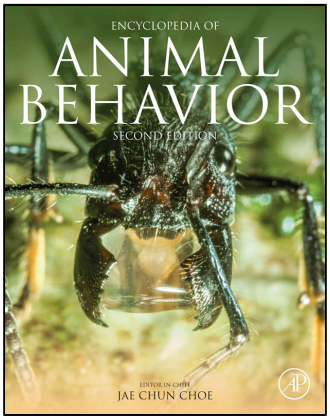

From Flanagan, S.P. (2019). Quantitative Genetics of Behavior. In: Choe, J.C. (Ed.), Encyclopedia of Animal Behavior, (2nd ed.). vol. 2, pp. 80–93. Elsevier, Academic Press.
ISBN: 9780128132517

Academic Press

Quantitative Genetics of Behavior

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Glossary

Adaptive landscape A multivariate surface that represents the mean fitness of a population as contours in relation to combinations of trait values. The adaptive landscape can incorporate concepts including phenotypic variation, selection, plasticity, genetic drift, epistasis, and speciation (Arnold *et al.*, 2001).

Allele One of a number of alternate forms of the same gene or genetic locus.

Epistasis The effect of one gene is dependent on the presence of another gene (i.e., the genetic background changes genotypic effects).

Fitness A description of an individual's reproductive success, fitness is the average contribution to the gene pool of the next generation that is made by individuals of the specific genotype or phenotype.

Genetic architecture The term typically refers to the number of genes underlying a trait, the way in which genes contribute to phenotypic variance (additivity, dominance, and co-dominance), the statistical interactions between those genes (epistasis) and their alleles (linkage disequilibrium), the physical relationships among genes (physical linkage), and the genetic correlations among traits.

Genotype The combination of alleles at a gene or genetic locus.

Indirect genetic effects The effects of another individual's genotype on the phenotypic trait value for another conspecific individual.

Linkage disequilibrium The non-random association of alleles at different loci. Linkage disequilibrium is a statistical association and does not necessarily imply that the loci are physically linked on a chromosome.

Non-additive genetic effects Factors influencing phenotypic variation that do not arise from additive genetic effects. Non-additive genetic effects include dominance, where one allele masks the effect of the other (recessive) allele, and incomplete dominance or co-dominance, where the heterozygous phenotype is intermediate between the two homozygous phenotypes. Additionally, interactions among genes cause epistatic effects, such as when genes are in a pathway or are physically linked on a chromosome.

Phenotype The trait value of an individual. The phenotype is determined by the combined effects of additive genetic effects, non-additive genetic effects, environmental effects, plasticity, gene-by-environment effects, and indirect genetic effects.

Phenotypic gambit The assumption that phenotypic correlations accurately represent the same patterns as genetic correlations, therefore assuming that the primary factors shaping phenotypic correlations are additive genetic effects (Brommer and Kluen, 2012).

Plasticity The ability of an organism to change its phenotype in response to the biotic or abiotic environmental conditions.

Pleiotropy One gene influences two or more different traits.

Polygenic The phenotype is determined by the effects from multiple genes.

Quantitative trait loci A section of the genome that correlates with variation in a phenotype. The quantitative trait locus (QTL) is usually linked to or contains the genes that influence the phenotype.

Abstract

Understanding the evolution of behaviors requires understanding how those behaviors are inherited. Quantitative genetics is perfectly suited for this task, because it partitions the variance in phenotypes within a population into heritable and non-heritable components. In this article, I describe how quantitative genetics approaches estimate these heritable components and outline commonly-used study designs for laboratory and wild populations. Furthermore, I describe the challenges unique to applying these methods to behaviors. Using a broad literature search, I provide an overview of what has been learned from the quantitative genetic study of animal behavior and I highlight fruitful areas for future research.

Keywords

Evolution; Genetic correlation; Genetic covariance; Genomics; Genotype-by-environment; G-matrix; Heritability; Indirect genetic effects; Maternal effects; Phenotypic correlation; QTL; Quantitative genetics; Relatedness; Repeatability; Selection

Encyclopedia of Animal Behavior, Second Edition, 2019, 80–93

Introduction

People have been studying the behavior of animals for centuries. Conspicuous animal behaviors, such as the mass migrations of monarch butterflies, fascinate people and capture their imaginations. Other behaviors, such as the movements of buffalo, historically were important to hunters and had more practical applications. Now, understanding behaviors has other practical applications, such as providing insight into human behavioral disorders and syndromes or minimizing negative interactions between humans and wildlife in suburban and rural communities (Alcock, 2009). Studying animal behavior is of further interest to biologists because behavior is the intersection of internal processes (such as genes, neurobiology, physiology) and external factors (such as abiotic environmental cues or the social context of an individual), and as such can be very dynamic (Ryan and Wilczyński, 2011). Importantly, the expression of behaviors can greatly impact the *fitness* of individuals – For example, a bold individual may secure a mate, or get eaten.

Understanding how behaviors have come to be, and how they may change in the future, requires knowing how behavioral traits are inherited. Many behavioral traits vary quantitatively – that is, they vary on a continuous scale and are not discrete – and as such their genetic basis (their mode of inheritance) is likely *polygenic*. Even discretized behavioral traits, such as aggressive vs docile behavioral syndromes, are likely under polygenic control (Boake, 1994). Additionally, many of the factors affecting the inheritance of behaviors arise from non-genetic effects, such as the social or ecological environments, and behaviors can exhibit great *plasticity*, changing rapidly within an individual's lifetime. Furthermore, behavioral traits are often comprised of multiple traits (Arnold, 1994). All of these elements impact the expression and evolution of behavioral traits, and to predict evolutionary outcomes we would like to differentiate between the roles each of these elements play. Evolutionary quantitative genetics is designed to peel apart these different sources of variation, and is therefore ideally suited for studying the inheritance patterns and evolution of behavioral traits.

The Quantitative Genetics Approach

What is Quantitative Genetics?

The fundamental concept in evolutionary quantitative genetics is comparing resemblance within families to resemblance among families to identify how much phenotypic variance originates from inherited material (genetics). Understanding these patterns of inheritance enables us to predict responses to selection, one of the key goals of evolutionary biology (Arnold, 1994). In an evolutionary context, selection is a phenotypic concept describing the statistical change in *phenotypes* from one generation to the next, and inheritance is a genetic concept describing how the effects of selection are transferred from parents to offspring (Arnold, 1994). Quantitative genetics focuses on phenotypes rather than *allele* frequencies – instead of analyzing genotypes, quantitative genetics focuses on a combination of selection and inheritance (Boake, 1994). A key advantage of the quantitative genetics framework is that it considers the action of selection on the traits with a direct link to fitness.

The evolutionary quantitative genetics approach relies on statistical descriptions of inheritance and the genetic basis of phenotypes at a population level. To understand how selection impacts the distribution of a given trait in a population, we need to consider both the inheritance of a trait and the strength of selection. A given trait value (z) has two major components: (1) The summation of the average effects of each gene, which define the genetic property of parents that produces resemblance among offspring (the additive genetic effects, a); and (2) the environmental effects (e):

$$z = a + e$$

At the population level, the variance in the trait (P) is similarly composed of the additive genetic variance (G) and the environmental variance (E):

$$P = G + E$$

Heritability (h^2) is the slope of the regression of offspring traits on the average trait values of the parents, and is equivalent to ratio of additive genetic variance to phenotypic variance: $h^2 = G/P$. The heritability is therefore a useful term describing how much of the trait variation arises from additive genetic effects (This measure of heritability is called narrow-sense heritability, because it refers only to the additive genetic variance. Broad-sense heritability (H^2) refers to the proportion of phenotypic variation that is genetic, including non-additive genetic effects). To measure how the mean trait value ($\bar{z}$) changes in response to selection, we first measure the difference in mean traits between successful and unsuccessful individuals to calculate the selection differential, s, and impose that strength of selection on the genetic component of the traits:

$$\Delta\bar{z} = h^2 s$$

This equation can be a powerful tool, but in the case of many traits – behavioral traits in particular – We must consider multiple traits simultaneously (Arnold, 1994).

The complexity added by considering multiple traits is that they are not usually independent. Therefore, it would be inappropriate, and would miss important information, if we were to simply apply $\Delta\bar{z} = h^2 s$ to each trait independently. Instead, we need to consider the correlations between traits. To do this, we make use of the additive genetic variance-covariance (or (co)variance for

short) (Remember that a correlation is a standardized covariance: $corr(X,Y) = \frac{cov(X,Y)}{\sigma_X \sigma_Y}$, where σ_X and σ_Y are the standard deviations of trait X and trait Y) matrix, **G**. (This G is bolded, rather than italicized as above, because vectors and matrices are presented in bold font in mathematics). In this symmetric matrix, the genetic variance for each trait is on the diagonal and the other elements are covariances between traits. The trait means can be similarly represented as a vector, and selection on each trait is also represented as a vector, which is called the selection gradient, β, and the change in trait means is:

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

Due to the rules of matrix multiplication, traits that have non-zero genetic covariances experience indirect selection from selection on their correlated traits (see Arnold, 1994 for an accessible and more comprehensive discussion about the mathematics behind these concepts).

These relationships between selection, genetics, and traits have important consequences for evolution. The trajectory of evolution can be constrained by genetic correlations. Genetic variation may not exist for one trait in the direction that is favored most by selection, so this may hamper the other trait's ability to follow the path towards the highest fitness. This is easiest to imagine by thinking about the *adaptive landscape* for two traits, where the trait values are on x and y axes and fitness is on the z axis – Fitness creates a landscape of peaks and valleys, where peaks are places with the highest fitness. However, the (co)variances among the two traits may be aligned so that the correlation makes it very difficult to climb up to the peak directly. Therefore, even though selection favors moving directly up towards the peak, the correlation between the traits forces them to move through lower-fitness trait combinations along a curved trajectory up towards the peak. The traits end up evolving along the lines of least resistance corresponding to where they have the most variation (Arnold *et al.*, 2001). The traits do what someone climbing a mountain is likely to do; a hiker will choose to stay on the already-built paths winding back-and-forth up a mountain, rather than forge a new path directly up the hill through a steep forest to the top.

A useful way to think about using quantitative genetics is by thinking about partitioning the variance in the phenotypes of interest. For example, let's consider mate choice in field crickets – Males sing to attract females, females exhibit preferences, and songs and preferences differ among species. Both male song and female preferences have an additive genetic basis (Bertram *et al.*, 2012; Blankers *et al.*, 2015), so the song of a male is in part determined by the genetic contribution from his father and mother. However, the ability for a male cricket to sing is costly (Mowles, 2014) and may be impacted by his physiological condition (Scheuber *et al.*, 2003) and the environment (Pires and Hoy, 1992). Individuals (each with its own genotype) may have different responses to the same environmental conditions, creating genotype-by-environment (GxE) interactions (Nystrand *et al.*, 2011). Additionally, the spermatophore provided by a male's father to his mother may impact his development or attractiveness, creating paternal effects (Head *et al.*, 2005; analogous maternal effects are also common in many species). Furthermore, there is additive genetic variation in female preferences, so females of different genotypes prefer males with different singing behaviors (Blankers *et al.*, 2015). Therefore, *indirect genetic effects* can emerge from females of specific genotypes preferring songs produced by males of specific genotypes. Using quantitative genetics approaches, Blankers *et al.* (2015) were able to piece apart the contributions of additive genetic variation and dominant genetic effects in male song and female preferences in two cricket species, *Gryllus rubens* and *G. texensis*, and identified variation from *epistasis* in female preferences. Armed with this knowledge, we can begin to understand how selection on phenotypes might actually result in evolutionary change. Although Blankers *et al.* (2015) focused on indirect genetic effects arising from female preferences and non-additive genetic effects, the quantitative genetics framework can incorporate many different types of ecologically and evolutionarily relevant factors impacting phenotypic variation, such as developmental processes (e.g., Stamps and Groothuis, 2010) and the indirect genetic effects arising from developing in the context of social interactions with parents and siblings (Kölliker, 2005).

Characterizing how phenotypic variation is partitioned, and the structure of the genetic components contributing to phenotypic variance, reveals the *genetic architecture* of traits. 'Genetic architecture' is a catch-all term describing the genetic basis of traits, and typically refers to the number of genes underlying a trait, the way in which genes contribute to phenotypic variance (additivity, dominance, and co-dominance), the statistical interactions between those genes (epistasis) and their alleles (*linkage disequilibrium*), the physical relationships among genes (physical linkage), and the genetic correlations among traits. The standard infinitesimal model of quantitative genetics assumes that the additive genetic value arises from an infinite number of unlinked genes, whose small effects are summed – This creates continuous genetic variation in the trait (Fisher, 1918). Clearly, the assumption of infinite unlinked loci is unrealistic, but this model can be a useful approximation for many traits, especially those with a polygenic basis. Considering *non-additive genetic effects* is important because they frequently contribute to variance in phenotypes and can impact the predicted responses to selection (Meffert *et al.*, 2002).

Two components of the genetic architecture that can critically impact inheritance and responses to selection are the physical location of genes and the multiple roles genes can play. The physical location of loci can create epistatic effects or even change patterns of inheritance if the genes are located on sex chromosomes. For instance, in the case of male singing behavior in crickets, Blankers *et al.* (2015) found that two of the male song traits had a bimodal distribution that was biased toward the maternal distribution in F_1 hybrids, indicating that those traits have some X-linked variation. Sex-linkage changes the patterns of inheritance such that genetic variance differs among sexes (Lynch and Walsh, 1998). Additionally, genes can shape phenotypes of multiple traits through *pleiotropy*, and this can create genetic correlations among traits. Common examples of genes with pleiotropic effects are the genes encoding hormones and their receptors, which often play major roles in behavioral phenotypes (McGlothlin and Ketterson, 2008).

What Approaches are Used to Study Quantitative Genetics in Animals?

To take an evolutionary quantitative genetics approach, researchers must estimate or know the relatedness among individuals in the study and use that relatedness to partition the variance in phenotypes into various genetic effects. The experimental design will determine how phenotypic effects can be partitioned because it determines which non-additive genetic effects can be differentiated from additive genetic effects. When choosing the appropriate experimental design, the primary consideration is whether the study can be conducted in wild populations or if it must be conducted in the laboratory. Studying populations in their natural habitats is generally preferable because it includes the environmental variance affecting phenotypes, variance that is relevant to the evolutionary trajectory for those phenotypes. Another deciding factor is whether multiple generations can be studied, or whether one is restricted to studying a single generation.

If both parents and offspring can be studied, the parent-offspring regression approach is often implemented. Parent-offspring regression directly estimates what quantitative genetics researchers want to know, which is the covariance between parents and offspring. Additionally, it is unbiased by selection on the parents (Lynch and Walsh, 1998). This approach requires assaying the behavior of interest in both the parents and their offspring and estimating the regression of offspring traits on parental traits (**Table 1**), which can be difficult for long-lived species with age-specific behaviors. If only the mother can be confidently identified, it is important to consider whether maternal effects might play a role in the system.

If a researcher can only assay a single generation, comparing siblings ("sibs") can be an effective way to partition the genetic variance. Half siblings have an expected covariance of ¼ of the total additive genetic variance, so estimating this value is relatively straightforward using a one-way analysis of variance (ANOVA; **Table 1**). However, half sibs experience common environmental effects, so pairing half-sib analyses with analyses of full siblings can decouple these effects (Arnold, 1994). Combined half-sib and full-sib analyses can also partition out dominance effects, although the power for estimating dominance effects is low (Lynch and Walsh, 1998). Most of these studies are conducted in laboratories, but under some circumstances these may be possible in the wild.

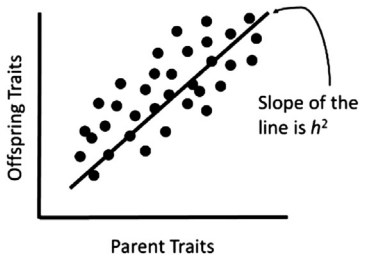
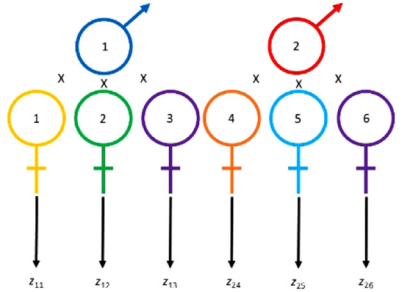
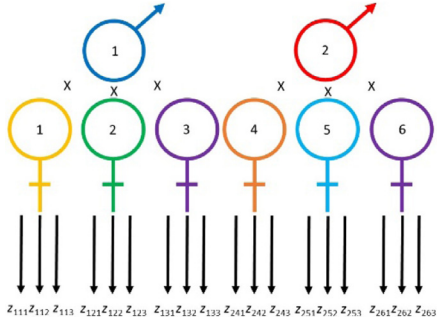
A whole class of experiments, the cross-classified designs, require the use of laboratory populations of animals. If both males and females can mate multiply, they can be paired up in a factorial multiple mating design (This is also referred to as the North Carolina Design II), where each female is mated with each possible male and vice versa (**Table 1**). Another version of this is when F_2 crosses resulting from inbred lines are mated in a fully factorial design, creating a scenario in which all segregating loci have a frequency of 0.5 (Lynch and Walsh, 1998). Inbred lines can be used in a number of different ways to isolate the additive and dominance components of the phenotypic variance, including in reciprocal crosses, F_2 backcrosses, and diallels (**Table 1**; Lynch and Walsh, 1998). Because these techniques require inbred lines, they have primarily been used in studies of behavior in insects such as *Drosophila melanogaster* and in mice. Diallels use males and females from the same inbred line, rather than from different lines, as the parents in crosses (**Table 1**). The diallel design enables the estimation of variance components when considering parental genotypes as random effects and identifying breeding values of parental genotypes when parental genotypes are treated as fixed effects (Lynch and Walsh, 1998). Because a full diallel requires a very large sample size, researchers often use partial diallels, where overlapping subsets of pairs are mated (**Table 1**). Although most cross-classified designs can be analyzed using two-factor ANOVAs, partial diallels generate complex standard error construction so a better analytical approach is to use general mixed models with restricted estimates of maximum likelihood (REML) (Lynch and Walsh, 1998) or Bayesian analyses (Visscher *et al.*, 2008).

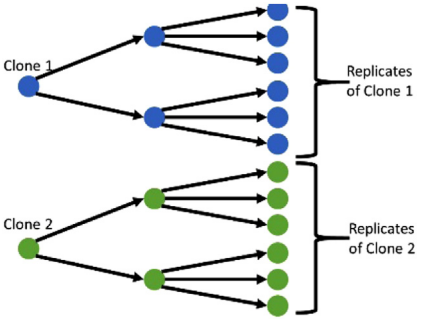
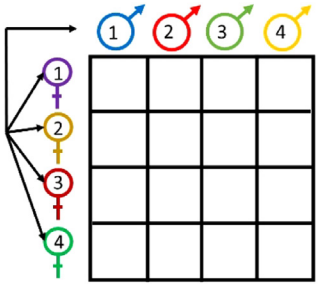
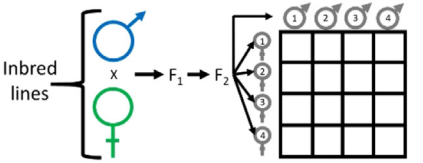
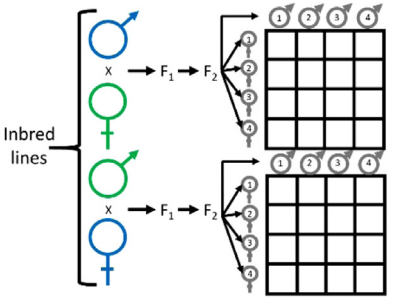
In sibling experiments and cross-classified designs maternal effects are not automatically differentiated from additive genetic effects (Note that maternal effects differ from maternal selection. Maternal effects impact offspring trait expression, whereas maternal selection impacts offspring fitness; Arnold, 1994). If both paternal and maternal half sibs are available, the maternal effects can be isolated through mother-offspring and father-offspring comparisons (Arnold, 1994). Otherwise, cross-fostering, F_2 hybrid crosses with more complicated experimental designs can isolate the effects of parental care and environmental effects from additive genetic variance (Arnold, 1994; see Lynch and Walsh, 1998 for detail on these experimental designs). Additionally, maternal effects commonly arise from parental care, so both maternal and paternal effects can occur in species with biparental care (Arnold, 1994); in such cases, it is important to consider both maternal and paternal effects in experimental design.

Genotype-by-environment effects may also be addressed through these laboratory-based experimental designs. By distributing paternal half sibs or isogenic lines across environments, a two-way ANOVA or estimates of covariances (Arnold, 1994) can identify the phenotypic effects of genes in isolation from the environment (Lynch and Walsh, 1998). However, the environments chosen for these experiments are critical – They must represent the environmental conditions characteristic of natural populations (Arnold, 1994). In a recent common garden experiment with a damselfly (*Lestes sponsa*), populations reared in unrealistic intermediate temperatures had dramatically different genetic (co)variances for a suite of life history traits than populations reared in simulated natural conditions (Sniegula *et al.*, 2018).

A criticism of laboratory studies is that they may not reflect the true genetic architectures found in the wild, so evaluating genetic constraints or trajectories in the lab may not be valid for an ecological or evolutionary context (Arnold, 1994). Long-term studies of wild populations using pedigrees have been successful for estimating quantitative genetic parameters responsible for behavior in a handful of species, such as bighorn sheep (e.g., Réale *et al.*, 2009) and blue tits (e.g., Brommer and Kluen, 2012). Although the complex relatedness among individuals cannot be analyzed using ANOVA, many iterative restricted maximum likelihood and Bayesian modeling approaches exist to estimate genetic (co)variances from known pedigrees (Visscher *et al.*, 2008; Gienapp *et al.*, 2017) (These methods are also applied to unbalanced designs with simpler relatedness associations (i.e., sibling analyses; Visscher *et al.*, 2008)). These approaches are further advantageous because they can accommodate complex pedigrees, such as those

Table 1 Quantitative genetic study designs that can be used to study behavior. The major study designs are further split into different types of studies (e.g., sibling comparisons are broken into half-sib, full-sib, and half-sib and full-sib), which are in italic font. Population type refers to whether laboratory or wild populations can be used for each study design. The number of generations ("No. gens") describes the minimum number of generations required for the analysis. The "Types of variance" column describes the components of phenotypic variance that can be described by the study design, or other comments if applicable. The Analysis approach describes the statistical analysis needed for the simplest of these designs, and the number of studies ("No. studies") shows how many studies of the quantitative genetics of behavior used each type of study design. The final column contains diagrams for some of the designs

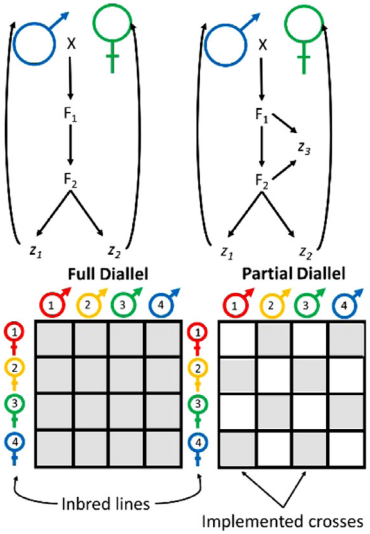
Study design	*Population type*	*No. gens*	*Types of variance*	*Analysis approach*	*No. studies*	*Diagram*
Parent-offspring Regression	lab, wild[a]	2	Dominance is not considered, but maternal effects are important	Regression	12	
Sibling comparisons						
Half-sib	lab, wild[a]	1	Cannot partition common environmental effects from additive genetic effects	ANOVA	16	
Full-sib	lab, wild[a]	1	Not useful for estimating heritability when done without half-sibs		15	
Half-sib and full-sib	lab, wild[a]	1	Partitions additive genetic effects from dominance, but estimating dominance effects lacks power	Nested ANOVA	10	
Common garden	lab	1	Estimates GxE effects	ANOVA/REML	10	
Cross fostering	lab, wild[b]	1	Decouple maternal or sire effects from additive genetic effects	ANOVA/REML	12	

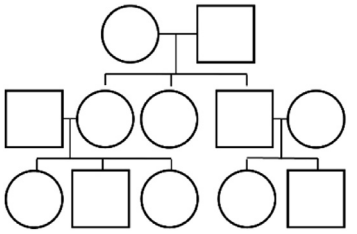
Clones	lab, wild	2	Only broad-sense heritability is relevant, and does not factor out maternal effects	ANOVA	6	
Cross-classified designs			Estimates additional variance components and provides better dominance estimates			
Factorial multiple mating	lab	1	Partitions paternal and maternal factors from dominance effects	Two-way ANOVA	7	
Factorial crosses from inbred lines	lab	3	Estimates dominance, but epistasis and gametic phase disequilibria are potential sources of bias.	Two-way ANOVA	9	
Inbred reciprocal cross	lab	2	Partitions the role of parental sex – Identifies mitochondrial or sex-linked effects	Two-factor ANOVA	2	

(Continued)

Table 1 Quantitative genetic study designs that can be used to study behavior. The major study designs are further split into different types of studies (e.g., sibling comparisons are broken into half-sib, full-sib, and half-sib and full-sib), which are in italic font. Population type refers to whether laboratory or wild populations can be used for each study design. The number of generations ("No. gens") describes the minimum number of generations required for the analysis. The "Types of variance" column describes the components of phenotypic variance that can be described by the study design, or other comments if applicable. The Analysis approach describes the statistical analysis needed for the simplest of these designs, and the number of studies ("No. studies") shows how many studies of the quantitative genetics of behavior used each type of study design. The final column contains diagrams for some of the designs—cont'd

Study design	*Population type*	*No. gens*	*Types of variance*	*Analysis approach*	*No. studies*	*Diagram*
Inbred backcross	lab	3	Additive and dominance effects can be estimated with similar precision	Two-factor ANOVA	10	
Diallel cross	lab	2	Estimate breeding values for parental genotypes and estimate variance components	ANOVA/REML	51	
Diallel common garden	lab	2	Estimates GxE effects in addition to the other genetic effects	ANOVA/REML	0	
Diallel cross fostering	lab	2	Decouples maternal effects from additive genetic effects	ANOVA/REML	0	
Complex relatedness						
Known Pedigrees	wild	≥2	Flexible – Can estimate many types of variance components, depending on design	REML/Bayes	40	
Relatedness	wild	1	Flexible – Can estimate many types of variance components, depending on design, but the effects of linkage are unclear	REML/Bayes	7	
Selection experiments	lab,wild[b]	3	Impose selection and measure the effects across generations, usually for multiple populations or genetic backgrounds	REML/Bayes	30	





Other					
Phenotypic Correlations	lab, wild	1	Assumes phenotypes reveal the same relationships as genetics	ANOVA or REML/ Bayes	9
Repeatability	lab, wild	1	Repeatability of a trait is the upper limit to heritability	linear models	7
Other breeding designs	lab, wild	1	Other more complicated uses of relatedness, sibship, and intercrossing – Can be used in a variety of scenarios	REML/Bayes	17
Genetic Architecture					
GWAS	lab, wild	1	Uses correlations among allele frequencies and trait values to identify potentially causal variants	Bayesian models	5
QTL Mapping	lab, wild	2	Uses crosses between individuals with different phenotypes to identify genomic regions that are segregating with the phenotype	ANOVA/Logarithm of odds	19

[a]Can be difficult in the wild.
[b]Requires experiments in natural population.

from wild-caught populations (Gienapp *et al.*, 2017). Additionally, molecular markers can be used to estimate variance components through comparisons of pairwise relatedness and pairwise phenotypic similarity. With the ease of generating thousands of markers distributed throughout the genome, molecular estimates of relatedness (Attard *et al.*, 2018) are facilitating the study of the quantitative genetics of behavior in wild populations in organisms without existing genomic resources (Gienapp *et al.*, 2017). Although the simplest models ignore non-additive effects, they can be extended to include dominance effects (Lynch and Walsh, 1998).

A further advantage of using genomic data to estimate relatedness is that researchers collect data that can also be used for isolating *quantitative trait loci* (QTLs) that underlie traits, simultaneously evaluating two forms of the genetic architecture (the patterns of inheritance and the gene function or location). Mapping QTLs can be an important goal for quantitative geneticists, because it moves us away from the strictly statistical framework of genetic (co)variances. In the genomic era, a form of QTL mapping called genome-wide association studies use single nucleotide polymorphisms (SNPs) generated from genotyping-by-sequencing or RNA-sequencing to identify genomic regions associated with the trait(s) of interest through correlations between SNP allele frequencies and phenotypes (Gienapp *et al.*, 2017). However, correlating QTLs with complex behavioral phenotypes is not always successful. For example, aggression in golden retrievers is highly heritable, but none of four candidate genes or 60,073 SNPs showed an association with aggression (van den Berg *et al.*, 2008).

Another approach, which has not been widely used to study behaviors, is to generate linkage maps and place QTLs associated with traits on those maps (**Table 1**). The key component to these studies is to create populations with linked loci in disequilibrium, using inbred lines, sibs and other relatives – although inbred lines have the fewest complications (Lynch and Walsh, 1998). Mapping QTLs in outbred populations is more difficult because many parents will not be informative, associations must be assessed in each set of relatives rather than averaged over all individuals, and these studies have lower power than studies of inbred lines (Lynch and Walsh, 1998). However, analyzing outbred populations characterizes QTLs segregating within a population, as opposed to between populations or lines, and therefore can be important. In general, because all parents are not informative, studies of outbred populations require large sample sizes (Lynch and Walsh, 1998), which may be a reason for their underuse in studies of the genetic architecture of behavioral traits.

If the response to selection can be directly measured, the Robertson-Price equation, $\Delta\bar{z} = \sigma_A(w,z)$, can be used to estimate the additive genetic covariance contributing to the trait. This equation describes the change in mean phenotype as the additive genetic covariance between the trait (z) and relative fitness (w). This method has the limitation that it if the trait is not under selection but has a genetic correlation to an unmeasured trait, then a substantial covariance can be observed – therefore, a multivariate approach to measuring selection is preferred (Pujol *et al.*, 2018).

In many cases, researchers are unable to estimate genetic (co)variances but are able to measure phenotypic changes and correlations. In theory, phenotypic (co)variances could represent genetic (co)variances. However, this *phenotypic gambit* is false when non-additive genetic or environmental effects play a substantial role in contributing to phenotypic variation (i.e., when heritability for a given trait is small; Brommer and Kluen, 2012).

Challenges to Applying the Quantitative Genetics Framework to Behaviors

Evolutionary quantitative genetics can be very useful for characterizing inheritance patterns, but it does have some limitations. A primary criticism is that using the genetic (co)variances to predict trait evolution requires that those (co)variances remain stable, which theoretical studies have shown is not always true (e.g., Jones *et al.*, 2003). Additionally, because genetic variances can be affected by factors such as demography and environmental effects (Pujol *et al.*, 2018), estimates of genetic (co)variances cannot necessarily be extrapolated to other populations (Visscher *et al.*, 2008). Indeed, many long-term studies of traits in natural populations have shown that genetic variance does not necessarily translate into evolutionary responses to selection, possibly due to statistical or biological effects (Pujol *et al.*, 2018). Nonetheless, partitioning the sources of variance in phenotype can help provide insight into the mode of gene action and the importance of non-additive effects, so the approach has merit, even if its ability to predict into the future is limited.

Estimating genetic (co)variances also has a number of statistical limitations. First, the statistical power is usually rather limited, and estimating heritability with a standard error of less than 0.1 usually requires a sample size of hundreds of individuals (Visscher *et al.*, 2008). Estimates of additive genetic variance can also be biased if alternative sources of variance, such as non-additive genetic variance or covarying environmental effects, are unaccounted for (Pujol *et al.*, 2018). The power to include these effects in analytical models may be missing if sample sizes are too small.

Applying evolutionary quantitative genetics to the study of animal behaviors has the additional challenge of requiring a scoring system that is simple, yet ecologically and evolutionarily relevant (Arnold, 1994). Behaviors are suites of traits, so a scoring system may need to be developed for each component trait. For example, male crickets serenade prospective mates with characteristic chirps and trills, with variation within and among species in the rate and duration of their songs, as well as the acoustic frequency. Researchers have used independent measurements of each of these song components, and female preferences for each component, within a multivariate framework to quantify male courtship and female preference behaviors (Bertram *et al.*, 2012; Blankers *et al.*,2015). Blankers *et al.* (2015) created hybrid crosses of two co-occurring species to characterize the covariation among these traits – in other words, how the traits come together to be the behavior of male song – and the patterns of inheritance. This quantitative genetics approach allowed Blankers *et al.* (2015) to infer that the song traits most strongly preferred by females are able to evolve independently (i.e., they did not covary significantly) but covary with non-preferred traits. Therefore, female preferences can

impose indirect selection on correlated traits. This insight into how male behavior (the suite of male song characteristics) may respond to selection imposed by female preferences gives researchers a better idea of how the species can differ substantially in song traits when female preferences do not differ as much between the species (Blankers *et al.*, 2015). Another option is to survey behavioral traits themselves. For example, Petelle *et al.* (2015) measured several behavioral traits (docility, sociality, and exploration) in wild yellow-bellied marmots. Using genetic markers to estimate relatedness, they were able to estimate heritability and genetic correlations between behaviors. Whether assaying component traits or the behavior itself, having a reliable and simple scoring system is critical.

Behavioral traits present the further challenge that they usually have low heritabilities (Mousseau and Roff, 1987), in part because they exhibit plasticity and can change rapidly and drastically based on the context of their expression. Additionally, repeatability places an upper bound on heritability, so researchers are recommended to use estimates of behavioral traits from averages of multiple behavioral trials to (Arnold, 1994). Applying this approach has also led to the characterization of animal personality, which are behavioral syndromes that show high within-individual repeatability.

What Have we Learned From Quantitative Genetics Studies of Behaviors?

Quantitative genetic studies of animal behaviors have been primarily conducted in model systems (32 studies of *Drosophila melanogaster*, 24 of *Mus musculus*, 12 of zebrafinches, *Taeniopygia guttata*, and 6 of *Caenorhabditis elegans*; **Table 2**) and in insects (118 of

Table 2 Species whose behavior has been studied in a quantitative genetics framework, grouped by class. Only species that were found in at least 3 or more different studies were included in the table. The bolded number in the third column is the total number of studies studying the associated class

Class	*Species*	*Number of studies*
Insecta		106
	Drosophila melanogaster	29
	Callosobruchus maculatus	5
	Gryllus firmus	5
	Nicrophorus vespilloides	4
	Plutella xylostella	4
	Tribolium castaneum	3
	Others	56
Mammalia		53
	Mus musculus	22
	Marmota flaviventris	5
	Mus doemsticus	5
	Macaca mulatta	3
	Rattus norvegicus	3
	Others	15
Aves		40
	Taeniopygia guttata	9
	Parus major	6
	Passer domesticus	5
	Gallus gallus domesticus	4
	Cyanistes caeruleus	3
	Others	13
Actinopterygii		25
	Poecilia reticulata	3
	Others	22
Arachnida		9
	Tetranychus urticae	4
	Others	6
Amphibia		7
Reptilia		6
Chromadorea		6
	Caenorhabditis elegans	6
Branchiopoda		4
	Daphnia spp.	4
Gastropoda		2
Malacostraca		2
Chlorophyceae		1
Rhabditophora		1
		262

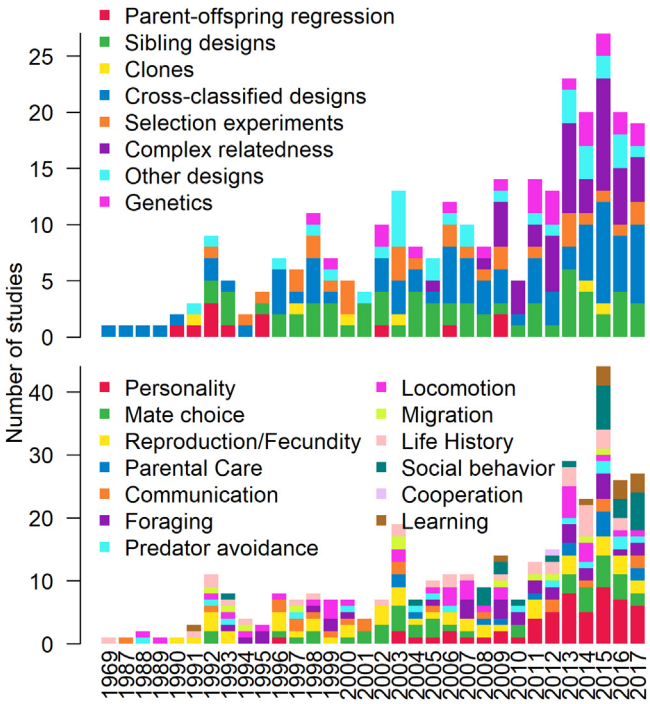


Fig. 1 This figure shows the number of studies using various study designs (top) and investigating different types of behaviors (bottom). Note that a single study may use multiple study designs (e.g., both a cross-classified design and a genetic approach to map QTLs) and might fall into multiple behavioral categories (e.g., both foraging and reproductive behaviors in phytophagous insects).

286 studies; **Table 2**), as revealed by a review of quantitative genetic studies of behavior in non-human animals (Supplemental Files 1, 2, Relevant Website). The bias is likely due to the fact that model species and insects can be more easily bred in laboratory conditions. However, modern genetic techniques have opened up the path for studying behaviors in wild populations because now relatedness can be estimated without the need for a known pedigree or any previous genetic resources (Gienapp *et al.*, 2017), which is reflected in the increase in the number of studies using complex relatedness in recent years (**Fig. 1**).

Four general topics have been commonly addressed in studies of behavior, namely the phenotypic gambit, indirect genetic effects, GxE, and plasticity. All nine of the studies relying solely on phenotypes (**Table 3**) assumed that the phenotypic gambit holds for animal behaviors. An additional eight studies estimated both phenotypic correlations and genetic correlations and compared the two. Of those, four found mismatches between phenotypic and genetic correlations, three identified correlations to be in the same direction, and one had a mismatch for some traits and concordance for others. Clearly the assumption that phenotypic correlations accurately represent the direction of genotypic correlations is unreliable. Therefore, whenever possible it is preferable to estimate genetic correlations rather than rely on phenotypic correlations for an evolutionary perspective on behavior.

Indirect genetic effects arise from interactions between individuals of different genotypes. Maternal effects are one of the most common forms of indirect genetic effects estimated and were the predominant form of indirect genetic effects studied on behavior and can impact personality traits (Taylor *et al.*, 2012; Petelle *et al.*, 2017). Generally maternal effects contributed to phenotypic variation, although a small number of studies found no evidence of maternal effects on behavioral traits (Forstmeier, 2005; Quinn *et al.*, 2016; Winney *et al.*, 2018). Other than maternal effects, the most predominant source of indirect genetic effects arose from aggressive (i.e., competition) or affiliative (i.e., courtship) social interactions (9 or 27 studies). In general, most studies including indirect genetic effects found these effects reduce heritability and can impact evolutionary trajectories (e.g., by contributing to evolutionary stasis, Santostefano *et al.*, 2017).

Like indirect genetic effects, gene-by-environment interactions also reduce the heritability of behaviors. The vast majority of GxE experiments measured changes in behavior in response to dietary manipulations. Many of the studies that tested for GxE focused on foraging behaviors (8 of 20, 40%), primarily in the context of specialization of feeding and oviposition in phytophagous insects (e.g., Forister *et al.*, 2007). The remainder of GxE studies tended to focus on the effects of the environment on mate choice behaviors and personality. Plasticity and GxE are often related, as significant GxE effects imply that there is heritability in the plasticity of the trait. Several studies have estimated the heritability of plasticity itself and genetic correlations between plasticity and other traits, although many simply asked whether plasticity in behavioral phenotypes exist. Behavioral plasticity is also a major focus of studies on personality; as attention to animal personality increases (**Fig. 1**), so will attention to the heritability of plasticity itself.

A broad range of behaviors have been studied using quantitative genetics, but the most-studied topic has been animal personality, or behavioral syndromes (**Table 3**), and this topic has increased in popularity since 2003 (**Fig. 1**). Animal personalities are described as repeatable behaviors within individuals, and can be measured as behavioral phenotypes, such as boldness/aggression

Table 3 The number of studies that analyzed each behavioral category and addressed each of the four topics. A single study can belong to multiple categories. A total of 262 studies were identified by the Web of Science search

Category	*Number of Studies*
Behavior	
Personality	58
Reproduction and Fecundity	45
Mate choice and Sexual selection	43
Locomotion	35
Foraging	33
Life history	33
Social behavior	27
Communication	22
Predator avoidance	17
Parental care	14
Learning	12
Migration	11
Cooperation	1
Topics	
Indirect genetic effects	27
Plasticity	20
GxE	20
Phenotypic gambit	8

and shyness/meekness. Some studies have shown that these personality traits are a multivariate behavioral phenotype composed of multiple, highly correlated behavioral traits (Oswald *et al.*, 2013). In many species these personality traits are heritable (blue tits, Brommer and Kluen, 2012; wandering albatross, Patrick *et al.*, 2013; rhesus macaques, Brent *et al.*, 2014; blue tits, Class *et al.*, 2014), but personality can also be impacted by rearing environment (Brommer and Kluen, 2012). However, whether there are fitness consequences for heritability is less clear – Aggressive parents in wild blue tit populations have offspring with higher probability of recruitment (Class *et al.*, 2014), but aggression in rheseus macaques has no impact on reproductive success (Brent *et al.*, 2014). Additionally, a meta-analysis suggests that behavioral syndromes may substantially constrain evolutionary trajectories of populations (Dochtermann and Dingemanse, 2013). Therefore, although heritable variation exists for personality traits, whether selection acts on those traits and whether the traits impact a population's ability to evolve are remaining questions.

Reproductive behaviors have been another major focus of quantitative genetic studies, likely because of their direct influence on fitness. Many of the studies focusing on reproductive behaviors investigated oviposition and nest site preferences (16 of 45, 35.6%), multiple mating (8 of 45, 17.8%), or copulation duration or mating rate (7 of 45, 15.5%). The remainder studied laying date or first age of reproduction (11.1%), sexual vs asexual mating behaviors (6.7%), and tradeoffs between other behaviors (e.g., aggression) with fecundity or similar traits, such as milk yield in cows (13.3%). Consistent with a review on male-female correlations in multiple mating (Gromko, 1992), the results from these studies present conflicting evidence for correlations between the sexes and for trade-offs between nest or oviposition preferences and locomotion, foraging, or other life-history traits. Despite the lack of a consensus regarding trade-offs, the majority of the studies of reproductive behaviors identified substantial additive genetic variation, in addition to non-additive effects, maternal effects, and other indirect genetic effects.

Another popular topic of study is mate choice and courtship behaviors (**Table 3**). A recent meta-analysis of the quantitative genetic basis of sexually selected traits, such as mate choice and behaviors, identified noticeable trends for both traits and preferences. Primarily, female preferences had lower heritabilities than sexually selected traits and the heritability of sexually selected traits decreased with increased strength of selection (Prokuda and Roff, 2014). This meta-analysis analyzed data for morphological, physiological, and behavioral traits and found that behavioral traits had the lowest heritability estimates for both sexually selected and non-sexually selected traits (Prokuda and Roff, 2014), consistent with previous results for behavioral traits (Mousseau and Roff, 1987). Consistent with these findings, the majority of the studies I reviewed here found modest heritabilities for traits and preferences, although some studies found no significant heritable component to display behaviors (Meffert *et al.*, 2002) or preferences (Schielzeth *et al.*, 2009). The vast majority of the studies in my dataset estimated genetic variance, heritabilities, genetic and phenotypic correlations, and selection on courtship behaviors and/or traits (24 of 43, 55.8%). These studies were testing the most basic requirement for models of sexual selection – that heritable variation exists – or aimed to characterize trade-offs or correlations among traits. An additional 9 studies (20.9%) tested specific models of sexual selection (e.g., the Fisher-Lande Process, the good genes model, or models of sexual conflict "Describing alternative models of sexual selection is beyond the scope of this article, but see Kokko *et al.*, 2006 for an overview"), one of which explicitly compared these models (Mühlhäuser and Blanckenhorn, 2004). Despite this attention to testing models of sexual selection, no clear consensus has emerged regarding the relationship between the genetic architecture of sexually selected traits or preferences and the mechanisms of sexual selection processes.

Foraging is another set of behaviors that directly impact fitness and have received attention (Table 3). The majority of foraging behaviors studied were preferences for specific food items (i.e., host specificity in phytophagous insects) and foraging behavior as part of a behavioral syndrome (20 of 33). The remaining studies investigated trade-offs between foraging behaviors and other traits (7 of 33) or the fitness consequences of preference or resistance to consumption of detrimental compounds (i.e., pesticides, 6 of 33). In general, foraging behaviors were heritable and often exhibited trade-offs with other traits and behaviors.

What Still Needs to be Answered?

As the previous section highlights, evolutionary quantitative genetics has revealed a great deal about behaviors. For example, much of the phenotypic variance does not arise from additive genetic variation, although most behaviors have some heritable components (Meffert *et al.*, 2002; Prokuda and Roff, 2014). Social interactions and plasticity can decrease heritability estimates when they manifest as indirect genetic effects and GxE interactions. Importantly, the genetic (co)variances and non-additive effects of behaviors, including animal personalities, can greatly impact the evolutionary trajectories of populations and species (Dochtermann and Dingemanse, 2013).

Despite this progress, there is much still to learn. Many of the studies highlighted here were conducted in laboratories, and heritability estimates in the lab may be substantially different from those in the wild (Arnold, 1994). Additionally, genetic (co)variances are often different among populations, but most studies focus on a single population – for obvious logistical constraints. Because of this, much of the theory regarding how genetic architecture impacts population differentiation and speciation has not been tested in wild populations. Similarly, although there has been much debate over mechanisms of sexual selection and mate preferences and sexually selected behaviors are highly studied, very few studies have attempted to resolve the sexual selection debate using quantitative genetics approaches.

A major gap in the knowledge of the quantitative genetics of behavior is the lack of linkages to genes (Bell and Dochtermann, 2015). The next critical step for the quantitative genetics of behavior will to begin to link genetic (co)variances with genomics. Does genetic variance arise from regulatory regions, coding regions, or non-coding regions? How does physical linkage impact epistatic effects? Supergenes have been shown to be involved in alternative reproductive tactics (Kupper *et al.*, 2016; Lamichhaney *et al.*, 2016; Winney *et al.*, 2018), suggesting that structural changes (i.e., inversions) and variation in recombination rate throughout the genome have the potential to drastically impact heritable behavioral variation in populations. So although we have learned a great deal about the heritabilities and genetic correlations of behaviors over the past several decades, the future is bright for integrating the quantitative genetics study of behavioral variation with genomics.

See also: **Evolution:** Cellular Epigenetics and Behavioral Evolution; Indirect Genetic Effects. **Genes and Behavior:** Animal Personalities and Behavioral Genetics; Behavioral Genetics; Evolutionary Behavioral Genetics; Studying the Genetics of Behavior in the Genomics Era.

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Relevant Website

https://github.com/spflanagan/literature-review/blob/master/docs/qgb_analysis.html. – Literature Review Analysis available on GitHub.