AN ABSTRACT OF THE THESIS OF

Dave F. Zirkle for the degree of Doctor of Philosophy in Zoology presented on January 10, 1990.

Title : A Quantitative Genetic Analysis of Fitness Characters in Tribolium confusum

Redacted for privacy

Abstract approved : _____
Peter S. Dawson

The objective of this study is to assess the quantitative genetic structure of fitness characters in Tribolium confusum flour beetles. Estimates of genetic variation and covariation for a series of fitness components (two measurements of survival, larval weight, development time, fecundity, and female productivity) were obtained from two populations of T. confusum (populations 1-1 and 1-2). Prior to sampling for the present experiment, the two populations had been maintained under identical, controlled culture conditions for over 10 years.

An analysis of genotypic means indicates extensive genetic differentiation has occurred between the two populations for all characters except development time. Population 1-1 contains relatively large amounts of additive genetic variation for both measurements of survival, larval weight, fecundity, and female productivity. Many of the genetic variance component estimates from population 1-2 are negative. Both populations contain significant amounts of nonadditive variation for fecundity and female productivity.

Population 1-1 also contains significant amounts of genetic covariation for several of the characters. Positive genetic covariation exists for the two measurements of survival, while the genetic correlation between survival and fecundity is large and negative. Many of the genetic correlation estimates from

population 1-2 are undefined since the variance component estimates are negative.

There appears to be a relationship between genetic variation and covariation for survival and fecundity in population 1-1. Significant amounts of genetic variation were detected for survival and fecundity in addition to a significant negative genetic correlation between these characters. This result is consistent with the theoretical expectation that antagonistic pleiotropy among fitness components can maintain genetic variation in the individual characters.

This type of genetic structure is consistent with the shifting balance theory of evolution and could explain the substantial amount of genetic differentiation observed between the two populations. That is, strong negative genetic correlations among fitness components can not only maintain genetic variation in individual characters, but also generate a complex adaptive landscape where stochastic forces operating in conjunction with systematic forces can produce large population differences among the various traits.

A Quantitative Genetic Analysis of Fitness Characters In *Tribolium confusum*

by
Dave F. Zirkle

A THESIS
submitted to
Oregon State University

in partial fulfillment of
the requirements for the
degree of

Doctor of Philosophy

Completed January 10, 1990

Commencement June 1990

APPROVED:

Redacted for privacy

Professor of Zoology in charge of major

Redacted for privacy

Chairperson of Department of Zoology

Redacted for privacy

Dean of Graduate School

Date thesis is presented January 10, 1990

Typed by Dave F. Zirkle

Acknowledgments

I am grateful to my wife, Donna Zirkle, for her patience and support during this endeavor. I thank Pete Dawson, William Hohenboken, and Russell Riddle for many illuminating discussions on the topic of quantitative genetics and Charles King, Art Boucot, Chuck Davis, Joe Beatty, Monica Geber, Paul Bellaty, and Debbie Rossi for their support and help in completing this project. I also thank Pete Dawson, Steve Knapp, Charles King, Kenneth Rowe, and Ralph Berry for their review of this manuscript. Finally, I thank Pete for instilling in me an appreciation for the field of population genetics, for his continual support through both good and bad times, and most of all, his friendship.

TABLE OF CONTENTS

LIST OF TABLES

List of Tables continued

List of Tables continued

List of Tables continued

A QUANTITATIVE GENETIC ANALYSIS OF FITNESS CHARACTERS IN *TRIBOLIUM CONFUSUM*

INTRODUCTION

Clearly, it is important to quantify the amount of genetic variation for fitness components in order to predict the course of evolution for such traits under the influence of natural selection. However, if antagonistic pleiotropy is common, then it also becomes necessary to estimate genetic correlations among fitness components in order to make accurate evolutionary predictions. To provide this type of information, I have obtained estimates of genetic variation and covariation for a series of fitness components in two populations of flour beetles (*Tribolium confusum*). The objectives of the present study are three-fold.

(1) How much genetic variability for characters closely related to fitness is present in populations subjected to long-term evolutionary forces? A corollary of this objective involves an attempt to accurately partition the total genetic variance for fitness characters into additive and nonadditive components.

(2) What are the genetic relationships among fitness components in populations subjected to long-term evolutionary forces? More specifically, what are the signs and magnitudes of the genetic correlations among characters closely related to fitness?

(3) What is the relationship between genetic variation and covariation for fitness components in populations of *T. confusum*? That is, does antagonistic pleiotropy, expressed as negative

genetic correlations among fitness components, play an important role by setting boundaries and constraints on the evolutionary process such that genetic variation for individual traits is maintained?

Several investigators have shown that natural populations contain relatively large amounts of genetic variation for characters closely related to fitness (reviewed by Istock, 1983, 1984; Dingle, 1984). However, Robertson (1955) has presented reasons for expecting the total genetic variance of fitness components at equilibrium to be composed primarily of nonadditive genetic variation. Unfortunately, with the exception of a few studies (e.g. Dawson, 1965; Via, 1984), most individuals have not partitioned the total genetic variance of fitness components into additive and nonadditive components. Furthermore, most previous estimates of genetic variance components for fitness characters have been characterized by rather large sampling errors. The present study was designed to overcome these weaknesses by obtaining precise estimates of additive and nonadditive components of genetic variance for a number of fitness characters.

Antagonistic pleiotropy expressed as negative genetic correlations has been proposed as a mechanism capable of maintaining additive genetic variation for individual fitness characters (e.g. Caspari, 1950; Lerner, 1954; Dickerson, 1955; Robertson, 1955; Wallace, 1959, Wright, 1977; Rose, 1982, 1983, 1984a, 1984b, 1984c; Dawson and Riddle, 1983; Istock, 1983, 1984; Dingle, 1984; Riddle, Dawson, and Zirkle, 1986). The present study is also designed to investigate this hypothesis by comparing the amounts of additive genetic variation for individual fitness components and the signs and magnitudes of the additive genetic correlations among these characters.

LITERATURE REVIEW

I. Scientific Background

The two fundamental tasks of population genetics are a description of and an explanation for the genetic composition of a population. Very early in the history of the field, important descriptive parameters for undertaking the former task were readily agreed upon. These parameters are allele and genotype frequencies in a population, or other derived measures, and are based on the genetic mechanisms discovered by Mendel. The latter task of providing an explanation for the genetic composition of a population involves establishing a theoretical basis for the field of population genetics. The work of R.A. Fisher (1918, 1930), Sewall Wright (1921, 1931), and J.B.S. Haldane (1932) combined the genetic mechanisms discovered by Mendel with principles of evolution to arrive at what is generally considered to be the theoretical foundation of population genetics. This "classical" theory (reviewed by Provine, 1971; Lewontin, 1974) emphasized the dominant role of natural selection in evolution and ascribed importance to mutation as a source of genetic variation on which natural selection could act. However, the vast majority of mutations were considered to be deleterious and rapidly removed from a population, while only a small portion of mutations were advantageous, and these were quickly fixed in the population by the action of natural selection. This view of natural selection led to the prediction that individuals in a population should be genetically uniform (i.e. homozygous for the "wild type" allele) and consequently populations should exhibit little genetic variation. Basically, the only genetic variation present in a population was

due to deleterious alleles held at low frequencies as a result of the joint effects of natural selection and mutation (e.g. Haldane, 1927), and this variation was viewed as an imperfection or "genetic load" on the population (e.g. Muller, 1950).

Initially, there was little discrepancy between "classical" theoretical predictions and empirical observations of the genetic structure of populations. This initial agreement between theory and observation was due to the fact that only visible mutations could be observed by early population geneticists and these were almost always deleterious. Thus, empirical research was capable of focusing on only a small subset or portion of the total genetic composition of a population. Furthermore, it was precisely this class of variation (i.e. deleterious) that one would expect to be under the influence of strong natural selection, and therefore, to closely fit the homogeneous population structure predicted by "classical" theory.

It was not until the discovery of gene arrangements in Drosophila pseudoobscura (Sturtevant, 1926), and other sibling species, that population geneticists had a different and more "natural" class of variation to study. Dobzhansky (reviewed 1970) showed that the genetic structure of D. pseudoobscura populations, as measured by gene arrangements, was inconsistent with the "classical" theory since almost all natural populations were found to be polymorphic for various gene arrangements. Subsequent population cage experiments indicated that natural selection could play a significant role in maintaining variation for gene arrangements in natural populations of D. pseudoobscura (e.g. Dobzhansky, 1947, 1948, 1956; Dobzhansky and Levene, 1948; Dobzhansky and Pavlovsky, 1953).

Based on these observations, a new school of thought now known as the "balance" theory, was proposed by Dobzhansky (reviewed by Dobzhansky, 1955). In contrast to the "classical" theory, the "balance" theory emphasized that natural selection acted to

maintain genetic variation in a population. Therefore, individuals in a population were considered to be genetically dissimilar (i.e. often heterozygous for different alleles) and consequently populations were heterogeneous and contained large amounts of genetic variation.

One of the underlying assumptions of the "balance" theory was that fitness values or selection coefficients were not constant but varied spatially or temporally. Mather (1953, 1955) called this mode of selection "disruptive" while Dobzhansky (1970) used the term "diversifying" selection. Theoretical investigations of this phenomenon have been conducted (reviewed by Christensen and Feldman, 1975; Felsenstein, 1976; Hedrick, Ginevan, and Ewing, 1976; Hedrick, 1986). Other types of natural selection incorporating variable fitness values as a function of frequency, density, and sex have also been investigated. The basic conclusion of these theoretical investigations is that variable selection coefficients can potentially maintain genetic variation in a population. That is, the conditions for a stable polymorphism are generally less restrictive for cases involving variable selection coefficients than for models incorporating constant fitness values.

With the advent of electrophoresis and its application to population genetics, it became possible to obtain accurate estimates of genetic variation for a much larger portion of the genome. Most estimates indicated that natural populations contain large amounts of genetic variation, much greater than previously anticipated (e.g. Harris, 1966; Lewontin and Hubby, 1966). The average proportion of polymorphic loci within a species was found to be approximately 30%, while the average heterozygosity of an individual was around 10% (reviewed by Nevo 1978; Nevo, Beiles, and Ben - Shlomo, 1984). Proponents of the "balance" theory took these findings as prima facie evidence for their position. In order to maintain the integrity of the "classical" theory, supporters of this position advanced what has become to be known as the "neoclassical" or "neutralist" hypothesis (reviewed by Kimura and

Ohta, 1971; for an historical review see Provine, 1971; Lewontin, 1974). This position claimed that the variation revealed through electrophoretic techniques was irrelevant to the organism and produced no differences in fitness. The presence of allozyme variation in a population was the result of the interaction of random genetic drift, mutation, and gene flow, but not natural selection. Thus, the observed allozyme variation was considered to be neutral with respect to fitness, however, adaptive evolution did occur at other loci through the action of natural selection which was still viewed as a "purifying" force. The "selectionist - neutralist" debate (reviewed by Lewontin, 1974) has stimulated a tremendous amount of theoretical and experimental research concerning the importance of natural selection in determining the amounts and patterns of allozyme variation in natural populations. However, at the present time, there is no clear consensus on which of the two positions is essentially correct.

As with the single locus developments discussed above, the field of quantitative genetics has progressed in a similar manner. Important descriptive parameters such as phenotypic, genotypic, and environmental variances and their derived components were developed very early in the history of the field (Fisher, 1918). The theoretical foundation for quantitative genetics was developed concurrently with single locus theory, since most polygenic models are multi-locus extensions of this basic theory (Fisher, 1918; Wright, 1921; Haldane, 1932). Theoretical considerations again led to the prediction that individuals within a population should be genetically similar and that populations should exhibit little genetic variation for polygenic characters closely related to fitness (e.g. Fisher's Fundamental Theorem of Natural Selection). Also, as with the single locus case, there was an initial agreement between theoretical predictions and empirical observations. In this case, the cause of the agreement was based on the type of population studied by quantitative geneticists. Classical population geneticists have generally concentrated on natural populations, or laboratory populations sampled from nature and maintained to

preserve their natural genetic composition. In contrast, quantitative geneticists have focused on applied plant and animal breeding techniques, and therefore, have utilized domesticated populations subjected to intense artificial selection and/or inbreeding, or laboratory populations descended from a small number of founders. These types of populations are expected to contain small amounts of genetic variation and, therefore, match the genetic structure predicted from theoretical considerations.

Subsequent quantitative genetic studies of laboratory populations sampled from nature and maintained to preserve their genetic variability have revealed an entirely different pattern. Many of these populations have been found to contain relatively large amounts of quantitative genetic variation for a wide variety of characteristics (reviewed by Istock, 1983, 1984; Dingle, 1984). Thus, though occurring later, the same discrepancy between theoretical predictions and empirical observations that developed with respect to single locus cases, also occurred in the field of quantitative genetics.

At this point, the "classical" hypothesis becomes untenable, since many of the quantitative characters analyzed are generally considered to be closely related to fitness, and therefore, should be under the influence of natural selection. As a result, it is unreasonable to claim that this genetic variation is neutral with respect to fitness, as was done for the case involving allozyme variation. To date the "classical" school has not attempted to account for the large amounts of quantitative genetic variation found for fitness components in several populations.

In order to account for the large amounts of quantitative genetic variation found to exist in populations, theoretical models incorporating variable selection coefficients as a function of environmental heterogeneity have been developed as in the single locus case (Bulmer, 1971; Slatkin, 1978; Via and Lande, 1985). Again, it is possible to show theoretically that variable fitness

values can potentially maintain polygenic variation in a population. Experimental tests of this hypothesis have only recently been conducted. MacKay (1980, 1981) reported a positive association between environmental heterogeneity and estimates of polygenic variation in experimental populations of D. melanogaster subjected to either constant or variable environments. In contrast, Zirkle and Riddle (1983) and Riddle, Dawson, and Zirkle (1986) found no evidence that polygenic variation for fitness traits in populations of flour beetles (Tribolium castaneum and T. confusum) was maintained by environmental heterogeneity. Thus, unequivocal evidence that a major portion of the observed polygenic variation is maintained by variable selection coefficients is still lacking.

A number of evolutionary models have been developed that examine the balance between mutation and weak stabilizing selection as a mechanism capable of maintaining quantitative genetic variation in a population (Latter, 1960; Kimura, 1965; Bulmer, 1972; Lande, 1975; Turelli, 1984). However, mutation rates must be very high and selection very weak in order to account for the levels of polygenic variation found in populations. Unfortunately, the difficulties involved in estimating parameters with such small values makes it unlikely that this issue will be experimentally resolved (Turelli, 1984).

Antagonistic pleiotropy, expressed as negative genetic correlations between characters, has also been proposed as a mechanism capable of maintaining quantitative genetic variation in a population (e.g. Caspari, 1952; Lerner, 1954; Dickerson, 1955; Robertson, 1955; Wallace, 1959, Wright, 1977; Rose, 1982, 1983, 1984a, 1984b, 1984c; Dawson and Riddle, 1983; Istock, 1983, 1984; Dingle, 1984; Riddle, Dawson, and Zirkle, 1986). Theoretical models of the evolution of characters subjected to pleiotropy have been developed (Bulmer, 1973; Charlesworth, 1980; Lande, 1980, 1982a, 1982b; Rose, 1982, 1983; Slatkin, 1982; Gillespie, 1984). Specifically, with respect to the maintenance of variation, Rose (1982, 1983) has shown theoretically that large amounts of

additive genetic variation for individual fitness characters can be maintained when there are negative genetic correlations among the various components. In particular, recessive deleterious gene action tends to generate stable polymorphisms under a variety of conditions.

Most of the experimental evidence for negative genetic correlations between characters comes from studies involving domesticated animals subjected to artificial selection for a single characteristic (reviewed by Falconer, 1981). Indirect evidence for negative genetic correlations among fitness components in laboratory populations of Drosophila has been reported (Gowen and Johnson,1946; Hiraizumi, 1961). Dawson (1966), Rose and Charlesworth (1981b), Rose (1984), and Luckinbill et al. (1984) observed correlated responses to artificial selection on fitness components that appeared to be the result of negative genetic correlations. Direct estimates of negative genetic correlations between early fecundity and longevity have been reported by Rose and Charlesworth (1981a) in D. melanogaster.

In contrast, Temin (1966), Giesel (1979), Giesel and Zettler (1980) and Simmons, Preston and Engels (1980), found positive genetic correlations among fitness components, while Mukai and Yamazaki (1971) reported no evidence for negative genetic correlations. However, these studies used highly inbred strains, and therefore, positive genetic correlations are expected since the value of fitness components in these cases depends greatly on the number and severity of recessive deleterious alleles that have been fixed as a result of inbreeding (Rose, 1981a, 1984b). Bell (1984a, 1984b), Stearns (1983), and Murphy et al. (1983) also reported positive genetic correlations among fitness components in non - inbred laboratory populations. However, Service and Rose (1985) have criticized these experiments because they involve assays of individuals subjected to novel environments which can produce artificial genetic correlations that tend to be positive.

Thus, it appears that differential success has been achieved with respect to the two fundamental tasks of population genetics. In many ways, the field of population genetics is "descriptive" science rather than a "predictive" one. Great strides have been made in providing a genetic description of the genetic composition of populations, particularly with respect to electrophoretic variation, although descriptions of polygenic variation are becoming more commonplace. However, the second task of providing an explanation for the genetic composition of populations has proved more formidable. In reality, theoretical explanations for the genetic composition of populations abound and far exceed the empirical knowledge base of the field. Experimental examination of this immense body of theory is sorely needed, and it is with this realization that the present investigation was undertaken.

II. Organism

Flour beetles of the tenebrionid family, Tribolium, have a world-wide distribution. There are 26 species in the genus (Hinton, 1948), but almost all genetic and ecological studies have utilized T. castaneum or T. confusum.

Tribolium can be reared on a variety of different flour types, thus, it is easy to vary the environment by altering the culture medium. In addition, all life-history stages can easily be recovered by passing the culture medium through a sieve of the appropriate size. Also, males and females can be easily sexed and separated during the pupal stage without the use of ether. These simple techniques combined with the short generation time of Tribolium (approximately 4-5 weeks under our laboratory conditions) make it an ideal organism for ecological and genetic studies. King and Dawson (1972) have presented a detailed review of Tribolium population biology.

The life cycle of T. confusum has been described in detail by several individuals (e.g. Good, 1933; Park, 1934) and will only be briefly discussed. Males have been reported to be fertile within 24 hours after eclosion (Erdman, 1964). Females are capable of being fertilized within 20 hours after eclosion and begin to deposit fertile eggs approximately 100 hours later (Dawson, 1964). At the time of peak fecundity, approximately 2 weeks after eclosion, females can produce 10 to 20 eggs per day under favorable culture conditions in our laboratory (personal observations). The development time of eggs is approximately 4-6 days at 29 C (Young, 1970). The number of larval instars is generally seven or eight, but can vary from as few as six to as many as 12 (Good, 1933). Depending on the culture conditions and the genetic strain,

egg-to-pupa development time is approximately 20-35 days (Young, 1970), however, the length of the pupal period is relatively constant at about 5 or 6 days. Under favorable culture conditions in our laboratory, mean life-span is approximately 5 to 6 months (personal observations).

CHAPTER 1

Introduction

A primary question for population geneticists is "Why is there so much genetic variation?" Recent interest in this problem has focused primarily on the need to provide an explanation for the large amounts of genetic variation revealed through electrophoretic techniques (reviewed by Christensen and Feldman, 1975; Felsenstein, 1976; Hedrick, Ginevan, and Ewing, 1976; Hedrick, 1986). In contrast, consideration of the mechanisms maintaining polygenic variation in populations has not received the same attention. This seems rather odd since quantitative genetic studies have revealed that populations contain relatively large amounts of polygenic variation for a wide variety of characteristics (reviewed by Istock, 1983, 1984; Dingle, 1984).

Theoretical models incorporating variable selection coefficients as a function of environmental heterogeneity have shown that spatial and temporal variation in the environment can potentially maintain polygenic variation in a population (Bulmer, 1971; Slatkin, 1978; Via and Lande, 1985). However, unequivocal experimental evidence supporting this hypothesis is lacking. MacKay (1980, 1981) reported a positive association between environmental heterogeneity and estimates of polygenic variation in laboratory populations of D. melanogaster. In contrast, a series of experiments from this laboratory (Dawson and Riddle, 1983; Zirkle and Riddle, 1983; Riddle, Dawson, and Zirkle, 1986) found no evidence that polygenic variation for fitness characters in populations of flour beetles (Tribolium castaneum and T. confusum) was maintained by environmental heterogeneity.

Several evolutionary models have been developed that examine the balance between mutation and weak stabilizing selection as a mechanism capable of maintaining quantitative genetic variation in a population (Latter, 1960; Kimura, 1965; Bulmer, 1972; Lande, 1975; Turelli, 1984). However, mutation rates must be very high and selection very weak in order to maintain the levels of polygenic variation found in populations. The difficulties involved in obtaining reliable estimates of these parameters makes it unlikely that this issue can be experimentally resolved (Turelli, 1984).

Antagonistic pleiotropy, expressed as negative genetic correlations between characters, has also been proposed as a mechanism capable of maintaining polygenic variation in a population (e.g. Caspari, 1952; Lerner, 1954; Dickerson, 1955; Robertson, 1955; Bulmer, 1973; Wright, 1977; Charlesworth, 1980; Lande, 1980, 1982a, 1982b; Rose, 1982, 1983, 1984a, 1984b, 1984c, 1985; Slatkin, 1982; Dawson and Riddle, 1983; Istock, 1983, 1984; Dingle, 1984; Gillespie, 1984; Riddle, Dawson, and Zirkle, 1986). Specifically, with respect to the maintenance of variation, Rose (1982, 1983) has shown theoretically that large amounts of additive genetic variation for individual fitness components can be maintained when there are negative genetic correlations among the various characters.

Indirect evidence for negative genetic correlations among fitness components in laboratory populations of Drosophila has been reported (Gowen and Johnson,1946; Hiraizumi, 1961). Other investigators have observed correlated responses to artificial selection on fitness components that appeared to be the result of negative genetic correlations (Dawson, 1966; Rose and Charlesworth, 1981b; Rose,1984; Luckinbill et al., 1984). Direct estimates of negative genetic correlations between early fecundity and longevity have been reported by Rose and Charlesworth (1981a) in D. melanogaster.

In contrast, much of the genetic variance for fitness characters could be due to deleterious recessive alleles maintained at low frequencies by mutation balancing selection. In this case, since deleterious alleles would be likely to have adverse effects on several components of fitness, the genetic correlations among these characters would be positive. Temin (1966), Giesel (1979), Giesel and Zettler (1980) and Simmons, Preston and Engels (1980), found positive genetic correlations among fitness components, while Mukai and Yamazaki (1971) reported no evidence for negative genetic correlations. However, these studies used highly inbred strains, and therefore, positive genetic correlations are expected since the value of fitness components in these cases depends greatly on the number and severity of recessive deleterious alleles that have been fixed as a result of inbreeding (Rose and Charlesworth, 1981a; Rose, 1984b). Bell (1984a, 1984b), Stearns (1983), and Murphy et al. (1983) also reported positive genetic correlations among fitness components in non - inbred laboratory populations. However, Service and Rose (1985) have criticized these experiments because they involved assays of individuals subjected to novel environments which can produce artificial genetic correlations that tend to be positive.

Clearly, it is important to quantify the amount of genetic variation for fitness components in order to predict the course of evolution for such characters under the influence of natural selection. However, if antagonistic pleiotropy is common, then it also becomes necessary to estimate genetic correlations among fitness components in order to make accurate evolutionary predictions. To provide this type of information, I have obtained estimates of genetic variation and covariation for a series of fitness components in two populations of flour beetles (T. confusum). The objectives of the present study are three-fold. First, obtain estimates of genetic variation for a series of fitness components in populations of T. confusum. A corollary of this objective involves an attempt to partition the total genetic

variance of fitness characters into additive and nonadditive components. Second, obtain estimates of genetic covariation among fitness components in populations of I. confusum. More specifically, what are the signs and magnitudes of the genetic correlations among characters closely related to fitness? Third, investigate the relationship between genetic variation and covariation for fitness components in populations of I. confusum. That is, does antagonistic pleiotropy expressed as negative genetic correlations among fitness components, play an important role by setting constraints on the evolutionary process such that genetic variation for individual traits is maintained?

Materials and Methods

Experimental populations: The present study utilized two populations (hereafter referred to as populations 1-1 and 1-2) of the "confused" flour beetle, T. confusum. The populations were initiated in 1969 from random samples of 500 adults taken from a heterogeneous stock, the Oregon Synthetic Population (Ore). The Ore population was synthesized from systematic crosses using individuals obtained from both laboratory and wild sources.

The samples taken from the Ore population were randomly assigned to one of eight different flour types (for further details, see Dawson and Riddle, 1983; Riddle, Dawson and Zirkle, 1986). Each flour treatment was replicated twice. For the present study, I used the two replicate populations subjected to the corn flour treatment. Both populations were maintained through time by transferring immature stages (eggs, larvae, and pupae) to fresh corn flour at eight - week intervals. The populations were maintained under identical conditions of temperature and humidity. When sampled for the present study, the two populations had been subjected to over 70 transfer cycles.

Experimental procedures: Information concerning the following characters was obtained: (1) fecundity, measured as the number of eggs produced by a single female over a 96 hr collection period; (2) 28 - day survival, measured as the proportion of individuals surviving from a given number of eggs, 28 days from the first day of the egg collection period; (3) larval weight, measured as the mean weight of ten randomly-selected larvae, 28 days from the first day of the egg collection period; (4) 35 - day survival, measured as for (2) but at 35 days; (5) development time, measured as the proportion of pupae present from the number of surviving

individuals, 35 days from the first day of the egg collection period; and (6) female productivity, a composite character of individual females, measured as the product of fecundity, 28 - day survival, and larval weight. All portions of the experiments were conducted in an incubator in total darkness at 29° C and 60% relative humidity. Both populations were maintained on corn flour that was prepared by sifting through a #5 mesh silk bolting cloth for general use and a #9 mesh silk bolting cloth for use as an egg collection medium.

In order to expand the populations, 200 adults from each of the populations were randomly collected from several of the eight - week transfer cycles. These adults were placed in jars containing 30 g of fresh flour for a four - day egg collection period. Approximately 35 days later, pupae were separated by sex, and stored at low density in 6 - dram shell vials containing 6 g of flour. One week after eclosion, ten males from each population were individually mated to four females for a period of seven days in 6 - dram shell vials containing 3 g of fresh flour. Fertilized females were then placed individually in 6 - dram shell vials containing 6 g of fresh flour for a 96 - hr egg collection period. An excess of medium was used in order to reduce egg cannibalism by the adult female. Females were then removed from the flour and the number of eggs deposited was scored. At this time, 25 eggs were randomly selected from each parental female and placed in 6 - dram shell vials containing 6 g of fresh flour. Twenty - four days later (28 days from the first day of the egg collection period), the number of surviving larvae from each full - sib group was determined. On the same day, ten randomly selected larvae from each full - sib family were weighed in a group to the nearest 0.01 mg on a micro - analytical balance. All surviving larvae were then returned to the original vials. One week later (35 days from the first day of the egg collection period), the vials were inspected again, and the number of surviving larvae and pupae present was scored. At this time, pupae were by sex, and stored at low density in 6 - dram shell vials containing 6 g of fresh flour. Four females

were randomly selected from each full - sib group and scored for fecundity following the same experimental procedures used for the parental generation. However, in this case, pedigreed females were randomly mated to single males from the same population and generation. This mating scheme produced a series of first cousin and half - first cousin families with minimal inbreeding. The entire experimental design was repeated 32 times (blocks) over a two - year period.

In order to provide estimates of female productivity among the offspring, some of the first - cousin families were scored for 28 - day survival and larval weight (blocks 1-20) following the procedures used in the previous generation.

Statistical procedures: An analysis of variance model (SAS v. 5,18; Proc GLM) was utilized to test for differences between the means of the two populations for all of the measured characteristics. Table 1 contains a sample analysis of variance model with expected mean squares. The same model was used for data collected from both generations of the study. The model included the main effects of blocks and populations and the two - way interaction of blocks and populations. Blocks were treated as a random effect with populations as a fixed effect. The main effect of blocks and the two-way interaction were tested over the mean square error while the effect of populations was tested over the interaction term.

Since the data were unbalanced for all measured characteristics, random nested analysis of variance models (SAS v. 5.18; Proc VARCOMP) with unequal subclass numbers were used to estimate variance components. Negative variance component estimates were set equal to zero in order to estimate genetic parameters. F-tests from the analysis of variance models were used to determine statistical significance of the genetic parameters.

The following model based on a half - and full - sib design (HS/FS) was used to estimate the heritability of fecundity and female productivity. Subscripts are applicable to a balanced design. A sample analysis of variance table with expected mean squares and genetic expectations is presented in Table 2.

$$Y_{ijkl} = \mu + b_i + s_{j(i)} + d_{k(ij)} + e_{l(ijk)}$$

where Y_{ijkl} = the record of the i^{th} progeny of the k^{th} dam mated to the j^{th} sire in the i^{th} block

μ = grand mean

b_i = the effect of the i^{th} block

$s_{j(i)}$ = the effect of the j^{th} sire in the i^{th} block

$d_{k(ij)}$ = the effect of the k^{th} dam mated to the j^{th} sire in the i^{th} block

$e_{l(ijk)}$ = random error attributable to variation among individuals.

Heritability estimates were obtained from four times the sire, four times the dam, and twice the sum of the sire and dam components of variance as ratios of the total phenotypic variance. The sire component of variance contains $^1/_4$ of the additive genetic variance and $^1/_{16}$ of the additive x additive genetic variance plus higher order interaction terms. The dam component of variance estimates $^1/_4$ of the additive genetic variance, $^1/_4$ of the dominance genetic variance, various fractions of the interaction variance, and variance due to maternal effects and common environment. The sire plus dam components of variance contain $^1/_2$ of the additive genetic variance, $^1/_4$ of the dominance genetic variance, various fractions of the interaction variance, and variance due to maternal effects and common environment. Estimates of nonadditive variance were obtained from 4 times the difference between the dam component of variance and the sire component of variance.

Approximate standard errors of the heritability estimates were first calculated using Searle (1971). These estimates were compared to an approximation suggested by Dickerson (1969). Since the two methods produced similar results, the simpler method developed by Dickerson was used.

$$\text{var}\ (h^2_s) = (16\ \text{var}\ V_s)/(V_s + V_d + V_w)^2$$
$$\text{var}\ (h^2_d) = (16\ \text{var}\ V_d)/(V_s + V_d + V_w)^2$$
$$\text{var}\ (h^2_{s+d}) = 4\ [\text{var}\ V_s + V_d + 2\ \text{cov}(V_s, V_d)]/(V_s + V_d + V_w)^2.$$

The following model based on a half - sib design (HS) was used to estimate the heritability of 28 - day survival, 35 - day survival, larval weight, and development time. Subscripts are applicable to a balanced design. A sample analysis of variance table with expected mean squares and genetic expectations is given in Table 3.

$$Y_{ijk} = \mu + b_i + s_{j(i)} + d_{k(ij)}$$

where Y_{ijk} = the mean of the progeny of the k^{th} dam mated to the j^{th} sire in the i^{th} block

μ = grand mean
b_i = the effect of the i^{th} block
$s_{j(i)}$ = the effect of the j^{th} sire in the i^{th} block
$d_{k(ij)}$ = the effect of the k^{th} dam mated to the j^{th} sire in the i^{th} block.

Heritability estimates were obtained from four times the sire component of variance as a ratio of the total phenotypic variance. The sire component of variance estimates $^1/_4$ of the additive genetic variance and $^1/_{16}$ of the additive x additive genetic variance plus higher order additive x additive interaction terms.

Approximate standard errors of the heritability estimates for an unbalanced design were obtained as follows (Swiger et al., 1964).

$$S.E\ (h^2_s) = 4\sqrt{2}\ (n_t - 1)\ (1-t)^2\ [1 + (k_1 - 1)\ t]^2 / k_1^2\ (n_t - s)\ (s - 1)$$

where
- s = number of sires
- n_t = total number of progeny
- k_1 = coefficient of expected mean square
- t = $V_s/V_s + V_w$.

The following intra - block and intra - sire regression model of offspring mean on dam (D/O) was also used to estimate the heritability of fecundity and female productivity (Turner and Young, 1969).

$$Y_{ijk} - b_i - s_{ij} = \mu + \beta\ (X_{ijk} - X) + e_{ijk}$$

where
- Y_{ijk} = the mean of the progeny of the k^{th} dam mated to the j^{th} sire in the i^{th} block
- b_i = the effect of the i^{th} block
- s_{ij} = the effect of the j^{th} sire in the i^{th} block
- μ = grand mean
- β = the regression coefficient of Y on X
- X_{ijk} = the record of the k^{th} dam mated to the j^{th} sire in the i^{th} block
- X = phenotypic mean
- e_{ijk} = deviation of the offspring means.

Heritability estimates were obtained from twice the offspring - dam regression coefficients. The covariance of offspring and dam contains $^1/_2$ of the additive genetic variance and $^1/_4$ of the additive x additive genetic variance plus higher order additive x additive interaction terms and $^1/_2$ of the variance due to maternal

effects. Standard errors of these estimates were obtained from twice the standard error of the regression coefficient corrected for the block and sire differences removed (Turner and Young, 1969). Heritability estimates with a lower confidence limit greater than zero were considered to be statistically significant.

Analogous methods were used to estimate the genetic correlations among the various traits. However, in these cases, nested analysis of covariance models were utilized to estimate the appropriate components of covariance. Standard errors of the various correlation estimates are summarized in Scheinberg (1966), Hammond and Nicholas (1972), and Norton (1974). Parametric confidence intervals for the genetic correlation estimates were calculated as ± 1.96 times the standard error of the estimate. Genetic correlation estimates with confidence limits not overlapping zero were interpreted as statistically significant.

Results

The phenotypic distributions of sire and dam means appeared non - normal for all characteristics except fecundity in both populations. Variances also appeared unequal across blocks for all characteristics except fecundity in both populations. In order to improve the symmetry of the distributions and stabilize variances, an arcsin square root transformation was used for the characteristics measured by proportions, while a base 10 logarithmic transformation was used for larval weight and female productivity. Therefore, all characteristics were transformed prior to analysis, except for fecundity which was analyzed in the original scale.

Phenotypic Parameters

Table 4 presents the means and standard errors for all of the characteristics in each population by generation. The analysis of variance results are given in Table 5. The main effect of blocks is significant for all characteristics in both generations ($P < 0.0001$ for most characteristics). The block x population interaction term is not significant for any of the characteristics in the first generation, however, this term is significant for all characters in the second generation ($P < 0.0001$ for most characteristics) except fecundity.

The two populations differ significantly with respect to 28 - day survival ($P < 0.0001$) in both generations and 35 - day survival in the first generation ($P < 0.0001$). Population 1-2 has a 6% higher survival rate than population 1-1 at 28 days in both generations and an 11% higher rate of survival at 35 days. Survival rates are substantially higher in both populations at 28 days than at 35 days. At day 28, all individuals are late instar larvae, while at day 35

more than half of the surviving individuals have pupated. The substantially lower survival rate during this one - week period may be due to cannibalism of early pupating individuals by larvae (Dawson, 1975).

The two populations also differ significantly with respect to mean larval weight in both generations ($P < 0.0001$). A random sample of ten larvae from population 1-1 is approximately 2 mg heavier than a comparable sample from population 1-2 in the first generation and 1.3 mg in the second generation.

The main effect of populations is significant for fecundity in both the parental and offspring females ($P < 0.0001$). Parental females from population 1-1 produce approximately 5 more eggs and offspring females 2 more eggs during the 96 - hr collection period than individuals from population 1-2.

There is no evidence for differences between the two populations for development time ($P = 0.1379$). Approximately 60% of the surviving individuals pupate by day 35 in both populations.

The populations also differ with respect to productivity for both the parental ($P < 0.0001$) and offspring females ($P = 0.0183$). Parental females from population 1-1 produce approximately 16% or 10 mg more biomass among their offspring than population 1-2 females while offspring females from population 1-1 produce approximately 4% or 2.5 mg more biomass.

Since both populations were tested in a common environment, these results indicate that the two populations are genetically differentiated with respect to all of the measured characteristics except development time. In summary, population 1-2 individuals exhibit a higher survival rate (both survival measurements), lower fecundity, and a smaller larval weight than individuals from population 1-1. The differences among these characteristics also

contribute to the result that population 1-1 females produce more biomass among their offspring than females from population 1-2.

Variance Component Analysis

Half - Sib Analysis: Estimates of heritability from both populations with standard errors are presented in Table 6. The analysis of variance results from which these estimates are derived are presented in the Tables 10-17.

Population 1-1 exhibits heritability estimates that are significantly greater than zero for 28 - day survival and larval weight ($P < 0.05$). These results demonstrate the presence of genetic variation for both of these characteristics in population 1-1. Since these estimates are obtained from the sire component, the variation is more than likely additive genetic variation. Heritability of 28 - day survival is 0.33 ± 0.16, while the heritability of larval weight is 0.36 ± 0.15. Heritability estimates are not significantly different than zero for either 35 - day survival or development time in population 1-1.

Estimates of heritability are not significantly different from zero for any of the characteristics measured in population 1-2. In fact, all sire variance component estimates are negative except for mean larval weight. A comparison of heritability estimates from the HS design indicates that there is no evidence for differences between the two populations for any of the characteristics.

Half - and Full - Sib Analysis: Estimates of heritability from both populations with standard errors for fecundity and female productivity are presented in Table 7. The heritability estimates are partitioned into sire, dam, and sire plus dam effects. The analysis of variance results from which these estimates are derived are given in Tables 18-21.

All estimates of heritability from population 1-1 are significantly greater than zero for fecundity ($P < 0.05$), demonstrating the presence of genetic variation for this fitness component. The estimate from the sire component is 0.32 ± 0.09 while the estimates from the dam (1.06 ± 0.10) and sire plus dam (0.69 ± 0.05) are significantly larger. For population 1-2, the heritability estimate from the sire component is not significantly different from zero, while the estimates from the dam (0.56 ± 0.09) and sire plus dam (0.28 ± 0.03) components are significant ($P < 0.05$). The two populations are significantly different ($P < 0.05$) with respect to all three heritability estimates.

All the estimates of heritability from population 1-1 are significantly greater than zero for female productivity ($P < 0.05$), indicating the presence of genetic variation for this composite character. The estimate from the sire component is 0.28 ± 0.12 while the estimates from the dam (1.45 ± 0.15) and sire plus dam (0.86 ± 0.07) are significantly larger. For population 1-2, the estimate of heritability from the sire component is not significantly different from zero, while the estimates from the dam (1.19 ± 0.14) and sire plus dam (0.60 ± 0.05) components are both greater than zero ($P < 0.05$). The two populations are significantly different ($P < 0.05$) based on a comparison of heritability estimates from the sire plus dam components.

Dam - Offspring Analysis: Estimates of heritability from both populations with standard errors for fecundity and female productivity are presented in Table 8. Since the effects of blocks and sires were not significant, these terms were removed from the model and an overall estimate of heritability was obtained from the regression of offspring mean on the value of the dam. Tables 22-25 contain the results of the regression analysis.

The heritability estimates for fecundity are significantly greater than zero ($P < 0.05$) in both population 1-1 (0.26 ± 0.09) and 1-2 (0.30 ± 0.10). The estimates for female productivity are also

significantly different than zero ($P < 0.05$) for both populations, and similar to the estimates from the sire plus dam components of the half - and full - sib design. The two populations are not significantly different based on a comparison of heritability estimates for either fecundity or female productivity in this analysis.

Covariance Component Analysis

Half - Sib Analysis: Estimates of genetic correlations for population 1-1 with standard errors are presented in Table 9 for all pairs of characteristics from the HS design. The analysis of covariance results from which these estimates are derived are given in Tables 26-45.

For population 1-1, several of the genetic correlation estimates are near zero. However, the correlations involving 28 - day survival with 35 - day survival and fecundity are significantly different from zero ($P < 0.05$). As expected, the genetic correlation between 28 - and 35 - day survival is large and positive ($+0.70 \pm 0.35$). In contrast, the correlation between 28 - day survival and fecundity is large and negative (-0.54 ± 0.27). This result indicates that a significant portion of the genetic covariation between these two characters is due to alleles affecting the traits in opposite directions. The same relationship was observed for 35 - day survival and fecundity, though the correlation is not significant (-0.69 ± 0.48). Several of the genetic correlation estimates between the individual characteristics and female productivity are positive. However, this result is not unexpected since female productivity is a composite character, and therefore, correlated with the individual characters. For population 1-2, the genetic correlation estimates are mathematically undefined since the sire variance component estimates are negative for all characteristics, except larval weight. Therefore, a comparison of the genetic covariance structure between populations is not possible.

Discussion

The present study was conducted to obtain estimates of genetic variation and covariation for a series of fitness components in two "replicate" populations of flour beetles. Replication in this case means that the two experimental populations were derived from large, random samples of individuals obtained from the same base population and that the populations have experienced identical extrinsic environmental conditions since their inception. By utilizing "replicate" populations, it is not only possible to estimate genetic parameters within each population, but also to compare the quantitative genetic structure of the two populations. This type of comparison permits one to assess the amount of divergence that has occurred between two populations subjected to the same extrinsic environment and, therefore, to some degree, similar evolutionary forces.

A comparison of mean values indicates that the populations are genetically differentiated with respect to fecundity, larval weight, female productivity, and both measurements of survival. The pattern of differentiation indicates that individuals from population 1-2 are smaller with higher survival but lower fecundity than individuals from population 1-1. This pattern also contributes to the greater female productivity observed for population 1-1. In contrast, there is no evidence for differences between the populations for development time.

Large differences between the populations for fecundity and weight measurements obtained at various stages of the life cycle have been previously reported (Dawson and Riddle, 1983; Riddle, Dawson, and Zirkle, 1986). Krause and Bell (1972) reported significant differences between two unrelated populations of T. castaneum for larval survival and female productivity

measurements obtained under conditions different from the present study. Genetic differences between individuals and populations are known for development time in Tribolium (e.g. Dawson and Riddle, 1983). However, it should be noted that the method of quantifying development time in the present study was substantially different from the procedures used in other studies. In the present study, development time was measured on a family basis as the percent of individuals pupating by a particular day, rather than actually scoring individuals. On the other hand, the fact that no differences were observed for development time could be due to the fact that this character is known to be under the influence of strong stabilizing selection in some laboratory populations of Tribolium (Dawson, 1975).

One of the more interesting results to emerge from recent quantitative genetic studies is the presence of relatively large amounts of additive genetic variance for fitness characters in natural populations (reviewed by Istock, 1983, 1984; Dingle, 1984). Several of the estimates obtained from the present study confirm this observation, particularly those involving population 1-1. Estimates of heritability from the HS design indicate that population 1-1 contains significant amounts of genetic variation for 28 - day survival and larval weight. Heritability estimates from the sire component of the HS/FS design also indicate that population 1-1 contains significant amounts of genetic variation for fecundity and female productivity. Since these estimates were obtained from the sire component, the variation is most likely additive genetic variation. In contrast, there is no evidence for the presence of genetic variation in population 1-2 for any of the characteristics measured from the sire component of the HS and HS/FS designs. Estimates of heritability obtained from the D/O design which include additive genetic variance in addition to other sources of variation also demonstrate the presence of significant variation for fecundity and female productivity in both populations.

The fact that most of the variance component estimates from the sire component are negative for population 1-2 deserves attention. The occurrence of negative estimates has been reported several times in the literature (e.g. ElRouby and Penny, 1967; Robinson, Comstock, and Harvey, 1955; Sentz, 1971; Williams, Penny, and Sprague, 1965). Common explanations for negative variance component estimates involve sampling error (Searle, 1971), inadequate genetic model, assortative mating (Lindsey, Lonnquist, and Gardner, 1962; Hallauer and Miranda, 1981), or competition effects among individuals (Falconer, 1981). Bridges and Knapp (1987) reported high probabilities of obtaining negative variance component estimates under a wide variety of experimental conditions, particularly for estimates from the dam component of variance from nested mating designs. Interestingly, the estimates from the dam component are large and positive in the present study while the sire variance component estimates are generally negative for population 1-2.

Unfortunately, the cause of the negative estimates in the present study is unknown at this time. For the purpose of discussion, the heritability estimates will be interpreted as equal to zero. The negative variance component estimates also make it difficult to interpret the genetic covariance structure of population 1-2. The genetic correlation estimates from this population are undefined for the purpose of discussion.

Very little is known about how the total genetic variance for fitness characters is partitioned into additive and nonadditive components. Robertson (1955) presented arguments for expecting the total genetic variance of characters closely related to fitness to be composed primarily of nonadditive components. The few cases where partitioning of the total genetic variance for fitness characters has been possible seem to confirm this prediction (e.g. Dawson, 1965; Via, 1984). The response of fitness characters to inbreeding also suggests that nonadditive effects comprise a significant portion of the total genetic variance (reviewed by

Falconer, 1981). A comparison of heritability estimates from the different sources indicates the presence of nonadditive variance for fecundity and female productivity in both populations. For both populations, the heritability estimates for fecundity and female productivity from the dam and sire plus dam components of variance are significantly larger than the estimate from the sire component. This comparison suggests the importance of nonadditive genetic variance and/or common environment for these characters. The contribution of nonadditive variance to total phenotypic variance is approximately 70% for fecundity and 56% for female productivity in population 1-1, while the corresponding estimates for population 1-2 are 81% and 85%. A comparison of the heritability estimates from the sire component of variance and the D/O analysis suggests that maternal effects may not be an important source of variance for fecundity, but a large component of variance for female productivity in both populations.

Since fitness is a compound character, natural selection must act simultaneously on several traits. Dickerson (1954) was the first to show that simultaneous selection for two characters can eventually produce a negative genetic correlation between the selected traits. This result is due to the fact that pleiotropic alleles which affect both characters in the same direction will be rapidly fixed by selection, while alleles which affect the traits in opposite directions will remain unfixed. Thus, most of the genetic covariance between the two characters will be due to pleiotropic alleles at intermediate frequencies and the genetic correlation will be negative. One consequence of a negative genetic correlation is that individual characters may exhibit large amounts of genetic variation, but simultaneous selection for both characters results in no response. Specifically, with respect to the maintenance of variation, Rose (1982, 1983a, 1983b) has shown theoretically that large amounts of additive genetic variation for individual fitness characters can be maintained when there are negative genetic correlations among the various components.

In contrast, much of the genetic variance for fitness characters could be due to deleterious recessive alleles maintained at low frequencies by mutation balancing selection. Since deleterious alleles would be likely to have adverse effects on several characters, the genetic correlations among fitness components would tend to be positive (Temin,1966; Giesel, 1979; Giesel and Zettler, 1980; Simmons, Preston and Engels, 1980; Bell, 1984a; 1984b; Stearns, 1983; Murphy et al., 1983).

It is also known that gametic phase disequilibrium can produce genetic correlations among characters (Lewontin, 1974), however, given the long history of the populations in the same environment and that strong epistatic interactions are required to maintain significant disequilibrium, the genetic correlations detected in the present study will be interpreted as due to pleiotropic effects. Genetic recombination should gradually breakdown all correlations among characters due to linkage except for those maintained by the strongest of epistatic interactions.

The genetic correlation estimates from the HS design for population 1-1 confirm the presence of negative genetic correlations among fitness characters. These estimates indicate that this population contains a significant amount of negative genetic covariation for 28 - day survival and fecundity. The same relationship exists between 35 - day survival and fecundity, though the correlation is not significant. There also appears to be a relationship between the estimates of genetic variation and covariation in population 1-1. That is, significant amounts of genetic variation were detected for 28 - day survival and fecundity in addition to a significant negative genetic correlation between these characters. Simultaneous selection for these two characters would produce no response, even though both of the characters exhibit substantial amounts of genetic variation. Thus, it is possible that a portion of the genetic variation for 28 - day survival and fecundity in population 1-1 is maintained by the negative genetic correlation detected between these characters.

Since a strong negative genetic correlation was observed, these results are inconsistent with the hypothesis that most of the genetic variation for these fitness characters is the result of deleterious recessive alleles maintained at low frequencies by mutation balancing selection. In addition, the heritability estimates for many of the fitness components, particularly from population 1-1, are substantially larger than would be predicted from a balance between mutation and selection.

Genetic correlation estimates from the HS design also indicate the presence of positive genetic covariation for 28 - day survival and 35 - day survival in population 1-1. This is not surprising, since it is likely that many of the same genes affect both of these characters in the same direction, particularly since the estimates were obtained only seven days apart.

The relative independence of genetic variance for development time detected in the present study has also been observed in other insect species (reviewed by Dingle, 1984). Strong genetic correlations between development time and other fitness components would tend to "tie up" genetic variance contributing to evolutionary flexibility, particularly for colonizing species. The genetic uncoupling of development time from other fitness characters can maintain the flexibility necessary for evolutionary response to environmental uncertainty.

The major forces of evolution include natural selection, mutation, gene flow, and random genetic drift. However, very little is known about the roles and relative importance of these various forces in the overall evolutionary process. At present there are essentially two opposing views which differ in the evolutionary role ascribed to stochastic forces. The shifting balance theory of evolution, proposed by Wright (1977, 1978), emphasizes the importance of both stochastic forces and systematic forces in the evolutionary process. In contrast, other individuals (e.g. Fisher, 1930; Ford, 1964) have argued that effective population sizes are

generally large enough that stochastic forces become insignificant and, therefore, natural selection assumes a dominant role in the evolutionary process. A central feature of Wright's shifting balance theory of evolution is the assumption that many alleles contributing to overall fitness exhibit pleiotropic effects. Furthermore, the theory also assumes that many polygenic traits have intermediate optima with respect to fitness. Under these assumptions, it is possible to generate complex adaptive landscapes with multiple peaks, valleys, and saddles. In many cases the peaks will not correspond to points of equal fitness and a population may become "stranded" on a submaximal fitness peak. According to the shifting balance theory of evolution, it is during this phase that random genetic drift may assume a significant evolutionary role. Allele frequency changes as the result of random genetic drift can cause a population to shift position and come under the influence of another peak in the multiple peak adaptive landscape. At this time, natural selection occurs, carrying the population to the summit of the new adaptive peak.

Riddle, Dawson, and Zirkle (1986) presented results consistent with Wright's shifting balance theory of evolution. Significant genetic differentiation among a series of replicate populations subjected to various environmental treatments was observed that was inconsistent with a model based on stochastic or systematic forces acting alone. In our experimental design, replicate populations were established to serve as a measure of random divergence between populations subjected to the same environmental treatment. Therefore, replicate populations were treated as a random factor and differences between replicates were initially ascribed to random genetic drift. Under these conditions, the differentiation among populations subjected to different environmental treatments did not appear adaptive, and therefore, natural selection was ruled out as the dominant evolutionary force contributing to the observed differences. At the same time, correlations between population means and population size for the observed characters were not significant, suggesting

that random genetic drift was not a significant evolutionary force either. However, in a complex adaptive landscape, replicate populations may follow different trajectories in adapting to the same environmental treatments. Under these conditions, differences between replicate populations would represent the combined effects of random genetic drift and selection, and therefore, replicates should be treated as fixed effects. The problem is that we really don't know which model is correct.

The two replicate populations used in the present study were a subset of the populations utilized in the original study (see Riddle, Dawson, and Zirkle, 1986). The results from the present study confirm our earlier observations. There appears to be a substantial amount of genetic differentiation between the two populations, even though the populations have been maintained on the same flour type and experienced the same extrinsic environmental conditions. There also appears to be a relationship between genetic variation and covariation with a strong negative genetic correlation between two fitness characters more than likely subjected to natural selection. This type of genetic structure is consistent with Wright's shifting balance theory of evolution and could explain the substantial amount of genetic differentiation observed between the two replicate populations in the present study and the large differences previously detected among the entire set of experimental populations. That is, strong negative genetic correlations among fitness components can not only maintain genetic variation in individual characters, but also generate complex adaptive landscapes where stochastic forces operating in conjunction with systematic forces can produce large population differences among the various traits. Thus, the original assumption that replicate populations represent only random divergence may have been incorrect.

If antagonistic pleiotropy is common, then it becomes necessary not only to quantify the amount of genetic variation for fitness components, but also to estimate genetic correlations among

fitness characters in order to provide accurate descriptions of the genetic composition of populations. The issue becomes not simply whether fitness components are genetically based, but rather, how the genome is organized with respect to fitness characters. Any explanation of the genetic structure of populations depends on understanding both the genetic variation for fitness components, since it is this variation on which natural selection acts, and the genetic covariance structure, since this structure will impose limits on the magnitude and direction of evolutionary change. That is, an understanding of both genetic flexibility and genetic constraint is required. Furthermore, antagonistic pleiotropy can lead to complex adaptive landscapes, and under these conditions an understanding of stochastic forces, mediated through population dynamics, and their interaction with systematic forces is also required in order to provide an explanation for the genetic composition of a population.

SUMMARY

Estimates of genetic variation and covariation for a series of fitness components (two measurements of survival, larval weight, development time, fecundity, and female productivity) were obtained from two populations of T. confusum (populations 1-1 and 1-2). Prior to sampling for the present experiment, the two populations were maintained under identical, controlled culture conditions for over 10 years.

An analysis of genotypic means indicates extensive genetic differentiation has occurred between the two populations for all characters except development time. Individuals from population 1-2 are smaller with higher survival, but females have lower fecundity than individuals from population 1-1. This pattern of differentiation also contributes to greater female productivity in population 1-1.

Population 1-1 contains relatively large amounts of additive genetic variation for survival, larval weight, fecundity, and female productivity. Many of the variance component estimates from population 1-2 are negative. Both populations contain significant amounts of nonadditive variation for fecundity and female productivity which appears to be due to nonadditive genetic variance and/or common environmental effects and maternal effects.

Population 1-1 also contains significant amounts of genetic covariation for several of the characters. Positive genetic covariation exists for the two measurements of survival, while the genetic correlation between survival and fecundity is large and negative. The genetic correlation estimates from population 1-2 are undefined since the variance component estimates are negative.

There appears to be a relationship between genetic variation and covariation for survival and fecundity in population 1-1. That is, significant amounts of genetic variation were detected for survival and fecundity in addition to a significant negative genetic correlation between these characters. This result is consistent with the theoretical expectation that antagonistic pleiotropy among fitness components can maintain genetic variation in the individual characters.

This type of genetic structure is consistent with the shifting balance theory of evolution and could explain the substantial amount of genetic differentiation observed between the two populations. That is, strong negative genetic correlations among fitness components can not only maintain genetic variation in individual characters, but also generate complex adaptive landscapes where stochastic forces operating in conjunction with systematic forces can produce large population differences among the various traits.

Table 1

Sample analyis of variance table with expected mean squares based on a balanced design.

Variance source	df	MS	EMS
Blocks (B)	b-1	MS_b	$V_e + rpV_b$
Populations (P)	p-1	MS_p	$V_e + rV_{bp} + rbK^2_p$
B x P	(b-1)(p-1)	MS_{bp}	$V_e + rV_{bp}$
Error	bp(r-1)	MS_e	V_e

b = number of blocks; p = number of populations; r = number of replications; MS = mean square; EMS = expected mean square; V = variance due to random effects; K^2 = fixed treatment effect.

Table 2

Sample analyis of variance table with expected mean squares for the HS/FS design.

Variance source	df	MS	EMS
Blocks	b-1	MS_b	
Sires	b(s-1)	MS_s	$V_w + k_2V_d + k_3V_s$
Dams/Sires	bs(d-1)	MS_d	$V_w + k_1V_d$
Progeny/Dams/Sires	bsd(r-1)	MS_w	V_w

b = number of blocks; s = number of sires per block; d = number of dams per sire; r = number of progeny per dam; MS = mean square; EMS = expected mean square; V = variance component; k_i = coefficients of expected mean squares.

Estimation of variance components:

$V_w = MS_w$
$V_d = MS_d - MS_w/k_1$
$V_s = MS_s - (MS_w + k_2MS_d)/k_3$.

With unequal numbers per subclass $k_1 \neq k_2$.

Table 3

Sample analyis of variance table with expected mean squares for the HS design.

Variance source	df	MS	EMS
Blocks	b-1	MS_b	
Sires	b(s-1)	MS_s	$V_w + k_1 V_s$
Progeny/Sires	bs(d-1)	MS_d	V_w

b = number of blocks; s = number of sires per block; d = number of dams per sire; MS = mean square; EMS = expected mean square; V = variance component; k_i = coefficients of expected mean squares.

Estimation of variance components:

$V_w = MS_w$
$V_s = MS_s - MS_w/k_1$.

Table 4

Means ± standard errors of the measured characters in both populations by generation.

Generation 1						
Population	28 - day survival (%)	35 - day survival (%)	Development time (%)	Mean larval weight (mg)	Fecundity (eggs/female)	Female productivity
1-1	1.12 ± 0.007	0.89 ± 0.006	0.89 ± 0.012	1.36 ± 0.003	42.20 ± 0.178	1.86 ± 0.004
1-2	1.20 ± 0.007	1.00 ± 0.006	0.87 ± 0.012	1.32 ± 0.003	36.73 ± 0.178	1.81 ± 0.004

Generation 2				
Population	28 - day survival (%)	Mean larval weight (mg)	Fecundity (eggs/female)	Female productivity
1-1	1.13 ± 0.011	1.35 ± 0.009	39.08 ± 0.283	1.83 ± 0.008
1-2	1.21 ± 0.011	1.32 ± 0.009	36.76 ± 0.283	1.81 ± 0.008

Table 5

Analyses of variance of the measured characteristics for both populations by generation.

		Generation 1					
Source of variation	df	28 - day survival	35 - day survival	Development time	Mean larval weight	Fecundity	Female productivity
Block (B)	31	0.1497****	0.1315****	0.2813****	0.0127**	103.91*	2.4916****
Population (P)	1	2.9380****	6.1764****	0.3113	0.7168****	13493.82****	1.7914****
B x P	31	0.0451	0.0324	0.1342	0.0063	30.25	0.0129
Error	1740	0.0376	0.0365	0.1169	0.0066	64.62	0.0258

		Generation 2			
Source of variation	df	28 - day survival	Mean larval weight	Fecundity	Female productivity
Block (B)	19	0.2565****	0.0205****	238.96****	0.0892****
Population (P)	1	6.2345****	0.7351****	2375.94****	0.2967*
B x P	19	0.0967****	0.0425****	9.23	0.0445**
Error	4391	0.0338	0.0052	28.10	0.0209

* $P < 0.05$; ** $P < 0.01$; **** $P < 0.0001$

Table 6

Heritability estimates from the sire component of variance of the HS design ± standard errors for both populations.

Population	28 - day survival	35 - day survival	Development time	Mean larval weight
1-1	0.33 ± 0.16 *	0.14 ± 0.15	0.00 ± 0.15	0.36 ± 0.16 *
1-2	0.00 ± 0.15	0.00 ± 0.15	0.00 ± 0.15	0.06 ± 0.15

* $P < 0.05$

Table 7

Heritability estimates from the sire, dam, and sire plus dam components of variance of the HS/FS design ± standard errors for both populations.

Population	Fecundity			Female productivity		
	sire	dam	sire + dam	sire	dam	sire + dam
1-1	0.32 ± 0.09 *	1.06 ± 0.10 *	0.69 ± 0.05 *	0.28 ± 0.12 *	1.45 ± 0.15 *	0.86 ± 0.07 *
1-2	0.00 ± 0.04	0.56 ± 0.09 *	0.28 ± 0.03 *	0.00 ± 0.06	1.19 ± 0.14 *	0.60 ± 0.05 *

* $P < 0.05$

Table 8

Heritability estimates from the D/O regression ± standard errors for both populations.

Population	Fecundity	Female productivity
1-1	0.26 ± 0.09 *	1.46 ± 0.08 *
1-2	0.30 ± 0.10 *	1.30 ± 0.08 *

* $P < 0.05$

Table 9

Genetic correlation estimates from the HS design ± standard errors for population 1-1.

Character	28 - day survival	35 - day survival	Development time	Mean larval weight	Fecundity	Female productivity
28-day survival	-	+0.70 ± 0.35 *	undefined	+0.53 ± 0.32	-0.54 ± 0.27 *	+0.40 ± 0.31
35-day sirvival		-	undefined	-0.27 ± 0.50	-0.69 ± 0.48	+0.01 ± 0.59
Development time			-	undefined	undefined	-0.78 ± 0.89
Larval weight				-	-0.08 ± 0.24	+0.16 ± 0.42
Fecundity					-	+0.44 ± 0.22 *
Female productivity						-

* $P < 0.05$

Table 10

Analysis of variance of 28 - day survival from the HS design for from the half- sib design for population 1-1.

Variance source	df	ms	Variance component	Percent
Block	31	0.0874	0.0014	3.19
Sire	274	0.0489	0.0035	8.05
Error	572	0.0388	0.0388	88.75

Table 11

Analysis of variance of 35 - day survival from the HS design for population 1-1.

Variance source	df	ms	Variance component	Percent
Blocks	31	0.0932	0.0017	3.79
Sires/Blocks	274	0.0464	0.0017	3.75
Progeny/Sires/Blocks	572	0.0416	0.0416	92.46

Table 12

Analysis of variance of development time from the HS design for population 1-1.

Variance source	df	ms	Variance component	Percent
Blocks	31	0.2825	0.0057	4.20
Sires/Blocks	274	0.1273	-0.0006	0.00
Progeny/Sires/Blocks	572	0.1289	0.1289	95.79

Table 13

Analysis of variance of mean larval weight from the HS design for population 1-1.

Variance source	df	ms	Variance component	Percent
Blocks	31	0.0065	0.0000	0.00
Sires/Blocks	274	0.0074	0.0006	9.09
Progeny/Sires/Blocks	572	0.0057	0.0057	90.90

Table 14

Analysis of variance of 28 - day survival from the HS design for population 1-2.

Variance source	df	ms	Variance component	Percent
Blocks	31	0.1024	0.0027	7.00
Sires/Blocks	275	0.0289	-0.0022	0.00
Progeny/Sires/Blocks	578	0.0353	0.0353	92.99

Table 15

Analysis of variance of 35 - day survival from the HS design for population 1-2.

Variance source	df	ms	Variance component	Percent
Blocks	31	0.0710	0.0015	4.83
Sires/Blocks	275	0.0270	-0.0015	0.00
Progeny/Sires/Blocks	578	0.0313	0.0313	95.16

Table 16

Analysis of variance of development time from the HS design for population 1-2.

Variance source	df	ms	Variance component	Percent
Blocks	31	0.1249	0.0008	0.75
Sires/Blocks	275	0.1030	-0.0007	0.00
Progeny/Sires/Blocks	578	0.1051	0.1051	99.25

Table 17

Analysis of variance of mean larval weight from the HS design for population 1-2.

Variance source	df	ms	Variance component	Percent
Blocks	31	0.0131	0.0002	3.03
Sires/Blocks	275	0.0071	0.0001	1.54
Progeny/Sires/Blocks	578	0.0068	0.0068	95.42

Table 18

Analysis of variance of fecundity from the HS/FS design for population 1-1.

Variance source	df	ms	Variance component	Percent
Blocks	31	478.03	2.58	3.37
Sires/Blocks	274	194.13	5.90	7.72
Dams/Sires/Blocks	572	126.48	19.52	25.53
Progeny/Dams/Sires/Blocks	2634	48.42	48.43	63.36

Table 19

Analysis of variance of fecundity from the HS/FS design for population 1-2.

Variance source	df	ms	Variance component	Percent
Blocks	31	514.73	4.09	6.49
Sires/Blocks	275	61.83	-1.90	0.00
Dams/Sires/Blocks	578	83.79	8.26	13.09
Progeny/Dams/Sires/Blocks	2655	50.74	50.74	80.41

Table 20

Analysis of variance of female productivity from the HS/FS design for population 1-1.

Variance source	df	ms	Variance component	Percent
Blocks	19	0.0512	-0.0002	0.00
Sires/Blocks	172	0.0680	0.0017	6.91
Dams/Sires/Blocks	358	0.0488	0.0088	36.12
Progeny/Dams/Sires/Blocks	1650	0.0138	0.0138	56.96

Table 21

Analysis of variance of female productivity from the HS/FS design for population 1-2.

Variance source	df	ms	Variance component	Percent
Blocks	19	0.0824	0.0005	2.79
Sires/Blocks	170	0.0218	-0.0013	0.00
Dams/Sires/Blocks	358	0.0364	0.0057	28.92
Progeny/Dams/Sires/Blocks	1644	0.0135	0.0135	68.27

Table 22

Regression analysis of fecundity from the D/O design for population 1-1.

Variance source	df	ms
Dams	1	578.2974
Error	876	75.4797

Table 23

Regression analysis of female productivity from the D/O design for population 1-1.

Variance source	df	ms
Dams	1	7.7405
Error	876	0.0221

Table 24

Regression analysis of fecundity from the D/O design for population 1-2.

Variance source	df	ms
Dams	1	472.9509
Error	883	52.3546

Table 25

Regression analysis of female productivity from the D/O design for population 1-2.

Variance source	df	ms
Dams	1	4.0493
Error	883	0.0174

Table 26

Analysis of covariance of 28 - day survival and 35 - day survival from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	0.0726	0.0017
Sires/Blocks	274	0.0247	0.0017
Progeny/Sires/Blocks	572	0.0198	0.0198

Table 27

Analysis of covariance of 28 - day survival and development time from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	-0.0912	-0.0029
Sires/Blocks	274	-0.0121	-0.0007
Progeny/Sires/Blocks	572	-0.0100	-0.0100

Table 28

Analysis of covariance of 28 - day survival and mean larval weight from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	-0.0005	-0.0001
Sires/Blocks	274	0.0032	0.0008
Progeny/Sires/Blocks	572	0.0010	0.0010

Table 29

Analysis of covariance of 28 - day survival and fecundity from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	-1.7487	-0.0568
Sires/Blocks	274	-0.1860	-0.0779
Progeny/Sires/Blocks	572	0.0370	0.0370

Table 30

Analysis of covariance of 35 - day survival and development time from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	-0.1250	-0.0035
Sires/Blocks	274	-0.0275	-0.0032
Progeny/Sires/Blocks	572	-0.0183	-0.0183

Table 31

Analysis of covariance of 35 - day survival and mean larval weight from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	0.0054	0.0002
Sires/Blocks	274	0.0012	-0.0003
Progeny/Sires/Blocks	572	0.0020	0.0020

Table 32

Analysis of covariance of 35 - day survival and fecundity from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	-2.0252	-0.0696
Sires/Blocks	274	-0.1124	-0.0688
Progeny/Sires/Blocks	572	0.0850	0.0850

Table 33

Analysis of covariance of development time and mean larval weight from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	-0.0016	0.0000
Sires/Blocks	274	-0.0023	0.0000
Progeny/Sires/Blocks	572	-0.0022	-0.0022

Table 34

Analysis of covariance of development time and fecundity from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	2.3612	0.0880
Sires/Blocks	274	-0.0564	-0.0133
Progeny/Sires/Blocks	572	-0.0152	-0.0152

Table 35

Analysis of covariance of mean larval weight and and fecundity from the HS design for population 1-1.

Variance source	df	mcp	Covariance component
Blocks	31	-0.1758	-0.0073
Sires/Blocks	274	0.0240	-0.0047
Progeny/Sires/Blocks	572	0.0375	0.0375

Table 36

Analysis of covariance of 28 - day survival and 35 - day survival from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	0.0485	0.0012
Sires/Blocks	275	0.0140	-0.0013
Progeny/Sires/Blocks	578	0.0178	0.0178

Table 37

Analysis of covariance of 28 - day survival and development time from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	-0.0497	-0.0014
Sires/Blocks	275	-0.0109	0.0018
Progeny/Sires/Blocks	578	-0.0161	-0.0161

Table 38

Analysis of covariance of 28 - day survival and mean larval weight from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	0.0068	0.0003
Sires/Blocks	275	-0.0005	-0.0005
Progeny/Sires/Blocks	578	0.0009	0.0009

Table 39

Analysis of covariance of 28 - day survival and fecundity from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	-0.0185	0.0014
Sires/Blocks	275	-0.0561	-0.0338
Progeny/Sires/Blocks	578	0.0412	0.0412

Table 40

Analysis of covariance of 35 - day survival and development time from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	-0.0704	-0.0017
Sires/Blocks	275	-0.0245	0.0012
Progeny/Sires/Blocks	578	-0.0280	-0.0280

Table 41

Analysis of covariance of 35 - day survival and mean larval weight from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	0.0060	0.0002
Sires/Blocks	275	0.0007	0.0003
Progeny/Sires/Blocks	578	-0.0002	-0.0002

Table 42

Analysis of covariance of 35 - day survival and fecundity from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	-0.0008	0.0026
Sires/Blocks	275	-0.0705	-0.0286
Progeny/Sires/Blocks	578	0.0119	0.0119

Table 43

Analysis of covariance of development time and mean larval weight from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	0.0022	0.0001
Sires/Blocks	275	0.0005	-0.0002
Progeny/Sires/Blocks	578	0.0011	0.0011

Table 44

Analysis of covariance of development time and fecundity from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	-0.3404	-0.0157
Sires/Blocks	275	0.0897	0.0814
Progeny/Sires/Blocks	578	-0.1446	-0.1446

Table 45

Analysis of covariance of mean larval weight and fecundity from the HS design for population 1-2.

Variance source	df	mcp	Covariance component
Blocks	31	-0.7788	-0.0272
Sires/Blocks	275	-0.0261	-0.0224
Progeny/Sires/Blocks	578	0.0385	0.0385

LITERATURE CITED

Becker, W.A. 1975. A Manual of Quantitative Genetics. Washington State Univ. Press, Pullman.

Bell, G. 1984a. Measuring the cost of reproduction. I. The correlation structure of the life table of a plankton rotifer. Evolution 38: 300-313.

Bell, G. 1984b. Measuring the cost of reproduction. II. The correlation structure of the life tables of five freshwater invertebrates. Evolution 38: 314-326.

Bridges, W.C. and Knapp, S.J. 1987. Probabilities of negative estimates of genetic variance. Theor. Appl. Genet. 74: 269-274.

Bulmer, M.G. 1971. Effects of selection on genetic variability. Am. Natur. 105: 201-211.

Bulmer, M.G. 1972. The genetic variability of polygenic characters under optimizing selection, mutation, and drift. Genet. Res. 19: 17-25.

Bulmer, M.G. 1973. Maintenance of genetic variability of polygenic characters by heterozygous advantage. Genet. Res. 22: 9-12.

Caspari, E.G. 1952. Pleiotropic gene action. Evolution 6: 1-18.

Charlesworth, B. 1980. Evolution in Age-Structured Populations. Cambridge Univ. Press, London.

Christensen, F.B. and Feldman, M.W. 1975. Subdivided populations: a review of the one and two-locus deterministic theory. Theor. Popul. Biol. 7: 13-38.

Dawson, P.S. 1964. Age at sexual maturity in female flour beetles, Tribolium castaneum and T. confusum. Ann. Entom. Soc. Amer. 57: 1-3.

Dawson, P.S. 1965. Estimation of components of phenotypic variance for developmental rate in Tribolium. Heredity 20: 403-417.

Dawson, P.S. 1966. Correlated response to selection for developmental rate in Tribolium. Genetica 37: 63-77.

Dawson, P.S. 1975. Directional versus stabilizing selection for developmental time in natural and laboratory populations of flour beetles. Genetics 80: 773-783.

Dawson, P.S. and Riddle, R.A. 1983. Genetic variation, environmental heterogeneity, and evolutionary stability. In Population Biology: Retrospect and Prospect (eds. C.E. King and P.S. Dawson). pp. 147-170. Columbia Univ. Press, New York.

Dickerson, G.E. 1955. Genetic slippage in response to selection for multiple objectives. Cold Spring. Harb. Symp. Quant. Biol. 20: 213-224.

Dickerson, G.E. 1969. Techniques for research in quantitative animal genetics. In Techniques and Procedures in Animal Production Research. Am. Soc. Anim. Prod. Publication.

Dingle, H. 1984. Behavior, genes, and life histories: complex adaptations in uncertain environments. In A New Ecology: Novel Approaches to Interactive Systems. (eds. P.W. Price,

C.N. Slobodchkoff, and W.S Gaud). pp. 169-194. Wiley, New York.

Dobzhansky, Th. 1947. Genetics of natural populations. XIV. A response of certain gene arrangements in third chromosome of Drosophila pseudoobscura to natural selection. Genetics 32: 142-160.

Dobzhansky, Th. 1948. Genetics of natural populations. XVI. Altitudinal and seasonal changes produced by natural selection in certain populations of Drosophila pseudoobscura and Drosophila persimilis. Genetics 33: 158-176.

Dobzhansky, Th. 1955. A review of some fundamental concepts and problems of population genetics. Cold Spring Harb. Symp. Quant. Biol. 20: 1-15.

Dobzhansky, Th. 1948. Genetics of natural populations. XXV. Genetic changes in populations of Drosophila pseudoobscura and Drosophila persimilis in some locations in California. Evolution 10: 82-92.

Dobzhansky, Th. 1970. Genetics of the Evolutionary Process. Columbia Univ. Press, New York.

Dobzhansky, Th. and Levene, H. 1948. Genetics of natural populations. XVII. Proof of operation of natural selection in wild populations of Drosophila pseudoobscura. Genetics 33: 537-547.

Dobzhansky, Th. and Pavlovsky, O. 1953. Indeterminate outcome of certain experiments on Drosophila populations. Evolution 7: 198-210.

ElRouby, M.M. and Penny, L.H. 1967. Variation and covariation in a high oil population of corn (Zea mays L.) and their implications in selection. Crop Sci. 7: 216-219.

Erdman, H.E. 1964. Sexual precocity of male flour beetle, Tribolium castanem Herbst, and the influence of temperatures on reproduction during early adult life. Can. Entom. 96: 656-659.

Felsenstein, J. 1976. The theoretical population genetics of variable selection and migration. Ann. Rev. Genet. 10: 253-280.

Fisher, R.A. 1918. The correlation between relatives on the supposition of Mendelian inheritance. Trans. Roy. Soc. Edinburgh. 52: 399-433.

Fisher, R.A. 1930. The Genetical Theory of Natural Selection (1st ed.). Clarendon Press, Oxford.

Falconer, D.S. 1981. Introduction to Quantitative Genetics (2nd ed.). Longman, New York.

Ford, E.B. 1964. Ecological Genetics. Wiley and Sons, New York.

Giesel, J.T. and Zettler, E.E. 1980. Genetic correlations of life-historical parameters and certain fitness indices in Drosophila melanogaster: r_m, r_s, diet breadth. Oecologia 47: 299-302.

Giesel, J.T. 1979. Genetic co-variation of survivorship and other fitness indices in Drosophila melanogaster. Exp. Gerontol. 14: 323-328.

Gillespie, J.H. 1984. Pleiotropic overdominance and the maintenance of genetic variation in polygenic characters. Genetics 107: 321-330.

Good, N.E. 1933. Biology of the flour beetles Tribolium confusum Duv. and T. ferrugineum Fab. J. Agric. Res. 46: 327-334.

Gowen, J.W. and Johnson, L.E. 1946. On the mechanism of heterosis. I. Metabolic capacity of different races of Drosophila melanogaster for egg production. Am. Natur. 80: 149-179.

Haldane, J.B.S. 1927. A mathematical theory of natural and artificial selection. Part V. Selection and mutation. Proc. Camb. Phil. Soc. 23: 838-844.

Haldane, J.B.S. 1932. The Causes of Evolution (1st ed.). Harper and Bros., London.

Hallauer, A.R. and Miranda, J.B. 1981. Quantitative genetics in maize breeding. Iowa State Univ. Press, Ames.

Hammond, K. and Nicholas, F.W. 1972. The sampling variance of the correlation coefficients estimated from two-fold nested and offspring-parent regression analyses. Theor. Appl. Genet. 42: 97-100.

Harris, H. 1966. Enzyme polymorphisms in man. Proc. Roy. Soc. B 164: 298-310.

Hedrick, P.W. 1986. Genetic polymorphism in heterogeneous environments: a decade later. Ann. Rev. Ecol. Syst. 17: 535-566.

Hedrick, P.W., Ginevan, M.E., and Ewing, E.P. 1976. Genetic polymorphism in heterogeneous environments. Ann. Rev. Ecol. Syst. 7: 1-32.

Hinton, H.E. 1948. A synopsis of the genus Tribolium Macleay with some remarks on the evolution of its species-groups (Coeleoptera, Tenebrionidae). Bull. Ent. Res. 39: 13-55.

Hiraizumi, Y. 1961. Negative correlation between rate of development and female fertility in Drosophila melanogaster. Genetics 46: 615-624.

Istock, C.A. 1983. The extent and consequences of heritable variation for fitness characters. In Population Biology: Retrospect and Prospect (eds. C.E. King and P.S. Dawson). pp. 48-67.

Istock, C.A. 1984. Boundaries to life history variation and evolution. In A New Ecology: Novel Approaches to Interactive Systems. (eds. P.W Price, C.N. Slobodchkoff, and W.S. Gaud). pp. 143-168. Wiley, New York.

Kimura, M. 1965. A stochastic model concerning the maintenance of genetic variability in quantitative characters. Proc. Natl. Acad. Sci. USA 54: 731-736.

Kimura, M. and Ohta, T. 1971. Theoretical Aspects of Population Genetics. Princeton Univ. Press.

King, C.E. and Dawson, P.S. 1972. Population biology and the Tribolium model. Evol. Biol. 5: 133-227.

Krause, E. and Bell, A.E. 1972. A genetic study of biomass in Tribolium. I. Path coefficient analysis of base populations. Can. J. Genet. Cytol. 14: 181-193.

Lande, R. 1975. The maintenance of genetic variability by mutation in a polygenic character with linked loci. Genet. Res. 26: 221-235.

Lande, R. 1980. The genetic covariance between characters maintained by pleiotropic mutations. Genetics 94: 203-215.

Lande, R. 1982a. A quantitative genetic theory of life history evolution. Ecology 63: 607-615.

Lande, R. 1982b. Elements of a quantitative genetic model of life history evolution. In Evolution and Genetics of of Life Histories (eds. H. Dingle and J.P. Hegmann). pp. 21-29. Springer-Verlag, New York.

Latter, B.D.H. 1960. Natural selection for an intermediate optimum. Aust. J. Biol. Sci. 13: 30-35.

Lerner, I.M. 1954. Genetic Homeostasis. Wiley and Sons, New York.

Lewontin, R.C. 1974. The Genetic Basis of Evolutionary Change. Columbia Univ. Press, New York.

Lewontin, R.C. and Hubby, J.L. 1966. A molecular approach to the study of genic heterozygosity in natural populations. II. Amount of variation and degree of heterozygosity in natural populations of Drosophila pseudoobscura. Genetics 54: 595-609.

Lindsey, M.D., Lonnquist, J.H., and Gardner, C.O. 1962. Estimates of genetic variance in open-pollinated varieties of corn-belt corn. Crop Sci. 2: 105-108.

Luckinbill, L.S., Arking, R., Clare, M.J., Cirocco, W.C., and Buck, S.A. 1984. Selection for delayed senescence in Drosophila melanogaster. Evolution 38: 996-1003.

MacKay, T.F.C. 1980. Genetic variance, fitness, and homeostasis in varying environments: An experimental check of the theory. Evolution 34: 1219-1222.

MacKay, T.F.C. 1981. Genetic variation in varying environments. Genet. Res. 37: 79-93.

Mather, K. 1953. The genetical structure of populations. Symp. Soc. Exp. Biol. 7: 66-95.

Mather, K. 1955. Polymorphism as an outcome of disruptive selection. Evolution 9: 52-61.

Mukai, T. and Yamazaki, T. 1971. Genetic structure of natural populations of Drosophila melanogaster X. Developmental time and viability. Genetics 69: 385-398.

Muller, H.J. 1950. Our load of mutations. Am. J. Hum. Genetics 2: 111-176.

Murphy, P.A., Giesel, J.T., and Manlove, M.N. 1983. Temperature effects on life history variation in Drosophila simulans. Evolution 37: 1181-1192.

Nevo, E. 1978. Genetic variation in natural populations: patterns and theory. Theor. Popul. Biol. 13: 121-177.

Nevo, E., Beiles, A. and Ben - Shlomo, R. 1984. The evolutionary significance of genetic diversity: ecological, demographic, and life - history correlates. In Evolutionary Dynamics of Genetic Diversity. Lecture Notes in Biomathematics 53 (ed. G.S. Mani). pp. 13 - 213. Springer - Verlag, New York.

Norton, N.E. 1974. Analysis of family resemblance. I. Introduction. Am. J. Hum. Genet. 26: 318-330.

Park, T. 1934. Observations on the general biology of the flour beetle Tribolium confusum. Quart. Rev. Biol. 9: 36-54.

Provine, W.B. 1971. The Origins of Theoretical Population Genetics. Univ. of Chicago Press.

Riddle, R.A., Dawson, P.S., Zirkle, D.F. 1986. An experimental test of the relationship between genetic variation and environmental variation in Tribolium flour beetles. Genetics 113: 391-404.

Robertson, A. 1955. Selection in animals. Cold Spring Harb. Symp. Quant. Biol. 20: 225-229.

Robinson, H.F., Comstock, R.E., and Harvey, P.H. 1955. Genetic variances in open pollinated varieties of corn. Genetics 40: 45-60.

Rose, M.R. 1982. Antagonistic pleiotropy, dominance, and genetic variation. Heredity 48: 63-78.

Rose, M.R. 1983. Further models of selection with antagonistic pleiotropy. In Population Biology. (eds. H.I. Freedman and C. Strobeck). pp. 47 - 53. Springer - Verlag, New York.

Rose, M.R. 1984a. Artificial selection on a fitness component in Drosophila melanogaster. Evolution 38: 516-526.

Rose, M.R. 1984b. Genetic covariation in Drosophila life history: Untangling the data. Am. Natur. 123: 565-569.

Rose, M.R. 1984c. Laboratory evolution of postponed senescence in Drosophila melanogaster. Evolution 38: 1004-1010.

Rose, M.R. 1985. Life history evolution with antagonistic pleiotropy and overlapping generations. Theor. Popul. Biol. 28: 342-358.

Rose, M.R. and Charlesworth, B. 1981a. Genetics of life history in Drosophila melanogaster. I. Sib analysis of adult females. Genetics 97: 173-186.

Rose, M.R. and Charlesworth, B. 1981b. Genetics of life history in Drosophila melanogaster. II. Exploratory selection experiments. Genetics 97: 187-196.

Scheinberg, E. 1966. The sampling variance of the correlation coefficients estimated in genetic experiments. Biometrics 22: 187-191.

Searle, S.R. 1971. Linear Models. Wiley and Sons, New York.

Sentz, J.C. 1971. Genetic variances in a synthetic variety of maize estimated by two mating designs. Crop Sci. 11: 234-238.

Service, P.M. and Rose, M.R. 1985. Genetic covariation among life - history components: the effect of novel environments. Evolution 39: 943-945.

Simmons, M.J., Preston, C.R., and Engels, W.R. 1980. Pleiotropic effects on fitness of mutations affecting viability in Drosophila melanogaster. Genetics 94: 467-475.

Slatkin, M. 1978. Spatial patterns in the distribution of polygenic characters. J. Theoret. Biol. 70: 213-228.

Slatkin, M. 1982. Pleiotropy and parapatric speciation. Evolution 36: 263-270.

Stearns, S.C. 1983. The genetic basis of differences in life-history traits among six populations of mosquitofish (Gambusia affinis) that shared ancestors in 1905. Evolution 37: 618-627.

Sturtevant, A.H. 1926. A crossover reducer in Drosophila melanogaster due to an inversion of a section of the third chromosome. Biol. Zentral. 46: 697-702.

Swiger, L.A., Harvey, W.R., Everson, D.O., and Gregory, K.E. 1964. The variance of intraclass correlation involving groups with one observation. Biometrics 20: 818-826.

Temin, R.G. 1966. Homozygous viability and fertility loads in Drosophila melanogaster. Genetics 53: 27-46.

Turelli, M. 1984. Effects of pleiotropy on predictions concerning mutation-selection balance for polygenic traits. Genetics 111: 165-195.

Via, S. 1984. The quantitative genetics of polyphagy in an insect herbivore. I. Genotype-environment interaction in larval performance on different host plant species. Evolution 38: 881-895.

Via, S. and Lande, R. 1985. Genotype-environment interaction and the evolution of phenotypic plasticity. Evolution 39: 505-523.

Wallace, B. 1959. The role of heterozygosity in Drosophila populations. Proc. 10th Intern. Cong. Genetics 1: 408-419.

Williams, J.C., Penny, L.H., and Sprague, G.F. 1965. Full-sib and half-sib estimates of genetic variance in an open-pollinated variety of corn, Zea mays L. Crop Sci. 5: 125-129.

Wright, S. 1921. Systems of mating. I-V. Genetics 6: 111-178.

Wright, S. 1931. Evolution in Mendelian populations. Genetics 16: 97-159.

Wright, S. 1977. Evolution and the Genetics of Populations, Volume 3. Univ. of Chicago Press.

Wright, S. 1978. Evolution and the Genetics of Populations, Volume 4. Univ. of Chicago Press.

Young, A.M. 1970. Predation and abundance in populations of flour beetles. Ecology 51: 602-619.

Zirkle, D.F. and Riddle, R.A. 1983. Quantitative genetic response to environmental heterogeneity in Tribolium confusum. Evolution 37: 637-639.