Deleterious Effect of the Melanotic Tumour on the Survival Rate of Drosophila melanogaster Female Flies

Author: Kosuda, Kazuhiko

Source: Zoological Science, 13(6) : 821-824

Published By: Zoological Society of Japan

URL: https://doi.org/10.2108/zsj.13.821

ZOOLOGICAL SCIENCE 13: 821–824 (1996)

Deleterious Effect of the Melanotic Tumour on the Survival Rate of *Drosophila melanogaster* Female Flies

Kazuhiko Kosuda

Biological Laboratory, Faculty of Science, Josai University, Sakado, Saitama 350-02, Japan

ABSTRACT—The effect of melanotic tumour formation on the survival rate of female flies was investigated for a period of five weeks after eclosion in the C-104 mutant strain of *Drosophila melanogaster*, found in a natural population in Budapest. Melanotic tumours developed only in female flies in the vicinity of spermathecae. The incidence of tumour formation was higher in the dead females than in the living ones. This fact explicitly indicated that tumour formation has a deleterious effect on the survival rate and thus the life span of its carriers. It also implied that non-self recognition or self-defense reaction is accomplished at the expense of the organisms. The reason for the maintenance of this melanotic tumour gene in a natural population is discussed.

INTRODUCTION

Many different melanotic tumours have been described in *Drosophila melanogaster* since the first report by Bridges (1916). In general, the melanotic tumour phenotype shows variable expressivity and low penetrance (Sparrow, 1978; Carton *et al.*, 1992). Melanotic tumours typically appear in third instar larvae shortly before pupation. The tumours are observed as irregular shaped dense black masses within the body cavity of the larva, usually floating in the haemocoel and present as single or multiple masses. There is a general agreement that melanotic tumours develop from the aggregation and melanization of haemocytes (Lindsley and Grell, 1967; Salt, 1970).

In previous reports, Kosuda (1988, 1990) identified a melanotic tumourous strain named C-104, in *Drosophila melanogaster*, in which tumours formed only in adult females exclusively in the vicinity of spermathecae. The incidence of tumour formation also was found to increase as the female aged (Kosuda, 1990). The genetic factor responsible for this melanotic tumour formation was named *tu-91k*, and assigned to the second chromosome of *Drosophila melanogaster* (Kosuda, 1992).

Melanin production and cellular encapsulation of parasites by haemocytes are both considered to be typical non-self recognition responses and self-defense systems in insects and other invertebrates (Salt, 1970; Carton *et al.*, 1978; Nappi and Carton, 1986; Rizki and Rizki, 1984). If melanotic tumour formation is a self-defense mechanism, it may have advantageous influences in its carriers. It is an interesting question as to whether melanotic tumour formation has a beneficial or deleterious effect on the survival rate and other fitness components of its carriers. In a review of melanotic tumours, Sparrow (1978) stated that adult tumours are benign and that there is no evidence they affect adult viability. In the present investigation, the effect of melanotic tumour formation on adult viability was examined.

MATERIALS AND METHODS

The melanotic tumour strain, C-104, homozygous for the second chromosome was used. The strain was derived from a natural population of *Drosophila melanogaster* in Budapest, Hungary in 1986. Female flies of the C-104 strain were kept together with males of the same strain for five weeks at 29°C. The frequency of melanotic tumour development increases as the female flies get older and the tumour incidence is accelerated by high temperature (Kosuda, 1990). Flies were maintained in vials and transferred every two or three days to fresh vials. At the time of transfer, dead female flies were randomly taken and individually examined for the presence of melanotic tumours under the microscope. Then each female fly was classified and assigned to one of three categories, according to the numbers of spermatheca attaced or enveloped by the melanotic tumours (Fig.1a-c). Three experiments were repeated at different times. The incidence of melanotic tumour formation was also investigated in living flies that were arbitrarily sampled. Indices of melanotic tumour development in dead and living flies were compared. If the melanotic tumour had a deleterious effect on adult viability/longevity, the incidence would be higher in the dead than in the living flies.

RESULTS

Data from three replicated experiments concerned with incidence of melanotic development in the living and dead female flies at different ages during the five weeks are combined into Table 1. The incidence of tumour formation on an individual basis, Ratio I, is remarkably higher in dead flies than in living females at all ages. The incidence on a spermatheca basis, Ratio II, is also considerably higher in the

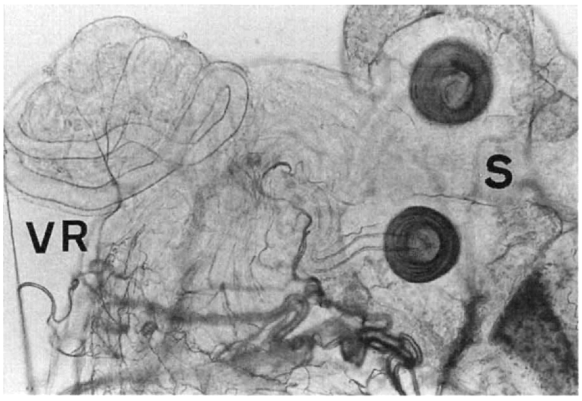

Fig. 1a. Normal spermathecae (S) in the control strain, Canton-S. VR: ventral receptacle.

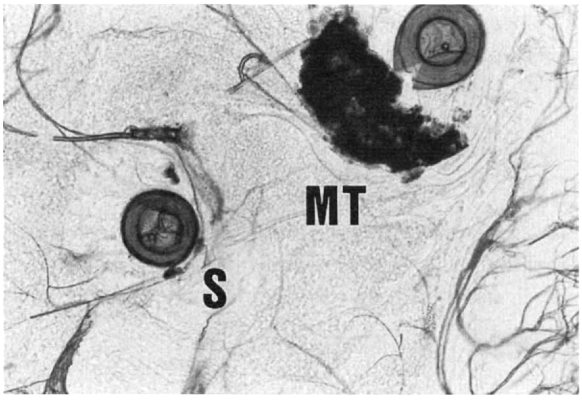

Fig. 1b. One spermatheca (S) out of two is encapsulated by the melanotic tumour (MT).

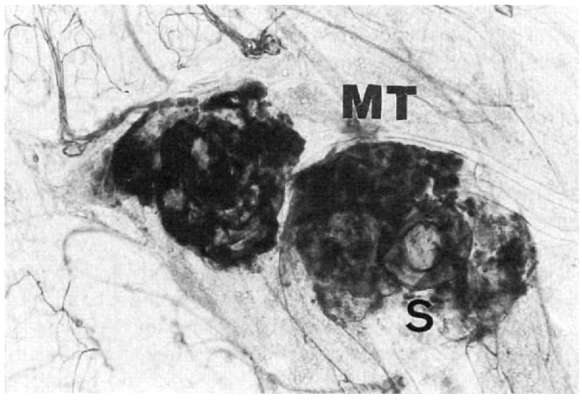

Fig. 1c. Both spermathecae (S) are encapsulated by the melanotic tumour (MT).

dead females than in living flies. Thus the rate of mortality of old female flies carrying melanotic tumours is much higher than that of non-carriers of tumours.

The results of a two-way analysis of variance for melanotic tumour formation on an individual basis is given in Table 2. Both the treatment (living or dead) and age are shown to be statistically higher significant. These fact indicate that melanotic tumour formation may have a deleterious effect on the survival rate and consequently may lead to a reduction in the longevity of its carriers.

The proportion of melanotic tumour development in old living females 5 weeks after eclosion was 0.281 on an individual basis, whereas no tumours formed in young flies one week after eclosion. On the other hand, the proportion of dead females with melanotic tumours increased with age from 0.021 to 0.506. The incidence of tumour formation on a spermatheca basis, Ratio II, also increased from 0.0 to 0.165 for living females, and from 0.011 to 0.315 for dead flies.

The probability of death can not be obtained from the number of living and dead individuals at the respective ages in Table 1, since living and dead female flies were randomly sampled.

DISCUSSION

The results in Tables 1 and 2 may be explained without considering the deleterious effect of the melanotic tumours on viability, if it occurs following death of female flies, since insect haemolymph is known to melanize after death. However, this possibility can be rejected since the incidence of melanotic tumours was previously proven not to increase following death in a preliminary experiment.

Table 1 indicates explicitly that the incidence of melanotic tumours increases with age both for living and dead females. This fact was also reported for living flies by Kosuda (1990). These findings may imply that melanotic tumours are liable to develop in female flies that get old and then die earlier. The viability of such individuals should also be low. It is expected that accelerated senescence should necessarily accompany the reduction in viability. Deleterious effects of melanotic tumour formation on viability also are supported by the observation that the proportion of males in the living flies increased as they get old (data not shown).

From an evolutionary perspective, it is interesting that melanotic tumour formation decreases the survival rate. It suggests the possibility of metabolic trade-off; energy reserves may be reduced in tumourous flies due to energy utilization in melanotic encapsulation of apparent “non-self”, a potentially valuable survival response, and hence this leads to the reduction in adult viability and then a shorter life span.

The phenoloxidase cascade may play an indispensable role in self or non-self recognition system and melanotic tumour formation (Ashida and Yamazaki, 1990; Ratcliffe *et al.*, 1984; Rizki *et al.*, 1985). Phenoloxidases oxidize phenolic compounds to the corresponding quinones, which are very reactive and therefore harmful to organisms (Ashida and Yamazaki, 1990). Therefore, melanin production may have deleterious effects on the survival rate of female flies carrying melanotic tumours in the C-104 strain.

The present study shows that melanotic tumour formation decreases adult viability and then longevity of female flies. However, this does not affect fitness at all, since melanotic tumours develop mainly in old females. In fact, the C-104

Table 1. Deleterious effect of melanotic tumour formation on female viability in the C-104 of *Drosophila melanogaster*

Age (weeks)	No. of female flies				Total	Ratio I	Ratio II
		No. of spermatheca attached or encapsulated by melanotic tumours					
		0	1 (A)	2 (B)			
1	Alive	81	0	0	81	0	0
	Dead	46	1	0	47	.021	.011
2	Alive	90	15	3	108	.166	.097
	Dead	113	36	11	160	.294	.181
3	Alive	283	69	20	372	.239	.147
	Dead	210	120	65	395	.468	.316
4	Alive	190	59	19	268	.291	.181
	Dead	223	160	70	453	.508	.331
5	Alive	74	24	5	103	.281	.165
	Dead	44	34	11	89	.506	.315

Ratios I and II are the proportion of tumour formation on an individual basis and a spermatheca basis, respectively. They are calculated by the following formulae; Ratio I = (A + B)/Total and Ratio II = (A + 2B)/(2 × Total), respectively.

Table 2. Two-way analysis of variance for the proportion of melanotic tumour formation on an individual basis

Sources	S. S.	d. f.	M. S.	F
Alive/Dead	723.14	1	723.14	66.47**
Age	6290.84	4	1572.71	144.55**
Interaction	190.33	4	47.58	4.37*
Error	217.63	20	10.88	
Total	7421.95	29		

Inverse sine transformation is made for Ratio I. * and ** indicate significance level of 5% and 1%, respectively.

mutant strain can be maintained in our laboratory without any difficulties.

The melanotic tumour in the C-104 strain seems to be subjected to little or no natural selection, as the tumours develop mainly at an old age at 29°C. In fact, the C-104 strain is derived from a natural population. The genetic factors responsible for geriatric traits, such as Huntington's chorea, diabetes, cancer, prebyopia and baldness are expected to be maintained in human populations, since they are expressed in aged individuals after they have passed through their reproductive periods. They are almost free from the effects of natural selection, even though they have deleterious effects on their carriers (Kosuda, 1990). Consequently, the genetic variance is expected to be greater in aged individuals than in the young. In fact, Kosuda (1985) observed experimental verification that the phenotypic variability increased with age for the male mating activity in *D. melanogaster*. Recently, strong evidence of an increase in genetic variability with age was presented for mortality rates of *D. melanogaster* (Hughes and Charlesworth, 1994). Furthermore, Hughes (1995) confirmed that additive genetic variance in aged males is statistically larger than that in young ones for mating activity in *D. melanogaster*.

ACKNOWLEDGMENTS

I am indebted to A. J. Nappi, Loyola University Chicago, U. S. A. and D. D. F. Skibinski, University of Wales Swansea, U. K., for their kind reading of the manuscript and valuable suggestions. I express my sincere thanks to anonymous reviewers for useful comments.

REFERENCES

Ashida M, Yamazaki HI (1990) Biochemistry of phenoloxidase system in insects: with special reference to its activation. In "Molting and Metamorphosis" Ed by E Ohnishi, H Ishizuka, Japan Sci Soc Press/Springer-Verlag, Tokyo/Berlin, pp 239–265

Bridges CB (1916) Non-disjunction as a proof of the chromosome theory of inheritance. Genetics 1: 1–52

Carton Y, Bouletreau M, Van Alphen J, Van Lenteren J (1978) The *Drosophila wasps*. In "The Genetics and Biology of *Drosophila* Vol 3e" Ed by M Ashburner, L Carson, JN Thompson, Academic Press, New York, pp 347–394

Carton Y, Frey F, Nappi A (1992) Genetic determinism of the cellular

immune reaction in *Drosophila melanogaster*. Heredity 69: 393–399
Hughes KA (1995) The evolutionary genetics of male life histry characters in *Drosophila melanogaster*. Evolution 49: 521–537
Hughes KA, Charlesworth B (1994) A genetic analysis of senescence in *Drosophila melanogaster*. Nature 367: 64–66
Kosuda K (1985) Ageing effect on male mating activity in *Drosophila melanogaster*. Behav Genet 15: 297–303
Kosuda K (1988) The melanotic tumour formation in female flies of *Drosophila melanogaster*. Jap J Genet 63: 572–573
Kosuda K (1990) Ageing and temperature effects on tumour development in *Drosophila melanogaster* females. Gerontology 36: 121–125
Kosuda (1992) Chromosomal assignment of the geneic factor, *tu-91k*, responsible for a melanotic tumour in the *Drosophila melanogaster* adult female. Genet Sel Evol 24: 561–565
Lindsley DL, Grell EH (1967) Genetic variations of *Drosophila melanogaster*. Carnegie Institution Publication No. 627. Carnegie Institution, Washington
Nappi AJ, Carton Y (1986) Cellular immune responses of *Drosophila*. In "Immunity in Invertebrates" Ed by M Brehelin, Springer-Verlag, Berlin, pp 171–187
Ratcliffe NA, Leonard C, Rowley A (1984) Prophenoloxidase activating, non-self recognition and cell co-operation in insect immunity. Science 226: 557–559
Rizki TM, Rizki RM (1984) The cellular defense system of *Drosophila melanogaster*. In "Insect Ultrastructure Vol 2" Ed by RC King, H Akai, Plenum Press, New York, pp 579–604
Rizki TM, Rizki RM, Belloti RA (1985) Genetics of *Drosophila* phenoloxidase. Mol Gen Genet 201: 7–13
Salt G (1970) The cellular defence reaction of insects. Cambridge Monographs in Experimental Biology No. 16. Cambridge University Press, Cambridge
Sparrow JC (1978) Melanotic 'tumours'. In "The Genetics and Biology of *Drosophila* Vol 2b" Ed by M Ashburner, TRF Wright, Academic Press, New York, pp 277–313

(Received March 28, 1996 / Accepted August 26, 1996)