Rotten, stale, fresh: three flavors of heritability

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Abstract

The concept of heritability in behavioral genetics is different from the more standard concept used in biology. The former is a statistical measure of the proportion of genetic variance relative to the total phenotypic variance of a trait in a population, the latter refers to the transmission of phenotypic traits across generations via the transmission of an underlying causal substrate (genes). It will be argued that the behavioral-genetic concept is a generally useless quantity, while the standard biological concept is overly narrow and implies a false picture of the significance of genes in development. By suitably expanding standard heritability into a general causal concept based on its role in evolution, we will arrive at a general view of development that recognizes the causal parity of all determinants of phenotypic traits and shows why the behavioral genetic dichotomy of genes vs environment is fundamentally misguided. Some implications for criminology and the social sciences will be addressed.

Keywords: heritability, behavioral genetics, genes, development, evolution, environment

Introduction

The goal of a science of human beings is to understand what makes us into what we are, and to use that understanding to improve ourselves. Behavioral genetics is no exception: its ultimate concerns are to learn about the kinds of causes that shape us and what they imply for the possibility of positive change.

However, the particular way behavioral genetics has traditionally raised and answered those questions has been the subject of continuing controversy. Indeed, the pages of *Criminology* have hosted one of the latest instalments of this debate (Barnes et al. 2014; Burt & Simons 2014; Burt & Simons 2015; Moffitt & Beckely 2015; Wright et al. 2017), and we believe it is fair to say that it did not end in mutual understanding.

The alleged problems addressed by critics are diverse: they range from statistical assumptions made in estimating heritability, the meaning of those estimates, their relationship to the causes of human traits, to the conceptual distinction between nature (genes) and nurture (environment) underlying behavioral genetics' core methodology. On a more abstract level, these issues translate into doubts about whether the methods used by behavioral geneticists to answer their questions are appropriate and whether these questions even make sense in the first place.

In this paper we have two aims: to diagnose the situation, and to offer a way out - indeed, what we consider to be the *right* way out. The diagnosis will involve showing why the concept of heritability in behavioral genetics lacks utility. The way out then

leads from recognizing the relationship of heritability in the behavioral genetic sense to its common meaning in biology to generalizing this biological notion into a principled concept of heritability based on its causal role in evolution. In the process, an adequately contemporary view of heritability and development, united by causal principles, will emerge, and will complete the diagnosis of why the traditional behavioral genetic approach has become obsolete.

Fortunately, to follow these considerations requires neither very specific nor particularly modern knowledge of biology (contrary to some recent critiques of behavioral genetics that stress the importance of molecular biological knowledge such as epigenetics [e.g. Burt & Simons 2015; Charney et al. 2012]). They all follow from basic insights that were available, in principle, to scholars for at least a century. This is also why an essentially complete formulation of the present view was possible for psychologist and philosopher Susan Oyama already in 1985 (Oyama 1985).

This paper will focus exclusively on the classical methods of behavioral genetics - variance partitioning into a genetic and one or two environmental components - and will ignore more modern approaches involving the actual measurement of specific genes or environmental factors (i.e. it focusses on G vs E, and ACE models). Even though some behavioral geneticists may now disavow the classical methods, their results constitute the main body of literature in the field and have, and still are, exerting a massive influence on both scientific as well as popular thinking. The nature-nurture dichotomy still looms large, and heritability estimates are still widely cited as quantitative measures of the influence of genes on human traits.

The origins of nature and nurture

As a first approximation, it may be said that Charles Darwin shattered the complacent view of his predecessors that God was taking care of guaranteeing the similarity of parents and offspring. After Darwin, the fact of parent-offspring resemblance found itself in urgent need of a non-theistic explanation. Theories of biological inheritance sprang up, and the role of environment and upbringing called for clarification. Darwin's cousin Francis Galton was perhaps the first to explicitly bring biology (the innate qualities of a person, which he called "nature") into opposition with the environmental influences that impinge on a person after birth (the acquired qualities he called "nurture"). As is well known, the biologically inherited part became later identified with genes. The view that emerged saw human development as the battleground of two fundamental forces: genes and environment. The question became: which of the two was stronger, and: how could their relative strengths be measured?

Galton had already suggested that identical twins would be useful to address these questions, as their virtual indistinguishability from birth indicated that their biological inheritances (genes) were the same. The degree to which they turned out different should then be a pure measure of the influence of environment.

By the 1930s, the statistical tools necessary to decompose the effects of nature and nurture had become available in the form of the analysis of variance. It allowed to quantify how much of the variance in an organismic trait was "due to" the variance in any number of explanatory variables. For example, the height to which a plant would grow was expected to depend both on its genotype and on the environment, such as the amount of water in the soil. In a simple experimental design, an agriculturist could plant seeds of different genotypes and expose each genotype to different amounts of water. After the plants had grown to their full height, she would measure how much height varied among the different genotypes (averaged across all water conditions), obtaining the amount of genetic variance. Similarly, she would measure the variation in height across the different levels of exposure to water (averaged across all genotypes)

and obtain the environmental variance. If the effects of genotype and water were independent of each other, summing the two variances would yield the total variance in height across all the plants. The ratio of the genetic variance to the total height variance (a number between 0 and 1) could be taken as a measure of the relative strength of the effect of differences in genes on differences in plant height. This number became known as the “heritability” of a trait (Lush 1937). The ratio of environmental variance to height variance was the complement of heritability and was taken to indicate the strength of environmental differences on trait differences. Comparing these two ratios would supply the answer to Galton’s question: which was stronger, nature or nurture?

Variance partitioning in humans

The way to obtain these numbers in humans is, however, importantly different from the example above. The facts that monozygotic (“identical”) twins share all of their genes, and that dizygotic (“fraternal”) twins share half of their genes can be used as a shortcut to estimating genetic and environmental variances (how this is done mathematically will not concern us here). The simplest partitioning includes only a term for genes (G) and one for everything else, called environment (E). The total variance in some phenotypic trait (P) is then simply the sum of the genetic and environmental variance:

$$P = G + E$$

A slightly more complex approach is to split the environmental variance into two: one term for environmental influences that make twins more similar to each other (called “shared environmental effects”) and one for environmental influences that do not make twins more similar (called “non-shared environmental effects”). This is called the ACE model, where A stands for (additive) genetic effects, C for shared and E for non-shared environmental effects.¹

What is crucial to understand is that apart from the trait of interest (e.g. intelligence, antisocial behavior, political preference), *nothing is measured* in a classical behavioral genetic study. The subjects’ genotype is not quantified or identified in absolute terms. All that enters the calculations is the assumption that *all* the genetic variance of monozygotic (MZ) twins contributes to their phenotypic similarity (since they share all of their genes), while only *half* of the genetic variance of dizygotic (DZ) twins contributes to their phenotypic similarity (since they share only half of their genes). Environmental influences are not measured at all. Instead, the term for the environment (E) is simply the remainder after the phenotypic (P) and genetic (G) variances have been calculated ($E = P - G$).

This has an important corollary: since the environment is treated as a leftover category, any environmental differences that produce the same pattern of effects as do genetic differences will be counted as genetic effects, not environmental ones. This is true even if the environmental differences actually *cause* or *causally contribute to* the differences in phenotype.

Perhaps the most striking example of this is the fact that the phenotypic effects of the common egg cell which MZ twins develop from is entirely neglected in twin study designs. MZ twins are created when one single fertilized egg splits in two and the two resulting daughter cells each grow to become a separate individual, while DZ twins stem from two different egg cells having been fertilized by two different sperm cells. Twin

¹We will not deal with the assumption of additivity and the concomitant omission of an interaction term (G x E) from the basic variance partitioning scheme in classical behavioral genetics. We believe the assumption of additivity is a significant flaw that might well render heritability estimates useless on its own. Our intention here is to show the fatal problems with heritability even granting the unimportance of G x E interactions.

studies make use of the fact that in MZ twins, the two daughter cells will have the same genome - copies of the parent cell's genome - but ignore that all the non-genetic rest of the two cells (called the "cytoplasm") also stems from the common parent cell and will therefore also be the same or very similar, whereas the two cells from which DZ twins develop are likely to have less similar cytoplasms. The implication is that in addition to (and in conjunction with) their common DNA, their common cytoplasm is a hot candidate for making MZ twins more similar to each other than DZ twins. To the extent this effect operates in the 2:1 fashion specified by the mathematical formalism of an ACE model, it will be subsumed under the genetic component A, while in reality being a shared environmental effect belonging to component C. The result will be high values of A and low values of C, such as repeatedly found in behavioral genetics studies.

One context in which this matters is the continued debate about the causes of individual or racial IQ differences (see e.g. recent debate in *Vox* [Haider 2017a; Haider 2017b; Turkheimer, Harden & Nisbett 2017a; Turkheimer, Harden & Nisbett 2017b]). If twin-based methods of identifying sources of variation confound genetic with cytoplasmic effects, the importance of genes for IQ will have to be re-evaluated. The same is true in another important context: the debate about the relative importance of parents vs. peers, where behavioral genetic findings have given rise to dramatic claims about the global inefficacy of parenting (Boutwell 2015a; Boutwell 2015b; Boutwell 2017a; Boutwell 2017b; Boutwell & Khan 2016; Harris 1998; Rowe 1994). In fact, what makes cytoplasmic confounding particularly nasty is that there is no known way of disentangling genetic from cytoplasmic effects. The problem affects the entire body of research on twins and heritability. Everything, from antisocial personality to political preferences, that has been claimed to be heritable could potentially be an effect of cytoplasmic similarity, or, more realistically, a joint effect of genetic and cytoplasmic similarity.

The interactionist challenge

A more fundamental charge is sometimes leveled at variance partitioning in the context of developmental causes, one that has been considered by a number of critics to be the final nail in the coffin of behavioral genetics (Burt & Simons 2014; Burt & Simons 2015; Moore 2003). This "interactionist challenge" states that the development of organismic traits is a process of interacting causes that are inextricably entangled, such that their individual causal contributions cannot be separated, let alone quantified. Therefore, attempting to put numbers on the relative importance of genes and environment for a trait is doomed to fail. Genes and environmental causes are *both* necessary (and none by itself sufficient) to build a phenotype, it is said, and if that is the case, it is impossible to quantify their relative contributions. Variance partitioning with regard to the causes of development is fatally flawed.

For example, in their recent challenge to behavioral genetics, Burt and Simons (Burt & Simons 2015) put the case as follows:

"[G]enes and environments are involved in an interpenetrating and interdependent dynamic relationship that renders the attempt to demarcate separate influences— the goal of heritability studies—illogical at both the individual and population levels" (p. 105).

The argument is often illustrated by analogy. One is owed to Richard Lewontin (Lewontin 1974), who has us imagine two men building a brick wall, where one is laying the bricks and the other is applying the mortar. The two men are analogous to two different developmental causes, and the brick wall represents a phenotypic trait. After their work is done, does it make sense to ask which of the two men has contributed more to the finished wall? The answer is no, because both their contributions are necessary, but not by themselves sufficient, to build the wall.

Another popular analogy is the area of a rectangle (analogous to a trait) and its two causes, length and width. Does it make sense to ask which of them, its length or its width, contributes more to the rectangle's area? Obviously not. Both length and width are necessary for the area to even exist, so none can be considered more important. Trying to quantify their relative contributions makes no sense.

This example makes the point of inseparable, "interacting" causes beautifully, but nevertheless, it must have driven at least some behavioral geneticists to exasperation, as it earned the designation "damned rectangle" (Harris 2006). So how do these frustrated scholars object to the argument?

First, there is some common ground: the defender of behavioral genetics agrees with the critic's characterization of how causes operate in development (Barnes et al. 2014; Rutter 2006)². Second, however, she correctly notes that this characterization pertains to how *individual* traits are caused in *an individual organism*, i.e. to the *etiology* of traits. It says nothing about what causes *differences* in traits between *different* individuals. Third, she points out that that's what behavioral geneticists are interested in and what they use variance partitioning for. Fourth, she claims that *variance partitioning is still possible, even in the case of inextricably interacting developmental causes*.

This last claim is correct. In their behavioral genetics textbook, Plomin et al. (2008, p. 85) even use the "damn rectangle" itself to show that, although both its length and width are necessary for any *individual* rectangle's area to exist, a *population* of rectangles may not only show variation in length and width, but these two variations may well contribute unequally to the variation in area. In other words, in a particular population of rectangles, the variation in length might be much greater than that in width, and therefore contribute much more to the variation in surface area. In the context of the causes of differences (variation) in a population, length and width are not inseparable: either one of them can exhibit larger variation than the other one and therefore contribute more to differences in the trait of interest, in this case surface area.

Having established the feasibility of variance partitioning for interacting causes, the crucial question becomes: can it actually tell us anything useful about such causes? Critics of behavioral genetics may think not; they may think that variance partitioning is irrelevant, or even illogical, when it comes to questions of trait etiology (Burt & Simons 2014; Burt & Simons 2015; Moore 2003). However, far from being irrelevant, variance partitioning is actually one of the major scientific tools to identify causes in general, including the causes of organismic traits. It is the core of two of the most widespread statistical methods in all of the natural sciences: ANOVA and linear regression, both employed nearly universally in experimental and observational studies to provide clues about the causes of human and animal traits. Crucially, variance partitioning provides these clues not only when causes are operating independently, but also in the case of interacting developmental causes.

We want to show this now using two simple examples that capture the essence of the interactionist challenge, i.e. the presence of two causes that are both necessary, but none by itself sufficient, to bring about (or *co-constitute*) a phenotype. (The restriction to two causes is merely for ease of exposition with no loss of generality.)

As in the example of the rectangle, imagine two causes, say two different types of molecules A and B in the human body, that interact to produce a third molecule C, which is the outcome, or trait, or phenotype of interest. Both molecules are necessary to produce the outcome, just as both length and width are necessary to form the area of a rectangle. Further, as in the case of the rectangle, assume that the total amount of C

²From Barnes (2014): "What this necessarily means is that for a single individual, his or her genes and environment in interaction contribute to his or her phenotypic score..." (p. 615).

produced is the (mathematical) *product* of the amounts of A and B. To link this to the familiar formalism of linear regression, we can write $y_C = x_A * x_B$, where y_C = outcome (amount of molecule C produced), and x_A/x_B = predictor variables (amount of molecule A and B, respectively). This formula expresses the variance partitioning model used to analyze the data.

The data, remember, embody the interactionist constraints just mentioned: when there is no molecule A and/or no molecule B (i.e. when $x_A = 0$ and/or $x_B = 0$), there will be no molecule C produced ($y_C = 0$). This corresponds to the requirement in the rectangle case that when there is no length and/or no width, there can be no rectangle, let alone any surface area.

Simulating such data and analyzing it using linear regression is straightforward (see Stata code in Appendix). As expected, an analysis including an interaction term ($x_A * x_B$) recovers the multiplicative relationship between the amount of molecule C and that of molecules A and B. The model would therefore correctly indicate molecules A and B as possible causes of molecule C, and would also correctly infer that the amount of C produced is the mathematical product of the amounts of molecule A and B going into the process. It would therefore correctly represent the interactionist constraint that whenever one or both of A and B are missing, there will be no outcome at all (since $y_C = x_A * x_B = 0$ whenever $x_A=0$ or $x_B=0$).

When an additive model ($y_C = x_A + x_B$) is applied to the data, it will still detect the positive dependency of the outcome on both predictors and identify both of them as possible causes.

The upshot is that the interactionist charge that variance partitioning is useless in the case of interacting developmental causes is invalid. Analysis of variance on the population level can, in principle, identify the causes of traits in individuals, even if these causes are "non-separable" in the sense of being jointly necessary, but singly insufficient, to cause a trait. There's nothing illogical or categorically wrong about this endeavor. Employing variance partitioning *per se* is not what makes heritability estimates useless. They are useless for a quite different reason.

Heritability in behavioral genetics: relevance to causes of traits

It is not uncommon for technical terms used in science to have a pre-existing, colloquial meaning. "Heritability" is an example. Its meaning in behavioral genetics is very different from that in common language, including its common meaning in biology. As mentioned at the beginning, behavioral genetic heritability (which we will henceforth call "heritability_{BG}") is a single number estimated from twin studies that denotes the proportion of total variance in an outcome variable that is genetic variance. In other words, it is simply the ratio of genetic to total phenotypic variance. As such, its value lies between 0 (no heritability_{BG}) and 1 (complete heritability_{BG}). In the context of variance partitioning as discussed above, heritability_{BG} is the relative size of the variance component due to genes, where "genes" are one of two (or three) causes being considered (the other being "environment").

Before discussing the implications of the different meanings of "heritability", we need to assess the utility or lack thereof of heritability_{BG} for identifying the developmental causes of traits. We have shown that variance partitioning *per se* can be useful for identifying such causes, but that is not at all the same as showing that *estimating heritability_{BG}*, which is the classical behavioral genetic way of variance partitioning, is useful for this. In fact, the very reason why heritability_{BG} estimates are useless is because they are a particularly unhelpful way of variance partitioning.

The main problem is that the two (or three) sources of variance in behavioral genetic studies are such broad, unspecific and unsystematic categories as to make it virtually impossible to draw any useful causal inferences from them. The ACE model seems to make a potentially useful distinction between two kinds of environmental effects (shared vs. non-shared), but these effects are identified in terms of whether they make twins more similar to each other or not. They *do not* identify any *actual* environmental conditions.³ Even assuming that heritability_{BG} precisely estimates the proportion of genetic variance, all a non-zero value tells us is that genes make a difference to a trait in a population. It leaves us entirely in the dark about which specific genes are involved in the formation of that trait (Sober 2001).

It may be objected that the simple statement that genes contribute to a trait (that a trait “is genetic”) already constitutes significant knowledge. Thus, the behavioral geneticist Michael Rutter (Rutter 2006) writes

“...the overall pattern demands acceptance of the importance of genetic influences. That is, the findings are incompatible with a zero genetic effect”. (p. 60)

Frankly, however, that genes are involved universally in the formation of traits is one of the fundamental facts of developmental biology. There was never a reason to assume that a ubiquitous cellular component whose contributions to most aspects of cell life have been extensively documented would somehow magically fail to affect some classes of traits. Another behavioral geneticist, Eric Turkheimer, calls this a “pointless null hypothesis” (Turkheimer 2011).

Looking at variance partitioning in any field other than behavioral genetics will show how differently it is used. For example, a study of obesity may measure intake of fat, carbohydrates and proteins and find a statistical effect of carbs. This result gives a useful pointer to possible causes, and a follow-up study might then look at different types of sugary foods to narrow down the causes of the effect. Studies all over the natural sciences use experimental manipulations or observational variables specific enough to give potentially useful hints for further investigation. In contrast, knowing that some variance in a trait is genetic, even knowing the amount of that variance, provides no useful knowledge and no idea how to proceed further.

As far as the identification of causes is concerned, heritability_{BG} tells us nothing useful. Nevertheless, the brute fact of genetic involvement as indicated by a positive heritability_{BG} estimate might be seen to carry significance beyond this: it might tell us something about the malleability of a trait.

³The shared environmental effect C is often understood as those influences that are shared among twins (notably the family environment, see e.g. Rowe [1994], p. 53), whereas the non-shared environmental effect E is taken to be those influences and experiences that are unique to each twin (e.g. peers). Behavioral genetics studies have repeatedly found that C is small, while E is quite substantial. This has been taken to show that the whole family environment - including everything that parents do - has very little effect on children’s development, while it is those experiences outside the home, e.g. peer groups, that mostly shape their phenotypes (Rowe 1994; Harris 1998). If this were true, it would have dramatic implications for parents, schools and any kind of intervention targeted at improving children’s abilities and behavior.

However, what these variance components really stand for are environmental influences that make twins *more similar* (C) to each other or not (E). Better names for these effects would be something like “similar-making” and “not similar-making”. Although it may seem self-evident to some that common experiences, including much of what happens in the home, will have similar effects, making the twins more alike, and that unique experiences (such as with peers) will make them different, this is not at all a given (Rutter 2012). While plausible-sounding, it is pure speculation, and as such, nothing that could bear the weight of far-reaching conclusions about the effectiveness of parenting. Finally, it should be obvious on reflection that without measuring anything about the *actual* environments encountered by the study subjects, it is hard to conclude anything about their effects. A mere mathematical formalism cannot achieve this.

Heritability in behavioral genetics: relevance to malleability

According to a still common view, a genetic involvement means that a trait, being inherited and therefore “inborn”, is more hard-wired, more fixed, and less amenable to change than a trait that is environmentally caused. Therefore, the mere fact of genetic involvement is taken to be much more consequential than, for example, the fact of the involvement of diet, in causing a trait.

No such thing is true, however. Whether genes are or are not involved in the causation of a trait says nothing at all about how difficult to change it is. In other words, genetically influenced traits are not *a priori* more difficult to change. It is always an *empirical* question how malleable a trait is, and the answer may not only be different for different traits, but even for different individuals carrying the same trait.

Perhaps, if a genetically influenced trait could only be altered by changing the relevant genes, an argument could be made that due to the relative inaccessibility and the structural and functional complexity of the genome, changing such a trait would always be hard compared to changing other traits whose etiology does not involve genes. But changing a genetically influenced trait does not necessarily require changing the relevant genes. Traits are always multi-causal, and each of the involved causes, whether genetic or environmental, is, in principle, an access point for causal manipulation.

A famous example is that of the disease phenylketonuria (PKU). It involves a faulty gene that fails to produce an enzyme required to break down a certain amino acid. If left untreated, the amino acid accumulates in the body, leading to early mental retardation. There is a treatment that essentially cures the disease, but it has nothing to do with genes. It consists of a special diet that is free of the offending amino acid, so that a toxic build-up is prevented. A causally simple environmental intervention cures a genetic disease. Many other such examples could be found, all illustrating the same underlying principle: if multiple causes co-determine an outcome, manipulating any of them can potentially change the outcome. This is true regardless of whether any of the involved causes is genetic or not.

It is important to realize that heritability_{BG} estimates are not informative about contexts such as this, in which a treatment for a disease trait, or an intervention to improve a beneficial trait (e.g. intelligence) is sought. In particular, the question of whether there could be an environmental intervention to change the trait in a desired direction is not addressed by heritability_{BG}, since such an as yet unknown intervention cannot be part of the environment of the population from which the heritability_{BG} estimate was derived. For example, before a dietary treatment became available, the heritability_{BG} of PKU was high, as environmental variation in the disease phenotype was low. This meant nothing, however, for the prospects of finding a successful environmental intervention.

Heritability_{BG} is useless as an indicator of a trait’s malleability. Genetic involvement *per se* does not determine malleability, and even the fact that a trait such as PKU is “genetic”, i.e. originates with a gene defect, does not allow any *a priori* conclusions about how difficult to change it is, and where best to intervene to change it.

A word on genetic determinism

We have little doubt that many scientists, including behavioral geneticists, would agree with these considerations, and that most would reject any explicit statement of genetic determinism. However, one would learn very little about the true extent of gene deterministic beliefs from such explicit denials. The issue is more subtle, because notions of genetic determinism mainly exist in unreflected language use and

assumptions, and in certain persistent research practices. We will give examples of each of these.

Regarding research practices, the strong dominance of genetic research in biomedicine over the past decades (e.g. see Human Genome Project [HGP], the gene therapy boom, and today's personalized/precision medicine) is testimony to the fact that knowledge of genes is seen as informative about human traits in ways that knowledge of other developmental causes is not (Gamma 2013; Gamma 2017). The "decoding" of the genome, for example, was hailed as the gateway to a unique source of knowledge about the human body that would eventually allow us to eradicate the root causes of all disease (e.g. Bill Clinton, cited in Collins 2010).

Regarding the use of language, genes are still widely described in terms that suggest they have a privileged role in development. For example, it is often granted to genes that they can constitute a disposition or propensity for a disease or behavior. However, the same is almost never said for non-genetic causes of development. There is no hormone-based disposition for substance abuse or a dietary propensity for anti-social behavior. A similar way of privileging genes is to say that they determine a pre-existing, inborn potential, e.g. regarding intelligence, whose boundaries cannot be overcome by environmental influences (Lewontin 2000, p. 26).

The most striking asymmetry in language use with regard to the causes of development, however, is the so-called *information metaphor*, i.e. the notion that genes contain instructions for building a body, while other developmental factors are just passively following the genetic command (Sterelny & Griffiths 1999, p. 100)⁴. The information metaphor is easily the most important vehicle for genetic determinism among scientists and is virtually ubiquitous.⁵ At the same time, the only place we know instructions to literally exist and have causal effects is in the realm of human minds and human interactions, and it is hard to take seriously the notion that a molecule could *literally* give instructions to other molecules. Accordingly, scholars examining this idea (mostly philosophers of science) have almost uniformly come to reject it (Godfrey-Smith 1999; Godfrey-Smith 2008; Griffiths 2001; Griffiths & Gray 1994; Johnston 1987; Keller 1995; Neumann-Held 2006; Nijhout 1990; Moss 2004; Oyama 1985; Sarkar 1996; Šustar 2007). It follows that there are little grounds for believing that genes control or direct or code for traits. Their role in development is not categorically different from that of other trait causes (Griffiths & Gray 2001; Griffiths & Knight 1998).

The information metaphor often goes hand in hand with other unreflected assumptions, e.g. the idea that the genetic influence on traits is somehow prior to any kind of non-genetic influence (Smith 1992). Also, it will later be seen (see section "Heritability, extended") that the almost universal equation of "heritable" with "genetic" also implies a form of genetic determinism.

Given our characterization of the situation, it should only be expected that an explicit denial of genetic determinism will often co-exist, in the same person, with language and/or behavior that implies some variety of genetic determinism. In our experience, this occurs frequently and one of us has elsewhere examined Richard Dawkins' views as

⁴Sterelny & Griffiths (1999) characterize this view as follows: "The genes are the only things that contain information; they are the blueprint or program for building the organism... The other material causes of development are only building blocks, which are assembled according to the genetic plan."

⁵Some examples: the behavioral geneticist Brian Boutwell writes about "the reality that genes provide the information for building and running biological machinery like the human body" (Boutwell & Barnes 2017). Famous psychologist Steven Pinker speaks of "deep and elegant principles, like the insight that life depends on a molecule that carries information, directs metabolism, and replicates itself" (Pinker 2013) and renowned evolutionary biologist and blogger Jerry Coyne believes that "[genes] are the bearers of heredity, the switches of development, and the coders of bodies." (Coyne 2016)

a particularly striking example (Gamma 2013).⁶ We mention these examples to counter a frequent objection to charges of genetic determinism, namely that no serious scientist endorses this position. But explicit denials can perfectly co-exist with implicit forms of genetic determinism and sometimes seem little more than superficial lip service to be discharged of before one can continue to focus on the real business of genes.

Much more could be said about this, but to conclude this section, suffice it to say that a full rejection of any form of genetic determinism would certainly entail accepting the statement that to know that genes are involved in causing a trait is exactly as little or as much informative as to know that proteins are involved in causing a trait. If the latter is considered to be a near-truism and virtually non-informative (as we suspect it would be by many), the very same is true for the equivalent statement about genes. If readers feel reluctant to accept this, they might have lingering traces of genetic determinism in their system.

Heritability in behavioral genetics: relevance to biological heritability

In biology, heritability is most commonly understood as the recurrence of a trait across generations due to genetic inheritance. We will henceforth call this notion “heritability_{Bio}”. To say that a trait is heritable_{Bio} means that it exists in parents and recurs in offspring because the latter receive from their parents the genes that give rise to the trait. Heritability_{Bio} therefore incorporates not only the aspect of “passing on” something to someone, but also a particular causal mechanism for doing so. That mechanism is genetic inheritance, because in the standard view of biology, genes are seen as the only causal substrate for trait inheritance. In “The Selfish Gene”, Dawkins puts it bluntly by stating that “genetic factors replicate themselves, blemishes and all, but non-genetic factors do not” (Dawkins 1976, pp. 98/99). Later in this article, we will try to show that divorcing heritability from any particular causal mechanism leads to a theoretically motivated, fundamental, and integrative concept that is immensely useful in capturing the wide gamut of inheritance phenomena that are known to exist beyond genetic reproduction.

First, however, the question: what is the relationship between heritability_{BG} and heritability_{Bio}? Why do behavioral geneticists call a statistical measure of the proportion of genetic variance in a trait “heritability”?

The answer is that, given the view that heritability_{Bio} is based exclusively on the transmission of genes, measuring the influence of genetic differences on trait differences in a population would simultaneously capture the extent to which such trait differences are biologically inherited. While superficially plausible, it is clear that the process by which heritability_{BG} estimates are arrived at does not even cast so much as a glimpse at the process of genetic transmission from parents to offspring. It is not surprising, then, that the behavioral genetic measure of heritability sometimes diverges sharply from the biological concept.

For example, consider traits such as bipedality (having two legs) or pentadactyly (having five fingers) in humans. These are paradigmatic features of the “design” of *Homo sapiens* and universally considered to be highly heritable. However, almost all variation in these traits is environmental, stemming from accidents, and therefore heritability_{BG} is basically zero.

⁶A similar disconnect between explicit and non-explicit thinking about the effects of genes among biologists was proposed by Stotz and Griffiths (2004).

The inverse case is possible, too: consider the trait of wearing skirts, which, as most would agree, is primarily culturally determined and not heritable_{Bio}. Wearing skirts, however, is almost perfectly correlated with sex, which itself is usually considered genetically determined. Giving precedence to genetic over environmental variation, even when the two correlate, the heritability_{BG} of wearing skirts would turn out to be very high. Analogous considerations would show that racism and sexism are also highly heritable, even if in reality they were entirely sociocultural phenomena.

How do these discrepancies occur? In the case of bipedality, what goes wrong is that there is genetic causation, but no genetic variation. And without genetic variation, heritability_{BG} is zero. In the other cases, there is environmental causation and therefore correlation, but it is congruent with a genetic correlation, so the effect is ascribed to genes and heritability_{BG} becomes high.

These examples illustrate that behavioral genetic heritability does not track biological heritability as commonly understood. This is yet another way in which the heritability_{BG} concept proves to be useless, and, what's more, confusing.

In conclusion, then, heritability_{BG} is useless for determining the specific causes of a trait, for indicating the malleability of a trait, and for tracking heritability as commonly understood in biology.

Truth is not decided by authority, but it should at least be known that authorities in (the philosophy of) biology share this verdict (Bateson 2001; Feldman & Lewontin 1975; Keller 2010; Lewontin 2006; Sober 2001), even if possibly being more polite about it than we are here. We specifically mention these scholars because it is philosophers of biology who have done most of the work in developing the conceptual basis of biology, particularly addressing issues such as the role of genes in evolution and development (see for example the collection of essays in Neumann-Held & Rehmann-Sutter 2006). But also prominent behavioral geneticists acknowledge that estimating heritability_{BG} in itself is not very useful. According to Michael Rutter (Rutter 2006), "It is not that, on its own, it matters very much whether the heritability is 20 percent or 80 percent" (p. 89), while Eric Turkheimer finds variance partitioning as practised by behavioral genetics to be without "scientific content" entirely (Turkheimer 2011, p. 598).

Towards a truer view: extending standard biological heritability

The story might end here. It might be thought that, all right, the behavioral genetic way of variance partitioning has been unhelpfully unspecific, and its concept of heritability unfortunately confusing. Take that away, and everything else is still fine. But it turns out that the common biological concept of heritability is seriously wanting itself, and that by fixing it, we can arrive at an elegant, unified view of evolution based on a principled definition of heritability. This definition will abstract from contingent, particular mechanisms of inheritance and instead focus on the *causal role* of heritability in Darwinian evolution. It will be called heritability_X (the "X" stands for "extended").

Why was Darwin able to formulate his theory of evolution by natural selection without knowing anything about the actual mechanisms of biological inheritance? Because the actual mechanisms do not matter for the theory, only the fact of heritability itself, understood as the fact that parental traits tend to recur in the offspring generation or, simply put, that offspring tend to resemble their parents.

This basic observation is one part of the "holy trinity" of Darwinism, the three criteria that allow for evolution by natural selection: phenotypic variation, differential fitness, and heritability (Sterelny & Griffiths 1999, p. 32). Together they specify a causal recipe for evolution by natural selection. Importantly, this recipe is abstract enough to leave its

physical implementation open. It is defined, rather, by the *causal roles* it specifies. As long as these roles are implemented in any way in any system, the system will exhibit Darwinian evolution.

In the world of organisms, the recipe specifies that there must be variation in a trait among a population of individuals. Some of the variants cause its carriers to have higher fitness, i.e. to produce more offspring than carriers of other variants. Finally, the trait in question must be heritable, i.e. offspring of carriers of the “successful” trait must be more likely to have the trait themselves. As a result, the trait increases in frequency in the offspring generation.

This three-step process is one iteration of the cycle of Darwinian evolution, and it will repeat as long as there’s a fitness differential and no countervailing forces. The successful trait will continue to increase in the population across many generations, which is another way of saying that it evolves by natural selection.

The causal role of heritability in Darwinian evolution

Nothing in this account depends on the particularities of the mechanism by which offspring gets to be similar to parents. Rather, the heritability requirement is defined by a particular causal role. This role consists of causing parental traits to recur in offspring. This can happen in potentially many ways, but, following Oyama (1985), we suggest that these can be subsumed under three broad categories, using one simple background assumption.

The assumption is that, since traits are multiply caused, the presence of the same or similar causes (in different organisms of the same population) will tend to result in the same or similar traits. Put somewhat sloppily, the idea is simply “same input - same output”. This is certainly plausible.

A parental trait will therefore recur in offspring to the extent that the same causes that gave rise to the trait in parents are also present in offspring. For a trait to be inherited, therefore, the same or very similar “trait-building” causes need to be present in both parents and offspring. Oyama (1985) identifies three broad ways in which this can happen: first, the trait-building causes simply persist across generations; second, the trait causes are physically copied and transported from parents to offspring; and, third, the trait causes are reconstructed in each generation anew. These categories should cover most, if not all, of the possibilities of trait inheritance.

To make the idea more concrete, we’ll give examples of each of the three categories. *Persistent* trait causes include fixed features of the environment such as sunlight, oxygen, gravity, and humidity. These are always there, and will exert their effect in every generation. *Copied* trait causes include the familiar case of DNA, which is physically replicated before passed on to offspring. DNA methylation patterns, if reliably copied across generations, would be another example. *Reconstructed* trait causes are probably the major category. They include all trait causes that recur in every generation without having been directly physically copied or having been always there. Examples are diverse and include the womb (present in every generation, but reconstructed from “parts” in every generation anew), nutrition, social interaction with parents and conspecifics, behavioral habits, cultural norms and practices, and language.

As these examples show, heritability in the evolutionary relevant sense is more accurately described as “repeated presence across generations”, because this removes the narrow focus on something physical having to be passed on from parents to offspring. Here, however, we will stick to the shorter term “heritability_X”.

Non-genetic inheritance in nature

At this point, it is natural to ask: what is the empirical evidence for non-genetic inheritance in humans? The answer is that it is all around. It is less a case of having to discover novel non-genetic inheritance mechanisms (although that is part of it, as for example in epigenetic inheritance), but one of many basic and well-known observations falling into place as soon as they are put into a new conceptual framework. That oxygen and sunlight are reliably present in every generation of our species is a simple fact. That every child starts out from a cell containing its mother's cytoplasm and begins growing up in the rich environment of a womb is another one. That diets can stay stable for long periods of time in local populations is also well-known. That offspring in each generation experiences very similar maternal behavior, and will soak up local cultural norms and practices and learn the local language is no secret, either.

Vastly more than just DNA is stable across generations (Griffiths & Gray 2001; Griffiths & Gray 1994; Jablonka & Lamb 2005; Jablonka & Lamb 2007; Jablonka & Raz 2009; Oyama 1985). This should be uncontroversial. All that is needed then is to recognize that these other trans-generationally stable trait causes meet the heritability_x requirement for Darwinian evolution just as genes do. They all fulfill the causal role of being reliably present in repeated generations, poised to contribute to the building of the phenotype. They are thus heritable in the one sense that matters, the sense that enables evolution by natural selection.

In human beings, culture will be the major source of non-genetic inheritance (Acerbi & Mesoudi 2015; Richerson & Boyd 2008). Factors such as how we treat our children, how we school them, what norms and values we teach or spread, what behaviors and habits our lifestyle encourages or discourages, how and where we house, how and where we work, what societies we build, what political and legal systems we have, what technologies we use, what we eat, whether and how we exercise, all this and more is part of a large package of culture children in every generation are shaped by. These packages will be different across parts of the globe, and different parts of the package will have different "time constants", i.e. will change faster in time than others. But all will to some extent contribute to the stability or similarity of the phenotypes related across generations by descent. It is plausible to assume that culture will be more strongly involved in shaping mental as opposed to physical traits, but then behavioral genetics has always had a primary interest in mental traits such as cognition, affect, personality and psychiatric disorders.

Non-genetic inheritance is also found in animals with little or no culture. First, persistent trait causes belonging to the physical, geological or biological environment affect most animals just as they do humans. So do, second, reconstructed trait causes like wombs and eggs, a population- or species-specific diet, social interaction with parents and conspecifics, specific forms of housing etc. Third, there are other, more particular non-genetic inheritance systems in animals that are described in detail in the work of geneticists Jablonka & Lamb (2005; 2007). The subject matter, particularly in humans, far transcends the current fashionability of "epigenetics" (here narrowly understood as chemical and structural modifications of DNA).

Heritability, extended

An extended view of heritability also resolves a contradiction inherent in popular notions that deny genetic determinism, whilst also finding nothing wrong with the standard biological concept of heritability based exclusively on genetic inheritance. The problem - not often recognized - becomes evident in a phrase we used earlier to characterize heritability_{Bio}: "To say that a trait is heritable means that it exists in parents

and recurs in offspring because the latter receive from their parents the genes that give rise to the trait.”

This mechanism of inheritance can only work as described if having the right genes more often than not causes the trait in question, regardless of the configuration of other, non-genetic trait causes. In other words, it can only work with a liberal measure of genetic determinism. When genetic determinism is rejected, however, a “paradox” pops up: how could trans-generationally similar genes “give rise to” trans-generationally similar traits *without other trait causes also being trans-generationally similar*?

If genes don’t have special deterministic or instructive powers, how could the mere presence of certain genes guarantee the formation of a certain trait, irrespective of the status of other, non-genetic co-determinants of the trait?

The answer is it could not. Development is paradigmatically a matter of multiple causes interacting to create a phenotype. If only one type of cause (genes) were stable across generations, and all others were free to vary arbitrarily, there would be no way of making sure that offspring develop the same traits as their parents.⁷ It is here that the interactionist challenge really bites!

As soon as this is accepted, the pieces fall into place: trait heritability in the evolutionarily relevant sense can only be achieved if *all* or *most* or *many* trait causes are stable across generations - certainly not if only *one* kind of cause is stable. The rest is re-description: we call *all* trans-generationally stable trait causes *inherited*, because that’s what they are in the sense that is relevant to Darwinian evolution (Griffiths 2001; Griffiths & Gray 1994; Griffiths & Gray 1997; Griffiths & Gray 2001)⁸. And we call *all* trans-generationally stable traits *heritable*, because that’s what they are in the sense that is relevant to Darwinian evolution. What we end up with is a view in which many or most traits are heritable, and many or most causes of traits are inherited, at least to some extent. In her book-length treatment of the nature-nurture question, philosopher of science Evelyn Fox Keller (2010) summarizes this new view of heritability:

“...let us acknowledge that... almost all human traits are transmitted from one generation to the next ... [and] let us also accept the fact that the mechanisms of transmission are very varied. They may be genetic, epigenetic, cultural, or even linguistic.” (p. 80)

Heritability, development, evolution: a new view

Both the behavioral genetic and the standard biological concept of heritability are obsolete because they are based on the false view that only genes are inherited, or at least that it is only genetic inheritance that matters. That view is outdated and, as far as the relevant facts are concerned, no more than highschool biology is needed to reject it. A deeper understanding of heritability and development can only follow from an extended notion of heritability which has a principled causal basis in evolutionary theory.

To summarize, the present paper makes the following points about heritability:

⁷It follows that to equate “heritable” or “inherited” with “genetic”, which is almost universally done by scientists and lay persons alike, is to tacitly accept some form of genetic determinism, because it is granted to genes alone to ensure the formation of the traits that are inherited.

⁸“...we should define “inheritance” so that something is inherited just if it passes from generation to generation in such a way that evolution can act on its variant forms. Hence, every element of the developmental matrix which is reliably replicated in each generation and which plays a role in the production of the evolved life cycle of the organism counts as something which is inherited.” (Griffiths 2001)

Heritability_{BG} is rotten: first, it is confusing and misleading, because it does not track common notions of biological heritability, but mistakenly equates “heritable” with “genetic”. Second, it is not useful in its own right: it tells us nothing new (that both genes and environment are involved nearly universally in the formation of traits is a truism), it does not permit the identification of specific causes of traits, instead sticking with unhelpfully broad categories, and it does not allow any conclusions about whether or which interventions to improve undesirable traits will be successful. The perceived significance of knowing whether or not a trait is “genetic” is an illusion based on the lingering influence of genetic determinism.

Heritability_{Bio} is stale: the commonsense meaning of biological heritability improves on heritability_{BG} in actually being about the transmission of traits from one generation to the next. However, it is stuck with the old and false idea that trait heritability is based exclusively on genetic inheritance, which is untenable once any deterministic powers of genes (such as the ability to give instructions) are rejected.

Heritability_X is fresh: the way out of this jungle of antiquated ideas is to go back to square one and start from first principles, by remembering why the notion of heritability has become important in biology in the first place. It is because of the causal role it plays in evolution by natural selection, a role that is not supported only by genetic inheritance, but by any mechanism that ensures that many or most developmental causes of a heritable trait are reliably present in generation after generation. Hence, a principled notion of heritability - heritability_X - is based on the evolutionarily relevant requirement that traits reliably recur in every generation. It leaves possible implementing mechanisms open, in accordance with the fact that different such mechanisms are not only conceivable, but actually operational. In human beings, cultural inheritance is the most varied and important such mechanism, but others are possible, and many have in fact been described in various species of animal (Jablonka & Lamb 2005).

The view of evolution that emerges is one in which an interacting network of internal and external trait causes envelops and permeates the developing organism, spanning the generations and leading to the reliable recurrence - the heritability - of traits, or to trait variation, depending on the stability or variability of the involved network components.⁹

This connects with a view of development in which traits are always the product of multiple, interacting causes, none of which is more determinative of the outcome than any other. Heritable traits do not remain stable across generations because there is one special trait cause that instructs the developmental process, but because a sufficient number of diverse trait causes are inherited so that the process results in a stable phenotype. The principle “same input - same output” applies.

This picture makes sense of fundamental observations about the world, in particular the human world: that a vast array of conditions and stimuli that impinge on us are present in every generation, from the obvious such as sunlight and oxygen to the subtle such as cultural norms of behavior. Although it is certainly impressive that the DNA molecule is faithfully copied from parents to offspring, it is not only genes that provide stable inputs to the developmental process. Faithful replication is required neither for trait heritability nor for biological or cultural evolution (Henrich et al. 2008; Richerson & Boyd 2008, p. 83 ff). Rather, there are gradients of similarity of inherited trait causes that lead to gradients of similarity in inherited traits. All these processes, whether genetic or non-genetic, contribute to the cumulative evolution of human phenotypes.

⁹Some traits will then be selected for because they confer a reproductive advantage to their carriers, but in highly cultured species such as ours, some traits might gain ground due not to the reproductive proliferation of its bearers, but because cultural causes of the trait become prevalent for independent reasons, making the trait more frequent. Either way, phenotypes in the population will change in response, constituting evolution.

Implications for criminology and beyond

Farewell to heritability_{BG}

Churning out heritability estimates has become an industry in behavioral genetics, including criminology (Mason & Frick 1994; Miles & Carey 1997; Rhee & Waldman 2002; Walters 1992). The most obvious implication of the present view is that the production of such estimates should be discontinued. Along with that, their consumption and use by other disciplines should be abandoned as well. Heritability estimates are literally useless: they constitute a waste of time, money, and intellectual resources, by tying research to a false dichotomous view of heritability and development.

Genetic confounding

A second implication, closer to the heart of criminology, is that whenever the term “genetic” is used in reference to genes as the (supposedly) only substrate of biological inheritance (as opposed to genes as particular molecules playing particular causal roles in development), it should be replaced by terms such as “heritable” or “hereditary”. This follows from the fact that heritability_x has a much broader basis than just genes.

What this means is that it can no longer just be *assumed* that the similarity of, e.g., MZ twins is due only to shared genes. It might be due to genes *and* something else, or to something else *entirely*. “Heritable” is not congruent with “genetic”, it is much broader. In the absence of further evidence, it must be considered an open question what inheritance mechanism might account for some “heritable effect” found in a behavioral genetics study.

Where this becomes relevant in criminology, and the social sciences in general, is the issue of “genetic confounding”. The idea here is that social/environmental explanations of traits, typically in a within-family context, are subject to alternative explanations due to genetically inherited similarities. Here is an example, taken from Moffitt & Beckely (2015): suppose you want to test the social/environmental hypothesis that a mother’s hostile parenting causes antisocial, aggressive behavior in her child. If you look at mother-child-dyads from different families and find that children of more hostile mothers display more aggressive behavior, you might be tempted to conclude in favor of your hypothesis. However, this would be premature, because you could just be measuring the fact that children of hostile mothers might have inherited from them a genetic propensity to be more hostile and aggressive themselves. There would then be no causal arrow pointing from hostile parenting to a child’s aggressive behavior. Instead, there would be a common genetic cause for the hostile behavior in both the mother and the child. The presence of such a common genetic cause would be a confound for any social/environmental explanation, and this is what “genetic confounding” refers to.

Twin designs, in particular “discordant twin designs”, have been proposed as a solution to such confounding (Moffitt & Beckely 2015). The idea is to find MZ twins who are discordant for the trait of interest. Since they still share their genes and the resulting phenotypic similarity, their *dissimilarity* in the target trait cannot be explained by their genetic *similarity*. This opens the door for alternative explanations in terms of *non-heritable_{Bio}* social and environmental causes, such as the hostile parenting from the above example (which, by the way, is based on research actually conducted by Caspi et al 2004).

This approach is a good one, a useful and legitimate use of twins for research, but it is often misdescribed as controlling only for genetically caused similarity. Since trans-

generational similarity in humans is usually caused by many more kinds of inherited traits and trait causes than just genes, this study design actually controls for *any* inherited similarities that potentially affect the target trait. In short, it controls for “*hereditary_x*” confounding, which is broader than just genetic confounding.¹⁰

The misdescription as mere genetic confounding implies that genes alone would constitute a sufficient explanation of a target trait. Behavioral genetic accounts often seem to suggest that inherited genes by themselves can constitute sufficient causal routes to traits, which are alternative to and independent of any social/psychological/environmental route. That, for example, genes constitute an alternative explanation of why a child develops antisocial behavior - alternative to an explanation in terms of parenting style. Often, a reference to some “genetic propensity” (for, e.g., aggressive behavior, Moffitt & Beckely 2015) is considered a satisfactory specification of such an alternative.

However, in their appropriately downgraded role, genes alone cannot constitute full-blown causal routes to traits, as they are but one component of a diverse causal network, all of whose parts make necessary contributions to any developmental outcome. Assuming that there could be a purely genetic account of a trait is a kind of category mistake.¹¹

In fact, there is a striking explanatory asymmetry in the popular dichotomy between genes and the social/environmental: while genes cannot constitute sufficient causal explanations for traits, social processes, being on a much higher organizational level than genes that includes the outcomes of many different types of trait cause, *can* constitute full-blown explanations, or at least can come much closer to being complete explanations. This is not favoring social causation due to some ideologically prior commitment, but simply the result of the choice of causal-explanatory opponents made in behavior genetics and other social sciences, a choice that is so extremely lopsided as to pit a single molecule against the multitude and diversity of causal processes that together constitute the “social” or “environmental”.

As far as genes are relevant to the explanation of a trait such as antisocial behavior, they need to be woven into a fuller account that appeals to the numerous different types of relevant causes. The wildly popular tendency to oppose genes and the social to each other is a dead end, because human development is the paradigmatic place in which causes interact and are integrated in the formation of a phenotype.

Genetic propensities

The appeal to genetic “propensities” and the like is of no help in this situation. Once it is accepted that genes play no special role in development, it follows that if genes can constitute propensities, so, in principle, can other causes of development. For example, it might legitimately be said that a mutation in the BRCA1 gene constitutes a genetic propensity for breast cancer in women, given that affected women have an elevated risk for such cancers. Then, however, any situation in which a non-genetic cause increases the chance of developing a certain trait must also be considered as one in which the non-genetic cause constitutes a propensity. For example, a compromised immune system (due, e.g. to AIDS) constitutes an immunological propensity for infectious diseases, smoking constitutes a behavioral propensity for lung cancer, and growing up

¹⁰The statistician Carlin and colleagues (2005) put this in simple terms: “Twins are of special interest because they provide naturally matched pairs where the confounding effects of a large number of potentially causal factors (such as maternal nutrition or gestation length) may be removed by comparisons between twins who share them.” (p. 1089)

¹¹The criminologist Brian Boutwell asks “Is crime genetic?” and answers his own question with “Scientists will never know because they are afraid to ask” (Boutwell 2017a). But crime cannot just be “genetic”, because genes are simply not the type of entities that could ever constitute sufficient explanations for behavioral or cognitive traits. There’s no need to be afraid of anything here.

in poor neighborhoods constitutes a socio-economic propensity for criminal behavior (assuming that all these causal effects are established).

Alternatively, one might altogether discard talk of propensities, but then the ban must apply to any and all kinds of trait cause. What's no longer possible is to privilege genes as the unique bearers of propensities.

Our vote would go to the abandonment of propensities, as they tend to be used as explanatory shortcuts that impede a deeper understanding. To illustrate, let's look at another example from Moffit & Beckley (2015) that they describe in the following way: "Imagine testing whether prison causes crime by comparing twin siblings who go to prison against their co-twins who do not. Research attempting to test the iatrogenic influence of incarceration on postrelease criminal recidivism is bedeviled by the possibility that longer prison sentences might selectively be given to individuals carrying greater criminal propensity".

According to Moffit & Beckley, the discordant twin design is supposed to avoid the bedevilment: being monozygotic, the twin siblings are assumed to either both *have* or *not* have the genetic propensity. Therefore, if we now find that post-release, the imprisoned co-twin commits more crimes than the non-imprisoned one, this difference may indeed be caused by imprisonment, since we can rule out a pre-existing criminal propensity as the explanation. That's how the thinking goes.

But there are problems with this argument. A propensity only matters if it causes something in the world. In a study of twin pairs discordant for imprisonment, the imprisoned co-twins, if they have a propensity for crime, must, *on average*, have committed more crimes than someone without that propensity. If the non-imprisoned co-twins share this propensity, they, too, would be expected to have committed an above-average number of crimes.

The first problem is that if it is just *assumed* that the twins share the same propensity because they're twins, then it's this very assumption - and not the data - that does the explanatory work. And if the twins indeed share the propensity, then the non-imprisoned co-twins' criminality levels at baseline should, on average, match those of their imprisoned siblings. On the other hand, if the non-imprisoned co-twins do not share the criminal propensity, then the design does not control for such a propensity.

The second problem is that the fact that all the twin pairs in this study are discordant for an indicator (imprisonment) of the level of criminality for which they are both assumed to have a propensity is *prima facie* evidence *against* them both having the propensity. Put differently, to the extent the twins are discordant for criminality it becomes less credible that they in fact share a propensity for criminality.

We propose that instead of - or at least in addition to - speculating about propensities, one should try to measure whatever one thinks subjects have a propensity for. If you believe MZ twins share a criminal propensity, include measures that capture the results of such a propensity: if present, it should, on average, produce higher levels of criminal behavior. If, out of a sample of hundreds or thousands of individuals with an assumed propensity for committing crimes, no one has *actually* committed more than an average number of crimes, then what point is there in postulating a propensity?

Another dichotomy: internal vs. external

Taking a step back, we think what is really hard to let go of is the idea that there is something inherited - something that is *internal*, more or less hard-wired, biological - which is opposed to things *external* - outside and not belonging to the person, more changeable and volatile. And that the internal part constitutes our nature, is more fundamental and stable than our environments and cultures, and has been shaped

through millions of years of biological evolution. Hence the seductive appeal of imagining a battle of two forces, one represented by inherited genes inside, the other by everything outside of us, which we lump into a single big category, the environment. The justification for completely flattening the environment in this way is that all that presumably matters about it is that it is *not inherited*, is not shaped by Darwinian evolution, and is therefore not contributing systematically to our nature.

But this is not true: biology can change quickly and even natural selection can occur on time scales as small as years or even months (Gibbs & Grant 1987; Herrel et al. 2008; Losos et al. 2006; Reznick et al. 1997). Environments and cultures, on the other hand, can be stable for centuries and exhibit selection processes that fit the Darwinian bill as much as do genetic systems. They evolve. A simple view that locates our basic mental and behavioral traits inside the body and considers everything outside as less fundamental is therefore misguided.

We believe that ultimately, the separation into “internal” and “external” runs afoul of the same problem that besets the forced split into genes and environment: the two are tied together in a dense web of causal interactions that *jointly* determines developmental outcomes, and that is *jointly* subjected to evolution by natural selection. Our search must be for complete explanations of traits that integrate inside and outside influences.

We agree, however, that in light of the obvious physical boundaries of individual organisms and their autonomy and agency, especially in the human case, questions about the relative roles of internal and external causes of development seem natural. Clearly, this is an area that requires more thinking, especially conceptual and theoretical clarification by philosophers or philosophically-minded scientists. But it is equally clear that out-dated concepts of heritability and absurd dichotomies pitting one type of biomolecule against whole environments does nothing to help sort out these questions, but rather obscures them.

Conclusions

The behavioral genetic conception of heritability - what we have called “heritability_{BG}” - has (at least) three major flaws: first, it is confusing and misleading, because it does not track common expectations about biological heritability; second, it has no useful application in human research: it does not provide insights beyond truisms about the multi-causal nature of development, it does not identify specific causes of traits, it is silent on the potential success of new therapeutic interventions, and it has no bearing on the malleability of traits.

Third, what heritability_{BG} shares with the common understanding of biological heritability (“heritability_{Bio}”) is the popular misconception that heritability is based exclusively on genetic inheritance. In other words, both versions confuse “heritable” with “genetic”. This is both theoretically and empirically false: considering the origin and causal role of heritability in Darwin’s scheme of evolution by natural selection shows that, conceptually, any mechanism that guarantees that traits reliably recur across generations will fulfill the heritability requirement of evolution. Empirically, non-genetic inheritance has been amply documented in nature, and many fundamental observations about the stability of cultural and physical environments can be re-interpreted as phenomena of inheritance in the Darwinian sense. This leads to an extended, causally defined concept of heritability we call “heritability_X”.

Heritability_X incorporates the insight that most trait causes - genetic and non-genetic - recur in every generation and are therefore heritable in the relevant sense, which is as trans-generationally stable enablers of Darwinian evolution. In the old, gene-centered

view of life, inheritance was exclusively genetic, and this required that genes also had the power to guarantee the proper development of heritable traits. The “information metaphor” was the way this was supposed to work: genes embody instructions or even a (computer-like) program to direct the formation of traits.

However, there was never any solid theoretical and empirical reason to believe in the information metaphor, and every reason to reject it, as close scrutiny by philosophers of science has amply demonstrated (Godfrey-Smith 1999; Godfrey-Smith 2008; Griffiths 2001; Griffiths & Gray 1994; Johnston 1987; Keller 1995; Neumann-Held 2006; Nijhout 1990; Moss 2004; Oyama 1985; Sarkar 1996; Šustar 2007). In a new, updated view of life, traits from the parental generation are successfully re-created in offspring because a sufficient number of parental trait causes - genetic and non-genetic - are inherited and serve as “inputs” to offspring development. Same or similar input tends to produce same or similar outputs.

This perspective does justice to the most fundamental observation about development: that it is paradigmatically a process of different kinds of causes interacting constantly to produce traits or phenotypes, and that, in general, these causes are all jointly necessary, but not by themselves sufficient, to generate a certain outcome. There is a complete “causal parity” (Griffiths & Knight 1998) of different developmental causes, with none of them playing an *a priori* more significant or more determinative role than any other.

The causal parity of genes and other developmental factors also implies that genes cannot constitute sufficient causal routes to traits, let alone provide complete explanations of traits. Full-blown explanations will integrate various kinds of causes across different levels of organizational hierarchy, and across the divide between the internal and the external. Appeals to genetic propensities, common in criminology, are not helpful in this endeavor, as genes are not unique in their potential to constitute dispositions for illnesses or behaviors.

Criminologists now have a tremendous chance to shed useless, outdated ideas, adopt a view that represents the cutting edge of modern knowledge about evolution, inheritance and development, and take a leading role in reforming their own discipline as well as the social sciences at large. The expected rewards are deeper explanations, and a truer understanding of what it is about human biology and social circumstance that makes some of us into thieves, murderers, or corporate psychopaths. Is there any reason to hesitate?

Appendix

Stata code for the examples in the section “The interactionist challenge” can be downloaded from https://github.com/alexgamma/3_flavors_heritability_stata_code

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