nd a variable region that may be important for
y and nonvariability both important?

n comment on the interpretation of the correla-
and measure of whole-animal performance and
ould be interpreted as an indication that there is
per se, or they could simply reflect inbreeding
ion structure. What is your feeling on this?

h the inbreeding explanation. For one thing, the
lationship has been most dramatically demon-
s, and various bivalve species, are the kinds of
ect to have very large effective population sizes.
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est this experimentally. The answer depends on
ochemical genetic mechanism, much as we have
(e.g., LDH and LAP).

9 Genetic correlation and the evolution of physiology

STEVAN J. ARNOLD

Correlation . . . has no doubt played a most important part [in shaping structures during evolution], and a useful modification of one part will often have entailed on other parts diversified changes of no direct use.

C. Darwin (1859, p. 199)

Statement

Genetic coupling between physiological traits can be studied by analyzing the individual variation that resides within populations. Genetic coupling or correlation can deflect the course of adaptive evolution and cause temporary maladaptation in physiology and other traits. Genetic models indicate that maladaptive phases of physiological evolution could last many thousands of generations.

Introduction

Variation within natural populations has been neglected by physiologists despite its evolutionary importance (Endler, 1986; Chapter 7). Comparative physiology has usually focused on contrasts between populations, species, and higher taxa, and variation within populations has been overlooked. Yet, as Darwin realized, heritable differences within populations are molded by natural selection to produce geographic and specific differences. The study of intrapopulational variation can reveal ongoing processes of selection as well as genetic constraints on the microevolutionary transformation of populations. My aim is to discuss the insights we might get by quantitative genetic analysis of variation in physiology. The discussion is necessarily speculative because several interesting genetic avenues have not been explored. My focus will be on coupling between physiological traits. Genetic coupling, commonly measured as genetic correlation, may constrain the evolution of physiological traits and can lead to counterintuitive evolutionary outcomes.

In the following sections, I begin by describing two different approaches to studying the genetics of physiology. Next I outline the essential concepts of heritability and genetic correlation which underlie one of those

approaches, namely quantitative genetics. I briefly discuss the evolutionary importance of those concepts and move on to a discussion of the different manifestations of genetic correlation. This discussion relies on many hypothetical examples because so little quantitative genetic work has focused on physiological traits. Nevertheless, genetic correlations in physiology seem likely in many circumstances. Next I consider some detailed examples (again, mostly hypothetical) of how genetic correlations might have a major impact on physiological evolution. Finally I argue that we need more quantitative genetic studies of physiology and describe some possible directions and practical considerations.

Background

Two complementary genetic approaches

Genetic studies of physiology fall into two general categories that differ in outlook: the gene-to-physiology viewpoint and the physiology-to-genetics perspective. The two approaches are complementary and answer different questions, yet they have hardly ever been used in combination. The gene-to-physiology approach begins with the observation of natural variation of a particular gene locus, commonly assayed by protein electrophoresis, and then shows the effects of that variation on physiology and fitness (Chapters 5 and 8). Watt, Carter, and Donohue (1986), for example, review a particularly elegant example of this approach. They trace the impact of phosphoglucose isomerase variation in butterfly populations on flight capacity, survivorship, and mating success. The gene-to-physiology approach is aesthetically pleasing because it can reveal an entire pathway from gene to fitness. More importantly, the approach yields the information necessary to model change in the frequency of a gene that affects physiology.

In contrast, the physiology-to-genetics approach focuses on physiological attributes that may be affected by numerous genes rather than on the physiological effects of a particular gene. The aim is to understand how the evolution of one physiological trait will affect the evolution of other traits. For example, deliberate selection for large body size in house mice results in larger mice, but these mice also have smaller interscapular pads of brown adipose tissue (Sulzbach and Lynch, 1984). This example illustrates the general principle that evolutionary modification of one element in a physiological system can have reverberating effects on other elements in the system. Such reverberations can be anticipated by studying resemblance among relatives in physiological traits. Thus the physiology-to-genetics approach can succeed at its goal of predicting interactions during evolution without tracing pathways to particular genes. The approach does not tell us which particular genes are associated with variation i
genes affect that variation.

Likewise the gene-to-physiology approa
genes affect a particular physiological trait.
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Quantitative genetics and the importance o

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ticular genes are associated with variation in physiology or even how many genes affect that variation.

Likewise the gene-to-physiology approach does not tell us how many genes affect a particular physiological trait. Furthermore, because the main goal is to trace a particular pathway from genotype to phenotype, the gene-to-physiology approach does not tell us whether physiological traits are likely to interact and affect one another's evolution.

Quantitative genetics and the importance of genetic correlation

Quantitative genetics is the genetic discipline that uses what I have called the physiology-to-genetics approach. The discipline focuses on the inheritance of traits whose variation is affected by many genes and by the environment. Many physiological attributes are probably polygenic, so quantitative genetics is a natural analytical tool.

The basic idea in quantitative genetics is to predict the attributes of offspring from the characteristics of their parents. A hypothetical example of resemblance in body size between offspring and parents in a population of mice is shown in Figure 9.1. Each point represents the average body size of the offspring plotted against the average body size of their parents, all sizes measured in adulthood. In this example, offspring strongly resemble their parents in body size. If we randomize environments in both generations, so as to eliminate environmental causes of parent-offspring resemblance, we can show with a little algebra that the observed resemblance is due to the additive properties of the many genes that might affect body size (Fisher, 1918). The slope of the best-fit line that predicts offspring values from those of their parents is known as *heritability*. The slope turns out to be mathematically equivalent to a measure of variation in the genetic values of the parents, referred to as *genetic variance* for body size.

The plot of offspring against their parents also enables us to visualize the consequences of selection. Suppose the plot represents all the potential parents and their hypothetical offspring but only a fraction of the potential parents actually survive to breed and produce offspring. In particular, imagine that only the pairs of parents in the top 20% of average body masses actually breed. We can readily deduce the consequences of this selection. In Figure 9.2, we have the same plot of offspring-parental resemblance as in Figure 9.1. The vertical dotted line marks the average body size of the actual, selected parents. The distance between these lines measures the strength of selection on body size, shown with the heavy arrow. The dotted horizontal line shows the body size of offspring expected from the average of all potential parents. This is the body size that would prevail in the next generation, and in all succeeding generations, if no selection were imposed. The solid horizontal line shows the size of offspring expected from the selected parents. These offspring are larger than the offspring of all potential parents, as shown by

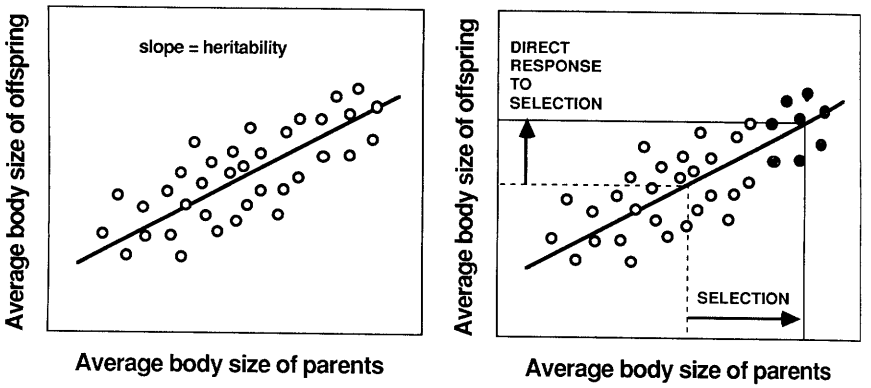

FIGURE 9.1 (Left) The average body size of offspring plotted against the average body size of their parents. Each point in this hypothetical plot represents the average value for offspring and the average for their two parents. The slope of the least squares regression of offspring on parents estimates the heritability of the trait.

FIGURE 9.2 (Right) Direct response to selection on body size. A subset of the hypothetical parents actually survive and produce offspring. These surviving parents are indicated with solid dots. The strength of selection on body size (horizontal arrow) can be visualized as the distance between the average body size of all potential parents (vertical dotted line) and the average body size of the actual parents (vertical solid line). The direct response to selection is a function of the strength of selection and the heritability of the trait.

the vertical arrow. This change in the body size of the next generation is known as the "direct response to selection" on body size. If there were no heritability of body size, the slope of the best-fit line would be zero, and there could be no response to selection.

Plots assaying offspring-parental resemblance can also be used to visualize genetic coupling between characters. Figure 9.3 shows the body size of progeny plotted against a second hypothetical trait, size of brown adipose pads, of parents. Trends in such plots reflect genetic coupling if common environmental effects are absent or have been eliminated by design. In Figure 9.3, positive genetic effects on body size tend to be coupled with negative genetic effects on size of adipose pad, resulting in an overall negative trend in the data (more precise genetic meanings of coupling will be discussed later). The slope of the best-fit line through the data reflects covariance between genetic values for the two traits or "genetic covariance." In Figure 9.3, the genetic values for adipose pad size are expressed by parents whereas genetic values for body size are expressed in offspring. The "genetic correlation" between

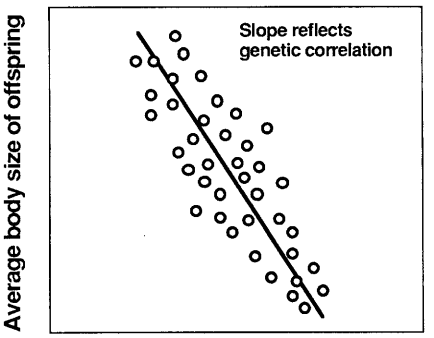

FIGURE 9.3 (Left) Average body size
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FIGURE 9.4 (Right) Correlated respo
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Quantitative genetics has been an import
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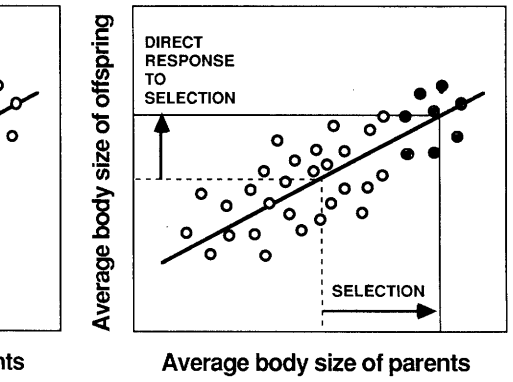

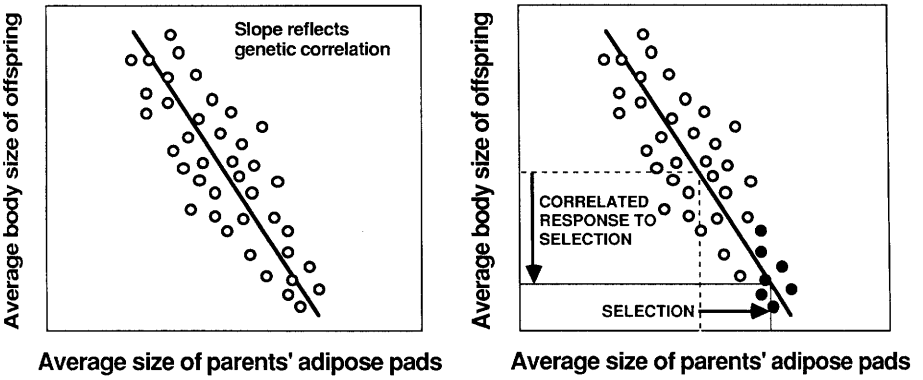

FIGURE 9.3 (Left) Average body size of offspring plotted against a second hypothetical character of their parents, size of brown adipose pads. The trend in the data reflects genetic coupling or genetic correlation between the two traits. In this case the genetic correlation is negative.

FIGURE 9.4 (Right) Correlated response of body size to selection on adipose pads. Actual parents of the next generation are indicated with solid dots. Selection favoring large adipose pads (horizontal arrow) causes a decrease in the body size of offspring because of the negative genetic correlation between the two traits.

two traits is simply a standardized genetic covariance that varies within the limits -1 to $+1$. In Figure 9.3, the genetic covariance is negative.

As a consequence of genetic covariance, selection on one trait will cause evolution in other traits. In Figure 9.4 we have the same offspring-parent data as in Figure 9.3. Imagine that selection is imposed on adipose pads, so that only the parents in the upper 20% of pad sizes survive and produce offspring. Because of the negative genetic correlation, these selected parents will yield progeny with smaller body sizes (solid horizontal line) than the average of potential parents (their expected progeny is shown with a dotted horizontal line). The change in body size in the next generation due to selection on adipose pads is referred to as a "correlated response to selection" on adipose pads.

Quantitative genetics has been an important conceptual framework for plant and animal breeders. Quantitative genetics has been useful because it enables the breeder to summarize the inheritance of phenotypic traits and to predict how those traits will respond to selection. For these same reasons, quantitative genetics can be useful to the physiologist who is interested in the evolution of physiology. The phenotypic resemblance between offspring and parents, as described in Figures 9.1 and 9.3, is only a rough summary of the underlying genetic system, but it captures the essential information

needed to predict the consequences of selection. In recent years, quantitative genetics has been increasingly used by evolutionary theorists. The main thrust of this work has been to see how multiple phenotypic traits will respond to long-term patterns of selection (Lande, 1976, 1979, 1980a).

The importance of genetic correlations for evolution has been revealed by applied and theoretical lines of inquiry. Breeders and experimentalists have had to take genetic correlations into account time and time again in trying to interpret the results of deliberate selection (Dickerson, 1955; Robertson, 1980; Falconer, 1981). In addition, recent theoretical models have shown that genetic correlations can have major, sometimes nonintuitive effects on the course of evolution (e.g., Lande, 1979, 1980a). The essential point, revealed by both approaches, is that genetically coupled traits will not evolve independently. Thus to understand the evolution of physiological systems, we must consider the possibility of genetic coupling between elements in the system.

Sources of genetic correlation

The genetic coupling between traits, described by a genetic correlation, can arise from two sources: pleiotropy and linkage disequilibrium (Falconer, 1981). A single gene may affect many traits in an organism, a phenomenon called "pleiotropy." Two traits may be genetically correlated because of the summed action of many genes with pleiotropic effects. The contributions of alleles at one locus to a genetic correlation are shown in Figure 9.5. The ubiquity of multiple effects by single mutations suggests (Figure 9.5) that pleiotropy is the rule rather than an exceptional mode of gene action (Wright, 1968). "Linkage disequilibrium" refers to correlation between the allelic effects of different loci (Crow and Kimura, 1970). So, for example, a negative genetic correlation between body size and fat pad size might arise because alleles at one locus increase body size and those alleles occur most frequently in conjunction with alleles at a second locus that tend to decrease size of fat pads (Figure 9.6). The contributions of pleiotropy and linkage disequilibrium to a genetic correlation can be separated with an elaborate series of crosses. Both such crosses and computer simulations indicate that pleiotropy, rather than linkage disequilibrium, is usually the major determinant of genetic correlation (Bulmer, 1974). In routine work in quantitative genetics, where the main issue is response to selection, the two sources of correlation in genetic effects are simply lumped together and summarized as a genetic correlation.

Phenotypic correlations versus genetic correlations

If we measure two traits, say body size and size of brown adipose pads, in each individual in a population, we could readily compute the correlation between the traits. Such similar correlations, based on individual values

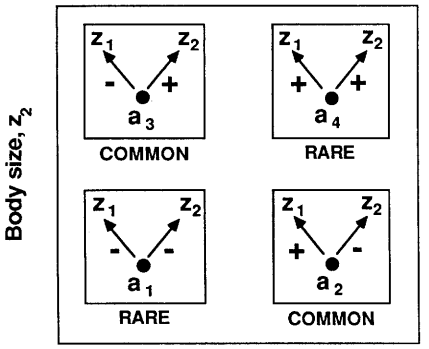

FIGURE 9.5 (Left) Genetic correlatio
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size, denoted z_2. Two of the alleles, a_2
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FIGURE 9.6 (Right) Genetic correlati
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effects on body size. The a_1 allele tend
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a_2b_2) are rare, a negative genetic corre

within a generation, are known as "phenotyp
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Detecting and measuring genetic correlatio

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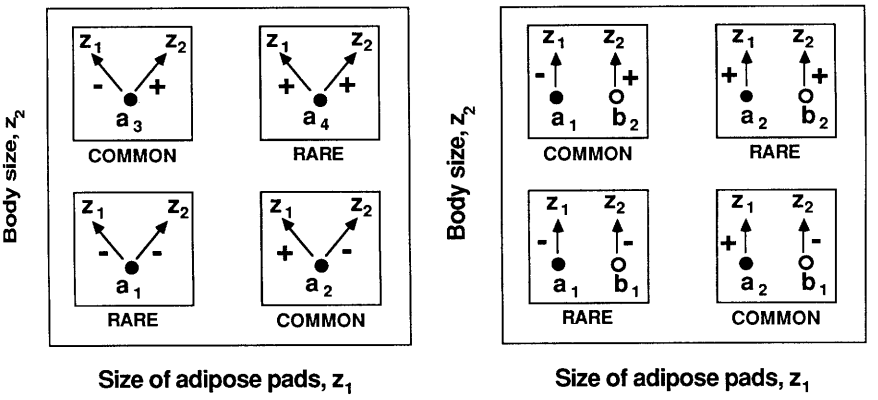

FIGURE 9.5 (Left) Genetic correlation due to pleiotropy. Four alleles at a hypothetical locus exist in a population and are denoted a_1, a_2, a_3, and a_4. Each affects both size of adipose pads, denoted z_1, and body size, denoted z_2. Two of the alleles, a_2 and a_3, are common in the population. Because these alleles have effects of different sign on the two traits, they produce a negative genetic correlation between size of adipose pads and body size.

FIGURE 9.6 (Right) Genetic correlation due to linkage disequilibrium. Two alleles at a particular hypothetical locus have different effects on body size. The a_1 allele tends to decrease size of adipose pads while the a_2 allele tends to increase size of adipose pads. Two alleles at a second hypothetical locus have contrasting effects on body size. If genotypes with contrasting signs (a_1b_2 and a_2b_1) are common in the population while genotypes with complementary signs (a_1b_1 and a_2b_2) are rare, a negative genetic correlation will be produced.

within a generation, are known as "phenotypic correlations." Why couldn't we use phenotypic correlations to assess the degree of genetic coupling between traits? The answer is that phenotypic correlations reflect both genetic and environmental effects on the traits. In extreme cases the genetic and environmental effects may be opposite in sign (Falconer, 1981). For example, mice with more access to food may be both larger in size and have larger adipose pads for nutritional reasons. If such nutritional effects are large, the phenotypic correlation between the traits may be positive even though the genetic correlation is negative.

Detecting and measuring genetic correlations

Genetic correlation can be assessed by assays of phenotypic resemblance among known relatives or by selection experiments. For example, we could measure one trait in parents, measure the other trait in offspring, and assess their correlation (Figure 9.3). This procedure will work so long as parents

and offspring do not share environmental features that lead to nongenetic correlation of the characters. Alternatively, offspring can be reared apart from parents, so that environmental causes of offspring-parent resemblance are disrupted. Many other combinations of relatives can give comparable genetic information [e.g., sets of progeny, each set with the same mother and father (full sibs) or with the same father but different mothers (parental half-sibs)]. A large part of the literature in quantitative genetics is devoted to the problem of devising experimental breeding plans that give efficient and reliable estimates of genetic variances and covariances (Cockerham, 1963; Falconer, 1981; Becker, 1984).

Deliberate selection on experimental populations can also reveal genetic correlation and assess its magnitude (Robertson, 1980; Falconer, 1981). The basic procedure is to select on one trait, usually for a few to many generations, and see if other traits respond to that selection. Such experiments can produce correlated responses to selection and give dramatic evidence for genetic coupling. For example, the practical question of whether selection for rapid growth in domestic animals is likely to have deleterious effects on reproduction has been investigated with selection experiments (e.g., Bradford, Barkley, and Spearow, 1980). Often selection experiments include unselected control lines, so that random or systematic effects of the environment can be disentangled from response to selection. A selected line gives only a single estimate of genetic correlation so replicate selection lines are needed in order to estimate standard errors (Hill, 1980).

Varieties of genetic correlation

The aim of the following survey is to suggest some physiological situations in which genetic correlation will have interesting evolutionary consequences, rather than to attempt an inventory of quantitative genetic studies of physiology. Thus a common feature in the examples that follow is the possibility of antagonistic responses to selection, which arise when selection acts on genetically coupled traits and produces conflicting results. In most of the examples, I will consider homologous traits that are likely to show positive genetic correlation due to pleiotropy. For example, body size at one age may be genetically the same character as body size at a later age, and consequently the two traits may show a large positive genetic correlation. Antagonism in adaptation can arise when selection acts in different directions on the two traits, for example, when large size is favored at one age and small size at another age. Likewise, antagonism can occur when selection acts in the same direction on two traits that have a negative genetic correlation (Figures 9.2 and 9.4).

Correlation between characters expressed a

An important generalization, emerging from
phology, is that homologous traits, traits in c
that are functionally linked, often show posit
sometimes they show positive genetic correla
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Sometimes unexpected genetic correlatio
behavioral example. Chemoreception is a m
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and fish). Litters from the same population d
moreceptive responses to virtually all of these
a consequence of heritable differences becaus
the tongue-flicking responses of newborn sn
mother's diet during gestation. Arnold (1981b
between responses to different prey by analy
scores of litters. Not surprisingly, responses
showed positive genetic correlations. Litters t
treefrog odor also showed strong responses
correlations, however, were unexpected. In
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Correlation between characters expressed a

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Correlation between characters expressed at the same age

An important generalization, emerging from studies of correlation in morphology, is that homologous traits, traits in close spatial proximity and traits that are functionally linked, often show positive phenotypic correlations and sometimes they show positive genetic correlations as well (Olson and Miller, 1958; Lande, 1980b; Cheverud, 1982). Because so little quantitative genetic work has been done with physiology, we do not have comparable generalizations for physiological traits.

Sometimes unexpected genetic correlations occur, as in the following behavioral example. Chemoreception is a major sensory modality used by garter snakes to recognize prey species. Even naive, newborn garter snakes will give an active tongue-flicking response to cotton-tipped swabs that are laden with prey odors. Sometimes such swabs elicit prey attack. Arnold (1981a, 1981b) tested the tongue-flicking response of newborn snakes to the odors of several natural prey species (slugs, leeches, salamanders, anurans, and fish). Litters from the same population differ significantly in their chemoreceptive responses to virtually all of these prey. This variation is probably a consequence of heritable differences because Burghardt (1971) showed that the tongue-flicking responses of newborn snakes are not influenced by the mother's diet during gestation. Arnold (1981b) estimated genetic correlations between responses to different prey by analyzing covariation in the average scores of litters. Not surprisingly, responses to different species of anurans showed positive genetic correlations. Litters that showed strong responses to treefrog odor also showed strong responses to tadpole odor. Some genetic correlations, however, were unexpected. In particular, litters that reacted strongly to slug odor also reacted strongly to leech odor. The genetic correlation between responses to these unrelated prey was very high (nearly .9) and may reflect the sharing of a critical chemical cue. Thus suppose that a chemical substance on the surface of slugs triggers tongue-flicking and prey attack. If there is heritable variation in responsiveness to that substance and if the same substance is found on leeches, we would expect a genetic correlation between chemoreceptive responses to these two prey.

Correlation between characters expressed at different ages

Many studies have estimated genetic correlations between the same morphometric character measured at different ages. Generally, genetic correlations are higher when the ages are close together (e.g., Leamy and Cheverud, 1984). For example, in a laboratory rat population, the genetic correlation between mass at two and four weeks of age is nearly 1, but between the ages of two and forty-seven weeks it has fallen to .4 (Cheverud, Rutledge, and Atchley, 1983).

Another prospect is that one type of character expressed at one age may be correlated with another type of character expressed at a different age. For

example, in holometabolous insects, a larval trait might be genetically correlated with a phenotypically dissimilar trait in adults (e.g., if the traits share developmental or metabolic pathways so that pleiotropy gives rise to genetic correlation). For example, Palmer and Dingle (1986) detected a positive genetic correlation between adult wing length and developmental time by selecting on wing length in an hemipteran. By implication, then, evolution of increased flight performance through selection for longer wings might frustrate the evolution of a short larval period in this species.

Correlation between characters expressed in males and females

Homologous traits in males and females often show high genetic correlation. For example, the correlation between body size in males and females may be .9 or higher in birds, mammals, and *Drosophila* (Lande, 1980a). (All the studies to date have been done on species that show only modest sexual dimorphism in size.) The result is not surprising when we realize that new mutations will tend to give comparable effects in males and females. Consequently, when selection acts differently on the sexes, the possibility for antagonistic responses to selection is very great.

Sexual dimorphism is an indication that selection acts differently in males and females (Darwin, 1874). For example, in most anurans, males actively call to attract mates, while females are silent and rarely vocalize. Recent studies have shown that male vocalization is energetically expensive, resulting in as much as a twenty-five-fold elevation of metabolic rate (Bucher, Ryan, and Bartholomew, 1982; Taigen and Wells, 1985). In a hylid frog, this sexual difference in energy expenditure is paralleled by a striking sexual dimorphism in size of trunk musculature and in activity levels of oxidative enzymes (Taigen, Wells, and Marsh, 1985). Citrate synthase activity in male trunk musculature is seventeen times greater than in females. Recent theoretical models suggest that positive genetic correlation between the sexes in trunk musculature and in citrate synthase activity could have delayed the evolution of sexual dimorphism in these traits. Those models provide a genetical perspective on the evolution of sexual differences in physiology and are discussed in the next section.

Correlation between characters expressed in different environments

Some of the genetic difficulties that thwart adaptation to alternative environments can be analyzed by measuring genetic correlation between traits expressed in different environments (Falconer, 1952, 1981; Robertson, 1959; Via and Lande, 1985). The simple step of defining a trait, say milk yield, in two environments as separate characters has helped to solve practical problems in animal breeding, and it opens new ecological vistas as well. Suppose we select for increased milk yield in dairy cows maintained on good pastures and succeed in improving the breed in this environment. Will the breed show

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Genetic correlation between traits in altern
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Correlation between direct genetic and mater

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improved milk yield when also maintained on a poor pasture or will their milk yield be worse than that of the original stock? This question can be approached by asking whether there is a positive or a negative genetic correlation between milk yield in the two environments. A high positive correlation indicates that the same genes promote milk yield in both nutritional environments, while a negative correlation indicates that selective improvement in one environment may cause a genetic loss in milk yield in the other environment.

Genetic correlation between traits in alternate environments is a current issue in studies of how arthropods are able to adapt to each of several host plant species (Via, 1984, 1986; Futuyma and Peterson, 1985; Lofdahl, 1985). Genetic correlation between arthropod performance on different host plants has been investigated by selection experiments and by analysis of covariation within populations. Gould (1979) raised herbivorous mites for many generations on two separate host plant species: one that yielded good mite growth and one that was mite-resistant. The line on the mite-resistant host improved in performance (developmental rate and survivorship) over a twenty-one-month period. However, by transferring mites back to the permissive host, Gould found that its performance there had slightly deteriorated. The result suggests a negative genetic correlation between performance on the two hosts. In contrast, Rausher (1984) did not find a negative genetic correlation between host plant performances within tortoise beetle populations. A large number of such case studies will be needed before we will know whether genetic trade-offs are important in the evolution of host plant specialization in arthropods.

Correlation between direct genetic and maternal genetic effects

In many organisms with maternal care, the behavior and physiology of the mother can profoundly affect the offspring's phenotype. Milk supply or feeding at the nest affects offspring growth, for example. Because growth rate is of great economic importance in domestic mammals, much experimental and theoretical work has focused on the genetic analysis of maternal effects (Dickerson, 1947; Willham, 1963, 1972; Falconer, 1965; Cheverud, 1984a; Riska, Rutledge, and Atchley, 1985). The resulting concepts and findings are directly applicable to the problem of analyzing the evolution of a physiological trait that experiences or produces a maternal effect.

The critical first step is to realize that the maternal effect itself may have a genetic basis. Thus milk supply may be heritable to some degree. Consequently, when we consider offspring growth rate, we can imagine two classes of genetic effects: direct effects due to genes that act in the zygote and affect its growth, and maternal effects that act in the mother and affect the offspring's growth by changing the offspring's maternal environment. We also have the possibility that the two kinds of genetic effects, direct and maternal,

might be correlated. Experimental work with mammals, for example, has shown that such correlations exist. Furthermore, when the correlation between direct and maternal genetic effects is positive, selection on maternal performance can amplify the effects of selection on offspring growth. However, when the correlation is negative, the evolution of maternal performance may be antagonistic to the evolution of offspring growth, and vice versa.

Negative correlation between direct genetic and maternal genetic effects on body mass and growth have been found in both mice and cattle (Riska et al., 1985). In some cases the negative correlation may result from a trade-off between offspring size and offspring numbers. Thus, larger female mice tend to have larger litters but smaller offspring (Falconer, 1965). Because of the trade-off between offspring size and numbers, a genetic tendency for larger size will be associated with a negative pleiotropic effect on offspring size (mediated through the maternal effect of body size on litter size and hence on offspring size). Antagonism between the two kinds of genetic effects cannot be mediated through litter size in all cases, however, because the negative genetic correlation has been found in some cases even when litter sizes were held constant (Riska et al., 1985).

Correlation between direct genetic and maternal genetic effects is conceivable in many circumstances and could have an important impact on the evolution of maternal (or paternal) care. For example, in viviparous natricine snakes, body temperature during pregnancy affects the scalation and number of vertebrae of developing offspring (Fox, 1948; Fox, Gordon, and Fox, 1961; Osgood, 1978). A quantitative genetic perspective on maternal effects raises the issues of (1) whether gravid females show phenotypic and genetic variation in thermoregulation, (2) whether natural variation in maternal thermoregulation (contrasted with the dramatic temperature differences imposed in perturbation experiments) affects offspring thermoregulation and vertebral numbers, and (3) whether there is a genetic coupling between maternal thermoregulation and number of vertebrae. A correlation between direct genetic effects on thermoregulation and maternal genetic effects on number of vertebrae would cause selection on vertebral numbers to affect the evolution of thermoregulation. Conversely, selection on thermoregulation would affect the evolution of number of vertebrae.

Carey (1980) has stressed the need for studies of how wide-ranging bird species meet the challenge of greater water loss through the eggshell at higher elevations. Within species, water loss appears to be buffered against the effects of altitude. Carey proposed four kinds of maternal effects as candidates for adaptations to elevation: (1) a change in porosity of the shell, which reduces water vapor conductance through the eggshell; (2) an increase in initial water content; (3) an increase in shell thickness; and (4) parental behavior that changes the egg microenvironment and thereby reduces water loss. Phenotypic effects of the zygotic genome might also affect water loss.

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Evolutionary consequences of genetic cor

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Antagonism between direct and correlated

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For example, properties of the chorioallantoic membranes, which are constructed by the embryo, might affect water loss. Returning to our theme of genetic correlation, we can ask whether direct genetic effects on embryonic membranes are correlated with maternal genetic effects on the eggshell and its environment. With positive correlation, maternal adaptation might reinforce zygotic adjustments to high elevation, but with negative correlations, maternal and zygotic adjustments might work against each another and delay adaptation.

Maternal effects complicate the detection and measurement of genetic variances and covariances. The complication arises because we are interested in genetic effects that act at two stages of the life cycle (in offspring and in mothers). Consequently, to analyze the inheritance of maternal effects one needs a breeding design that produces mothers of known relationship as well as offspring of known relationship (Eisen, 1967; Rutledge, Robinson, Eisen, and Legates, 1972).

Evolutionary consequences of genetic correlation

The major detrimental effect of genetic correlation is to delay adaptation. Departures from evolutionary optima caused by genetic correlation may be temporary, but they may last for hundreds of thousands of generations. To understand these effects, we need a more precise concept of adaptive antagonism.

Antagonism between direct and correlated responses to selection

When two traits are genetically coupled, selection on one trait may interfere with the adaptive evolution of the other trait. We can readily see how such antagonism can arise by referring to the standard quantitative genetic equations for one generation of response to selection. By convention, z refers to the value of a trait (e.g., thermal tolerance, wing length, citrate synthase activity) in a population; $\bar{z}$ is the average value of the trait in the population, and $\Delta\bar{z}$ is the change in the average value of the trait from one generation to the next, due to natural selection. If selection acts only on a single trait then

$$\Delta\bar{z} = G\beta \tag{9.1}$$

where G is the genetic variance of the trait and β represents the magnitude of natural selection (Falconer, 1981). In other words, the amount of change in a trait due to selection is proportional to the product of *both* the strength of selection *and* the genetic variance. Equation 9.1 is but a mathematical representation of directional natural selection (e.g., Figure 9.2), with which ecological physiologists are familiar. Of course, traits may not behave independently. Thus, if we consider the values of any two correlated traits, z_1 and

z_2, with their respective genetic variances (G_{11} and G_{22}) and selection coefficients (β_1 and β_2):

$$\Delta\bar{z}_1 = G_{11}\beta_1 + G_{12}\beta_2 \quad (9.2)$$
$$\Delta\bar{z}_2 = G_{22}\beta_2 + G_{12}\beta_1 \quad (9.3)$$

where G_{12} is the "genetic covariance" between traits 1 and 2 (Lande, 1979). The general implication of these equations is that the evolutionary change in a trait is the sum of two components. The first is the "direct response to selection" (e.g., $G_{11}\beta_1$ for trait 1), which gives the shift in the mean of z_1 due to selection acting directly on that trait. The second component, termed the "correlated response to selection" (e.g., $G_{12}\beta_2$ for trait 1), is the shift in the mean of z_1 due to selection acting on the correlated character, z_2. Likewise, the response to selection by z_2 is composed of a direct response ($G_{22}\beta_2$) and a correlated response ($G_{12}\beta_1$).

Importantly, depending on the relative magnitude and sign of the direct response and the correlated response, the correlated response may reduce the direct response to natural selection or override it entirely. In either of these situations, the population is in a kind of genetic treadmill, with part or all of the genetic advance in each character being canceled in each generation by selection acting through the correlated character. For example, the direct response to selection on body size (Figure 9.2) is overridden by the correlated response of body size to selection on adipose pads (Figure 9.4). Consequently, a smaller body size evolves even though selection favors large body size.

We have few examples of antagonism between physiological features of animals, for such phenomena have rarely been studied. However, by turning to breeders' attempts to select artificially for multiple traits in agricultural species, we can readily appreciate how important antagonism might be in affecting the outcome of natural physiological adaptation. In some cases, repeated artificial selection fails to alter a trait after an initial large change, not because the genetic variance is small, but because of negative genetic correlations between the traits. In chickens, for example, a negative genetic correlation between egg production and body size frustrated attempts to produce chickens that were both large and laid many eggs (Gyles, Dickerson, Kinder, and Kempster, 1955). Such genetic trade-offs are common in crop species, and Dickerson (1955) has referred to the resulting cancellation of direct and correlated responses as "genetic slippage." Dickerson anticipated current interest in genetic trade-offs by students of life-history evolution by pointing out that deliberate selection for multiple objectives bears many similarities to selection on major fitness components.

Genetic slippage can also arise from antagonistic selection. In the garter snake example described earlier, newborn snakes from two populations were tested for chemoreceptive responses to slugs and leeches (Arnold, 1981a, 1981b). In a population allopatric with slugs, naive newborn snakes hardly react to slug or leech odors, even though leec
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Counterintuitive evolutionary trajectories and maladaptation

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react to slug or leech odors, even though leeches are a minor constituent of the natural diet (the principal prey are fish and anurans). In a population sympatric with slugs, naive newborn snakes give strong feeding reactions not only to slugs, the principal natural prey, but also to leeches, even though leeches are never encountered in nature. The enigmatic reaction of these slug-sympatric snakes to leeches might represent a correlated response to selection for slug recognition. I tested this supposition using the data reported in Arnold (1981a, 1981b) and equations given in Lande (1979). The results indicate that the reactions of slug-sympatric snakes to leeches, while strong, are not strong enough to be simply a correlated response. To account for the population difference in reaction to the two prey, and taking into account the strong positive genetic correlation in both populations, I needed to invoke both selection for slug reaction and selection against leech reaction. The results indicated that antagonistic selection on positively coupled traits might be responsible for the geographic differences.

Counterintuitive evolutionary trajectories and temporary maladaptation

In the preceding sections, we considered the possibilities that direct and correlated responses to selection might cancel so that no evolution occurs even though selection favors improvement. We also considered a case, in garter snakes, in which the population seemed to have evolved in the opposite direction to selection on one of two traits. More extreme possibilities exist. It is conceivable that negative genetic correlations could cause evolution in the opposite direction to the force of selection on both traits. Cheverud (1984a) shows that this nonintuitive result is possible if there is a strong negative genetic correlation between direct genetic and maternal genetic effects. For instance, let us return to the earlier example of thermoregulating female snakes that affect the number of vertebrae in their embryos. Even if selection favored a higher body temperature for thermoregulating mothers as well as more vertebrae in their embryos, a sufficiently large negative genetic correlation could cause the evolution of lower maternal temperatures and fewer vertebrae. Such bizarre evolution is possible in the short run, but we need a model of long-term response to selection, as well as single generation equations, to predict ultimate outcomes.

Lande's (1979, 1980a) models for the evolution of genetically coupled traits make some predictions about the time-course and direction of long-term evolution. The general result from Lande's models is that genetic correlation tends to delay adaptation (adaptation being conceived as proximity to the hilltop on the adaptive landscape). Those models examine the effects of genetic correlation on the evolution of trait averages by holding the genetic variations and covariance(s) constant. This assumption of constancy is not outrageous because both theoretical and empirical studies indicate that the

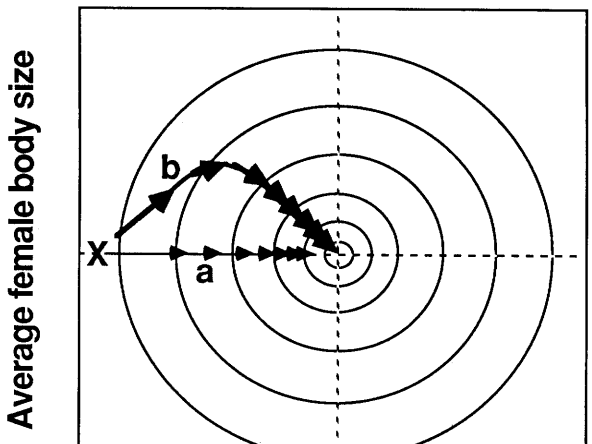

FIGURE 9.7 The evolution of two genetically coupled traits, body size in females and males. A population with a bivariate mean at the position shown by the X (middle left) experiences a changed environment that favors the same female body size but a larger male body size. The new adaptive landscape is represented by the concentric circles which are contours of average fitness in the population. The fitness maximum is located at the center of the concentric circles, and the dotted lines show optimal female and male body sizes. If there is no genetic correlation, the population will evolve along the straight path (a). A strong, positive genetic correlation will cause the population to evolve along the curved trajectory. Arrowheads are drawn at intervals of a few hundred generations. (After Lande 1980a.)

trait means evolve on a much faster time scale than genetic variances and covariances (Lande, 1976). The models treat the common situation in which selection favors some intermediate optimum for each of the two characters. Thus the population is visualized as evolving on a hill-shaped surface in a space whose two horizontal axes are the population averages for the two evolving traits, with average population fitness as the vertical dimension. Mean fitness in the population is at a maximum at the hilltop, and, it can be shown, the population tends to evolve toward that hilltop (Figure 9.7).

Delay in adaptation due to genetic correlation can be appreciated by considering the evolution of sexual dimorphism. Consider the case of body sizes of the two sexes, homologous traits that show high genetic correlation between males and females. Suppose the environment changes so that now

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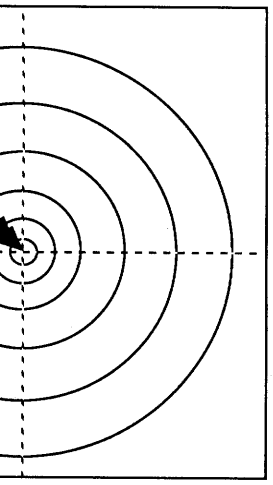

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selection favors a larger body size in males but the original female body size is still at its optimum (Figure 9.7). In the absence of genetic correlation (and with comparable genetic variances in the two sexes), average body size in the sexes would evolve directly toward the new adaptive peak. That is, average female size would stay constant and average male size would increase. Genetic correlation causes a curved evolutionary trajectory. Initially the population evolves rapidly in the direction of increased male and female body size, for in that direction the population possesses the most genetic variance. During this rapid phase, the females may evolve above optimal body size as the males continually evolve larger, always toward their optimum. The maladaptive evolution of the females toward a larger than optimal size is due to a correlated response. The correlated response to selection for larger males temporarily overwhelms the direct response to selection for females, which would otherwise tend to maintain females at their original body size. Finally, a slow process of sexual differentiation ensues during which males gradually increase in size and females slowly decrease in average size. This differentiation is slow since there is little genetic variability in the direction of sexual dimorphism because of the strong genetic coupling between the sexes (Lande, 1980a). The second phase of sexual differentiation could last tens or even hundreds of thousands of generations (Lande, 1980a). Throughout that phase the average female is larger than the optimum. Thus the effect of genetic correlation is to cause the evolutionary trajectory to curve and to delay adaptation. Dissimilar genetic variances in the two traits can have comparable effects (Leutenegger and Cheverud, 1985).

The important message is that the delay in adaptation may be exceedingly long. Consequently, when considering physiological traits that are likely to be genetically coupled, we must seriously entertain the possibility that our study population has excessive or deficient average trait values. Genetic correlation can cause long-lasting disharmony between the population and its environment.

Populations adapting to two different environments may also experience curved evolutionary trajectories and temporary maladaptation due to genetic correlation. Via and Lande (1985) model the situation in which individuals reside in one of two environments throughout their lives and experience corresponding differences in selective pressures. Adaptation can be much delayed if traits that confer success in the two environments are genetically coupled. In particular, if one of the environments is common (e.g., one host plant of a sedentary, herbivorous insect), adaptation to that environment may dominate the first phase of evolution. During that phase, the population may evolve traits deleterious to success in the second environment (e.g., a second host plant species), because correlated responses to selection in the common environment swamp direct responses to selection in the rare environment. Thereafter, a slow process of adaptation to the rare environment may ensue.

Even when the two environments are equally common, genetic correlation can deflect the course of evolution and promote temporary maladaptation.

Long-term evolutionary solutions

Genetic correlation may cause temporary maladaptation, but other traits may ameliorate or even remove the constraints imposed by genetic coupling. Some long-term solutions to the examples of genetic constraint that we previously discussed include (1) the evolution of age-specific or sex-limited expression of traits, and (2) the evolution of the ability to discriminate among hosts or prey that differ in their fitness consequences for the predator. Such mechanisms may eventually lessen the impact of genetic correlation or even eliminate its effects. Nevertheless, specificity in expression and modification of behavior and physiology do not evolve overnight. As new ensembles of traits evolve to meet new environmental challenges, the population will repeatedly confront the constraints and consequences of genetic coupling. Modifications that help retrieve the population from the slow phase of adaptation imposed by genetic coupling may themselves consist of genetically correlated traits and so will experience their own slow evolutionary phase.

The evolution of genetic correlation

In the preceding discussions we took genetic correlation as a given and examined its evolutionary consequences. Yet, genetic correlations themselves evolve, so the constraints they impose are unlikely to last forever, a point sometimes overlooked in arguments for the ever-lasting power of developmental constraints. Thus Charlesworth, Lande, and Slatkin (1982) refute S. Gould's (1980) and Alberch's (1980) arguments that developmental constraints can impose long-term evolutionary stasis by pointing to the success of selection experiments in moving population averages of numerous traits far beyond the limits of variation in the initial population. In addition, the long-term effect of selection may be to change the pattern of genetic and developmental constraints (Schmalhausen, 1949; Waddington, 1957; Cheverud, 1984b).

Genetic models indicate that selection can shape genetic correlations. The pattern of genetic correlation (sign and magnitude) represents a balance between selection, mutation, migration, and other factors (Lande, 1976, 1980b, 1984; Turelli, 1985). Because so many factors influence the evolution of genetic correlations, generalizations about when genetic correlations are likely to be weak or strong, positive or negative, will probably emerge from empirical rather than from theoretical work.

Unexplored genetic issues in ecophysiology

There is a great need for quantitative studies o
edge of the heritabilities and genetic correlat
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Heritability is an important issue confronting
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Most geneticists would expect physiological tra
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Schmalhausen, 1949; Waddington, 1957; Chev-

hat selection can shape genetic correlations. The
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Because so many factors influence the evolution
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positive or negative, will probably emerge from
heoretical work.

Unexplored genetic issues in ecophysiology

There is a great need for quantitative studies of physiological traits. Knowledge of the heritabilities and genetic correlations of physiological traits is critical to our understanding of the evolution of physiology, yet we have extremely few estimates for natural populations. Most of the relevant past work has been focused on applied problems or basic genetic issues other than the evolution of physiology in nature. Thus when quantitative work has included physiological traits, it usually has had the goal of improving commercial livestock breeds rather than elucidating evolutionary processes. Much of the most sophisticated empirical work in quantitative genetics has been designed to explore basic genetic issues (e.g., how does the heritability of a trait change as a function of the age when the trait is measured?). To accomplish such genetic goals, arbitrary traits are often used (e.g., bristle numbers and wing variation in *Drosophila*). As a consequence, the results seem far from concerns of the ecophysiologist. The perceived distance is an illusion. Much can be learned from the genetic literature about conceptualizing and executing genetic studies of physiology. At the same time, studies of physiology can bring a much needed ecological and evolutionary focus to quantitative genetics.

Physiology is an attractive field for quantitative genetic work for a number of reasons. First, the functional significance of many traits is well understood. The traits are not mere markers of an underlying genetic system, but play specific, known roles in adaptive processes. Second, comparative work is often available and can reveal which traits are evolutionarily liable. Third, many physiologists work with animals sampled directly from nature so that specific natural reference populations can be established for genetic work. In combination, these attributes mean that by studying the inheritance of evolutionarily liable physiological traits we can assess the joint roles of selection and inheritance in pursuing evolutionary problems. In other words, the genetic results can be placed in a specific ecological and evolutionary framework.

Heritability is an important issue confronting the physiologist. Because so little work has been done, heritability remains an active, unresolved issue. Most geneticists would expect physiological traits to show heritable variation in a natural population, but this expectation is merely an educated guess derived from more extensive work on morphological traits. For physiological traits there is virtually no data base, so we do not know whether genetic material for natural selection to act on is abundant or sparse. The extensive experience of animal breeders shows that heritabilities of linear dimensions (conformation) are often in the high range (70% or higher). Such traits respond rapidly to selection and are presumably capable of rapid evolution in nature. We have no comparable generalizations for physiology.

Genetic correlation is a second unresolved issue. We can expect many physiological traits to be genetically coupled because pleiotropic gene action is so common (Wright, 1968). Despite the fact that genetic correlation could play a major role in the evolution of physiological systems, the relevant correlations are a virtually unexplored field of study. For example, a trade-off between burst speed and capacity to sustain running performance might be a reasonable expectation because different, perhaps conflicting physiological systems might support these two kinds of performance. The genetic manifestation of the trade-off is a negative genetic correlation and thus is the parameter that will tell us whether there will be an evolutionary trade-off. The genetic basis of such trade-offs, revealed in genetic correlation, is unexplored.

Genetic correlations with potentially major evolutionary effects are particularly promising candidates for study. Thus high genetic correlations cause correspondingly large deflections of evolutionary trajectories (Figure 9.7). We have already reviewed several instances in which strong genetic coupling is likely: between traits at successive ages; between sexually homologous traits; between the same trait expressed in different environments. A further criterion for genetic correlations having major evolutionary effects is that of antagonism between direct and correlated responses to selection. Thus the genetic correlations just listed are likely to be both strong and positive. If selection favors, say, large trait values at one age but small values at the next oldest age, then trait differentiation will be much slowed because of antagonism in adaptation. In general, the genetic system will have a major impact on adaptation when genetic correlations and selective pressures differ in sign.

The minimal requirements for doing quantitative genetic work are replicated sets of known relatives from a specific population. The requirement of a specific, natural reference population can be easy to satisfy and is critical to interpreting the results. If animals are pooled from a variety of localities, the genetic parameters that are estimated may reflect a totally artificial population entity unlike any particular, local population in nature. The sampled animals should be randomly sampled from a local, interbreeding population to maximize the value of the results. Ability to breed the animals through a succession of generations is desirable but not necessary. For many difficult-to-breed animals it is nevertheless practical to collect series of gravid females and arrange for egg-laying or birth of broods under uniform laboratory conditions. Heritabilities and genetic correlations can be estimated for such animals by analyzing variation within and among broods or by regression of progeny averages on mothers' trait values (Falconer, 1981). Roughly speaking, dozens of families, each composed of several individuals, are needed if heritabilities and genetic correlations lie in the low range. The capacity to breed animals of known parentage permits more powerful estimation as well as selection experiments.

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In contemplating a first project in quantitative genetics, it may be reassuring to consult with a colleague who is already familiar with the formidable jargon and technical literature. The complexities of the field are more apparent than real. Likewise, consultation with a statistician may be useful in planning to estimate heritabilities and genetic correlations. How many sets of mothers and offspring will be needed? Falconer (1981) and Becker (1984) are extremely useful introductory texts.

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Discussion

POWERS: You said that the effect of temperature on vertebral number is an environmental factor and yet you are looking at its genetic component. Could you elaborate a little on why you are addressing it and how you are addressing it relative to environmental versus genetic factors?

ARNOLD: What I was showing is the reasonable prospect, on the basis of studies on other vertebrates, that there is a maternal effect of temperature on vertebral numbers in garter snakes. There are two extreme possibilities. One is that this maternal effect is completely nongenetic. Another is that there is some heritable component to the maternal effect, that is to say, that there is some heritable variation in temperatures that females are selecting during pregnancy. If that is the case, then vertebral number could have two heritable components: a component due to genes that affect vertebral numbers directly, and a second category of genes that affect maternal thermoregulation and influence indirectly the trait in question. So we have the possibility of a complicated genetic basis for vertebral numbers, and also the possibility of a genetic correlation between vertebral numbers and maternal performance. Then the two traits could evolve as a consequence of variation in genes affecting vertebral number directly, genes affecting maternal thermoregulation directly, and genes that have pleiotropic effects on both traits. When we consider the large number of traits that are influenced by maternal

performance or paternal performance, this k
general, particularly in viviparous vertebrates

FLORANT: If Siamese cats are kept at cold t
ears of the kittens are much darker than in c
the garter snakes. I can see why Siamese cats n
mother was in a cold environment, but I do
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ARNOLD: An issue that I did not even try to
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in so many species of teleost fish, for example.
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populations, we know there is stabilizing sel
that individuals that deviate in either directio
suffer a decrement in fitness. My hypothesis w
shaped curve in garter snakes, that zone of st
coincide with the stabilized mean of the verteb
is a hypothesis I am using to motivate the field

FUTUYMA: This is reminiscent of the theory o
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might be just a nonadaptive developmental ano

ARNOLD: Yes, that is possible.

FUTUYMA: It is a case where you would like
norm of reaction is an adaptation, or if the ends
a pathology of some kind.

ARNOLD: I am not dealing with the difficult p
reaction evolves. If we take that as a given for t
that thermoregulation in pregnant females mig
norm of reaction. Given that there is stabilizin
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on that zone of stability.

BENNETT: I would like to point out that the
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could evolve as a consequence of variation in
nber directly, genes affecting maternal thermo-
es that have pleiotropic effects on both traits.
number of traits that are influenced by maternal

performance or paternal performance, this kind of situation might be quite general, particularly in viviparous vertebrates.

FLORANT: If Siamese cats are kept at cold temperatures, the paws and the ears of the kittens are much darker than in controls. This seems parallel to the garter snakes. I can see why Siamese cats might want darker paws if their mother was in a cold environment, but I do not understand why a garter snake would want more vertebrae in a warmer environment.

ARNOLD: An issue that I did not even try to address was the evolution of the norm of reaction and why we might have a "U"-shaped norm of reaction in so many species of teleost fish, for example. There is a zone of stability: at the bottom of the "U," these populations can experience quite an excursion of temperatures and not see an impact on vertebral numbers. In garter snake populations, we know there is stabilizing selection on vertebral numbers: that individuals that deviate in either direction from the population mean suffer a decrement in fitness. My hypothesis would be that if there is a "U"-shaped curve in garter snakes, that zone of stability might have evolved to coincide with the stabilized mean of the vertebral number distribution. That is a hypothesis I am using to motivate the field and laboratory work.

FUTUYMA: This is reminiscent of the theory of canalization, the notion that there has been selection to mold a developmental system that will produce the "right" phenotype in the usual range of environmental conditions. The plateau at the bottom of this "U" could be the target that development is shooting toward, but the high vertebral number at an extreme temperature might be just a nonadaptive developmental anomaly.

ARNOLD: Yes, that is possible.

FUTUYMA: It is a case where you would like to know whether the entire norm of reaction is an adaptation, or if the ends of that norm of reaction are a pathology of some kind.

ARNOLD: I am not dealing with the difficult problem of how the norm of reaction evolves. If we take that as a given for the moment, we can imagine that thermoregulation in pregnant females might evolve in relation to that norm of reaction. Given that there is stabilizing selection on the vertebral numbers, there might be selection on maternal thermoregulation to move in on that zone of stability.

BENNETT: I would like to point out that the functional consequences of these morphological features can easily be tested. In the first pass, if one thinks of locomotion as being a function of vertebral number, it is easy to ask whether animals with more vertebrae locomote better in cold environments than in warm ones.

BARTHOLOMEW: Is what you are studying different from examining the nature of the fit between the maternal behavior of a mammal and the physiological state of the infant at birth?

ARNOLD: No, I think that you could use the same framework for viewing the evolution of maternal performance of any kind and target offspring phenotypes. In this case, this approach makes us look at thermoregulation in a perspective that I had not entertained before. I think the framework of quantitative genetics gives us an agenda of issues that will help us focus attention on different aspects of the problem.

FUTUYMA: A reaction norm is a geneticist's jargon for the form of the relationship between some feature of the organism and the environmental conditions in which the organism has been reared or kept for some time. A quantitative geneticist may approach this by taking, for example, performances at various temperatures as being in themselves characters, and ask how, from individual to individual, these characters are correlated with one another. If you plot different genotypes, would you find that they show different reaction norms? This could help you determine whether or not the entire reaction norm constitutes an adaptation. Do you interpret every part of the curve as being adaptive? Or do you assume that perhaps some of this curve is simply a correlated nonadaptive consequence of other parts of the curve?

BENNETT: Certainly the extremes of the curve are nonadaptive: if you freeze it or boil it, the system is going to come apart and those are not adaptations but structural consequences. When people do these studies, they try to phrase them in terms of natural environmental exposure and what the animal really experiences.

HUEY: Your question borders on an issue that we have not addressed, which is, if you get selection on one part of the performance curve, how does that affect other parts of the curve? If you get selection for higher tolerance, does that drag along the lower parts of the curve; or, if you get selection on the variance, how does that affect traits?

JACKSON: I think that it is going to depend on the type of function you are looking at. Some functions are simple reactions to change in temperature, such as heart rate. Upon those may be built the effect of temperature on other physiological control mechanisms that may affect heart rate, such as the autonomic nervous system. I think the issues of the direct effect of temperature, regulatory mechanisms existing within the animal, adaptations, and longer-term changes are all going to play a part.

DAWSON: There is in some cases a correlation between, for example, thermal preferenda and lethal temperature in lizards. Paul Licht's work showed that one set of enzymes, the myofibrillar ATPases, exhibit thermal differen-

tiation, but alkaline phosphatase had such a high that it did not show any diversification. It relat and how centrally they are involved, be nonpermissive.

BISHOP: We were working on a bivalve and we ten percent protein turnover. If the organism t acids and accumulated them, it could survive, an in the LAP [leucine aminopeptidase] activity. animals with genetically higher LAP activities the face of osmotic stress. However, because th they had a higher protein turnover activity wh actually selected against the animal. So when character, it depends upon all of the stresses tha

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tiation, but alkaline phosphatase had such a high thermal denaturation point that it did not show any diversification. It relates to specific characteristics and how centrally they are involved, being either permissive or nonpermissive.

BISHOP: We were working on a bivalve and we calculated that there is about ten percent protein turnover. If the organism trapped all of the free amino acids and accumulated them, it could survive, and you need really no increase in the LAP [leucine aminopeptidase] activity. Then Koehn found that the animals with genetically higher LAP activities would adapt more rapidly in the face of osmotic stress. However, because they had higher LAP activities, they had a higher protein turnover activity when they were starved, which actually selected against the animal. So when you talk about an adaptive character, it depends upon all of the stresses that the animals face.