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BRIEF COMMUNICATION

Using Variation in Heritability Estimates as a Test of G × E in Behavioral Research: A Brief Research Note

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Abstract
Better characterization of the sources of phenotypic variation in human behavioural traits—stemming from genetic and environmental influences—will allow for more informed decisions about how to approach a range of challenges arising from variation, ranging from societal issues to the treatment of diseases. In particular, understanding how the environment moderates genetic influence on phenotypes (i.e., genotype–environment interactions, or G × E) is a central component of the behavioral sciences. Yet, understanding of this phenomenon is lagging somewhat, due in part to the difficulties of detecting G × E. We discuss the logic behind one of the primary ways to detect G × E: comparing heritability estimates across environments. Then, we highlight some pitfalls, with an emphasis on how very strong G × E can sometimes be undetectable using this method when high heritability is present in multiple environments. We conclude by forwarding some initial, yet tentative, suggestions for how best to address to the problem.

Keywords Gene by environment interaction · Heritability · Quantitative genetics · Behavioral genetics

Introduction

For decades now, scientists have consistently uncovered evidence suggesting that variation across a wide range of human behavioral traits is partly accounted for by genetic variation (Plomin et al. 2013; Polderman et al. 2015; Turkheimer 2000). Equally well-documented is evidence that environmental factors also explain significant amounts of variance in complex phenotypes (Polderman et al. 2015). Thus, studying the constituent components of trait variance—genetic and environmental—is key for understanding individual differences. Yet, researchers have also long recognized the need to examine how one component might moderate the effects of the other (i.e., a gene–environment interaction or G × E). Gene–environment interactions occur when the effect of genetics on a phenotype become more (or less) pronounced, depending on environmental variation, or vice versa. Given the impact that studying G × E has had on understanding phenotypic diversity in a host of organisms across biology (Bradshaw 1965; West-Eberhard 1989, 2003; Via et al. 1995; Schlichting and Pigliucci 1998; Ghalambor et al. 2007; Karlsson et al. 2010), increased clarity about this particular form of gene–environment interplay in human beings will undoubtedly lead to a better understanding of human diversity.

Difficulties in measuring variation in human traits arise in part from the quantitative nature of these traits. As with most phenotypic traits in most organisms, many key human traits vary continuously owing to the action of multiple genetic loci and numerous environmental effects, as well as the interactions of genes at different loci, both with one another and with the environment (Falconer and MacKay 1996). Thus, the study of G × E in humans is fraught with a variety of methodological and practical difficulties that can be more easily sidestepped in other corners of biology (via the use of

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experimental manipulation). Primarily, a multitude of ethical and practical constraints limit, and often preclude, the use of experimental designs and require the use of indirect methods for detecting G×E for human behavioral traits (Barnes et al. 2014). One primary approach for detecting G×E is to compare heritabilities of a trait across the environments of interest. To preview an example we utilize below, researchers in the psychological sciences have long suspected that socioeconomically deprived environments might moderate genetic influences on general cognitive abilities in human beings (Tucker-Drob and Bates 2016). Known as the Scarr-Rowe hypothesis, this topic has elicited much attention from researchers, and has been tested largely by comparing heritability estimates of cognitive ability across varying levels of socioeconomic success.

Despite the fairly common use of comparing heritabilities across environments to detect gene–environment interactions, there is an important concern embedded within this approach. From our vantage point, this concern remains under-appreciated by experimentalists in biology, and is underemphasized by researchers utilizing classical twin based designs to test for G×E. The primary issue is that, while this approach can certainly be used to suggest the presence of a G×E, a lack of variation in heritabilities across environments cannot be used to reject the possibility that an interaction exists. Yet, it is not uncommon to reach just such a conclusion when testing for interactions. Moreover, the reliance on comparing heritabilities to detect G×E without concurrently using other methods to confirm results is an approach that, while useful, may need to be revisited. The goal of this paper is to outline the logic behind the use of comparing heritabilities to detect gene–environment interactions, and draw attention to why it should not be used exclusively to conclude a lack of G×E.

Environmental variation and G×E

Environmental variation is an important source of phenotypic variation in human traits (Polderman et al. 2015). However, how much a phenotype for an individual differs across environments, and the nature of how the environment influences that individual's phenotype, can depend on the genetic background of the individual (Roff 1997). In other words, not all genotypes respond to environmental change in the same way, and this difference in how genotypes respond to environmental variation is referred to as G×E. Thus, when G×E is present, there is no one single function that can predict how a specific change in the environment generates a corresponding change in phenotype for all genotypes in a population (Roff 1997) because genotypes differ in either the degree or polarity of change across environments.

Let's consider an example where the environment of focus is pathogen presence/absence. If we expect that G×E is present, not all genotypes exposed to the pathogen will respond in the same way (Box 1). Instead, we expect the change in the level of health is minimal for some individuals because specific allelic variants confer resistance to infection, and the change in health is more dramatic for individuals without those allelic variants who fall gravely ill upon pathogen exposure. Yet more individuals may experience intermediate declines in health because of partial resistance conferred by allelic variants (Box 1). This model of G×E can be expressed using graphs, illustrated in the figure in Box 1[1] Graphs of this nature indicate the phenotype that corresponds to each genotype measured in the environments of focus; the resulting lines that connect the phenotypes of each genotype across environments are referred to as norms of reactions, or reaction norms. The presence of non-parallel reaction norms (i.e., reaction norms that differ in slope)—most dramatically seen between G1 and the other two genotypes—is the primary indication of G×E.[1]

Box 1: Key definitions, and a visual and verbal explanation of G×E

Environmental variation The proportion of phenotypic variance resulting from differences in the environment to which individuals are exposed.

Plasticity The expression of more than one phenotype by a single genotype, with expression dependent on environmental conditions. Plasticity is the mechanism that generates environmental variation.

Heritability The proportion of phenotypic variance that results from additive genetic variation among individuals. Because heritability is a proportion, it is unitless. It also is inversely related to the amount of environmental variation, such that total variance = 1.0. Heritability is environmentally specific such that it can be close to 1.0 measured in one environment and 0.0 in another. For

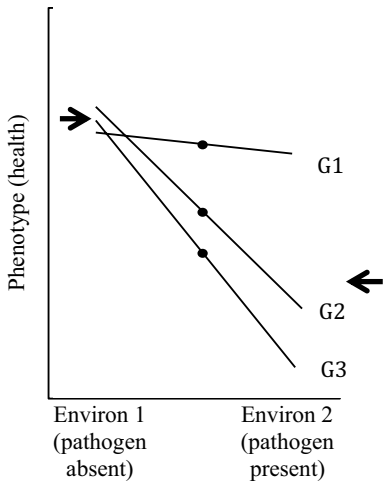
[1] It is worth pointing out that debates about the nature, prevalence, and importance of G×E are not new in behavior genetics, and some of the very points we touch on here have been discussed previously. See for instance, Sesardic (1993), and the exchanges sparked from around the topic of non-additive genetic effects and reaction norms. Moreover, behavioral geneticists have, for decades, acknowledged pitfalls when testing for G×E and have been suggesting supplementary methods, so neither is this component of our paper particularly novel (see, for instance, Plomin et al. 1977). Our intention, then, is to revive interest in the topic across fields where the discussion has either faded, or has yet to take hold in general (e.g., criminology, sociology, etc.).

example, if the environment is 'pathogen present,' not all individuals will necessarily get exposed to the pathogen, and so this variation in exposure will generate variation not captured by genetic differences among individuals.

Norms of reaction Descriptions of phenotypic expression, typically individual genotypes sampled, across a range of environments. Also referred to as a 'reaction norms.'

Explanation of box figure

The accompanying figure depicts the phenotypes of three genotypes across two different environments. In the pathogen example from the introduction, the phenotype is the level of health, and the environments are pathogens absent and pathogens present. Therefore each genotype has its own reaction norm that shows how healthy it is when pathogens are present versus absent. The line corresponding to G1 (genotype 1) indicates that this genotype is fairly healthy in both pathogen free and pathogen present environments. The lines corresponding to G2 and G3 show lower overall health when pathogens are present, likely because of allelic variants that confer less resistance to infection.



This figure can be analyzed for environmental variation. If we compare mean health between the two environments, we see greater mean health when pathogens are absent, indicating an effect of the environment on phenotype.

We can also determine if genetic variation is present within a single environment—indicated by variation among genotypes in their phenotype. In the pathogen absent environment, there is very little variation among genotypes, indicating very low genetic variation. In the pathogen present environment, there is significant variation among genotypes in health; assuming good estimates of phenotype and little variance within a genotypes, this indicates high genetic variation in health. Finally, if we consider heritability across all environments, we look at differences in the overall height of the reaction norms (i.e., differences in the mean phenotype of each of the genotypes) and see intermediate heritability. When genetic variation is present, the trait is said to be heritable, meaning that some of the variation among individuals in that environment is due to underlying genetic differences.

Finally, this figure demonstrates significant G×E in health. The slopes of the reaction norms differ among genotypes, which means that each genotype responds in a different way to a change in the pathogen environment. Note that, from a mathematical standpoint, genetic correlations of a trait expressed in different environments would be less than 1, with 1 indicating a retained rank-order of phenotype values across genotypes in different environments.

The logic of using variation in heritabilities to detect G×E

The data required to produce the graphs in Box 1 are often not readily available to behavioral geneticists studying human subjects, as one needs knowledge of *exact* genotypes and precise measurements of the environment to generate them. However, the Classic Twin Design—a variant of a classic quantitative genetics breeding design—does not require specific knowledge about genotypes or environments and can be readily applied to test for the presence of G×E as a source of phenotypic variation by comparing heritabilities across environments (Barnes et al. 2014; Plomin et al. 2013; Tucker-Drob and Bates 2016). Thus, a reliance on comparing heritabilities across environments as an indication of the presence/absence of G×E has become common practice. The key to understanding why this approach is viable for *detecting* G×E, and also why this approach is not viable for *rejecting* G×E, is the environment-specificity of heritability estimates.

Heritability is the proportion the total variance in a trait that is accounted for by genetic differences among individuals, i.e., the additive genetic variance that contributes to the overall phenotypic variance in a sample (Roff 1997; Plomin et al. 2013). Therefore, the measure of heritability always varies between 0.0 (when none of the phenotypic variation in a sample is due to genetic differences among individuals) and 1.0 (when all of the variation is due to genetic difference among individuals). Heritability can vary across samples for two primary reasons: the amount of genetic variation in

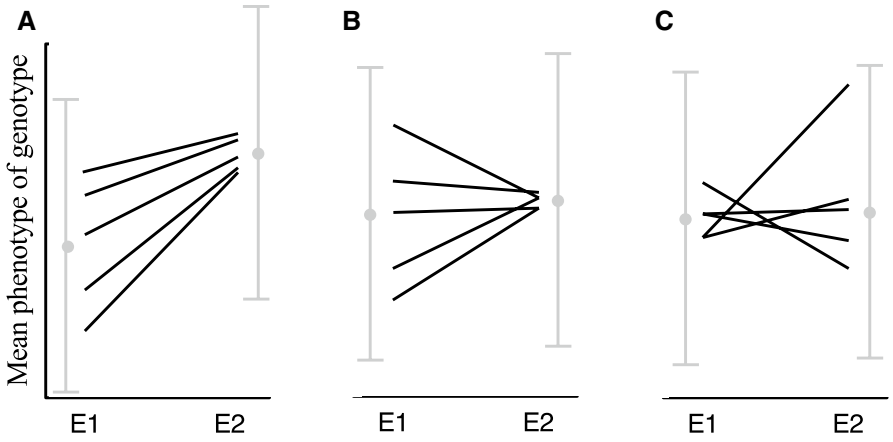


Fig. 1 Three graphs illustrating non-parallel reaction norms generated by unequal heritability across two environments (E1 and E2). Phenotypic variance of the overall sample within each environment is equal for all scenarios, and indicated by the gray mean ± SE bars for each environment

each sample differs, or the amount of environmental variability experienced by individuals within each sample differs. Low heritability—when most of the variation in a trait is explained by environmental factors—can occur when very little overall genetic variation is present among individuals in a sample. As a result, there are few genetic differences among individuals that can generate phenotypic differences. Heritability is also typically low when the environment is highly variable, in which case environmental factors account for most of the phenotypic differences among individuals (Roff 1997). Conversely, heritability has the most potential to be high when: the sample is genetically diverse (and genetic differences strongly impact phenotypic differences), and the environment is highly uniform across individuals (in which case, any phenotypic differences that arise among individuals can be attributed to genetic differences rather than the environment).

If we assume that individuals across two environments from which we sample comprise genetically-similar backgrounds, any difference in heritability across environments will be due to a difference in the magnitude of the influence of the environment on phenotype across those environments. In other words, the environment alone generates more phenotypic diversity across individuals in one environment than in the other. If this is the case, then the average differences among different genotypes will be greater in one environment than in the other. As a simple mathematical inevitability, this necessarily means that genotypes will differ from one another in the magnitude and/or slope of change in phenotype from one environment to the other.

Graphically, the pattern generated is one of non-parallel reaction norms, a key diagnostic of the presence of G × E. Figure 1 illustrates some of the different patterns in which heritability estimates vary across environments. In each graph, we assume similar total phenotypic variance across environments, but a difference in the mean phenotype among genotypes (each represented by a reaction norm). The greater the spread in the mean values of a genotype (or family, which is often the case for classic quantitative genetics breeding designs), the higher the heritability. In Fig. 1a, b, the heritability is greater in environment 1 versus 2, and vice versa for Fig. 1c, with each Fig. 1a–c illustrating a variant of the classic 'fan-shaped' interactions indicative of G × E (Dick 2011).

Human behavioral scholars have relied heavily on the use of variation in the heritability of traits across environments derived from Classic Twin Designs as evidence of G × E (Tucker-Drob and Bates 2016). The usual drawbacks apply to estimating heritability from these types of data—high estimation errors (Roff 1997), and inflated estimates when parents and offspring share a similar environment (Roff 1997)—which is not uncommon in humans, for which transmission of the environment can be quite high (i.e., passive gene–environment correlations; see Plomin et al. 2013; Purcell 2002; Davey-Smith and Hemani 2014). However, of greater concern is a lack of diagnosing G × E when heritabilities do *not* vary across environments.

In Fig. 2—again, assuming overall phenotypic variance remains constant across environments, genetic variance accounts for a similar amount of the variation present in both environments—we observe no difference in heritability across environments (Fig. 2). Without knowing the exact phenotypic values for each genotype, and instead relying solely on comparing heritabilities, we would conclude G × E is absent. However, G × E is clearly present, as indicated by non-parallel reaction norms (Fig. 2c being the most extreme example). Consider a perfect illustration of this type of scenario from outside the human literature. Using isolines, Ingleby et al. (2013) demonstrated G × E in cuticular hydrocarbon expression across two environmental gradients: diet and temperature. They also found very similar heritabilities for the same traits within each of the low and high diet and temperature environments (Ingleby et al. 2013). Without information about the phenotypes of individual isolines, a simple comparison of heritabilities could have led to an improper conclusion that G × E was absent. There are very

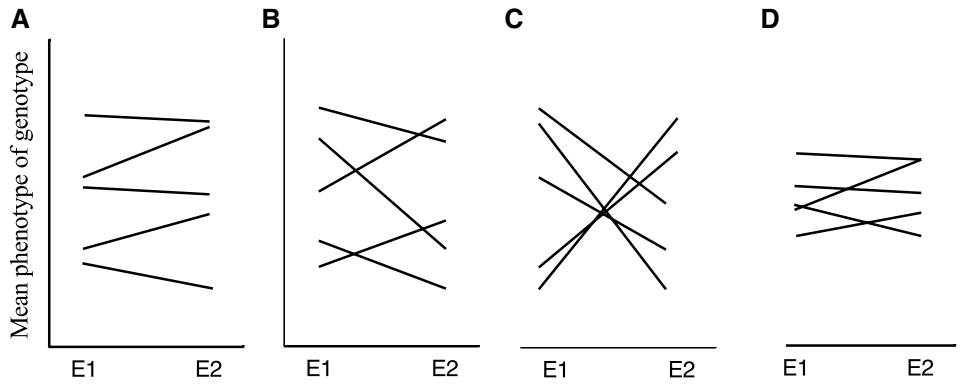


Fig. 2 Four scenarios in which heritability estimates are similar within each environment, but significant G×E is present. For each scenario, we consider a situation in which genotypes are represented in equal frequencies within and among environments, and variance within each genotype is low (with each genotype depicted by a reaction norm that illustrates the average phenotype for that genotype in each environment). **a** High heritability overall, similarly high heritability estimates within each environment, moderate G×E. **b** High heritability overall, similarly high heritability estimates within each environment, high G×E. **c** Low overall heritability, similarly high heritability estimates within each environment, very high G×E. **d** low-moderate heritability overall, similarly low-moderate heritability within each environment, low-moderate G×E

few examples in which this type of data has been generated (potentially related to publication bias against negative results and/or experimental constraints, particularly in humans), so it is impossible to determine how common it is for this pattern to emerge. For this reason, we believe it is worth employing at least some extra caution when interpreting patterns of variation in heritabilities until we gain a better understanding of the frequency of these types of results.

Reconsidering a lack of G×E: a brief example

The implementation of the heritability comparison approach as evidence for G×E in Humans is well-illustrated using the closely scrutinized *Scarr-Rowe hypothesis*, which concerns the moderating effects of socio-economic status on the heritability of general intelligence (g) (Scarr-Salapatek 1971; Tucker-Drob and Bates 2016). Variation in measures of intelligence is partly heritable—moderately so early in life, and highly so later in adulthood (Plomin and Deary 2015). Intelligence also shows an important environmental component of variation; for instance, caloric and nutritional deficits can have an adverse impact on intellectual development (Ritchie 2015). Recognizing the deleterious effect of such nutritional and caloric deprivations, several decades back Scarr-Salapatek (1971) described the possibility that variation for intellectual ability among low socio-economic status individuals might be predominately accounted for by environmental factors. Among higher socio-economic groups—within which there are presumably more uniform environmental conditions across individuals—genetic differences should account for a larger portion of the variance. Recent studies using larger and more representative datasets than the original tests of the hypothesis have produced some supportive evidence that the environment does indeed moderate the heritability of intelligence (Tucker-Drob and Bates 2016).

The *Scarr-Rowe hypothesis* provides a good example for the current discussion, in particular because the body of evidence pertaining to it was recently systematically reviewed. Tucker-Drob and Bates (2016) uncovered an interesting pattern in the findings: G×E has been replicated in samples based in the United States, but G×E was generally absent in non-American samples. As the authors note, one plausible explanation (among others) for this pattern is that the socio-economic environment of Europe and elsewhere varies less (i.e., is more egalitarian) than in the United States, and thus is incapable of explaining as much of the variance in intelligence. Additionally, the possibility remains that the effects emerging in U.S. samples are statistical artifacts, and additional studies may reveal them as such (the sensitivity analyses of Tucker-Drob and Bates (2016), however, suggest the effects in the U.S. are indeed robust). Importantly, it is also possible that G×E might in fact exist in some non-American samples where heritbaility estimates across SES do not vary. However, because the main test utilized is to compare heritabilities across socio-economic environments, and owing to the reasons discussed above, this approach may not reveal some patterns of G×E. Given that testing for variation in heritability across environments cannot be used as a definitive rejection of G×E, we discuss approaches to supplement this primary means of testing for G×E below. Note that our intention in discussing Tucker-Drob and Bates (2016) above is not to critique their conclusions, but rather to reference

a closely scrutinized and widely known G×E from human behavioral genetics.

Further considerations

To reiterate: accepting the presence of G×E using a comparison of heritabilities across environments is *not*, generally speaking, problematic. Moreover, thoughtful approaches have been suggested to effectively parse some of the key factors than can bias toward finding an interaction (such as gene–environment correlation; see Purcell 2002).[2] The emphasis of our discussion revolves around what considerations should emerge when heritabilities do *not* vary across environments. Unfortunately, a solution to this concern is not readily at hand. The advent of modern genotyping techniques in humans has extended approaches to measuring G×E to molecular genetic studies examining specific candidate polymorphisms in combination with measured environments (Caspi et al. 2002). Following this, an explosion of candidate G×E work proliferated across academic journals (Chabris et al. 2015; Davey-Smith and Hemani 2014; Duncan and Keller 2011). However, the constraints of small (non-representative) samples, limited statistical power, as well as common false positive results have resulted in a large-scale shift toward more powerful and robust molecular approaches such as genome wide association studies (GWAS; see Chabris et al. 2015). The best approach moving forward may be, when possible, to perform a follow-up test for a lack of G×E with additional methods including polygenic scores (derived from robust GWAS analyses) (Conley 2016), coupled (perhaps concurrently) with the use of classical twin approaches. Assuming that some amount of agreement emerges across methods, one might reasonably assume the true absence of G×E when heritability estimates fail to differ across environmental exposures.

Concluding thoughts

Studying whether certain environments moderate the effects of genes (and vice versa) on various phenotypes is a central component of the behavioral sciences. As modern genotyping procedures become more efficient, it has become considerably easier for research to take advantage of large genome-wide analyses (GWAS) when testing for G×E via the use polygenic scores (for an overview, see Conley 2016). While GWAS based analyses will doubtless be critical for G×E research, these studies too have limitations (see Carlson et al. 2013)—such as limited coverage across diverse ancestral populations and phenotypic data that may not yet include traits that are available in extant twin data. As a result, researchers will likely continue using classical twin methods to test hypotheses about G×E in human subjects where experimental designs are often difficult or impossible to implement. What we suggest in the current research note is that researchers should be cautious about inferring that a G×E is absent when heritability estimates do not differ significantly across environments, and add additional tests when feasible.

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Compliance with ethical standards

Conflict of interest Kasey D. Fowler-Finn and Brian B. Boutwell declare that they have no conflict of interest.

Statement of human and animal rights This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent For this type of study formal consent is not required.

References

Barnes JC, Wright JP, Boutwell BB, Schwartz JA, Connolly EJ, Nedelec JL, Beaver KM (2014) Demonstrating the validity of twin research in criminology. Criminology 52(4):588–626

Bradshaw AD (1965) Evolutionary significance of phenotypic plasticity in plants. Adv Gener 13:115–155

Carlson CS, Matise TC, North KE, Haiman CA, Fesinmeyer MD, Buyske S, Schumacher FR, Peters U, Franceschini N, Ritchie MD, Duggan DJ, Spencer KL, Dumitrescu L, Eaton CB, Thomas F, Young A, Carty C, Heiss G, Marchand LL, Crawford DC, Hindorff LA, Kooperberg CL, Page Consortium (2013) Generalization and dilution of association results from European GWAS in populations of non-European ancestry: the PAGE study. PLoS Biol 11(9):e1001661

Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, Taylor A, Poulton R (2002) Role of genotype in the cycle of violence in maltreated children. Science 297(5582):851–854

Chabris CF, Lee JJ, Cesarini D, Benjamin DJ, Laibson DI (2015) The fourth law of behavior genetics. Curr Dir Psychol Sci 24(4):304–312

Conley D (2016) Socio-genomic research using genome-wide molecular data. Ann Rev Sociol 42:275–299

[2] It is worth mentioning that a biometric—or twin based—approach to testing for the presence of G×E in human data involves examining either differences in heritability estimates across environments, or differences in (raw) additive genetic variance across environments. For researchers using the approach described by Purcell (2002), it is the second strategy that is being employed.

Davey Smith G, Hemani G (2014) Mendelian randomization: genetic anchors for causal inference in epidemiological studies. Human Mol Genet 23(R1):R89–R98
Dick DM (2011) Gene–environment interaction in psychological traits and disorders. Annu Rev Clin Psychol 7:383–409
Duncan LE, Keller MC (2011) A critical review of the first 10 years of candidate gene-by-environment interaction research in psychiatry. Am J Psychiatry 168(10):1041–1049
Falconer DS, Mackay TFC (1996) Introduction to quantitative genetics, 4th edn. Longman, Harlow
Ghalambor CK, McKay JK, Carroll SP, Reznick DN (2007) Adaptive versus non-adaptive plasticity and the potential for contemporary adaptation in new environments. Funct Ecol 21(3):394–407
Ingleby FC, Hosken DJ, Flowers K, Hawkes MF, Lane SM, Rapkin J, Dworkin I, Hunt J (2013) Genotype-by-environment interactions for cuticular hydrrocarbon expression in Drosophila simulans. J Evolution Biol 26:94–107
Karlsson K, Eroukhmanoff F, Svensson EI (2010) Phenotypic plasticity in response to the social environment: effects of density and sex ratio on mating behaviour following ecotype divergence. PLoS ONE 5(9):e12755
Plomin R, Deary IJ (2015) Genetics and intelligence differences: five special findings. Mol Psychiatry 20(1):98–108
Plomin R, DeFries JC, Loehlin JC (1977) Genotype–environment interaction and correlation in the analysis of human behavior. Psychol Bull 84(2):309
Plomin R, DeFries JC, Knopik VS, Neiderheiser J (2013) Behavioral genetics, 6th edn. Worth Publishers, New York
Polderman TJ, Benyamin B, De Leeuw CA, Sullivan PF, Van Bochoven A, Visscher PM, Posthuma D (2015) Meta-analysis of the heritability of human traits based on fifty years of twin studies. Nat Genet 47(7):702–709
Purcell S (2002) Variance components models for gene–environment interaction in twin analysis. Twin Res Human Genet 5(6):554–571
Ritchie S (2015) Intelligence: all that matters. Hodder & Stoughton, Hachette
Roff DA (1997) Evolutionary quantitative genetics. Chapman and Hall, New York
Scarr-Salapatek S (1971) Race, social class, and IQ. Science 174(4016):1285–1295
Schlichting C, Pigliucci M (1998) Phenotypic evolution: a reaction norm perspective. Sinauer Associates, Sunderland
Sesardic N (1993) Heritability and causality. Philos Sci 60(3):396–418
Tucker-Drob EM, Bates TC (2016) Large cross-national differences in gene × socioeconomic status interaction on intelligence. Psychol Sci 27(2):138–149
Turkheimer E (2000) Three laws of behavior genetics and what they mean. Curr Dir Psychol Sci 9(5):160–164
Via S, Gomulkiewicz R, De Jong G, Scheiner SM, Schlichting CD, Van Tienderen PH (1995) Adaptive phenotypic plasticity: consensus and controversy. Trends Ecol Evol 10:212–217
West-Eberhard MJ (1989) Phenotypic plasticity and the origins of diversity. Annu Rev Ecol Syst 20:249–278
West-Eberhard MJ (2003) Developmental plasticity and evolution. Oxford University Press, New York