

Johnston has done extensive research on these desert species and has reported to us that mating always stopped when temperature rose above 25°C or when humidity fell below 25%, usually at 10 a.m.) November is a rather mild month and provides the right temperature conditions for prolonged mating activity. Yet we have seen that *D. mettleri* does not use afternoon time efficiently. Apparently this would reduce the reproductive potential of this species but since numbers of *D. mettleri* are high, although not as high as of *D. nigrospiracula*, this may be irrelevant. As we mentioned above, both species have a very similar morphology, but the more we study their ecology, the more distinct we find they are. This suggests that most of their differentiation has occurred at the level of behavior, and morphology only plays a role on the characters involved directly with sexual isolation. More investigation is needed to know the adaptive (or non-adaptive) meaning of the differential mating activity reported here. However, as far as we know, this is the first time that such differential behavior has been described from a natural population of *Drosophila*.

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References: Fellows, D.P. and W.B. Heed 1972, *Ecology* 53:850-858; Kaneshiro, K.Y., H.L. Carson, F.E. Clayton and W.B. Heed 1973, *Amer. Natur.* 107:766-774; Margalef, R. 1974, *Ecologia*, Ediciones Omega, Barcelona.

Gamo, S., M. Ogaki and E. Nakashima-Tanaka. University of Osaka Prefecture, Japan. Anesthetics resistance in *D. melanogaster*.

We reported that ether resistance of Eth strain at 24 hours age of adult flies was completely dominant over sensitivity and that maternal or cytoplasmic effects were negligible. The locus of the major gene(s) for the ether resistance is around 61 on the third chromosome, and the

minor genes are on both the X and the fourth chromosomes (Ogaki et al. 1967).

We have investigated effects of chloroform and halothane on the Eth and the bw;st;sv<sup>n</sup> strains. Mortality of the Eth strain was 1.3% in females and 1.7% in males, and that of the bw;st;sv<sup>n</sup> strain was 82% in females and 90% in males in 30 seconds treatment with chloroform. The LT<sub>50</sub> (50% lethal time) of the Eth strain for halothane treatment was 7 minutes in females and 9.5 minutes in males; that of the bw;st;sv<sup>n</sup> strain was 3.5 minutes in females and 3 minutes in males. Thus, it can be said that a cross-resistance to ether, chloroform and halothane is found in the Eth strain. Reciprocal crosses between the Eth and bw;st;sv<sup>n</sup> strains showed that the resistance to chloroform, as well as to ether, was completely dominant over sensitivity and that maternal or cytoplasmic effects were negligible. But the resistance to halothane was an intermediate trait with no maternal or cytoplasmic factors. A major gene with respect to chloroform resistance is located on the X chromosome, and a minor gene(s) on the second chromosome. However, the major gene(s) for halothane resistance is located on the right end of the third chromosome, which may be different from the major gene of ether resistance, and minor genes are on the X and second chromosomes. Therefore, resistances to ether, chloroform and halothane may be controlled by different genes.

Gausz, J., A.A.M. Awad and H. Gyurkovics. Biological Research Center of the Hungarian Academy of Sciences, Szeged, Hungary. New deficiencies for the kar locus of *D. melanogaster*.

The heat-shock inducible puff regions of 87A and 87C on the right of the 3rd chromosome have a special importance in studying gene regulation. A detailed genetic analysis of these loci is going on in our laboratory. As a rather limited number of deficiencies were only available for these loci (Ish-Horowicz 1977; Costa 1977), we

screened for new deficiencies in the region. Males homozygous for bw eye color mutation on the 2nd chromosome were irradiated with 4000 r X-rays (150 kV, 0.5 mm Al-filter, 1000 rads/minute). After irradiation the flies were immediately mated en mass to virgin females homozygous for bw on the 2nd and cu and kar on the 3rd chromosome. The interaction of bw and kar results in a very characteristic light brown eye color, making it possible to distinguish the putative deficiencies and point mutations from the wild type progeny. After scoring 63,000

F<sub>1</sub> progeny 28 kar and 8 cu stocks were recovered over the TM3 balancer. The kar stocks were crossed to Df(3R)kar<sup>3J</sup> and Df(3R)kar<sup>3Q</sup> deficiencies deleting the kar locus and 10 of the established stocks proved to be single kar point mutations. All the isolated cu stocks produced cu progeny when crossed to cu point mutation, i.e., they represented new cu alleles. It is in accordance with the presence of a minute locus nearby to cu locus (Lindsley et al. 1972).

All the putative kar deficiencies were crossed to ru cu ca flies and 3rd instar larvae from the progeny were dissected and salivary gland chromosome preparations were made (Yoon et al. 1973) to determine the breakpoints of the deficiencies. With the aid of these deficiencies, we mapped the coding locus for kar to 87C8 band.

The new stocks carrying a deficiency in the 87A-C region were mated to In(3R)Na flies and 3rd instar larvae from the progeny were heat-treated (30 minutes at 37°C) to show either the presence or absence of 87A and 87C puffs. The data are listed in Table 1.

Table 1. List of new kar deficiencies and their breakpoints.

Deficiency	Breakpoints		Formation of puffs	
	Proximal	Distal	87A	87C
Df(3R)kar <sup>Sz-5</sup>	86E20-F1 ;	87F3-4	-	-
Df(3R)kar <sup>Sz-8</sup>	87C1-3 ;	87D14-15	+	+
Df(3R)kar <sup>Sz-11</sup>	87C7-8 ;	87E5-6	+	+
Df(3R)kar <sup>Sz-12</sup>	87B1-3 ;	87C8-9	+	-
Df(3R)kar <sup>Sz-13</sup>	86E6-7 ;	87C9-D1	-	-
Df(3R)kar <sup>Sz-15</sup>	87B1-2 ;	87E1-2	+	-
Df(3R)kar <sup>Sz-16</sup>	87C1-2 ;	87C9-D1	+	+
Df(3R)kar <sup>Sz-21</sup>	87C6-7 ;	87C8-9	+	+
Df(3R)kar <sup>Sz-23</sup>	86E6-7 ;	87C9-D1	-	-
Df(3R)kar <sup>Sz-27</sup>	87C7-8 ;	87E12-13	+	+
Df(3R)kar <sup>Sz-29</sup>	87C3-4 ;	87C9-D1	+	+
Df(3R)kar <sup>Sz-28</sup>	87C7-8 ;	87E9-10	+	+
Df(3R)kar <sup>Sz-30</sup>	87B2-4 ;	87D2-3	+	-
Df(3R)kar <sup>Sz-31</sup>	86C6-7 ;	87C9-D1	-	-
Df(3R)kar <sup>Sz-33</sup>	87C1-2 ;	87E4-5	+	+
Df(3R)kar <sup>Sz-37</sup>	87C5-6 ;	87D14-E1	+	+
Df(3R)kar <sup>Sz-40</sup>	87B2-3 ;	87D1-3	+	-
Df(3R)kar <sup>Sz-72</sup>	87E1-3 ;	87F13-14	+	+

References: Costa, D. et al. 1977, DIS 52:140; Ish-Horowicz, D. et al. 1977, Cell 12:643-652; Lindsley, D.L. et al. 1972, Genetics 71:157; Yoon, J.S. et al. 1973, Experientia 29:639.

Gershenson, S. Institute of Molecular Biology and Genetics, Academy of Sciences of the Ukrainian SSR, Kiev 252627, USSR. Additional data on putative insertion mutations in wild populations of *D. melanogaster*.

In several recently published papers (Golubovsky 1977, Golubovsky et al. 1977, Green 1975, Ising and Ramel 1976, Ivanov and Golubovski 1977, Rasmussen and Green 1974) interesting data have been presented on unstable heredity variations in *D. melanogaster* presumably caused by insertion mutations. The significance of these data is increased by the fact that at least one of these

mutations, the sex-linked recessive mutation singed bristles (sn), was found in several wild populations of this insect. In this connection I think it worthwhile to draw attention to similar findings (seemingly the first of this kind) made by me more than 40 years ago. These findings were described in a report of the genetical laboratory of the Institute of Zoology (Academy of Sciences of the Ukrainian S R) which was published in May or June, 1941. Because of the subsequent invasion of Hitler's army, this book reached only very few libraries in the USSR and no reprints have been prepared of the papers contained therein so that they remain unknown to most geneticists. Here follows a brief summary of a part of my paper (Gershenson 1941) published in this book.