Chapter 3

Estimation of Genetic Parameters

3.1 Introduction

This experiment measured the genetic variance and covariance components of life-history traits of a population of *Tribolium castaneum* recently derived from the wild and studied under optimum environmental conditions.

A number of designs for the estimation of phenotypic and genetic parameters were considered. The use of inbred lines was rejected because they are not representative of the original population from which they are drawn, and can generate positive genetic correlations. A parent-offspring regression would be difficult because of the problem of standardisation of conditions for both parents and offspring, particularly for the measurement of developmental rate and reproductive indices. In addition, sib analyses with optimal structures are preferable to offspring-parent regressions for the estimation of low heritabilities, approximately 25% and less, when an equal total number of individuals are measured (Robertson 1959). Since the number of individuals measured was the limitation in this experiment and heritabilities of fitness components were expected to be low, a sib analysis was chosen over a parent-offspring regression. Two common approaches to sib analysis are the heirarchial and diallel designs.

Sheridan, Frankham, Jones, Rathie and Barker (1968) compared these two designs for partitioning variance and estimating genetic parameters for various bristle number characters of *Drosophila melanogaster* and recommended "the use of the diallel analysis is advisable for estimating genetic parameters and partitioning variation, especially when epistasis, sex-linkage, or maternal effects are present". As there is evidence that epistasis is an important

component of some traits in *Tribolium* (Goodwill 1975; Goodwill and Walker 1978), the diallel design was deemed to be most appropriate.

Thus a diallel analysis was used to obtain estimates of phenotypic and genetic parameters for a number of major life-history characters such as: time to pupation, time to adult emergence, body weight, growth rate, peak fecundity in a 24 hour period, day of peak fecundity, total lifetime fecundity, day of death, adult life-span, reproductive life-span and various indices of the reproductive schedule.

These estimates were used to evaluate:

1) the veracity of the reproductive cost theorem; and

2) the numerous explanations for the evolution of senescence.

As the beetles used in this analysis were third generation descendents of adults collected from the wild and efforts were made to retain the population's original character as much as possible once transferred to the laboratory, it was possible to examine and conjecture about the evolutionary significance of the genetic variance-covariance matrix and the genetic constraints on life-history evolution.

Based on these estimates of genetic variation and correlation between traits, predictions were made of expected correlated responses to selection for developmental rate in either direction. In a later experiment developmental rate was selected in both directions, thus it was possible to compare these predictions with the actual outcome of a selection programme.

3.2 Materials and Methods

3.2.1 Experimental Procedures

Sections 2.2 and 2.3 contain descriptions of the methods and materials used for the generations preceeding the diallel analysis. Throughout this study the same laboratory husbandry procedures are used unless otherwise stated.

Figure 3.1 is a diagrammatic presentation of the experimental procedure, whilst Figure 3.2 gives the chronological sequence of procedures. Both figures are supplementary to the text.

The crossing scheme was based on that described by Lerner (1950) and utilised 54 units of two sires x two dams, intercrossed within each unit. As demonstrated by Dawson (1965b), it is possible to use this design with *Tribolium castaneum,* because in a single-pair mating

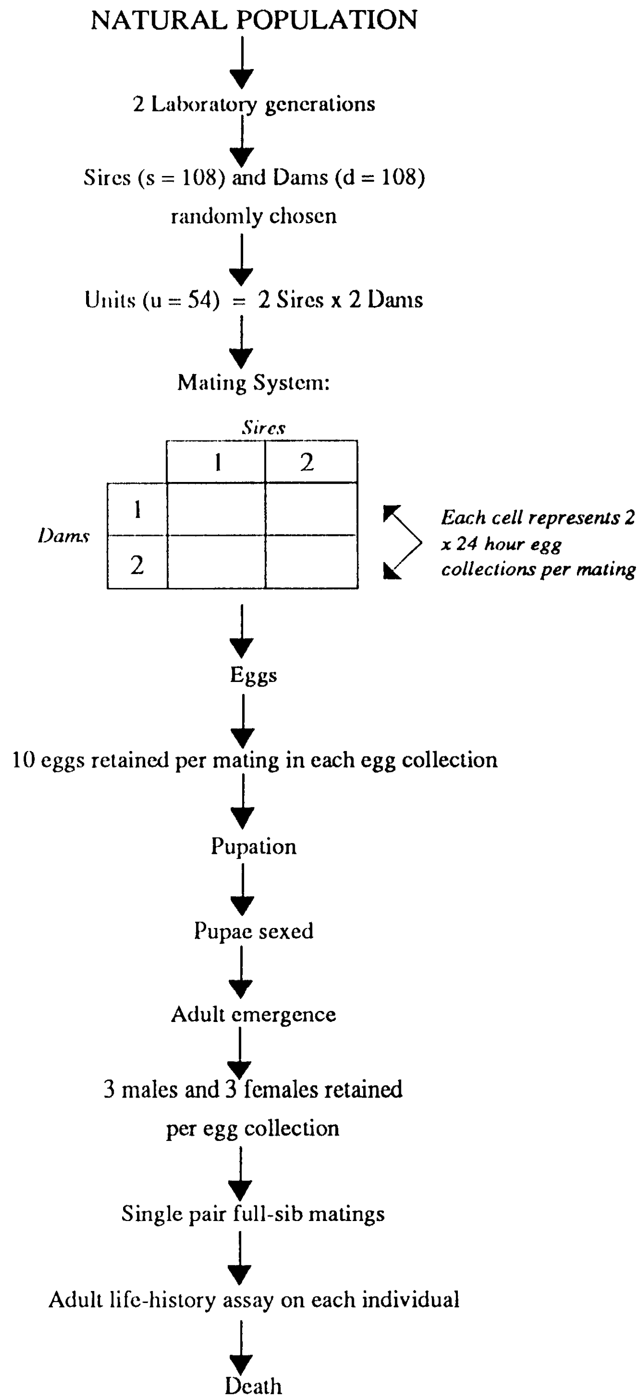

Figure 3.1: Schematic presentation of the Diallel experiment

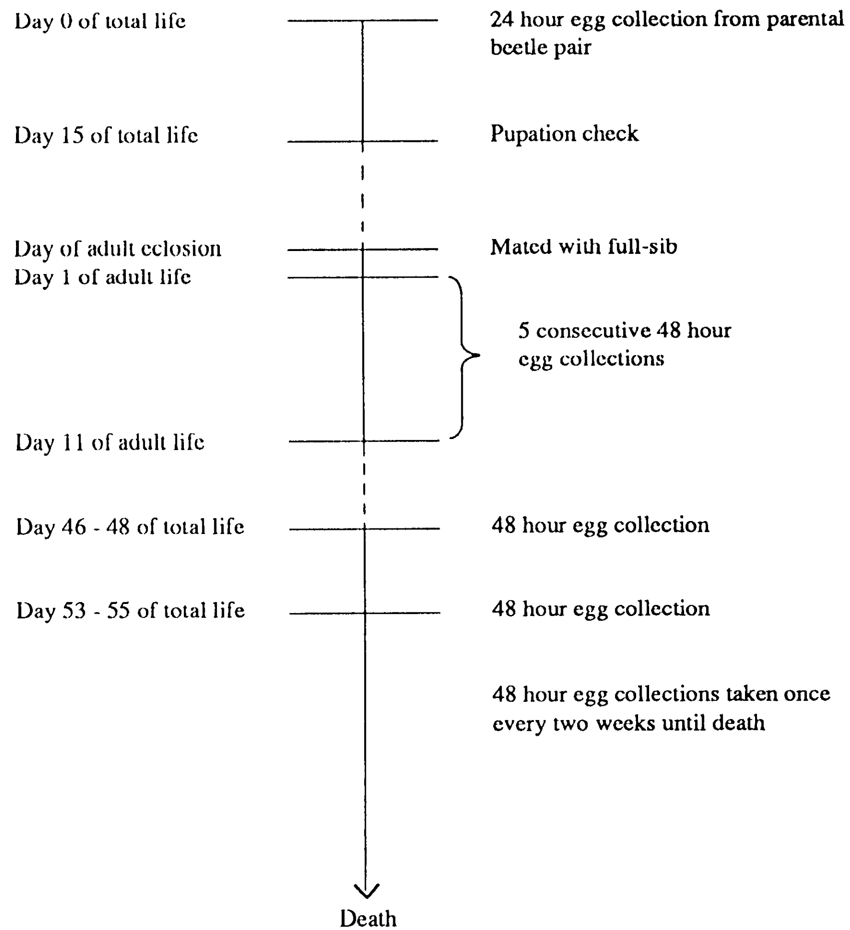

Figure 3.2: Chronological sequence of measurements taken on individual female beetles

if the first male is replaced by a second male, after a few days all eggs laid by the female will have been fertilised by the second male: i.e. "sperm precedence" (Schlager 1960).

The number of offspring needed to give good estimates of heritabilities and genetic correlations was estimated prior to the commencement of the experiment. Fortunately, the optimum structure for the measurement of heritability is also the optimum for the measurement of the genetic correlation between two characters (Robertson 1959). No formula was found for a diallel design, but the optimal design of an hierarchal design is 3 or 4 dams per sire with the number of offspring per dam equal to $\frac{2}{h^2}$ (Robertson 1959), when it is desired to estimate the correlations from sire and from dam components with equal precision. This formula, $\frac{2}{h^2}$, was used as a rough guide for the diallel design.

Examples of estimates of heritability for *T. castaneum* vary from 0.11 to 0.38 for developmental rate (Dawson and Riddle 1983); 0.16 to 0.35 for 30 day adult weight (Okada and Hardin 1967;1970) and 0.36 for four day virgin egg lay (Orozco and Bell 1974). Taking 0.2 as a reasonable average heritability, $2/0.2 = 10$ progeny are needed.

As separate estimates of heritabilities of some traits should be made for each sex and some traits can only be measured on females, each dam must produce 10 females and 10 males. Individual females at peak egg production can produce over 20 eggs per day but usually produce between 10 and 20 eggs (see Bhat and Bhat 1974b). It was assumed then, conservatively, that one should be able to obtain 10 eggs per female per 24 hours.

The probability of obtaining 3 males and 3 females from (likely) 10 eggs per female per 24 hour egg collection was 0.8907, whilst the probability of obtaining 4 pairs was 0.6563 and of 2 pairs 0.9346. To achieve adequate numbers of progeny it was decided that there was not much benefit and alot more work and confusion in taking 3 egg collections per mating and getting two pairs per egg collection, than trying to obtain 3 pairs per egg collection and collecting twice from each mating. This last procedure would at its optimum produce 12 females and 12 males per dam, leaving a margin of 2 progeny for sires and dams who produced less than the optimum of 3 males and 3 females at any or all egg collections.

From previous work, it was determined that 1200 female progeny were the maximum possible workload that could be handled. 54 units would produce 1296 females if all dams produced 3 pairs of progeny but death and loss would ensure that numbers would be below 1200.

It was decided to use 108 dams and 108 sires, but because of possible loss through mortality and infertility, 180 males and 180 females were initially randomly selected from

the newly emerged adults of the second generation and set up as single pairs. These beetles were individually weighed and the weights recorded (Appendix 2).

After 8 days, all pairs were transferred into fresh 2 g media for 24 hours (Egg collection 1 — day 0 of total life), then removed and put into fresh media for another 24 hours (Egg collection 2 — day 0 of total life). Both sets of vials were retained. The eggs laid in each vial were counted, recorded (Appendix 2) and a maximum of 10 eggs from each egg collection from each pair were seeded into vials containing fresh 2 g media.

A maximum of 10 eggs was set so that density, number of larvae/gram media, would have little effect on the developmental rate of larvae (Dawson 1965b; Howe 1962), for as larval crowding gets more severe, the rate of larval development decreases. However, the effects of density decrease with a decrease in handling and sifting of larvae (Kence 1973; Mertz and Robertson 1970), because the major factor inhibiting pupation at high densities appears to be tactile stimuli resulting from mutual jostling of larvae (Kence 1973). The earlier the flour in the cultures is sifted and the more frequent the interference with the cultures, the more drastic is the prolongation in developmental period and decrease in survival (Kence 1973). To minimise the effects of density and handling, eggs were left undisturbed to develop until day 15.

Immediately after the first two egg collections 108 pairs were selected at random after elimination of four pairs when one member had died, two pairs which produced no eggs and one pair which produced abnormal eggs (lacking the normal mucilaginous covering). All other pairs produced at least four eggs in each of the two periods.

The 108 pairs were divided into 54 groups consisting of two pairs per unit and the dams were exchanged between sires within units. Thus sire 1, who had been mated to dam 1, was now mated to dam 2, whilst dam 1 was mated to sire 2. Sixteen days later all pairs were transferred into fresh 2 g medium and two twenty-four hour egg collections were set up as described above (egg collections 3 and 4). The sixteen days delay guaranteed the second sire's sperm precedence. Within this period there should have been no parental age effect on developmental time or longevity between the first and last egg collection (Dawson 1965b; Soliman and Lints 1975). However, one female died within this period so one unit was missing progeny from one mating for the analysis.

Thus within the diallel crossing scheme there are two 24 hour egg collections from each female per male. The two replicates of each mating were set up to allow computation of common environmental effects on developmental time and to increase the total numbers

of progeny (see section 3.2.2 for *a priori* estimation of experimental numbers required to give precise estimates of genetic parameters). From each day (block) of egg collection, a maximum of 108 vials were obtained. Within each of the 4 blocks the 108 vials were randomised within a tray, and the four trays were placed into the incubator at random on the same shelf.

From day 15 of each egg collection, vials were checked daily for pupae. Pupae were sexed, and their day of pupation and sex recorded. Pupae were placed into vials so they could be protected from cannibalism and identified by their sire, dam, sex and day of emergence. Larvae that had not pupated by day 26 were discarded. Day 26 was determined as the cutoff point for all egg collections, as observations of egg collection 1 at day 26 showed:

1) less than 1% of the progeny remained as larvae; and

2) all remaining larvae were abnormally small and showed no sign of pupation.

Four days after the earliest pupae pupated, pupae were checked daily for adult emergence. The day of adult emergence for each individual was recorded.

Three males and three females, or as many as were available, were selected from each vial across the range of pupation. This was so the subsample of progeny would not be prejudiced by unconscious selection for early, late or intermediate developing beetles. Extra males were retained and set up in a single-sex culture.

Beetles were randomly paired with a full-sib of the opposite sex and set up as single pairs in 0.5 g medium. It was assumed that full-sib matings should not affect oviposition rates of females, though the hatchability of eggs laid might be affected by inbreeding. There was only one restriction on the full-sib mating and that was that females were mated with males that emerged on the same day or were at the most one day younger. If necessary spare full-sib males from the replicate block were mated with their sisters but were excluded from analysis. *Tribolium castaneum* males are mature on the second day of imaginal life (Erdman 1964), whilst females can be fertilised within 3 hours after eclosion and lay fertile eggs at 96 to 108 hours of age (Dawson 1964). Virgin females lay eggs at a much lower rate than fecundated females (Bhat and Bhat 1974a). If males were substantially younger than females, fertilisation of females could not have been guaranteed and this might have affected the initial number of eggs laid by some females.

All full-sib matings between progeny from one egg collection were randomised and kept together as one unit in the incubator. Thus there were four blocks of vials, each block

corresponding to an original egg collection.

The first egg collection from each female was initiated the day after eclosion of the female, when both male and female were transferred into a vial containing 1 g medium. This and subsequent egg collections were for 48 hours and females laid their eggs in 1 g medium.

The 48 hour time period was deemed more appropriate than 24 hours because:

1) It was no longer necessary to synchronise larval age.

2) Frequent handling depresses oviposition (Mertz and Robertson 1970; Reynolds 1944).

3) Longer egg lay periods are less likely to experience environmental variance than shorter periods, and thus the repeatability of rate of egg lay is increased (Orozco and Bell 1974). For fecundated females the egg lay period could not be extended beyond 48 hours due to hatching of eggs (Orozco and Ruano 1970).

4) Cannibalism is no more of a problem for 48 hour egg lays than 24 hour egg lays, particularly if the food is replaced at every sieving, as it was in this experiment (Howe 1962).

Eggs from the 48 hour collections were counted, their numbers recorded and then discarded. 48 hour egg collections were repeated four more times until day 11 of the adult life of each female. By day 11, females should have attained peak egg lay rate. To synchronise the egg collections of all females from one block, the next egg collection spanned days 46-48 of total life. The next 48 hour collection was days 53-55 and from thence egg collections were taken once every two weeks until the female's death.

When adults were transferred for egg collection, dead adults were removed and the day they were found recorded as their day of death.

Sexual activity may shorten the life-span of *T. castaneum* males (Spratt 1980) and of females (Lavie and Stern 1978; Lloyd and Park 1962; Spratt 1980). Therefore, to standardise culturing conditions, if the male partner of a pair outlived the female, a female beetle of a black mutant strain was placed with him until his death. The original stock of the black mutant strain was supplied by Dr. DuWayne C. Englert, Dept. Zoology, Southern Illinois University, U.S.A., in July 1982 and subsequently maintained in large numbers in 400 g medium, changed monthly. Handling of the pair continued as before but eggs were not retained and counted.

If a male died before his female partner, he was replaced by another male from the single-sex culture set up previously. Since egg collections were continued for the female, a male of the same population was used as her mate, as there were less likely to be physical differences such as copulation frequency etc. which may have affected the female's oviposition rate or survival. If a female was left without a mate for a week or so because of death, her oviposition rate should have been unaffected as it takes three to four weeks for a significant drop in egg production to occur (Bhat and Bhat 1974a).

Adults were weighed at day 70 of total lifespan. It was originally intended to weigh them at adult eclosion but this proved impossible to manage, thus it was decided to weigh all beetles at the one age, day 70, when time would be available to do so.

One problem was encountered with the physical layout of the experiment in the incubator which affected analysis of the data. Due to lack of space in the incubator at crucial times of the experiment, the progeny of the two replicates of one mating were placed on the same shelf in the incubator. Thus the two matings were separated. The consequence of this design fault is that any environmental differences between shelves would confound the estimation of the within diallel unit interaction term. However, environmental differences between shelves were probably small or non-existent as compared with the within unit interaction term. Firstly, shelf 1 was directly above shelf 2 and only separated by nine centimetres; secondly, the trays containing vials were perforated and sitting on racks so there was good air flow; and thirdly, the incubator was fan-forced so conditions should have been fairly uniform throughout the incubator and able to equilibriate quickly after disturbance, such as the door opening. A preliminary experiment using developmental rate, an environmentally sensitive trait, had also confirmed that no environmental trends existed across or between shelves of the incubator (Appendix 3). Thus it appears to be valid to assume that the interaction term was not confounded by environment.

3.2.2 Variables and Statistical Analyses

Male variables used were:

1) PUPN = time interval between day 0 and pupation (days).
2) ADULT = time interval between day 0 and adult eclosion (days).
3) WT70 = adult body weight 70 days after day 0 (mg).
4) GR = WT70/ADULT (mg/day).

5) DEATH = time interval between day 0 and death (days).

6) ALS = adult life-span — time interval between eclosion and death (days).

GR is not growth rate but is a comparable index because of the determinate size of adult *T. castaneum* and the highly significant product-moment correlation coefficient between weight on the day of adult emergence and weight on day 70 (Table 3.1).

The variables ADULT, WT70, DEATH and GR were filtered by the analysis program to exclude individuals with missing values for these variables. This filtering excluded all beetles who died before day 70. Beetles who died very early in adult life were likely to have died from non-senescent causes such as developmental injuries (Lavie 1981). This was confirmed by an examination of histograms of death and adult life span. Both histograms were bimodal with a very small peak following adult emergence separated by well over 40 days from a much larger peak. Excluding beetles who died before 70 days only excluded these very early deaths, which being non-senescent were not of interest. The exclusion had the benefit of removing outliers for DEATH and ALS, and more closely approximating conditions of normality for these traits. Following filtering, the data of all variables appeared unimodally distributed.

Tests of the nature and amount of departure from normality were done on all variables as:

1) measures of skewness and kurtosis;

2) Bartlett's test of homogeneity of variances between sire groups; and

3) independence of mean and variance using values of sire groups.

Table 3.1: Comparisons between bodyweight measurements of beetles (n = 42) weighed on day of adult eclosion and 70 days after day 0.

Day of measurement	Average weight	σ_{n-1}	R	r^2
Adult eclosion	1.5571	0.1612	0.8055	0.6488
70	1.6069	0.1864		

Only GR showed no departure from normality. All other variables showed various degrees of skewness; kurtosis; heterogeneity of variances; and dependence of means and variances of sire groups. However, Bartlett's test is very sensitive to nonnormality and may have indicated the presence of skewness and kurtosis rather than heteroscedasticity (Sokal and Rohlf 1981a).

For genetic analyses one must be careful of transformations of data to achieve normality because transformations of scale can influence, remove or reduce variance attributable to epistasis or dominance (Falconer 1981). Transformations are less necessary when the coefficient of variation (CV) is low, for analyses of variance do not rely on a strictly normal distribution and can be carried out on untransformed data when the CV is not above about 20%. All CV's were below 20% except for DEATH and ALS. Log(x + 1) and square root transformations did not improve normality of any of the characters, and, in light of the above, it was decided not to transform any of the male variables.

A computer program using methods of linear interpolation between egg collections and triangulation was used to estimate the number of eggs laid by each female in her lifetime. It also subdivided each individual's reproductive schedule into quarters to enable comparisons to be made between early and late egg lay. Bhat and Bhat (1974b) found strong phenotypic correlations (0.777 correlation coefficient) between eggs laid early in life between the time interval 7 and 11 days after adult emergence and total production in the 32 days following adult emergence. To evaluate the predictive value of early life indices for total lifetime production, the sum of eggs for various time periods early in adult life span were estimated.

The female variables were:

1) PUPN = time interval between day 0 and pupation (days).
2) ADULT = time interval between day 0 and adult eclosion (days).
3) WT70 = adult bodyweight 70 days after day 0 (mg).
4) GR = WT70/ADULT (mg/day).
5) DEATH = time interval between day 0 and death (days).
6) ALS = time interval between eclosion and death i.e. adult life-span (days).
7) EGGTOT = the total number of eggs produced in the lifetime of each female (eggs).
8) MAXEGG = the maximum number of eggs laid by a female in a 48 hour period (egg/day).

9) MAXDAY = day of peak egg production i.e. MAXEGG (day).
10) LASEGG = last egg collection from which eggs were collected (day).
11) RLS = time interval between eclosion and LASEGG i.e. reproductive life-span (days).
12) DAY011 = total number of eggs laid within the 11 days following eclosion (eggs).
13) DAY711 = total number of eggs laid in the time interval 7 and 11 days following eclosion (eggs).
14) EGG54 = total number of eggs laid during the 48 hour period between days 53 and 55 (egg/day).
15) Q1R = total number of eggs laid in the first quarter of RLS (eggs).
16) Q2R = total number of eggs laid in the second quarter of RLS (eggs).
17) Q3R = total number of eggs laid in the third quarter of RLS (eggs).
18) Q4R = total number of eggs laid in the fourth quarter of RLS (eggs).
19) ADFR = average daily fecundity for RLS (egg/day).
20) ADFQ1R = average daily fecundity for Q1R (egg/day).
21) ADFQ2R = average daily fecundity for Q2R (egg/day).
22) ADFQ3R = average daily fecundity for Q3R (egg/day).
23) ADFQ4R = average daily fecundity for Q4R (egg/day).
24) Q1L = total number of eggs laid in the first quarter of ALS (eggs).
25) Q2L = total number of eggs laid in the second quarter of ALS (eggs).
26) Q3L = total number of eggs laid in the third quarter of ALS (eggs).
27) Q4L = total number of eggs laid in the fourth quarter of ALS (eggs).
28) ADFL = average daily fecundity for ALS (egg/day).

29) ADFQ1L = average daily fecundity for Q1L (egg/day).
30) ADFQ2L = average daily fecundity for Q2L (egg/day).
31) ADFQ3L = average daily fecundity for Q3L (egg/day).
32) ADFQ4L = average daily fecundity for Q4L (egg/day).
33) MED1 = the number of eggs produced before the population median for egg production (day 93) (eggs).
34) MED2 = the number of eggs produced after the population median for egg production (day 93) (eggs).

The genetic correlation between these last two variables is an indicator of overall reproductive cost i.e. if an individual produces more eggs early relative to other individuals, does it produce relatively less later on because of lower production and/or earlier death?

If a maximum number of eggs was laid in more than one egg collection period, MAXEGG was calculated as the mean between the two or more egg collection days. This is not an unreasonable procedure as the reproductive schedule of *T. castaneum* is characterised by a plateau of peak egg production.

Survival of progeny to adulthood was not included as a trait among the female variables even though it is an important measure of fitness. This was because it was expected to be depressed as a result of the full-sib matings. Comparisons between progeny of full-sib matings and progeny of females mated to unrelated males confirmed this expectation. The average proportion of larvae and pupae alive at day 20 for full-sib matings was 71.00%, σ_{n-1} = 21.11, whilst for non-related matings it was 87.48%, σ_{n-1} = 22.18. As the inbreeding depression may have affected genotypes differentially, survival of progeny was not used as an index.

As with the males, various variables were filtered (ADULT, WT70, DEATH, GR, MAXDAY, LASEGG and EGG54) to exclude beetles with missing values and those that died before day 70. Filtering was particularly important for female variables because the analysis program could not differentiate between zeros and missing values and included missing values in the analysis as zeros. Filtering on the above variables removed individuals with missing values from the analysis but ensured that zeros, when they were relevant values for variables such as reproductive indices, could be included in the analysis.

Only 21 females were excluded and all died before day 70. Eighteen of these died before 26 days after adult emergence and only 5 of the 20 laid any eggs. The removal of these

outliers eliminated the bimodality and reduced the coefficient of variation for DEATH and ALS, and, as for the males, more closely approximated conditions of normality. All variables had unimodal distributions.

Tests of normality were done for all female variables. Only Q2R, Q3R and Q1L met all requirements of normality. GR for females exhibited some heterogeneity of variance. Transformations did not improve normality except for the traits MAXDAY, ADFQ4R and Q4R, where the square root was used. No other variables were transformed though some had quite high coefficients of variation particularly the reproductive indices for ALS as compared with those for RLS.

The computer program REG87 (Gilmour 1987) was used to calculate the variance and covariance components of all variables.

The original model assumed was:

$$Y_{ijklm} = \mu + U_i + L_j + P_k + LxP_{jk} + S(U)_{l(i)} + D(U)_{m(i)} + SxD(U)_{lm(i)} + e_{ijklm}$$

where Y_{ijklm} = phenotypic value of the mth beetle;

μ = value of the overall mean;

U_i = the random effect of the ith unit;

L_j = the fixed effect of the jth incubator shelf;

P_k = the fixed effect of the kth position on an incubator shelf;

LxP_{jk} = the interaction effect of the jth shelf and the kth position;

$S(U)_{l(i)}$ = the random effect of the lth sire nested within the ith unit;

$D(U)_{m(i)}$ = the random effect of the mth dam nested within the ith unit;

$SxD(U)_{lm(i)}$ = the interaction effect of the mth dam mated to the lth sire nested within the ith unit; and

e_{ijklm} = random errors associated with each observation (assumed to be real and independent with expectations equal to zero).

However, the positioning of the experimental vials had aliased L and SxD(U) i.e. the same contrasts were used to estimate both terms. As explained in section 3.2.1 it was assumed that the interaction term was not confounded by environment, thus all terms containing L were removed from the model and shelf effects were ignored for the purposes of analysis. Reg87 also absorbed Units for ease of analysis. Thus the model used by the program was:

$$Y_{iklm} = \mu + P_k + S(U)_{l(i)} + D(U)_{m(i)} + SxD(U)_{lm(i)} + e_{iklm}$$

The expected number of progeny per dam per mating was 6, so the average number of progeny per dam per mating was used as an approximation for deriving k coefficients (Falconer 1981):

$$\frac{Number\ of\ actual\ records\ of\ progeny\ (1082♂)\ (1086♀)}{Number\ of\ Units\ (54)\ x\ Number\ of\ matings\ per\ Unit\ (4)} \approx 5 \text{ for both}$$

males and females. Thus the k coefficients were 10 for the sire and dam components and 5 for the interaction component.

If calculated variance components are negative they are effectively zero. Thus when traits had a negative interaction variance component it was interpreted that there was no genetic variation for the trait from this source. In such cases interaction was not fitted and SxD(U) was included in error. Reg87 was then able to give estimates of heritabilities and genetic correlations for these traits. If the interaction variance component was positive then it was not possible to drop SxD(U), and Reg87 would only give the variance and covariance components. Genetic parameters were calculated using the standard formulae of Falconer (1981) and Becker (1984). For heritability:

$$h_S^2 = \frac{4\widehat{\sigma}_S^2}{\widehat{\sigma}_S^2 + \widehat{\sigma}_D^2 + \widehat{\sigma}_\gamma^2 + \widehat{\sigma}_\epsilon^2}$$

$$h_D^2 = \frac{4\widehat{\sigma}_D^2}{\widehat{\sigma}_S^2 + \widehat{\sigma}_D^2 + \widehat{\sigma}_\gamma^2 + \widehat{\sigma}_\epsilon^2}$$

$$h_{S+D}^2 = \frac{2(\widehat{\sigma}_S^2 + \widehat{\sigma}_D^2)}{\widehat{\sigma}_S^2 + \widehat{\sigma}_D^2 + \widehat{\sigma}_\gamma^2 + \widehat{\sigma}_\epsilon^2}$$

where h_S^2 = heriability estimate based on estimated sire component;

h^2_D = heritability estimate based on estimated dam component;

h^2_{S+D} = combined heritability estimate from the estimated sire and dam components based on the resemblance between full-sibs;

$\hat{\sigma}^2_S$ = estimated sire component of variance;

$\hat{\sigma}^2_D$ = estimated dam component of variance;

$\hat{\sigma}^2_\gamma$ = estimated interaction variance component; and

$\hat{\sigma}^2_e$ = estimated within-progeny variance component.

For genetic correlations between traits X and Y:

$$r_S = \frac{\widehat{cov}_S}{\sqrt{\widehat{\sigma}^2_{S(X)}\widehat{\sigma}^2_{S(Y)}}}$$

$$r_D = \frac{\widehat{cov}_D}{\sqrt{\widehat{\sigma}^2_{D(X)}\widehat{\sigma}^2_{D(Y)}}}$$

$$r_{S+D} = \frac{\widehat{cov}_S + \widehat{cov}_D}{\sqrt{\widehat{\sigma}^2_{S(X)} + \widehat{\sigma}^2_{D(X)}}\sqrt{\widehat{\sigma}^2_{S(Y)} + \widehat{\sigma}^2_{D(Y)}}}$$

where r_S = genetic correlation estimate based on sire variance and covariance component estimates;

r_D = genetic correlation estimate based on dam variance and covariance component estimates;

r_{S+D} = genetic correlation estimate based on the combination of sire and dam variance and covariance component estimates;

$\widehat{cov}_S$ = estimated sire component of covariance;

$\widehat{cov}_D$ = estimated dam component of covariance;

$\widehat{\sigma}^2_{S(X)}$ = estimated sire variance component of X;

$\widehat{\sigma}^2_{S(Y)}$ = estimated sire variance component of Y;

$\widehat{\sigma}^2_{D(X)}$ = estimated dam variance component of X; and

$\widehat{\sigma}^2_{D(Y)}$ = estimated dam variance component of Y.

The significance of variance and covariance components was determined by F-tests. If sire or dam variance components were negative for a particular trait, the respective paternal or maternal heritability and genetic correlations were undefined. When calculating total genetic variance for the trait, the negative variance component was set to zero. This enabled calculation of a heritability estimate based upon the other (if positive) parental variance component. The calculation of standard errors for such heritability estimates was also made possible by this assumption.

Standard errors of heritabilities and genetic correlations were calculated by the use of a GWBASIC program, HM3SE, written by Dr. Arthur Gilmour of the N.S.W. Department of Agriculture. The program used appropriate formulae given by Becker (1984) for standard errors of heritabilities and genetic correlations estimated from intraclass correlations. Values produced by this program were accurate, as no approximations were involved in their calculation.

One-tailed t-tests were used to test whether estimated heritabilities were significantly greater than zero, whilst two-tailed t-tests were used to test whether the estimated genetic correlations were significantly different from zero.

Genetic expectations of the components of variance (Table 3.2) have been taken from Becker (1984). Mode and Robinson (1959) showed that genetic covariance can be partitioned in a manner analogous to genetic variance, and the genetic expectations of variance and covariance components were similar. The genetic expectations have been simplified by ignoring genes with different effects in the two sexes as well as non-additive sex-linked and additive x sex-linked effects.

Comparisons of heritability estimates from paternal half-sisters and maternal half-brothers identified the presence or absence of maternal effects. Comparisons between paternal half-brothers and paternal half-sisters provided estimates of sex-linkage. The interaction components provided upper limits for estimates of dominance and epistasis.

If maternal and paternal half-sib estimates of heritability for one sex were similar, the two estimates of heritability from the sire and dam components respectively were assumed to be not biased by maternal effects or, in the case of half-brothers, by sex-linkage, and equally reliable. In this case the heritability derived from the combination of sire and dam components is the best estimate. This is because the combination is based on the resemblance between full-sibs, and estimates based on full-sib families, if unbiased, are more precise than those based on half-sib families (Falconer 1981). Maternal and paternal

Table 3.2: Genetic expectations for heritability and variance components estimates.

Estimate	**Genetic component**							
	V_A	V_{AA}	V_{AAA}	V_{AS}	V_D	V_{AD}	V_{DD}	V_M
Heritability								
Paternal half-brother	1	$\frac{1}{4}$	$\frac{1}{16}$					
Paternal half-sister	1	$\frac{1}{4}$	$\frac{1}{16}$	2				
Maternal half-brother	1	$\frac{1}{4}$	$\frac{1}{16}$	2				4
Maternal half-sister	1	$\frac{1}{4}$	$\frac{1}{16}$	1				4
Variance components								
Sire × Dam components								
for male offspring		$\frac{1}{8}$	$\frac{3}{32}$		$\frac{1}{4}$	$\frac{1}{8}$	$\frac{1}{16}$	
Sire × Dam components								
for female offspring		$\frac{1}{8}$	$\frac{3}{32}$		$\frac{1}{4}$	$\frac{1}{8}$	$\frac{1}{16}$	

V_A = additive variance
V_{AA} = additive × additive variance
V_{AAA} = additive × additive × additive variance
V_{AS} = additive sex-linked variance
V_D = dominance variance
V_{AD} = additive × dominance variance
V_{DD} = dominance × dominance variance
V_M = maternal effects variance

half-sib estimates of genetic correlations between two traits can be compared and analysed in the same way as estimates of heritability.

3.3 Results

3.3.1 Population Sampling

Before analysis of data commenced, a check was made that the sample of progeny used in the diallel design was representative of the population. The distribution of developmental time (egg to pupa) was compared (Table 3.3) between those beetles that were kept to be set up as single pairs (Group 1) and those that were discarded (Group 2).

Table 3.3: Comparison of developmental rates of beetles retained for the diallel analysis (Group1) with the developmental rates of discarded beetles (Group 2).

Group	**Mean**	**Standard deviation**	**n**
1	20.789	1.558	2193
2	20.712	3.138	1403

The means were not significantly different, but the variances were highly significantly different. There appeared to have been some bias in the sampling procedure against individuals with extreme expressions of the trait. Since the extent of developmental variability is usually assumed to be a direct reflection of the extent of genetic variability present in the population (Arking and Clare 1986), it is possible that the genetic variation present for other life-history traits was restricted.

3.3.2 Means and Standard Deviations

The means, standard deviations and coefficients of variation of the filtered traits are given for each sex in Tables 3.4 and 3.5.

Some coefficients of variation are quite high, particularly for variables derived from the division of adult life span into quarters. The analysis of variance is robust to deviations from normality, but estimates of variance components for traits with high coefficients of variation may not be accurate.

Table 3.4: Means, standard deviations(SD) and coefficients of variation(CV) of male characters for 1052 beetles that lived beyond 70 days.

Character	**Mean**	**SD**	**CV (%)**
PUPN	20.608	1.475	7.157
ADULT	25.386	1.474	5.806
WT70	1.574	0.218	13.850
GR	0.062	0.008	12.903
ALS	313.764	79.596	25.368
DEATH	339.150	79.582	23.465

3.3.3 Heritabilities of Male Traits

Heritability estimates for male traits are given in Table 3.6. Analyses of variance and variance components are presented in Appendix 7.

If the paternal and maternal half-sib heritability estimates were similar, then, as explained before, the heritability derived from the combination of sire and dam components was treated as the best estimate. If dissimilar, the paternal half-sib heritability is most precise because for males it was unbiased by maternal effects, and is the heritability estimate referred to when discussing the heritability of a particular trait.

Table 3.7 gives the upper limits for the proportion of phenotypic variance which was due to dominance or epistasis for male variables which had positive interaction variance components: ALS and DEATH. If epistasis is assumed to be negligible then the interaction component represents $\frac{1}{4}$ of the dominance present for the trait; whilst if dominance is assumed to be negligible, it represents $\frac{1}{8}$ of the epistasis (see Table 3.2). Thus, multiplying the proportion of total phenotypic variance due to interaction variance by four gives the upper limit for the proportion of phenotypic variance due to dominance; and multiplying it by eight, the upper limit for the proportion of phenotypic variance due to epistasis.

For time to pupation (PUPN) and adult emergence (ADULT), the maternal half-sib heritability estimates were three times the paternal half-sib estimates. The differences must have been due to sex linkage and/or maternal effects. As the heritability estimates of these traits for females were similar, the differences could not have been due to sex linkage. It appears then that maternal effects inflated the heritability estimates for maternal half-brothers. Thus the heritabilities (h_S^2) for developmental rate were moderate and significant.

Table 3.5: Means, standard deviations and coefficients of variation of female characters for 1090 beetles that lived beyond 70 days.

Character	Mean	SD	CV (%)
PUPN	20.977	1.614	7.694
ADULT	25.665	1.600	6.234
WT70	1.755	0.247	14.074
GR	0.068	0.009	13.235
ALS	190.300	57.954	30.454
DEATH	215.941	58.171	26.938
EGGTOT	1620.430	541.642	33.426
MAXEGG	15.506	2.666	17.193
MAXDAY	61.019	27.095	44.404
LASEGG	196.358	52.156	26.562
RLS	170.717	52.002	30.461
DAY011	89.055	23.541	26.434
DAY711	48.933	12.512	25.570
EGG54	13.403	3.284	24.502
Q1R	497.746	185.398	37.248
Q2R	512.895	177.111	34.532
Q3R	416.771	151.934	36.455
Q4R	193.022	99.671	51.637
ADFR	9.559	2.019	21.121
ADFFQ1R	11.535	2.377	20.607
ADFQ2R	12.089	2.562	21.193
ADFQ3R	9.955	2.896	29.091
ADFQ4R	4.658	2.341	50.258
Q1L	556.564	205.908	36.996
Q2L	541.005	194.073	35.873
Q3L	379.582	181.218	47.741
Q4L	143.283	113.218	79.017
ADFL	8.745	2.325	26.587
ADFQ1L	11.616	2.387	20.549
ADFQ2L	11.571	2.948	25.477
ADFQ3L	8.478	3.819	45.046
ADFQ4L	3.316	2.652	79.976

Table 3.6: Heritability estimates (±SE) for male life-history traits.

Traits	h^2_S	h^2_D	h^2_{S+D}
PUPN	0.300 ± 0.107**	0.911 ± 0.184**	0.605 ± 0.094**
ADULT	0.264 ± 0.101**	0.940 ± 0.187**	0.602 ± 0.095**
WT70	0.481 ± 0.135**	0.631 ± 0.155**	0.556 ± 0.089**
GR	0.555 ± 0.142**	0.748 ± 0.167**	0.652 ± 0.090**
ALS	0.143 ± 0.150	0.291 ± 0.169*	0.217 ± 0.128*
DEATH	0.143 ± 0.150	0.283 ± 0.168*	0.213 ± 0.129

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.7: The upper limits for the proportion of phenotypic variance which is due to dominance or epistasis for male variables.

Character	$\frac{\sigma^2_\gamma}{\sigma^2_T} \times 100^*$	**% Dominance**	**% Epistasis**
ALS	7.402	29.607	59.214
DEATH	7.490	29.959	59.918

* Proportion of total phenotypic variance due to interaction variance.

Heritabilities were low and insignificant for DEATH and Adult Life Span (ALS). Comparison between maternal half-brother and paternal half-sister estimates, whose genetic expectations are the same in the absence of maternal effects, revealed the influence of maternal effects. Small amounts of sex-linkage were also present. (The presence of sex-linkage can be determined by differences between paternal half-brother and half-sister heritability estimates, and between maternal half-brother and half-sister estimates.)

DEATH had a significant interaction component whilst that for ALS was close to significance. Both characters displayed substantial amounts of dominance or epistasis. It is impossible to differentiate between the respective amounts of dominance and epistasis by the use of the diallel design alone.

WT70 and GR both had moderately high heritabilities,with possibly small amounts of sex linkage and/or maternal effects present. Examination of female estimates (Table 3.8) did not confirm the presence of sex linkage and/or maternal effects for WT70, thus h^2_{S+D} was the best heritability estimate. For GR, maternal effects were present, which was not surprising considering the large influence of maternal effects on the heritability of adult emergence time.

3.3.4 Heritabilities of Female Traits

Heritability estimates for female traits are given in Table 3.8. Table 3.9 gives the upper limits for the proportion of phenotypic variance which was due to dominance or epistasis. Analyses of variance and variance components are presented in Appendix 8. All female variables had positive interaction variance components.

For female half-sib heritability estimates, paternal half-sisters have twice the amount of sex linkage as maternal half-sisters. However, maternal half-sister estimates are biased fourfold by maternal effects, whilst paternal half-sisters have none. For traits which may only be measured on females, differences between heritability estimates may be difficult to ascribe to either sex linkage and/or maternal effects (see Table 3.2). Large maternal effects could hide the existence of sex linkage or vice versa, or each could balance the other. These problems of interpretation were borne in mind when differences between estimates were interpreted according to genetic expectations.

Differences between estimates also could have been due to experimental error, thus caution was used in interpreting any small difference between estimates as due to either sex-linked variation or maternal effects. If differences did exist the paternal half-sister

heritability estimate was regarded as the more precise estimate of heritability.

Unlike males, time to pupation and adult emergence did show some dominance and/or epistasis though the interaction components were not significant.

Only a few of the traits had significant interaction components (see Appendix 8). These were DEATH, ALS, MAXDAY, Q2R, Q3R, Q2L, MED1 and MED2.

Of particular interest is MAXDAY which appeared to have no heritable variation but a very large interaction component. Dominance represented up to 65%, or additive x additive epistasis represented up to 130% of the phenotypic variance. The impossibly large epistatic estimate suggests that dominance comprised most of the interaction component.

Most of the reproductive traits exhibited low to moderate heritabilities. At the higher end of the scale were DAY711 (h^2_{S+D}), ADFR (h^2_S) and ADFQ2R (h^2_S). The heritabilities for these three traits were still lower than those for the non-reproductive traits WT70 (h^2_{S+D}) and GR (h^2_S). The traits are all linked by their dependence upon the number of eggs laid at peak egg production and/or the length of time peak egg production was maintained. ADFQ2R probably contained a large part of the plateau of peak egg production for most beetles.

Maternal effects appeared to be present for many of the early adult life reproductive indices such as MAXEGG, EGG54, Q1R, ADFQ1R, Q1L, ADFQ1L and MED1. Sex-linked variation was usually apparent in later life traits and traits influenced by later life factors, such as Q3R, Q4R, ADFR, ADFQ2R, ADFQ3R, Q3L, ADFL, ADFQ2L and ADFQ3L. Sex linkage might also have been present for early adult life traits but hidden by larger maternal effects.

There were some exceptions to the general rule. LASEGG, RLS, DEATH and ALS are late life characters but all were influenced by maternal effects. Assuming sex-linked variation was negligible, the proportion of maternal effects for LASEGG and RLS was 0.06, whilst for DEATH and ALS it was 0.07. If sex linkage was present then these were underestimates of maternal effects. An explanation for the presence, though small, of maternal effects could be that the length of reproductive and adult life spans were modified by early life trait(s) that did experience maternal effects.

A final observation was that the additive genetic variance of fecundity did not increase with age. Since $4\sigma^2_S \approx V_A$, examination of the sire components for the traits ADFQ1R, ADFQ2R, ADFQ3R and ADFQ4R should have revealed any change in additive genetic variation. There was an increase in additive genetic variation from ADFQ1R to ADFQ2R but

Table 3.8: Heritability estimates (±SE) for female life-history traits.

Traits	h^2_S	h^2_D	h^2_{S+D}
PUPN	0.247 ± 0.126*	0.785 ± 0.190**	0.516 ± 0.114**
ADULT	0.228 ± 0.117*	0.792 ± 0.185**	0.510 ± 0.110**
WT70	0.521 ± 0.151**	0.559 ± 0.157**	0.540 ± 0.103**
GR	0.520 ± 0.149**	0.881 ± 0.189**	0.701 ± 0.107**
ALS		0.184 ± 0.170	
DEATH		0.184 ± 0.171	
EGGTOT	0.203 ± 0.154	0.217 ± 0.157	0.210 ± 0.125
MAXEGG	0.350 ± 0.148*	0.519 ± 0.169**	0.434 ± 0.115**
MAXDAY			
LASEGG	0.087 ± 0.135	0.320 ± 0.165*	0.203 ± 0.122
RLS	0.088 ± 0.135	0.313 ± 0.164*	0.200 ± 0.122
DAY011	0.310 ± 0.150*	0.223 ± 0.139	0.267 ± 0.113*
DAY711	0.364 ± 0.148**	0.386 ± 0.151**	0.375 ± 0.110**
EGG54	0.238 ± 0.142	0.353 ± 0.158*	0.296 ± 0.116**
Q1R	0.045 ± 0.130	0.289 ± 0.161*	0.167 ± 0.120
Q2R	0.137 ± 0.157	0.171 ± 0.162	0.154 ± 0.132
Q3R	0.184 ± 0.167	0.065 ± 0.153	0.125 ± 0.134
Q4R	0.108 ± 0.149		
ADFR	0.456 ± 0.164**	0.287 ± 0.144*	0.372 ± 0.115**
ADFQ1R	0.264 ± 0.152*	0.370 ± 0.166*	0.317 ± 0.122**
ADFQ2R	0.423 ± 0.166**	0.273 ± 0.148*	0.348 ± 0.119**
ADFQ3R	0.290 ± 0.155*	0.124 ± 0.135	0.207 ± 0.116*
ADFQ4R			
Q1L		0.212 ± 0.162	
Q2L	0.064 ± 0.166	0.120 ± 0.174	0.092 ± 0.144
Q3L	0.335 ± 0.155*	0.141 ± 0.131	0.238 ± 0.114*
Q4LL	0.069 ± 0.144	0.030 ± 0.140	0.049 ± 0.122
ADFL	0.355 ± 0.155*	0.178 ± 0.134	0.267 ± 0.113*
ADFQ1L	0.284 ± 0.150*	0.396 ± 0.165*	0.340 ± 0.120**
ADFQ2L	0.375 ± 0.151**	0.199 ± 0.129	0.287 ± 0.108**
ADFQ3L	0.236 ± 0.148	0.060 ± 0.126	0.148 ± 0.113
ADFQ4L			
MED1	0.318 ± 0.163*	0.401 ± 0.174*	0.360 ± 0.128**
MED2	0.181 ± 0.156	0.218 ± 0.161	0.199 ± 0.128

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.9: The upper limits for the proportion of phenotypic variance which is due to dominance or epistasis for female variables.

Character	$\frac{\sigma_I^2}{\sigma_T^2} \times 100^*$	**% Dominance**	**% Epistasis**
PUPN	2.388	9.552	19.105
ADULT	0.794	3.175	6.350
WT70	0.066	0.266	0.531
GR	1.281	5.123	10.246
ALS	9.392	37.569	75.138
DEATH	9.532	38.127	76.254
EGGTOT	6.731	26.923	53.846
MAXEGG	3.432	13.729	27.458
MAXDAY	16.189	64.760	129.510
LASEGG	5.514	22.056	44.112
RLS	5.442	21.768	43.536
DAY011	3.120	12.482	24.963
DAY711	2.284	9.136	18.273
EGG54	3.901	15.603	31.206
Q1R	5.004	20.017	40.034
Q2R	8.532	34.130	68.259
Q3R	9.036	36.142	72.284
Q4R	6.168	24.670	49.340
ADFR	3.595	14.381	28.762
ADFQ1R	5.670	22.680	45.361
ADFQ2R	4.697	18.787	37.574
ADFQ3R	4.206	16.825	33.650
ADFQ4R	6.126	24.545	49.090
Q1L	6.736	26.945	53.890
Q2L	11.731	46.922	93.844
Q3L	3.295	13.180	26.360
Q4L	6.014	24.056	48.112
ADFL	3.115	12.459	24.918
ADFQ1L	4.971	19.882	39.764
ADFQ2L	1.753	7.010	14.021
ADFQ3L	3.402	13.607	27.214
ADFQ4L	8.683	34.733	69.466
MED1	7.267	29.066	58.132
MED2	7.510	30.041	60.082

* Proportion of total phenotypic variance due to interaction variance.

this stayed substantially at the same level for ADFQ3R and dropped to zero for ADFQ4R (see Appendix 8). Late life characters do not apparently exhibit greater additive genetic variation than early life indices. The indices for mean number of eggs laid per day for various quarters of the reproductive life-span were chosen as the best measures of fecundity. Fecundity indices for adult life-span were confounded by the length of post-reproductive periods, whilst the indices for total number of eggs laid in a quarter of reproductive life-span were influenced by the length of reproductive life-span. Using the mean number of eggs laid per day eliminated the contributing factor of the length of reproductive life-span.

The means and standard deviations of ADFQ1R, ADFQ2R, ADFQ3R and ADFQ4R were independent so transformation was unnecessary. Mertz (1975) found that mean fecundity and its standard deviation were negatively correlated but this feature of the data disappeared when fecundity was evaluated over longer time intervals as was done here.

In summary, fitness traits in this *T. castaneum* population mostly have low to medium heritabilities and do not exhibit much dominance and/or epistatic variation, with some notable exceptions e.g. DEATH, ALS, MAXDAY, Q2R, Q3R, Q2L and ADFQ4L.

3.3.5 Genetic Correlations between Male Traits

Genetic correlation estimates between male traits are presented in Table 3.10. For traits whose maternal genetic correlation estimates were unbiased by sex linked covariation and/or maternal effects, the combined estimate should be most accurate on theoretical grounds.

Two-tailed t-tests were used to test whether estimates were significantly different from zero. Estimates of genetic correlations are subject to large sampling errors and are therefore usually not precise. Standard errors were large for most estimates, so emphasis in discussion for both male and female traits was placed on estimates of large magnitude, rather than only discussing genetic correlations which were significantly different from zero. Genetic correlations of large magnitude were likely to have been significant if the size of the experiment had been increased to the point that sampling errors were minimised.

Furthermore, when heritabilities are low, less than or equal to 0.20, estimates of genetic correlations and their sampling variances are likely to be biased upwards. The bias increases with an increase in the genetic correlation (when heritability is fixed) but is decreased by large sample sizes, $N \geq 1000$, such as used here (Van Vleck and Henderson 1961). Thus the significance of genetic correlations between characters with low heritabilities is difficult to determine, particularly when the magnitude of the genetic correlation is large, and so

Table 3.10: Genetic correlation estimates (±SE) for male traits from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
PUPN–ADULT	0.993 ± 0.013**	0.996 ± 0.004**	0.995 ± 0.005**
PUPN–WT70	0.219 ± 0.225	0.328 ± 0.167	0.286 ± 0.133*
PUPN–GR	-0.085 ± 0.233	-0.206 ± 0.180	-0.163 ± 0.141
PUPN–ALS	-0.018 ± 0.439	-0.482 ± 0.274	-0.344 ± 0.266
PUPN–DEATH	0.010 ± 0.437	-0.454 ± 0.279	-0.316 ± 0.268
ADULT–WT70	0.276 ± 0.224	0.300 ± 0.169	0.285 ± 0.133*
ADULT–GR	-0.028 ± 0.240	-0.237 ± 0.177	-0.167 ± 0.142
ADULT–ALS	0.003 ± 0.409	-0.453 ± 0.264	-0.320 ± 0.250
ADULT–DEATH	0.031 ± 0.407	-0.425 ± 0.269	-0.290 ± 0.252
WT70–GR	0.953 ± 0.023**	0.855 ± 0.050**	0.897 ± 0.029**
WT70–ALS	0.080 ± 0.373	0.126 ± 0.272	0.108 ± 0.236
WT70–DEATH	0.091 ± 0.373	0.140 ± 0.275	0.120 ± 0.238
GR–ALS	0.081 ± 0.380	0.378 ± 0.265	0.265 ± 0.239
GR–DEATH	0.082 ± 0.380	0.378 ± 0.268	0.264 ± 0.242
ALS–DEATH	1.000 ± 0.000**	1.000 ± 0.000**	1.000 ± 0.000**

* $P \leq 0.05$, ** $P \leq 0.01$

possibly biased itself.

DEATH and ALS had heritabilities less than 0.2, so genetic correlations between these and other characters were probably biased.

The only significant genetic correlations from combined sire and dam components were for PUPN-ADULT, PUPN-WT70, ADULT-WT70, WT70-GR and ALS-DEATH. All other correlations were of very low magnitude. There is then a relationship between slow developmental rate and greater body weight.

The very high positive genetic correlations and low standard errors between ALS and DEATH (r_{S+D}), and between PUPN and ADULT (r_{S+D}) indicate that they were measures of the same trait, that is longevity and developmental rate respectively. The genetic architecture of the paired traits was also very similar as would be expected.

3.3.6 Genetic Correlations between Female Traits

Tables 3.11 to 3.31 give the genetic correlation estimates between characters measured on females. Maternal and paternal half-sister estimates for each trait are presented. Also included are genetic correlation estimates based on combined sire and dam components of variance and covariance. If paternal and maternal half-sister estimates were dissimilar, then the paternal half-sister genetic correlation estimate was the best estimate of genetic correlation as it was unbiased by maternal effects.

Reproductive indices based on ALS are not presented. Their coefficients of variation were in some cases very large (Table 3.5) and their values were confounded by varying lengths of post-reproductive periods. Genetic correlation estimates for reproductive indices based on reprodutive life span (RLS) were more precise and easier to interpret, whilst providing the information sought.

Many of the genetic correlations recorded here were "part-whole", that is, some of the traits were not derived independently of each other, particularly many of the reproductive indices. Where one was only a small fraction of the other as DAY711 was of EGGTOT, then genetic correlations suffered little to no bias and were amenable to interpretation. More confounded genetic correlations, such as between ADFR-ADFQ1R, were only included as detail. Some, such as Q*R-ADFQ*R (* = 1, 2, 3 or 4), were omitted from the results because they did not provide meaningful information.

Also omitted were all genetic correlation estimates for MAXDAY and ADFQ4R, as sire and dam variance components were negative for both traits, thus all genetic correlation estimates were undefined.

Table 3.11: Genetic correlation estimates (±SE) for female traits, PUPN–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
PUPN–ADULT	0.993 ± 0.018**	0.989 ± 0.007**	0.990 ± 0.008**
PUPN–WT70	0.431 ± 0.234	-0.039 ± 0.220	0.122 ± 0.175
PUPN–GR	0.195 ± 0.282	-0.494 ± 0.170**	-0.284 ± 0.165
PUPN–ALS		-0.056 ± 0.369	
PUPN–DEATH		-0.000 ± 0.365	
PUPN–EGGTOT	0.127 ± 0.445	-0.094 ± 0.337	-0.016 ± 0.291
PUPN–MAXEGG	0.362 ± 0.308	-0.083 ± 0.233	0.057 ± 0.203
PUPN–LASEGG	-0.062 ± 0.662	0.199 ± 0.267	0.140 ± 0.279
PUPN–RLS	-0.110 ± 0.667	0.154 ± 0.273	0.094 ± 0.283
PUPN–DAY011	0.376 ± 0.330	-0.024 ± 0.305	0.127 ± 0.235
PUPN–DAY711	-0.009 ± 0.344	-0.252 ± 0.253	-0.161 ± 0.218
PUPN–EGG54	0.535 ± 0.369	-0.201 ± 0.264	0.030 ± 0.238
PUPN–Q1R	0.281 ± 0.814	-0.002 ± 0.290	0.048 ± 0.310
PUPN–Q2R	-0.032 ± 0.583	-0.019 ± 0.374	-0.023 ± 0.347
PUPN–Q3R	0.130 ± 0.482	-0.330 ± 0.751	-0.093 ± 0.395
PUPN–Q4R	0.381 ± 0.620		
PUPN–ADFR	0.246 ± 0.301	-0.418 ± 0.294	-0.133 ± 0.223
PUPN–ADFQ1R	0.270 ± 0.366	-0.273 ± 0.278	-0.097 ± 0.249
PUPN–ADFQ2R	0.049 ± 0.340	-0.383 ± 0.321	-0.191 ± 0.245
PUPN–ADFQ3R	0.259 ± 0.363	-0.601 ± 0.501	-0.181 ± 0.291

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.12: Genetic correlation estimates (±SE) for female traits, ADULT– *, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
ADULT–WT70	0.414 ± 0.234	–0.054 ± 0.218	0.102 ± 0.174
ADULT–GR	0.177 ± 0.282	–0.508 ± 0.166**	–0.304 ± 0.162
ADULT–ALS		0.006 ± 0.356	
ADULT–DEATH	0.062 ± 0.352		
ADULT–EGGTOT	0.066 ± 0.455	–0.063 ± 0.326	–0.018 ± 0.284
ADULT–MAXEGG	0.317 ± 0.311	–0.168 ± 0.232	–0.019 ± 0.204
ADULT–LASEGG	–0.017 ± 0.648	0.294 ± 0.260	0.226 ± 0.273
ADULT–RLS	–0.066 ± 0.650	0.250 ± 0.265	0.181 ± 0.277
ADULT–DAY011	0.398 ± 0.321	–0.153 ± 0.312	0.057 ± 0.236
ADULT–DAY711	0.041 ± 0.339	–0.321 ± 0.246	–0.189 ± 0.213
ADULT–EGG54	0.459 ± 0.367	–0.281 ± 0.262	–0.054 ± 0.238
ADULT–Q1R	0.337 ± 0.845	0.048 ± 0.281	0.097 ± 0.299
ADULT–Q2R	–0.049 ± 0.582	0.019 ± 0.360	–0.003 ± 0.334
ADULT–Q3R	0.011 ± 0.506	–0.344 ± 0.749	–0.150 ± 0.400
ADULT–Q4R	0.136 ± 0.602		
ADULT–ADFR	0.158 ± 0.311	–0.555 ± 0.298	–0.246 ± 0.228
ADULT–ADFQ1R	0.273 ± 0.366	–0.335 ± 0.274	–0.142 ± 0.248
ADULT–ADFQ2R	–0.002 ± 0.350	–0.513 ± 0.327	–0.284 ± 0.251
ADULT–ADFQ3R	0.143 ± 0.374	–0.830 ± 0.578	–0.343 ± 0.312

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.13: Genetic correlation estimates (±SE) for female traits, WT70–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
WT70–GR	0.970 ± 0.023**	0.887 ± 0.044**	0.917 ± 0.028**
WT70–ALS		0.186 ± 0.376	
WT70–DEATH		0.183 ± 0.376	
WT70–EGGTOT	0.355 ± 0.325	0.522 ± 0.303	0.441 ± 0.248
WT70–WT70–MAXEGG	0.727 ± 0.157**	0.779 ± 0.124**	0.753 ± 0.109**
WT70–LASEGG	–0.050 ± 0.488	0.167 ± 0.288	0.091 ± 0.266
WT70–RLS	–0.069 ± 0.486	0.172 ± 0.291	0.087 ± 0.268
WT70–DAY011	0.701 ± 0.209**	0.703 ± 0.247**	0.699 ± 0.181**
WT70–DAY711	0.282 ± 0.240	0.389 ± 0.220	0.337 ± 0.175
WT70–EGG54	0.474 ± 0.261	0.724 ± 0.188**	0.611 ± 0.174**
WT70–Q1R	0.378 ± 0.724	0.610 ± 0.253*	0.504 ± 0.274
WT70–Q2R	0.408 ± 0.406	0.598 ± 0.363	0.509 ± 0.311
WT70–Q3R	0.514 ± 0.362	0.632 ± 0.790	0.539 ± 0.377
WT70–Q4R	0.302 ± 0.472		
WT70–ADFR	0.531 ± 0.193**	0.531 ± 0.224*	0.526 ± 0.158**
WT70–ADFQ1R	0.470 ± 0.249	0.740 ± 0.176**	0.617 ± 0.164**
WT70–ADFQ2R	0.562 ± 0.193**	0.573 ± 0.224*	0.562 ± 0.159**
WT70–ADFQ3R	0.651 ± 0.238**	0.395 ± 0.379	0.534 ± 0.231*

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.14: Genetic correlation estimates (±SE) for female traits, GR–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
GR–ALS		0.140 ± 0.345	
GR–DEATH		0.111 ± 0.344	
GR–EGGTOT	0.382 ± 0.339	0.417 ± 0.297	0.430 ± 0.248
GR–MAXEGG	0.725 ± 0.173**	0.765 ± 0.126**	0.750 ± 0.114**
GR–LASEGG	-0.056 ± 0.481	-0.007 ± 0.262	-0.021 ± 0.248
GR–RLS	-0.065 ± 0.479	0.017 ± 0.264	-0.006 ± 0.249
GR–DAY011	0.668 ± 0.230**	0.691 ± 0.250**	0.664 ± 0.188**
GR–DAY711	0.309 ± 0.236	0.483 ± 0.192*	0.406 ± 0.159*
GR–EGG54	0.428 ± 0.273	0.773 ± 0.176**	0.639 ± 0.172**
GR–Q1R	0.332 ± 0.735	0.497 ± 0.250	0.441 ± 0.272
GR–Q2R	0.485 ± 0.444	0.500 ± 0.359	0.492 ± 0.320
GR–Q3R	0.572 ± 0.397	0.696 ± 0.880	0.582 ± 0.406
GR–Q4R	0.298 ± 0.475		
GR–ADFR	0.557 ± 0.201**	0.727 ± 0.202**	0.624 ± 0.152**
GR–ADFQ1R	0.448 ± 0.263	0.797 ± 0.172**	0.659 ± 0.167**
GR–ADFQ2R	0.645 ± 0.202**	0.744 ± 0.218**	0.676 ± 0.161**
GR–ADFQ3R	0.691 ± 0.257**	0.744 ± 0.425	0.676 ± 0.242**

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.15: Genetic correlation estimates (±SE) for female traits, ALS–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
ALS–DEATH		0.989 ± 0.002**	
ALS–EGGTOT		0.679 ± 0.314*	
ALS–MAXEGG		–0.170 ± 0.416	
ALS–LASEGG		0.802 ± 0.162**	
ALS–RLS		0.813 ± 0.159**	
ALS–DAY011		–0.265 ± 0.554	
ALS–DAY711		–0.153 ± 0.437	
ALS–EGG54		–0.130 ± 0.478	
ALS–Q1R		0.529 ± 0.341	
ALS–Q2R		0.527 ± 0.443	
ALS–Q3R		1.000 ± 0.726	
ALS–Q4R			
ALS–ADFR		–0.179 ± 0.492	
ALS–ADFQ1R		–0.090 ± 0.490	
ALS–ADFQ2R		–0.562 ± 0.581	
ALS–ADFQ3R		–0.307 ± 0.698	

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.16: Genetic correlation estimates (±SE) for female traits, DEATH–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
DEATH–EGGTOT		0.675 ± 0.316*	
DEATH–MAXEGG		–0.179 ± 0.418	
DEATH–LASEGG		0.817 ± 0.156**	
DEATH–RLS		0.825 ± 0.154**	
DEATH–DAY011		–0.273 ± 0.557	
DEATH–DAY711		–0.170 ± 0.437	
DEATH–EGG54		–0.145 ± 0.480	
DEATH–Q1R		0.531 ± 0.340	
DEATH–Q2R		0.527 ± 0.443	
DEATH–Q3R		0.979 ± 0.710	
DEATH–Q4R			
DEATH–ADFR		–0.210 ± 0.493	
DEATH–ADFQ1R		–0.109 ± 0.494	
DEATH–ADFQ2R		–0.589 ± 0.590	
DEATH–ADFQ3R		–0.353 ± 0.700	

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.17: Genetic correlation estimates (±SE) for female traits, EGGTOT– *, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
EGGTOT–MAXEGG	0.420 ± 0.329	0.296 ± 0.318	0.349 ± 0.255
EGGTOT–LASEGG	0.352 ± 0.646	0.832 ± 0.135**	0.644 ± 0.224**
EGGTOT–RLS	0.348 ± 0.646	0.846 ± 0.131**	0.651 ± 0.222**
EGGTOT–DAY011	0.550 ± 0.404	0.528 ± 0.446	0.537 ± 0.348
EGGTOT–DAY711	0.215 ± 0.394	0.339 ± 0.368	0.279 ± 0.304
EGGTOT–EGG54	0.657 ± 0.369	0.445 ± 0.348	0.537 ± 0.290
EGGTOT–Q1R	1.242 ± 0.852	0.972 ± 0.058**	0.967 ± 0.062**
EGGTOT–Q2R	1.032 ± 0.076**	1.047 ± 0.061**	1.040 ± 0.059**
EGGTOT–Q3R	0.963 ± 0.066**	1.119 ± 0.483*	0.987 ± 0.089**
EGGTOT–Q4R	1.799 ± 0.952		
EGGTOT–ADFR	0.749 ± 0.245**	0.124 ± 0.441	0.463 ± 0.253
EGGTOT–ADFQ1R	0.664 ± 0.301*	0.478 ± 0.315	0.560 ± 0.250*
EGGTOT–ADFQ2R	0.649 ± 0.270*	−0.003 ± 0.497	0.350 ± 0.289
EGGTOT–ADFQ3R	0.552 ± 0.364	−0.371 ± 0.821	0.175 ± 0.413

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.18: Genetic correlation estimates (±SE) for female traits, MAXEGG–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
MAXEGG–LASEGG	–0.734 ± 0.833	–0.199 ± 0.321	–0.352 ± 0.339
MAXEGG–RLS	–0.748 ± 0.830	–0.194 ± 0.323	–0.354 ± 0.341
MAXEGG–DAY011	0.825 ± 0.143**	1.065 ± 0.144**	0.931 ± 0.107**
MAXEGG–DAY711	0.741 ± 0.150**	0.915 ± 0.088**	0.835 ± 0.091**
MAXEGG–EGG54	0.924 ± 0.113**	0.970 ± 0.073**	0.952 ± 0.076**
MAXEGG–Q1R	0.385 ± 0.650	0.430 ± 0.255	0.399 ± 0.267
MAXEGG–Q2R	0.481 ± 0.390	0.349 ± 0.348	0.404 ± 0.292
MAXEGG–Q3R	0.477 ± 0.351	0.122 ± 0.610	0.308 ± 0.344
MAXEGG–Q4R	0.612 ± 0.494		
MAXEGG–ADFR	0.936 ± 0.064**	0.857 ± 0.095**	0.877 ± 0.064**
MAXEGG–ADFQ1R	0.978 ± 0.066**	0.997 ± 0.046**	0.989 ± 0.047**
MAXEGG–ADFQ2R	0.973 ± 0.068**	0.895 ± 0.100**	0.915 ± 0.067**
MAXEGG–ADFQ3R	0.880 ± 0.161**	0.648 ± 0.298*	0.742 ± 0.172**

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.19: Genetic correlation estimates (±SE) for female traits, LASEGG– *, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
LASEGG–RLS	0.999 ± 0.002**	0.999 ± 0.001**	0.999 ± 0.001**
LASEGG–DAY011	-0.156 ± 0.621	-0.095 ± 0.416	-0.110 ± 0.371
LASEGG–DAY711	-0.617 ± 0.649	-0.200 ± 0.331	-0.326 ± 0.314
LASEGG–EGG54	-0.389 ± 0.769	-0.111 ± 0.363	-0.190 ± 0.375
LASEGG–Q1R	0.299 ± 1.363	0.803 ± 0.131**	0.713 ± 0.204**
LASEGG–Q2R	0.262 ± 0.914	0.848 ± 0.149**	0.641 ± 0.263*
LASEGG–Q3R	0.113 ± 0.912	1.000 ± 0.639	0.498 ± 0.393
LASEGG–Q4R	1.359 ± 1.042		
LASEGG–ADFR	-0.328 ± 0.590	-0.444 ± 0.380	-0.363 ± 0.338
LASEGG–ADFQ1R	-0.577 ± 0.918	-0.049 ± 0.370	-0.205 ± 0.397
LASEGG–ADFQ2R	-0.504 ± 0.698	-0.580 ± 0.421	-0.504 ± 0.384
LASEGG–ADFQ3R	-0.621 ± 0.731	-0.867 ± 0.607	-0.660 ± 0.441

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.20: Genetic correlation estimates (±SE) for female traits, RLS–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
RLS–DAY011	-0.175 ± 0.617	-0.089 ± 0.419	-0.113 ± 0.372
RLS–DAY711	-0.617 ± 0.643	-0.188 ± 0.333	-0.320 ± 0.316
RLS–EGG54	-0.410 ± 0.769	-0.099 ± 0.365	-0.189 ± 0.376
RLS–Q1R	0.282 ± 1.387	0.811 ± 0.129**	0.715 ± 0.204**
RLS–Q2R	0.263 ± 0.907	0.858 ± 0.148**	0.647 ± 0.261*
RLS–Q3R	0.113 ± 0.907	1.029 ± 0.667	0.510 ± 0.390
RLS–Q4R	1.350 ± 1.032		
RLS–ADFR	-0.334 ± 0.586	-0.423 ± 0.382	-0.355 ± 0.338
RLS–ADFQ1R	-0.589 ± 0.913	-0.034 ± 0.371	-0.200 ± 0.398
RLS–ADFQ2R	-0.503 ± 0.689	-0.564 ± 0.421	-0.495 ± 0.383
RLS–ADFQ3R	-0.626 ± 0.724	-0.839 ± 0.601	-0.650 ± 0.440

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.21: Genetic correlation estimates (±SE) for female traits, DAY011– *, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
DAY011–DAY711	0.895 ± 0.066**	0.935 ± 0.065**	0.910 ± 0.052**
DAY011–EGG54	0.881 ± 0.166**	0.891 ± 0.164**	0.871 ± 0.135**
DAY011–Q1R	0.831 ± 1.098	0.490 ± 0.368	0.527 ± 0.349
DAY011–Q2R	0.554 ± 0.524	0.560 ± 0.524	0.552 ± 0.432
DAY011–Q3R	0.515 ± 0.468	0.692 ± 1.029	0.566 ± 0.515
DAY011–Q4R	0.908 ± 0.798		
DAY011–ADFR	0.843 ± 0.167**	1.075 ± 0.221**	0.936 ± 0.157**
DAY011–ADFQ1R	0.933 ± 0.118**	0.976 ± 0.117**	0.942 ± 0.096**
DAY011–ADFQ2R	0.818 ± 0.186**	1.050 ± 0.241**	0.911 ± 0.173**
DAY011–ADFQ3R	0.678 ± 0.280*	1.000 ± 0.495*	0.787 ± 0.282**

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.22: Genetic correlation estimates (±SE) for female traits, DAY711– *, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
DAY711–EGG54	0.771 ± 0.176**	0.889 ± 0.118**	0.833 ± 0.116**
DAY711–Q1R	0.280 ± 0.728	0.387 ± 0.304	0.330 ± 0.311
DAY711–Q2R	0.178 ± 0.484	0.298 ± 0.428	0.242 ± 0.365
DAY711–Q3R	0.159 ± 0.440	0.457 ± 0.792	0.263 ± 0.421
DAY711–Q4R	0.521 ± 0.618		
DAY711–ADFR	0.713 ± 0.176**	0.961 ± 0.168**	0.818 ± 0.137**
DAY711–ADFQ1R	0.824 ± 0.130**	0.950 ± 0.077**	0.891 ± 0.081**
DAY711–ADFQ2R	0.689 ± 0.192**	0.841 ± 0.198**	0.752 ± 0.156**
DAY711–ADFQ3R	0.538 ± 0.288	0.976 ± 0.471*	0.697 ± 0.264*

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.23: Genetic correlation estimates (±SE) for female traits, EGG54–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
EGG54–Q1R	0.756 ± 0.895	0509 ± 0.285	0.542 ± 0.301
EGG54–Q2R	0.714 ± 0.477	0.441 ± 0.404	0.556 ± 0.356
EGG54–Q3R	0.699 ± 0.436	0.318 ± 0.688	0.507 ± 0.424
EGG54–Q4R	1.091 ± 0.804		
EGG54–ADFR	1.023 ± 0.140**	0.969 ± 0.128**	0.974 ± 0.107**
EGG54–ADFQ1R	0.958 ± 0.092**	1.007 ± 0.058**	0.987 ± 0.061**
EGG54–ADFQ2R	1.038 ± 0.146**	0.968 ± 0.141**	0.982 ± 0.115**
EGG54–ADFQ3R	0.965 ± 0.256**	0.734 ± 0.384	0.823 ± 0.243**

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.24: Genetic correlation estimates (±SE) for female traits, Q1R–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
Q1R–Q2R	1.068 ± 0.438*	0.935 ± 0.086**	0.910 ± 0.094**
Q1R–Q1R–Q3R	1.131 ± 0.854	1.100 ± 0.715	0.880 ± 0.251**
Q1R–Q4R	3.384 ± 4.937		
Q1R–ADFR	1.074 ± 1.330	0.147 ± 0.380	0.394 ± 0.313

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.25: Genetic correlation estimates (±SE) for female traits, Q2R–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
Q2R–Q3R	0.953 ± 0.172**	1.277 ± 0.712	1.033 ± 0.170**
Q2R–Q4R	2.331 ± 1.695		
Q2R–ADFR	0.872 ± 0.403*	0.222 ± 0.468	0.558 ± 0.313

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.26: Genetic correlation estimates (±SE) for female traits, Q3R–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
Q3R–Q4R	1.723 ± 0.885		
Q3R–ADFR	0.833 ± 0.248**	-0.004 ± 0.874	0.559 ± 0.301

* $P \leq 0.05$, ** $P \leq 0.01$

Table 3.27: Genetic correlation estimates (±SE) for female traits, Q4R–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
Q4R–ADFR	0.800 ± 0.367*	-0.189 ± 3.769	0.575 ± 0.467

* P ≤ 0.05, ** P ≤ 0.01

Table 3.28: Genetic correlation estimates (±SE) for female traits, ADFR–*, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
ADFR–ADFQ1L	1.137 ± 0.137**	0.904 ± 0.130**	1.004 ± 0.099**
ADFR–ADFQ2L	1.052 ± 0.046**	1.101 ± 0.083**	1.071 ± 0.052**
ADFR–ADFQ3L	0.957 ± 0.051**	0.925 ± 0.127**	0.942 ± 0.061**

* P ≤ 0.05, ** P ≤ 0.01

Table 3.29: Genetic correlation estimates (±SE) for female traits, ADFQ1R– *, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
ADFQ1R–ADFQ2R	1.045 ± 0.117**	0.799 ± 0.157**	0.908 ± 0.102**
ADFQ1R–ADFQ3R	1.088 ± 0.273**	0.671 ± 0.408	0.868 ± 0.260**

* P ≤ 0.05, ** P ≤ 0.01

Table 3.30: Genetic correlation estimates (±SE) for female traits, ADFQ2R– *, from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
ADFQ2R–ADFQ3R	1.034 ± 0.133**	1.185 ± 0.349**	1.081 ± 0.166**

* P ≤ 0.05, ** P ≤ 0.01

Table 3.31: Genetic correlation estimates (±SE) for female traits MED1 and MED2 from paternal (r_S) and maternal (r_D) half-sibs and from the combined sire and dam (r_{S+D}) components of variance and covariance.

Traits	r_S	r_D	r_{S+D}
MED1–MED2	0.152 ± 0.472	-0.086 ± 0.461	0.020 ± 0.381
MED1–MAXEGG	0.897 ± 0.076**	0.939 ± 0.052**	0.921 ± 0.049**
MED2–MAXEGG	0.063 ± 0.438	-0.093 ± 0.388	-0.027 ± 0.329

* P ≤ 0.05, ** P ≤ 0.01

Standard errors were large particularly for reproductive variables. It was not surprising that some of the genetic correlation estimates between reproductive variables were greater than one, since each variance and covariance was estimated with error.

Differences between the male paternal and maternal half-sib estimates revealed that maternal effects and/or sex-linked covariation contributed to many of the genetic correlations. Comparison of male and female genetic correlation estimates was necessary to determine which effects were present. For example, differences between the maternal half-brothers and paternal half-sisters' genetic correlation estimates revealed that maternal effects possibly diminished the magnitude of maternal half-sib genetic correlation estimates between PUPN-WT70, PUPN-GR, ADULT-GR, PUPN-ALS, ADULT-ALS, PUPN-DEATH and ADULT-DEATH. In contrast, maternal effects may have had positive contributions to GR-ALS and GR-DEATH. The sire variance components for females were negative for DEATH and ALS, thus genetic correlations between these and other traits could not be estimated.

Comparisons between paternal half-brothers and paternal half-sisters demonstrated sex-linkage was a positive factor for PUPN-WT70, PUPN-GR and ADULT-GR. Though ALS and DEATH appeared not to have any sex-linked covariation by this comparison, maternal half-brother estimates were substantially greater in magnitude than paternal half-sister estimates, indicating negative sex-linked effects for PUPN-ALS, ADULT-ALS, PUPN-DEATH and ADULT-DEATH; and positive effects for GR-ALS and GR-DEATH.

The lack of apparent difference between paternal half-brothers and half-sisters' genetic correlation estimates for DEATH/ALS and other traits, could have been due to the undefined nature of the genetic correlations for paternal half-sisters. Another explanation has been investigated in the discussion.

The partitioning of genetic covariation was obviously only a very rough guide to the architecture of the genetic correlations, due to the high sampling errors of covariance components, and the small size of variance components for most traits. Genetic correlations with large standard errors were the result, thus some differences between genetic correlations, estimated from different variance components for the same trait, were expected due to error. This problem was even more severe for the genetic correlations between traits that could only be measured on females.

Comparison with male estimates was impossible for female reproductive traits so the causal components could not be identified when there were differences between maternal and paternal half-sisters' genetic correlation estimates. For example, if the paternal genetic

correlation estimate between two traits was greater than the maternal genetic correlation estimate, the difference could have been due to either positive sex-linked covariation between the traits or maternal effects influencing the traits antagonistically (Table 3.2). If both sources of covariation were present they could balance each other's effects so that paternal and maternal estimates appeared similar.

An example of this was the genetic correlation estimate ADULT-WT70. In males the estimates for ADULT-WT70 were similar, but for females the paternal estimate was much greater than the maternal half-sister estimate, suggesting positive sex-linked covariation and/or antagonistic maternal effects. The difference between paternal half-brothers' and half-sisters' estimates comfirmed the presence of positive sex-linkage, whilst some antagonistic maternal effects could have been responsible for the difference in estimates between maternal half-brothers and paternal half-sisters. PUPN-WT70 had a similar genetic architecture, so it was likely that ADULT-WT70 experienced both positive sex-linked covariation and antagonistic maternal effects. The conclusion about the estimates for males was that both effects had cancelled each other out, so to speak, and similar genetic correlation estimates for sire and dam components were the result.

There were so many results available for females, that only a summary of the major points of interest has been attempted here.

MAXDAY, ALS, DEATH and ADFQ4R had negative sire variance components, thus paternal genetic correlations for these traits were undefined. DEATH and ALS had positive dam variance components so that maternal half-sister genetic correlation estimates were obtained, some of which were significant. As explained before these maternal estimates are difficult to interpret.

GR and WT70 were significantly and positively correlated with many of the reproductive variables. PUPN and ADULT only approached significance with EGG54 but otherwise had genetic correlations of low to moderate magnitude with other traits. Since ADULT and PUPN had positive genetic correlations of moderate, though insignificant, magnitude with WT70, it was likely that they would be correlated with some fecundity indices. There was a very high positive correlation between WT70 and GR, but not between ADULT and GR. ADULT had a much higher genetic correlation with WT70 than with GR. The value of WT70 must have had a much greater effect on the value of GR than the value of ADULT.

Correlations for GR and WT70 with all other traits except developmental duration were extremely similar. I conclude that there was a physiological connection between WT70 and

fecundity indices, which GR reflected, being determined largely by WT70.

The relationship between developmental duration and ALS or DEATH could not be determined, but the relationship with RLS, a variable closely linked to longevity, was antagonistic, though of very low magnitude and not approaching significance.

RLS and LASEGG were highly positively correlated ($r_{S+D} = 0.999 \pm 0.001$) and their correlations with other traits were very similar, so they are treated in this discussion as the one trait. RLS had large negative correlations with some important indices of fecundity: MAXEGG, DAY711, ADFQ1R, ADFQ2R and ADFQ3R. These were the only large negative correlations found between female variables. They were not significant but this was not unexpected because of the low heritabilities of RLS and LASEGG. RLS had a large positive correlation with Q4R but this was expected because the larger the last quarter of RLS, the larger Q4R would be. The value of Q4R was probably determined more by the number of days that it incorporated than the rate of egg lay by the individual beetles. This conclusion was suggested by the positive, but small, correlations between RLS-Q1R (r_S), RLS-Q2R (r_S) and RLS-Q3R (r_S), compared to the much larger negative correlations between RLS-ADFQ1R (r_S), RLS-ADFQ2R (r_{S+D}) and RLS-ADFQ3R (r_S). Of course, a large last quarter of RLS was more likely to include higher egg-laying rates within its time interval. Any genetic correlations with Q4R, however, were suspect because of its low heritability (Table 3.8). Conclusions from these results were that beetles with higher rates of egg-lay appeared to have shorter reproductive life-spans.

Interestingly enough, the maternal half-sister genetic correlation estimates for DEATH-ADFQ2R and ALS-ADFQ2R were both negative and of relatively high magnitude. DEATH and ALS were also negatively correlated, but insignificantly, with ADFQ1R and ADFQ3R. The negative correlation was either due to negative maternal effects or positive sex-linked covariation. Maternal effects were unlikely to be a source of covariation for these two later life traits and sex-linked covariation was present between life-span indices and other traits in males, thus positive sex-linkage was probably responsible.

The negative genetic correlation estimates between DEATH/ ALS and fecundity indices corroborate the existence of a negative relationship between fecundity and life-span. This relationship is general and not limited to trade-offs between early fecundity and reproductive life-span.

All genetic correlations between fecundity indices were positive, some of very large magnitude. The magnitude of correlations between ADFQ1R, ADFQ2R and ADFQ3R

were very high and significant. These traits were also all highly correlated with other reproductive traits such as: MAXEGG, DAY011, DAY711, EGG54 and ADFR. The genetic correlation estimates of ADFQ4R were indefinite but Q4R was strongly and positively correlated with all other fecundity indices. The correlations with early life variables such as DAY011, DAY711, MAXEGG and EGG54, even with reservations about the validity of genetic correlation estimates involving Q4R, indicate that early and late life fecundity were not negatively correlated even if genetic variation for late life fecundity was minimal.

These results suggest that individuals laid eggs at a rate which was genetically predetermined, and that as they aged their rate of egg lay with respect to other individuals in the population remained at the same level. Thus beetles with a high rate of egg production early in life maintained their relatively high levels throughout their life-spans. There did not appear to be any trade-offs between early and late life fecundity which explained the finding that RLS was negatively correlated with both early and late life fecundity indices.

The low and insignificant genetic correlation estimate between MED1 and MED2 was suggestive that the trade-off between fecundity and reproductive life-span was balanced in MED2. The high positive correlation between MED1 and MAXEGG ($r_{S+D} = 0.921 \pm 0.049$) confirmed that beetles with high rates of egg production laid more eggs than low producers before the median. (MAXEGG has a genetic correlation of $r_{S+D} = 0.877 \pm 0.064$ with ADFR.) Since the number of eggs laid after the median was unrelated to the number of eggs laid before, it was deduced that for some, but not the majority of, beetles with high rates of egg lay, their shortened reproductive life-spans had the effect of decreasing their total egg output after day 93 to less than that of beetles with low rates of egg lay.

Rate of egg lay had little to do with the total number of eggs laid by an individual after the median. Overall beetles with high rates of egg production produced more eggs than beetles with low rates (EGGTOT-ADFR $r_S = 0.749 \pm 0.245$).

3.4 Discussion

3.4.1 The Cost of Reproduction

For the purposes of this discussion it was assumed that the number of eggs laid by an individual female was a direct measure of the number of its progeny represented in the next generation if it had been mated to a male drawn at random from the population. For this to be a valid assumption, the number of eggs laid by a female must have been unaffected

by being mated to her brother. If egg lay were depressed, different genotypes could be differentially affected, and the calculation of genetic variances for fecundity traits may have been affected.

For example, beetles with high rates of egg lay might have had their egg lay rate relatively more depressed by inbreeding than beetles with low rates, and thus genetic variation would have been minimised. However, a comparison between females mated to full-sibs and those mated to unrelated males revealed no significant differences between the means and variances for number of eggs laid on day 68.

If the number of eggs laid is a direct measure of the number of progeny represented in the next generation in a randomly mating population, then there should be no trade-off between fecundity and survival of offspring from egg to adulthood. Survival of offspring up to day 20 was positively correlated with fecundity in the control population set up for the selection experiment, so the number of eggs laid by a female was assumed to be a satisfactory measure of its contribution to the next generation.

Since rate of egg lay was unaffected by inbreeding depression, the number of eggs laid during a time interval by a female, relative to other females of the diallel analysis, was a measure of her reproductive investment, provided egg size and composition are constant. Therefore, the relationships between early and late life fecundity or survival were used to determine if there was a cost to reproduction.

On the other hand, survival costs were present, as indicated by negative genetic correlations between various fecundity indices and reproductive life-span, and between ADFQ2R and ALS (most probably through sex-linked covariation). A highly significant positive maternal half-sister genetic correlation between ALS and RLS ($r_D = 0.813 \pm 0.159$) was further reason to interpret the negative genetic correlations between fecundity and RLS as demonstrative of survival costs.

Beetles which had high rates of egg lay maintained their advantage throughout most, if not all, of their reproductive life-spans. Their total eggs laid were greater than lower producers but this selective advantage must be weighed against the selective disadvantage of a shorter reproductive life-span. Since overwintering capacity is an important feature of the survival of *T. castaneum* populations in the wild, a longer reproductive life-span might be quite an advantage for an individual. Such a cost should more properly be termed a "reproductive survival" cost.

To confirm the universality of reproductive costs for *T. castaneum,* other natural populations need to be examined in a range of environments. The results of Sokal (1970) confirm the presence of survival costs in two laboratory populations of *T. castaneum.* Mertz (1975) selected for early fecundity and did enhance it at apparently the expense of late fecundity and adult life-span. However, he found no correlation between the reduction in longevity and the fecundity response, and concluded the reduction in longevity was unrelated to selection for early fecundity. His conclusions though may have differed if he had examined the relationship between fecundity and reproductive life-span. He did note that some of the later life "fecundity differences could be attributed to the short postreproductive period which portends death". Thus his data were suggestive that reproductive life-span had shortened and its shortening was at least partially responsible for the decrease in late life fecundity.

McRae (1988), using two laboratory temperatures and two *T. castaneum* populations recently derived from the wild, found negative genetic correlations between early reproductive traits and adult life-span but positive correlations between early and late life fecundity indices within both populations at both temperatures. McRae (1988) found heritable genetic variation for adult life-span, which I did not, and so presented genetic correlations between early fecundity indices and adult life-span. However, the genetic correlations between RLS and ALS approximated to 1 for both populations in both temperatures, and genetic correlations between these two and other traits were very similar so he did not present the correlations for RLS. My results confirm his findings, and in conjunction with the previously mentioned studies suggest that survival costs are a feature of the life-history of *T. castaneum.* The fact that McRae (1988) found negative genetic correlations in both environments, indicates that my assumption that fundamental genetic constraints are operational in all "realistic" environments was valid and justifies my use of a single set of experimental conditions.

Some of McRae's (1988) negative genetic correlations between ALS and reproductive indices were significant. This appears to be due to his standard error estimates which were only approximations and which he acknowledged were minimal. I used no approximations to calculate standard errors in this work. As well, a greater number of female individuals were measured and recorded than by McRae (1988) (1090 compared to less than 830). McRae's (1988) levels of significance should be treated with caution. Further experiments with *T. castaneum* will have to use larger numbers than used here to obtain better estimates of

genetic correlations between fecundity indices and RLS.

Though genetic correlations between fecundity indices were high and positive, they did not approach unity, so modification of reproductive schedule should be possible by selection. Many, particularly early life fecundity indices had moderate levels of additive genetic variation, so should be able to respond to changes in natural selection pressures deriving from ecological variables and/or mortality schedule of adults or juveniles (see Morris 1986).

In populations, such as laboratory populations, where high egg production has a greater selective advantage than longer reproductive life-span, negative genetic correlations could well appear between early and late life fecundity. Early and late life fecundities could both experience strong directional selection up to the point where only alleles with antagonistic effects on both traits would be left segregating. Wallinga and Bakker (1978) induced a trade-off situation between present and future reproduction by selecting for large litter size in mice.

Though a cost to reproduction does appear to exist in *T. castaneum* populations, it would be unwise to assume that it is the only internal constraint influencing the reproductive schedule. Bell (1984a) has proposed that perhaps present and future reproduction are positively correlated within any given niche, whereas age-specific reproduction is negatively correlated between niches. Thus one individual may produce more offspring in one environment, but do badly in another. If the population is dispersed over a number of niches, or is a colonising species, then natural selection will act to create some intermediate phenotype which "bet-hedges" its chance of reproduction across all niches. Baldwin and Dingle (1986) have found some indications that negative genetic correlations between expressions of the same life-history trait measured at different temperatures do exist for at least some of the important fitness traits in natural populations of *Oncopeltus fasciatus*. On the other hand, McRae (1988) has shown that across-environment genetic correlations between character states were positive for both reproductive and non-reproductive traits in two populations of *T. castaneum*.

Some researchers have suggested that there is a determinant relationship between growth rate/body size and reproduction (Koufopanou and Bell 1984; Reznick 1983). It has been proposed that selective forces do not act upon reproduction, but growth rate/size has evolved with corresponding adjustments in the reproductive schedule, either because of the developmental interrelationship of all life-history traits (Reznick 1983), or because it is

a necessary adaptation to counterbalance the changes in developmental rate (Tinkle, Wilbur and Tilley 1970).

It is well known that in ectotherms large females often have greater longevity and higher fecundity, and large males usually have enhanced mating success (Butlin and Day 1985; Gilbert 1984a; Hinton 1981, Vol.1 for examples among insect species; Mousseau and Roff 1987). Dunham and Miles (1985) have also found many important life-history variables scale allometrically with body size in squamate reptiles.

In this study growth rate and weight in particular were significantly and positively correlated with many fecundity indices. McRae (1988) found that genetic correlations between bodyweight and fecundity indices were always positive at 37°C for both *T. castaneum* populations. One population did exhibit some negative genetic correlations between bodyweight and fecundity at 33°C but these were very small and not significant.

Though a determinant relationship between growth rate/body size and reproduction cannot be rejected on the basis of data here, "it is at least as logical to explain variation in body size as an allometrically forced result of selection on life histories" (Dunham and Miles 1985). Fecundity has been shown to respond to direct selection (Mertz 1975; Rose and Charlesworth 1981b; Wallinga and Bakker 1978; and many others), so it isn't likely that changes in size are solely, if at all, responsible for changes in the levels of reproduction. Furthermore, fecundity and body size are insignificantly or negatively correlated in birds and mammals (Mousseau and Roff 1987) which suggests that the relationship between growth rate/size and fecundity is not universal. The relationship between weight and the reproductive schedule probably reflects the physiological/developmental interrelationship of the traits (Gilbert 1984c) and variation in the reproductive schedule is not necessarily due to variation in body size at all (Leather 1988). Body size may also act as a constraint on levels of reproduction through developmental disruption if either trait is expressed at very high levels, as demonstrated by Gilbert (1984c).

Evidence from many other species in many different environments, needs to be assessed before reproductive cost can be deduced to act as a universal constraint on life-histories. Worthy of further investigation is the relationship between weight and the reproductive schedule, and the possible genetic correlations between age-specific reproduction in different environments.

3.4.2 Interpretation of Genetic Variances and Covariances

Any quantitative genetic analysis is specific to a particular population in a particular environment at a particular time (Barker and Thomas 1987; Falconer 1981). Estimates of heritabilities and genetic correlations are only useful for predicting potential responses to selection in the same environment, because estimates can be biased by genotype-environment interaction components or changing effects of genes.

The laboratory environment was a realistic if optimal one for *Tribolium castaneum* (see section 2.2), so that it should not have exposed previously hidden genetic variation or changed gene expression. There should have been minimal linkage phase disequilibrium and the population was probably at demographic equilibrium (sections 2.2 and 2.3), so estimates of heritabilities and genetic correlations should have been stable and realistic for this population in this particular environment.

Dawson (1965b) estimated the heritability of pupation time in a *T. castaneum* population using a diallel design. h^2_S was 0.13, h^2_D was 0.41 and the proportion of maternal effects 0.07. He also found substantial dominance for the trait if epistasis was assumed negligible. The population used here had higher heritabilities for both sexes but within the established range (Dawson and Riddle 1983), and higher maternal effects (h^2_D males — h^2_S females/4 = 0.166). Levels of dominance and/or epistasis did differ between the sexes for developmental rate: time to pupation and adult emergence in males both had negative interaction variance components, whilst these variance components were positive in females. Since Dawson (1965b) did not distinguish between males and females in his analysis, it is not surprising that he concluded pupation time per se exhibited dominance.

As both sexes had significant levels of additive genetic variation for pupation time, it should be possible to predict future short-term response to selection for developmental rate with a reasonable degree of accuracy.

Past selection history was not very amenable to deduction because it was impossible to determine if heritabilities and genetic correlations had been altered by the move from the wild into the laboratory either by genotype-environment interaction or changes in gene expression. Constant laboratory conditions minimised environmental variance, so estimated heritabilities may have been larger than if estimated in the wild. Conclusions about past selection history are tentative, as are inferences drawn about the maintenance of genetic variation.

Low additive genetic variance for a trait can be due to past strong directional selection

upon the trait. Life-history traits are expected to have experienced strong directional selection, so it is not surprising that most of them exhibited low to moderate levels of additive genetic variation. Lewontin (1965) predicted that populations of colonisers will have low amounts of additive genetic variance for developmental time but relatively higher amounts for fecundity. Generally this maxim did appear to be true for this population of *T. castaneum.*

Fitness characters are also expected to have large amounts of dominance variation (Falconer 1981; Robertson 1955; Travis, Emerson and Blouin 1987). Most of the reproductive and life-span traits had substantial amounts of non-additive genetic variance, which was probably predominantly dominance. However, it was impossible to differentiate between dominance and epistasis, and epistasis may be as important as dominance for some traits in *Tribolium* (Goodwill 1975,1978), so it is impossible to confirm the presence of dominance variation.

Developmental rate and size indices had relatively low levels of nonadditive genetic variation as compared with most of the other traits, thus they are unlikely to display substantial amounts of dominance.

DEATH, ALS and MAXDAY all had negative sire variance components for females but extremely large estimates of non-additive genetic variance. Clare and Luckinbill (1985) have shown that a stressful environment for *Drosophila melanogaster* larvae affects the expression of genes controlling aging so that genes for short life which are dominant in an optimal environment, display additive inheritance in suboptimal environments. Arking and Clare (1986) have suggested that stressful environments decanalise phenotypes and reveal the hidden additive genetic variation of developmentally buffered traits. This hidden genetic variation in unstressed environments may appear as dominance variation. Lack of additive genetic variation and large estimates of non-additive genetic variance for the three traits, indicate that their additive genetic variation may be suppressed by developmental buffering systems in optimal conditions, but not in suboptimal environments. This inference must be tested experimentally. Developmentally buffered characters have usually experienced strong selection and it is highly likely that timing of reproduction and timing of death would be important components of life-history strategies.

The low additive genetic variation and (possibly) high dominance variation of longevity (DEATH, ALS) and timing of reproduction (MAXDAY) is suggestive, that even if their genetic variation is not suppressed by developmentally buffering systems, these characters

have experienced strong selection. For a colonising species such as *Tribolium castaneum,* timing of attainment to peak reproduction would be a particularly important component in determining its success as a coloniser.

It is easy to explain low additive genetic variance for fitness traits but not so easy to explain higher levels as exhibited by WT70, GR, DAY711, ADFR and ADFQ2R. The usual explanation is that selection has been weak or non-existent but this is unlikely for these important life-history characters. The estimates of additive genetic variance are probably not unusual in their magnitude: the heritability estimate for DAY711 was similar to that of Orozco and Bell (1974) for virgin egg lay between days 7 and 11 following adult emergence. Their pooled estimate was 0.37 $\pm$ 0.02, as compared with the estimate here of 0.375 $\pm$ 0.110.

In the past selection history of the population, fluctuating selection pressures and genotype-environment interactions, products of environmental heterogeneity, may have been responsible for maintaining the additive genetic variation of fitness components (Mitchell-Olds 1986; Murphy *et al.* 1983). If environmental heterogeneity were responsible, it is surprising that more traits did not have substantial amounts of additive genetic variation. It cannot be ruled out as a responsible factor nor can balancing selection and pleiotropic overdominance, nor mutation-stabilising selection. None of these theorems for the maintenance of genetic variation was tested here.

However, antagonistic pleiotropy could have been the responsible mechanism. There were negative genetic correlations between RLS–DAY711 (r_S = –0.617 $\pm$ 0.066), RLS–ADFQ2R (r_{S+D} = –0.495 $\pm$ 0.383) and RLS–ADFR (r_S = –0.334 $\pm$ 0.586) which may have acted as "constraints" on the loss of genetic variation. DAY711, ADFQ2R and ADFR also had large positive genetic correlations with other traits which were negatively correlated with RLS (e.g. MAXEGG–RLS). Positive genetic correlations may also act as constraints if selection pressures on traits are antagonistic e.g. a decrease in ADULT, which is favoured by natural selection, would adversely affect fecundity and weight.

When life-history traits are highly intercorrelated, as was found here, then negative genetic correlations between a few important characters are probably sufficient to maintain additive genetic variance at moderate levels for many of the other correlated characters. Analyses of covariance between life-history traits must be comprehensive in the number and type of traits measured to determine whether antagonistic pleiotropy is a possible causal mechanism for conserving variation.

Mitchell-Olds (1986) concluded from experimental data that antagonistic pleiotropy was not a constraint on response to natural selection by fitness components of *Impatiens capensis*. Yet he did not examine the genetic correlations between reproductive life-span or adult life-span and other life-history traits. It is doubtful then whether his conclusion was valid. This criticism of incompleteness is pertinent to most of the work which has found only positive genetic correlations between major fitness characters (e.g. Bell 1984a,b; Butlin and Day 1985; Druger and Matzke 1977; Murphy *et al.* 1983; Stearns 1983a; Wu 1981).

Of course antagonistic pleiotropy may not be responsible for maintaining additive genetic variation for fitness components in every population of all species, and it could quite probably even when existent, work in conjunction with one or more of the other mechanisms mentioned beforehand.

One of the objections to the role of the universality of antagonistic pleiotropy was that life-history characters do not exhibit dominance (Rose, Service and Hutchinson 1987). Dominance of effect on single fitness components was seen as a crucial aspect of the ability of antagonistic pleiotropy to maintain variation. Yet, for the Echarina population, though GR and WT70 had low amounts of non-additive genetic variation, ADFR, DAY711 and ADFQ2R all exhibited reasonable amounts, part of which was more than likely due to dominance. As mentioned before, a number of life-history traits exhibited quite large amounts of non-additive genetic variation. Other workers have also found that life-history characters in other species exhibit high levels of dominance (Emerson, Travis and Blouin 1988), so I believe the objection of Rose *et al.* (1987) is not pertinent.

Rose *et al.* (1987) rejected the sole role of antagonistic pleiotropy as a force maintaining genetic variation, when they found that there was no antagonistic relationship between early fecundity and subsidiary characters contributing to longevity. This work though has more to do with the evolution of senescence than with the maintenance of genetic variation, as it is obvious that relationships between characters associated with longevity and early fecundity are only a small proportion of the many that make up a life-history.

Many of the genetic correlations between the fitness components were large and positive, in contrast to the expected low and/or large negative genetic correlations due to strong directional selection upon one or both traits. Most of the large positive correlations were between characters that were obviously related such as fecundity indices. The exceptions were positive correlations of quite high magnitude between WT70 or GR and many fecundity indices. Genetic variation was probably not eliminated, even though one could expect strong

Table 3.32: Predictions for correlated responses to selection for increased and decreased duration of time to adult emergence.

Traits	decreased duration of adult emergence	Increased duration of adult emergence
PUPN	Decrease	Increase
WT70	Decrease	Increase
Fecundity	Decrease	Increase
RLS	No effect	

directional selection to be acting on both traits, because of the negative genetic correlations between WT70 and developmental rate, and between fecundity and RLS.

Selection for fast and slow developmental rate should only produce a small response in characters correlated with developmental rate. Even though some genetic correlations were reasonably high, the genetic variation for most life-history traits was low and most traits would be unable to respond rapidly, if at all, to changing selection pressures. Predictions given in Table 3.32 are purely qualitative, as the degree to which traits would respond to selection depends on the genetic variance-covariance matrix which is quite complex.

GR would be expected to change in the same direction as WT70, but as it has a very low correlation with ADULT, its response should be slower. These predictions are based on the surmise that selection would not reveal hidden genetic variation for developmental rate or any of the other life-history traits.

3.4.3 Evolution of Senescence

The results of this experiment can be used to test the veracity of a number of hypotheses concerning the evolution of senescence. Many of the genetic correlation estimates referred to in this section are not significantly different from zero, but in recognition of this problem, hypotheses are never proved or disproved on the basis of one estimate. I looked for patterns as revealed by a number of estimates to assess the validity of conjectures.

The results do not support the Unitary hypothesis of senescence, as a number of negative genetic correlations between major life-history traits were found. In particular there were negative relationships between fecundity and reproductive life-span, and between developmental rate and fecundity. It could be argued, as a bolster to the Unitary hypothesis,

that the population had experienced a stable "wild" environment, that selection had been consistent and unidirectional and all alleles were fixed except those with antagonistic effects. This is unlikely as some genetic correlations between life-history traits were large and positive, and some life-history traits displayed substantial amounts of additive genetic variation.

The Unitary hypothesis of senescence has served a purpose in drawing attention to the "universal pleiotropy" between life-history traits. Beyond this it gives no understanding of the mechanisms which maintain senescence as a universal feature of life-histories. Other hypotheses are more specific and amenable to investigation by further experimentation as they offer concrete predictions.

The case for accumulation of mutations with age as primarily responsible for senescence, has also been weakened. Rose and Charlesworth (1981a) have stated that absence of increasing additive genetic variance for fecundity with age was sufficient basis to reject the Mutation Accumulation theory of senescence. As stated previously, no such increase was found for this population of *Tribolium castaneum.* Rose and Charlesworth's (1981a) predictions for the Mutation Accumulation hypothesis have been criticised, but the detection here of substantial genetic correlations between early and late life history characters, in particular RLS and fecundity, further undermines the hypothesis. If senescence were the result of accumulated mutations then RLS, a manifestation of senescence and prefiguring imminent death, should not be influenced to any great extent by genes which influence early fecundity.

Predictions of the Developmental hypothesis were not tested but some results were witness to the epigenetic nature of longevity. PUPN, ADULT, WT70 and GR had very low, insignificant negative genetic correlations with RLS (DEATH and ALS were indeterminant), but these were expected in optimum constant conditions (Economos and Lints 1986a,b; Mayer and Baker 1984).

The negative genetic correlations based on dam components for males between DEATH/ALS and PUPN/ADULT, were much greater than those for females. The differences may have been due to negative sex-linked covariation, but this sex-linked covariation was not revealed by comparisons between paternal half-brothers' and half-sisters' genetic correlation estimates. Another explanation could be that longevity was more developmentally buffered in females than males. Mertz (1975) found that selection affected longevity of males far more than longevity of females in a population of *T. castaneum.* Economos and Lints (1986b) also

found that in *Drosophila melanogaster* after disturbing developmental processes by increasing amplitude of oscillation of temperature, female life-span was correlated more highly with growth rate than was male life-span. These results are suggestive of a generally stronger relationship between developmental processes and life-span for females than for males. The difference between male and female genetic correlations also could be the result of the operation of different genes in the two sexes. Luckinbill, Graves, Reed and Koestawang (1988) have shown in *D. melanogaster* that genes determining longevity are nonidentical in males and females.

When Lints and Hoste (1974) proposed that a non-genetic maternal effect governed longevity, they were perhaps not completely in error as maternal effects are present for DEATH, ALS and RLS. Other workers have found that ageing appears to be caused by changes in the cytoplasm and loss of mitochondrial DNA could be the key event in this process (Fleming 1986; Massie 1986). Thus mitochondria, inherited only from the mother, may be the source of these "maternal effects", and if so, would provide an explanation for maternal influence on late life characters.

Antagonistic pleiotropy as a basis for senescence gains some credence from this experiment. Firstly, reproduction was more variable among older individuals (Table 3.5), so late fecundity was probably less important than early fecundity (Bell 1980). Secondly, there were negative genetic correlations between early fecundity and reproductive life-span, whilst all defined fecundity indices were positively correlated with each other. It appears unlikely that senescence was the result of genes that had beneficial effects on life-history traits early in life but deleterious ones later in life, otherwise one would expect negative genetic correlations between early life fecundity indices and ADFQ3R. If the relationships here are present for all *T. castaneum* populations, selection for early fecundity in *Tribolium* should depress longevity. Sokal (1970) and Mertz (1975) found that longevity was depressed through selection for early viability and fecundity respectively. Selection for late fecundity would either:

a) increase late fecundity at the expense of longevity; or

b) increase late fecundity and because of indirect selection for late-life viability, increase longevity and reproductive life-span. This increase in reproductive life-span may depress early fecundity.

This second alternative may be the explanation for the apparent response to selection

for late fecundity in populations of *D. melanogaster* by Rose and Charlesworth (1981b) and Luckinbill and Clare (1985). Selection for early fecundity in *D. melanogaster* did not depress longevity; but Luckinbill and Clare (1985) have hypothesised that longevity may have a minimum threshold in populations of species selected for high early fecundity such as *Drosophila*.

A negative genetic correlation between reproduction and reproductive life-span is one of the postulates of proof for the Running-out-of-Program (RP) hypothesis. The RP hypothesis does not require a negative genetic correlation between early fecundity and life-span, but a generally negative relationship between fecundity and life-span. McRae (1988) has confirmed this relationship in other populations of *T. castaneum*.

Cutler's RP hypothesis also proposes an epigenetic component to longevity which has been confirmed by, among others, Clare and Luckinbill (1985), Economos and Lints (1986a,b) and Luckinbill and Clare (1985). Experimental evidence here does not prove or disprove a genetic link between longevity and duration of development, so Sacher's and Cutler's versions of the RP hypothesis cannot be compared for evaluation. Clare and Arking (1986) have suggested that Cutler's (1982) postulate that life-span is determined by the time of action and degree of expression of specific longevity determinant genes, is consistent with the change of expression of genes when individuals are exposed to stressful environments, to reveal substantial additive genetic variation for longevity in *Drosophila*.

As mentioned in the previous section, the genetic architecture of longevity in *T. castaneum* would certainly be compatible with this hypothesis of suppression of genetic variation in optimal conditions by developmental buffering systems. Even if the genetic variation is not developmentally buffered, age at death does appear to have experienced strong directional selection, and thus it is difficult to imagine that it is merely the result of indirect selection as postulated by other evolutionary theories of senescence.

At this point, it appears that the relationship between development and longevity is in need of elucidation; and that, the RP hypothesis of senescence may be the most fruitful avenue for further exploration by workers interested in the evolution of senescence.