

Chromosomal assignment of the genetic factor, *tu-91k*, responsible for a melanotic tumour in the *Drosophila melanogaster* adult female

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Summary – Chromosomal transfer experiments were carried out to assign *tu-91k*, the genetic factor responsible for the organ-specific and female-limited adult melanotic tumour in *Drosophila melanogaster*. It was found that the second chromosome has a predominant effect and *tu-91k* is semidominant in its phenotypic expression.

melanotic tumour / *Drosophila melanogaster* / chromosome assignment / semi-dominance

Résumé – Assignment chromosomique du facteur génétique *tu-91k*, responsable du développement d'une tumeur mélanique chez la femelle de *Drosophila melanogaster*. Des expériences de transfert chromosomique ont été réalisées en vue d'assigner le facteur génétique *tu-91k*, responsable d'une tumeur mélanique spécifique d'un organe et limitée à la femelle adulte de *Drosophila melanogaster*. Il a été trouvé que le deuxième chromosome a un effet prédominant et que *tu-91k* est semi-dominant dans son expression phénotypique.

tumeur mélanique / *Drosophila melanogaster* / assignation chromosomique / semi-dominance

INTRODUCTION

Many different melanotic tumour strains have been described in *Drosophila* and other insects since Bridges (1916) first reported one in *Drosophila melanogaster*, but only in a rather small portion have the tumour genes been analysed in detail (Gateff 1978, 1982; Sparrow, 1978). Many of the difficulties of analysing melanotic tumour mutants stem from the fact that the phenotype is very variable and they often have

low penetrance. In previous papers, Kosuda (1990, 1991) reported a new melanotic tumour strain, C-104, in *D melanogaster*, in which the penetrance is rather high. The tumour in the C-104 strain has several unique characteristics (Kosuda, 1991). As this melanotic tumour develops at an adult stage, whereas most other tumours appear in larval stages, especially in the 3rd instar larvae shortly before pupation, this tumour can be classified as an adult one. It can only be detected under the microscope in female flies, especially in the vicinity of the spermathecae. In other words, its expression is sex-limited and organ-specific. Tumours often become macroscopically visible as dense black bodies within the abdominal cavity when they fully develop. The incidence of tumour development was shown to increase with age (Kosuda, 1990). To test the role of major chromosomes of *D melanogaster* in the development of melanotic tumours, chromosomal transfer experiments were carried out in the present study.

MATERIALS AND METHODS

The melanotic tumour strain C-104, used in this study is a highly inbred one and is derived from a natural population in Budapest, Hungary. The 2 major autosomes and the X chromosome of the C-104 strain were independently made homozygous by a routine procedure utilizing "balancer" chromosomes.

Complicated inversions contained in these balancer chromosomes prevent recombination between the homologous chromosomes, and thus preserve the original genetic content of the chromosome in the C-104 strain. Second and third chromosomes were marked with Cy In (2LR) *CyL/Pm* and with In (3LR) TM3 *Ubx/Sb*, respectively. The sex chromosome was also marked with the Muller-5 chromosome. The mating scheme is presented in figure 1. Female flies homozygous for respective chromosomes were maintained for 2 wk at 29°C to facilitate ageing, by transferring these flies to fresh vials containing standard cornmeal yeast media every 2 or 3 d. After 2 wk, female flies were singly dissected in Ringer solution for *Drosophila* under the microscope and were examined under the microscope for presence or absence of melanotic tumours in the vicinity of both spermathecae (figs 2a,b,c).

RESULTS AND DISCUSSION

Numerical results are given in table I. The principal genetic factor causing melanotic tumours in the aged females is located on the second chromosome, as is explicitly shown in table I.

The genetic factor responsible for the development of female specific melanotic tumours in the C-104 strain is designated *tu-91k*. The 3rd chromosome and X chromosome are apparently not involved in the expression of the melanotic tumour development in the C-104 strain. It is possible that modifier loci are involved on other than the second chromosome, as the genetic control of other melanotic tumours has generally been found to be multifactorial (Barigozzi *et al* 1960; Burnet and Sang, 1964; Mampell, 1967; Lindsley and Grell, 1968; Belt and Burnet, 1972; Sparrow, 1974, 1978). It seems evident that the presence of a second chromosome from the C-104 strain alone is sufficient to induce melanotic tumour formation, since

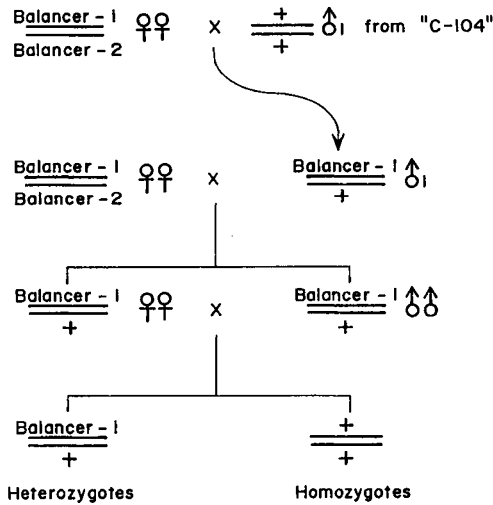


Fig 1. Mating scheme. Muller-5, Cy and Ubx chromosomes were represented by Balancer-1 of the 3 major chromosomes of *D melanogaster*, respectively. Muller-5, Pm, and Sb chromosomes were also employed as Balancer-2. Chromosomes of the C-104 strain were indicated by +.

the proportion of flies carrying melanotic tumours among those homozygous for the second chromosome is not less than that in the original C-104 strain as reported in Kosuda (1990), although reduction in the phenotypic expression is usually expected in such crosses owing to changes in the genetic background.

The heterozygous effect of *tu-91k* on the expression of this organ-specific and female-limited adult melanotic tumour was also studied using 3-wk-old female flies. The *tu-91k* heterozygous females were obtained by crosses between the C-104 strain and *Cy In (2LR)CyL Pm* or Carton - S strain. The results are summarized in table II. Ratios I and II showing the proportion of melanotic tumour-developing females or spermathecae in heterozygous females are 0.179 and 0.095 on the average, respectively. On the other hand, ratios I and II in homozygotes are 0.281 and 0.186, respectively. Since these figures in heterozygous females are almost half of those in homozygotes, *tu-91k* seems to be semi-dominant. In other words, the degree of dominance (h) seems to be nearly 0.5.

The present results confirm that the melanotic tumour development in the C-104 strain of *D melanogaster* is genetically controlled and the hereditary mode is relatively simple and the second chromosome has a predominant effect. The major gene responsible for this melanotic tumour mutation on the second chromosome is named *tu-91k*. It should be noted that the *tu-91k* gene is not recessive as are other melanotic tumour mutant genes but its phenotypic expression is semi-dominant.

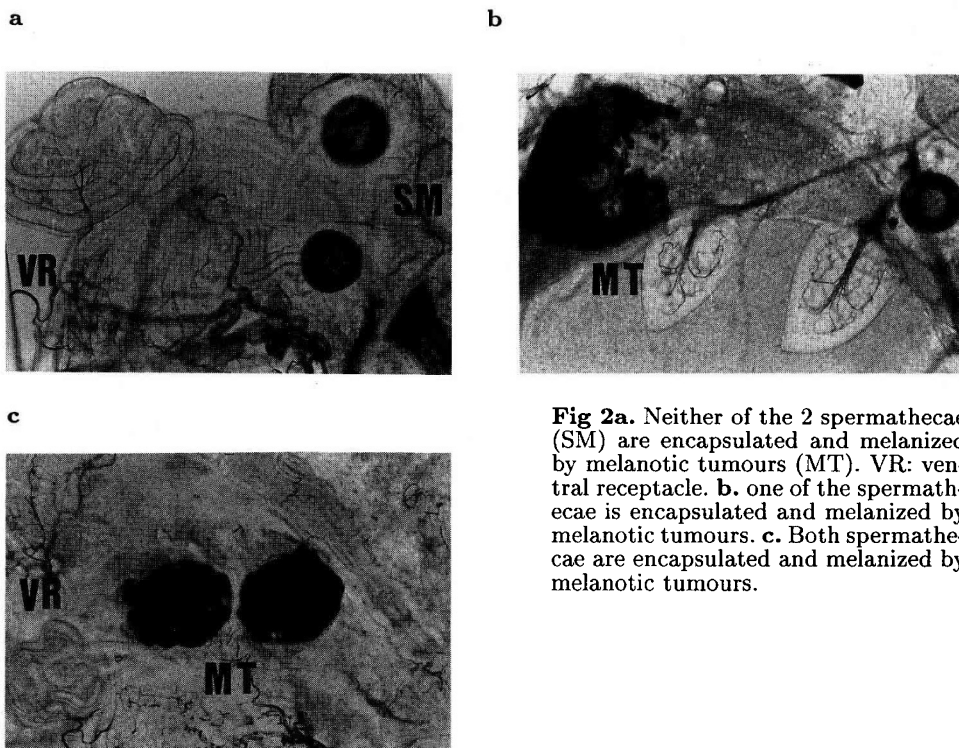


Fig 2a. Neither of the 2 spermathecae (SM) are encapsulated and melanized by melanotic tumours (MT). VR: ventral receptacle. **b.** one of the spermathecae is encapsulated and melanized by melanotic tumours. **c.** Both spermathecae are encapsulated and melanized by melanotic tumours.

Table I. Melanotic tumour development in female flies homozygous for X, 2nd and 3rd chromosomes. Homozygous chromosomes were extracted from the tumourous C-104 strain. Figures in the table express number of female flies carrying no, 1 and 2 tumour-bearing spermathecae, together with total number of females examined. Experiments a, b, and c were carried out at 3 different times.

		<i>No tumour</i>	<i>1 tumour</i>	<i>2 tumours</i>	<i>Total</i>
X-chromosome	Exp a	38	0	0	38
	Exp b	45	0	0	45
	Exp c	44	0	0	44
	Total	127	0	0	127
2nd chromosome	Exp a	26	17	13	56
	Exp b	45	20	3	68
	Exp c	39	14	6	59
	Total	110	51	22	173
3rd chromosome	Exp a	46	0	0	46
	Exp b	49	0	0	49
	Exp c	39	0	0	39
	Total	134	0	0	134

Table II. Heterozygous effect of the melanotic tumour gene, *tu-91k*, in the C-104 strain. Ratios I and II are indicative of the proportions of tumour-development on an individual basis and on a spermatheca basis, respectively. For further details, see Kosuda (1990). + Chromosome represents the 2nd chromosome of Canton-S strain.

	No tumour	1 tumour	2 tumours	Total	Ratio I	Ratio II
Heterozygotes						
<i>tu-91k/Cy (2LR) CyL</i>	112	19	1	132	0.152	0.080
<i>tu-91k/+</i>	374	81	5	460	0.187	0.099
Total	486	100	6	592	0.179	0.095
Homozygotes						
<i>tu-91k/tu-91k</i>	141	37	18	196	0.281	0.186

REFERENCES

- Barigozzi C, Castiglioni MC, DiPasquale A (1960) A complex genotype controlling the production of melanotic tumors (pseudotumors) in *Drosophila*. *Heredity* 14, 151-162
- Belt AL, Burnet B (1972) Experimental modification of the dominance relations of a melanotic tumor gene in *Drosophila melanogaster*. *Genet Res* 20, 115-135
- Bridges CB (1916) Non-disjunction as a proof of the chromosome theory of inheritance. *Genetics* 1, 1-52
- Burnet B, Sang JH (1964) Physiological genetics of melanotic tumors in *Drosophila melanogaster*. II. The genetic basis of response to tumorigenic treatment in the *tu* and *tu bw*; *st su-tu* strains. *Genetics* 49, 223-245
- Gateff E (1978) Malignant and benign neoplasms of *Drosophila melanogaster*. In: *The Genetics and Biology of Drosophila* (Ashburner M, Wright TRF, eds) Academic Press, London, vol 2b, 181-276
- Gateff E (1982) Cancer, gene and development: the *Drosophila* case. *Adv Cancer Res* 37, 33-74
- Kosuda K (1990) Ageing and temperature effects on tumour development in *Drosophila melanogaster*. *Gerontology* 36, 121-125
- Kosuda K (1991) The tumour formation in *Drosophila melanogaster* females. *Dros Inf Serv* 90, 123-124
- Lindsley DL, Grell EH (1968) *Genetic Variation of Drosophila melanogaster*. Carnegie Institution, Washington
- Mampell K (1967) Genetics and environmental control of melanotic tumours in *Drosophila*. *Genetica* 37, 449-465
- Sparrow JC (1974) The genetics of some second chromosome melanotic tumour mutants of *Drosophila melanogaster*. *Genet Res* 23, 13-21
- Sparrow JC (1978) Melanotic tumours. In: *The Genetics and Biology of Drosophila* (Ashburner M, Wright TRF, eds) Academic Press, London, vol 2b, 277-313