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Published in: Hereditas

DOI: 10.1111/j.1601-5223.1991.tb00337.x

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Document Version
Publisher's PDF, also known as Version of record

Publication date: 1991

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):

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A locus in Drosophila melanogaster affecting heat resistance

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(Received February 7, 1991. Accepted April 9, 1991)

Temperature is an important environmental factor for Drosophila melanogaster and polymorphism for temperature resistance is very likely to occur (Parsons 1973). Many studies are published about heat resistance in Drosophila melanogaster, but most of them concern the heat shock response (e.g., Lindquist 1986). Little attention is paid to the variation in heat resistance itself, though such studies can reveal additional information about the mechanisms of heat resistance and climatic adaptations. Above this, conditional lethal mutations can play a role in agricultural pest control (e.g., Hedrick 1984).

The first report of variability for heat resistance in Drosophila melanogaster is from Hosgood and Parsons (1968), who found differences between iso-female lines and ascribed these to (unknown) polymorphic genes. Morrison and Milkman (1978) succeeded in selection for decreased resistance, and showed that the gene or genes responsible for this decrease could be localized for the greater part at the second chromosome. The present article describes the genetical localization of a natural mutant for decreased heat resistance in Drosophila melanogaster: a recessive heat sensitive lethal on the second chromosome, l(2)h.s.

Experiments

For all experiments the flies were reared at 25°C and about 50% R.H. on standard medium (Oudman et al. 1991) and tests were performed at 35°C and about 90% R.H. to prevent desiccation.

The mutant was discovered in the wild type laboratory strain Groningen 83, which was founded in 1983 with 403 females from a fruit market in Groningen, the Netherlands. Fig. 1 shows survival at 35°C of males from a polymorphic strain of Drosophila melanogaster. 35°C (not shown) did not have two phases of mortality, but had normal curves with mortality starting at about 48 hours. When survival at 25°C was followed from a strain that had two mortality phases at 35°C, a normal survival curve was observed (Fig. 2), as was the fact at 29°C (not shown). Thus, the two phases of mortality at 35°C seemed to be caused by a polymorphism for survival at high temperature that did not influence longevity at lower temperatures.

For further analysis eight inbred strains were derived from the substrain mentioned above by at least four subsequent sister-brother crosses, and tested for survival at 35°C. Table 1 shows that two strains (1 and 7) had strongly decreased heat resistance compared to the other strains. This effect was similar for males and females. In a resistant strain some flies might die during the first day, due to ‘normal’ mortality, as would be the fact at any other temperature. Because comparison of these strains in later generations gave similar results (not shown), it was concluded that the inbred strains were true-breeding for a genetic factor determining heat resistance.

A cross between a sensitive and a resistant strain (not shown) yielded a resistant strain; thus sensitivity was a recessive trait. Chromosome analysis was performed with the aid of the balanced marker stock

Fig. 1. Survival at 35°C of males from a polymorphic strain of Drosophila melanogaster.
Hereditas 114 (1991)

for the second and third chromosome SM5 Cy: TM3 Ser (LINDSEY and GREGOR 1968). The observation that homozygous sensitive adults never survived for longer than 16 hours at 35°C, while resistant flies, if in good condition, normally survived much longer, was used in the chromosome localization to infer the resistance of flies. The heat sensitive strain 7 was crossed with the marker strain according to the scheme in Fig. 3. F2 males were tested for survival during 16 hours at 35°C. From the survival percentages in Fig. 3 it can be deduced that heat sensitivity is a recessive character located on the second chromosome.

Localization experiments with the recessive mutants cinnabar (cn. II 57.5) (all standard locations according to LINDSEY and GREGOR 1968) and brown (bu. II 104.5) (not shown) induced that the character was located just left of cn. Final localization on the second chromosome was performed with the recessive mutants purple (pr. II 54.5) and cn. Females of the heat sensitive strain 7 were crossed with males pr cn. F2 females (heterozygotes in which recombination could occur) were backcrossed to pr cn males. In the F2 four phenotypes occurred: wild type, purple, cinnabar, and purple/cinnabar (Table 2). In the F2 a recombination percentage between pr and cn of 2.3 % was found, instead of the expected 3 %, which is significantly different (χ² = 3.96, 0.04<P<0.05). This difference might be caused by different viability of the recombinant genotypes, but there were no other indications for such differences. To determine the frequency of sensitivity in the F2 phenotypes, a number of individual males from each of the F2 phenotypes were crossed with sensitive females from strain 7, and their progeny were kept apart. These F3 flies were tested for survival at 35°C. If sensitivity was present in an F2 male in heterozygous state, mortality of F3 progeny was expected to be 50 %. If sensitivity was not present in the F2 male, 100 % survival of F3 progeny was expected. At least 70 flies per cross were tested at 35°C. Table 2 shows the number of the F2 phenotypes and the results of the F3 tests. Because the ratio sensitive/non-sensitive was reversed between the recombinant phenotypes (pr and cn) and between the non-recombinant phenotypes (wild type and pr cn), ap-

Table 2. Percentage adult mortality after various periods of time at 35°C in eight inbred strains of Drosophila melanogaster in individuals tested.

<table>
<thead>
<tr>
<th>Strain</th>
<th>Females, hours at 35°C</th>
<th>Males, hours at 35°C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12</td>
<td>24</td>
</tr>
<tr>
<td>1</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>8</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>
Table 2. Occurrence of heat sensitivity in F3 males from crosses of F2 males with heat sensitive females

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Number in F2</th>
<th>Number F3 tested</th>
<th>Resistant</th>
<th>Sensitive</th>
</tr>
</thead>
<tbody>
<tr>
<td>wild type</td>
<td>1233</td>
<td>121</td>
<td>0</td>
<td>121</td>
</tr>
<tr>
<td>cinnabar</td>
<td>29</td>
<td>27</td>
<td>10</td>
<td>17</td>
</tr>
<tr>
<td>purple</td>
<td>29</td>
<td>27</td>
<td>18</td>
<td>9</td>
</tr>
<tr>
<td>purple/cinnabar</td>
<td>1208</td>
<td>121</td>
<td>121</td>
<td>0</td>
</tr>
</tbody>
</table>

Apparently only one locus is involved. The locus is located between pr and cn, closer to pr than to cn. The location on a standard second chromosome is 54.5 + (9 + 10)/54*3 = 55.6. The locus is a conditional, heat sensitive, recessive lethal on the second chromosome: l(2)hs.

Conclusion and discussion

l(2)hs is a natural, heat sensitive, recessive lethal of Drosophila melanogaster, with map position II 55.6. Adult flies, homozygous for l(2)hs, never survive 16 hours at 35°C, while wild type flies, if in good condition, usually survive much longer.

No recessive heat sensitive mutants were known from the autosomes of Drosophila melanogaster (Lindsley and Zimm 1986). Suzuki (1970) described a number of mutations sensitive to 29°C, but these were recessive mutations on the X-chromosome and dominant lethals on the second and third chromosome. Possibly l(2)hs is the same locus that influenced survival in the experiments of Morrison and Milkman (1978), who only did a chromosome localization.

About the mechanism we can only speculate at the present time. Enzyme inactivation or tissue- or cell damage both are possible, perhaps intermediated by the absence of heat shock protein synthesis. No heat shock protein loci are known in the region near II 55.6 (Ashburner and Bonner 1979). A number of mutants that influence the expression of the heat shock response are known from the second chromosome, but they are not exactly localized (Parker-Thornburg and Bonner 1987). Studies of the molecular and physiological mechanism of the locus and the temperature range of sensitivity will be necessary.

Up to now l(2)hs is only found in the Groningen 83 population. Because the mutant has not been discovered before it is probably rare in nature, but further studies on the occurrence are necessary.

Acknowledgements. — The investigations were supported by the Foundation for Biological Research (BION), which is subsidized by the Netherlands Organization for Scientific Research (NWO: grant 811–436–104). I like to thank B. J. Zwaan for his contribution of the 25°C survival curve, R. Bijlsma and W. van Delden for comments on the manuscript, and H. Mulder for preparing the drawings.

References


