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Genetic architecture of behavior in fishes and the response to selection

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A basic assumption of behavioral ecology is that the variation observed in behavioral traits is shaped by selection. The pattern and rate of response of a trait to selection depends on the presence of genetic variation, its mode of inheritance and genetic correlations with other traits. Quantitative genetic analysis provides a methodology to examine the genetic architecture of behavioral trait variation in natural populations.

Studies of fish behavior have documented substantial additive and non-additive genetic variation. Genetic architectures with important implications for patterns of response to selection such as sex-linkage and pleiotropy have been uncovered. Morphology and behavior are often inherited as a suite of traits which may share a common genetic basis. However, environmental factors can yield correlated variation in both morphology and behavior and experimental study separating the inheritance of each is necessary to determine if their genetic basis is held in common. Quantitative genetic analysis permits the prediction of microevolutionary patterns of local populations over limited time scales.

1. Introduction

Genetic variation in a form malleable to selection must be present for behavior to evolve (Cade 1984) and evolutionary models generally assume that such variation is readily available (O'Donald 1980, Dominey 1984). However, it is likely that the genetic system constrains patterns of evolution at times (Arnold 1987, Loeschcke 1987) and behavioral traits are not exempt from this possibility. There is ample evidence of genetic variation for behavioral traits in fish (Huntingford 1984, Noakes 1986) and here I focus on those aspects of the genetic system that influence the response of behavioral traits to selection. I will attempt to identify the genetic issues relevant to evaluating the potential for a response to selection, describe how they are studied, and present some examples from fishes illustrating how genetical study can illuminate evolutionary analyses.

2. Background

2.1. What is genetic architecture?

Genetic architecture is the mode of inheritance of a trait including the number of genes involved, the amount of additive genetic variation present, and the amount of non-additive genetic variation resulting from dominance, epistasis, and pleiotropy. Environmental factors before and after birth add to this complicated picture. It is the goal of quantitative genetics to decompose phenotypic variation into portions attributable to these factors. The relative importance of each of these factors in behavioral variation will determine the magnitude and rate that behavior can respond to selection.

The portion of the genetic architecture that responds most readily and permanently to selection is the additive genetic variation. Additive genetic variance is that genetic variance that arises from allelic substitutions whose effects are independent of genotype. It's magnitude is a function of the effect on the trait of each allele in combination with all other alleles and their relative frequencies (Hedrick 1983). The narrow sense heritability, which is used to predict the response to selection, is the ratio of this additive genetic variation to the total phenotypic variation (see also Hartl 1981: 151–155).

Non-additive genetic variation refers to the aspects of genetic architecture that do not respond readily to selection. For example, dominance variance is the genetic variance that results when the phenotypic value of a heterozygote is not intermediate between the two homozygotes at a given locus. Thus, dominance variance arises from allelic substitutions whose phenotypic effects are not independent of genotype at those loci. While dominance variance results from interactions of the different alleles of individual genes, epistasis describes the interaction among alleles at different genes. Epistasis is the portion of the genotypic variance that is dependent upon the genotypes of other genes. The broad sense heritability is the ratio of all additive, dominance and epistatic genetic variation (the total genetic variation) to the total phenotypic variation.

Genetic correlation is another aspect of genetic architecture that can influence patterns of inheritance and the response to selection. A genetic correlation between two or more traits can arise from pleiotropy or linkage. Pleiotropy arises when a single gene influences more than one trait while linkage occurs as a result of the non-random assortment of genes. Linkage is often considered a temporary cause of genetic correlation (Falconer 1981:281; but see Davies & Workman 1971). Genetic correlation may delay adaptation (Lande 1979, 1980) because the response to selection of one trait is dependent upon its genetic correlation to others that may be experiencing antagonistic selection pressures (Falconer 1981, Bulmer 1985, Arnold 1987).

Sex-linkage refers to genes located on the sex chromosomes and may have important implications in predicting a response to selection on some traits, especially if sexual dimorphism is involved. In the "textbook" case, the heterogametic sex is haploid at these loci while the homogametic sex remains diploid or lacks them altogether. This pattern of inheritance renders the heterogametic sex readily shaped by selection on a trait influenced by a single gene of major effect and not subject to recombination. A rapid response to selection is possible because such a gene can not be masked in a heterozygotic state in the heterogametic sex (Hedrick 1983). Patterns of transmission of such sex-linked genes must be studied separately between the sexes (Bulmer 1985) and epistasis with autosomal genes is common. In the case of a polygene on the X-chromosome, evolution is not accelerated relative to autosomes unless directional dominance is present (Charlesworth et al. 1987). Sex-linkage may be mimicked by autosomal genes with expression limited to one sex. However, these sex-limited traits follow the same patterns of inheritance and expected response to selection as autosomes.

In fishes, the inheritance of sex-linked genes does not necessarily conform to this "textbook" pattern. For example, in *Xiphophorus maculatus* some populations have three types of sex chromosome present, certain genes may be present on all of the sex chromosomes, and crossing over between sex chromosomes is known (Kallman 1984). Fishes display remarkable diversity in sex-determining mechanisms (Kirpichnikov 1981, Bull 1983) rendering it is unwise to assume the "textbook" pattern of inheritance in sex-linked traits.

For example, both male and female heterogamety has been noted even with both cases occurring in the same species (Kallman 1984).

Environmental conditions are seldom spatially or temporally constant in nature and can greatly alter the phenotypic expression of a genotype. Environmental effects can influence phenotypes before and after birth. Maternal environmental effects may be related to the mother's nutritional state or physical environment through variation in traits such as gestation period, egg size, or yolk quality. Maternal environmental effects may also be related to the mother's genotype. Accounting for shared maternal environment effects is especially difficult but this is a potentially significant source of correlation among siblings in some non-behavioral traits (DeFries et al. 1967, Riska et al. 1985). Environmental conditions can have marked effects on fish development in ways that may be genotype-dependent, so-called genotype-environment interactions (Trexler 1989). Heritability and genetic correlation may change dramatically across environments (e.g. Travis et al. 1990a).

2.2. Number of genes involved

A basic question in genetic architecture is whether a trait is the product of many genes with small (minor) individual effects or a single gene or few genes with major effects that may also be influenced by other genes with smaller effects (Falconer 1981). Without excruciating study of the segregation of phenotypes it is not possible to estimate with precision the number of genes involved in variation of a trait. An impression can be obtained by examining the distribution of phenotypes to search for discrete categories but this should not be confused with proof of Mendelian or quantitative inheritance. Environmental effects can obscure the appearance of discrete genotypic categories. In studying a trait whose phenotype is discrete, one must keep open the possibility that in fact its basis is polygenic but its expression results from an all-or-nothing response to a genetic threshold (Bulmer 1985, Gianola 1979a).

The majority of behavioral traits examined display continuous variation indicative of a polygenic basis (Ewing & Manning 1967, Huntingford 1984). However, as noted above, such data can be misleading. In fact, most characters are probably subject to control from several or many genes and variation at each may have the potential of either major genic effects or minor effects (Mather & Jinks 1982:30). The number of genes yielding variation and distribution of their effects can greatly influence evolutionary dynamics (Carson & Templeton 1984, Hedrick 1983, Rathie & Nicholas 1980, Templeton 1980, 1981). Luckily, from an analytical standpoint the number of genes and their relative contribution may not be so important as quantitative genetic analysis is based on models derived from Mendelian genetics. The types of results to be expected from analysis will differ between single gene and polygenic systems in level of dominance, gene interaction, and specificity of gene action (Mather & Jinks 1982). Ultimately, interpreting the implications of results from a quantitative genetic analysis of a trait determined by an unknown number of genes in the light of long term phenotypic evolution must be done cautiously.

2.3. The response to selection and genetic architecture

Many studies have documented that there is genetic variation for behavioral traits (Huntingford 1984, Noakes 1986) without differentiating additive from non-additive components. This is comparable to noting that the broad sense genetic variation is greater than zero. However, broad sense heritability provides little information as to what one should expect about the response of a trait to selection. This is because the relative amount of additive genetic variation can not be determined from the broad sense heritability and, over the short term, the response to selection of non-additive variation depends on population size (see below).

Estimates of the amount of additive genetic variation and genetic correlations permit predictions of the response to selection under conditions of weak selection and large, random mating populations (Lande 1979). Additive genetic variation for behavior is probably ubiquitous as demonstrated by the diversity of behavioral patterns that appear to have evolved in natural populations in response to local conditions. Non-additive genetic variation has also been documented for behavioral traits (e.g. dominance: Arnold 1981b, epistasis: Bateson & D'Udine 1986, and pleiotropy: Riechert & Maynard Smith 1989), but has not been well explored in any group. The nonadditive portion of the total genetic variation will respond to selection, though slowly (dominance variance) or impermanently (epistatic variance) (Bulmer 1985) if the population size is large. Population bottlenecks can cause dominance and epistatic components of variance to be converted into additive variance (Goodnight 1987, 1988). The final response to selection can thereby be governed by an unpredictable mix of the initial additive and nonadditive components (Cockerham & Tachida 1988). Thus, in some natural populations dominance and epistatic variation may ultimately contribute to the long-term response to selection. Maternal effects, inherited or environmental, can speed, retard, or change the direction of the response to selection depending on the form they take (Kirkpatrick & Lande 1989). However, I am not aware of an example of a maternal effect on offspring behavior.

Phenotypic correlations may link morphological and behavioral traits but the evolutionary implications of such linkage is dependent on their genetic architecture. Examples of such correlations include alternative reproductive modes and body size (Gross 1984, Farr 1989), jaw, gill morphology, and/or body shape and feeding behavior (Roberts 1974, Kornfield & Taylor 1983, Wooten 1984, Grudzien & Turner 1984a, b, Ehlinger & Wilson 1988), and the number of lateral scutes and aggression and predator avoidance behavior (Huntingford 1981, Reist 1983). In such cases, the genetic basis of behavioral phenotypes must be studied in conjunction with morphological ones to establish their relationship and make predictions about the effects of selection on either trait.

There are several possible sources of morphological and behavioral phenotypic correlations. Two genetic relationships are possible. The traits could stem from separate genes that are linked or the basis of both morphological and behavioral characteristics may stem from a common source which is itself under genetic control (i.e., linkage or pleiotropy, respectively). An example of the latter would be if the same hormone(s), whose regulation is under genetic control, could be influencing both traits (Bakker 1985, Travis et al. 1990b). On the other hand, it is possible that a factor of the external environment is influencing both traits independently. Finally, behavior may shape morphology, or the converse, with variation of the "shaping" trait under genetic or environmental control. For example, behavior which induces physical stress on morphological structures may lead to different developmental pathways in genetically similar individuals (Meyer 1987). Thus suites of morphological and behavioral traits may share a common genetic architecture, a common environmental basis, or some combination of the two. The differences in these relationships have implications when attempting to evaluate past and future evolutionary trajectories. Establishing the type of genetic relationship between two or more traits requires an experimental research program (or pedigree analysis generally unavailable from natural populations).

3. Methods for study

The genetic basis of trait variation can be studied through common garden experiments, selection experiments, crosses between inbred lines, or the correlations between relatives from outbred populations (Bulmer 1985). Though desirable from the standpoint of genetic analysis, inbred lines are seldom available to behavioral ecologists and may even be undesirable (Fuller 1983, Houde 1988a). Also, the development of inbred lines and selection experiments require that several generations be maintained in controlled conditions which is difficult or impractical for many species. In fact, inbred or true breeding lines are not an absolute necessity if the genetic variation within stocks is small compared to that between them (Mather & Jinks 1982). Ultimately, the choice of analytical methodology must be based on the questions asked and the level of resolution needed in the answers obtained.

Analysis of the distribution of phenotypes produced from crosses between different species permits study of the genetic differences that separate them (e.g., Clark et al. 1954, Clarke et al. 1984, Ferguson & Noakes 1983, Parzefall 1989). It is possible to estimate the number of segregating genetic factors which contribute to phenotypic differentiation from such studies. This is a minimal estimate of the number of genes involved in differentiation (Lande 1981). F_1. F_2, and backcross generations with sample sizes of at least 20, 100 and 100, respectively, are required to apply this technique with reasonable accuracy (Lande 1981). The interpretation of such studies is limited to differences between the species compared. It is not informative regarding within population genetic variation or the impact of selection in most natural situations. Also, the production of hybrids may inflate the genetic variance by disrupting each species' genetic background and creating novel gene combinations with unpredictable epistatic effects (Wetherington et al. 1989a, b).

The minimal requirements for study of genetic architecture are that individuals can be selectively mated and their offspring can be raised to an age appropriate for behavioral screening. The traits studied are generally described as modal or fixed action patterns, behavior that is stereotyped to a degree that it can be readily identified and screened in a large number of individuals (Barlow 1968, Manning 1975, Noakes 1986). In Appendix I provide an overview of some techniques used to examine genetic architecture and a brief discussion about choosing among research designs.

4. Genetic architecture and fish behavior

The presence of a genetic basis for behavior has been documented for a variety of fish species and a diversity of behavioral traits (Table 1). However, there are few examples of study into more than the most basic issues of genetic architecture of the behavior of fishes. My intention here is to focus on three types of behavior which illustrate the value of knowledge of genetic architecture or

Table 1. Summary of some recent studies of the genetic architecture of behavior in fish. See Huntingford (1984) and Noakes (1986) for additional references. Methods column notes the experimental design used in each study, the names of most designs are explained in the appendix. CG = common garden experiment, IL = crossing of inbred lines, BIP = biparental progenies design, NCI = North Carolina I design, BSEL = bidirectional selection experiment.

Species	Behavior	Method	Reference
Phoxinus phoxinus	predator avoidance	CG	Magurran 1990
Oncorhynchus tshawytcha	homing	CG	McIsaac & Quinn 1988
O. tshawytcha	rheotaxis and aggression	CG	Taylor & Larkin 1986, Taylor 1988
Oncorhynchus kisutch	swimming performance	CG	Taylor & McPhail 1985
Salmo salar	swimming performance	CG	Hurley & Schom 1984
Astyanax mexicanus	foraging	BIP	Schemmel 1980
Poeciliopsis monacha-lucida	aggression	CG	Keegan-Rogers & Schultz 1984
Xiphophorus nigrensis	courtship	CG	Zimmerer & Kallman 1989
Poecilia reticulata	courtship	IL, CG	Farr 1983, Farr & Peters 1984, Luyten & Lilley 1985
P. reticulata	female mate choice	CG	Breden & Stoner 1987, Houde 1988
P. reticulata	schooling	CG	Seghers 1974, Breden et al. 1987
Poecilia mexicana	aggression	BIP	Parzefall 1986
Poecilia latipinna	courtship	NCI	Travis et al. 1990b
Gasterosteus aculeatus	courtship	CL	Sevenster & t'Hart 1974
G. aculeatus	aggression	BSEL	Bakker 1985, 1986, Bakker & Sevenster 1989
Macropodus opercularis	aggression	BSEL	Francis 1984
M. opercularis	behavioral responses	BSEL	Gervai & Csanyi 1985, Csanyi & Gervai 1986

where more research seems promising to me. These are sexual behavior, aggression, and foraging. I do not intend to provide an exhaustive review of these topics but instead wish to point out the types of results obtainable from genetic analysis and to stimulate discussion about selected issues relevant to the evolution of behavior.

4.1. Sexual behavior

Some species of poeciliids display a suit of traits associated with male sexual behavior which can be described as alternative reproductive tactics (Farr 1989). Two modes of male behavior can be identified (Farr 1989). One mode is to use courting in an attempt to elicit female complicity in mating by choice while the other, thrusting, attempts to circumvent female choice. Secondary sexual characters are often associated with these different modes of male behavior and body size variation may or may not be related as well. The genetic architecture of these tactics has been studied in detail in a few species of poeciliids. An inherited basis for developmental correlates of alternative mating strategies (size and age at maturity) have been documented in other fishes, e.g. *Oncorhynchus kisutch* (Iwamoto et al. 1984), *Salmo salar* (Gerde 1984), and *Lepomis macrochirus* (Gross, personal communication), but inheritance of the behaviors was not studied to my knowledge.

Ten species of xiphophorine fishes are known to exhibit a sex-linked genetic polymorphism which influences the timing of release of gonadotropic hormone from the pituitary gland thereby regulating the onset of maturation (Kallman 1989). Most of these species have XY sex determination and the so-call P gene (pituitary gene) regulating this process has been mapped to the Y chromosome. Two species have more complicated sex-determining mechanisms and P gene factors in these species are known on both chromosomes (Kallman 1989). Associated with the timing of maturation in these species is variation in size at maturity. Recently, Zimmerer & Kallman (1988, 1989) have studied the genetic basis of male sexual behavior in *Xiphophorus nigrensis* which is linked to male size and allelic variation at the P gene.

Large male *X. nigrensis* court females while small males use thrusting behavior in an attempt to sneak copulations. Intermediate sized males mix the two courtship patterns (Ryan & Causey 1989). Using males known to possess different P gene alleles (*L*, *II*, *I*, *s* in decreasing order of size), Zimmerer & Kallman (1989) determined that males with the P alleles leading to the three largest sizes (*L*, *II*, *I*) all courted females. Males with the allele for smallest size at maturity (*s*) would use both sexual modes while alone with a female and switched solely to a sneaking (thrusting) mode when in the presence of larger males. When the smallest males from the *I* genotype were placed in competition with the largest males of the *s* genotype, such that their sizes overlapped, the *I* males failed to switch to the sneaking strategy. In such situations the *s* males sneaked. Thus, there is a genotype-dependent control of sexual behavior in *X. nigrensis*.

Allelic variation at the P locus on the Y-chromosome of *X. nigrensis* and associated loci affect a complex of fitness traits which include size, color, and sexual behavior. Similar variation can be seen in many other members of the genus and, as noted below, may have a similar effect in other poeciliids. Also, some linkage relationships of allozyme proteins have been shown to be conserved across different species of poeciliids (Morizot & Siciliano 1984, Mulvey & Trexler, unpublished data). Unfortunately, homology of such a genetic system is difficult to prove though plausible (Kallman 1989, Travis et al. 1990a).

Natural populations of guppies (*Poecilia reticulata*) display variation in color patterns and courtship behavior much of which appears to be sex-linked. Many, though not all, color patterns of guppies have been shown to be Y-linked traits (i.e., patrilineal inheritance, guppies have XY sex determination) (Kirpichnikov 1981). Y-linkage also plays a prominent role in the inheritance of courtship and thrusting behavior in guppies (Farr 1983). In addition, autosomal genes acting epistatically modify or "fine-tune" the basic Y-linked pattern. The autosomal effects differ between strains but explained a surprisingly consistent amount of total phenotypic variation in three measures of courtship behavior and one of thrusting when examined in three separate sets of crosses (around 10%) (Farr & Peters 1984). In addition, Farr (1983) found a genetic correlation between color patterns in one of the strains and the level of certain courtship behavior.

Patrilineal inheritance and potential Y-linkage has been shown to be important in a third poeciliid, the sailfin molly (*Poecilia latipinna*) (Travis et al. 1990a, b). Male courtship behavior and dorsal fin shape are interrelated traits that vary allometrically in sailfin mollies (Farr et al. 1986). This species displays a wide range of male size within and among populations (Trexler 1989); large males may be three times the length and ten times the weight of small males. The sailfin molly shares a P gene-like genetic system with *Xiphophorus* (Travis 1989a). Using a mating design with 22 sires mated to two dams each, the slope of the regression of size of sire on the size of his sons at maturity is not significantly different from one (Travis et al. 1990a). The slope of this regression equals the heritability for a sex-linked trait (Bulmer 1985). In contrast to males, there seems to be no genetic basis to variation in size or age at maturity in females within the population studied. There were no significant differences among sire families and no meaningful dam effects for female offspring (Travis et al. 1990a).

The sexual behavior of the sires used in this experiment was documented as was that of all male progeny produced (Travis et al. 1990b). The results produced the appearance that male behavior was inherited with body size. This result would be consistent with the findings of genotype-dependent courtship behavior in male *X. nigrensis*. However, as a separate treatment, siblings of those fish used to explore size and age at maturity were raised for four months in a cool room. Thus, growth in these fish was stunted before they were moved to warmer conditions where development could be completed. This treatment produced small sized males with the large male genotype. These stunted males behaved as small males, they courted females infrequently. All fish were raised in complete isolation from other fishes throughout life and were tested in single male-single female trials. It is possible that males can "feel" their size hormonally (Liley 1969, 1972, Liley & Stacey 1983) and this dictates courtship behavior. Hence, the P genotype of mollies may control their behavior but indirectly through its influence on endocrine function.

The poeciliids described above may be somewhat anomalous as the traits described are, or appear to be, Y-linked and most sex-linkage is associated with X-chromosomes (Bull 1983). However, all sexually dimorphic traits require sex-limited gene expression (at least) and sex-linked variation is common where it has been looked for (Bull 1983). As noted above, sex-linkage (with certain architectures) has implications for the response to selection.

4.2. Aggression

Genetic variation for aggressive behavior in the form of biting, pushing, and various displays has been noted for several species of fish (Table 1) and can have important ecological consequences (e.g., Keegan-Rogers & Schultz 1984). Aggressiveness displayed in a variety of different contexts has been examined for a common basis.

Aggressiveness displayed under different conditions and at different points of the life cycle may share positive, negative or no genetic correlation. Bakker (1985, 1986) and Bakker & Sevenster (1989) have examined the relationships of aggressive behaviors displayed in different contexts in threespine sticklebacks, *Gasterosteus aculeatus*, through selection experiments. Male dominance ability and territorial aggression did not respond equally to four generations of bidirectional selection for dominance. Dominance tests involved contests between two males for a single territory; territorial males were introduced simultaneously to a small aquarium and the contests were prolonged until a single winner was apparent (Bakker 1985). Territorial aggression was scored when an opposing male of the same size and age was suspended in a glass tube outside of the tank of the male being tested (Bakker 1985). Similar results were obtained by Francis (1984) after five generations of bidirectional selection for dominance in paradise fish (*Macropodus opercularis*) using similar behavioral assays to those described for sticklebacks.

Genetic correlations among some aggressive behavioral patterns were observed in Bakker's experiments. For example, courtship aggressiveness responded to selection on dominance ability in a similar way to territorial behavior

(Bakker 1985). Courtship aggression was measured as the response of a male presented with a ripe female in a glass tube (Bakker 1985) and the test for territorial behavior is described above. Bakker (1985) interprets the common response of these traits as indicative of a common genetic basis. Subsequent studies revealed that courtship aggression responded to selection on both juvenile and territorial aggression (Bakker & Sevenster 1989). Interestingly, sexual activity was enhanced by selection to reduce juvenile aggression and reduced by selection to reduce territorial aggression. Bakker & Sevenster (1989) interpret these results as effects of selection on the pituitary-gonadal axis. For example, selection on territorial aggression (male behavior) may result from selection acting on androgens (based on changes in kidney size which is, in turn, related to the production of a glue used in nest construction and under androgenic control) (Bakker 1986). Selection on juvenile aggressiveness seems to act on gonadotropic hormones (based on correlated responses in age at sexual maturity, age of onset of juvenile aggression, and female reproductive condition) (Bakker 1986).

Morphological variation may influence the level of aggression a fish receives from conspecifics and its social status (Tinbergen 1951). Many species of *Xiphophorus* share a male secondary pigment trait of black vertical bars associated with agonistic behavior. The intensity of the black bars heightens in socially dominant fish. Zimmerer & Kallman (1988) determined that the bar pattern of *X. nigrensis* has a polygenic basis but that individuals with the *s* allele of the P gene (see section 4.1) also have a factor on their Y chromosome which suppresses the expression of bars. The smallest males with the *l* allele of the P gene have bars though their size overlaps with the largest of those with the *s* allele at the P locus. The suppression of this "aggressive" coloration may help *s* males avoid conflicts with males of the larger genotypes which they would surely lose. Thus, the alternative mating strategies of this species result from variation in a suite of behavioral and morphological traits. The "strategies" are the sum of this variation which is organized by direct or indirect relationship (through linkage or pleiotropy) with variation at the Y-linked P locus (Zimmerer & Kallman 1989). A similar relationship between male color variation and aggression may be present in sticklebacks (Bakker & Sevenster 1983, Bakker 1985).

Genetic linkages among behavioral traits and between behavior and morphology are probably common and can have important implications for evolution. However, phenotypic correlations do not necessarily reflect genetic correlations (Bakker & Sevenster 1989) so the genetic relationships among traits can only be ascertained by explicit study.

4.3. Foraging behavior

Foraging behavior is an area where few genetic analyses have been performed. There is evidence for genetic variation in these behavioral traits (Table 1) and it can have important ecological consequences (e.g., Schenck & Vrijenhoek 1986, Wetherington et al. 1989a). More study in this area should be rewarding. For example, little is known about how foraging components such as functional and aggregative responses are inherited and evolve. It is generally thought that these two components of prey finding involve separate modes of foraging possibly relying on different senses. It seems reasonable that these modes would evolve together, but how? Perhaps there are genetic correlations between these behavioral traits so their evolution is linked. In addition, are there genetic relationships between foraging behavior and predator avoidance behavior? It seems likely that components of these two different behavioral modes could be under antagonistic selection. The compromise, risk sensitive foraging, is the topic of recent interest (e.g., Ibrahim & Huntingford 1989) but an implicit assumption of such studies is that the components of this behavior are free to evolve as necessary. Is this so?

Threespine sticklebacks (*G. aculeatus*) in a single drainage on Vancouver Island display morphological and behavioral variation associated with foraging (Lavin & McPhail 1985, 1986). Three feeding morphs can be identified (openwater, littoral, and intermediate) which differ as a result of both genetic and environmental factors (Lavin & McPhail 1987). A cluster analysis of the genetic correlations of eight trophic characters indicated that variation in two character suites, head shape and gill raker structure, differentiated these morphs. Lavin & McPhail (1987) interpret the observed weak genetic correlations between gill raker architecture and head shape as adaptive. Selection can operate relatively independently on these structures to produce optimal phenotypes for use of different food regimes. Feeding behavior was not scored in these fish so the genetic architecture of this behavior remains unknown.

Complex suites of morphological and behavioral solutions to ecological problems have been observed in fish foraging systems. Little is known about the genetic mechanisms behind these adaptations and their study should prove rewarding.

5. Conclusions

Architectures of both many genes with small individual effects and single genes with major effects and epistatic modifiers have been noted for fish behavior. Phenotypes stemming from these architectures are expected to evolve at different rates. Polygenic inheritance is probably most common in general (Lande 1981) and may be so for fish behavior (Parzefall 1989) but few data are available for behavior. Neither can be assumed from phenotypic data alone.

"Suites" of genetically correlated traits may permit complex adaptations by evolving in concert or resist adaptation because of antagonistic selection pressures on different components of the suite. Patterns of genetic correlations can thus be informative in studies of adaptation (Lavin & McPhail 1987). Morphological and behavioral traits may be involved and a common hormonal basis has been implicated in some cases. Genetic analysis to the second generation or beyond is required to determine if these suites result from pleiotropy or linkage (Butlin & Ritchie 1989).

It is easy to imagine that pleiotropy is important in behavior. Many apparently different behavioral traits share common features when decomposed to modal units. A common effector system controls jaw movements used in courtship displays, eating, retrieving larvae, and respiratory movements in cichlid fishes (Barlow 1968). Selection on the rate or threshold for any one of these actions could influence all of them if their central nervous system control is homologous. Barlow (1968) points out that many identifiable behavioral components reappear in very different contexts. He provides the example of a quivering behavior seen at the outset of swimming, in courtship, and in interindividual grooming in the orange chromide. Again, selection on the threshold or rate of one of these behavioral traits could conceivably influence all of them via a common genetic basis (see Manning 1975). In a similar vein, Halliday & Arnold (1987) have suggested that a behavior in one sex (multiple mating in females) could arise as the non-adaptive result of a genetic correlation to a trait under selection in the other sex (male sexual behavior) (see also Sherman & Westneat 1988, Arnold & Halliday 1988). A genetic correlation between the sexes for a new mutation is to be expected and is slow to dissipate (Lande 1980, Arnold & Halliday 1988). The viability of this explanation depends on whether the population in question is near the onset of sexual selection or at equilibrium, by which time sex-limited expression should evolve (Lande 1980).

The genetic basis of apparently similar behavior in members of the same genera may not be identical. Variation at both a major sex-linked gene and autosomal polygenes may be responsible for alternative courtship behavior in the poeciliids *P. latipinna* and *X. nigrensis*. However, *P. latipinna* with a large male genotype appear to be more flexible in their courtship patterns than those in *X. nigrensis*. (Caution must be exercised here as the context in which behavior was screened differed greatly in these studies and can alter results [Travis & Woodward 1989].) Similar alternative mating tactics have been noted in other fishes (e.g. salmonids and centrarchids) and the morphological correlates have a genetic basis. These systems may provide an excellent opportunity to examine the long-term evolution of behavior. It would be interesting to learn if these alternative strategies have arisen repeatedly within groups and if the genetic basis is preserved across species. New techniques from molecular biology provide the potential to revolutionize this type of research (Atchley & Newman 1989).

It is reasonable to ask if the information relevant to selection gleaned from quantitative genetic analysis is worth the trouble for a behavioral ecologist. The generality of its results is limited to the population from which stocks are drawn at the time that they are collected (Mitchell-Olds & Rutledge 1986). This is because the interpretation of parameters like heritability and genetic correlation are dependent upon gene frequencies in the study population and environmental conditions. The ability to make predictions about the response to selection from the narrow sense heritability depends on several factors (Mitchell-Olds & Rutledge 1986). One is the constancy of the heritability and the genetic correlations over time. The level of constancy is unknown (Turelli 1988) though relative constancy is believed to obtain over "short spans of evolutionary time" (Ayers & Arnold 1983, Lofsvold 1986). But, small effective population sizes and strong selection (Lande 1980) or a major gene that is an important source of genetic variation (Mitchell-Olds & Rutledge 1986) may yield changes in these parameters over relatively short time periods. Components of nonadditive genetic variation may be "recruited" into the response to selection in ways not anticipated by simple formulations if population bottlenecks arise (Cockerham & Tachida 1988). Also, the environment must not change enough to yield changes in heritability and genetic correlations from genotype-environment interactions (Service & Rose 1985). In general, quantitative genetic parameters must be re-estimated for each population studied and each environment they experience (Mitchell-Olds & Rutledge 1986).

The value of quantitative genetic analysis comes in two forms. For study of the potential for local adaptations by specific populations, the methods described here are useful. Genetic architecture, and especially heritability and genetic correlations, permit predictions over the short term with assumptions that can be evaluated and may even be fulfilled. These parameters are probably not useful in considering macroevolutionary patterns (Riska 1989). Perhaps of more importance, theoretical models underscore that genetic architecture is a critical factor in the dynamics of evolution. The value of different models will therefore be dependent on what genetic architectures actually exist in nature. Clearly, we can not be confident of a general understanding of evolution in nature until we know what assumptions about genetic architecture are valid.

From a logistic standpoint, behavior is not an attractive field for the application of quantitative genetic methods because the traits are frequently difficult or impossible to score on many individuals raised under controlled conditions. In spite of this challenge, there are many instances where the information gleaned from such study can provide deepened insight into the evolutionary process.

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Appendix: methods for study of the genetic basis of behavior

This appendix is intended to provide an introduction to some techniques used to obtain genetic information useful in predicting the response of a trait to selection. I have emphasized a quantitative genetics approach because of its potential for application to natural fish populations. Other techniques have been used successfully to examine genetic architecture but they require the identification of mutant phenotypes and/or the use of inbred strains, or the maintenance of stocks in captivity for many generations (Manning 1975, Atchley & Newman 1979).

A1. Common garden experiments

The most frequently used approach to document the presence of a genetic basis for behavior is a common garden or reciprocal transplant experiment (Primack & Kang 1989). By raising fishes from two or more populations under common environmental conditions, a genetic basis can be inferred for any persistent differences (Clausen et al. 1948). By rearing fish from different lineages or populations under two or more common conditions norms of reaction can be compared (Trexler 1989). Norms of reaction are the profiles of environmental influence on phenotype and their heterogeneity among populations indicates that environmental variation affects the populations uniquely. Such experiments are best done with fish in their second or third generation of controlled laboratory maintenance to standardize maternal environmental effects among genotypes. When populations are to be compared, family lineages within populations should be randomized in the laboratory to eliminate the effects of common family environments as much as possible.

Common garden experiments provide evidence of the presence or absence of genetic variation (i.e., if the broad sense heritability is greater than zero), under the assumption that maternal effects are negligible. They can provide an informative dimension to analysis if multiple environments are incorporated into the study. Common garden experiments do not permit one to explore the genetic architecture of a trait or make predictions about how or if it will respond to selection.

A2. Mating designs

There are four general experimental designs used to examine the genetic basis of behavior in outbred populations: the production of biparental progenies, and the North Carolina 1, 2, and 3 designs. These experimental designs provide estimates of the total genetic variance or of the variance attributable to the various genetic components discussed in the text. Their interpretation must be viewed with several assumptions in mind (see Cockerham 1963, Mitchell-Olds & Rutledge 1986) and is limited to the population from which stocks are drawn, at the time that they are drawn, and to the environment(s) used for the study. Maternal environmental effects should be standardized as much as possible. The following experimental designs are discussed in detail in Cockerham 1963, Mather & Jinks 1982, and Becker 1984.

Randomly mated biparental progenies (BIPs)

This design requires that randomly chosen males and females be mated and their offspring raised. Each male is mated to one female and each female to one male. This results in a collection of full sib families. From the analysis of variance, the mean squares attributable to variation within families is the total genetic variance from additive and non-additive sources including dominance, the common family environment, and epistasis (though the epistatic component is small). If enough families are used, a parent-offspring regression can be employed to estimate the additive genetic variation, excluding non-additive effects.

North Carolina 1

Half-sib correlations do not include variation from dominance or common family environment and the epistatic component is negligible (only 1/64th is represented). The N. C. 1 design mates individual males or females to two or more opposite gender individuals resulting in a collection of half and full-sib families. Thus, this design permits and estimation of the additive variance from two sources which can be pooled (Gianola 1979b), the correlation of half-sibs and the parent-offspring regression (if sample sizes permit). Also, an estimate of non-additive variance by comparison of full and half-sib correlations is possible.

North Carolina 2

The full N. C. 2 design mates all of n males with m females to yield n X m families. Thus, both half and full sib families are generated due to both maternal and paternal parents. (This design differs from the diallel cross which also requires crossing each individual to itself.) This design permits tests for the reciprocity of genetic patterns between the sexes. By selectively mating pairs of males and females in a "chain block" design it is possible to reduce the sample size somewhat while salvaging the power of this design. Pruning of the design retains the ability to estimate the covariances of full and half sibs (Cockerham 1963). The chain block design enhances the ability to detect differences among sires or dams by increasing the number used, but at a loss of statistical power for tests of interactive effects (Travis et al. 1987).

North Carolina 3 or Triple Test Cross

N. C. designs 1 and 2 provide tests for the presence of dominance but poor estimates of its level (Mather & Jinks 1982) and no estimate of epistasis. The N. C. 3 design is the best design for studying non-additive effects including the potential to examine epistatic effects (Mather & Jinks 1983, Bulmer 1985). The approach is to develop two "tester" lines by selecting individuals from the "high" and "low" extremes of the trait under scrutiny. It is wise to maintain these for two or three generations under constant conditions to equilibrate maternal environments (J. Travis, personal communication). This may also serve to assure as much as possible that these lines segregate for different alleles in all genes acting on the trait. When this is accomplished the tester lines are crossed to produce a hybrid F_1 generation. These three lines, two testers and their hybrid, are then crossed to individuals from the study population and these progeny are examined for patterns of phenotypic variance.

A3. Choosing a design

The logistics of raising fish under controlled conditions often renders the more complex designs unworkable. Do the simple BIP or N. C. 1 designs provide enough information to be worth the trouble? The additive genetic variance can be estimated from all four designs if adequate sample sizes are possible. However, the BIP design without enough families for parent-offspring regression may be of limited value. The broad sense heritability alone provides little more information than obtained from a well controlled common garden experiment. I feel that a common garden experiment with two environments bracketing those inhabited in nature provides more information than a comparably-sized BIP study with too few families for a useful parent-offspring regression. Both experiments indicate if the broad sense heritability differs from zero. The two environment common garden experiment also indicates if genetic effects are consistent across environments. Mather & Jinks (1983, p. 248) observe that the BIP design with parent-offspring regression may be more informative than the N.C. 1 design without enough sires for parent-offspring regression. This results from the large standard errors to be expected about estimates of heritability obtained by correlation methods. The relative precision of parent-offspring regression and half-sib correlations depend on the number of sire families and family sizes. With small family sizes, parent-offspring regression is more precise than half-sib correlation (see Gianola 1979b for tables indicating optimal allocation of experimental resources).

Given logistically realistic sample sizes and heritabilities between 0.2 and 0.6, half-sib correlation and parent-offspring regression may only tell if the additive genetic variation is greater than zero with a standard level of confidence (Klein et al. 1973). Current methods of estimating standard errors about these parameters are only approximate and better methods using resampling techniques need to be developed (Via 1984, Mitchell-Olds & Rutledge 1986, Knapp et al. 1989). Estimates of genetic correlations are less precise than those of heritabilities (i.e., larger sample sizes are needed to estimate genetic correlations than to estimate heritability with comparable precision). Klein et al. (1973) recommend that estimates of genetic correlations should not be attempted with less than 400 families of size four! This claim must be balanced with the observation that the standard errors about genetic correlations are inversely related to the level of genetic correlation and the heritability of the traits involved. Hence, Arnold (1981a) was able to identify genetic correlations with moderate to small standard errors for garter snake prey preferences and other traits, all with high heritabilities and high genetic correlations.

The N. C. 1 design permits one to estimate narrow sense heritability two ways and to asses the potential role of dominance variance. Its use precludes the gathering of reciprocal information about both sexes, however, it is possible that at this stage of study interest may focus on one sex or the reciprocity of inheritance may be known. The N. C. 2 design is necessary when patterns of inheritance between the sexes is at issue. Modifying this as a chain block can permit statistically powerful tests of a priori genetic hypotheses while maintaining a logistically feasible experiment size (e.g. see Woodward et al. 1988). Obviously, N. C. 3 is the method of choice to explore non-additive genetic variation in detail but requires a substantial research commitment.