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Genetic and Maternal Variation for Heat Resistance in Drosophila from the Field

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ABSTRACT

In Drosophila, field heritability estimates have focused on morphological traits and ignored maternal effects. This study considers heritable variation and maternal effects in a physiological trait, heat resistance. Drosophila were collected from the field in Melbourne, Australia. Resistance was determined using knockdown time at 37°. *Drosophila melanogaster* was more resistant than *Drosophila simulans*, and males tended to be more resistant than females. Field heritability and maternal effects were examined in *D. simulans* using the regression of laboratory-reared F_1 and F_2 onto field-collected parents. Males from the field were crossed to a laboratory stock to obtain progeny. The additive genetic component to variation in heat resistance was large and significant, and heritability was estimated to be around 0.5. A large maternal effect was also evident. Comparisons of regression coefficients suggested that the maternal effect was not associated with cytoplasmic factors. There was no correlation between body size (as measured by wing length) and heat resistance. Unlike in the case of morphological traits, the heritability for heat resistance in nature is not less than that measured in the laboratory.

THE ability of insects to counter environmental stresses is an important factor in determining their abundance and distribution. Responses to temperature extremes are likely to be particularly important because the distributions of many insects are constrained by extremes of heat and cold. Genetic variation in physiological resistance to temperature extremes and other climatic stresses will therefore help to determine the ability of insect populations to maintain their distributions by adapting to future climatic changes (HOFFMANN and PARSONS 1991; HOFFMANN and BLOWS 1993; LYNCH and LANDE 1993).

Genetic variation in heat resistance in Drosophila has been examined using both direct selection for increased heat resistance and indirect natural selection in the laboratory (*e.g.*, MORRISON and MILKMAN 1978; STEPHANOU and ALAHIOTIS 1983; QUINTANA and PREVOSTI 1990; HUEY *et al.* 1992). The responses suggest that there may be a significant heritable component to variation in physiological heat resistance. However, laboratory estimates are not necessarily relevant to field conditions, particularly if mortality levels are used as the sole measure of heat resistance. It is likely that there will be many effects of heat, such a reduction in fecundity and mating activity, long before mortality occurs (HOFFMANN and BLOWS 1993). Ideally, heritability needs to be measured in natural populations rather than in laboratory environments (RISKA *et al.* 1989). Unfortunately, this is difficult in most species, including Drosophila, where individuals cannot be followed across generations in the field.

Nevertheless, an estimate of heritability in nature can be obtained from a comparison of laboratory-reared offspring and their parents caught in the field, as first suggested by PROUT (1958). This estimate will be equivalent to the actual heritability in nature if there are no genotype-environment interactions and if the additive genetic variance is the same in the laboratory and the field (LANDE 1987). This method has been employed to estimate the heritability of wing length and abdominal bristle number in *Drosophila melanogaster* (COYNE and BEECHAM 1987), thorax length in *Drosophila buzzatii* (PROUT and BARKER 1989; RUIZ *et al.* 1991) and courtship song characters in *Drosophila montana* and *Drosophila littoralis* (ASPI and HOIKKALA 1993). The general conclusion of these studies is that heritability seems to be lower in nature than in the laboratory.

In this study, we investigate the genetic component of heat resistance in field-collected *Drosophila simulans*. We have measured heat resistance as the time flies take to be knocked down a tube, as described in HUEY *et al.* (1992). The principal advantage of this method is that it allows progeny to be obtained subsequent to measurement of heat resistance as flies are rarely killed or sterilized. The knockdown assay of heat resistance appears to reflect overall physiological tolerance and to be correlated with conventional assays of heat resistance (HUEY *et al.* 1992). By comparing the knockdown times of male and female parents and their laboratory-reared offspring, we examined the additive genetic variance for resistance. However, because we measured knockdown resistance on separate groups of F_1s, we could not

estimate the phenotypic variance in the progeny needed to obtain the lower bound for heritability in the field (see RISKA *et al.* 1989).

An important aspect of this work is to examine maternal effects generated under field conditions. These effects can bias estimates of heritability and genetic correlations, as similarities between mother and offspring may not only result from the transmission of nuclear genes (LANDE and PRICE 1989). The genotype of the mother can influence the environment experienced by her offspring before and after egg laying, such that environmental and genetic differences in one generation are manifested as phenotypic differences in the next generation (MOUSSEAU and DINGLE 1991), and this has been demonstrated for heat resistance in *D. melanogaster* (W. D. CRILL and R. B. HUEY, personal communication). Maternal effects may also arise because of the transmission of cytoplasmic factors such as mitochondrial DNA or because the maternal environment influences the progeny phenotype.

A subsidiary aim is to investigate the association between knockdown resistance and the body size of the flies. Female Drosophila may be more resistant than males to heat (HUEY *et al.* 1992), and there is some evidence that increased stress resistance in females may be equated to the size difference between the sexes (PARSONS 1973). Geographical clines reported for body size in Drosophila are thought to be due to climatic factors (COYNE and BEECHAM 1987) and a correlation between heat resistance and body size has often been assumed (TANTAWY and MALLAH 1961; ANDERSON 1973).

MATERIALS AND METHODS

Field collections: Flies of an unknown age were collected from four suburban sites in Melbourne, Australia, in early June 1992, using traps containing oranges. Flies were collected on three separate occasions and were tested for knockdown resistance within a few hours after removal from the traps. Flies were held at 25° before being placed into the knockdown tube.

Heat stress: Flies were heat stressed in a controlled temperature room at 37 ± 1°, with a relative humidity of 25 ± 2%. A glass knockdown tube (Figure 1) was used, based on the design of HUEY *et al.* (1992). The tube was placed directly beneath an overhead light. Flies remained at the top of the tube because of positive phototropism and negative geotropism exhibited by Drosophila. Flies were allowed to settle before the shutter was opened, and fell down the tube as they succumbed to heat stress. To ensure that flies did not simply wander out, movement down the tube was impeded by baffles.

Flies falling into a collecting vial were incapacitated to the extent that they were unable to fly or walk up the tube. This vial was replaced at 30-s intervals and each individual fly given a knockdown time according to when it fell into the collecting vial. Collecting vials were placed in a container maintained at around 25° by ice, to prevent death and sterility from heat stress. At least 20 flies were tested in a knockdown tube, but no attempt was made to control absolute numbers as preliminary tests revealed that mean knockdown time was not influenced by fly density. This apparatus provides a repeatable measure of heat resistance. Pilot experiments indicated that runs on the same day with flies from the same population had mean knockdown times that were not more than 30 s apart.

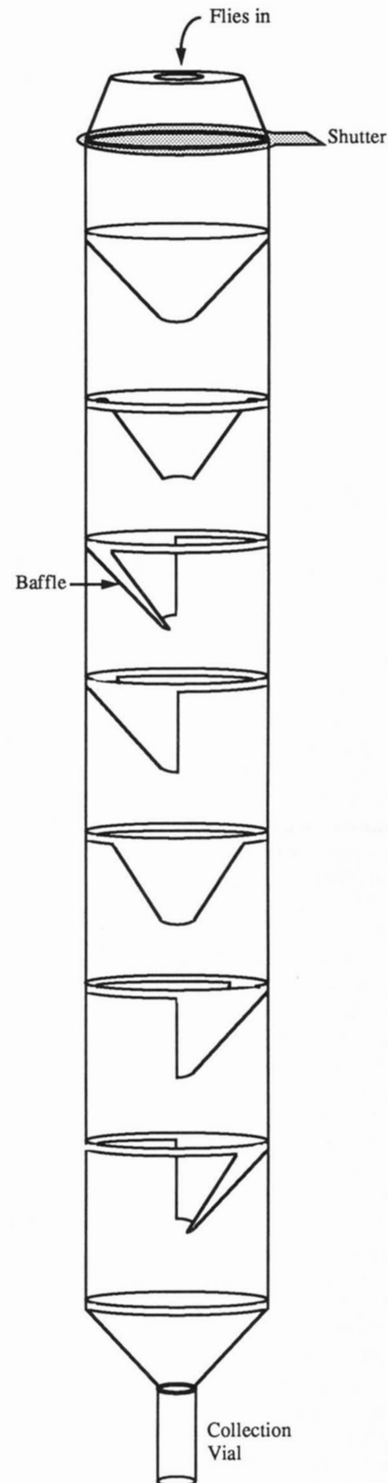

FIGURE 1.—Knockdown tube for measuring heat resistance of Drosophila.

After being stressed, flies were sorted according to sex and species under CO_2 anaesthesia. Although other species were also collected from the field, *D. melanogaster* and *D. simulans* were the only species for which 37° was appropriate to measure heat resistance.

The heat resistance of a control line was determined at the

TABLE 1

Heat resistance of field-collected flies

	No. of flies	Mean knockdown time (min)	Range	Variance (min^2)	Coefficient of variation (%)
D. simulans					
Males	111	1.8	0.5–8.5	2.06	78.5
Females	135	1.6	0.5–4.5	0.95	59.9
D. melanogaster					
Males	19	7.8	1.5–18.5	25.84	65.6
Females	18	6.6	1.0–17.5	17.35	63.4

beginning of each testing day to control for variation in temperature and relative humidity between days. Only one control run was carried out because environmental conditions were constant on a particular day. A *D. melanogaster* isofemale line was used as the control line. This line had been maintained in the laboratory for two years and originated from Cairns, Australia. The control line was cultured in 300-ml glass bottles with 80 ml of yeast medium at a density of 20–30 adults per bottle. During eclosion, bottles were cleared of flies each morning and newly eclosed flies were collected in the afternoon. Prior to stressing, flies were kept for four days at a density of 70–100 per bottle, so all control flies were 4 days old at the time they were tested for resistance.

Laboratory generations: All laboratory rearing was carried out at 24–25° under continuous light. Field females were set up individually in 30-ml vials with 10 ml of a dead yeast-sucrose medium sprinkled with live yeast to stimulate oviposition. A total of 135 *D. simulans* isofemale lines were obtained. After 4 days the field females were transferred to fresh vials to obtain a replicate. After a further 4 days the females were removed and frozen to be used for the body size analysis (see below).

The same procedure was followed with the field males, except that they were mated to virgin females from a *D. simulans* isofemale line. This line had been maintained in the laboratory for 2 years, and was derived from a single female collected from Melbourne in 1990. A total of 96 lines were obtained from the field-collected *D. simulans* males.

F_1 flies were collected over a period of three days from each replicate. They were maintained at a constant density of 15–25 flies per vial, placed on fresh medium 2 days prior to stressing, and heat stressed when 4–6 days old. As it was not feasible to stress the progeny of each line separately, offspring of lines with the same maternal knockdown times (or paternal times in the case of comparisons with the male parents) were pooled for stressing. Because of the need to control for age effects, runs were only done with offspring emerging at the same time. This usually meant that several runs were carried out for each parental knockdown time. In total there were 40 runs with F_1s from the female parents and 46 runs with F_1s from the male parents. These runs were executed over a number of days and hence the mean knockdown value for a run was corrected using the appropriate value of the control line in a multiple regression (described below). Note that each run and not each line was treated as an independent data point in the regression analyses.

After the F_1s from field-collected females had been stressed, 10 males and 10 females from each run were chosen at random and pairs of flies were set up in vials to produce the next generation. This means that flies were mated to their sibs or to unrelated individuals whose mother had the same knockdown time. Upon eclosion, the F_2 generation was treated in the same manner as the F_1s to obtain mean knockdown scores. Progeny from field-collected males were not bred to the F_2 generation.

Estimating genetic components: Regressions of offspring onto male or female parents/grandparents were used to estimate variance components. Mean knockdown times of progeny were used in the regression analysis. The regression coefficient (b) can be related to the narrow-sense heritability with the assumption of no genotype by environment (G × E) interactions. Because day-to-day variation in environmental conditions affected the phenotypes of progeny, a multiple regression was performed to eliminate this source of variance before the parent-offspring regression was carried out (see SOKAL and ROHLF 1981). Day-to-day variation was determined from knockdown times of the control line tested each day. We subsequently calculated the expected mean of the laboratory progeny, based upon the knockdown time of the field parents. For the F_1 to F_2 comparisons, day-to-day variation in two generations of flies had to be controlled. For this purpose, the F_1 values were corrected using the equation

$$Y_{adj} = Y - bx,$$

where b is the regression coefficient obtained from the first step of the parent-F_1 multiple regression and x is the control mean (SOKAL and ROHLF 1981, p. 493). Using the adjusted F_1 values, multiple regressions were performed as above.

Body size: Body size was measured by examining the third longitudinal vein, from its intersection with the anterior crossvein to the wing tip. This measure is highly correlated genetically and phenotypically with overall size (ROBERTSON and REEVE 1952). The wings of flies were removed using fine forceps, mounted on microscope slides and measured using an image analyzer (Trace). Each vein was measured on two separate occasions to ensure that the technique provided a valid measurement. A Pearson correlation coefficient of 0.988 between scores for these repeat measurements ($P < 0.001$, n = 62) indicated that the method was highly repeatable.

RESULTS

Species, sex and size effects: Mean knockdown times of field-collected *D. melanogaster* and *D. simulans* are given in Table 1. *D. melanogaster* seems to be more resistant to heat than *D. simulans*. The variance of knockdown times was also examined to compare variability in resistance. The knockdown times of *D. melanogaster* are more variable than those of *D. simulans*. However, this species difference disappears once coefficients of variation are compared (Table 1).

Sex differences in *D. simulans* were investigated by comparing the knockdown times of males and females in all runs. Males tended to be more resistant to heat than females; when all the F_1 and F_2 data are considered,

TABLE 2

Regression coefficients corrected for differences in variances between the sexes and corresponding "heritability" estimates (assuming no maternal effects)

Regression	$b \pm$ SE	P	h^2
From field females			
Dam-son	0.40 ± 0.10	0.0008	0.80 ± 0.20
Dam-daughter	0.35 ± 0.07	0.0001	0.70 ± 0.14
Dam-grandson	0.14 ± 0.09	0.0625	0.28 ± 0.18
Dam-granddaughter	0.15 ± 0.07	0.0180	0.30 ± 0.14
Son-grandson	0.21 ± 0.11	0.0287	0.22 ± 0.12
Son-granddaughter	0.17 ± 0.09	0.0287	0.18 ± 0.09
Daughter-grandson	0.28 ± 0.11	0.0092	0.29 ± 0.12
Daughter-granddaughter	0.21 ± 0.10	0.0168	0.22 ± 0.10
From field males			
Sire-son	0.24 ± 0.09	0.0095	0.48 ± 0.18
Sire-daughter	0.23 ± 0.08	0.0075	0.46 ± 0.16

Probabilities are for one-tailed *t*-test to determine if regression coefficients are greater than 0.

males had a higher mean knockdown time than females in 166 out of 172 runs (Sign test, $P < 0.001$). In addition, *D. simulans* males had a greater variance in knockdown time than females in 158 out of 172 runs (Sign test, $P < 0.001$). In contrast to these results, Huey *et al.* (1992) found that *D. melanogaster* females were relatively more resistant to knockdown than males.

The relationship between size and heat resistance in field *D. simulans* was investigated by computing Pearson correlation coefficients between knockdown time and wing length of the females. Correlation coefficients for three collections were 0.02 ($N = 62$), -0.07 ($N = 9$) and 0.10 ($N = 49$). None of these coefficients are significant, so there is no evidence that size (as measured by wing length) influenced knockdown resistance, consistent with the results of Oudman *et al.* (1988). We therefore did not measure wing size in progeny generations.

Parent-offspring regressions and variance components: Multiple regressions were performed using the knockdown times of parents, F_1 and F_2 progeny, and the control line. The difference in phenotypic variance between the sexes biases across-sex regressions, which we corrected by multiplying by the appropriate ratio of phenotypic standard deviations. For example, the regression of dam to male offspring is adjusted by the ratio of female to male standard deviations (Falconer 1989). The adjusted regression coefficients and their standard errors are given in Table 2. One-tailed *t*-tests were carried out to determine if regression coefficients were significantly greater than 0. Probabilities for these tests (Table 2) indicate that all regression coefficients are significant at least at the 0.05 level, with the exception of the dam-grandson regression which is almost significant.

To interpret these regression coefficients, we need to look at factors contributing to similarity between relatives. Maternal effects may arise from several sources (Willham 1963; Eisen 1967). They may arise from inheritance that is strictly maternal, as in the case of cytoplasmic factors that are passed on through the female parent. They may also arise because aspects of the maternal genotype (beyond that which is inherited) influence progeny phenotypes. Finally, maternal effects may be solely environmental, in that the environment experienced by a female influences the phenotypes of her progeny. These sources lead to three variance components: the maternal cytoplasmic variance (σ^2_{MC}), the maternal additive genetic variance (σ^2_{MA}), and the maternal environmental variance (σ^2_{ME}).

The components of variance contributing to covariances in the current mating design are given in Table 3. If there are no G × E interactions, then the sire-F_1 regressions should provide the best estimate of narrow-sense heritability because additive genetic variance should be the sole variance component (assuming no epistasis). An estimate of narrow-sense heritability, the proportion of phenotypic variance that is accounted for by additive genetic variance, can then be obtained by doubling the parent-offspring regression coefficient. In contrast, an estimate of narrow-sense heritability from doubling of the dam-F_1 regressions may be inflated by all three sources of maternal effects.

The F_1-F_2 regressions require adjustment for assortative mating because F_1 males were mated with F_1 females with a similar knockdown time due to inbreeding of the F_1 generation. This means that knockdown times of the F_1 parent not included in the regression are similar to those included in the regression. The covariance between offspring and parents can be defined as $\frac{1}{2}\sigma^2_A(1 + r)$, where r is the phenotypic correlation between mates (Falconer 1989, p. 178). The phenotypic correlation was estimated to be 0.91 on the basis of the correlation between knockdown times of the female and male F_1s. The F_1-F_2 covariance therefore included $0.955\sigma^2_A$. The dam-F_2 and F_1-F_2 regressions included maternal cytoplasmic variance as well as additive genetic variance. This is because F_1s from mothers with the same knockdown times were crossed to produce F_2s. Any cytoplasmic differences between field females affecting heat resistance were therefore passed to the F_1s and F_2s. Some of the maternal additive genetic variance and maternal environmental variance may also be carried over from field females to the F_2 generation, although this is ignored in Table 3.

The additive genetic variance component in the dam-grandchild comparison requires some explanation because this is usually given as $\frac{1}{4}\sigma^2_A$. Recall from the methods section that F_1s from field-collected mothers with the same knockdown times were mated together to produce F_2s. This means that two F_1s crossed to produce the F_2 generation may have the same mother, or else a different mother but with the same knockdown score. In either case, we have to adjust for assortative mating among

TABLE 3

Expectations of variance components in the current mating design

Comparison	Variance component: No G × E	G × E
Dam-F_1	$\frac{1}{2}\sigma_A^2 + \frac{1}{2}\sigma_{MA}^2 + \sigma_{MC}^2 + \sigma_{ME}^2$	$\frac{1}{2}\gamma_1\sigma_{AL}\sigma_{AN} + \frac{1}{2}\gamma_2\sigma_{MAL}\sigma_{MAN} + \gamma_3\sigma_{MCL}\sigma_{MCN} + \sigma_{ME}^2$
Dam-F_2[a]	$0.48\ \sigma_A^2 + \sigma_{MC}^2$	$0.48\ \gamma_1\sigma_{AL}\sigma_{AN} + \gamma_3\sigma_{MCL}\sigma_{MCN}$
F_1-F_2[b]	$0.955\ \sigma_A^2 + \sigma_{MC}^2$	$0.955\sigma_{AL}^2 + \sigma_{MCL}^2$
Sire-F_1	$\frac{1}{2}\sigma_A^2$	$\frac{1}{2}\gamma_1\sigma_{AL}\sigma_{AN}$

Maternal additive effects and maternal environmental effects are assumed to be passed from flies collected in nature to the first laboratory generation but not to the second laboratory generation. The F_1-F_2 comparison only involved progeny from field females. γ_1, γ_2, γ_3 = additive genetic correlation, maternal genetic correlation and maternal cytoplasmic correlation respectively between a trait in nature and the same trait in the laboratory. σ_A^2 = additive genetic variance; σ_{MA}^2 = maternal genetic variance; σ_{MC}^2 = maternal cytoplasmic variance; σ_{ME}^2 = maternal environmental variance; σ_{AL}^2, σ_{AN}^2 = additive genetic variance in the laboratory and in nature; σ_{MAL}^2, σ_{MAN}^2 = maternal genetic variance in the laboratory and in nature; σ_{MCL}^2, σ_{MCN}^2 = maternal cytoplasmic variance in the laboratory and in nature.

[a] Estimate of 0.48 based on the assumption that F_1s had different mothers and on a correction for the phenotypic correlation between mates in the F_1s (see text).

[b] Estimate of 0.955 based on correction for phenotypic correlation between mates in the F_1 generation (see text).

the F_1s. Consider first the situation where F_1s producing the F_2 generation are assumed to have a different mother. Following CROW and KIMURA (1970, p. 158) and assuming additive genetic effects, the grandparent-child covariance is given as

$$\mathrm{cov} = \sigma_A^2 \frac{1+r}{2} \frac{1+A}{2}$$

In this equation, r is the phenotypic correlation between the F_1 parents, and A is the correlation between genic values of the field parents given as $r\sigma_A^2/\sigma_T^2$. A is used instead of r because only the genic part of the correlation contributes to the variance of the F_2s. If we assume no assortative mating in the field flies, then $A = 0$ and the covariance becomes $0.48\sigma_A^2$ because $r = 0.91$ (see above).

Now consider the situation where F_1s are assumed to have the same mother. In this case, we can determine the dam-grandchild covariance by carrying out a pedigree analysis. If there are no dominance effects or epistasis, the covariance between two relatives (X, Y) is given by $r\sigma_A^2$, where $r = 2f_{XY}$ and f is the consanguinity coefficient (CROW and KIMURA 1970, p. 138). In our design, F_1s have the same parents and mate together to produce F_2s. A path analysis can be used to determine f, the inbreeding coefficient of a hypothetical offspring between the dam and an F_2 in this pedigree. There are three individuals in the path from the dam to the F_2 via an F_1, and two possible paths because two F_1s mate to produce the F_2s. The consanguinity coefficient is therefore given as $(\frac{1}{2})^3 + (\frac{1}{2})^3$ or $\frac{1}{4}$, resulting in a covariance of $\frac{1}{2}\sigma_A^2$. This estimate does not need to be corrected for assortative mating in the F_1s, which only arises as a consequence of inbreeding imposed by our experimental design. Because $\frac{1}{2}\sigma_A^2$ is close to the $0.48\sigma_A^2$ value estimated above, it does not matter much whether F_2s are assumed to arise from matings between F_1s with different mothers (but with the same phenotype) or between F_1s with the same mother.

We have so far ignored non-additive genetic effects and differences between laboratory and field environments. Additivity may be a reasonable assumption based on information about the genetic architecture of stress resistance traits in general (HOFFMANN and PARSONS 1991). However, when comparing field flies and their offspring raised in the laboratory, there is the possibility of genotype-environment interactions resulting in different variance components in the field and laboratory. We can modify the notation employed in RISKA *et al.* (1989) to incorporate all nuclear and cytoplasmic heritable effects. Following these authors, we use γ to represent the correlation for heritable effects between a trait in nature and the same trait in the laboratory. The variance components can be represented as a product of this correlation, the laboratory standard deviation and the standard deviation in nature. If there are no genotype-environment interactions, $\gamma = 1$ and the variance components are the same in the field and the laboratory. The expected variance components for the parent/grandparent-offspring comparisons, allowing for differences between the laboratory and field conditions, are given in Table 3.

The mean estimates of the regression coefficients (from Table 2) are: 0.38 for dam-F_1, 0.15 for dam-F_2, 0.21 for F_1-F_2 and 0.24 for sire-F_1. Because the sire-F_1 regression coefficients are not less than that of the dam-F_2 and the F_1-F_2 coefficients, cytoplasmic factors do not seem particularly important in the inheritance of heat resistance in *D. simulans*. Note that the F_1-F_2 comparisons only involved the progeny of field females because progeny from field males were not bred to the F_2 generation. The higher values of the dam-F_1 coefficients compared to the sire-F_1 coefficients are therefore probably due to maternal additive genetic variance and maternal environmental effects rather than cytoplasmic variance.

The higher heritability estimates obtained from the sire-F_1 regressions compared to the F_1-F_2 regressions suggest that γ_1 is close to 1.0 and that the additive genetic

standard deviation for flies raised in nature is greater than that for flies raised in the laboratory. Therefore, the additive genetic variance measured in the laboratory is a conservative estimate of that in nature. To obtain a lower bound for heritability in nature, an accurate estimate of the phenotypic variance of laboratory progeny is required (RISKA *et al.* 1989). Unfortunately, small temperature changes while heat stressing different batches of laboratory progeny preclude pooling data from F1s to obtain an overall estimate of the phenotypic variance.

DISCUSSION

The best estimate of heritability for heat resistance in the Melbourne population of *D. simulans*, as provided by the sire-son regression, is 0.48 ± 0.18. This high narrow-sense heritability suggests that the population we studied has a high potential to adapt if the climate becomes warmer. This result contrasts with laboratory studies on heat resistance in *D. melanogaster* which suggest fairly low levels of genetic variation (*e.g.*, MORRISON and MILKMAN 1978; KREBS and LOESCHCKE 1994) although HUEY *et al.* (1992) obtained a heritability estimate of 0.28 for knockdown resistance in this species. Perhaps *D. simulans* have a higher heritability for heat resistance than *D. melanogaster*, although this seems unlikely judging by comparisons between these species for other stress resistance traits (*e.g.*, COHAN and HOFFMANN 1989; HOFFMANN and PARSONS 1993). Another possibility is that the heritability is higher in field populations than in laboratory populations because of inbreeding or the small number of founders often used to initiate and maintain laboratory populations.

Apart from providing evidence for high levels of genetic variance, this study also demonstrates a strong maternal effect for field-collected flies. Both sons and daughters were more similar to their mothers than their fathers. Because this effect was not passed down to the F_2 generation, cytoplasmic inheritance does not seem to be important. This contrasts with the results of STEPHANOU and ALAHIOTIS (1983) who found that cytoplasmically inherited factors largely determined an indirect selection response for heat resistance in *D. melanogaster.*

The environment experienced by field females or a delayed influence of nuclear genes could contribute to the maternal effect. One possible source of environmental influence is field acclimation. The heat resistance of Drosophila adults can be increased by prior exposure of adults or larvae to non-lethal temperatures (*e.g.*, LEVINS 1969). It is possible that such an increase may be passed to F_1 offspring. In Drosophila, environmental effects associated with acclimation that are passed to the next generation are known for resistance to cold (M. WATSON, personal communciation) and heat (W. D. CRILL and R. B. HUEY, personal communication). Other possible sources of environmental influences include disease and nutritional factors passed on from mothers to progeny which may indirectly influence heat resistance. Nuclear effects may arise because of proteins and long-lived mRNA present in the egg cytoplasm that exert their effects during development.

The results indicate that the heat resistance of *D. melanogaster* is higher than that of *D. simulans* when flies from the field are compared. This difference is consistent with the higher resistance of *D. melanogaster* to other stresses such as desiccation and ethanol (PARSONS 1983). Although the ecological significance of this result is not clear, *D. simulans* numbers in temperate Australia tend to increase in autumn and winter whereas *D. melanogaster* numbers peak in summer when heat stress is at a maximum (MCKENZIE and PARSONS 1974; NIELSEN and HOFFMANN 1985).

It has often been suggested that heritability in nature will be less than that measured in the laboratory, mainly because of greater environmental variability under field conditions. This seems to be supported by field estimates of size-related traits in several Drosophila species (COYNE and BEECHAM 1987; PROUT and BARKER 1989; HOFFMANN 1991; ASPI and HOIKKALA 1993). However, our results suggest that heritabilities need not be lower for physiological traits. If this finding holds for other traits, laboratory estimates may not be accurate indicators of the potential of natural populations to respond to directional selection on physiological traits.

Our high heritability estimates raise a number of questions about the evolution of heat resistance in *D. simulans* populations. Because levels of resistance should change readily under directional selection, higher resistance levels would be expected in populations frequently exposed to hot periods. This prediction can be tested by geographical comparisons of *D. simulans* populations which occur over a range of climatic conditions. Mean heat resistance in populations might also be expected to change seasonally as populations experience hot conditions in summer. Such seasonal genetic changes have been detected in Drosophila populations for body size (*e.g.*, TANTAWY 1964; THOMAS 1993) which seems to have a lower heritability under field conditions (*e.g.*, COYNE and BEECHAM 1987). Obviously, predictions about seasonal changes depend on the knockdown assay for heat resistance being relevant to the survival of Drosophila adults at high temperatures under field conditions.

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