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Invited Review

Epigenetics for behavioral ecologists

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Environmentally dependent behavioral variation may play a critical role in several ecological and evolutionary phenomena, in particular, rapid adaptation to novel and changing environments. Although it is clear that the expression and inheritance of environmentally dependent animal behaviors can be mediated by epigenetic mechanisms—factors that influence gene expression without modifying the DNA sequence, per se—our understanding of epigenetic processes underlying behavioral variation in natural populations is limited. This is, in part, due to the difficult nature of characterizing epigenetic mechanisms and processes in genetically heterogeneous populations that experience variable environments. In this review, we first highlight the advances that have been made toward understanding molecular epigenetic mechanisms underlying behavioral variation, and their potential role in ecological and evolutionary processes. We then propose approaches and systems that will be amenable to the study of behavioral epigenetics in natural populations. Although well-executed studies in this emerging field will be challenging and few, they have the potential to shed new light on several outstanding ecological and evolutionary questions. *Key words*: DNA methylation, environmental variation, inheritance, maternal effects, rapid evolution, transgenerational. *[Behav Ecol]*

INTRODUCTION: EPIGENETICS FOR BEHAVIORAL ECOLOGISTS

Both genotype and environment contribute to variation in behavior, physiology, morphology, growth, life history, and demography (Pigliucci 2001). Moreover, much, if not most, variation in phenotype arises when the 2 factors interact (Sih et al. 2004; Miner et al. 2005; Owens 2006, see Figure 1). Several recent studies have argued that some behavioral variation, and phenotypic plasticity in general, is mediated by *epigenetic* mechanisms, molecular-level processes (e.g., DNA methylation, histone modification, RNAi) that modify gene expression but do not change DNA sequence and may lead to heritable change in phenotype (Nicotra et al. 2010; Richards et al. 2010; Martin et al. 2011; Richards 2011). Although elegant laboratory studies have shed light on epigenetic mechanisms that contribute to behavioral variation (Weaver et al. 2004, 2005; Anway et al. 2005; Crews et al. 2007; Skinner et al. 2008), the implications of such mechanisms for ecological and evolutionary processes are largely unstudied. For example, the glucocorticoid receptor (GR) promoter in rat hippocampi is differentially methylated depending on how mothers care for their young (Weaver et al. 2004, 2005). Cross-fostering has demonstrated that variation in maternal care impacts rat pup stress reactivity and coping behavior; isogenic rats that received little care as pups are anxious and neophobic as adults, whereas those that received ample care are bolder and neophilic. More importantly from an epigenetic perspective, these behaviors are correlated with the methylation status of the GR promoter; greater methylation of the GR promoter results in less GR expression and a greater disposition for anxiety (Meaney 2001; Weaver et al. 2004). Unfortunately, the dynamics of these epigenetic effects in natural field settings have yet to be investigated.

Personalities (stable differences in behavior between individuals; Dingemanse and Rèale 2005; Duckworth and Badyaev 2007) similar to rat coping styles are common in wild vertebrates, and in many situations have ecological and evolutionary relevance. Variation in personalities can be maintained within or among populations when different personalities confer selective advantages to individuals experiencing different environmental conditions (Sih et al. 2004). For instance, high conspecific densities (or low per-capita resource levels) should favor more aggressive personalities, whereas low conspecific densities should favor less-aggressive personalities (Zielinski et al. 1991). Thus, environmentally induced variation in behavior, which may be epigenetically based, might be adaptive when densities rapidly fluctuate over time or across different environments. Further, such variation might allow populations to persist under *novel* environmental conditions including habitat modification or climate change (Rapp and Wendel 2005; Nicotra et al. 2010). These initially induced epigenetic modifications may be heritable because of genomic reprogramming of the germline caused by the "shock" of novel conditions (*sensu* McClintock 1984; see also Rapp and Wendel 2005). Even somatic epigenetic modifications might become heritable if they promote their own re-establishment in each generation (e.g., through parental effects Day and Bonduriansky 2011). Additionally, environmentally dependent epigenetic effects underlying a certain behavior may facilitate the fixation of genetic variants such that the behavior becomes constitutively expressed when it is adaptive (e.g., a more aggressive phenotype in a competitive, static environment), or more sensitive to environmental change when it is conditionally adaptive (a process referred to as "genetic accommodation"; *sensu* West-Eberhard 2003). There is currently little empirical evidence to determine the prevalence or relevance of such epigenetically facilitated behavioral change.

In this review, we have 2 goals: (1) to build on recent progress in ecological epigenetics (Bossdorf et al. 2008) by

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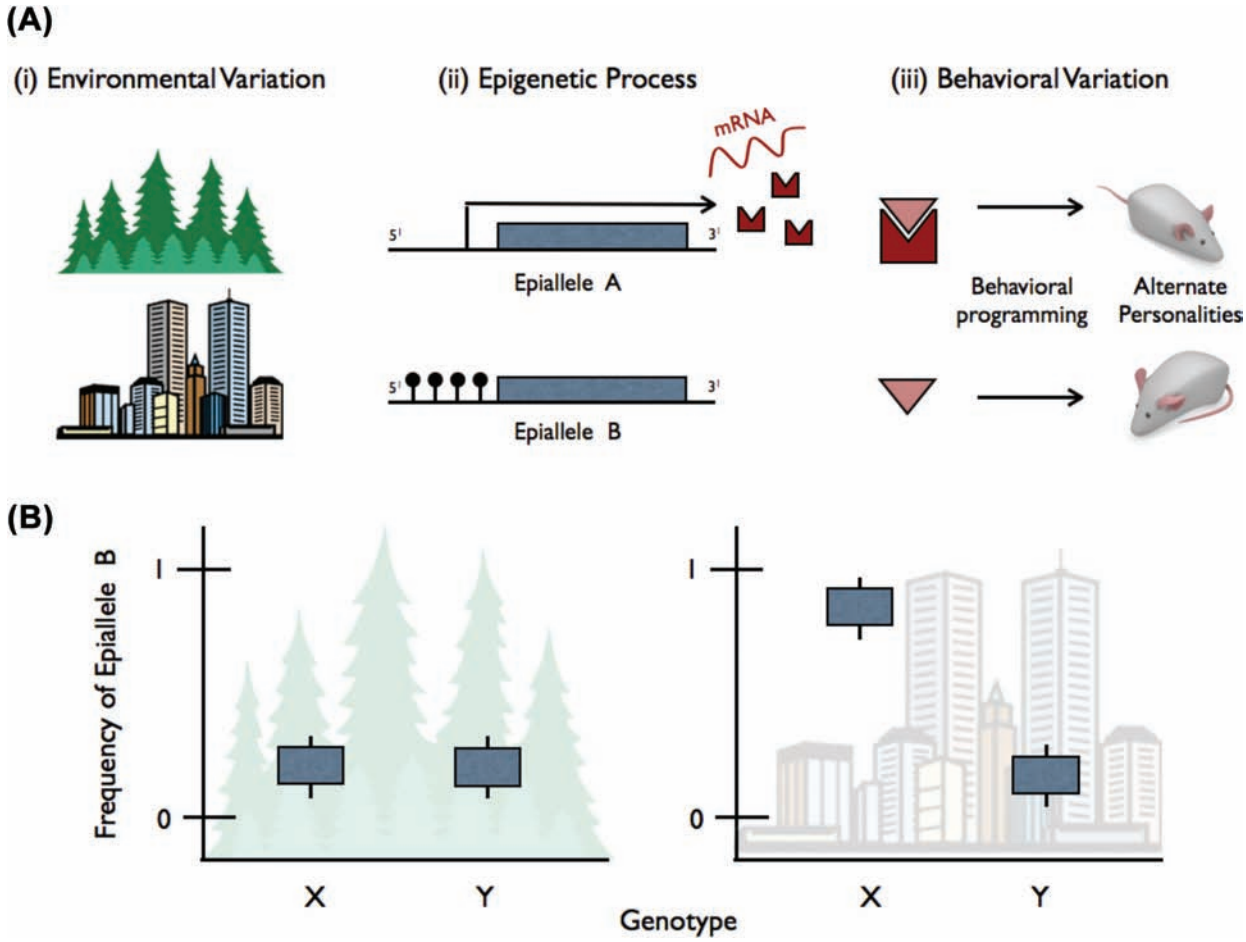

Figure 1
Behavioral variation is often generated from gene by environment interactions, some of which are influenced by epigenetic mechanisms, molecular factors that modify gene expression but do not change the DNA sequence, *per se*. For instance, young rats (panel A) might be sensitive to environmental variation (i) that induces alternate methylation patterns in gene regulatory regions; (ii) DNA methylation generally reduces the accessibility of DNA to transcription factors and thus prevents the production of gene products. In this example, the gene product is a hormone receptor that has an organizational role in adult behavior; altering hippocampal expression of this hormone receptor during a certain developmental window can influence whether an individual is more or less responsive to stressors later in life (*sensu* Weaver et al. 2004) (iii). Thus, "epialleles" are alternate, stable epigenetic states of the same gene that can generate behavioral variation, and can differ between individuals exposed to different environments. Further, the inducibility of epigenetic variation in behavior might depend on the genetic background of an individual (e.g., in panel B, environmentally induced epigenetic variation occurs in genotype X but not Y; rat images provided by the Genetic Science Learning Center [http://learn.genetics.utah.edu/content/epigenetics/rats/]).

developing ideas for behavioral ecological epigenetics, and (2) to devise a framework for determining whether molecular epigenetic mechanisms contribute to ecologically and evolutionarily relevant behavioral variation. Several recent plant studies have demonstrated compelling relationships between molecular epigenetic variation and ecological processes, (Lira-Medeiros et al. 2010; Herrera and Bazaga 2011; Scoville et al. 2011; Richards et al. 2012), yet animal behavior studies have lagged. Below, we review relevant work in the emerging field of ecological epigenetics, as well as the advances made in behavioral epigenetics of model organisms. Then, we highlight approaches and study systems that will be particularly insightful for behavioral ecologists. Finally, we close by discussing future empirical and conceptual challenges for the nascent field of behavioral ecological epigenetics. We expect that meeting these challenges will shed light on questions such as, What are the relative contributions of epigenetic and genetic variation to behavioral evolution during rapid adaptation to novel environments? If epigenetic changes underlie environmentally induced behaviors, can they be stabilized by subsequent genetic changes? Do populations that experience fluctuating environments retain flexibility via epigenetic variation that functions independently of genetic variation (i.e., the so-called "pure" epigenetic variation; Richards 2006), or does selection on genetic variation render these behaviors more sensitive to environmental change?

THE EMERGING FIELD OF ECOLOGICAL EPIGENETICS

DNA methylation is the most studied molecular epigenetic effect (Richards and Elgin 2002; Lippman et al. 2004; Feng et al. 2010), and several ecological studies have documented genome-wide changes in methylation patterns using a methylation-sensitive variant of the amplified fragment length polymorphism—MS-AFLP—protocol. For instance, studies have demonstrated correlations between genome-wide methylation patterns and different habitats (*Viola*, Herrera and Bazaga 2010, 2011; *Laguncularia*, Lira-Medeiros et al. 2010;

Dactylorhiza, Paun et al. 2010; *Metschnikowia*, Herrera et al. 2012; Richards et al. 2012). In another study, plants exposed to simulated herbivore or pathogen damage had up to 30% change in genome-wide methylation polymorphisms compared with genetically identical controls, and some of the environmentally induced epigenetic modifications persisted across generations (Verhoeven et al. 2010).

In addition to genome-wide studies of methylation changes, the study of "epialleles"—alternate, stable epigenetic states of the same gene—could provide ecologists with a tool for characterizing epigenetic processes within and across rapidly evolving populations (Kalisz and Purugganan 2004). Epialleles alter gene expression (Figure 1) and may vary in state between individuals with different environmental histories. In theory, epialleles could be used as proxies for behavioral phenotypes generated by environmental variation. If heritable epigenetic variation plays a role in adaptation, then local differences in habitat characteristics may select for different epialleles in different populations. As with genetic polymorphisms, this selection will result in population-level associations between epialleles, behaviors, and ecological habitat characteristics.

Some basic principles of ecological epigenetics garnered from plant and yeast studies suggest that the application of epigenetic markers at a genome-wide or gene-specific level may be a powerful tool for characterizing the evolutionary potential of environmentally generated behavioral variation. First, phenotypic variation may be the result of environmentally induced epigenetic variation within generations (Herrera et al. 2012). Second, phenotypic variation might also reflect environmentally induced epigenetic variation inherited between generations (Scoville et al. 2011). Third, the ability of an organism to produce epiallelic variation may depend on the genotype of the individual (Figure 1; Scoville et al. 2011). Still, the use of plant studies as a guide for behavioral ecological epigenetics should be done with caution. There are important distinctions between molecular epigenetic processes in plants and animals that might influence what techniques are used to study them, as well as their ecological or evolutionary implications. First, germ-line segregation occurs relatively early during development for many animals. Subsequently, there are comparatively fewer opportunities for environmental modification to be passed through the germline (Jablonka and Raz 2009; Jablonka and Lamb 2010; also see Anway et al. 2005; Franklin et al. 2010). Second, animal behavior partially determines what environmental cues organisms experience as well as the environments to which their offspring are exposed. In some cases, adult behaviors create an environment for the offspring that mimics environmental cues experienced by the parents, thereby inducing similar phenotypes and, potentially, epigenetic marks in their offspring without necessarily changing the germline. These parental effects form the basis for an alternate system of inheritance that is perhaps less important for plants (but see Donohue 2005).

ADVANCES IN BEHAVIORAL EPIGENETICS: WHAT DO WE KNOW?

A critical goal for behavioral epigenetics, and ecological epigenetics in general, is to identify variation that affects phenotype and is heritable, because natural selection will ultimately act on heritable variation that alters the timing, degree, or sensitivity of a behavioral response among individuals. Some epigenetic variants are more likely to fit these criteria due to their persistence over the lifetime of the individual and their tendency, once induced, to be transmitted between generations, regardless of subsequent environmental change (Tal et al. 2010). Identifying these environmentally induced heritable epigenetic variants is challenging because epigenetic variation may also include marks that are limited to context-specific induction within generations that are reset with no memory of past environments (Sung and Amasino 2004; Tal et al. 2010). Below, we illustrate different types of behavioral variation and discuss which are most likely to be underlain by environmentally inducible and heritable epigenetic variation.

Behavioral variation

Reversibility of behavioral responses to external and internal cues is an intrinsic characteristic of behavior (Duckworth 2009). The level of behavioral expression might also vary with the environmental context or the state of an individual (i.e., behavioral plasticity; Duckworth 2009). For instance, in some birds, the expression of male aggression is dependent on the season (breeding) and the state of the individual (reproductive; Rohwer and Wingfield 1981). Finally, behavioral responses can be modified or refined by environmental experience (i.e., through learning), resulting in behavioral flexibility.

Fleeting epigenetic marks that mediate transitions between behavioral states may not be evolutionarily relevant if they are equivalent across individuals. In contrast, epigenetic factors that influence the timing, degree, or sensitivity of a behavioral response and that vary among individuals *can* be selected. For instance, selection might act on epigenetic variation among individuals that makes them more or less aggressive, renders their responses more or less sensitive to seasonal and physiological conditions, or expands a behavioral repertoire. Such epigenetic modifications of behavior might reflect underlying genetic variation, environmental variation, or a combination of both. Moreover, there is compelling evidence that environmentally induced epigenetic marks that modify behaviors can be transmitted between generations. For instance, female mice that experienced environmental enrichment learned to negotiate a water maze faster than control mice, and the offspring of these mice also learned faster even though they had never experienced environmental enrichment themselves (Arai and Feig 2011). Because epigenetic modifications can be involved in memory consolidation within generations (Day and Sweatt 2011), it is plausible that a propensity for learning can be transmitted between generations via epigenetic mechanisms.

There are at least 2 ways in which induced epigenetic behavioral variation can be maintained across generations. First, environmentally induced epigenetic marks can be inherited directly through the germline. Second, such marks can be inherited indirectly through the soma of the offspring that are developing *in utero*, or when environmentally induced adult behaviors reproduce similar environmental cues for the offspring, thereby propagating the inducing stimuli. Below, we discuss recent advances made toward understanding these processes in the laboratory (these studies and more are summarized in Appendix A).

Modes of inheritance

Germline inheritance

One of the first examples of inherited epigenetic behavioral variation involved the effects of the fungicide, vinclozolin, on rats (Anway et al. 2005). Offspring of pregnant rats exposed to this endocrine disruptor (i.e., an environmental chemical that disrupts hormone function) exhibited marked differences in anxiety-like behavior and learning capabilities (André and Markowski 2006; Skinner et al. 2008). Importantly, anxiety-like behavior persisted through the F3

offspring, even in the absence of vinclozolin. The effects of maternal vinclozolin exposure can potentially influence the development and resulting phenotypes of F1 and F2 generations as their somatic and germ-line methylation patterns are established *in utero* (Faulk and Dolinoy 2011). However, in this study, somatic patterns of methylation in the F3 offspring from the vinclozolin-exposed lineage would have been free from direct exposure, suggesting that vinclozolin reprograms the germline. Further, DNA from the sperm of the offspring from the vinclozolin-exposed lineage possessed altered methylation in several genes compared with controls (Anway et al. 2005; Stouder and Paoloni-Giacobino 2010), suggesting a mechanism for the transmission of the behavioral phenotype.

In another study, Franklin et al. (2010) subjected rats to chronic and unpredictable maternal separation. As adults, these pups developed depression-like syndromes and possessed modified epigenetic patterns in their sperm, specifically in the regulatory regions of candidate genes for depressive behaviors. Not only did maternal separation alter the behavior and epigenetic state of genes in the germline of the separated males, similar patterns of behavior and epigenetic marks were found in the offspring of these males. Because the separated males in this study were mated to control females, it is unlikely that the offspring phenotypes were influenced by maternal behavior or physiology; rather, these epigenetic modifications were likely transmitted through the male germline. To further corroborate this finding, strikingly similar patterns of epigenetic modifications were found in the brains of female F2 progeny from separated males. Aside from the behavioral effects induced by vinclozolin and maternal separation, however, there are relatively few empirical examples of direct, environmental modification of the germline in animals (Appendix A; Crews 2010).

Soma-to-soma inheritance

Most examples of epigenetic inheritance of behavioral traits occur via "soma-to-soma" transmission (i.e., developmental interactions between offspring or between parents and offspring that do not pass through the germline; Jablonka and Lamb 2010). Although induced epigenetic modifications to the germline are maintained by an organism's own mitotic and epigenetic machinery, and are therefore relatively resistant to further environmental changes (Crews 2010), epigenetic changes to somatic tissues that are passively inherited (e.g., certain diet-derived substances) or maintained by higher-level processes (e.g., parental care) are probably more susceptible to erosion in the absence of the inducing environment. Nevertheless, soma-to-soma transmission may play a dominant role in behavioral ecological epigenetics: although the germline has a relatively short sensitive window for epigenetic modification, somatic epigenetic modification can occur at any time, from embryogenesis through adulthood (Skinner and Guerrero-Bosagna 2009).

In litter-bearing animals, for example, prenatal hormonal conditions have distinct effects on adult behavior contingent on position *in utero*. Siblings develop next to each other, and each produces sex steroids that can diffuse through amniotic fluid and affect littermates on either side. In several eutherians, females flanked by 2 males (2M females) have higher levels of circulating testosterone, are more aggressive, and are more behaviorally sensitive to testosterone treatments in adulthood than females of the same litter who were flanked by 2 females (0M females, Clark et al. 1993; Ryan and Vandenbergh 2002). Further, high population densities experienced by mothers exacerbate 2M characteristics in their pups (Zielinski et al. 1991). These environmental effects can potentially be perpetuated through several generations because, as adults, 2M females are also more likely to produce more male pups than female pups, increasing the probability that their own female pups will be surrounded by males *in utero* (Ryan and Vandenbergh 2002).

Gonadal hormones such as testosterone are known to influence epigenetic processes such as DNA methylation and histone acetylation in developing brains (Nugent and McCarthy 2011), providing a potential mechanism for the emergence of adult aggression in 2M females. For instance, the promoter region of estrogen receptor α (ERα) is more heavily methylated in neonatal female rats than in male rats, or female rats that have been exposed to masculinizing doses of estradiol (in males, estradiol derived from testosterone is responsible for behavioral masculinization via activation of ERα; Kudwa et al. 2006). Although these patterns disappear during development, they remerge in the same manner with exposure to adult gonadal steroids (i.e., adult female rats have higher levels of methylation relative to adult males and estradiol-treated females). These results suggest a role for hormonally mediated epigenetic determination and maintenance of sexual behaviors (Martin et al., 2011).

Because some induced behaviors have a tendency to be re-established in each generation through parental effects on offspring, the postnatal environment created by parental care has been of particular interest to researchers (Day and Bonduriansky 2011). As mentioned earlier, variation in rat maternal care can influence the parenting behavior of their pups (via methylation of hippocampal glucocorticoid receptor expression; Champagne et al. 2003; Weaver et al. 2004), thus producing a behavioral mechanism of inheritance. A similar phenomenon has been observed in mice: individuals that experienced maternal separation early in life exhibited reduced stress-coping and learning abilities as adults, concurrent with increased expression of the arginine vasopressin (AVP) gene in the neurons of the paraventricular nucleus (Murgatroyd et al. 2009). Increased *Avp* expression was, in turn, facilitated by hypomethylation of the *Avp* promoter. The behavioral effects of early life maternal separation were reversed when mice were treated with an AVP antagonist, suggesting a causal link between the epigenetically maintained expression of this gene and impaired coping abilities in adult mice. Interestingly, AVP may also have a critical role in parental behavior (Bester-Meredith and Marler 2003). California mouse pups (*Peromyscus californicus*) fostered to parents of a species that exhibits less parental care (white-footed mouse, *P. leucopus*) exhibited reduced parental care as adults, and lower levels of AVP in the bed nucleus of the stria terminalis, a brain region associated with parental care (Bester-Meredith and Marler 2003). Thus, epigenetic modification of AVP may provide a mechanism for behavioral inheritance in rodents.

The examples above provide compelling evidence for environmentally induced epigenetic modification of behavior, and the transmission of such modifications across generations. But under what environmental conditions does epigenetic regulation of behavioral variation evolve, and how does such inheritance influence subsequent evolution?

Ecological and evolutionary implications

Given the ample empirical evidence that epigenetic variation underlies variation in many environmentally dependent behaviors in laboratory animals, how is epigenetic variation relevant to ecological and evolutionary phenomena, and how is it different from genetic variation? In some ways, heritable epigenetic modifications should act as genetic mutations. However, in contrast to genetic variants, environmentally induced epigenetic variants can exist at higher initial frequencies than genetic variants as several individuals can acquire inducible epigenetic variants at once (West-Eberhard 2003). Thus, heritable epialleles underlying adaptive traits may arise and go to fixation faster than spontaneous genetic mutations.

Further, epialleles have the opportunity to test more genetic backgrounds, allowing them to explore different epistatic interactions. In short, the ability to occur in several individuals at once suggests that epigenetic behavioral variation can facilitate population persistence when environmental change is too rapid for genetic variation to arise (Price et al. 2003; Bonduriansky and Day 2009).

Aside from their high initial frequencies in populations, epigenetic variants differ from genetic variants with respect to their reversibility or permanence. The permanence of epigenetic variants (and the behavioral phenotypes they produce) may be influenced by the environmental conditions in which they evolve. Heritable, epigenetically based behaviors that produce intermediate-term responses to environmental pressures might be particularly advantageous in fluctuating environments (i.e., temporally patchy environments; Jablonka et al. 1992; Lachmann and Jablonka 1996), specifically when there is a lag time between the induction and the expression of a behavior (Jablonka et al. 1995; Lachmann and Jablonka 1996). For instance, adult female rats might become aggressive only some time after population densities increase, and thus express a mismatched behavior in the interim (Lachmann and Jablonka 1996). The selective stress of a mismatched behavior would be mitigated for offspring if they were programmed for aggression even before experiencing the inducing cue. Thus, epigenetic variation that facilitates such intermediate-term responses might be selected when it allows developing organisms to produce behaviors most appropriate for future environments.

On the other hand, some environmental changes might be so rapid or drastic that inherited behaviors fail to produce adaptive outcomes. When an environmentally induced epigenetic variant (and the behavior it produces) is adaptive under certain conditions, and maladaptive under others, the reversibility of the variant will determine whether it is appropriate or mismatched for current conditions. For example, mouse pups born to mothers experiencing high population densities will be more aggressive as a result of elevated maternal testosterone, which might be advantageous for individuals experiencing high population densities (Zielinski et al. 1991). However, if the population suddenly becomes less dense, the inherited behavior might no longer be advantageous, and may even become a liability. In some situations, aggressive behavior impacts fitness in non-competitive environments through its detrimental effects on parental care (Duckworth 2006; Duckworth and Badyaev 2007).

The risk of producing a behavior that is mismatched to an individual's conditions is particularly relevant to organisms experiencing environments that are unpredictable and fluctuate within generations (i.e., fine-grained environments Jablonka et al. 1995; Piersma and Drent 2003; Young and Badyaev 2007). Under such conditions, selection will favor mechanisms that provide a wide variety of behavioral phenotypes such as bet-hedging (producing mixed, stable phenotypes within broods; Bonduriansky et al. forthcoming), phenotypic memory (a single parental phenotype is inherited by offspring), and learning (Snell-Rood et al. 2010). Induced behavioral phenotypes that are inherited are favored in rapidly fluctuating environments (in addition to temporally patchy environments) because even though some behaviors carried over from previous environments will be maladaptive, at least some individuals will express behaviors fit for the current environment (Cooper and Kaplan 1982; Jablonka et al. 1995). Likewise, learning might provide all the behaviors necessary to endure a changing environment (Wright et al. 2010). Empirical examples suggest that both phenotypic memory (Weaver et al. 2004) and an inherited propensity for learning (Champagne et al. 2008) might be mediated by environmentally induced epigenetic factors, and can thus be targets of selection.

The degree to which epigenetic modifications resist subsequent environmental changes, once induced, can have a profound effect on evolutionary dynamics (Jablonka et al. 1995; Tal et al. 2010; Bonduriansky et al. forthcoming); Day and Bonduriansky 2011; Geoghegan and Spencer 2011). When induced epialleles are transmitted with high fidelity (i.e., they maintain their epigenetic state regardless of subsequent environmental change), they are more likely to accumulate and contribute to stable equilibria (i.e., phenotypic variation) than when they are environmentally labile (Day and Bonduriansky 2011; Geoghegan and Spencer 2011). However, what we know about the relative stability of induced epigenetic variation is restricted to specific variants induced under laboratory conditions. On one hand, germline epigenetic modifications caused by vinclozolin can be stable through 5 generations (Anway et al. 2005) and on the other hand, dietary factors or social enrichment can reverse epigenetic modification caused by maternal care within a generation (Weaver et al. 2005; Champagne and Meaney 2007; Champagne 2008).

One way to address this empirical lacuna is to produce inbred animal lines that vary in epigenetic state via environmental or chemical manipulation, or that vary specifically at genetic loci involved with chromatin control (i.e., epiRILs; *sensu* Johannes et al. 2009). This could shed light on the relative stability of epialleles (Johannes and Colomé-Tatché 2011) and how this stability might depend on the number of generations in which a population experiences the inducing environment (Tal et al. 2010). However, there are at least 2 caveats to such an approach. First, creating inbred lines from wild populations can take several generations, limiting this method to organisms with short generation times (e.g., insects), clonal organisms (e.g., *Daphnia* or *Caenorhabditis*—water fleas and nematodes, respectively), or model organisms that can be generalized to natural systems. Second, natural environmental variation experienced by individuals is far more complex than what can be produced using experimental manipulations. Nevertheless, determining inducibility and stability of epigenetic variation in behavior caused by natural environmental variation will be critical for understanding its role in evolutionary phenomena.

In addition to the transmissibility of induced epigenetic variants, the relationship to fitness will determine whether variants are retained by selection (Pál and Miklós 1999). For instance, the epigenetic modifications in rats caused by maternal exposure to vinclozolin might persist for generations, but might also be lost from populations rapidly because they cause male offspring to seem unattractive to potential mates (Crews et al. 2007). Conversely, behavioral variants that are favorable can allow for the evolution of genetic modifiers that enhance their expression (genetic accommodation; West-Eberhard 2003), which in turn promotes population persistence or divergence under changing or novel environmental conditions (Pál and Miklós 1999). Selection on genetic variation underlying the regulation of the induced behavior itself might result in the behavior becoming fixed (Pál 1998) or more sensitive to environmental cues (West-Eberhard 2003). Selection on epigenetic variation in behavior can also influence the epigenetic and genetic evolution of *correlated* traits, for at least 2 reasons. First, behavioral responses expose associated traits to novel or relaxed selection regimes (Price et al. 2003; Bonduriansky and Day 2009; Lahti et al. 2009). Second, when behavior facilitates population persistence under novel environmental conditions, the new environment per se may alter the expression of associated traits (Via and Lande 1985; Falconer and Mackay 1996; Badyaev et al. 2002), thus

generating or limiting opportunities for selection to occur. In short, environmentally dependent behavioral responses mold both selective pressures and the phenotypic variation on which those pressures act. Unfortunately, we still know little about the mechanistic basis of behavioral responses to novel or changing environments, and can only assume that their expression involves an epigenetic component.

As future studies begin to reveal the various mechanisms that mediate epigenetic inheritance, we will better understand and predict their evolutionary dynamics. Diverse mechanisms likely have different properties of induction and inheritance. The effects of parental care on offspring behavior might be more sensitive to subsequent environmental variation (Champagne and Meaney 2007) than RNA-based inheritance (Rassoulzadegan and Cuzin 2010) and certain germ-line modifications (Anway et al. 2005). Epigenetic mechanisms might also differ in how they covary with genetic variation (Day and Bonduriansky 2011). Nevertheless, nongenetic mechanisms of inheritance are compatible with evolutionary models and can, in many cases, be treated equivalently insofar as we empirically determine their influence on fitness, stability within generations, and stability between generations (Day and Bonduriansky 2011).

APPROACHES

When it comes to characterizing epigenetic variation in behavior in natural populations, there are at least 2 places to start. One might start with a genome-wide survey of epigenetic markers (e.g., MS–AFLP) and then correlate the presence of these marks with the presence of a particular behavioral variant. Alternatively, one might identify candidate genes that have previously been linked to behavioral variation, and then experimentally assess whether the expression of those genes can be epigenetically modified in a way that reflects ecologically relevant conditions; in essence, a "trial-and-error" approach. Each approach has benefits and shortcomings, but both face a serious hurdle when applied to field-collected specimens. It becomes difficult to identify epigenetic variation that is independent of underlying genetic variation; that is, some epigenetic marks might vary perfectly with genetic variation, regardless of environmental conditions (Richards 2008). In such cases, correlations between epigenetic and environmental variation might exist as a result of selection on underlying genetic variation (e.g., epigenetic "hitchhiking"; Richards 2011). Thus, establishing whether the environment plays a causal role in the expression of epigenetic marks that affect evolutionarily relevant behavioral phenotypes, independently from genetic variation, requires experimental manipulation.

To this end, we suggest a two-pronged approach (Figure 2). The first element involves a comparative assessment of populations encountering different and ecologically relevant environments and their corresponding behavioral phenotypes and epigenetic states. This descriptive approach is necessary to establish the adaptive value of a behavior in different

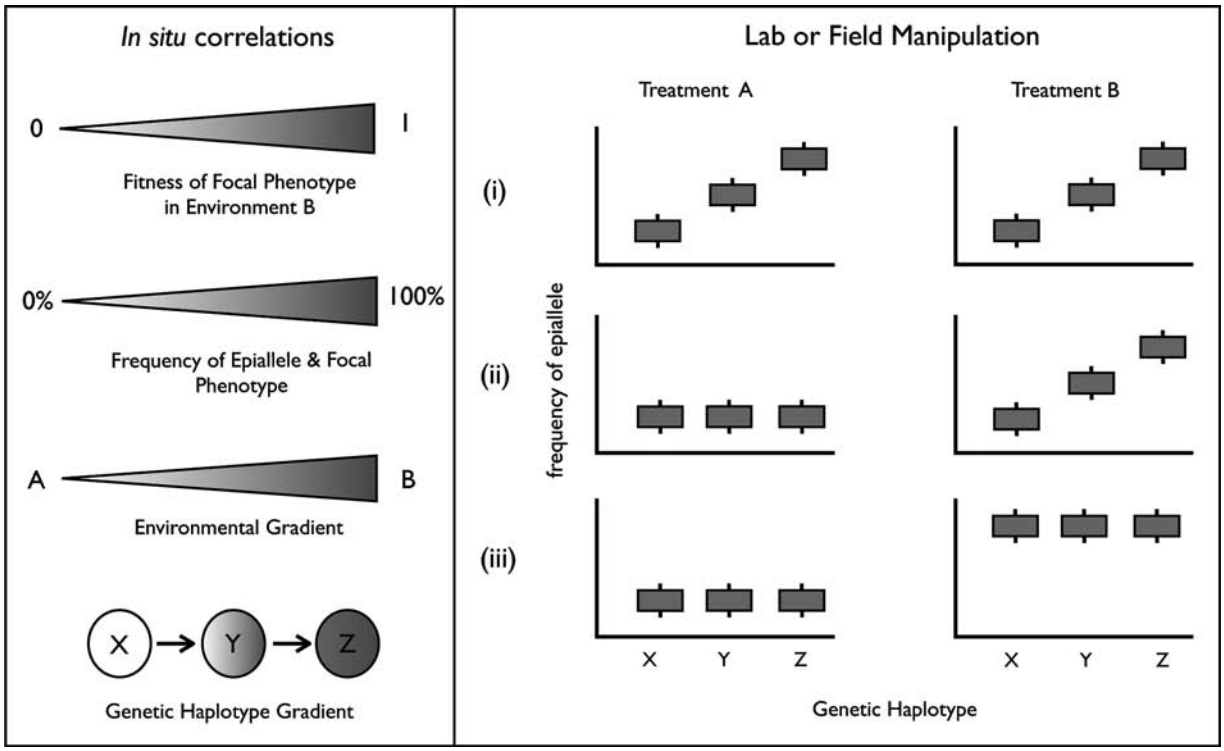

Figure 2
A twofold approach for estimating the relative contributions of genetic and epigenetic variation to behavior in natural populations. A comprehensive research program would establish the environmental determinants of a behavioral phenotype in natural populations (left panel). Thereafter, characterizing the source of epigenetic variation—whether it is environmentally or genetically dependent—requires controlled environmental manipulations during development (right panel). This manipulation can be accomplished either by raising organisms from different populations in the lab, or by manipulating their natal environments in the field. Individuals from the same population would constitute the same "genetic haplotype" (e.g., X, Y, & Z), although spreading genetically similar individuals (e.g., siblings) over different treatments (e.g., Treatments A & B) would provide more control for genetic effects on behavioral phenotypes. Patterns of behavior among populations may be generated by obligate epigenetic variation that is solely dependent on genetic variation (i), facilitated epigenetic variation that depends on both genetic and environmental variation (ii), or pure epigenetic variation that can be environmentally induced in any genotype (iii).

environments. However, by assessing wild-caught animals, the researcher's ability to ascribe the environment as a causal agent of epigenetic states and their corresponding behaviors is compromised. Such individuals have a history of unique experiences, many of which could impact behavioral differences. Addressing causality is thus more easily and accurately approached in a controlled laboratory or psuedo-natural setting, where the environmental history of an organism and its parents are known. Thus, the second element involves establishing the potential for environmentally inducible epigenetic variations to occur via experimental manipulation.

As with all studies of adaptive traits, one could begin by identifying behavioral phenotypes that vary among populations experiencing different environments. Such environmental heterogeneity may arise when novel habitats are colonized (e.g., during a biological invasion) or when environmental conditions change (e.g., global warming). Ideally, one would then demonstrate that behaviors are adaptive for environments in which they are found; for example, by demonstrating associations between those behaviors and fitness. Finally, by regressing the presence of a behavioral phenotype on the presence of a given epiallele or epiallelic combination (*sensu* Herrera and Bazaga 2011; Herrera et al. 2012), one might also establish a relationship between epigenetic variation and the behavioral pattern of interest. However, heterogeneous environments also coincide with genetic differentiation; individuals within environments might be more genetically similar to each other than to those in different environments due to local adaptation or neutral isolation by distance (Nei 1972). This population genetic structure (represented as "genetic haplotypes" in Figure 2) challenges our ability to characterize genetically independent epigenetic variation; spurious correlations might arise between epigenetic variation and environmental conditions because environments and genetic variation are also correlated (although see approaches using outlier analysis in Schrey et al. 2012; Richards et al. 2012). Thus, it is necessary to judge the capacity of distinct genetic populations to induce environmentally dependent epigenetic modifications via experimental manipulation.

Determining whether a behavior is epigenetically influenced can be accomplished using cross-fostering designs or environmental manipulations (Cheverud and Moore 1994). Such manipulations can be performed in the field or laboratory (using animals derived from natural populations). If these manipulations are conducted in the laboratory, great care must be taken to replicate ecologically or evolutionarily relevant stimuli. Alternatively, one could attempt to reproduce epigenetic patterns that are observed in natural populations through the use of chemicals (e.g., trichostatin A, a deacetylase inhibitor) or molecular tools (e.g., RNAi), which might be particularly useful when epigenetic marks underlying behavioral variation are an integrated response to several environmental cues. In either case, such manipulations cannot usually be conducted on genetically identical animals, preventing results from being genetically independent. For natural populations, genetically similar animals may be the closest proxy for genotypic identity that one can obtain (Stamps and Groothuis 2010). If the closely related individuals (e.g., siblings) can be parsed among environmental treatments, these replicate individuals can provide some control of genetic variation, and therefore a more accurate appreciation of environmentally inducible and genetically independent epigenetic variation.

Potential results

The epigenetic and behavioral responses we would expect from performing cross-fostering or environmental manipulations on developing individuals from different populations are presented in Figure 2. Obligate epigenetic variation is strictly determined by genetic variation, regardless of the environment in which individuals are raised (Richards 2006). That is, behavioral phenotypes and obligate epigenetic variation should covary among populations as a result of the genetic differences underlying both. Facilitated epigenetic variation (*sensu* Richards 2006) arises when genotypes influence the probability that an epiallele and associated behavioral phenotype will be expressed in response to an environmental stimulus. Finally, some epialleles are induced by environmental stimuli across all genotypes ("pure" epigenetic variation; i.e., they are not correlated with genetic variation), and we would expect that raising individuals from different populations under common conditions would result in similar epigenetic marks and associated behaviors.

One caveat to this approach is that it might be easy to miss a key epigenetic modification-driving behavior, regardless of the technique being used. On one hand, genome-wide scans for epigenetic variation are especially susceptible to missing a critical locus that is environmentally sensitive and regulated by epigenetic machinery, as markers like AFLP are random fragments that are not easily associated with functional genes. On the other hand, although candidate epigene approaches begin with more informed targets, they generally are restricted to a few putative mechanisms (Weaver et al. 2004). Both approaches are complicated by the fact that epigenetic modifications are tissue specific and sometimes cell specific. Even within tissues and cells, marks can be transient but impactful if their appearance happens in a particular window of time (e.g., spermatogenesis). Further, the destructive sampling required for collecting tissues precludes the measurement of individual-level variation under different environmental conditions. For behavioral ecology, these factors present unique challenges (e.g., Trainor et al. 2008). In spite of these challenges, the results from such experiments can engender exciting new questions with testable hypotheses.

SYSTEMS FOR EPIGENETIC BEHAVIORAL ECOLOGY

To some degree, animal systems used for genetic studies of behavioral ecology (reviewed in Fitzpatrick et al. 2005) will also be appropriate for epigenetic investigation. Many of these systems benefit from the availability of sequenced genomes, making candidate gene approaches more feasible. Conveniently too, several of these systems exhibit some degree of environmental sensitivity in gene–behavior relationships. Although many behaviors are environmentally influenced, there has been little to no investigation of whether molecular epigenetic mechanisms are involved. Put differently, there are ample opportunities for behavioral ecologists to investigate the epigenetic basis of behavioral variation.

A second characteristic of animal systems that should be considered for epigenetic studies pertains to the time of divergence between populations or species exhibiting different behavioral traits. For the behavioral ecologist asking, "What epigenetic mechanisms gave rise to the behavioral phenotype in question", systems undergoing *recent* environmental change would be most appropriate. In particular, the short timescale and geographic scope over which humans facilitated biological invasions should provide a wealth of opportunities; invading individuals often experience very different environments from their host ranges, and the source, age, and direction of such invasions are often known (Moczek 2007; Moczek et al. 2011). In many cases, the experimental

power of such approaches is twofold, because the behavioral responses of invaders to their new communities also influence the behavior of resident organisms (e.g., Phillips and Shine 2006). Here, we describe animal systems that we find particularly promising for behavioral ecological epigenetics, including those that are most experimentally tractable (e.g., with sequenced genomes and genetic tools such as RNAi) and those that have undergone recent ecological and behavioral divergence.

Systems

Honeybees (genus *Apis*) develop as either nonreproductive workers or reproductive queens, and this differentiation is mediated by the consumption of a royal jelly diet (Miklos and Maleszka 2011). Whereas queens are aggressive and spend their entire lives bound to the hive, workers spend most of their lives foraging for resources that will sustain their colony. These divergent behaviors correlate with different patterns of methylation among bee brains, and can be reproduced by experimentally manipulating methyltransferase (the enzyme responsible for methyl group maintenance) with RNA interference technology (RNAi; Kucharski et al. 2008). Honeybees have a fully sequenced genome, making them a useful system for determining the role of epigenetic mechanisms in behavioral plasticity at a gene-specific (Toth et al. 2007; Toth and Robinson 2007) or genome-wide level. However, it is not clear what role environmentally dependent behavior played in the evolution of honeybee sociality.

Other social insects may be more ideal for investigating the epigenetic origins of behavioral variation. Halictine bees (sweat bees) are a primitively eusocial clade in which environmental correlates of social behavior *are* known. Generally, populations of *Halictus rubicundus* vary their social behavior based on environmental variation: solitary behavior is most often associated with harsh conditions, such as high altitude and latitude (Ulrich et al. 2009). For instance, when nests from a harsh site in Ireland were transplanted to a mild site in Britain, nearly half of the nests became social. This type of social plasticity might also exist in other lineages of Halictine bees; in southern Greece, a population of *Halictus sexinctus* contains both common and eusocial colonies, although it is not known whether this variation is due to a genetic or an epigenetic polymorphism (Richards et al. 2003). Given the wealth of genes implicated in environmentally dependent social behavior (Whitfield et al. 2006), it would be interesting to determine whether any of these candidates are epigenetically controlled and responsible for environmentally sensitive sociality in Halictine bees.

Voles (genus *Microtus*) vary in the degree to which they engage in social monogamy. Although rodents are typically promiscuous, monogamy is thought to have evolved in certain populations and species in response to resource limitation (McGuire et al. 1993). Monogamy has evolved in at least some populations of prairie (*M. ochrogaster*), pine (*M. pinetorum*), and mandarin (*M. mandarinus*) voles, and this variation has proven useful for understanding gene–brain–behavior relationships in an ecologically relevant context. Importantly, it has been appreciated for decades that vole social behaviors can be influenced by early life experience. For instance, female voles raised without a father display low spontaneous parental behavior relative to those raised by both parents (Ahern and Young 2009). Likewise, as mentioned earlier, cross-fostering experiments have demonstrated the nongenetic transfer of behavioral traits in California mice (Bester-Meredith and Marler 2003) and rats (Weaver et al. 2004). Thus, it appears that in some rodent species, environmentally induced adult behaviors can be transmitted to offspring via parental care. However, there is currently no evidence of epigenetic mechanisms mediating behavioral transmission in voles. Further, little is known about the evolutionary and environmental history of different species and populations, or whether ancestral lineages possessed behavioral plasticity. Thus, systems undergoing contemporary divergence would be more appropriate for investigating the epigenetic origins of behaviors.

Certain avian systems are amenable for investigating the contribution of epigenetic inheritance to rapid evolutionary change because several studies have focused on the types of behaviors that allow birds to invade novel habitats (e.g., Wyles et al. 1983). In general, avian species with relatively large brains and innovative behavioral repertoires are more successful at invading new areas (Sol et al. 2002). As discussed earlier, learning and behavioral flexibility can provide an individual with a range of behaviors that are potentially adaptive for unpredictable environmental conditions. Further, the ability of an individual to learn is dependent on early life conditions (Champagne et al. 2008), and learning could be mediated by epigenetic mechanisms (Arai and Feig 2011). In spite of this compelling connection between environmentally induced epigenetic variation and avian personalities that are adept at colonizing novel environments, the relationship between epigenetic variation and colonization ability has not been tested.

House sparrows (*Passer domesticus*) are a tractable system for exploring the potential relationship between environmentally dependent epigenetic effects on learning or behavioral flexibility and the colonization of novel habitats. A native of Eurasia, *P. domesticus* has spread to every continent except Antarctica. Although most human-facilitated introductions of *P. domesticus* probably occurred during the 19th century, some recent introductions coincided with human development of certain areas (Anderson 2006). During the 1950s, birds derived from South Africa colonized Mombasa, a city on the eastern coast of Kenya. Since then, house sparrows spread inland and reached the western border of Kenya in just the last few years (Anderson 2006). Thus, populations along this gradient differ in age. We have found that genome-wide methylation polymorphism is maintained in Kenyan populations even though genetic variation is dramatically reduced compared with older established populations (Schrey et al. 2011). Additionally, ongoing research in our labs is investigating whether birds at the front of the invasion—which are most likely to benefit from exploratory behavior—differ in the methylation status of their glucocorticoid receptor promoter, which might correlate with an individual's propensity to exhibit fearless or exploratory behavior (Meaney 2001).

A similar, recent invasion has occurred in the house finch (*Carpodacus mexicanus*), whose ancestral range prior to 1850 was confined to the west coast of North America (Hill 2002). The house finch was introduced to New York in the 1930s, and subsequently spread across all of the Eastern United States, southern Canada, and recently as far as northwestern Montana (Badyaev 2009). This successful colonization was made possible by environmentally modified reproductive behavior; specifically, the time at which females began egg incubation. Egg incubation is facilitated by the hormone prolactin, which also governs the order in which males and females of a clutch are produced (Badyaev et al. 2005). Extreme conditions (extreme temperatures or novel diseases, Badyaev and Oh 2008) result in variable incubation timing as well as variable levels of prolactin, ultimately resulting in sex-biased clutches (Badyaev et al. 2002, 2005). Sex-biased clutches, in turn, generate offspring with variable morphologies that appear to be crucial for survival in these novel environments (Badyaev et al. 2002).

An interesting evolutionary outcome of these environmental effects in *C. mexicanus* is that female behavior may have undergone genetic accommodation in populations established for longer periods of time. In recently established populations, females breeding for the first time produce sex-biased ovulation approximately proportional to the environmental stimulus, and fine-tune this induced effect over subsequent breeding events such that their reproductive behavior becomes environmentally independent (Badyaev and Oh 2008). In more established populations, sex-biased ovulation order is produced even by naive females in a precise, environmentally independent fashion (Badyaev and Oh 2008). Because this system possesses replicate, novel populations and a detailed record of behavioral data, it would be ideal for answering evolutionary questions about environmentally induced epigenetic variation in behavior.

CONCLUSIONS AND FUTURE DIRECTIONS

There are a few general principles that will likely be important for the nascent field of behavioral ecological epigenetics. The first is that environmental effects on behavior are often transmitted to offspring via the soma rather than the germline (Jablonka and Raz 2009). There are far more examples of soma-to-soma transmission than germ-line transmission among laboratory studies (Appendix A), and this bias might be more dramatic if complex environmental inputs were considered. For instance, this review did not cover cultural inheritance and niche construction, 2 more broadly defined, nongenetic processes that might have profound, non-germline effects on behavior and are common in nature (Avital and Jablonka 2000; Odling-Smee et al. 2003, Odling-Smee 2010). It is likely that, in many cases, these non-germ-line processes interact to produce heritable, molecular-level epigenetic variation that might influence behavioral traits (Danchin et al. 2011).

Secondly, the selective consequences of environmentally generated behaviors depend both on the inducing and the selecting environment. For instance, a study by Champagne et al. (2008) demonstrated that optimal learning in mice pups is achieved when they are tested under the conditions in which they were raised; mice raised under stressful conditions learn best when they are challenged under stressful conditions, and the reverse is true for mice who were raised under nonstressful conditions (Champagne et al. 2008). Therefore, to extend our understanding of epigenetically controlled behaviors into real-world situations, we must understand the degree to which inducing and selecting environments overlap.

Third, a major challenge in ecological behavioral epigenetics will be determining at what level we should identify epigenetic modifications. The epigenetic markers that contribute most to among-individual differences in behavior—and ultimately fitness—will be most relevant to evolutionary processes. The task of identifying these targets can be complex. Early life experiences interact with adult experiences, learning, and genetic variation to produce what we characterize as individual differences in behavior (Champagne and Meaney 2007; Champagne 2008). Thus, the most fruitful research programs will be those that identify *key* epigenetic modifications: those that best predict individual differences in a behavioral phenotype and ultimately become the targets of natural selection. Fortunately, there are a wealth of ecological, neuroendocrinological, and molecular studies that can inform our choices of genetic targets, tissues, developmental windows, environmental cues, and organismal systems.

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Appendix A

Mode of transmission	Taxa	Offspring behavioral phenotypes	Parental environment	Most proximate mechanism for transmission	Reference
Germline modification					
	Rattus norvegicus (rat)	Increased and decreased anxiety-like behaviors in female and male rats, respectively	Maternal exposure to the fungicide vinclozolin	Reprogramming of the germline via methylation	Skinner et al. 2008, 2010
	Rattus norvegicus (rat)	Female aversion to males whose ancestors were exposed to vinclozolin	Great-grandmother's exposure to the pesticide, vinclozolin	Possibly altered expression of major histocompatibility complex genes and major urinary protein 4 genes through methylation	Anway et al. 2005; Crews et al. 2007
	Rattus norvegicus (rat)	Depressive-like behaviors	Chronic and unpredictable maternal separation	Reprogramming of the male germline via methylation	Franklin et al. 2010
Soma-to-Soma transfer					
Allocation to Egg or Embryo					
	Mus musculus (mouse)	Hyperphagia (excessive consumption)	Neonatal overfeeding (through litter reduction)	Unknown	Schmidt et al. 2000
	Mus musculus (mouse)	Hyperphagia (excessive consumption)	Maternal overnutrition during pregnancy	Unknown, but thought that abnormally high levels of leptin lead to leptin resistance and affect hypothalamic functions	Samuelsson et al. 2008; Kirk et al. 2009
	Mus musculus (mouse)	Hyperphagia (excessive consumption) and reduced locomotion as the offspring compared to control rats	Maternal undernutrition during pregnancy	Unknown, however, neonatal treatment with the adipokine leptin reverses this developmental programming	Vickers et al. 2005
	Mus musculus (mouse)	Hyperphagia (excessive consumption)	When mouse dams possess a retrotransposon upstream of the agouti gene, their pups are subject to an obesity syndrome, and this effect may be ameliorated by maternal methionine supplementation	DNA hypo- and hypermethylation of the transposon in the *agouti* promotor region can allow and suppress the obesity syndrome, respectively	Wolff et al. 1998; Waterland & Michels 2007
	Mus musculus (mouse)	Aggressive behavior in females flanked by 2 males vs. those flanked by 2 females *in utero*	The effects of intrauterine position are enhanced by maternal stressors such as intense light, heat, and crowding	The sex of litter mates and corticosterone produced by mothers in response to stressors	Montano et al. 1991; Zielinkski et al. 1991; Ryan & Vandenbergh 2002
	Sialia mexicana (Western bluebird)	Aggressive males and non-aggressive males	Low resource availability causes mothers to produce more sons earlier, which promotes aggressive, dispersive offspring behavior	Maternal modification of the order in which male and female offspring are produced during oogenesis	Duckworth 2009
	Gasterosteus aculeatus (three-spined sticklebacks)	Tighter or looser shoaling behaviour: an antipredator defence	Mothers exposed to predators produce offspring with tighter shoaling behavior	Unknown; possibly via egg size or composition and possible involving egg corticosterone	Giesing et al. 2011
	Apis mellifera (honey bees)	Sexually productive queen and infertile worker bees. Workers are navigationally proficient and queens are colony-bound	The expression of the morphs is mediated by larval nutrition; that is, queens are raised on a specialized diet called "royal jelly"	More genes are methylated in queen bees than worker bees; the authors hypothesize that methylation is controlling the alternate splicing of certain genes	Lyko et al. 2010; Kucharski et al. 2008

Mode of transmission	Taxa	Offspring behavioral phenotypes	Parental environment	Most proximate mechanism for transmission	Reference
	Onthophagus taurus (dung beetles)	Males that fight for (major) or sneak copulations with (minor) females - major males provide food for their young and minor males provide no parental care	The expression of morphs is mediated by larval nutrition; that is, the amount of dung provided in an individual's brood mass	A spike of the hormone ecdysone that would normally cause horn growth is inhibited by juvenile hormone in small males, where size is assessed by some mechanism - possibly abdominal stretch receptors	Hunt & Simmons 1997; Moczek & Emlen 1999; Emlen & Nijhout 1999; Moczek & Emlen 2000
	Schistocera gregaria (desert locust)	Gregarious and solitary behaviors	Maternal crowding	Crowded females produce modified egg foam (containing an alkylated L-dopa analogue) that causes otherwise solitary offspring to become gregarious	Miller et al. 2008
Parental Behavior					
	Rattus norvegicus (rat)	An enhanced HPA (hypothalamic-pituitary-adrenal) response to stressors. Individuals with heightened HPA responses are more fearful than those with attenuated HPA responses.	Naturally occurring variations in maternal care in the first week of life are associated with changes in brain and behavior that persist until adulthood.	Maternal licking and grooming behavior result in alternations of DNA methylation of the *NR3C1* (glucocorticoid receptor) promoter region, and other genic regions on chromosome 18	Champagne et al. 2003; Weaver et al. 2004; McGowan et al. 2011
	Sehirus cinctus (burrower bug)	Begging behavior	Provisioning behavior: A cross fostering experiment demonstrated that female burrower bugs respond to signals from offspring in poor condition by providing more food	Unknown	Agrawal et al. 2001
	Microtus ochrogaster (prairie vole)	Spontaneous alloparental behavior and partner preference formation	Presence or absence of a father: A cross fostering experiment demonstrated that offspring reared without fathers demonstrated low alloparental behavior and delayed onset of partner preference behavior	Decreased overall parental licking and grooming may result in increased oxytocin content in the paraventricular nucleus and higher corticotropin releasing factor content in the dorsal raphe of offspring	Ahern & Young 2009
	Peromyscus californicus (California mice)	Pup retrieval behavior	Male Caliornia mice cross-fostered to the less parental *P. lecuopus* (white-footed mouse) display fewer instances of pup retrival (parental) behavior	Lower production of arginine vasopressin in the bed nucleus of stria terminalis (BNST) - there is a correlation between the expression of this neurotransmitter in the BNST and parental behavior in *Peromyscus*	Bester-Meredith and Marler 2003
Social Learning					
	Rattus rattus (Black rat)	Ability to strip pine cones	Social learning during early development (likely from mothers)	Learning	Aisner & Terkel 1992
	Tursiops sp. (bottlenose dolphins)	Using marine sponges as foraging tools ("Sponging")	Social learning (likely from mothers)	Learning	Krützen et al. 2005
	Sturnus vulgaris (European starling)	Ability to solve a novel foraging problem	Social context where faster learners "teach" slower learners how to solve the novel foraging problem	Learning	Boogert et al. 2008

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