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BEHAVIORAL SYNDROMES: AN INTEGRATIVE OVERVIEW

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ABSTRACT

A behavioral syndrome is a suite of correlated behaviors expressed either within a given behavioral context (e.g., correlations between foraging behaviors in different habitats) or across different contexts (e.g., correlations among feeding, antipredator, mating, aggressive, and dispersal behaviors). For example, some individuals (and genotypes) might be generally more aggressive, more active or bold, while others are generally less aggressive, active or bold. This phenomenon has been studied in detail in humans, some primates, laboratory rodents, and some domesticated animals, but has rarely been studied in other organisms, and rarely examined from an evolutionary or ecological perspective. Here, we present an integrative overview on the potential importance of behavioral syndromes in evolution and ecology. A central idea is that behavioral correlations generate tradeoffs; for example, an aggressive genotype might do well in situations where high aggression is favored, but might be inappropriately aggressive in situations where low aggression is favored (and vice versa for a low aggression genotype). Behavioral syndromes can thereby result in maladaptive behavior in some contexts, and potentially maintain individual variation in behavior in a variable environment. We suggest terminology and methods for studying behavioral syndromes, review examples, discuss evolutionary and proximate approaches for understanding behavioral syndromes, note insights from human personality research, and outline some potentially important ecological implications. Overall, we suggest that behavioral syndromes could play a useful role as an integrative bridge between genetics, experience, neuroendocrine mechanisms, evolution, and ecology.

INDIVIDUAL HUMANS SHOW consistent differences in their behavioral tendencies. Compared to others, some people are relatively assertive, or bold, or friendly, or deceptive. Analogous patterns of individual variation have been documented in several primates, domesticated animals, laboratory rodents, and a scattering of other animals (Gosling and John 1999; Gosling 2001). In humans, these differences have been termed personality types (Pervin and John 1999). In other taxa, they have been referred to as coping styles, temperaments, behavioral tendencies, strategies, syndromes, axes, or constructs (Gosling 2001). From an ecological and evolutionary view, an underlying theme of these related concepts is that they refer to suites of correlated behaviors that can include those expressed either within a given behavioral context (e.g., foraging behaviors in different habitats) or across different contexts (e.g., feeding, antipredator, mating, contest, and dispersal contexts). In evolutionary ecology, suites of correlated characters are commonly referred to as syndromes (e.g., life-history syndromes, dispersal syndromes), thus we refer to suites of correlated behaviors as behavioral syndromes.

An example of a behavioral syndrome that has been documented in several species is an aggression syndrome (Huntingford 1976; Riechert and Hedrick 1993). Although all individuals alter their aggression levels depending on the context (feeding, mating, predator avoidance), some are consistently more aggressive than others across contexts. Analogous consistent between-individual differences in behavior have been noted for activity (Henderson 1986; Sih et al. 2003), shyness/boldness (Wilson et al. 1994; Fraser et al. 2001), fearfulness (Boissy 1995), and reactivity (Koolhaas et al. 1997). Most extant literature examines proximate (e.g., genetic, neuroendocrine, developmental) bases of these syndromes. Our focus is on the evolution and ecological importance of behavioral syndromes. We also review relevant literature on proximate bases and suggest that behavioral syndromes could play a useful role as a bridge that integrates genetic, physiological, ecological, and evolutionary approaches to studying behavior.

The reason why behavioral syndromes have critical implications for evolution and ecology is simple. The existence of behavioral syndromes implies correlations between behaviors expressed in different contexts; i.e., what an individual does in one context is coupled with what it does in other contexts. When traits are correlated, single traits (here,

behavior in any single context) do not evolve in isolation. Instead the suite of correlated traits (here, the behavioral syndrome) evolves as a package (Price and Langen 1992; Lynch and Walsh 1998). In particular, the correlations can generate tradeoffs across contexts that can play a major role in evolution.

A useful analogy can be drawn between life-history evolution and the evolution of behavioral syndromes. Life-history correlations (e.g., the cost of reproduction expressed as a negative correlation between current reproduction and future reproduction or survival) clearly play a major role in shaping life-history evolution (Roff 1992; Stearns 1992). Due to tradeoffs generated by these correlations, individuals typically do not attempt to maximize their reproductive effort or survival in any given year. Instead, depending on the selection regime, tradeoffs can favor delayed reproduction, limited reproduction in any given year, and senescence (Roff 1992; Stearns 1992). If we look at any one of these traits in isolation, it can appear suboptimal; however, across the organism's lifetime, these traits can be part of an optimal life history. In addition, the combination of spatiotemporal variation in selection regimes and life-history tradeoffs can explain the maintenance of life-history variation within and among species (Roff 1992; Mangel and Stamps 2001; Orzack and Tuljapurkar 2001).

Returning to behavior, consider a species with an aggression syndrome where some individuals are more aggressive than others in more than one context. More aggressive individuals that do well in situations where aggressiveness is called for (e.g., in competition for food or mates) might be unsuitably aggressive in situations where caution or care is more appropriate (e.g., in the presence of a dangerous predator or in a parental care context). Conversely, less aggressive individuals should do well in situations where low aggression is appropriate, but might fare poorly in situations where aggression is favored. Following the analogy from life-history evolution, these tradeoffs should have three important, general implications for behavioral ecologists. First, behaviors that are part of a syndrome should not be studied in isolation. Understanding what individuals do in any

given context requires an understanding of their correlated behaviors in other contexts. This is true for behaviors that are similar (e.g., aggression during feeding and during mating) and behaviors that are seemingly unrelated (e.g., aggression and dispersal). Second, the existence of behavioral tendencies that carry over across contexts (e.g., the aggression syndromes described above) could mean that individuals show suboptimal behavior (when judged in an isolated context) in some, perhaps many, situations. Third, the notion that individuals do well in some contexts and poorly in others could help to explain the maintenance of individual variation in behavior.

Our goal here is to provide a conceptual overview on the study of behavioral syndromes. We discuss: 1) terminology and basic empirical designs for studying behavioral syndromes; 2) examples of how behavioral syndromes might shape ecologically important behaviors; 3) the evolution of behavioral syndromes; 4) proximate mechanisms (genetics, experience, and neuroendocrine bases); 5) insights from the study of human psychology; and 6) ecological implications (e.g., for population or community ecology, conservation biology).

TERMINOLOGY AND METHODS

As noted above, trait correlations and the resulting tradeoffs are at the heart of many issues in modern evolutionary ecology (Figure 1). To illustrate how behavioral syndromes relate to traditional concepts about tradeoffs in behavioral ecology, we distinguish between within- versus across-situation conflicts involving one or more behavioral contexts. By a *context,* we mean a functional behavioral category—e.g., feeding, mating, antipredator, parental care, contest, or dispersal contexts. A *situation* is a given set of conditions at one point in time. Different situations could involve different levels along an environmental gradient (e.g., different levels of predation risk or different food levels), or different sets of conditions across time (e.g., the nonbreeding season versus the breeding season, or juvenile versus adult stages). Correlations can involve behaviors expressed in:
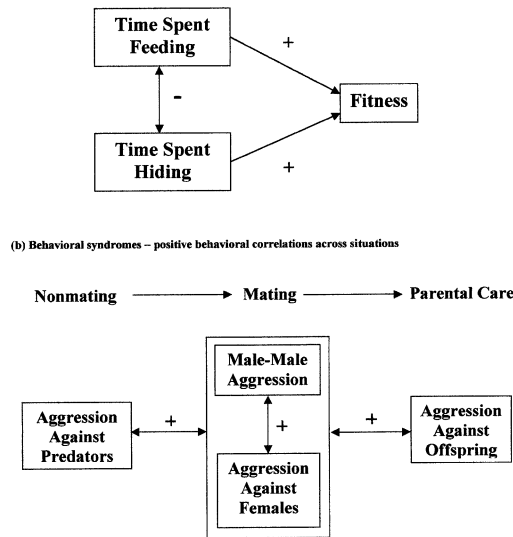
FIGURE 1. TYPES OF BEHAVIORAL CORRELATIONS THAT GENERATE CONFLICTS
    Two types of behavioral correlations that generate conflicts: (a) negative behavioral correlations produced by time budget conflicts within any given situation; e.g., conflicts between feeding and hiding; (b) positive behavioral correlations across situations produced by behavioral tendencies that carry over across situations.

1) different contexts within the same situation (e.g., feeding activity and mating activity in one set of ecological conditions); 2) the same context, but in different situations (e.g., feeding activity in the presence versus absence of predators or the voracity of juveniles versus adults); or 3) different contexts in different situations (e.g., aggression toward conspecifics in the absence of predators versus feeding activity in the presence of predators).

    One major source of behavioral correlations involves time budget conflicts within a given situation; these exemplify the first type of correlation noted above—correlations for different behavioral contexts within a given situation. Simply because individuals have a limited amount of total time, more time spent on one activity tends to result in less time spent on other mutually exclusive activities. The various elements of a time budget thus tend to be *negatively correlated* (Figure 1a). For example, often animals cannot feed while hiding and vice versa. If these are the main activities in a time budget, then time spent hiding versus feeding should be negatively correlated. As a result, even though both

activities, taken in isolation, should increase fitness, organisms cannot simultaneously maximize both. Instead, they must adaptively balance these conflicting demands (e.g., Sih 1980, 1987; Houston et al. 1993; Lima and Bednekoff 1999). Time budget conflicts play a major role in modern thinking about adaptive behavior.

    Behavioral correlations within a given situation can also be generated by individual variation in behavioral tendencies. For example, individuals might exhibit a general attack syndrome that, within a feeding context, tends to produce positive correlations among individuals in their probability of attacking different food types in one habitat. "High voracity" individuals should have high overall feeding rates, but might also frequently err by attacking prey that are unpalatable, dangerous, or otherwise difficult to capture or handle. Positive correlations associated with behavioral syndromes can mask negative correlations due to time budget conflicts. For example, if some individuals are generally more active than others, this could result in a positive correlation between mating and feeding activity within a given time period

(rather than the expected negative correlation based on time budget constraints for individuals with identical activity levels).

Behavioral tendencies can also carry over to cause correlations between behaviors exhibited in the same context but across different situations. For example, individual variation in general activity could result in a positive correlation between foraging activity in the presence versus absence of predators. Individuals that feed most actively during periods of safety might also continue to feed relatively actively and thus take the greatest risks when predators are present (Sih et al. 2003). The result would be a tradeoff between foraging and predator avoidance due to an activity carryover across situations rather than the usual time budget conflict within a situation.

Most interestingly, behavioral syndromes can involve different behavioral contexts expressed across different situations. For example, as noted earlier, an aggression syndrome might result in positive correlations between aggression levels in intrasexual competition, male-female conflict, feeding, antipredator, and/or parental care contexts (Huntingford 1976; Riechert and Hedrick 1993; Figure 1b). Across-situation correlations could involve behaviors that seem similar but are measured in different situations (e.g., aggression in contests versus tendency to attack dangerous prey), or different yet plausibly related behaviors (e.g., aggression in contests versus parental feeding of offspring).

Of course, whether a correlation is positive or negative depends on how the variables are defined. For example, the correlation between mating activity in the absence of predators and behavior in the presence of predators depends on whether the latter is defined as time spent hiding or time exposed. If individuals exhibit consistent activity levels across situations, mating activity without predators will be positively correlated with exposure, but negatively correlated with refuge use. Throughout this paper, we define behaviors such that higher activity (or aggression or boldness) results in larger values; thus a consistent behavioral tendency produces a positive correlation.

To quantify a behavioral syndrome, we need at least two observations of behavior (preferably in different contexts or situations) for each of a set of individuals. With these data we can quantify two distinct aspects of a behavioral syndrome: *within*-individual versus *between*-individual consistency in behavior. Within-individual consistency refers to the tendency for any given individual to exhibit consistent behavior across observations (e.g., for an individual to be generally aggressive). In principle, this can be quantified for a single individual, independent of the behavior of others. Between-individual consistency refers to consistent differences among individuals in behavior (e.g., rank-order consistency in aggressiveness) expressed statistically as a *behavioral correlation*. If the study involves repeated observations of the same type of behavior in the same context and situation, we refer to behavioral consistency as *repeatability* (Boake 1994). A *behavioral syndrome* is a suite of correlated behaviors across multiple (two or more) observations—most interesting, if it involves multiple contexts or situations. Within the syndrome, each individual has a *behavioral type* (e.g., more versus less aggressive individuals).

Behavioral syndromes can be most interesting when they result in less than optimal behavioral plasticity (limited behavioral plasticity). For example, a shy/bold syndrome (Wilson et al. 1994) is particularly interesting if bold individuals are inappropriately bold in situations where they should be cautious. The existence of between-individual consistency need not imply within-individual consistency or limited plasticity. It is possible, for example, for individuals to differ consistently in mean aggressiveness, but for all individuals, including those that are more aggressive, to be highly plastic in their aggressiveness across contexts. If, however, individuals show limited behavioral plasticity, we refer to this as a *behavioral carryover* or *spillover*.

These distinctions can be illustrated using plasticity plots (Figure 2a) and correlation plots (Figure 2b). These plots are analogous to reaction norm (2a) or genetic correlation plots (2b) of quantitative genetics (Via and Lande 1985; Roff 1997), except that for our purposes each line (2a) or point (2b) can rep-
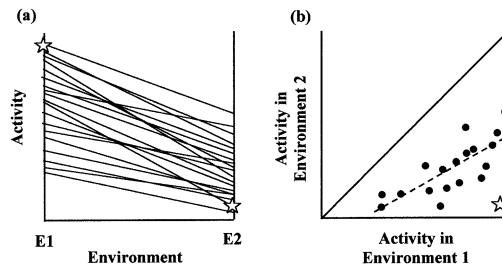
FIGURE 2.   GRAPHS OF BEHAVIORAL CORRELATIONS
   Graphical representations of behavioral correlations borrowed from the literature on adaptive plasticity: (a) plasticity plots (reaction norms if each line is for a different genotype); (b) correlations across environments. E1 and E2 might represent, for example, activity in the absence and presence of predators. The stars are the optima for each environment.

resent either a genotype or simply an individual. Different behavioral types are represented as different lines (2a) or points (2b). In our example, due to stabilizing selection, there is an optimal trait (represented by the stars) in each environment. A fixed behavior would appear as a flat line (slope = 0) in a plasticity plot, and a point on the 45-degree line in a correlation plot. A positive behavioral correlation is shown as a line of positive slope in Figure 2b, and as a tendency for plasticity lines to be parallel in Figure 2a. It is possible for behaviors to be negatively correlated; however, our main emphasis is on positive correlations associated with behavioral syndromes. A behavioral carryover (less than optimal plasticity) is indicated if the slope of the plasticity line is less than the optimal plasticity, or if a point on the correlation plot is closer to the 45-degree line than is the optimal point. A behavioral correlation poses a potentially important constraint if no plasticity line or point coincides with the optimum, due to behavioral carryovers.

   The basic empirical design for studying behavioral syndromes involves following a set of individuals across multiple contexts or situations in order to measure their behavior and, ideally, related traits (e.g., morphology, neuroendocrine profile) and performance (e.g., feeding rate, mating success, survival) in each context or situation. Although this design is simple, it represents a philosophical shift in how many of us study behavior. Many of us are specialists who focus on one type of behavior over a limited range of the organ-

ism's overall life. In Gould and Lewontin's (1979) terminology, we "atomize" the organism. The concept of behavioral syndromes inherently advocates a more holistic view of the organism's behavior over its lifetime. Organisms encounter many contexts and situations over their lifetime, many of which contribute to their overall fitness. If there are correlations across some of these contexts or situations, they need to be understood. In many fields, following individuals across contexts or situations is not the norm. For example, in order to satisfy statistical independence, predator-prey studies generally examine different individuals in different treatment conditions (e.g., presence versus absence of predators). We suggest that additional, new insights can be gained by studying the same individuals across contexts or situations. Studies of social behavior often follow individuals over a long time period, through multiple contexts and situations (e.g., territory establishment, intraspecific competition and mate choice, nesting and parental care); however, few studies have then examined carryovers or correlations across these contexts or situations.

   A matrix of correlations among multiple behaviors can contain more than one set of correlated behaviors. A multivariate analysis (e.g., factor analysis) might distinguish distinct suites of behaviors that represent one or more separate syndromes—e.g., an aggression syndrome and a fear syndrome (Budaev 1998; Gosling 2001). Personality psychologists have developed other statistical methods

to assess individuals across multiple contexts that should also be useful here (e.g., Campbell and Fiske 1959; Shrout and Fiske 1995; Briggs 1999). The literature on human personalities suggests that humans have four or five axes or *dimensions* of behavioral variation (see the later section on Human Personalities). Each individual exhibits a behavioral tendency on each dimension. Across the entire population, these dimensions should be uncorrelated; however, subsets of the population can show a correlated suite of tendencies—e.g., a "computer nerd" might be conscientious and introverted (Gosling, personal suggestion)—that represents a multidimensional behavioral type (Robins et al. 1996).

Path analysis (e.g., Pedhazur 1982; Scheiner and Callahan 1999; Sih et al. 2002) can quantify relationships between behavioral syndromes, other traits, and performance. If positive correlations are significant for behaviors within one basic context (e.g., positive correlations for attack tendency on different food types or at different food levels), but not between contexts (e.g., for attack tendency on food versus tendency to attack potential mates), the correlations are referred to as *domain specific,* whereas if the correlations occur across multiple contexts, they are termed *domain general* (Kagan et al. 1988; Wilson 1998). Although to us, domain-general syndromes seem particularly interesting, often domain-specific correlations should still be ecologically and evolutionarily important.

Up to this point, we have been discussing behavioral syndromes as a property of a single population. For some issues, however, we might be interested in contrasting behavioral syndromes or types for different populations or species. For example, do different dog breeds differ in behavioral type, or do dogs differ from cats? If the same behaviors have been quantified in different groups, then we can directly compare mean behavioral types across groups. For example, funnel web spiders from two populations differ in their overall willingness to attack conspecifics and food items: spiders from one population are more "aggressive" toward food and conspecifics than spiders from another population

(Riechert 1993). If, however, qualitatively different behaviors are exhibited by different taxa (e.g., different species or even more distantly related taxa), then attempts to contrast their levels of "aggression" or "activity" become problematic. Can aggression in squids be meaningfully compared to aggression in impala (for an early attempt to answer a similar question, see Glickman and Sroges 1966)? A related and potentially even more complicated issue is whether the same suites of traits are present in different groups, or whether the very structure of "personality" varies: aggression toward food and conspecifics might be tightly correlated in one group but unrelated in another. One broad approach asks whether factor analysis produces the same factors in different groups (Gosling and John 1999). For example, in humans, personality can be quantified in terms of five major axes: extroversion, neuroticism, openness, agreeableness, and conscientiousness (see section on Human Personalities). Studies on other animals (e.g., other primates, dogs) have asked whether these same five factors can be identified in these other animals. This approach, however, ignores more subtle differences between groups in correlation structures. An alternative approach, that to our knowledge has not been applied in animal behavior, is to use statistical methods drawn from evolutionary biology (e.g., confirmatory factor analysis) to compare correlation matrices from different groups (Steppan et al. 2002).

A Selective Review of Examples and Issues

Gosling's review of animal personalities (2001) noted four behavioral syndromes that have been quantified repeatedly in humans and "model organisms" (other primates, laboratory rodents, and domesticated mammals like dogs, cats, and farm animals): 1) aggression (tendency to attack other individuals); 2) general activity level; 3) sociability (tendency to seek out social interactions); and 4) fearfulness (nervousness, avoidance of novel stimuli) (Boissy 1995). Much of this work has focused on proximate (genetic, neuroendocrine) mechanisms. In contrast, behavioral

syndromes have received only limited attention in other organisms, and relatively few studies have examined syndromes from an evolutionary ecological perspective. Next, we review selected examples of behavioral syndromes studied in nonmodel organisms from an evolutionary ecological view.

For aggression syndromes, a notable study is the work by Riechert and coworkers on the funnel web spider, *Agelenopsis aperta*. Riechert and Hedrick (1993) showed that relative to less aggressive spiders, more aggressive ones showed higher attack tendencies on both prey and conspecific competitors (territory invaders), and a shorter latency to emerge from refuge after a simulated predator attack (i.e., reduced antipredator responses). Later work showed that aggressive spiders also tend to exhibit high levels of excessive, nonadaptive wasteful killing (Maupin and Riechert 2001), and some possibly nonadaptive sexual cannibalism (Riechert, personal observation). More aggressive genotypes were found in populations with a history of low food availability (relative to less aggressive populations; Riechert 1993), though gene flow among populations could result in overly aggressive animals in sites with high food levels (Riechert 1993). Controlled crosses between populations revealed a simple, single locus, sex-linked basis to aggressive tendency counteracted somewhat by an autosomal, quantitative (polygenic) level of fearfulness (Riechert and Maynard Smith 1989). Similar correlations between aggression in territorial and antipredator contexts have been documented in sticklebacks by Huntingford and coworkers (Huntingford 1976, 1982).

Another ecologically important syndrome involves activity correlations across situations. Activity level has been posited to be a key trait that links behavior to feeding rate, metabolic expenditures, and predation risk and, thus, to population/community dynamics (Sih 1987; Werner and Anholt 1993). While hundreds of experiments have quantified changes in average activity in response to changes in food availability or predation risk (Hassell 1978; Lima 1998), few studies have looked at activity correlations across contexts. For example, surprisingly few studies have quantified the activity of the same animals in the presence and absence of predators. Sih et al. (2003) studied the role of activity correlations across situations in determining predation rates and coexistence in a sunfish (predator)/salamander (prey) system. Streamside salamander larvae, *Ambystoma barbouri,* are found in streams with a mix of relatively permanent pools with predatory sunfish and ephemeral, fishless pools. Because larvae drift among pools, to survive to metamorphosis they must exhibit behavioral plasticity to cope with selection pressures in both pool types. Habitat ephemerality favors high activity that drives high feeding, growth, and developmental rates (Petranka and Sih 1987; Maurer and Sih 1996). In contrast, in fish pools, in the daytime larvae should show little or no activity, though they need to be somewhat active at night in order to move through a fish pool to drift out the downstream end (Sih et al. 1992). The optimal behavior should be to be very active in the absence of fish, moderately active in fish pools at night, and very inactive in fish pools in the day. Salamanders, however, exhibited positive activity correlations between the presence and absence of fish cues, and between activity in the day versus at night (Sih et al. 2003). Thus larvae that tended to be more active in fishless pools tended to also drift more among pools, and unfortunately also tended to be inappropriately active in fish pools in the day. The latter result at least partially explains the poor ability of these larvae to persist in fish pools or streams with too many fish pools (Petranka 1983; Sih et al. 1992). Full sib analyses suggested some heritability of activity syndromes in this system (Sih et al. 2003). Maintenance of genetic variation in activity types can apparently be explained by gene flow and spatiotemporal variation in selection pressures (Storfer and Sih 1998; Sih et al. 2003). On a larger taxonomic scale, Richardson (2001) found positive correlations across 13 species of anurans between the evolution of tadpole activity in the absence of predators versus in the presence of either of three predators (fish, newts, dragonflies). That is, species that evolve higher activity in the absence of predators also appear to simultaneously have higher activity in the presence of any of the three major predators.

Antipredator and feeding activity might be elements of a fearfulness, or shy/bold continuum (Wilson et al. 1993, 1994; Wilson 1998). This shy/bold syndrome has been examined by behavioral ecologists by comparing suites of behaviors and performance for individual animals that readily approach or are easily trapped by humans versus other individuals that avoid humans and traps. Bolder sunfish tend to also approach predators (engage in predator inspection), acclimate quickly to the laboratory, feed more on exposed and difficult to capture prey, and carry different parasites (Wilson et al. 1994). Bolder killifish disperse further and grow faster (Fraser et al. 2001), and bolder bighorn sheep tend to survive better in the field (Réale et al. 2000) than less bold ones.

A syndrome involving exploratory behavior, fearfulness, aggression, and response to environmental change has been identified in a number of species and termed the "proactive-reactive axis" (Hessing et al. 1994; Benus and Röndigs 1997; Koolhaas et al. 1997, 1999). Pro-active individuals manipulate or control their environments, while reactive individuals cope with their environments. Relative to reactive individuals, proactive individuals quickly explore their environment, readily form routines, and are more aggressive. Different "types" of individuals might do well in different environments. While proactive animals might outcompete reactive individuals in a relatively constant environment, it takes proactive individuals a long time to adjust to changing conditions. In contrast, reactive individuals pay close attention to their environment and, as a result, may be favored in more variable environments. Drent and colleagues have studied this proactive-reactive syndrome in the great tit (*Parus major*) in both the laboratory and field. They found correlations between exploratory behavior (Verbeek et al. 1994), foraging behavior (Drent and Marchetti, 1999; Marchetti and Drent, 2000), boldness/reactions to a novel environment (Verbeek et al. 1994; Drent and Marchetti 1999), aggressiveness/dominance in juveniles (Verbeek et al. 1996), responses to lost contests (Verbeek et al. 1999), and behavioral/physiological reactions to stress (Carere et al. 2001, 2003).

Recent work by this group showed that exploratory behavior and boldness are both repeatable and heritable (Dingemanse et al. 2002; van Oers et al. 2004), and respond to bidirectional artificial selection over the course of four generations (Drent et al. 2002). Finally, fast explorers showed greater dispersal in the field (Dingemanse et al. 2003). Thus reactivity appears to be a behavioral trait with substantial evolutionary and ecological implications.

Other possible examples of important behavioral carryovers emerged from attempts to explain apparently maladaptive behavior. In several cases, authors have suggested that an important general behavioral tendency has spilled over to produce inappropriate behavior in other similar contexts or situations. For example, Jamieson (1986) noted that host feeding of brood parasites (that often bear little resemblance to their own offspring) presumably reflects a spillover from strong selection favoring parental feeding of their own offspring. Brood parasites have obviously evolved adaptations to take advantage of this parental feeding tendency. Jamieson (1989) then took the additional step of suggesting that helping at the nest in cooperative breeders also represents a misguided spillover from parental feeding per se. This latter idea was met with considerable resistance (Emlen et al. 1991). Another well-known idea that involves spillover is the pre-existing sensory bias hypothesis (Ryan and Rand 1993), which posits that females prefer males that produce signals that are attractive for some other reason; e.g., signals that resemble feeding cues (Proctor 1991).

Spillover effects could extend across ontogeny. For example, Arnqvist and Henriksson (1997) suggested that excessive sexual cannibalism in fishing spiders (resulting in some females having a high percentage of infertile eggs possibly because they attacked all males that they encountered in their lifetime) could be the outcome of a general feeding aggression syndrome expressed over a lifetime. Juveniles that are more voracious should feed at a higher rate and thus grow faster and larger, ultimately into more fecund adult females (Johnson 2001). Adult females that continue to be more voracious should further enhance

their fecundity. If, however, there are positive correlations between attack tendencies on various potential prey including conspecific males (i.e., a "voracity syndrome"), then this could explain the observed excessive sexual cannibalism. Recent studies confirmed the existence of a voracity syndrome in another species of fishing spider (Johnson and Sih, personal communication).

Discussion of carryovers over ontogeny brings up the general issue of the temporal stability of behavioral correlations. The literature on humans, primates, and laboratory and domesticated animals includes considerable data on this issue (Gosling 2001); however, little information exists for other organisms. Clearly, a behavioral syndrome is likely to be particularly ecologically and evolutionarily important if it is consistent over a lifetime. We suggest, however, that even a short-term behavioral carryover should be important. In the sunfish-salamander system discussed above, we found reasonably strong correlations between activity in the presence of fish cues versus in the absence of cues a few days earlier or later, but weak correlations several weeks earlier or later in the larval period (Sih, personal communication). However, given that even an hour of inappropriately high activity in a fish pool in the day strongly increases larval mortality, a carryover that lasts for even a few weeks can be ecologically very important.

The definition of a behavioral syndrome can be extended beyond behavioral carryovers within a given individual to also include carryovers among individuals with the same or similar genotypes. For example, for a clonal species, a behavioral syndrome could involve a carryover between the feeding activity of some individuals to the mating activity of other clonemates. Taking this notion a step further, behavioral correlations can even carry over across the sexes (e.g., between brothers and sisters). For example, since sexual selection often favors males with high mating tendencies, Halliday and Arnold (1987) suggested that a genetic correlation between the sexes could cause some females to mate more frequently than necessary relative to their own fitness needs. A positive genetic correlation between mating drives of

male and female sibs has indeed been detected in *Drosophila* (Partridge 1994). Furthermore, the correlation across sexes could involve superficially unrelated, gender-dependent behaviors. For example, artificial selection in mice for highly aggressive, territorial males did not influence female territorial aggression (females do not typically show territorial aggression), but increased female proactivity expressed as an increased tendency to bury (rather than avoid) an electric prod (Koolhaas et al. 1999).

It is worth emphasizing that depending on the contexts or situations compared, behavioral correlations need not generate conflicts or tradeoffs. Conflicts occur when behavior is beneficial in some contexts or situations and costly in others. For example, high activity is often advantageous in feeding or mating contexts, but costly in an antipredator context. Similarly, aggressiveness might often be beneficial in a contest context, but costly if directed against offspring. However, no conflict arises across contexts or situations where the behavior is always favored. If, for example, high activity levels result in higher mating success across a range of different social situations (e.g., different densities or sex ratios), then a positive activity correlation across social situations does not result in a tradeoff in mating success across situations. Nonetheless, the correlation still has an important effect in that it increases variation among individuals in performance. In this social/mating example, a positive activity correlation increases variation in mating success and thus the opportunity for sexual selection. A study on water striders found that this "winners are winners, and losers are losers" effect of a positive activity correlation increased overall opportunity for sexual selection by almost 50% (Sih, personal observation).

It should also be noted that while our examples have emphasized positive behavioral correlations, the concept of a behavioral syndrome includes both positive and negative correlations. A strong negative behavioral correlation across contexts or situations still has the potential to be evolutionarily and ecologically important. For example, Hedrick (2000) showed that male crickets that have longer courtship calling bouts (i.e., take

greater risks while calling) compensate for this risky behavior by being more cautious when danger is imminent by exhibiting longer latencies to emerge from shelter when placed in a novel, potentially risky environment, and longer latencies to reinitiate calling after being interrupted by a predator cue. In general, adaptive behavioral compensation that reduces the costs of other costly (e.g., risky) behaviors might often generate negative behavioral correlations across contexts. A behavioral syndrome might then consist of a suite of positive correlations among similar behaviors that carry over across contexts or situations, mixed with negative correlations due to adaptive compensatory behaviors. Finally, some behaviors may not be significantly correlated at all. In particular, behavioral correlations might often be context specific or domain specific (Coleman and Wilson 1998). While different behaviors might be correlated within a feeding context or within an aggression context, these might represent different domains that are uncorrelated. Overall, it seems likely that a study that quantifies multiple aspects of behavior for the same set of individuals will yield a mix of positive, negative, and nonsignificant correlations.

Unfortunately, quantifying all these correlations can be a difficult, if not overwhelming, logistical task. How far afield do we need to go in our search for behavioral syndromes? One approach should be to examine correlations that have been shown to be important in the few systems that have been studied in this context (e.g., activity in the presence versus absence of predators, aggression in feeding, mating and contest contexts). Another approach could be to examine behavior in a broader spectrum of situations for systems with known individual variation in behavioral types for one focal context. We suggest, for example, contrasting a range of behaviors (feeding, antipredator, contest, mating) for alternative male mating types (Shuster and Wade 2003), dispersers versus site faithful individuals (Dingle 2001), producer versus scrounger foragers (Barnard and Sibly 1981; Giraldeau and Beauchamp 1999), ambush versus active foragers (Eckhardt 1979; McLaughlin 1989), or high versus low vigilance individuals. Dominant and subordinate individuals typically differ in a suite of behaviors—e.g., in foraging, antipredator, and mating contexts. Relatively little is known, however, about behavioral syndromes for other behavioral dichotomies. For example, we suspect that dispersing individuals might often be more bold or aggressive than site faithful ones. If so, how is this reflected in a broad range of behaviors across multiple environmental situations?

In sum, while existing examples suggest that behavioral syndromes might often be very important, the basic quantitative natural history of behavioral syndromes remains to be worked out for most nonlaboratory animals. Which behaviors are correlated across which contexts, and how stable are these correlations? Given that behavioral syndromes can be important, we next discuss approaches to understanding these syndromes

Evolutionary Issues

First, we examine the evolution of behavioral syndromes per se; i.e., the evolution of behavioral carryovers (that involve less than optimal plasticity) and behavioral genetic correlations. If these generate suboptimal behaviors, why do they persist? We then address the evolution of behavioral types within a syndrome. Which behavioral types (e.g., more versus less aggressive) should be found in any given environment? What conditions allow for the maintenance of genetic and individual variation in behavioral types within or among populations? Although no extant models explicitly address the evolution of behavioral syndromes, we draw on an analogous literature on the evolution of adaptive plasticity (e.g., Scheiner 1993; Sibly 1996; Schlichting and Pigliucci 1998; de Jong and Gavrilets 2000) to generate intuitively reasonable predictions and to provide guidelines on directions for future modeling.

evolutionary persistence of limited plasticity

What mechanisms might explain the evolutionary persistence of behavioral carryovers (i.e., of limited plasticity)? Although at first glance it might seem counterintuitive, under

realistic scenarios natural selection can favor the evolution of limited plasticity. If individuals have poor information about their environment—i.e., weak covariance between environmental cues that induce plasticity and the actual selective environment—then the optimal reaction norm (given the poor information) can have a much shallower slope (less plasticity) than the optimal reaction norm, given complete information (Tufto 2000). Using a stock market metaphor, given poor information about future market changes, "staying the course" can be more profitable than attempting to predict and "play the market." For an ecological example, if prey have poor information about the presence of dangerous predators, then even if predators are only occasionally present, prey might be forced to act as if predators posed a continual threat (Sih 1992); e.g., prey might evolve group living or ambush hunting as essentially fixed responses to predators that are only occasionally present. Luttbeg and Warner (1999) similarly showed, in a mating context, that while environmental variation favors learning and behavioral responses, relatively little response is expected if individuals have poor information or if they are subject to substantial time lags. These models, however, do not consider behavioral syndromes across multiple contexts. Further models should examine the basic logic outlined above in scenarios explicitly designed to include behavioral syndromes.

Alternatively, even if selection favors the decoupling of behavioral carryovers, they might still persist if they have a proximate (e.g., a genetic or neuroendocrine) basis that is difficult to break (see Proximate Mechanisms, below). Even if the proximate mechanism can be decoupled, if that decoupling takes a long time relative to the pace of environmental change, then low plasticity is favored (Padilla and Adolph 1996). Finally, limited plasticity should be more likely to persist if its cost is mediated by compensatory traits (e.g., other behaviors or morphological traits) that reduce the cost of the behavioral carryovers (DeWitt et al. 1999), or if individuals strongly avoid situations where they behave inappropriately (i.e., situation choice). Situation choice effectively shields individuals from exposure to the costs of behavioral carryovers; e.g., "geared up" individuals that would do poorly with predators can survive by avoiding habitats with predators.

EVOLUTION OF BEHAVIORAL GENETIC CORRELATIONS

While genetic correlations among life history and morphological traits have been well studied (Roff 1992), we know less about genetic correlations among behavioral traits. Genetic correlations have been treated in two main ways in evolutionary ecology. One view, exemplified by conventional life-history theory, treats genetic correlations as a source of constraint on the evolution of optimal traits. The assumption is that the correlation reflects some underlying mechanism that is difficult to decouple even over evolutionary time. For example, a genetic correlation might be due to pleiotropy (where one gene governs two or more traits), or to a deep, underlying physiological constraint (e.g., the inherent size/number tradeoff or an energy allocation tradeoff). An alternative view assumes that proximate mechanisms, including genetic mechanisms, can themselves evolve by natural selection. For example, if pleiotropy causes a maladaptive behavioral correlation, then selection ought to alter that pleiotropy (e.g., via genetic modifiers) to decouple the fitness-reducing correlation. Overall, natural selection ought to shape optimal genetic correlations to produce adaptive phenotypes with functionally integrated suites of traits (Cheverud 1996, 2000; Wagner et al. 2000). This view has been applied to understanding the genetics governing the development of integrated morphologies (Leamy et al. 1999; Klingenberg et al. 2001).

Our discussion of behavioral syndromes primarily falls within the "constraint" view of behavioral correlations. We emphasize scenarios where underlying proximate mechanisms produce behavioral genetic correlations across contexts that generate conflicts that cause suboptimal behavior in one or both contexts. Because of the conflict, no individuals or genotypes attain the unconstrained optimum (Figure 2). Instead, many

alternative behavioral types (e.g., a mix of high and low aggression or high and low activity genotypes) might all cope equally well—all exhibiting variations on "making the best of a bad situation." In contrast, the "optimal genetic architecture" view posits that natural selection should break up deleterious behavioral correlations, so that behaviors should be either decoupled (and thus free to evolve to the optimum in all environments) or correlated in an adaptive way. An adaptive behavioral correlation could reflect alternative optimal strategies for coping with two situations. For example, as noted earlier, male crickets that have longer calling bouts when predators are absent compensate by exhibiting stronger antipredator behavior when predators are present (Hedrick 2000); males with shorter calling bouts exhibit less response to predators. Although no explicit theory exists on the evolution of behavioral genetic correlations and behavioral syndromes, it should depend presumably on an interplay between the strength of selection (against or favoring the correlation) and the ease of decoupling the mechanisms underlying behavioral correlations.

WHICH BEHAVIORAL TYPE SHOULD BE FAVORED?

Optimality models predict optimal behaviors across a broad range of environmental contexts (Krebs and Davies 1996; Dugatkin and Reeve 1998; Houston and McNamara 1999; Clark and Mangel 2000). These models assume that genetic mechanisms governing behavior do not constrain the power of natural selection to drive the evolution of optimal behavior. Simple quantitative genetic models of adaptive plasticity confirm that although genetic correlations can influence evolutionary trajectories, ultimately if there are no limits to plasticity selection should indeed result in the evolution of optimal traits in all environments (Via and Lande 1985; Gomulkiewicz 1998). Behavioral syndromes and, in particular, behavioral carryovers, can produce a limit to plasticity, however. If constraints prevent individuals and genotypes from exhibiting optimal responses to environmental variation, which behavioral

types should be favored by selection in a particular environment? For example, consider prey activity in the absence and presence of predators. Prey that are more "geared up" feed and grow rapidly in the absence of predators but suffer high mortality when predators are present, while "geared down" prey survive relatively well when predators are present but do poorly in the absence of predators (e.g., Sih et al. 2003). Prey with intermediate activity profiles presumably show intermediate fitness in both habitats. What conditions should favor the evolution of "geared up" versus "geared down" versus intermediate activity types?

In theory, the fitness isocline method (Levins 1968; Leon 1993) could provide some insights (Figure 3). This method has been used to address optimal life histories (Pianka and Parker 1975; Roff 1992), given tradeoffs that produce a negative correlation between traits (e.g., between current reproduction and future survival) that are captured in a constraint function (Figure 3a). Only life-history combinations that are on or below this function are possible. Fitness isoclines are lines of equal fitness on a fitness landscape where overall fitness is higher for lines that are further from the origin (that have higher current reproduction and higher future survival). The optimal life history (indicated by the star) is found by looking for the point on the constraint function that yields the highest fitness. A conceptually similar construct has been used to analyze optimal diets of herbivores (via linear programming: Belovsky 1984), and in theory could be used to examine time budget conflicts in behavioral ecology, in general.

Behavioral syndromes, however, differ from the scenario in Figure 3a in two ways: 1) traits are often positively, rather than negatively, correlated; and 2) due to stabilizing selection (i.e., that accounts for time budget conflicts within each environment), there is an optimal behavior in each environment (Figure 3b). Despite these differences, the method still allows us to visualize the *optimal behavioral types* (represented by the double line) as the intersection between the constraint function (due to the behavioral carryover) and the highest attainable point on a
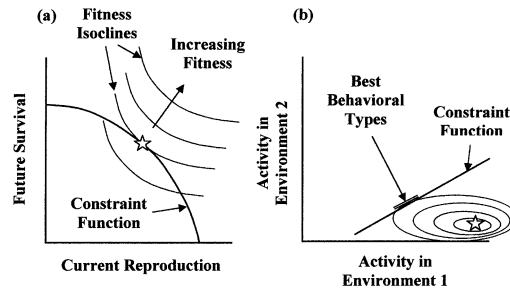
FIGURE 3.   FITNESS ISOCLINES AND SELECTION ON CORRELATED TRAITS
    A fitness isocline view of selection on correlated traits. (a) A standard view on the evolution of life histories given a cost of reproduction that results in a negative correlation between current reproduction and future survival (represented by the constraint function). Fitness isoclines are lines of equal fitness. Higher survival and reproduction yields higher overall fitness. The optimal life history is represented by the star. (b) An analogous view for behavioral syndromes that produce a positive correlation (shown by the constraint line) between activity in two environments (taken from Figure 2). The concentric ovals show a fitness landscape with a maximum (optimum in the absence of behavioral carryovers) at the star. The shaded region on the constraint function indicates the best behavioral types (all with similar fitness) given the behavioral carryover.

fitness landscape. In our example, a range of behavioral types yield similar highest fitness. To emphasize, while these optimal behavioral types exhibit suboptimal behavior (relative to an infinitely plastic optimum) in every situation, they still represent the "best of a bad situation" in that they show the best available strategy given the constraint of limited plasticity associated with the behavioral syndrome.

    While the fitness isocline method has some heuristic value, progress in modeling behavioral syndromes will require more explicit models that formalize specific scenarios and assumptions. Some guidelines can be found in models of adaptive plasticity (often life-history plasticity) in a variable environment (e.g., Houston and McNamara 1992; Moran 1992; Kawecki and Stearns 1993; Sibly 1995, 1996; Zhivotovsky et al. 1996; de Jong and Gavrilets 2000). In these models, the usual scenario is that individuals experience a developmental environment X that determines a focal trait that then influences fitness at a later time in the selective environment Y. For example, depending on whether individuals develop in the presence or absence of predators, they induce morphologies, life histories, or behavioral tendencies that then influence their subsequent fitness. These models typically assume that plasticity is irreversible. If the early environment induces a

trait that is not well adapted to the later selective environment, individuals cannot further adjust their trait. For example, if individuals grow up with predators and induce a spine (Tollrian and Harvell 1999), they cannot lose that spine even if they end up in a predator-free habitat where the spine reduces competitive ability. This clearly differs from the usual assumption in behavioral ecology that behaviors are infinitely plastic and reversible; however, it might not be unrealistic for behavioral syndromes. An individual's early experiences might shape its behavioral type that then results in relatively fixed (or at least less than infinitely plastic) behavioral tendencies across a range of contexts.

    The evolution of adaptive plasticity in the above scenario then depends heavily on the match (or mismatch) between the environments of development and selection (statistically, the covariance between X and Y) (de Jong and Gavrilets 2000). X and Y can be mismatched either because of spatial or temporal variation in the environment. Alternatively, even in a nonvarying environment, the focal trait can be mismatched to Y if X provides imprecise cues on Y (e.g., Sih 1992; Getty 1996; Tufto 2000). Drawing from models of adaptive plasticity, we suggest the following predictions on the evolution of behavioral types. First and fundamentally, the expected behavior in any given situation

depends on selection in all situations that are part of the same behavioral syndrome. That is, selection in each environment affects the evolution of behavioral types that then carries over to influence behavior in all other relevant situations. All else the same, the optimal behavioral type should be more heavily influenced by selection in: 1) environments that individuals experience more frequently; 2) environments with a stronger selection gradient—a stronger effect of behavior on fitness; and 3) higher quality environments—e.g., selection in source habitats outweighs selection in sink habitats (Holt and Gaines 1992; Holt 1996). Thus, for example, prey are likely to evolve a "geared up" activity type that does well in the absence of predators, even though it fares poorly with predators present (e.g., Sih et al. 2000, 2003), if prey spend most of their time in habitats without predators, if activity has a relatively weak relationship to risk, and if predators are so effective that habitats with predators are evolutionary sinks.

Given that the pattern of environments encountered by individuals matters, the evolution of behavioral types should depend on situation selection (Holt 1996; Zhivotovsky et al. 1996). If individuals choose situations that they do particularly well in, then as noted earlier this reduces the fitness costs of limited plasticity. Situation choice could drive the evolution of behavioral specialists, each primarily using their best habitats, with low genetic variation in behavioral types within each situation, but possibly the maintenance of high individual variation in the overall metapopulation.

Although the above predictions seem reasonable, we need theory developed specifically for addressing behavioral syndromes. Specifically, rather than assume that each individual goes through one cycle of irreversible, plastic response in each generation (the usual assumption in models of adaptive plasticity), we need models that allow for multiple episodes of reversible plasticity. Rather than assume instantaneous, infinite plasticity (the usual assumption in optimality models), we need models that include time lags and limits to plasticity (limits to the slope and perhaps height of plasticity functions). Padilla and Adolph (1996) considered two of these con-

siderations—reversible plasticity with time lags. They focused on conditions where plasticity is favored over fixed traits that are optimal for one environment but costly in another. Not surprisingly, they found that plasticity is favored if time lags are short relative to the rate of environmental change (see also Levins 1968). Their model allowed the plastic genotype to exhibit the optimal trait in each environment; i.e., they did not include a limit to plasticity, a notion that is at the heart of the idea of behavioral carryovers. Future work adding behavioral carryovers to models that examine reversible plasticity with time lags should be insightful.

MAINTENANCE OF VARIATION IN BEHAVIORAL SYNDROMES

The maintenance of variation is a major issue in evolutionary biology, in general (Futuyma 1998). Individual variation also has important ecological implications (Bolnick et al. 2003). Despite this, in behavioral ecology, the tradition has generally been to ignore individual variation and instead emphasize shifts in mean behavior in response to changing environments. Indeed, in a simple, pure optimality view, over time natural selection should result in little or no genetic variation among individuals—all individuals should be optimal in all environments. In reality, individual variation in behavior is probably ubiquitous (Clark and Ehlinger 1987; Wilson 1998). Some of this variation might persist because multiple optima exist within a single environment. Optimal behaviors depend on a balance of conflicting demands (tradeoffs). If individuals differ in their tradeoffs—e.g., due to differences in individual state (condition, energy reserves, size)—they can exhibit different optimal behaviors within the same environment (Houston and McNamara 1999; Clark and Mangel 2000; Mangel and Stamps 2001). Coexistence of multiple behaviors is probably often further enhanced by negative frequency dependence that has been modeled using game theory (Maynard Smith 1982; Dugatkin and Reeve 1998).

We suggest that behavioral syndromes might also play an important role in enhancing the maintenance of individual variation

in behavior. The basic rationale is simple. If behavioral carryovers are important, then no individual exhibits the optimal behavior in all situations. Different individuals should be best suited for different situations—all of which are part of the organism's overall life. Bold individuals might do well in some social or ecological situations, while shy individuals do well in others. A range of behavioral types can coexist in the long-term given suitable variation in contexts and environments (see Figure 3b where a range of behavioral types have similar fitness). The logic is analogous to theory on the persistence of behavioral or life-history polymorphisms in a variable environment; e.g., coexistence of specialists and generalists (Moran 1992; van Tienderen 1997), or of high mean/high variance versus low mean/low variance life histories (Orzack and Tuljapurkar 2001). One can think of it as "behavioral niche partitioning" (different types do well in different situations) in a variable environment.

Just as frequency dependence can enhance the coexistence of alternative mating, contest, or foraging tactics, the persistence of individual variation in behavioral types (e.g., different coping styles, aggression, or activity types) should presumably be increased by frequency dependent selection. Coexistence could involve mixed ESSs (where all types have equal fitness), or condition dependent strategies where high condition individuals attain higher fitness than low condition ones, but all exhibit a behavioral type that is a best solution given the individual's condition. Behavioral syndromes that revolve around social interactions (e.g., aggression, proactivity) where outcomes, benefits, and costs likely depend on the population's mix of behavioral types (e.g., on whether most individuals are highly aggressive, or unaggressive, or a mix of both) seem particularly ready for game theory analyses.

The above mechanisms based on multiple selective optima can have, but need not have, a genetic basis; e.g., behavioral variation due to condition dependence is often thought of as nongenetic. Based on a long history of evolutionary theory, genetic variation can be maintained by a variety of mechanisms including a mutation/selection balance, migration/selection balance, frequency dependence, and overdominance (Barton and Turelli 1989; Burger 1998; Hedrick 2000). The most relevant theory for behavioral syndromes should be the theory on mechanisms maintaining genetic variation in adaptive plasticity. Models on this issue confirm the usual expectations based on nonplastic traits (e.g., Via and Lande 1987; de Jong and Gavrilets 2000). Interestingly, de Jong and Gavrilets (2000) also predicted that greater environmental variation should actually reduce the amount of genetic variation in plasticity (e.g., in slopes and heights of plasticity functions) within populations. Their logic is that greater environmental variation exposes genotypes to potentially more serious mismatches resulting in stronger selection against suboptimal reaction norms.

OTHER EVOLUTIONARY CONSIDERATIONS

An exciting future area should be analyses of the joint evolution of behavioral syndromes and other fitness-related traits (e.g., life histories, morphology). This takes the "deatomizing" of traits a step further. Rather than simply look at correlated *behaviors* across contexts, we can look at functional integration of several types of traits across situations. A few studies have indeed focused on syndromes of multiple types of traits (e.g., Endler 1995; Dingle 2001). As with most aspects of behavior, however, previous studies have emphasized the joint evolution of morphology and behavior in one particular context (e.g., Brodie 1992; Smith and Skúlason 1996; DeWitt et al. 1999). A broadening of this approach to consider more behavioral contexts (predator-prey, aggression, mating, dispersal) will be rewarding; i.e., how do behavioral syndromes coevolve with other traits?

The existence of behavioral syndromes and their joint evolution with morphology and other traits could facilitate speciation (Wcislo 1989; Wilson 1998). In brief, if different behavioral types within a species evolve associated differences in morphology and other traits, this could enhance both disruptive selection against hybrids and assortative mat-

ing by behavioral type eventually resulting in speciation. For example, active versus sit-and-wait foragers within one species (that might also differ in their antipredator, mating, contest, and dispersal behaviors) should evolve different morphologies, physiologies, and life histories. Individuals that exhibit intermediate traits in this entire suite might generally fare poorly. In addition, the overall differences in a suite of traits could reduce mating encounters between the types (e.g., if they live in different habitats or evolve different life-history phenologies), and favor positive assortative mate choice. This basic scenario for speciation has been discussed in the context of trophic polymorphisms (Wilson 1998), but might apply more broadly to behavioral syndromes (and their other associated traits), in general.

Finally, an exciting future direction involves tracking the evolution of behavioral syndromes by contrasting behavioral types for related taxa in an evolutionary ecological framework (e.g., Dewsbury et al. 1982; Riechert and Hedrick 1993; Gosling 2001; Sih et al. 2003). We hypothesize that differences between species in behavioral type could have major effects on their ecology and evolution (see Ecological Implications). For example, work by Lefebvre and colleagues using a blend of the comparative method and experiments on focal species have revealed that bird taxa with large brains (relative to body size) tend to be less neophobic, better at problem solving, more likely to exhibit feeding innovations, and show greater evolutionary diversification and ecological invasiveness (Lefebvre et al. 1997; Sol and Lefebvre 2000).

In conclusion, formal theory on the evolution of behavioral syndromes remains to be developed. We first need simple models that address reversible plasticity in scenarios with limits to plasticity and perhaps correlations between the means and heights of reaction norms (i.e., specialists with more extreme mean traits should have less plasticity than generalists that have intermediate mean traits). We can then add other major elements of behavioral ecology (e.g., games, state dependence, explicit tradeoff functions), as well as more detailed aspects that are important for understanding limited plas-

ticity (e.g., response lags, imprecise information). Finally, future models should incorporate what is known about underlying mechanisms: genetic or physiological. A later set of models could look at the joint evolution of behavioral syndromes and other functionally related traits (e.g., morphology, life history).

PROXIMATE MECHANISMS

Although a thorough understanding of the mechanisms that underlie behavioral syndromes is important in its own right, we focus in particular on several key mechanistic issues that have potentially powerful evolutionary and ecological implications. Specifically, we address the role of proximate mechanisms in determining the existence and stability of behavioral correlations and limits to plasticity.

To understand the existence and stability of behavioral correlations, a critical issue is whether the behaviors are governed by *common* versus *independent* mechanisms. Consider a simple case where three behaviors, A, B and C, are positively correlated: individuals that are the most aggressive toward conspecifics (A) are also the boldest toward predators (B) and the most voracious foragers (C). In one possible scenario, A and B are both governed by mechanism X, while C is governed independently by mechanism Y (Figure 4a). Mechanism X might be a single gene with effects on multiple traits (pleiotropy), a hormone that affects more than one behavior, or an experience that influences multiple aspects of the phenotype. In this case, conditions that favor an increase in A will indirectly cause a change in B, and the correlation between A and B cannot be uncoupled without changing the underlying mechanism. In contrast, the correlations between A-C or B-C—e.g., due to a correlation, but not a causal link, between X and Y—are more easily broken apart because they are controlled by separate factors. For example, whereas both aggression toward conspecifics (A) and boldness to predators (B) might be affected by levels of a gonadal steroid, foraging behavior (C) might be regulated by an independent hormonal mechanism. In that case, we would
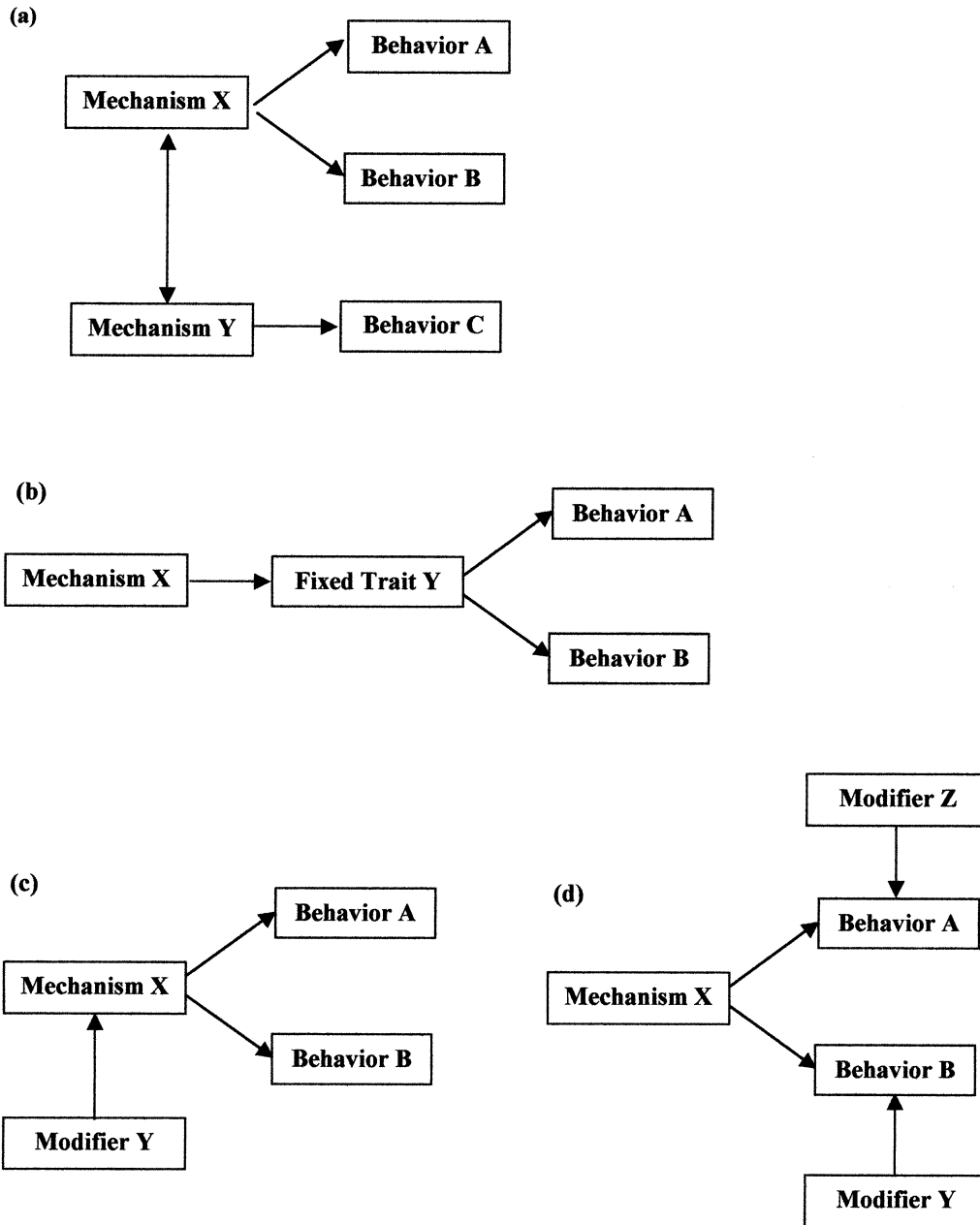
FIGURE 4.   MECHANISMS THAT GENERATE OR DECOUPLE BEHAVIORAL SYNDROMES

Mechanisms that can generate or decouple behavioral syndromes: (a) Behaviors A and B are governed by a common mechanism (e.g., pleiotropy), while behavior C is governed by a separate, independent mechanism Y. If X and Y are correlated (e.g., via linkage disequilibrium), this could result in correlations between A, B, and C. A and B should be difficult to uncouple, whereas it should be relatively easy to decouple C from A and B. (b) Mechanism X governs a fixed trait Y that then produces limited plasticity in behavior A. (c) The effect of a common mechanism X on behaviors A and B can be influenced by modifier Y; e.g., differential epistasis. (d) Effects of a common mechanism X on behaviors A and B are modified by independent modifiers Y and Z; e.g., tissue-specific hormone receptors.

expect the correlation between aggression and antipredator behavior to be more stable than the correlation between foraging behavior and aggression or antipredator behavior. Although, in reality, proximate control of behavioral syndromes likely involves complex webs of several interacting components that include multiple genes, experiences, and hormones, the key to the tightness of behavioral correlations should be the tightness of the interdependencies among these components.

To understand how proximate mechanisms might underlie limited behavioral plasticity, we focus on two main lines of logic. First, limited behavioral plasticity can be due to a tight (perhaps adaptive) connection between a relatively fixed trait (e.g., morphology or neurophysiology) and behavior (Figure 4b). For example, low activity individuals might lack the morphological or physiological capacity to move efficiently, and thus might be relatively inactive even when high activity is otherwise favored. Second, within-individual consistency in behavior could be the result of a behavioral positive feedback loop, often driven by learning. Choice of a given behavioral lifestyle in the short term might predispose individuals via learning to continue that lifestyle. Both of these mechanisms can cause time lags in an individual's behavioral response to environmental change; it can take time for individuals to learn that the environment has changed and to learn to adapt to a new environment, or behavioral plasticity might depend on an underlying physiological or morphological trait that takes time to change. As noted in the earlier section on the evolution of limited plasticity, long behavioral response lags (relative to the rate of environmental change) can favor limited plasticity (Sih 1992; Padilla and Adolph 1996). For both lines of logic, it should be useful to understand how they are governed by underlying mechanisms.

Below, we discuss three major types of mechanisms that likely influence behavioral syndromes: genetic, environmental experience, and neuroendocrine effects. We briefly review literature from diverse fields, many of which are relatively rarely read by behavioral ecologists (e.g., mouse and fly behavioral genetics, applied animal science, human psychology and behavioral genetics, behavioral endocrinology). We first discuss each type of mechanism separately, and then conclude by emphasizing that they interact; both genetics and experience often influence neuroendocrine mechanisms that govern behavior. Ultimately, an interdisciplinary approach that integrates these mechanisms should prove most powerful.

GENETICS

Information about the genetic basis of behavioral correlations and limited behavioral plasticity is critical for understanding the evolution of behavioral syndromes. Here, we discuss a broad range of genetic approaches that have been applied to these issues, albeit almost exclusively in a few model systems. In most taxa, little or no information exists on the genetics of behavioral syndromes.

Standard quantitative genetic methods (Lynch and Walsh 1998) can be used to measure behavioral genetic correlations and the heritability of behavioral types (Boake 1994). Given that behavioral syndromes involve connecting behaviors across environments, key issues are the genetic basis of and genetic variation in response to environmental variation, or reaction norms and gene x environment interactions (Via and Lande 1985; Via et al. 1995). The quantitative genetics of behavioral syndromes has long been a focal issue in the behavioral genetics of a few model systems (laboratory mice: Henderson 1986; Koolhaas et al. 1999; humans: Bouchard and Loehlin 2001). In laboratory mice, for example, parent-offspring regressions have quantified behavioral genetic correlations, and long-term selection lines have produced correlated changes in suites of behaviors (Sluyter et al. 1995; Bult and Lynch 2000). Similarly, in several domesticated species, artificial selection produced evolutionary changes in behavioral types such as fearfulness or tameness (Trut 1999).

Many aspects of the genetics of behavioral syndromes remain to be studied, however. Most importantly, only a few studies have looked at even the most basic aspects of the genetics of behavioral syndromes in nondo-

mesticated animals (Palmer and Dingle 1989; Riechert and Maynard Smith 1989; Dingemanse et al. 2002; Drent et al. 2002). Other quantitative genetic issues that are important, in theory, but are largely unstudied include the genetics of limited plasticity and the genetics of situation choice (e.g., a tendency for bold individuals to choose risky environments). To understand the evolution of limited plasticity, key issues are the heritability of plasticity per se (Schlichting and Pigliucci 1995) and the possibility that low behavioral plasticity might be genetically correlated with other relatively fixed traits (e.g., morphology). To measure a genetic tendency for a given behavioral type to experience some environments more than others—e.g., either due to situation choice by the focal individual or because its parents had a genetic tendency to provide a particular rearing environment—the relevant metric is the gene x environment correlation. While the behavioral G x E correlation has been well studied in humans (Rutter and Silberg 2002), it remains largely unstudied in other species.

Standard quantitative genetics provides only a statistical snapshot of a potentially dynamic, evolving genetic system. To better understand the mechanisms governing the evolution of behavioral syndromes, it is useful to know the genetic architecture that underlies genetic correlations and limited plasticity. The number of loci affecting behaviors and their relative importance, pleiotropy, dominance interactions among alleles at one locus, and epistatic interactions among loci (Cheverud 1996) might all influence behavioral syndromes. As noted above, a simple idea is that the stability of a genetic correlation between two behaviors should depend on whether it is caused by a common or independent genetic mechanism. A behavioral genetic correlation due to pleiotropy (a common mechanism for the two behaviors) should be harder to decouple than one based on linkage disequilibrium (a statistical relationship between mechanistically independent genes) (Figure 4a). However, genetic correlations due to pleiotropy can, at least in theory, also be broken up by selection on modifiers (i.e., other loci) that alter the plei-

otropy (Greenspan 2001). This possibility is referred to as differential epistasis (Cheverud 1996; Wagner et al. 2000; Figure 4c).

To estimate the number of loci and their relative importance in determining a given relationship, an invaluable tool is quantitative trait locus (QTL) analysis. If only a few major QTLs control a trait, a goal is to identify candidate genes, their function, and interactions among them. If focal behaviors are controlled by many QTLs of small effect, then a key issue is the pattern of pleiotropy underlying genetic correlations. Are all the correlations determined by one set of pleiotropic loci, or are different correlations governed by largely independent modules of pleiotropies (Cheverud 1996; Mezey et al. 2000). In theory, natural selection through differential epistasis could shape the genetic architecture to produce optimal sets of functionally integrated suites of traits (Cheverud 1996; Wagner et al. 2000) or optimal behavioral syndromes. Note that the "optimal" behavioral syndrome could still involve limited behavioral plasticity and apparently suboptimal behavior if, for example, information constraints prevent even the best individuals from rapidly tracking a changing environment (see sections on the evolution of limited plasticity and the sections on experience and neuroendocrine mechanisms). Some recent studies on model systems have indeed shown that behavioral genetic correlations can evolve (Bult and Lynch 2000), and other studies have applied QTL methods to examine the genetics of behavioral syndromes (Flint et al. 1995; Toye and Cox 2001; Turri et al. 2001). In particular, Turri et al. (2001) found that "anxiety" in mice might be the outcome of at least three separate behavioral tendencies—low activity per se, avoidance of aversive stimuli, and low exploration—governed by QTLs on separate chromosomes.

Finally, molecular genetic methods have revealed numerous single-gene effects on suites of behaviors in a few model systems (laboratory mice: Greenspan 2001; Bućan and Abel 2002; *Drosophila:* Sokolowski 2001). Perhaps the best example of a naturally occurring, single-gene behavioral polymorphism involves the *for* gene that influences

foraging activity in *Drosophila melanogaster* (Pereira and Sokolowski 1993). In *D. melanogaster,* larvae homozygous for the *for*R allele (rovers) have considerably longer foraging trails than individuals homozygous for the *for*s allele (sitters). Rovers and sitters differ in ways beyond larval feeding rate. Relative to sitters, rovers exhibit a higher tendency to encapsulate parasitoid wasp eggs (Hughes and Sokolowski 1996), and the higher activity of rovers persists into adulthood even though adults and larvae have very different means of locomotion and feeding. The fitness of rovers versus sitters depends on fly density—rovers are favored under high densities and sitters in low densities (Sokolowski et al. 1997)—and on wasp parasitism rates (Hughes and Sokolowski 1996).

Recent reviews on the genetics of behavior in laboratory mice (Greenspan 2001; Bućan and Abel 2002) and *Drosophila* (Sokolowski 2001) emphasize, however, some important limitations of molecular genetic studies. First, a methodological problem is that many molecular genetic studies involve drastic changes in gene expression (e.g., complete knockouts) that very possibly have qualitatively different effects on behavior than more subtle genetic variation. More relevant insights might come from further studies on the behavioral effects of relatively small changes in gene expression (e.g., hypomorphic mutations) or localized tissue-specific changes in gene expression (Greenspan 2001). Even more fundamental is the growing evidence that effects of any given single gene on behavior depend heavily on the genetic background (epistasis). For example, the effect of a given knockout mutation varies among genetic lines (Bućan and Abel 2002; Crabbe 2002). Behavioral syndromes appear to be under polygenic control with widespread pleiotropy and epistasis. In that case, molecular genetic studies that focus on one or a few genes might not be an efficient method for understanding the genetics of behavior. DNA microarrays that simultaneously assess gene expression for thousands of loci might yield suggestions for further directions to pursue, though the problem of interpreting microarray data is clearly nontrivial.

EXPERIENCE

In most cases, behavioral syndromes are likely to be determined not just by genes but also by individual experiences. What role does experience play in explaining differences among individuals in behavioral type, or changes in behavioral type over a lifetime? Conversely, how do behavioral types differ in the ways in which they learn from their experiences? Again, these issues have only been addressed in a few animals (e.g., humans and other primates). Below, we discuss some ideas drawn from either the limited literature on experience and behavioral syndromes (see Stamps 2003 for a more detailed review), or from the extensive literature on the effects of experience on a single behavior.

Experiences over the course of ontogeny can either produce or break up behavioral syndromes. On the one hand, experiences can start an individual on an ontogenetic trajectory that sets its later behavioral type. For example, stressful or traumatic early juvenile experiences can cause an individual to be anxious or fearful for the rest of its life. On the other hand, experience can also reshape or modify a behavioral syndrome. For example, while aggressive behavior might be correlated with antipredator behavior prior to any experience with a predator, subsequent experience with a predator might decouple this correlation by changing the individual's antipredator behavior without affecting aggressive behavior in the absence of risk.

The role of experience in building versus breaking up behavioral syndromes likely depends on the timing of experience over ontogeny. Early experiences might often build behavioral syndromes; that is, variations in early experience might produce individual differences in behavioral types. In species with extended parental care, parental behavior can obviously play in important role in determining the behavioral types of their offspring. For example, interactions between mothers and infants influence the development of personality in rats (Meaney 2001) and rhesus monkeys (Stevenson-Hinde et al. 1980; Suomi 1987). Even in species without extended parental care, parental choices (e.g., oviposition site choice or parental

investment in eggs) can have major impacts on offspring behavior. For example, maternal choices can produce small differences in the growth rate and behavior of juveniles that ultimately result in the development of discrete alternative mating strategies (Caro and Bateson 1986; Gross 1996; Emlen 1997). Of course, postnatal social interactions can also influence behavioral types. In house mice, juvenile coping styles are shaped more by their postnatal social environment (sex ratio: Benus and Henkelmann 1998) than by their mother's coping style (Benus and Röndigs 1997). In general, because the social environment experienced by developing organisms often influences their subsequent social behavior and life history (e.g., Rodd and Sokolowski 1995; White et al. 2002), we predict that future studies will reveal important effects of the social environment on the development of behavioral syndromes.

Two basic mechanisms could underlie a tendency for early experiences to canalize subsequent behavioral types. One mechanism involves the effects of early experience (e.g., habitat use) on the development of relatively fixed traits (e.g., morphology, physiology: Wainwright et al. 1991) that set an individual's subsequent lifestyle and behavioral type. Alternatively, early experiences might set off a positive feedback loop involving learning and tradeoffs between performance in different lifestyles (Immelmann 1975). If individuals that have learned a particular lifestyle cannot easily learn a new one, or if the cost of switching is large, then they might be constrained to maintain their current lifestyle.

Behavioral types, however, are not necessarily set for life. Some inference on the role of later experiences in altering behavioral types can be gleaned from the literature in comparative psychology on the stability of personalities over the life course. Ongoing experience can either enhance stability by reinforcing a package of traits, or experience might modify personality. Most studies looking at the stability of personality have found that some aspects of personality are stable while others are not (dairy goats: Lyons et al. 1988; wolves: MacDonald 1983; great tits: Verbeek et al. 1994, rhesus monkeys: Suomi et al.

1996; Capitanio 1999). From an evolutionary ecological perspective, it seems likely that selection should uncouple a behavioral syndrome through ontogeny when environmental conditions experienced by juveniles differ substantially from those experienced by adults, particularly if a behavioral syndrome that works well in juveniles does not work as well in adults. We might thus expect to see a decoupling of behavioral syndromes in species with complex life cycles or those which experience ontogenetic niche shifts during development (e.g., holometabolous insects, anurans).

While the above discussion has focused on how experience influences behavioral syndromes, the interaction also goes the other way: behavioral syndromes can also shape experience and influence learning. Interestingly, the relationship between behavioral types (e.g., boldness) and speed of learning varies, depending apparently on the syndrome and the type of task to be learned. One view is that bolder individuals experience more of their environment and thus learn more rapidly. Some studies indeed show that neophobia (avoidance of novel stimuli) is associated with slow learning of foraging tasks in birds (Greenberg 1990; Seferta et al. 2001; Webster and Lefebvre 2001). The literature on the proactive-reactive axis, however, emphasizes the opposite: proactive individuals (more aggressive, active, and bold) tend to form set routines and learn about environmental changes more slowly (Verbeek et al. 1994, 1996; Koolhaas et al. 1997). Proactive individuals try to manipulate situations, rather than react to them. In colloquial terms, they bluster through life at high speed and do not appear to notice subtle changes in their environment. In contrast, reactive individuals adapt to situations; they are more sensitive to environmental changes. Putting the two views together, it appears that bolder individuals might be better at learning novel tasks, while more shy and reactive individuals are better at sensing environmental changes within a familiar task. More work, however, is clearly needed to clarify generalities on the relationship between behavioral syndromes and learning.

In summary, much remains to be learned

about the effects of experience on behavioral syndromes outside of primates. Further empirical and theoretical work should address when experience is limiting versus flexible, and how the salience, duration, and timing of experience affect behavioral syndromes. A particularly exciting avenue might be the ongoing feedback between behavioral syndromes and learning.

NEUROENDOCRINE MECHANISMS

Because hormones regularly act on multiple target tissues, thus mediating "suites of correlated phenotypic traits" (Ketterson and Nolan 1999), it seems plausible that hormones might generate behavioral syndromes (e.g., testosterone levels might underlie aggression syndromes). Some studies indeed show associations between hormonal profiles and behavioral types. For example, different coping styles in house mice (*Mus musculus)* are associated with different neuroendocrine profiles. Proactive individuals show low hypothalamic-pituitary-adrenal axis reactivity in response to stress (low plasma corticosterone response) but high sympathetic reactivity (high levels of catecholamines), while the reverse is true for reactive individuals (reviewed in Koolhaas et al. 1997). In addition, there are differences between the two coping styles with respect to gonadal activity, disease vulnerability, and stress pathology. Other studies have measured the neuroendocrine basis of reactivity or fearfulness in other mammalian species (reviewed in Boissy 1995), and neuroendocrine correlates of the migratory syndrome (flight, delayed reproduction, flight muscle development) in insects (Fairbairn 1994; Zera and Denno 1997; Dingle 2001).

Recent work in behavioral endocrinology emphasizes, however, that the relationship between hormones and behavior is often far more complex and plastic than the simple notion that circulating levels of hormones govern behavior (Hews and Moore 1997; Ketterson and Nolan 1999). Hormonal impacts on behavior depend on many mediating factors. For example, variability in hormonal receptors (receptor density, specificity, binding affinities), metabolic properties of hormone synthesis and breakdown, binding globulins, and hormone-hormone interactions can all influence how a given hormone is related to a particular behavior or behavioral syndrome (Figure 4d). These mediating factors ought to allow for an easier decoupling of the tendency for hormones to produce behavioral syndromes.

For example, selection has apparently decoupled trait expression from the underlying hormonal pathway in the sex-role-reversed Wilson's phalarope (*Phalaropus tricolor*). Female aggression occurs without increases in plasma testosterone levels (Fivizzani et al. 1986). Instead, female phalaropes apparently increase their aggression levels by increasing levels of androstenedione, a testosterone precursor (Fivizzani et al. 1994). Selection acting independently on target tissue characteristics, such as enzyme conversion of androstenedione into testosterone, or variation in target-tissue receptors should yield fewer correlated responses than selection acting on overall levels of a circulating hormone such as testosterone. Altering the level of a hormone that mediates a suite of traits thus could be an effective way to change the timing of a syndrome, while altering receptors is more effective at changing individual traits. Situations where behavior seems to no longer be under the control of a traditional hormonal pathway should allow us to better understand evolutionary lability in the endocrine system that allows some decoupling of the correlated effects of a circulating hormone on a behavioral syndrome (Hews and Moore 1997).

Although the pathway from hormones to behavior can be complex, some insights can be gained by classifying hormonal effects into two main categories: organizational effects versus activational effects (Arnold and Breedlove 1985; Moore et al. 1998). Organizational effects are usually thought to act early in development by organizing brain anatomy and neurochemistry, and other aspects of morphology or physiology, that set the stage for later hormonal effects on behavior. Organization is thus traditionally associated with effects that are fixed for life (e.g., primary sexual differentiation through sex-specific gonadal development), or at least for some

significant amount of time. In contrast, activational effects of hormones are usually thought to act later in life; e.g., adult mating behavior might be activated by a hormonal surge. Both organizational and activational effects can come into play in a given system, although their relative importance likely varies across systems. In the context of alternative mating tactics, Moore et al. (1998) proposed a "relative plasticity hypothesis" that posits that organizational effects of hormones early in development produce fixed alternative phenotypes, while activational effects later in life govern plastic alternative phenotypes. Both effects can be important in one system. Once a male has been fixed into an organizational strategy (e.g., territorial versus satellite), activational hormone effects trigger the short-term expression of type-specific behavior patterns (e.g., sedentary satellite behavior versus wide-ranging mate search).

The comparison between organizational versus activational effects of hormones on single traits can be applied to behavioral syndromes. Organizational effects of hormones early in development often involve hormonal control of the development of fixed traits (e.g., morphology) that can then limit the range of behavioral plasticity and produce correlations that endure through ontogeny. In fact, recent work shows that early exposure to exogenous hormones from littermates (Clark and Galef 1995, 1998) and from the mother (Schwabl 1993, 1996; Adkins-Regan et al. 1995; Gil et al. 1999) can influence subsequent behavior.

In contrast, activational effects of hormones occurring later in development likely produce correlations of a shorter duration, which are more easily altered by modifications of the hormonal pathway, such as seasonal variations or tissue-specific sensitivity of hormone receptors (Soma et al. 2000; Tramontin and Brenowitz 2000). While activational effects, by definition, tend to involve less widespread impact on fixed traits, they might still limit behavioral plasticity in the short term whenever hormonal plasticity is less or slower than optimal behavioral plasticity. For example, levels of aggression in parental care and territorial defense might both be influenced by testosterone levels (Wingfield et al. 1990). If the animal moves quickly between the two contexts, but hormone levels change relatively slowly, the mechanism is not as plastic as it needs to be for the animal to instantly behave optimally in both situations. Aggression in the territorial defense context could then "spillover" into the parental context, resulting in behavior that might appear maladaptive if aggression was not viewed as a syndrome of correlated behaviors expressed in multiple contexts.

Most extant work on the neuroendocrine basis of behavioral syndromes has been done on model systems and domesticated animals, and has generally not addressed the evolutionary and ecological implications of hormones and behavioral syndromes. A line of research that looks particularly promising involves manipulating hormonal levels (at either an organizational or activational stage) to "phenotypically engineer" phenotypes outside the range of natural variation to investigate the causes and consequences of variation in behavior (Marler and Moore 1988; Sinervo and Huey 1990; Ketterson et al. 1996; Ketterson and Nolan 1999; Bell 2001). Further elucidation of the complex pathways that influence hormonal effects on behavior, including study of the many mediating factors and feedbacks, should reveal much about how and when behavioral syndromes are either generated or uncoupled.

INTEGRATING PROXIMATE MECHANISMS

Two themes that emerge are that: 1) while proximate mechanisms underlying behavioral syndromes have been studied in considerable detail in a few model systems, they remain largely unstudied in most animals; and 2) while a simple view of proximate mechanisms might suggest that they can easily generate behavioral correlations, in reality behavior might often be governed by complex regulatory networks that can, in principle, break up behavioral syndromes. Despite the potential for mechanistic decoupling of behavioral syndromes, these syndromes clearly exist and are likely widespread (Gosling 2001).

Although we discussed the three types of proximate mechanisms separately, they

clearly interact. Genes, experiences, and their interactions all influence neuroendocrine phenomena that, in turn, affect suites of behaviors. Behavioral geneticists studying model systems have emphasized how genes influence cellular and neuroendocrine mechanisms that underlie suites of behaviors (laboratory rodents: Boissy 1995; Koolhaas et al. 1999; Bućan and Abel 2002; *Drosophila:* Sokolowski 2001). For example, allelic variation in the *for* gene (the rover-sitter polymorphism) in *Drosophila* is associated with differences in expression of a cGMP-dependent protein kinase (PKG: Osborne et al. 1997) and in neurophysiology (Renger et al. 1999) that plausibly explain the behavioral differences. Individual experience also affects behavioral syndromes in these model systems; however, geneticists typically attempt to minimize (as opposed to study) the effects of environmental experiences on behavior (Bućan and Abel 2002). In contrast, behavioral endocrinologists often explicitly examine effects of experience on subsequent behavior through effects on the neuroendocrine system (e.g., Hews and Moore 1997). A few studies have looked at the genetic basis of differential response to experiences; e.g., fish from high predation localities respond more to the effects of experience with a predator than fish from low predation localities (Magurran 1990). The most powerful approach should be to integrate all of the above approaches.

An example of the integration of genetics, experience, and neuroendocrine bases of behavioral syndromes comes from the long-term studies on temperament in rhesus monkeys (*Macaca mulatta*) conducted by Suomi and colleagues. Rhesus monkeys show considerable variation in "reactivity," or fearfulness that is expressed in how individuals respond to novelty, environmental changes, and social challenges. Temperament influences social interactions (both agonism and affiliation) and exploratory behavior that mediates interactions with the nonsocial environment. Detailed longitudinal studies on captive monkeys showed that the expression of temperament depends on the interaction between genetics, experience, and the particular situation. Individual variation in temperament emerged most clearly under mildly stressful conditions, such as when infants were separated from their mothers or during physical or social challenges (Suomi 1987). Individual responses to mildly stressful situations were heritable and stable throughout development (Suomi et al. 1996). Temperament was also affected strongly by a mother's behavior toward her infant. Cross-fostering experiments showed that an individual's reactivity depends on the reactivity of its biological mother, the reactivity of its foster mother, and the conditions under which the individual's reactivity was measured (Suomi 1987). Reactivity of neonates was most strongly related to the biological mother's reactivity. In contrast, under stable social conditions, a juvenile's reactivity was best explained by its foster mother's reactivity. When a juvenile was exposed to an environmental challenge, however, the importance of its pedigree resurfaced. That is, the phenotypic expression of genetic differences was seen under stressful conditions, but not stable ones.

Individual variation in fearfulness in rhesus monkeys is associated with genetically-based differences in the autonomic nervous response and in serotonergic activity. For example, variation at the serotonin transporter gene regulatory locus (5-HTTLPR) is associated with variations in both serotonergic function and behavioral anxiety. The effects of genotype on serotonin function (Bennett et al. 2002) and behavior (Champoux et al. 2002) depend, however, on the environment and on individual experiences. For monkeys reared by their mother, there were no phenotypic differences between individuals that were homozygous versus heterozygous at the 5-HTTLPR locus. When monkeys were reared in a nursery, however, which was presumably more stressful for the monkeys, heterozygous and homozygous individuals differed in behavior and physiology (Champoux et al. 2002). Thus, understanding the physiological bases underlying variation in temperament helped to explain some of the complex patterns in the environment-dependent expression of genetic differences.

Human Personalities

The authors do not pretend to have detailed expertise on this huge literature.

Based on recent reviews representing a range of views (Mischel and Shoda 1998; Lubinski 2000; Tett and Guterman 2000; Bouchard and Loehlin 2001; Funder 2001), we offer a selection of insights for behavioral ecologists. First, the literature on human personality has a long history of using sophisticated, multivariate statistical methods (e.g., variations on factor analysis) to identify multiple personality axes. In contrast, relatively few studies of nonmodel animals have attempted to distinguish two or more distinct behavioral axes (e.g., Reichert and Maynard Smith 1989; Budaev 1998; Gosling 2001). In humans, some authors suggest (in some cases, begrudgingly) that the Big Five has emerged as the dominant paradigm. The Big Five characterizes human personalities in terms of scores on five axes: neuroticism, extroversion, agreeableness, openness, and conscientiousness. Comparative psychologists have attempted to identify and quantify analogs of the Big Five in animals (Gosling 2001). Regardless of whether we agree with the validity of the Big Five, behavioral ecologists studying behavioral syndromes could clearly benefit from borrowing statistical techniques and cautionary comments on their limitations from human personality studies.

Other insights come from attempts to integrate personality-based and situation-dependent views of human personality. The situation-dependent ("situationist") view posits that human behavior is so flexible that the entire concept of human personalities is misleading (Revelle 1995; Mischel and Shoda 1998). An integrative view suggests that whether a personality tendency is expressed might depend on the situation. For example, it has been suggested that variations in human personality might be most apparent during transitional events and around periods of stress (Caspi and Bern 1999). Alternatively, variations in aggressive tendencies might emerge most clearly in situations where low-moderate aggressiveness is normal (e.g., in the office, on the highway, playing sports, or at home), but might be masked in situations where either no one is aggressive (e.g., at church) or where everyone is aggressive (e.g., in hand-to-hand combat: Tett and Guterman 2000). The insight for behavioral

ecologists is that expression of behavioral syndromes might depend on the situations studied, and that our goal should be to understand which situations allow behavioral syndromes to emerge.

Another insight from integration emphasizes that people with different personalities tend to choose different situations (e.g., risk seeking or not: Buss 1987). In that case, even if people are highly flexible in their behavior in different situations, a significant amount of variation in overall behavior might still be explained by personality (i.e., by their choice of situations). The analogy for behavioral ecology is that the expression of behavioral syndromes could come through habitat choice for different situations, above and beyond correlated behavior across those situations. More aggressive individuals might not just be aggressive in contests against conspecifics (or fight rather than flee from predators), they might also spend more time seeking contests (or in risky habitats) than less aggressive individuals.

Finally, studies on humans offer the general insight that integrated interdisciplinary studies on behavioral syndromes should be valuable. With regard to proximate control of personality, personality scores (e.g., the Big Five) are heritable (e.g., Loehlin et al. 1998; Bouchard and Loehlin 2001; Funder 2001) and have some basis in brain structures and hormone levels (Funder 2001). Personality also depends on experiences and vice versa; i.e., there is an ongoing interaction over a lifetime between personalities and experiences (Scarr 1992; Lubinski 2000). With regard to effects of personality on individual performance, research in applied psychology examines relationships between personality and life outcomes (e.g., job performance, marital stability, tendency to not get depressed or involved with substance abuse, happiness). Interestingly, large-scale studies and meta-analyses suggest that out of the Big Five, the best predictor of overall positive life outcomes is conscientiousness (Barrick and Mount 1991; Tett et al. 1991; Soldz and Vaillant 1999). Overall, an integrative view that emerges is that a combination of genes and experience, mediated through effects on the brain and endocrine system, shape personal-

ities that along with specific contexts determine behaviors that then influence life outcomes. Development and application of a similarly integrative view, but with an explicit evolutionary overview (e.g., Buss 1991), should be a goal for the study of animal behavioral syndromes.

Ecological Implications

Finally, we return to the issue of the potential ecological consequences of behavioral syndromes. A simple generality is that since behavior often influences ecological patterns, behavioral syndromes should also often have important ecological implications. Two keys to how behavioral syndromes might affect ecological patterns are: 1) behavioral syndromes are a type of tradeoff that involves limits to plasticity and often suboptimal behavior (and these then influence ecological patterns); and 2) behavioral syndromes—e.g., correlations between reproductive, predator-prey, and dispersal behaviors—connect behaviors underlying three key components of species performance (births, deaths, and dispersal) that might otherwise be uncoupled. We focus on how these aspects of behavioral syndromes might affect some selected ecological issues.

Tradeoffs and limited plasticity often play a major role in population and community ecology. For population ecology, one can define the goal of the field as understanding factors that limit the distribution and abundance of organisms. A long-standing approach in ecology involves doing experiments to identify limiting factors (e.g., predation, competition, abiotic stress, lack of resources). A "limiting traits" approach (cf. Sih and Gleeson 1995) suggests that after identifying limiting factors, a useful focus should be to identify and understand limiting traits—e.g., inappropriate behaviors (or other traits) that explain the poor ability of a species to cope with limiting factors. If, for example, a species's distribution or abundance is limited by predation, what does the focal species do poorly to explain why it does not cope well with predators? This is a twist on the usual emphasis on adaptation and how adaptive traits shape species interactions. The

focus here is instead on key suboptimal traits. Limited plasticity associated with behavioral syndromes could clearly play a key role in this approach. An example, outlined earlier, involves how salamander activity syndromes help to explain why predatory sunfish limit the distribution and abundance of salamander larvae (Sih et al. 2003).

For community ecology, tradeoffs (e.g., between abilities to collect different resources, or between foraging versus antipredator or competition versus dispersal abilities) are a key to understanding patterns of species diversity and variation in community composition in space and time (MacArthur 1972; Connell 1975; Lubchenco 1978; Tilman 1988). That is, the fact that some species are better at some tasks (or in some habitats) while other species are more adept at alternative tasks is critical for explaining how species coexist, and which species dominate in any given situation. Again, it is plausible that limited plasticity associated with behavioral syndromes could underlie some of these tradeoffs; i.e., differences among species in general activity or aggression levels and their limited abilities to alter these levels could generate the tradeoffs that explain some patterns of community structure. A classical approach for connecting species' traits (as limited by tradeoffs) to patterns of coexistence focuses on niche partitioning (Pianka 1981; Abrams 1983). While a species's niche can be defined broadly to include how the species copes with all environmental dimensions, most analyses of niche partitioning have emphasized one or two dimensions (most commonly the feeding niche). The behavioral syndrome view suggests reexpanding the niche concept to include suites of correlated behavioral responses to multiple ecological factors. Coexistence might be better explained by species differences in their overall behavioral type than by resource partitioning per se.

Limited plasticity is presumably most critical immediately after a major environmental change. For many species, environmental change due to human disturbance—climate change, habitat loss and fragmentation, urbanization, spread of exotic species, chemical pollution—will probably be the single most important factor governing their future

persistence. A key issue is how well different species respond to human-induced change. Some species are thriving with humans (e.g., urbanized, invasive species), while others are being driven extinct. Sometimes both extremes of the ability to cope with human-induced change can be found within the same genus. We suggest that species differences in several aspects of behavioral syndromes could play important roles in determining their relative response to environmental change: 1) the species's average behavioral type; 2) time lags in individual behavioral response to new environments; 3) the degree of plasticity shown by individuals; and 4) between-individual variation in behavioral types within the species. Clearly, a species should be able to cope more effectively with a new environment if most individuals have a suitable behavioral type, or if individuals show rapid adaptive shifts in behavior. For example, for some taxa, more flexible, less neophobic species appear to respond more favorably to novel environments (Sol and Lefebvre 2000; Sol et al. 2002). However, even if most individuals in a species are not of an appropriate behavioral type for coping with an environmental change, the species can persist if it has large between-individual variation so that some individuals respond appropriately (Bolnick et al. 2003).

Regardless of whether environmental change is due to temporal variation in a particular site or dispersal to a new site, environmental change might often represent a strong, nonrandom bottleneck with respect to behavioral types. First, dispersal probabilities appear to often be associated with a dispersal syndrome or an aggression syndrome; e.g., either more aggressive individuals are more bold and disperse more readily, or more aggressive individuals drive out less aggressive ones. Second, the probability of surviving the environmental change might often be related to behavioral type. In any case, regardless of which behavioral aspect was important in determining success in getting through the bottleneck, the key is that if this behavior is part of a behavioral syndrome, it brings along with it an entire suite of correlated behaviors. If bold individuals tend to disperse, their boldness could spill over to influence their ecological interactions and impacts in the new habitat. Bottleneck selection on a behavioral syndrome could thus have critical, underappreciated impacts on various ecological phenomena in temporally or spatially variable environments.

For example, one major type of environmental change involves the spread of exotic species. Many studies have attempted to identify traits associated with invasive species (e.g., Lodge 1993; Mack et al. 2000). Although reviews have listed some behaviors, few studies have explicitly explored the behavioral mechanisms underlying species invasions (Holway and Suarez 1999). One notable exception suggested that invasive bird species tend to be less neophobic (more bold) and better at innovative problem solving than similar noninvasive species (Sol et al. 2002). Kolar and Lodge (2001) emphasized that to be an invasive pest, a species must be highly successful in each of three phases of the invasion process: it must disperse readily; grow well from an initially small propagule; and when it gets more abundant, have major impacts on the invaded community. Not all species have the ability to do well in all three phases; however, success in the three phases might be correlated if they represent parts of an overall bold/aggressive/active behavioral syndrome. Bold individuals disperse, aggressive individuals compete well even when rare, and aggressive/active individuals have major impacts on their community. The dispersal process per se might select for bold/aggressive/active individuals (i.e., only they disperse), who then have a particularly strong tendency to disrupt their invaded communities.

Behavioral syndromes associated with dispersal (Clobert et al. 2001; Dingle 2001) might also relate to another major type of environmental change: habitat loss and fragmentation. The ability of a species to persist in a fragmented habitat depends on both survival and reproduction within the remaining habitat, and movement between habitat fragments. It seems likely that fitness within habitat fragments depends on not just one type of behavior, but on an entire suite of behaviors (e.g., foraging, aggression, mating, parental care). Furthermore, both the ten-
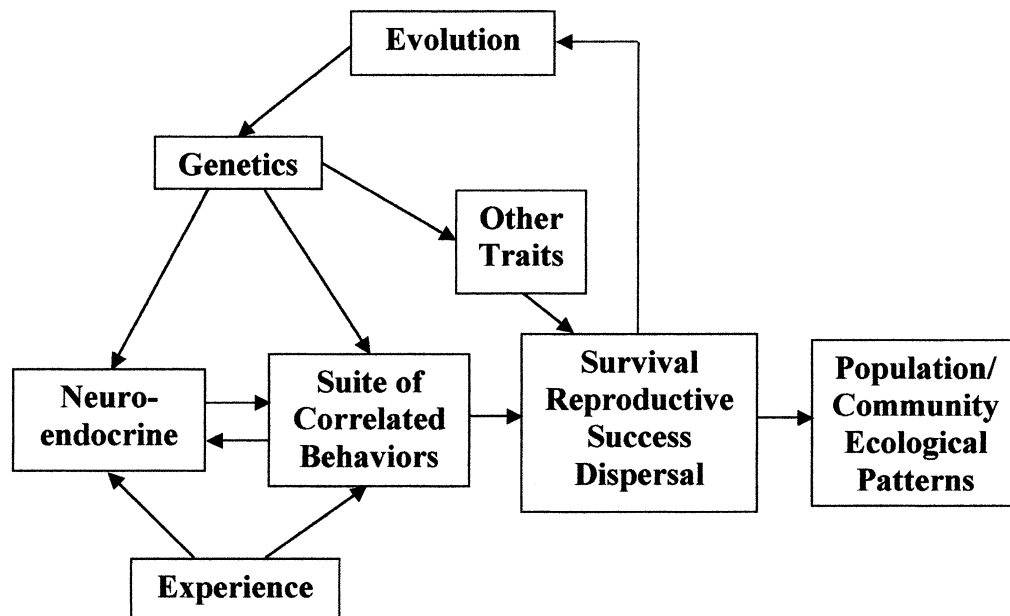
FIGURE 5. AN INTEGRATIVE OVERVIEW ON BEHAVIORAL SYNDROMES
Our integrative overview on behavioral syndromes.

dency and ability to disperse should depend on behavioral type (e.g., boldness: Fraser et al. 2001; aggressiveness: Chitty 1960). Correlations among these behaviors, if they exist, could play an important role in explaining the relative ability of different species to cope with habitat loss, or more generally, to persist in metapopulation or source/sink population structures. Finally, cycles in the prevalence of different behavioral types within populations driven by their relative ability to do well in different social conditions, and by their dispersal tendencies, could be associated with cycles in overall population abundance (Chitty 1960).

Concluding Remarks

Behavioral syndromes could play a useful role as a central core in interdisciplinary studies that integrate genetics, neuroendocrine, and developmental bases of behavior, and ecological consequences of behavior, all with an evolutionary overview (Figure 5). With regard to ecology, we suggest that while significant progress has been made in relating specific behaviors to specific aspects of ecology (e.g., behavior and predator-prey inter-

actions), a focus on behavioral syndromes yields the potential for connecting correlated suites of behaviors to not just one major ecological factor but to a broad set of factors relating potentially to birth, death, and dispersal rates—the three major factors that govern population dynamics. With regard to proximate mechanisms underlying behavior, it has long been the case that one of the most powerful and attractive reasons for studying the mechanistic bases of behavior (e.g., hormonal or experience effects) is the fact that these mechanisms have the potential to influence not just one behavior but suites of behaviors. Our suggestion is that it should be useful to explicitly connect our knowledge on mechanisms that govern suites of behaviors to the effects that these suites have on individual fitness and population/community dynamics. Finally, while there has been a long tradition of studying the genetics and evolution of correlated characters for other types of traits (morphology, life histories), this tradition has not been widely applied to suites of behavioral tendencies. In our view, it is unfortunate that while many have espoused the value of integrative research, the trend in fact is more

toward a drifting apart of the cultures of the mechanistic study of genetics and hormones, on the one hand, and larger-scale ecology on the other, with a reduced appreciation for the role of behavior in both. We suggest that a focus on behavioral syndromes offers the potential to refocus integration and generate new and exciting insights across these levels of study.

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