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Nurture might be nature: Cautionary tales and proposed solutions

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1 **Abstract**

2 Across a wide range of studies, researchers often conclude that the home environment and  
3 children’s outcomes are causally linked. In contrast, behavioral genetic studies show that parents  
4 influence their children by providing them with both environment and genes, meaning the  
5 environment that parents provide should not be considered in the absence of genetic influences,  
6 because that can lead to erroneous conclusions on causation. This article seeks to provide  
7 behavioral scientists with a synopsis of numerous methods to estimate the direct effect of the  
8 environment, controlling for the potential of genetic confounding. Ideally, using genetically-  
9 sensitive designs can fully disentangle this genetic confound, but these require specialized  
10 samples. In the near future, researchers will likely have access to measured DNA variants  
11 (summarized in a polygenic scores), which could serve as a genetic control, but that is currently  
12 not an option that is ideal or widely available. We also propose a work around for when  
13 genetically sensitive data are not readily available: the Familial Control Method. In this method  
14 one measures the same trait in the parents as the child, and the parents’ trait is then used as a  
15 covariate (e.g. a genetic proxy). When these options are all not possible, we plead with our  
16 colleagues to clearly mention genetic confound as a limitation, and to be cautious with any  
17 environmental causal statements which could lead to unnecessary parent blaming.

## 1 Nurture might be nature: Cautionary tales and proposed solutions

2 Most parents spend hours fretting over decisions about the environment they provide to  
3 their children, convinced that this environment has the most direct impact. The scientific  
4 literature mirrors this idea. Across a wide range of studies from many psychological domains,  
5 researchers often conclude that the environment parents provide and children's outcomes are  
6 causally linked, through **environmental transmission**. For example, a recent study examining  
7 the association of having a home library as an adolescent and later adult literacy, numeracy and  
8 technology skills drew our attention because of in-depth coverage in the Guardian ("Growing  
9 up", 2018). This study used a very rich and well-powered dataset, and found a correlation  
10 between the number of books in adolescents' homes and literacy performance in adulthood. They  
11 conclude that "growing up with home libraries boosts adult skills", inferring a causal connection  
12 (Sikora, Evans & Kelley, 2019). This is depicted in Figure 1. Here we discuss how the  
13 correlation between the environments parents provide, the "rearing environment", and their  
14 children's outcomes can indeed be fully due to a causal association, or importantly, can also be  
15 partly due to a **genetic confounding**, illustrated in Figure 2. After highlighting the problem, we  
16 suggest ways that psychological scientists can examine research questions related to the rearing  
17 environment and children's outcomes in ways that account for, or at least acknowledge, genetic  
18 confounding.

### 19 **Genetic control of exposure to the environment**

20 Decades of work from behavioral genetics show that children's traits are influenced by  
21 both genetic and environmental effects (Polderman et al., 2015; Turkheimer, 2000). Moreover,  
22 genetic influences are often seen on measures of the "environment", suggesting that the contexts  
23 surrounding children are partly under genetic control (Kendler & Baker, 2007). For example, a

1 meta-analysis found cumulative support for genetic influences on the parenting children received  
2 (Klahr & Burt, 2014). This idea, that there is genetic influence on exposure to environments, is  
3 called a **gene-environment correlation**. Specifically, there are three types of gene-environment  
4 correlations that can result in genetic confounds (Plomin, DeFries & Loehlin, 1977). First, a  
5 *passive gene-environment correlation* describes the situation where children receive both an  
6 environment they are raised in, as well as genetically influenced traits, from their parents. This  
7 means the environment that parents provide is confounded with genes they share with their  
8 children. Second, an *evocative gene-environment correlation* is when a person's genetically  
9 influenced trait elicits a specific environmental response, making that environmental response  
10 correlated to the person's genes. Third, an *active gene-environment correlation* describes how a  
11 person makes decisions to surround themselves with environments that are related to their  
12 genetically influenced traits. All three have the potential to cloud the true combination of genetic  
13 and environmental influences transmitted between parents and children (i.e., genetic  
14 confounding", but it is theorized that passive gene-environment correlations have a greater effect  
15 in childhood (Scarr & McCartney, 1983), the focus of our review.

16 To give an example of how (passive) gene-environment correlations can result in genetic  
17 confounding in studies focused on the rearing environment, a recent, high impact finding  
18 reported that parents with higher math anxiety have children with higher math anxiety, solely  
19 due to the home environment (Maloney, Ramirez, Gunderson, Levine & Beilock, 2015). The  
20 authors attribute helping with math homework as the causal environmental factor, concluding  
21 that parents with high math anxiety should not help with their children's math homework. This  
22 causal connection could exist, but equally parents with math anxiety also pass on genetic (and

1 environmental) risks related to both lower math cognition and higher math anxiety (Wang et al.,  
2 2014).

3 Another example, this time from the medical literature, examined the intergenerational  
4 transmission of smoking behavior from parents to adolescents, concluding that “the attitudes,  
5 beliefs, and behaviors toward [adolescent] cigarette use are learned through [parent] modeling”  
6 (Gilman et al., 2009). Again, this study focused on the environmental transmission from parents  
7 to offspring without accounting for the transmission of genes related to smoking behaviors and  
8 risk-taking behaviors (e.g. Vink et al., 2005). Furthermore, the authors conclude that smoking  
9 cessation interventions in adults can reduce smoking in subsequent generations. Parent-centered  
10 interventions might help to reduce adolescent smoking, but what is overlooked is that children  
11 carry their own genetic risks for smoking and direct intervention with the adolescents (e.g.  
12 Thomas et al., 2015) could more strongly influence their smoking behaviors.

13 We are certainly not the first to point out this familial transmission confound with the  
14 ecological literature. Indeed, nearly 40 years ago Scarr & McCartney proposed that “the human  
15 experience and its effects on development depend primarily on the evolved nature of the human  
16 genome” (Scarr & McCartney, 1983), and nearly 30 years ago Plomin and Bergeman addressed  
17 the prevalence of genetic confounding by illustrating that genetic influences are found on most if  
18 not all environmental measures (Plomin & Bergeman, 1991). Since then, several reviews have  
19 pointed to multiple examples from parental warmth to alcohol use to depression where causal  
20 pathways from parent behavior to child outcomes are reported, without accounting for genetic  
21 confounding (e.g. D’Onofrio et al., 2003; McAdams et al., 2014; Sherlock & Zietsch, 2018).  
22 These reviews have called for researchers to use caution with causal statements, and to address  
23 genetic confounding in their limitations. Further, they have asked for journal editors and

1 reviewers to be better watch-dogs in this endeavor; to insist that manuscripts adhere to these  
2 standards. However, based on our experience listening to conference presentations, reading press  
3 releases and newspaper articles, and often the majority of the discussion papers of these papers,  
4 we believe these guidelines are not yet being met.

5         We believe a reason why these previous reviews have not successfully changed minds  
6 and methods is because they have not given actionable solutions to researchers outside of  
7 behavior genetics. Therefore, when faced with doing nothing at all or publishing work with only  
8 a *potential* genetic confound, researchers have chosen the latter. Therefore, in the following  
9 section we will give many possible solutions, from gold-standard genetically sensitive designs, to  
10 design solutions that work in lieu of genetically-sensitive data, and finally, a renewed call for  
11 changes in reporting standards.

## 12 **What researchers *can* do**

13         The designs that we will discuss below present a not all-encompassing but global  
14 overview of genetically-sensitive designs and polygenic-scores designs, and include a genetic-  
15 proxy control design (the “Familial Control Method”), which we recommend when genetically-  
16 sensitive data are not available, as well as several other proxy control designs. These designs  
17 vary in how well they disentangle the genetic confound and in how challenging they are in terms  
18 of obtaining and analyzing the data.

### 19 ***Genetically-sensitive designs.***

20         Genetically-sensitive designs are ideal for studying genetic and environmental influences  
21 and their interplay. These designs take advantage of samples of related individuals that differ in  
22 genetic relatedness (e.g., monozygotic and dizygotic twins) or differ in environmental exposure  
23 (e.g., monozygotic twins reared apart). By far the most commonly used genetically-sensitive

1 design is the twin design. Heritability of a trait is indicated if monozygotic twins are more alike  
2 than dizygotic twins, and represents the additive genetic influences individuals inherit from their  
3 parents. With regard to studying the direct effect of specific aspects of the home environment,  
4 twin studies do not work for investigating the rearing environment that twin children share. For  
5 example, twin children growing up together are exposed to the same home library or household  
6 income, so monozygotic and dizygotic twin resemblance cannot be compared. However, a twin  
7 study can begin to separate the direct effect of the home environment in two cases. First, when  
8 child twins can report their own ratings of their home environment, therefore yielding estimates  
9 of the home environment that differentiate monozygotic and dizygotic twins. In this case, the  
10 extent to which aspects of children's rating of their home environment do not show entirely  
11 environmental influences, in other words, some heritability is measured on the "home  
12 environment", this infers that there is a genetic confound (Kendler & Baker, 2007). Using child  
13 twin ratings of the home, Hanscombe et al (2010) found that 22% of the variance of chaos in the  
14 home was attributable to genetic factors, suggesting that this "environmental" variable was  
15 partially genetically confounded.

16         The second way that the twin model can be used to identify the direct effect of the home  
17 environment is by focusing on the home environment that adult twins create. When twins are  
18 adults they can differ in how many books they own and the income of their household, so genetic  
19 and environmental influences on the home environments adults create can be studied. These  
20 studies quantify genetic and environmental influences on the home environment that parents  
21 provide (Kendler & Baker, 2007), but they do not quantify the influence of these home  
22 characteristics on outcomes in their offspring.

1 Genetically-sensitive designs that *can* address the direct effect of the rearing  
2 environment, after accounting for genetic confounding, are adoption studies and twin-family  
3 studies (see D’Onofrio et al., 2013). In an adoption design, resemblance between adopted  
4 children and their biological parents is due to heritability (plus the prenatal environment). In  
5 contrast, resemblance between adopted children and their adoptive parents is fully due to the  
6 environment that the parents have provided. Twin-family studies include twins and their family  
7 members, like young twins and their parents, or adult twins and their children. The latter,  
8 referred to as children-of-twins design, is particularly suitable to study the effect of children’s  
9 rearing environment, free of genetic confounding (McAdams et al., 2014). Put simply, consider a  
10 mother who has an identical twin sister. The mother’s son shares half of his genetic variants with  
11 his mother, but also with his aunt. If for a given trait he resembles his mother as much as his  
12 aunt, this suggests that the resemblance is fully due to shared genes. Conversely, if he is more  
13 like his mother than aunt, this demonstrates that the environment that the mother provides  
14 matters. There are even more complex extended twin family designs, described well in Keller,  
15 Medland and Duncan (2010). In sum, adoption and twin-family designs can rigorously assess  
16 whether the rearing environment is causally affecting children’s outcomes. However, the  
17 disadvantage is that they require access to such data, which are challenging to collect and  
18 analyze. We note for a reader interested in using twin data to better answer their questions about  
19 the direct role of the rearing environment, twin datasets are increasingly becoming publically  
20 available. For example, TwinLife (<https://www.twin-life.de/en>), TEDS  
21 (<http://www.teds.ac.uk/researchers>), NLSY kinship links (<http://nlsy-links.github.io/NlsyLinks/>),  
22 Netherlands Twin Register (<http://tweelingenregister.vu.nl/research>), and others are available  
23 online or via application. In addition, there is a data sharing culture in the behavioral genetics

1 community, and most will likely share when asked. We suggest that researchers consider using  
2 these resources to better test their research questions.

3 *Polygenic-scores designs.*

4 A new avenue to study intertwined genetic and environmental effects employs genome-  
5 wide polygenic scores (PGS). This method relies on genome-wide association studies (GWASs)  
6 which pinpoint genetic variants (i.e., alleles) that are linked to a trait. The most powerful GWAS  
7 to date ( $N > 1$  million) has identified 1,271 genetic variants associated with educational  
8 attainment (Lee et al., 2018). Each of them has a tiny effect, but these tiny effects can be  
9 summed in a PGS. The PGS, calculated for all (unrelated) individuals in an independent sample,  
10 explains 12% of the variance in educational attainment. Note that twin studies estimate the  
11 heritability of educational attainment at 40% (Branigan et al., 2013), so the PGS currently  
12 captures less than one third of this; the remainder is the “missing heritability” (Maher, 2008).

13 As we speak, novel methods are being designed to disentangle nature and nurture that  
14 draw on PGS. Below, we list some examples of recent developments. First, Dolan, Huijskens,  
15 Minica, Neale and Boomsma (2019) bring polygenic scores into the classical twin design. By  
16 doing so, one can estimate the gene-environment correlation, rather than assume it is absent.  
17 Second, Lee et al. (2018) and Selzam et al. (2019) found that for cognitive traits, the predictive  
18 power of a PGS within a family was about 50% lower than across unrelated individuals. The  
19 attenuation of the PGS’ predictive power within families suggests that passive gene-environment  
20 correlations (as captured by the PGS) contribute to children’s cognitive development. As a third  
21 example, both Kong et al. (2018) and Bates et al. (2018) separately proposed the same design  
22 incorporating parental and offspring PGS to disentangle environmental transmission from  
23 **genetic transmission** (i.e., account for the genetic confound between the home environment and

1 child outcomes). In both cases, the researchers split the genetic variants of the parents in half --  
2 those that the parent had and had not transmitted to the offspring -- and calculated for each half  
3 the PGS for educational attainment. Amazingly, both sets of parent PGS predicted adult  
4 offspring's educational attainment. The predictive value of the transmitted PGS was  
5 unsurprising, as this captures directly transmitted genetic effects. But the predictive value of the  
6 *non-transmitted* PGS was not certain, as this effect must be environmental. Kong et al. (2018)  
7 aptly coined this genetic effect through the rearing environment "genetic nurturing". Fourth,  
8 Wertz et al. (2018) incorporated both PGS of mothers and children, as well as direct measures of  
9 parenting. They showed that mothers' cognitive stimulation explained the relation of the  
10 maternal *non-transmitted* PGS to child educational attainment. This indicated that there is a  
11 direct environmental transmission of parenting on children's outcomes, unconfounded by  
12 correlated genetic transmission. Finally, de Zeeuw et al. (2019) used the full genetic-nurturing  
13 design (employing DNA of children and both parents) and found (thereby replicated) genetic-  
14 nurturing effects on adults' educational attainment. Crucially, for outcomes in childhood,  
15 academic achievement and ADHD-symptoms, they only found direct genetic effects; no genetic  
16 nurturing. They concluded that a large contributor to why the rearing environment predicts child  
17 outcomes may well be intergenerational transmission of genetic effects.

18         At the moment, measured genetic variants only explain small proportions of variance and  
19 the papers mentioned above are only a proof-of-principle. Therefore, at this moment, using PGS  
20 as a covariate when studying the rearing environment probably only accounts for a small portion  
21 of genetic confound. Nevertheless, these exciting developments will gain in strength when  
22 increasingly-larger GWASs of all sorts of traits yield more refined PGS. Parallel to this  
23 development, costs of genotyping are falling and the number of cohorts with genotype data is

1 growing (e.g., Evans et al., 2019). We predict that in the not-so-far future, using simply and  
2 cheaply collected genotypic information will become a regular part of the rearing environment  
3 researchers' data collection protocol, allowing researchers to partly control for genetic confounds  
4 in their models. We foresee that it will be easier to do so than rely on genetically-sensitive  
5 designs.

### 6 *Genetic-proxy control designs: the Familial Control Method.*

7 The designs discussed above are the current gold-standards. However, as these types of  
8 samples discussed above are not currently easy to collect and analyze, we propose here a useful  
9 work around (Hart, Ganley, Purpura, 2016; van Bergen, van Zuijen, Bishop & de Jong, 2017).  
10 Our colleagues can measure the same trait in the parent as the child, and use that as a covariate.  
11 That covariate then serves as a proxy for the familial transmission, including genetic  
12 transmission. In doing so, you have a proxy control for the familial effect. Hence, we term this  
13 method the **Familial Control Method**.

14 The Familial Control Method works especially well for traits that are mostly transmitted  
15 from parent to child through genes rather than the environment, like reading ability (Swagerman  
16 et al., 2017; Wadsworth et al., 2002)<sup>1</sup>. Van Bergen et al. (2017) capitalized on this in studying  
17 whether children's reading ability is influenced by the home literacy environment, like reading  
18 habits of the parents and the number of books in the home. Analyses consisted of straight-  
19 forward regression analyses, illustrated in Figure 3. The home literacy environment correlated  
20 with children's reading ability, but for most home-literacy indicators the effect was no longer

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<sup>1</sup> We note for the reader more familiar with the behavioral genetics literature than the familial transmission literature that within individuals, a trait, like reading ability, is often influenced by both genetic and environmental effects (e.g., Little, Haughbrook & Hart, 2017). However, when designs examine how a trait is transmitted from parents to children, often only genetic transmission is found. Environments can contribute to individual differences, but at the same time not impact why parents and children resemble each other.

1 significant after accounting for the reading ability of the parents. This suggests genetic confound  
2 rather than a genuine environmental effect. A similar approach was taken by Hart et al. (2016) in  
3 studying the effect of the home numeracy environment on children's math ability. When parent's  
4 math ability was included in the model, some effects of aspects of the home numeracy  
5 environment on children's math ability were attenuated, but most held up. Thus, doing more  
6 math-related activities with your children does seem to directly boost their math.

7         The Familial Control Method, using a parental trait as genetic proxy, is not watertight,  
8 and certain assumptions must be made for it to be effective for your research question. If, for a  
9 certain trait, parent-child resemblance is not only due to genetic transmission but also  
10 environmental transmission, the Familial Control Method can be too conservative, as it also  
11 takes away some of the variance due to true environmental effects. Additionally, as mentioned  
12 earlier, the trait measured in the parent should be the same as the trait measured in the child. This  
13 means that traits which are not at least reasonably the same in childhood as adulthood (i.e.,  
14 across birth cohorts and across the lifespan) would not work in this design. So the trait should be  
15 at least reasonably measurement invariant and relatedly, show reasonable genetic stability.  
16 Fortunately, for many phenotypes, children's phenotypes are simply developmental precursors to  
17 the adult phenotypes (e.g., for reading ability see Wadsworth et al., 2001, and for ADHD see  
18 Kan et al., 2013). A researcher must decide if the mentioned assumptions are appropriate for  
19 their trait of interest, but fortunately we do believe that these assumptions are reasonable for  
20 most to make. Finally, it is important to reiterate that the Familial Control Method serves as a  
21 proxy control for genetic influences on the studied trait which are shared within a (biologically-  
22 related) family. These genetic influences are where potential genetic confounds of the home  
23 environment would be.

1           We would like to argue that the parental trait is the best genetic proxy and that the  
2 Familial Control Method is currently feasible for many behavioral scientists. It does not require  
3 data of twin or adoption families, nor collecting DNA samples. In terms of prediction, parental  
4 traits capture more of the variance in children’s outcome than polygenic scores, so likely also  
5 capture more of the genetic confound. For the example of reading ability, it has been found that  
6 the abilities of the parents explain 21% of the ability of children (van Bergen et al., 2015). In  
7 comparison, polygenic scores (based on the educational-attainment GWAS) have been found to  
8 explain only 2-5% of reading ability in children (Selzam et al., 2017), and more recently 5-14%  
9 of “educational achievement” (including reading, writing, speaking, listening, and mathematics)  
10 in ages 7 to 16 years (von Stumm et al., 2019). Certainly this proportion of variance explained  
11 from simple polygenic scores is not trivial, and the predictive ability of polygenic scores is  
12 anticipated to increase in the coming years. However, for most behavioral scientists the trait in  
13 the parents is not only easier to measure, but currently also a better predictor. On a related note,  
14 the value of parental traits as predictors of child outcomes has been used for decades in studying  
15 precursors of developmental disorders. In such family-risk studies, children with a family history  
16 of say dyslexia, attention-deficit/hyperactivity disorder or autism are followed from an early age,  
17 before the disorder manifests itself. These children have an increased risk to develop the disorder  
18 (Snowling & Melby-Lervåg, 2016, Musser et al., 2014).

19           ***Other proxy control designs.***

20           Other proxy controls such as sociodemographic factors (e.g. SES) have been used, but  
21 these statistical adjustments are not capable of accounting for genetic confounding as adequately  
22 as the Familial Control Method, for several reasons. First, although sociodemographic factors  
23 such as educational attainment have been significantly associated with genetic factors through

1 twin studies (~20-40% Rietveld et al., 2013: Table S10; Branigan, McCallum, & Freese, 2013)  
2 and genome-wide association studies (~14%; e.g. Lee et al., 2018), the estimates are less than  
3 unity which indicates that genetic influences are not entirely responsible for individual  
4 differences in SES. Indeed, work examining the intergenerational transmission of SES has  
5 suggested that both genetic and environmental transmission occurs (Björklund, Jäntti & Solon,  
6 2007). In this scenario where SES is transmitted through genetic and environmental pathways,  
7 when controlling for SES in data analyses, a proportion of variance attributable to other  
8 background or environmental factors is also being controlled for in the model, leading to reduced  
9 associations between potentially important family-level predictors and child outcomes. In other  
10 words, you'd be throwing the baby out with the bathwater.

11         On the other hand, controlling for SES does not actually control for all of the genetic  
12 confounding. Say a researcher is interested in controlling for genetic confounding when  
13 examining the direct influence of books in the home on children's reading. Parental SES is a  
14 proxy for parental reading skill, but not a perfect correlate (average correlation is .26; Sirin,  
15 2004). Controlling for parental SES would not control for all of the potential genetic confounds  
16 on the association between books in the home and children's reading ability.

17         In conclusion, when controlling for SES, other potential sources of environmental  
18 variance are also being removed from the prospective models, and at the same time would not  
19 capture the extent of genetic confounding. We believe this would happen with other proxy  
20 control measures as well, outside of the Familial Control Method.

## 21 **Conclusion**

22         Here we have laid out numerous ways that genetic confounding can be controlled for  
23 when examining the rearing environment. We can certainly foresee times that none of these

1 options are possible. Therefore, we conclude that in those instances, our colleagues need to  
2 clearly mention the possible genetic confounding as a limitation, and to be cautious with any  
3 environmental causal statements which could lead to unnecessary parent blaming. To return to  
4 our first example, expecting all homes to have plenty of books is an idealistic goal, as it would  
5 surround all children with the opportunity to read if they wished. But unfortunately, having the  
6 opportunity to read as one wishes does not unlock the code of reading for all children. Reading is  
7 a skill that requires direct instruction and practice, and children with a family history of dyslexia  
8 themselves have a 45% chance of dyslexia despite adequate instruction and practice (Snowling  
9 & Melby-Lervåg, 2016). Simply having books around the home is not enough (Kim, 2007), yet  
10 the message that parents are getting is that it is (“Growing up”, 2018). The take home messages  
11 from that are that either parents who don’t have the resources for a home library are hurting their  
12 children, or parents with children struggling to read are at blame because they didn’t have quite  
13 enough books in the home. This is unfair and inaccurate. In the end, we believe that it is  
14 important to discover true environmental effects as well as how genes and environments  
15 interplay, especially when malleable, because then we can focus as field on creating and testing  
16 interventions that have a greater chance of directly improving children’s outcomes.

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## GLOSSARY

Environmental transmission (also called: Cultural transmission)	Transmission of traits from parents to their children by non-genetic means. It is used to describe when parents' traits impact their child's traits through the environment they create.
Genetic transmission	Transmission of traits from parents to their children by genetic means (i.e., children inherit genes from their parents for a given trait).
Genetic confounding	Here we use this term as a synonym for gene-environment correlation, describing a situation where the influence of parental traits on children's traits is not solely due to environmental transmission.
Familial transmission	Transmission of traits from parents to their children, both by genetic and non-genetic means. Familial transmission gives rise to parent-child resemblance.
Gene-environment correlation	Genetic influence on the exposure to the environment. There are three types: passive, evocative, and active (see text).
Familial Control Method	Using a measure of the same trait in the parent as the child as a covariate in models estimating the effect of the rearing environment. That covariate then serves as a proxy control for the genetic transmission effect.

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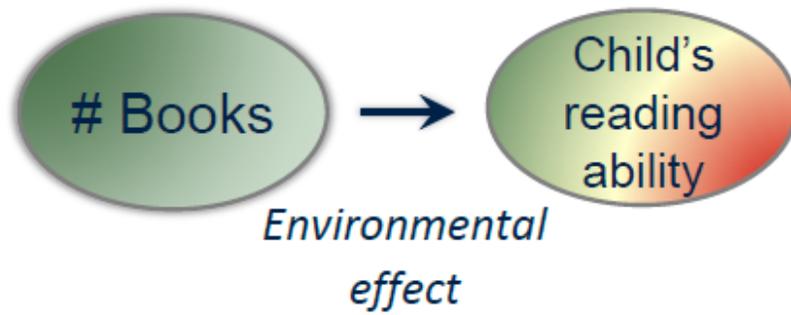
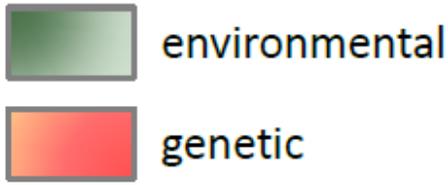
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Source of individual differences:



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*Figure 1.* An example of a direct environmental transmission effect. Number of books in the home is thought to be an environmental causal effect on children's reading ability.

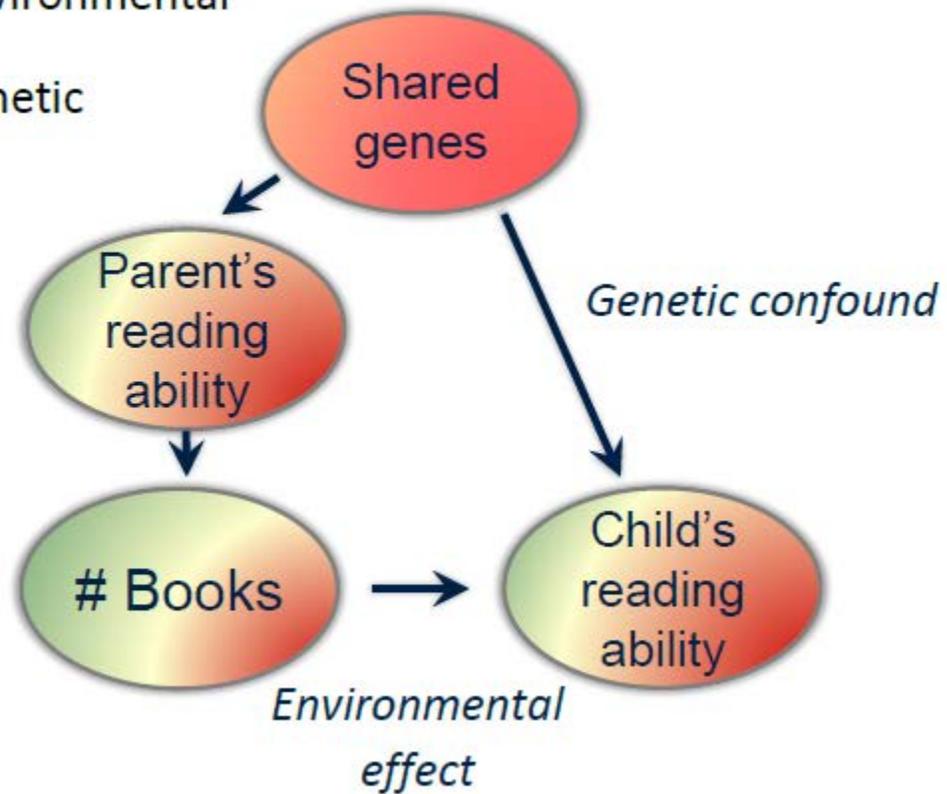
Source of individual differences:



environmental



genetic



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2 *Figure 2.* An example of how genetic confounding works (note, only one parent drawn, for  
3 simplicity). Parents share genes related to reading ability with their children, and also control the  
4 number of books in their home. It is important to note that the environmental effect may still  
5 have a causal role, even with genetic confounding.

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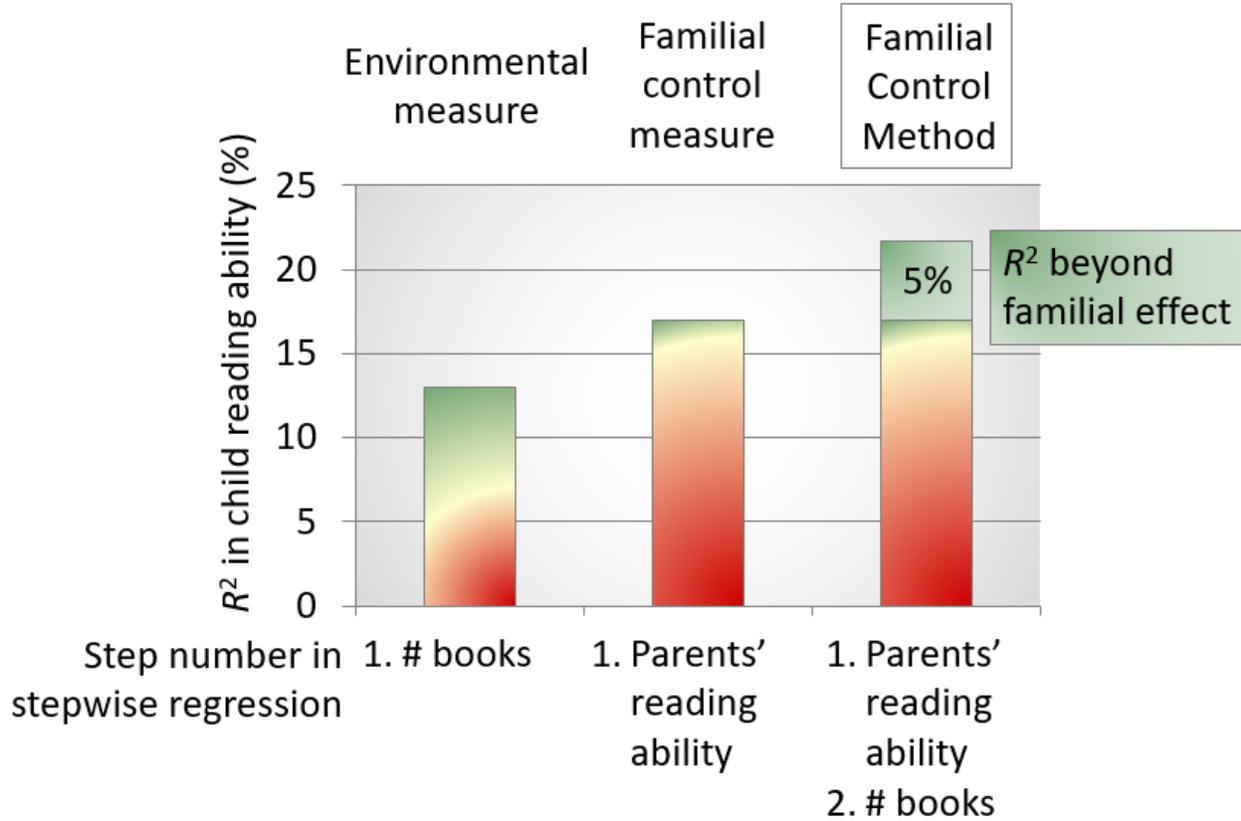
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3 *Figure 3.* Visualization of the Familial Control Method, in which a child outcome is predicted in  
4 a step-wise regression, with in the first step the familial control measure (i.e., the trait in both  
5 parents) and in the second step the measure of the environment. The findings that are depicted  
6 here come from van Bergen et al. (2017). The key question is whether the environmental  
7 measure explains variance beyond the familial effect, as this indicates a genuine environmental  
8 effect. In the example given, this was 5% and significant. This was negligible and non-  
9 significant for the other environmental measures reported in van Bergen et al. (2017).

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