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Heritability

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Synonyms

Heritability, as a technical term, does not have direct synonyms. However, closely associated terms include "transmissibility" and "inherited variance," both of which relate to aspects of heritability.

Definition

Broad-sense heritability, H2, measures the proportion of a trait's variance within a population attributable to all genetic differences, relative to the total variance of the trait. Narrow-sense heritability, h2, specifically quantifies the proportion of trait variance due to additive genetic factors, again in relation to the trait's total variance.

Introduction to Heritability

Heritability is a fundamental metric in genetics and most branches of evolutionary biology. It estimates the proportion of variance in a particular trait within a population attributable to genetic differences. Typically expressed as a percentage, it illuminates the genetic underpinnings of traits and their inheritance mechanisms.

The primary objective of heritability studies is to assess the influence of genetics on variations in traits, like height or intelligence, among individuals. Crucially, heritability does not indicate the degree to which an individual's trait is genetically determined. Rather, it assesses how much of the variation of a trait across a population can be attributed to genetic factors in this particular population. In a different population of the same species, or in the same population but in a different environment, or even in the same population in the same environment but a few generations later, the heritability of the same trait can be completely different.

In various research areas, heritability offers substantial insights. In breeding programs, it informs selection decisions by clarifying the genetic basis of traits. In medical genetics, it plays a role in assessing the contribution of genetic factors influencing disease risk. For evolutionary studies, heritability estimates can predict how traits might evolve over generations.

It is important to reiterate that heritability is not a constant value for any given trait. Instead, it varies depending on the population studied and the prevailing environmental conditions. The fundamental aspect of heritability is its role in quantifying the genetic contribution to variation in a trait within a specific population. Essentially, it measures the impact of genetic diversity on the variability of a trait, rather than determining whether a trait is entirely genetic in origin.

Historical Context and Development of Concept of Heritability

The concept of heritability has undergone significant evolution, expanding from its origins in agricultural breeding to becoming an indispensable tool for analyzing genetic influences across a broad spectrum of biological traits. This evolution reflects the wider progress in genetic research, particularly in understanding the interplay between genes and the environment.

The foundations of genetics, laid by Gregor Mendel in the nineteenth century, set the stage for future developments, even though Mendel himself did not use the term "heritability." The early twentieth century marked a pivotal era with contributions from eminent scientists like Ronald Fisher, Sewall Wright, and J.B.S. Haldane. Their work, which combined Mendelian principles with quantitative genetics, was vital for advancing our understanding of trait variation. This period also established a crucial differentiation between broad and narrow-sense heritability. Broad-sense heritability accounts for all genetic influences on trait variance, whereas narrow-sense heritability is confined to the additive effects of genes. Understanding this distinction is crucial for appreciating the diverse genetic factors that shape trait variability and their impact on selection. Significant advancements were made in the field with the development of molecular genetics and genome-wide association studies (Bush & Moore, 2012). These developments have enhanced our comprehension of heritability, connecting specific genetic variants with trait variation. They have also brought to light various debates and misconceptions about heritability, particularly in the context of the *nature* versus *nurture* discourse. These discussions seek to elucidate the relative contributions of genetics and environment in shaping traits, including behavioral traits.

Understanding Broad- and Narrow-Sense Heritability

A crucial aspect of heritability lies in understanding its two main forms: broad-sense heritability (H^2) and narrow-sense heritability (h^2). This distinction is not just a technicality but a fundamental aspect that influences how we interpret genetic data and its implications.

Broad-sense heritability (H^2) encompasses the total genetic variance in a trait, including additive genetic variance, dominance variance (the interaction between alleles at the same genetic locus), and epistatic variance (interactions between different genetic loci). H^2 provides an overall estimate of the genetic contribution to a trait's variance but does not distinguish between the types of genetic effects. In contrast, narrow-sense heritability (h^2) focuses specifically on additive genetic variance. Additive variance refers to the sum of the effects of individual alleles, which contribute to the trait independently of other alleles. This form of heritability is particularly important in the context of response to selection, as it predicts how a trait will respond to natural and artificial selection over generations. It is the additive genetic variance that typically drives evolutionary change on the level of a population or single species (i.e., on microevolutionary time scale,

see the chapter "Macro- and Micro-evolution"), making h^2 a key concept in evolutionary biology and breeding programs.

Understanding the difference between H^2 and h^2 is vital for interpreting studies in genetics and evolution. It helps in predicting the response of a trait to selection pressures and in making informed decisions in breeding and conservation efforts. This distinction also aids in clarifying common misunderstandings about genetic influence, as it emphasizes that not all genetic contributions to trait variation have the same evolutionary implications.

In genetics, comprehending the heritability of traits is essential to understanding how characteristics are transmitted across generations. This comprehension deepens when distinguishing between qualitative and quantitative traits. *Qualitative traits*, also known as categorical or discrete traits, are defined by distinct, separable categories. Examples include phenotypes like blood types in humans or flower colors in plants, which Gregor Mendel famously studied in his pea plant experiments. These traits are usually governed by a single gene or a small number of genes, leading to clear Mendelian inheritance patterns. The heritability of qualitative traits focuses on the likelihood of these traits being passed down in an unchanged form to the next generation, often exhibiting straightforward dominant or recessive patterns. In contrast, *quantitative traits* display continuous variation and do not fall into discrete categories. Traits such as height, weight, or intelligence are influenced by multiple genes (polygenic) and multiple environmental factors, resulting in a range of phenotypes. The heritability of quantitative traits involves assessing how much of the variability in these traits is due to genetic factors. This assessment is more complex than with qualitative traits, owing to the involvement of numerous genes and their interactions with the environment. The study of quantitative traits and their heritability was a key area of focus for the *biometricians*, who applied statistical methods to understand the inheritance of these complex traits.

The distinction between the heritability of qualitative and quantitative traits has practical implications in various fields, including agriculture and medicine. While quantitative traits often use regression analysis for heritability estimation, qualitative traits are more directly linked to Mendelian inheritance patterns. However, the distinction between these two types of traits is not clear-cut. With the advent of modern biology, we now understand that many traits previously considered purely qualitative are influenced by multiple genes and environmental factors, much like quantitative traits.

The expression of many qualitative traits is explained by the *threshold model*. This model, originally proposed by Francis Galton, accounts for the expression of qualitative traits, such as the occurrence or nonoccurrence of a specific disease in an individual. It posits that an underlying liability trait, a quantitative trait with normal distribution in a population, determines whether an individual develops a certain trait or disease based on whether their liability exceeds a threshold. This model, while phenotypic, can be integrated into a quantitative genetic framework by assuming that both the liability and the threshold position are influenced by multiple genes.

In humans, many diseases are viewed as threshold traits. Characteristics like blood pressure and personality traits have a continuum of values, where a point on this continuum marks the transition from "normal" to "disease." This shows that traits, even those manifesting in binary forms, have a complex, polygenic background.

The threshold model is key to understanding many traits that appear binary but are influenced by multiple genetic factors. In studies like wing dimorphism in crickets, it helped identify a polygenically influenced, continuous underlying trait determining morph expression. This model is relevant in human genetics for understanding traits and diseases that might seem to follow simple Mendelian patterns but are actually influenced by a broader genetic context.

The threshold phenomenon is an important mechanism explaining the relationship between qualitative and quantitative traits in genetics. It indicates that traits, even those appearing as binary outcomes, are affected by genetic and environmental factors. This concept enhances our understanding of heritability across both types of traits, emphasizing that qualitative traits are often, and likely mostly, underpinned by continuous genetic variance.

Fundamental Components of Heritability

In understanding both broad-sense heritability (H^2) and narrow-sense heritability (h^2), it is crucial to consider their key components: additive genetic variance, dominance, epistasis, and environmental factors.

Additive genetic variance, the cornerstone of narrow-sense heritability (h^2), refers to the outcome of the cumulative, mutually additive effects of individual alleles, either from the same or different loci, on a trait. "Cumulative" in this context means that the impact of each allele on the trait can be summed together. For instance, if one allele contributes a certain amount to the height of an organism and another allele contributes another amount, the total genetic influence on height is the sum of these individual contributions. This summative property is essential in understanding how traits evolve and respond to selection, as it quantifies the overall genetic contribution by considering each allele's effect as part of a collective whole.

Dominance variance, contributing significantly to the genetic variance included in broad-sense heritability (H^2), arises from interactions between alleles at the same genetic locus. In such scenarios, one allele can mask or modify the effect of another allele on the phenotype.

Epistasis involves interactions between genes at different loci, where the expression of one gene is influenced by one or more other genes. These interactions contribute to the complexity of the genetic architecture of traits and represent a very important component of broad-sense heritability (H^2). Epistasis is especially relevant in polygenic traits, where multiple genes contribute to a single phenotype. In this context, however, it is necessary to note that the vast majority of traits are polygenic, meaning they are influenced by multiple genes. Many biologists believe that a large portion of traits are actually omnigenic, meaning that they are influenced to varying degrees by all the genes of a given individual (Visscher et al., 2006).

Environmental factors also play a significant role in broad-sense heritability, albeit indirectly, through their interactions with genetic factors. Elements such as climate, lifestyle, and upbringing, while

not directly contributing to genetic heritability, profoundly impact the expression of genetic traits. These factors can alter gene expression, influencing the observable characteristics of an organism. In both broad-sense and narrow-sense heritability calculations, the variance attributed to environmental factors is included in the denominator as part of the total phenotypic variance. In the calculation of H^2, broad-sense heritability, the numerator encompasses all genetic variance, including additive variance, dominance, and epistasis. While gene-environment interactions influence the overall phenotype, they are generally considered as part of the environmental variance and do not directly contribute to the genetic variance in the heritability calculation.

The variance in environmental factors is a key reason why heritability estimates for individual traits are not constant but vary across different studies. Such fluctuations are further influenced by the genetic composition of the population studied, highlighting heritability's inherent dependence on the specific genetic and environmental context of a population.

Box 1 Environment-Environment Interactions

Besides gene-environment interactions, it is also important to briefly acknowledge the existence of environment-environment interactions. Just as the effects of individual genes do not simply add up, the influences of individual environmental factors do not accumulate in a straightforward manner either; the influence of some environmental factors can modify, and often significantly, the influence of others. For instance, in the absence of the parasite *Adelina tribolii* (Sporozoa), the beetle *Tribolium castaneum* outcompetes *T. confusum*. However, in the presence of this parasite, the outcome of this interspecific competition is reversed (Park, 1948). Similarly, under normal circumstances, the parasite *Trypanosoma otospermophili* reduces the viability of Richardson's ground squirrels (*Spermophilus richardsoni*), but when the squirrels are maintained in conditions of insufficient vitamin B6 supply, the parasitized individuals fare better than the non-parasitized ones (Munger & Holmes, 1988). Environmental factor interactions can complicate the determination of heritability and the course of evolution driven by natural and artificial selection.

Methods for Estimating Heritability

Measuring heritability is key for breeding and evolutionary studies. There are various methods, both traditional and modern, offering insights into broad-sense and narrow-sense heritability. Each method has its specific focus and advantages, which are important for proper selection and application.

Estimating Heritability Based on the Similarity of Related Individuals

An intuitive way to measure heritability is to compare the expression of a trait among relatives. When measuring heritability by comparing the expression of a trait among relatives, such as the height of parents and their offspring, statistical methods are typically employed to assess the strength of the relationship between these values. These methods include *correlation* and *regression* analyses. Although in practice these analyses often proceed without creating graphs, visualizing individual observations on a graph can be useful for illustrating the basic principle of these methods. In such graphs, the trait values of the parents would be represented on the x-axis, while the corresponding values in the offspring would be on the y-axis. In studies of sexually reproducing individuals, and when data is available, we use the average trait value of both parents (*midparent value*) for the x-coordinate. If we draw a line through these points, the slope of the line is the expected increase of offspring trait per increase of one unit along the midparent value. The steeper the line, the greater the heritability. The same number can be obtained by calculating Pearson's correlation (*r*) coefficient between parental and offspring traits. This coefficient indicates the proportion of variance in a trait that is determined by genes that are shared between a parent and an offspring. The estimate of heritability is then calculated as *r* multiplied by 2. This is because each parent shares only half of their genes with their offspring. While sometimes used as an estimate, *r* alone does not directly reflect narrow-sense heritability coefficient (h^2), since parents and offspring still share some between-allele interactions. Theoretically, h^2 values can range from 0 (no genetic influence) to 1 (all variance is genetically determined).

Measuring traits in parents and offspring at the same age and in the same environment can be challenging. This is particularly true for long-lived species where changes in environmental conditions can be significant. Therefore, in practice, correlations between siblings' traits are often assessed instead of correlations between parents' and offspring's traits. Siblings share half of their genes, similar to the parent-offspring relationship. In the case of half-siblings, who share only a quarter of their genes, the obtained *r* value is multiplied by 4.

An important source of information can also be the correlations between monozygotic and dizygotic twins. Monozygotic twins share 100% of their genes, while dizygotic twins share only 50%. Thus, differences between monozygotic twins are attributed to environmental factors, provided we overlook rare events such as mutations and somatic recombinations, while the differences between dizygotic twins are attributed to environmental factors and the greater genetic variance resulting from their sharing fewer genes in common. Similarly, by observing the correlations in traits of monozygotic twins raised together and those raised separately, it is possible to estimate the variance due to environmental factors and the effects of gene-environment interactions.

By comparing values obtained from full siblings and half-siblings or monozygotic with dizygotic twins, we can estimate how much of genetic variance is due to additive gene effects and gene interactions. If we have information on all kinship coefficients in a population, i.e., if we possess a full genealogical tree for the population under study or data from genome-wide studies that can be used for calculation of kinship coefficients, we can use contemporary multivariate statistical methods that allow for more precise heritability estimates using information about both close and distant relatives (Visscher et al., 2006).

Box 2 Understanding Regression to the Mean in Heritability Studies

Regression to the mean is a phenomenon that applies to many systems, not just biological ones. For example, if a specific measurement – be it the outdoor temperature taken by a thermometer or intelligence determined by an IQ test – significantly diverges from the norm in any direction, subsequent measurements often show less deviation, tending to move back toward the average. This occurs because the deviation includes not only the true value

of the measured quantity, such as ambient temperature or intelligence of the tested individual, but also random influences like a gust of wind, luck, or misfortune in guessing the correct answer in an IQ test. These random influences most likely will not repeat in the same way in subsequent measurements.

In biology, the law of regression to the mean was first described, perhaps, by Charles Darwin's cousin, Francis Galton, in his studies of the heritability of biological traits through regression analysis. Galton observed this phenomenon when he examined the relationship between the heights of parents and their offspring. He found that although children's heights were correlated with those of their parents, they were more likely to be closer to the average height of the population rather than the extreme heights of their parents. Aside from random external environmental influences, the expression of biological traits and thus the regression towards the mean is also affected by random influences of the internal environment, specifically the genetic background. Offspring inherit half of their genetic material from each parent, thereby possessing a 50% chance of acquiring any specific allele from either parent. When considering a rare allele associated with an atypical phenotype in one parent, this translates to a 50% likelihood of its transmission to the offspring. Conversely, there is an equal probability of inheriting the more prevalent allele for that trait, potentially leading to a phenotypic shift towards the population norm in half of the progeny, regardless of the gene's additive influence. However, the phenotypic expression of traits is not solely determined by single-gene effects but also by epistatic interactions among multiple genes. For instance, in cases where an extreme phenotype, such as notable deviations in stature, is attributable to epistasis involving several genes, the chance of offspring exhibiting a similar extreme phenotype diminishes. This reduction is due to the decreased probability of inheriting specific allelic combinations required for such multigene interactions – 25% for two-allelic interactions and 12.5% for three-allelic interactions. Therefore, epistatic interactions play a substantial role in hastening phenotypic regression to the mean in subsequent generations.

The regression to the mean caused by the dilution of gene interactions necessarily complicates the estimation of narrow-sense heritability. The outcome of correlation studies inevitably varies depending on whether the correlation is calculated based on traits of close or more distant relatives. When analyzing close relatives, such as parents and offspring, the genetic interactions are more pronounced due to the higher likelihood of shared alleles that contribute to these interactions. However, as we extend our analysis to more distant relatives, the impact of these gene interactions on trait expression diminishes. This is because the probability of distant relatives sharing the same combination of alleles involved in specific gene interactions decreases significantly. As a result, heritability estimates derived from close relatives might capture not only the direct effects of individual genes but also the effects of gene interactions. In contrast, estimates derived from distant relatives are more likely to reflect additive genetic variance (h^2), since the influence of gene interactions diminishes with greater genetic distance. In such cases, the result of a correlation study tends to approximate narrow-sense heritability. Only the application of this logic across several orders of relatedness allows to calculate h^2 stripped of all the interaction affects precisely.

Estimating Heritability Based on the Selection Experiments

A fundamentally different method that allows for the estimation of heritability is *the selection experiment.* In this approach, we first measure the average value of the trait being observed. Then, we apply selection pressure – for example, removing individuals with a high value of the trait from the population and subsequently measuring the average trait value in the remaining population. The difference in values before and after selection is called the *selection differential*, *S*. Next, we allow the selected individuals to interbreed, and in the very next generation, we again measure and calculate the average value of the trait. The difference between the average trait value before selection in the first generation and in the second generation is known as the *selection response, R*. The selection response is usually much smaller than the selection differential, and the ratio *R*/*S* is an estimate of narrow-sense heritability, h^2.

The issue with this method lies in the diminishing selection response across generations, as the genetic variance within the population becomes progressively exhausted, and as the representation of alleles with frequency-dependent effects on biological fitness is increasingly shifted out of their equilibrium frequencies, see *Box 3*. Eventually, the population may reach a selection plateau, where the selection response drops to zero. In this state, h^2 appears to be zero, even though alleles with additive effects on the trait still persist in the population, and correlation studies continue to sometimes show high narrow-sense heritability of corresponding traits.

Estimating Heritability Using Genome-Wide Association Studies

Another possibility to estimate heritability is offered by methods based on genome-wide association studies (GWAS) (Bush & Moore, 2012; Visscher et al., 2006). GWAS tests the association of hundreds of thousands of genetic variants (usually single-nucleotide polymorphisms, or SNPs) with a phenotype in a large sample of individuals. Several approaches can be used to process the obtained data (Bulik-Sullivan et al., 2015; Visscher et al., 2017).

GWAS is a relatively new technique, and it has not yet fully demonstrated all its capabilities. However, technological progress in this field is very rapid. Today, we can include tens of thousands of individuals in a study and, on a single microchip, monitor hundreds of thousands to millions of loci at once. However, current experiences show that to fully realize the potential of this method, it will be necessary to include even more individuals in the study and monitor even more loci. Results indicate that the number of genes involved in the expression of a single trait is enormous, with each explaining only a very small part of the variance in that trait, having a small effect size. Therefore, a really large population sample is required to demonstrate them. Currently, it is still true that the combined effect of all genes identified by GWAS is much smaller than the heritability of a given phenotypic trait estimated using classic heritability measurement techniques. However, this difference is gradually decreasing with the development of techniques that allow processing larger data sets, thus capturing the effects of genes with smaller impacts on the trait (Visscher et al., 2017). Consequently, the initially concerning "missing heritability" effect is gradually losing its disquieting aspect.

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Genetic Influences Across Trait Spectra

The study of trait heritability has revealed significant variations among different trait categories, uncovering discernible patterns. Generally, traits controlled by fewer genes and exhibiting simpler genetic architectures tend to have higher heritability. On the other hand, traits influenced by a larger number of genes (polygenic traits), especially those demonstrating complex genetic architectures, often exhibit lower heritability (Falconer, 1981). Also, traits directly affecting fitness, such as clutch size, typically have lower heritability compared to traits with indirect fitness impacts, such as body coloration or hunting behavior.

A comparative study on twins raised together and separately by Bouchard, Lykken, McGue, Segal, & Tellegen (1990) revealed that the highest heritability estimates were observed in morphological traits, with fingerprint ridge density showing an R-value (interclass correlation) of 0.96 and height an R-value of 0.93. The study also found significant heritability in other areas: cognitive abilities with R-values ranging from 0.73 to 0.88, physiological and psychophysiological variables between 0.54 and 0.82, personality factors at 0.49, and personal interests and attitudes ranging from 0.28 to 0.48. It was found that the correlation of traits measured in twins raised together was higher than in twins raised separately. An interesting exception was social attitudes, except for religious attitudes, where correlations were sometimes higher for twins raised separately than together. This may reflect an effort by children within a family to differentiate their opinions from each other and find their own opinion "niches," see the chapter "Birth Order".Similar findings were also observed in the extensive study by Mousseau and Roff (1987). Additionally, this study demonstrated that the heritability of traits varies across different taxa, typically being significantly higher in endothermic animals compared to ectothermic animals.

As anticipated, traits exhibiting greater reproducibility in measurement, meaning those yielding consistent results upon repeated assessments in the same individual, demonstrated higher heritability. For traits characterized by lower measurement reproducibility, particularly behavioral traits, it is advisable to conduct multiple measurements on the same individuals. Utilizing the average of these repeated measurements as the input for heritability calculations enhances the accuracy and reliability of the heritability estimates.

The heritability of many traits changes with the increasing age of individuals (Bergen et al., 2007; Kim et al., 2021). This phenomenon is particularly noticeable in human intelligence, which is often the subject of research interest. For example, a study conducted on 11,000 pairs of twins showed that the heritability of general cognitive ability increases significantly and linearly from 41% in childhood (9 years) to 55% in adolescence (12 years) and to 66% in young adulthood (17 years) (Haworth et al., 2010). The most likely explanation for this phenomenon is that in childhood, the main source of differences in intelligence among individuals is the environment, so the measured IQ test values depend mainly on how stimulating the environment in which the child grows up and how much attention the family pays to the child. Therefore, the measured heritability values are relatively low in childhood. In adulthood, on the other hand, genetic influences predominate, and the measured heritability significantly increases.

Heritability in Traits with Close and Distant Fitness Associations

The heritability of individual trait categories depends not only on their genetic architecture. It also strongly depends on how directly the traits affect the biological fitness of an individual. Traits directly affecting fitness, such as *life history traits* (clutch size, longevity, etc.) typically have lower heritability compared to traits with indirect fitness impacts, such as body coloration or hunting behavior (Mousseau & Roff, 1987). The traditional, Fisherian, explanation of this dependency is the combined effects of the directional selection (Fisher, 1958) and the process of stability-based sorting (Toman & Flegr, 2017). Genes, or more precisely alleles, that influence a trait with a strong direct impact on biological fitness either quickly become fixed (if they are advantageous for their carrier) or rapidly disappear from the population's gene pool (if they are disadvantageous for their carrier). In both cases, genetic variance disappears from the population. However, if these are alleles influencing a trait with an indirect impact on fitness or traits that affect fitness little or not at all, they can persist in the population for a long time. Their disappearance due to the effects of selection or genetic drift can be compensated by the continuous emergence of new mutations affecting the specific trait. For this reason, such traits consistently or even permanently exhibit high heritability.

Recent studies have questioned this explanation for the lower heritability of traits closely associated with fitness and proposed an alternative explanation more in line with empirical data. The authors point out that traits closely tied to fitness, especially life history traits, are influenced by numerous morphological and physiological traits, which are affected by both genes and the environment. In addition, life history traits are influenced by various environmental factors, both external (such as food sources) and internal. The internal factors include the influence of alleles at other loci, often referred to as genetic background. Since life history traits are further down the causal pathway from genes to phenotype than, e.g., morphological traits, they inherently have additional sources of variance. Therefore, for life-history traits, the ratio of variance explainable by genes with additive effects to the total variance in a given trait, i.e., narrow-sense heritability, is lower than in other traits (Price & Schluter, 1991). According to the new hypothesis, the lower heritability is not due to a lack of additive genetic variance (Va) but to greater environmental or nonadditive genetic variance (Kruuk et al., 2000; Merilä & Sheldon, 1999). Testing this hypothesis with real data revealed that, in accordance with the predictions of the new hypothesis, h^2 decreases as a trait's link to fitness increases, while no correlation exists between additive (or nonadditive) genetic variance and trait's link to fitness (Wheelwright et al., 2014). Geneticists attribute the presence of a substantial amount of additive genetic variance in traits closely linked to fitness to the fact that traits like fecundity or longevity are influenced by a larger number of genes compared to, for instance, morphological traits. This makes them a larger target for mutational processes (Houle et al., 1996). The continual influx of new mutations in these numerous genes provides a rich source of additive genetic variance, continuously replenishing what selection constantly removes in the same fitness closely associated traits.

Box 3 Gene Pool Elasticity in Directional Selection

Another possible reason for the seemingly counterintuitive observation that genes with additive effects are responsible for much of the variance even in traits under intense directional selection is *frequency-dependent selection* combined with *pleiotropy* – the fact that a single gene affects not just one trait but many. Alleles with negatively frequency-dependent effects on fitness are difficult to fix in or eliminate from a population, because they become more advantageous as they become rarer in the gene pool and less advantageous as they become more common. Such alleles arise rarely in a population but accumulate over time due to the universal process of *stability-based sorting* (Toman & Flegr, 2017). Due to pleiotropy, over time, most genes in a population's gene pool become interconnected in an elastic network (Flegr, 2010, 2013b). If selection pressure acts on a certain trait, it displaces the frequency of many alleles from their stable equilibrium, and because at least some of these alleles have negatively frequency-dependent effects on fitness, population increasingly resists the change in that trait, eventually stopping any change in the given trait. Although the population or whole species is subject to directional selection, it can only respond elastically and reversibly.

One of the many consequences of a population's stabilized gene pool is the inability to eliminate variance determined by genes with additive effects (V_a), even when the corresponding traits are subject to strong directional selection. Interestingly, the ability to respond to selective pressures only elastically and thus reversibly provides a species an advantage in changing environments where conditions fluctuate periodically or aperiodically (Flegr & Ponížil, 2018; Williams, 1975).

This "evolutionary freezing" (reduced or sometimes even nullified evolvability due to frequency-dependent selection) of a gene pool of a population is characteristic of sexually reproducing species. These species are typically diploid, permanently carrying two copies of each gene. If a diploid species stops reproducing sexually, it inevitably transforms into functionally haploid over time. In each locus, one of the alleles can undergo mutations that inactivate the mutated copy of the gene without penalty. This does not occur in sexually reproducing species, as recombination would result in the emergence of nonviable offspring carrying two inactivated copies of genes in many loci (Lewis & Wolpert, 1979).

Diploidy, the presence of two copies of each gene in every individual in a population, is a significant source of frequency-dependent selection. Many alleles are indeed beneficial when present in one copy in the genome, but disadvantageous in two copies. It is because new alleles typically arise through mutations that alter the function of their products. Very often, the product of the mutated allele ceases to perform or worsens in performing its original function. As long as the new allele is rare in the gene pool, it is almost exclusively found in the genomes of heterozygotes. In this case, the original function is ensured by the allele on the homologous chromosome, so the heterozygote benefits from having alleles that perform both the old and the new functions. This phenomenon is likely one of the reasons behind *heterozygote advantage*, which refers to the high viability of individuals produced by the crossbreeding of highly unrelated parents, resulting in many genes being in a heterozygous state. However, as the new allele becomes common, it increasingly appears in homozygotes, who may be unviable or at least have reduced fitness due to the absence of the original allele ensuring the original function.

Due to the high prevalence of frequency-dependent selection in sexually reproducing species, genetic variation persists long-term or permanently even in traits that have a direct and significant impact on biological fitness. The situation in species that reproduce long-term asexually, such as some reptile species, is not yet clear. It can be expected that in these species, variance determined by genes with additive effects will be smaller, especially in genes with an immediate impact on fitness, as predicted by the original Fisherian model-based hypothesis.

Heritability of Behavior

Technical Challenges in Behavioral Trait Analysis

Behavioral patterns form a crucial part of an individual's phenotype. Although an individual's behavior is controlled by their nervous system, genes play a role in shaping the development of this system and its current tuning, thereby indirectly influencing behavior. In comparison to other types of traits, determining the genetic contribution to the overall variance of a specific behavioral component is considerably more challenging.

The first hurdle lies in the technical aspect. Measuring behavioral traits often poses difficulties, mainly because the reproducibility of behavioral tests tends to be significantly lower compared to that of morphological or physiological traits. The stochastic noise inherent in measurement outcomes adds to the environmental variance of the trait, thereby artificially lowering the heritability values obtained. One pragmatic, albeit imperfect, approach to mitigate this issue is to conduct multiple measurements of the behavioral trait and calculate heritability using the average of these values.

The second obstacle also leans towards the technical realm. Behavioral traits that interest us often cannot be measured directly but must be assessed through indirect tests. For instance, when investigating the heritability of intelligence, we do not measure intelligence directly but rather a person's performance in an intelligence test. The same holds true for reaction speed or any personality trait. However, performance in these tests depends not only on the traits of our interest but also on numerous other attributes of the subjects, such as their motivation, competitiveness, and willingness to cooperate (Flegr, 2013a). For example, performance in an IQ test is only partially dependent on intelligence, just as the actual extroversion of a person is only partially reflected in the extroversion measured in a corresponding personality test. This reality partly explains why correlations between behavioral traits and individual biological factors are usually very weak; in published ecological and evolutionary studies, the studied factors typically explain only about 2–7% of the total variance of the target variable (Moller & Jennions, 2002). It is safe to assume that in unpublished studies, the percentage of explained variance would be even lower. In the study of the heritability of behavioral traits, this source of error contributes to the environmental variance of the trait – factors like motivation, cooperativeness, and competitiveness of individuals are environmental influences with respect to measured intelligence. As a result, the measured heritability of a trait is lower than its actual heritability. While the issue of low reproducibility in behavioral

tests can be mitigated through repeated measurements, the problem of interfering variables cannot be resolved in this way. In addition to stochastic noise, these variables can also introduce a systematic bias into the results. Measurement imprecision and the resulting stochastic noise often result in false-negative test outcomes, failing to detect an existing effect. Conversely, systematic bias can lead to false-positive test results, identifying an effect that does not actually exist.

Complex Pathways from Genes to Behavior

The third hurdle is substantive in nature. The pathway from gene to phenotypic trait is typically straightforward and brief for many morphological traits, often just involving the expression of a specific allele that controls the synthesis or lack thereof of a particular pigment. However, this directness is generally not characteristic of behavioral traits. For instance, at the beginning might be a gene for the synthesis of a pigment protecting the skin from UV light. If individuals have two nonfunctional alleles of this gene, exposure to direct sunlight can be problematic for them. Repeated sunburns can lead to behavioral adaptation – teaching them to stay in the shade. This is still a relatively simple case. However, we can continue. In the shade, they may encounter different types of prey than if they stayed in the sun – for example, certain species of rodents. If rodents in the area are infected with the parasite *Toxoplasma gondii*, the individual in the shade can easily become infected, see the chapter "Toxoplasma Infection". *Toxoplasma* in the body of the infected individual causes local inflammations, exposing the individual to mild chronic stress. If the infected individual is a woman, she responds to such stress by increasing extroversion, becoming more sociable, and seeking social contacts more frequently. If the infected individual is a male, he reacts to chronic stress in exactly the opposite way – becoming introverted and avoiding social contacts (Lindová et al., 2006). In both cases, studies conducted on a sufficiently large sample would reveal a genetic component of the behavior. However, the path from the damaged gene for the enzyme controlling pigment synthesis to the behavioral pattern – increased or decreased tendency to seek social contacts – is so long and complicated that it would be very difficult to detect its existence, let alone to trace it. Environmental influences and gene-environment interactions would overshadow the influences of genes indirectly affecting variation in social behavior, whether it be genes for the synthesis of the pigment, genes influencing an individual's dietary preferences, genes for resistance to infection by *Toxoplasma*, or genes on the Y chromosome determining that the individual will (with high probability) be male. Of course, in many cases, the path from a gene to a certain behavior is simpler than the hypothetical example outlined above. In many cases, however, it may be even more complicated. In any case, the path from genes to behavior is on average longer and more "twisted" than the path from genes to morphological or physiological traits.

Box 4 Influence of Behavior on Human Attitudes

Behavior, however, is not at the end of the chain of causes and effects. For example, in humans, behavior influences attitudes as well. It is usually assumed that the causality is the other way around, that attitudes influence behavior (Kim & Hunter, 1993), but it is a question of which dependence is stronger and which is primary. Human behavior is strongly influenced by attitudes and the hierarchy of life values, but it is also influenced by unconscious behavioral drives, and in many respects, especially in matters directly or indirectly related to reproduction, it is controlled by subcortical brain regions. In many cases, a person watches their behavior in certain situations, often with some disbelief. Given a preference to act in accordance with their attitudes and personal value hierarchy, a person gradually adjusts this hierarchy to better align with their observed actions, as described by Bem (1968, 1972) and Festinger (1957). This is because adjusting attitudes and personal value hierarchy is mostly much easier than adjusting those behavioral patterns that are controlled by older areas of the brain.

Given the complexity of gene-gene, gene-environment, and environment-environment interactions, it is usually challenging for researchers to determine the extent to which genetics and the environment influence attitudes, such as political leanings or social viewpoints. The intricate and often obscure pathways from genes to attitudes are difficult to trace, and it is frequently even challenging to recognize that genes were at the outset of the pathways leading to a specific attitude or its behavioral expression.

Nature or Nurture

A common area of inquiry for biologists and psychologists/sociologists is the extent to which genetics and the environment contribute to specific behaviors or cognitive abilities. This inquiry, often framed as the "*nature or nurture*" debate, is not only academically intriguing but also has significant practical implications. Understanding the genetic and environmental underpinnings of behaviors can inform strategies in mental health, education, and social policy.

As I have attempted to demonstrate above, this exploration is fraught with complexity. In almost all cases of behavioral traits, both genetic (nature) and environmental factors (nurture) play roles. The contribution of genes to behavior is not straightforward, often involving a myriad of genetic interactions as well as the interplay between genes and various environmental factors. These complexities make it challenging to even roughly delineate the influence of each component.

Moreover, the impact of environmental factors on behavior is usually very complex. It encompasses everything from prenatal exposure, early childhood experiences, socioeconomic status, and cultural background to current life circumstances. Each of these elements can profoundly influence behavior and cognitive abilities, often in ways that are intertwined with genetic predispositions.

Recent advances in genetic research, particularly in the field of epigenetics, have further blurred the lines between nature and nurture. Epigenetics studies how environmental factors can influence gene expression without altering the DNA sequence. This emerging field has revealed that environmental factors can have a lasting impact on an individual's genetic expression, which in turn affects behavior. Epigenetic changes, for instance, those induced by a traumatic experience, can in many cases be passed on to offspring and may thus manifest across several subsequent generations (Heard & Martienssen, 2014; Yehuda et al., 2000). The possibility of transgenerational transmission of epigenetic information further blurs the line between genes and environment, or between nature and nurture.

The intricate interplay between genes and the environment leads many experts to believe that the seemingly simple question of "nature or nurture" cannot be resolved, especially in the case of human behavior (Bradshaw & Ellison, 2009; Turkheimer, 2022). Instead, it is recognized that behavior is the product of complex, dynamic interactions between an individual's genetic makeup and their environmental experiences. This perspective underscores the need for multidisciplinary approaches to studying behavior, integrating insights from genetics, psychology, sociology, and neuroscience.

In conclusion, while the quest to understand the relative contributions of genetics and environment to behavior is ongoing and will likely continue indefinitely, it is increasingly clear that the interplay between these factors is complex and dynamic. In exploring human behavior, the distinction between genetic and environmental influences is becoming less clear-cut, indicating the importance of methodologies that consider both aspects in behavioral research. However, we cannot discount the possibility that similar insights may eventually be gained for other trait categories, where the "nature vs. nurture" debate is not as heated – lacking the same level of practical and ideological implications.

The Relationship Between Heritability and Evolvability

The primary goal of measuring the heritability of traits has been to obtain a parameter that would allow the prediction of how each trait responds to directed selection, whether it be natural selection or artificial selection in the breeding of plants and animals. Evolvability, in the context of biology and breeding, typically refers to the short-term ability of a trait to respond to selective pressure. From a macroevolutionary perspective, studied by paleontologists, both the extent to which a trait responds to selection at a given moment and the duration for which it can respond are crucial, though these parameters may not necessarily positively correlate. In breeding practice and ecological studies, the short-term ability to respond to selection plays a significantly more important role, partly because we cannot observe processes lasting thousands or millions of years in real-time. In the case of artificial selection, where we need to estimate the evolvability of the same trait across different populations, heritability is a usable parameter for assessing a trait's ability to respond to selection. However, the same purpose could be served by the amount of additive genetic variance itself, not just its proportion in the overall variance of the trait.

When comparing different traits, it is necessary to standardize additive genetic variance. Variance is calculated as the sum of squared deviations from the mean and will naturally vary dramatically between small traits and larger ones.

Traditionally, genetic variance across traits is standardized by dividing it by the total variance of the particular traits, thus effectively calculating heritability. However, this approach is not the only possible and perhaps not the optimal one. If a particular trait has high additive genetic variance, it usually means that its expression is influenced by a large number of internal factors – i.e., a large number of genes. If the expression of a certain trait can be influenced by many genes, it is likely that it can also be influenced by a large number of environmental factors, thus having high environmentally induced variance. A high number in the numerator of the heritability calculation formula is therefore offset by a high value in its denominator, resulting in traits with very different abilities to respond to selection having similar heritability values. Indeed, it is often observed that traits with low heritability, like life history characters closely related to fitness, may respond more effectively to selection than traits with high heritability.

A solution to this problem was proposed some time ago. It involves standardizing the amount of additive genetic variance not by dividing it by the total variance but by dividing it by the average size of the trait, i.e, by computing the *coefficient of variation* (Houle, 1992). A comprehensive meta-analysis showed that there is practically no correlation between heritability and evolvability calculated in this way – many traits with low heritability had high evolvability and vice versa. Given the expected correlation between genetic and environmental variance, it would seem that evolvability, calculated as V_a/mean, better reflects a trait's capacity to respond to selection at a given moment than heritability. However, this is not always the case. High environmental variance can also mask the genetic variance from selection, thereby reducing the effectiveness of selection.

In the scenario of *soft selection*, where a fixed percentage of, say, the smallest individuals is consistently culled from a population, the selection response is significantly higher if the deviation from the average size in most of these individuals is genetically based, rather than environmentally influenced. For instance, if environmental factors like food scarcity or diseases caused the size deviation in half of the culled individuals, the effectiveness of selection would be comparatively reduced. In the case of *hard selection*, such as when predators or breeders eliminate all individuals below a certain weight threshold, the efficiency of selection should not be influenced by the level of environmental variance within the population. Consequently, the practical implication is that studies should consistently monitor and document both heritability and evolvability.

As previously mentioned, macroevolutionary and microevolutionary potential – the ability of species to change over long-term or short-term time scales – are two completely different things that may not be related at all. In reality, however, the inability of a species to respond to selection in the short term, such as during a selection experiment, can have at least two fundamentally different causes. The first is the absence of additive genetic information, and the second, more likely, is *frequency-dependent selection*, which prevents the frequency of individual alleles from deviating too much from a stable equilibrium state, see *Box 3*. In both cases, the evolutionary response to equally intense selection gradually decreases over time. In the first case, this is because the additive component of genetic variance originally present in the population is gradually depleted; in the second, the deviation of certain alleles from the original equilibrium state gradually increases. Rare alleles (those against which selection was directed) become increasingly advantageous for their carriers, and abundant alleles (those in favor of which selection was directed) become increasingly less advantageous for their carriers. At some point, both these effects become so strong that the population ceases to respond to selection. Which of the two possibilities, depletion of additive variance or stabilization of the gene pool composition by frequency-dependent selection, is at play can be easily determined. If it is the first possibility, the mean phenotype of the population members remains the same; if it is the second, the mean phenotype in

subsequent generations returns initially quickly, later more and more slowly to the state before the start of selection (Flegr, 2010). The results of laboratory selection experiments tend to favor the latter possibility (Dobzhansky & Spassky, 1969).

In conclusion, evolvability on a microevolutionary time scale is fundamentally different from evolvability on a macroevolutionary time scale. Both types of evolvability likely differ significantly in the processes and parameters that limit them. Heritability plays a key role in short-term microevolutionary processes and specifically pertains to a population's ability to respond to selection at a given moment. Which parameter better describes evolvability, whether heritability or evolvability calculated as the trait's coefficient of variation, depends on several factors, including the type of selection the population is subjected to.

It is crucial to note that both heritability and evolvability are not static, even within a single population. They tend to decrease over time, potentially dropping to zero, either as the additive genetic variance in relevant traits is exhausted or as the gene pool's composition approaches its elasticity limits, which are determined by the competition of alleles with frequency-dependent effects on fitness.

Conclusions

In summarizing this chapter, we highlight the dynamic relationship between genetics and the environment in determining traits. Heritability, a crucial concept in genetics and evolutionary biology, is dynamic, with its influence fluctuating based on specific population characteristics and environmental factors.

The evolution of the heritability concept, stretching from its agricultural roots to its pivotal role in diverse scientific fields today, mirrors the broader advancements in genetic research, notably in molecular genetics and genome-wide association studies. This development underscores the importance of understanding heritability in its two forms: broad-sense (H^2) and narrow-sense (h^2). While H^2 encompasses all genetic variance, h^2 focuses specifically on variance caused by additive genetic factors, a distinction that is fundamental for accurately predicting how traits respond to different selection pressures in these advanced fields of study.

The interplay of genetic, epigenetic, and environmental influences on behavioral traits, especially in humans, raises complex questions about the extent to which behavior shapes attitudes and vice versa. This intricate relationship further complicates the enduring nature versus nurture debate, emphasizing the difficulty in disentangling the genetic basis of behavior from the impact of environmental experiences. Understanding this dynamic is pivotal, yet it remains an elusive goal due to the inherent complexity in defining the boundaries between innate predispositions and learned behaviors.

Finally, the concept of heritability is integral to understanding a trait's potential for evolutionary change. While pivotal in short-term evolutionary adaptations, heritability's influence is modulated by the dynamic nature of the gene pool. The response to selection, encompassing both immediate and long-term changes, depends on the interplay between genetic, epigenetic, and environmental elements.

Cross-References

- Birth Order
- Macro- and Micro-Evolution
- *Toxoplasma* Infection
- Xenoadaptations

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