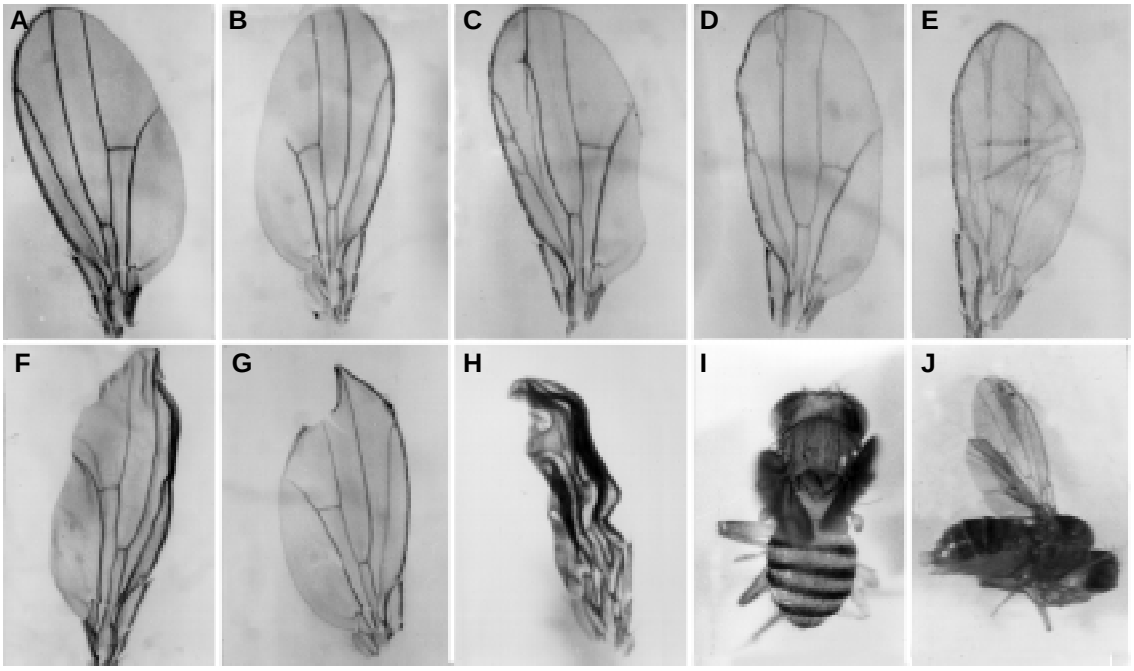

Figure 2 - Altered wing phenotypes encountered in the progeny of a WIP-11A x 17A2 cross of *D. willistoni* at 29ºC. (A) Wild wing; (B-E) irregular or incomplete vein formation; (E-G) incompletely expanded wings; (C, H) border of wings with reentrances; (I) abnormal wing position; (J) fly with different size wings.

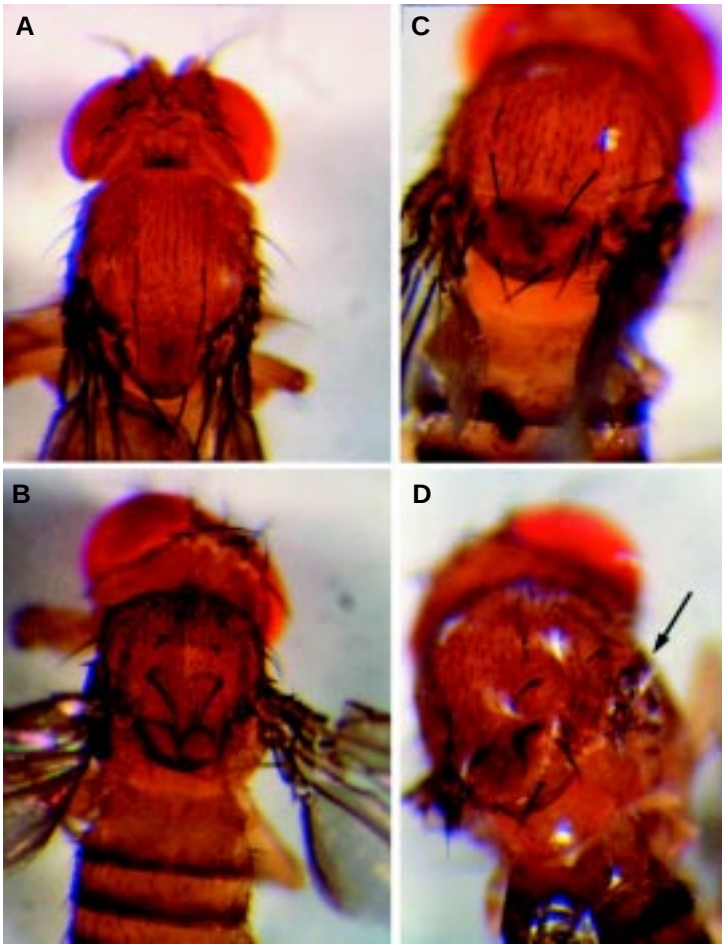

Figure 3 - Altered thorax and bristle phenotypes in the progeny of a WIP-11A x 17A2 cross of *D. willistoni* at 29ºC. (A) Wild phenotype; (B) abnormal bristles; (C) duplication of bristles (arrow); (D) altered bristles and thorax with a furrow (arrow).

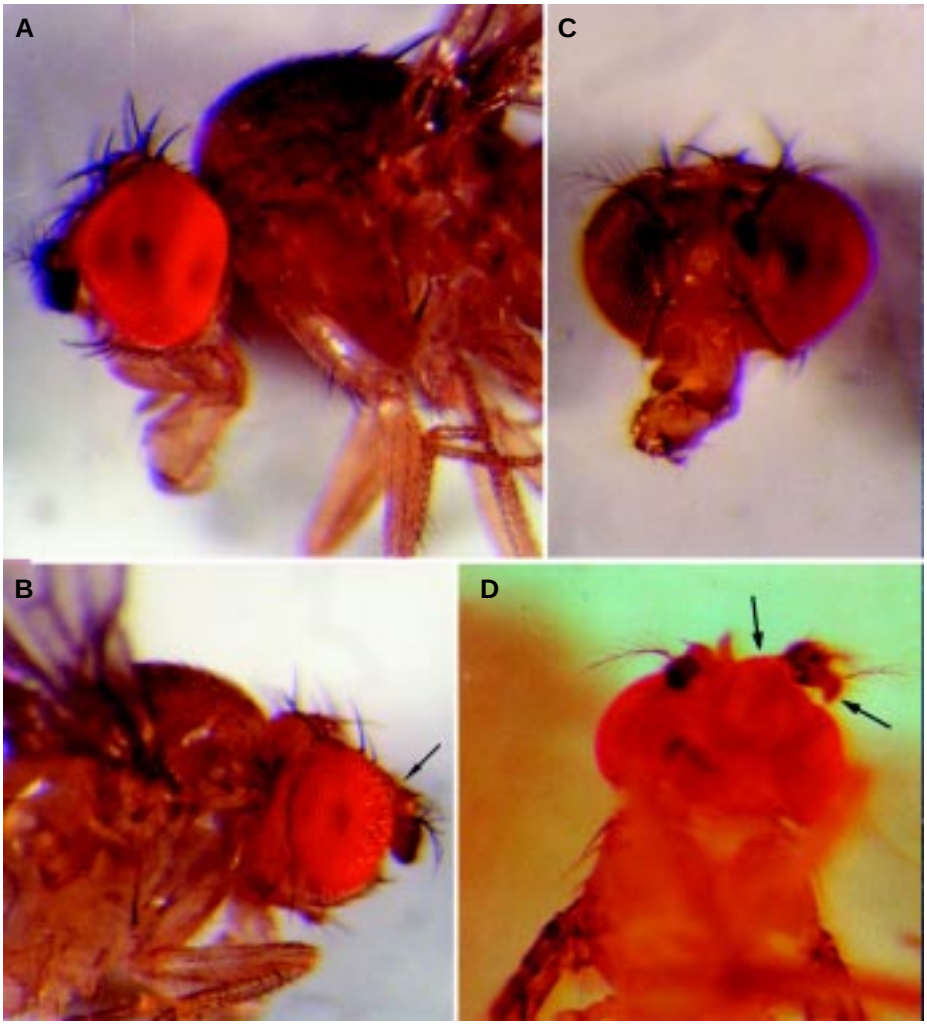

Figure 4 - Altered head and eye phenotypes in the progeny of a WIP-11A x 17A2 cross of *D. willistoni* at 29ºC. (A) Wild phenotype; (B) abnormal ommatidia (arrow); (C) different colored eyes; (D) unilateral duplication of head structures, including an extra eye and antenna (arrows).

revealed by Southern blots (Regner *et al.*, 1996), although their genomic positions differ sharply. *In situ* hybridization has shown that while 17A2 has several hybridization signals in the euchromatic arms, the signals in WIP-11A are concentrated mainly in the chromocenter.

Further studies on the regulation of *P* transposition need to be performed in species other than *D. melanogaster* before concluding whether this element is involved in the phenomenon described here.

In *D. nebulosa*, a close relative of *D. willistoni*, the *P* insertion site appears to be a complex sequence that is repeated three times in this species' centromeric heterochromatin (Lansman *et al.*, 1987). This finding led these authors to suggest that some species have *P* element insertional "hotspots" in their heterochromatic DNA such that capture in the heterochromatin of the chromocenter could prevent the mobilization of several transposons. Transposable element invasions and re-invasions would then result in periods of dysgenic effects related to the amount of complete elements present in the genome, followed by the accumulation of mobile sequences in heterochromatic hotspots and a reduction in dysgenic traits. Genomic location is possibly a strategy developed to control the effects of putative transposable element activity.

Clark *et al.* (1995) recently reported four major *P* element subfamilies in the *D. willistoni* and *D. saltans* species groups, indicating that the coexistence of more than one member of these subfamilies in the same genome is possible. The implications of such differences on the crossability of different populations remain unknown.

One additional feature that deserves mention is the continuum of phenotypes observed between the two extremes (normal and dysgenic) at 29ºC. These intermediate phenotypes were very rare in *D. melanogaster* (their occurrence was limited to crosses performed at temperatures below 25ºC) but frequent in *D. simulans* (Daniels *et al.*, 1989), and have also been reported for *D. mauritiana* (Montchamp-Moreau *et al*., 1991). However, *D. simulans* and *D. mauritiana* do not bear *P* elements under natural conditions; *P* elements were introduced into each either

by genetic transformation experiments or by interspecific crosses. Daniels *et al*. (1989) suggested that the expression of sterility in *D. simulans* tended to be more depressed at lower temperatures than *D. melanogaster* in similar conditions. Considering the results for *D. willistoni*, we suggest that a similar phenomenon promoted by the putative transposable element involved may occur in the latter species.

Previous studies on the origin of *P* elements suggested that these sequences are a new acquisition of *D. melanogaster,* but were harbored for a longer time in *D. willistoni* (Kidwell, 1983; Daniels and Strausbaugh, 1986; Daniels *et al*., 1990a; Houck *et al*., 1991). The latter species may have had to deal with *P* activity for a longer period than *D. melanogaster* which may not have had enough time to adapt to the effects of this elemental system. Indeed, Daniels *et al*. (1990a) suggested that hybrid dysgenesis may be different in these two species. Although our results do not constitute a thorough investigation of hybrid dysgenesis in *D. willistoni*, the results of gonadal sterility tests showed common basic features, suggesting that the differences between these two species may be more of a quantitative than a qualitative nature. Several types of evidence have led some to the conclusion that *P* element expression is influenced by certain host properties (Rio *et al*., 1986; O'Brochta and Handler, 1988; Rio and Rubin, 1988; Rio, 1990). These properties, however, do not seem to differ much between *D. willistoni* and *D. melanogaster*, since a complete *P* element from *D. willistoni* can promote transposition in *D. melanogaster* (Daniels *et al*., 1990a), and a complete *P* element of *D. melanogaster* can be excised from *D. willistoni* (O'Brochta and Handler, 1988).

Other transposable element families, such as *hobo*, are also able to promote gonadal dysgenesis similar to the *P* element in *Drosophila* (Blackman *et al*., 1987). However, *D. willistoni* has been reported to be devoid of *hobo* (Daniels *et al*., 1990b), thus weakening the possible involvement of *hobo* in the phenomenon we observed. However, we have recently reported weak *hobo* hybridization signals in the genome of some *D. willistoni* strains (including those here studied) (Loreto *et al*., 1998), that deserve future study.

It is possible that an unknown element harbored exclusively by some natural populations of *D. willistoni*, and/or that several unrelated transposable elements, as found in *D. virilis* (Petrov *et al*., 1995), could be responsible for the generation of phenomena such as those described here.

Other conditions, such as the presence of endocytobionts like the rickettsia *Wolbachia*, can promote intraspecific reproductive incompatibility. When this microorganism is present in one population and not in another, either no hybrids or fewer progeny are produced. This phenomenon affects females from one reciprocal cross, but not those from the other (for a review, see Hoy, 1994). This possibility is unlikely to explain the data reported here.

Considering this scenario, and the possible evolutionary consequences of the intraspecific incompatibility described here as well as the fact that environmental stress is capable of promoting transposon mobilization (Jouan-Dofournel *et al.*, 1996), we believe that exploration of the role of transposable elements as a source of genetic variability in natural populations of Neotropical *Drosophila* species has only just begun.

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RESUMO

Disgenesia gonadal dependente de temperatura foi encontrada na prole tanto de cruzamentos intra como interlinhagens, envolvendo duas populações de *Drosophila willistoni*. Uma delas é derivada de um velho estoque de laboratório e a outra, de uma amostra recentemente coletada de uma população natural. Tal fenômeno não havia ainda sido descrito em *D. willistoni* e nós sugerimos que a disgenesia gonadal encontrada seja causada por elementos transponíveis, como ocorre em muitas outras espécies de *Drosophila.*

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