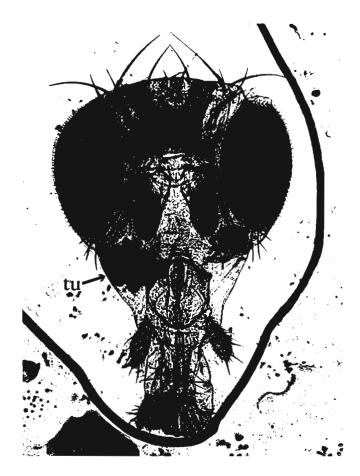
(acetone at 5.00 ppm concentration) and normal controls as well as in the chemically treated series.

When these data were statistically tested, by analysis of variance, it was found that these concentrations of Malathion were non-mutagenic to D. melanogaster. For the data on sex-linked recessive lethals, the 2x2 contingency test (Ehrenberg 1977) gave the same results. Consistent with this non-mutagenic effect of the insecticide, Mohn (1973) reported that Malathion did not induce mutations in E. coli (for 5-methyl tryptophan resistance) and Huang (1975) reported that it did not induce chromosomal aberrations in human hematopoeitic cell lines, though it inhibited their growth. Murthy (1979) reported non-induction of gene conversion in yeast and Degraeve et al. (1980) reported that Malathion neither induced forward mutations in yeast nor dominant lethals in mice. Contrarily, Wild (1975) reported chromosome breaks in humans who had had acute intoxication of this chemical; Sylianco (1978) and Chen et al. (1981) reported that Malathion induced micronuclei in mice and sister chromatid exchanges and cell cycle delay in chinese hamster cultured cells, respectively. Shiau et al. (1980) have also reported an increase in the induction of mutations in Bacillus subtilis and Salmonella typhimurium when they were treated with Malathion with 59 fraction.

In view of these highly contradictory results, more convincing investigations are needed to know the exact genotoxic potential of Malathion though it has been found to be non-mutagenic in D. melanogaster at the concentrations used.

References: Chen, H.H., J.L. Hsueh, S.R. Sirianni and C.C. Huang 1981, Mutation Research 88:307-316; Degraeve, N., J. Gilot-Delhalle, J. Moustschen, M. Moutschen-Dahman, A. Colizzi, M. Chollet and N. Houbrechts 1980, Mutation Research 74:201-202; Ehrenberg, L. 1977, in: Handbook of mutagenicity test procedures (Kilbey et al., editors), Elsevier/North Holland, Biomedical Press, 446-447; Huang, C.C. 1975, Proc. Soc. Exp. Biol. Med. 142:36-40; Mohan, G. 1973, Mutation Research 20:7-15; Murthy, M.S.S. 1979, Mutation Research 64:1-17; Shiau, S.Y., R.A. Huff, B.C. Wells and I.C. Felkner 1980, Mutation Research 71:169-179; Sylianco, C.Y.L. 1978, Mutation Research 53:271-272; Wild, D. 1975, Mutation Research 32:133-150; Würgler, F.E., F.H. Sobels and E. Vogel 1977, in: Handbook of mutagenicity test procedures (Kilbey et al., editors) Elsevier/North Holland, Biomedical Press, 335-373.



<u>Di Pasquale Paladino-Pasqua Cavolina, A.</u> Universiti da Palermo, Italy. A new melanotic tumor mutant, tu-pb, of Drosophila melanogaster showing unusual phenotypical manifestation.

A new melanotic tumor mutant, tu-pb, was discovered in a wild stock (S. Flavia) of Drosophila melanogaster.

The phenotype of tumorous tu-pb flies differes from that of other melanotic tumor stocks described so far. In fact, while melanotic tumors are usually visible as black masses free-floating in the abdomen, in tu-pb internal black masses are exclusively located on sides of the proboscis's base (Fig. 1); they are variable in number and size and may interest both or only one side; rarely larger tumors invade parts of the head.

The dissection of adults revels often melanotic masses bound the lateral-pharyngeal muscle, without having any structural relation with it (Fig. 2).

Fig. 1. Head of a tumorous tu-pb fly cleared in a fructose solution: tumors are visible as black masses on both sides of proboscis.

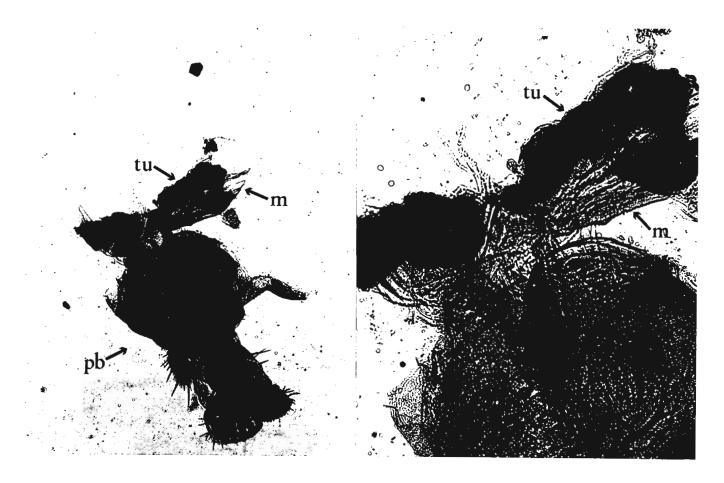


Fig. 2. Proboscis isolated by dissection from a head of a tumorour fly: melanotic masses appear bounding the lateral-pharyngeal muscle. pb=proboscis; m=lateral-pharyngeal muscle; tu=melanotic masses.

The examination of a large number of individuals at different stages during development has revealed that larvae or pupae, as well as imago at emergence, are always free from melanized masses; however, these become evident very shortly after emergence. In this respect too, tu-pb appears to differe from the other tumor stocks in which melanization is completed before pupation.

The penetrance is incomplete, very different between sexes and temperature dependent. In flies developing at 23.5° C, 30-50% of females and 2-10% of males are tumorous; when development takes place at higher (27° C) or lower (18° C) temperature, the manifestation of the tumor phenotype is almost completely suppressed.

The lack of tumorous F_1 progeny from reciprocal crosses between the wild-type strain Oregon-R and the tumoral stock tu-pb suggests recessiveness of the tumor gene/genes and no sex-linkage, while the appearance of tumors with very low frequency in the F_2 progeny is consistent with the hypothesis of more genes involved in the genetical determination.

Chromosomal location of the tumor factors was made crossing tu-pb to a balanced marked stock for the 2nd chromosome, SM5/Sp, and the 3rd chromsome, TM3/D.

 F_1 SM5/tu; TM3/tu females were then crossed with Sp/tu; D/tu brother males. Examination of the F_2 showed tumor manifestation in genotypes including together the 3rd chromosome tu-pb in homozygous condition and the 2nd chromosome tu-pb, even in the heterozygous form.

Therefore we suggest that for the tu-pb phenotype occurrence at least two genetical factors are involved, one recessive located on the 3rd chromosome and a dominant one located on the 2rd chromosome (see Table 1).

In order to map the recessive gene/genes on chromosome III, the mating scheme of Fig. 3 was performed: results obtained from the recombination experiment demonstrates that the major tumor gene is located between sr (62) and e^{S} (70.7), presumably closely e^{S} (see Table 2).

Heterozygous F_1 flies, derived from crosses between tu-pb and two second-chromosome bearing melanotic tumor genes strains, tu-48a and tu-g which manifest typical abdominal black masses, did not show tumors. In the F_2 the occurrence of tumors was not high, neither manifestation of the two tumor types occurred in the same individual.

From the results gathered so far, the tu-pb seems to be a new peculiar case of melanotic tumor manifestation in Drosophila melanogaster.

Table 1. Chromosomal location of tu-pb.

 F_2 from crosses: PP tu/SM5 ; tu/TM3 x of tu/Sp ; tu/D

Genotype			%	tu-pb	N. 99	% ♂♂ tu-pb	N. ජජ
2nd CHR.		3rd CHR.					
tu-pb/tu-pb	;	tu-pb/tu-pb		26.7	101	3.1	129
SM5/tu-pb	;	tu-pb/tu-pb		7.3	136	3.8	105
Sp/tu-pb	;	tu-pb/tu-pb		39.2	56	5.8	68
All other com	nbin	ations were f	ree o	f tumors			

Fig. 3. Matings made to map tu-pb on the 3rd chromosome:

- 99 tu-pb/tu-pb x of ru h th st cu sr e^S ca/TM3, ru Sb Ser e^S
- \$\$\$\$ tu-pb/ru h th st cu sr e^S ca x of ru h th st cu sr e^S ca/ru h th st cu sr e^S ca
- $\ref{eq:combinants}$ between the multiply-marked x of tu-pb/tu-pb and the tumor chromosome
- N.B. A maximum of 1/2 of the progeny may be homozygous for tu-pb. 2nd chromosome of the tumor stock is present at least in the heterozygous condition.

Table 2. Results of mapping the tumor gene tu-pb.

Recombinants							%	ያ የዩ tu-pb	Ν; ♀♀	% ơơ tu-pb	N. ơơ	Colt tu/N.Colt.
ru	+	+	+	+	+	+	+	19.28	586	4.59	609	10/10
ru	h	+	+	+	+	+	+	13.52	584	3.71	565	8/10
ru	h	th	+	+	+	+	+	27.41	62	1.69	59	1/1
ru	h	th	st	+	+	+	+	17.17	361	3.53	396	7/7
ru	h	th	st	cu	+	+	+	15.54	386	2.53	315	6/6
ru	h	th	st	cu	sr	+_	+	14.52	475	1.37	436	10/10
ru	h	th	st	cu	sr	e ^s	+	0.88	450	0.21	457	1/10
ru	h	th	st	cu	sr	e ^s	ca	0.00	560	0.00	546	0/10
+	h	th	st	cu	sr	e s	ca	1.72	406	0.00	408	1/10
+	+	th	st	cu	sr	e s	ca	0.00	638	0.00	573	0/11
+	+	+	st	cu	sr	e s	ca	0.00	124	0.00	131	0/2
+	+	+	+	cu	sr	e ^S	ca	0.00	367	0.00	383	0/7
+	+	+	+	+	sr	e ^s	ca	1.19	585	0.35	570	1/10
+	+	+	+	+	+	e^s	ca	5.14	583	1.91	521	5/10
+	+	+	+	+	+	+	ca	16.12	589	2.93	580	9/10
+	+	+	+	+	+	+	+	17.72	395	6.68	404	7/7