D. funebris, only about 1% of the flies showed an increased number of scutellar bristles and irregularities of the abdominal stripes, whereas in control cultures for other species, the percentage of affected flies was still smaller. Wing and vein anomalies were never observed.

The present simple experiment provides further evidence of the differential effect of the chemical compounds (thymidine) in organisms (different species of the genus *Drosophila*) with different genetical constitutions. The negative effect in *D. subobscura* and *Megaselia scalaris* may be due to either (a) thymidine not being taken up at all or (b) if taken up, acting as a normal constituent. Autoradiographic studies are needed to decide various points[2].

Zusammenfassung. Der Autor hat bereits früher über den teratogenen Effekt von Thymidin bei *D. melanogaster* berichtet. Die vorliegende Arbeit befasst sich mit demselben Phänomen bei anderen *Drosophila*-Arten und *Megaselia scalaris* (Phoridae). Während der teratogene Effekt bei *D. simulans*, *D. mercatorum* und *D. funebris* der gleiche wie bei *D. melanogaster* ist, tritt bei *D. subobscura* and *Megaselia scalaris* keine teratogene Wirkung auf. Die für die genannten Arten verwendete Thymidinkonzentration erweist sich für *D. hydei* als stark toxisch und verhindert die Verpuppung vollkommen. Das Ergebnis zeigt, dass verschiedene genetische Systeme sehr unterschiedlich auf ein und dieselbe chemische Substanz reagieren können.

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[2] This work was done in the Institute of Biology of the University of Vienna (Head: Prof. Dr. F. Mainx) and the author is thankful for guidance and laboratory facilities.

Polymorphism in Natural Populations of *Drosophila* for the Ability to Withstand Temperature Shocks

Single inseminated founder females of *Drosophila melanogaster* derived from the same population have been shown to lead to strains which differ genetically for several quantitative traits. Three are morphological (scutellar and sternopleural chaeta numbers, and egg length) and 2 are behavioural (mating speed and duration of copulation)[1-3]. It is likely that the differences between the strains are genetic in origin, arising from genetic differences between the founder females. This is direct evidence that the wild populations are polymorphic for genes (or polygenes) controlling these traits[4]. Even though it has been argued for many years[5] that polymorphism for polygenes is expected in outbreeding species, these results form its most general demonstration so far. The polymorphism for scutellar chaeta number has been exploited by Hosgood, MacBean and Parsons[6,7], who found that when directional selection for high chaeta number was based on those strains which had a high scutellar chaeta number, extremely rapid responses were obtained.

In this paper we extend our observations to a more physiological trait, namely the effect of a high temperature shock (33.5 °C for 24 h on flies 7 days of age) on survival at 7 days after the completion of the shock. Three strains which have been scored for the other traits mentioned above, and which were derived from single inseminated females collected at Leslie Manor, Victoria in December 1964 were used. Testing was carried out at the 7th, 18th, 30th and 40th generations in the laboratory by exposing 2 replicates of 25 flies of each sex to the temperature shock. Two further replicates were kept at 25 °C continuously for controls. Flies were cultured and stored as virgins on standard medium seeded with yeast at 25 °C for the 7 days prior to the temperature shock, and returned to 25 °C after the shock. Percentage mortalities at 7 days after the shock were calculated.

Mean percentage mortalities of control and temperature shocked males and females are given in Table I, and an analysis of variance of the data in Table II. In both sexes

Table I. Percentage mortality of control and temperature shocked flies at 7 days after the shock

		Control			Temperature shocked		
Strain		1	2	3	1	2	3
Generation	7 ♀	8	4	2	4	8	2
	♂	2	14	2	2	10	12
	18 ♀	0	0	4	4	0	10
	♂	10	4	0	4	16	8
	30 ♀	4	6	4	6	20	30
	♂	12	2	2	14	20	26
	40 ♀	4	4	2	50	76	2
	♂	4	4	8	42	98	12
Mean	♀	4.0	3.5	3.0	16.0	26.0	11.0
	♂	7.0	6.0	3.0	15.5	36.0	14.5

Each entry is the mean of 2 replicates of 25 flies.

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the temperature shock increased percentage mortality compared with the controls, and this difference was significant ($P < 0.001$). There was no significant difference in mortality between sexes, but mortality differed significantly between strains ($P < 0.001$) and generations ($P < 0.001$). Interactions involving temperature shock, strains, and generations were all significant, but all interactions involving sexes were non-significant. Because there were no significant differences between sexes, the data for males and females were pooled, and further analyses of variance carried out separately on the control and temperature shocked flies (Table III). For the control data, there were no significant differences between strains, generations or the strains × generations interaction, whereas all these components were significant for the temperature shocked flies ($P < 0.001$). Since the mortalities in the controls were similar in all strains and generations, the significant differences for the shocked flies indicate real differences between strains and generations. However, it is felt that more weight should be placed on the strain differences than on the generation differences, because of the possibility of gross effects between generations due to minor changes in microenvironment, e.g. minor temperature fluctuations in the incubator which had a range of ± 0.5 °C, although as the experiments extended over 40 generations it is possible that some genetic changes which influenced temperature sensitivity occurred in the laboratory.

Therefore, in agreement with the various other traits cited, the 3 strains tested have different mortalities following a high temperature shock. There is evidence in the literature for temperature sensitive strains, both in *Drosophila*[8] and in other species[9,10]. It seems as if each strain received certain genes (or polygenes) by chance and is almost completely unable to change them, so that the base population must be polymorphic for genes controlling the ability to withstand a high temperature shock. No differences in mortality occurred between strains in the controls, although differences might well have appeared, had deaths been scored over a longer period. This parallels some data of PARSONS[11] on longevity in *D. melanogaster*. Little difference was found between a series of hybrids derived from some inbred strains at the optimal temperature of 25 °C, but at the more extreme temperature of 29.5 °C, there was more variability of a genotypic nature, perhaps because the adaptation to this rather unfavourable environment depends on rather special gene combinations.

Temperatures of 33.5 °C would not be uncommon for short periods of time at Leslie Manor, Victoria, which is the locality of origin of the 3 strains under test. It is therefore reasonable that we should find evidence for some genetic control of the ability to withstand high temperatures. It is unlikely that genes conferring resistance to high temperature would become fixed in the population, because natural selection would not favour them, except at limited times during the year. However, because of the polymorphism, such genes would presumably be able to vary in frequency rapidly at appropriate times and enable adaptation to environmental changes as they occur. Furthermore, a polymorphic system of polygenes may allow colonization of new habitats, where temperature requirements may differ somewhat.

Table II. Analysis of variance of the percentage mortalities (after applying the angular transformation)

Sources of variation	Degrees of freedom	Mean square	Variance ratio
Temperature shock (T)	1	4836.8	72.52[b]
Sex (S)	1	203.2	3.05
Strain (St)	2	631.6	9.47[b]
Generation (G)	3	1402.7	21.03[b]
T × S	1	135.9	2.04
T × St	2	351.8	5.28[a]
T × G	3	1178.2	17.66[b]
S × St	2	100.3	1.50
S × G	3	77.4	1.16
St × G	6	454.3	6.81[b]
T × S × St	2	68.6	1.03
T × S × G	3	14.0	0.21
T × St × G	6	573.9	8.60[b]
S × St × G	6	144.2	2.16
Error	54	66.7	

[a] $P < 0.01$, [b] $P < 0.001$.

Table III. Analysis of variance of the percentage mortalities of control and temperature shocked flies separately (after applying the angular transformation)

Source of variation	Degrees of freedom	Control		Temperature shocked	
		Mean square	Variance ratio	Mean square	Variance ratio
Strains	2	21.4	0.65	420.6	14.15[a]
Generations	3	28.6	0.87	1093.7	36.79[a]
Strains × generations	6	47.5	1.44	388.4	13.06[a]
Error	12	32.9		29.7	

[a] $P < 0.001$.

Résumé. Chaque femelle *Drosophila melanogaster* sauvage, inséminée, isolée de son milieu naturel, a donné des descendances qui présentaient des différences génétiques concernant leur capacité à supporter le choc provoqué par de hautes températures. On peut donc émettre l'hypothèse que la population originelle de ces femelles est polymorphique quant aux gênes contrôlant cette aptitude.

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