

References: Bucheton et al. 1976, *Heredity* 36:305-314; Bucheton and Picard 1978, *Heredity* 40:207-223; Kidwell 1979, *Genet. Res.* in press; Pelisson 1978, *Genet. Res.* 32:113-122; Pelisson and Picard 1979, *Genetics* in press; Picard 1976, *Genetics* 83:107-123; Picard 1978, *Molec. Gen. Genet.* 164:235-247; Picard et al. 1977, *Biol. Cell.* 29:89-98; Picard et al. 1978, *Genet. Res.* in press.

Brncic, D. and Budnik, M. Universidad de Chile, Santiago, Chile. Colonization of *Drosophila subobscura* Collin in Chile.

In February 1978, Mr. H. Fenner of our laboratory collected for the first time in Chile *Drosophila subobscura* Collin, in an orchard near Puerto Montt (S.41°30'). A laboratory stock was established and our determination was con-

firmed by crosses with stocks from Bilbao (Spain) provided by Prof. Antonio Prevosti (Barcelona), and from Norway (Stock TX 2361-01 provided by the Univ. of Kansas). F₁ and F₂ were fully fertile in both tests. Photomicrographs of the giant salivary gland chromosomes of the larvae from the Chilean stock were studied by Prof. A. Prevosti (Barcelona), who kindly informs us that the band sequences correspond most probably to those observed in the Western Mediterranean Europe region (Meridional Spain), and in the Mediterranean coast of North Africa (Marruecos and Tunis). This first observation of *D. subobscura* in Puerto Montt (Chile) is significant, because *Drosophila* have been collected there practically every summer for the last 25 years.

Eight months later (November and December 1978), we collected *D. subobscura* in large numbers in the following places in Chile, that correspond to a north-south gradient of about 1200 km: Santiago (S.33°30'), Lake Rapel (S.34°15'), Talca (S.35°26'), Chillan (S.36°36'), Salto del Laja (S.37°10'), Los Angeles (S.37°28'), Pucon (S.39°15'), Valdivia (S.39°50') and Puerto Montt (S.41°30'). Most collections were made utilizing fermenting banana traps placed in orchards or gardens, with the exception of the Salto del Laja and Pucon localities in which the baits were placed in small natural forests of *Notofagus*. In addition, a few flies were collected in a fruit-vegetable store in Chillan City by sweeping the net over the fruits. In none of the above mentioned places was *D. subobscura* recorded before, indicating that it represents a newly introduced species coming most probably from the Palearctic zone. We have no information of the existence of the species in other places of Neotropical zone or in the Nearctic.

The quantitative data of the collections seems to indicate that the rapid invasion of *subobscura* has displaced some "domestic" species, particularly *D. simulans*, which was a very abundant species all over the central and south-central parts of Chile, and has now become a relatively rare species.

Stocks of *D. subobscura*, originated from the above indicated places, were sent to Prof. A. Prevosti of Barcelona for further research. [The authors would like to thank Mrs. Hertha Fenner and Mr. Gonzalo Gajardo from our Department and Prof. Eduardo del Solar from the Univ. Austral (Valdivia - Chile), who collected the flies at Puerto Montt, Lake Rapel and Valdivia respectively, supported by grants from PNUD/UNESCO (Proyect RLA 76/006) and Univ. of Chile (Proyect B 027 - 784).]

Bryant, M. L. and M. R. Murnik. Western Illinois University. The mutagenicity of herbicides in *Drosophila melanogaster*.

Our laboratory is interested in the potential mutagenicity of herbicides. Trifluralin (Eli Lilly Company) is an herbicide commonly used for weed prevention in soybean crops. It is a yellow liquid, miscible with water. Accord-

ing to several investigators (Andersen, Leighty, and Takahashi 1972; Shirasu, 1975), trifluralin is not mutagenic in any of four different microorganisms. Since tests in microorganisms test only for point mutations, we decided to test this herbicide in *Drosophila*. Male, wild type Oregon-R flies, fed as larvae 0.01 trifluralin (w/w in modified Carpenter's medium) were mated with virgin Basc females. The concentration of trifluralin used was the highest dose not toxic to the developing flies. The treated group produced 0.09% sex-linked recessive lethals, while the control group had 0.12% of these mutations. Thus, the results of these tests indicate that trifluralin does not produce sex-linked recessive lethals in *Drosophila*.

For a more complete testing program, we also used a chromosomal assay test system. Both larval fed and adult fed flies were used. Adult male flies (0-4 hours old) were starved for four hours, and then allowed to feed for 24 hours on 0.02% trifluralin (v/v) in a 1% sucrose (w/v) solution. The herbicide concentration used is about the LD₁₀ dose for adults. Males of the genotype $y^2 w^1 ct^6 f/sc^8 y^+ Y B^S$ which survived the feeding were mated with two virgin y/y females for two days each. Live transfer of males was made for five subsequent broods. Progeny of these matings were scored for loss, breakage, and nondisjunction of the X and Y chromosomes. There was no significant difference between the treated and control groups for any of the aberrations scored. However, the actual number of aberrations scored in each category was always higher in the treated group.

A larval fed group of $y^2 w^1 ct^6 f/sc^8 y^+ Y B^S$ males were mated within 24 hours of eclosion to y/y virgin females, and their progeny scored for aberrations. The results are in Table 1. The rate of XXY nondisjunction (0.12%) in the treated group is significantly different ($P < 0.01$) from that of the control group (0.04%). Data were analyzed according to the tables of Kastenbaum and Bowman.

Table 1. Chromosomal assay - larval feeding

	XXY nondisjunction		X or Y loss		N
	Number	Percent	Number	Percent	
Treated	16**	0.12	22	0.17	13152
Control	11	0.04	46	0.16	28767

** $p < 0.01$

the mitotic divisions of development. The larval fed treated group had significantly more mosaics than the control group at the 95% level.

In conclusion, although trifluralin does not appear to produce point mutations in microorganisms or *Drosophila*, it does appear to induce chromosome breakage and nondisjunction in *Drosophila*. The mechanism of chromosome aberration appears to be spindle apparatus malformation (Lignowski and Scott). In this case, larval feeding of flies was more efficient in demonstrating chromosome aberrations than was adult feeding.

References: Andersen, K.J., E.G. Leighty and M.T. Takahashi 1972, *J. Agr. Food. Chem.* 20:649-656; Kastenbaum, M.A. and K.O. Bowman 1970, *Mutation Res.* 9:524-526; Lignowski, E.M. and E.G. Scott 1972, *Weed Science* 20:267-270; Shirasu, Y. 1975, *Environ. Quality Safety* 4: 226-231.

Bulyzhenkov, V.E. and V.I. Ivanov. Institute of Medical Genetics, Moscow, USSR. Expression of Antennapedia⁵⁰ in triploid *Drosophila melanogaster*.

Dominant homozygous lethal homoeotic mutations of Antennapedia (Antp; 3-48.) locus cause transformation of proximal antennal segments into respective leg elements. The allele-specific interaction of Antp alleles with some other homoeotic genes in transforming the antennae (Bulyzhenkov, Ginter and Ivanov, 1975) suggested the mutations to be of missense type; thus the determinative products of mutant as well as of normal alleles should appear in cells of the antennal imaginal discs. In this case, a certain influence might be expected of an extra dose of Antp⁺ on the phenotypic expression of mutant alleles of this locus. In search for such influence the homoeotic transformation of antennae in triploid flies having in their genotype two doses of normal and a single dose of mutant allele was studied. Triploid females with normal third chromosomes and marked X-chromosomes ($z/z/FM7(y^{wa} lz B); +/+$) were crossed with diploid $FM7/Y; Antp^{50}/T(2;3)Xa$ males. The markers employed allowed us to distinguish between triploids, intersexes, and diploids. In preliminary tests complete penetrance of $T(2;3)Xa$ was shown. To estimate the rate of homoeotic transformation of antennae, the aver-

Another aberration recently scored in our laboratory is mosaicism. Larval fed males which produced progeny for the chromosomal assay also produced progeny of the genotype $+/B^S$, indicating that the long arm of the Y chromosome was broken and lost in one of