Bonamy R. Oliver & Alison Pike

University of Sussex

'To what extent does the heritability of known features of development such as intelligence limit the potential of policy that attempts to lessen intergenerational inequalities such as early intervention, social mobility, child poverty or the social class attainment gap'.

In the current report, we argue that the extent to which aspects of human development are heritable has little impact on the importance of policy that aims to ameliorate the adverse effects of environmental or intergenerational inequalities. We first address common misconceptions about heritability and behavioural genetics that can lead to inappropriate assertions about research findings and their implications for policy. We then move to demonstrate our vision of what behavioural genetics can offer early intervention science and attempts to lessen intergenerational inequality.

What is behavioural genetics?

Behavioural genetic research uses a variety of quasi-experimental designs to understand the role of genetic and environmental influence on variation in psychological traits such as general intelligence (e.g., Deary, Johnson, & Houlihan, 2009), disruptive behaviour (e.g., Rhee, & Waldman, 2002), and well-being (e.g., Bartels, 2015). The extent to which variance in a given trait is explained by genetic variance is termed ‘heritability’, and is explained further below. Shared environmental influences refer to experiences that contribute to sibling similarity, while non-shared environmental influences refer to differences in experience that contribute to differences between siblings in a family. The most common of these research designs are twin and adoption

studies. The twin design compares monozygotic (MZ: 'identical') and dizygotic (DZ: fraternal, 'not identical') twin similarity for specific psychological traits. On average MZ twins share 100% of their segregating genes, while DZ twins share on average only 50%, the same as brothers and sisters. Thus, the premise of the twin method is that, to the extent that members of MZ twin pairs are more similar to each other than are DZ pairs, a role for genetic influence is suggested; the more pair similarity for *both* MZ and DZ twins, the more shared environmental factors are implicated. In adoption studies, children living away from their biological parents in adoptive homes are examined. Here, the extent to which adopted children are similar to their biological parents suggests genetic influence, while similarity to their adoptive parents is evidence for environmental influence. In these ways, behavioural genetic designs allow the apportioning of trait variance into genetic as well as shared – most directly indexed through adoption studies -- and non-shared – most directly indexed through MZ twin differences -- environmental influences (once genetics have been accounted for).

Using such genetically-informed designs, scientists have shown almost every human trait to be at least partly heritable (Polderman et al., 2015). Indeed, findings from such studies are some of the most robust and replicable in the field of psychology (Plomin, DeFries, Knopik, & Neiderhiser, 2016). Behavioural genetics has become increasingly accepted and integrated across many areas of psychology, reaching more scientists, and thus expanding and developing in exciting and enticing ways. However, in the accelerated reach of research showing heritability for human development, there is a real and significant danger of common misconceptions in its interpretation and application.

Misconception 1: Heritability tells us about the cause of a trait in an individual

Heritability is perhaps best explained with an analogy. Let us suppose that we have two identical

plant seeds; amongst other things, light and water are essential for their growth. If both plants are given water (potential shared environmental influence) but one is kept in a pot on a window ledge and the other in the dark (potential non-shared environment), we would attribute any growth variation between the two plants to light. That is, differences in growth between the two plants would be due to differences in light exposure. Importantly, however, we would not say that light is the only cause of plant growth *per se*; instead, we understand that for the growth of each individual plant, water and light are both important.

Now let us suppose that we have two non-identical seeds (potential genetic influence) in a single pot that is watered and placed on the window ledge. Here, we might also expect to see some variation in plant growth. If we assume that we are able to control the within-pot environment well enough that both seeds in the pot receive equal amounts of water and light, the growth variation within pots would be explained by the genetic variation of the seeds -- heritability. Yet, environmental factors (light and water) would clearly continue to play an enormously important role in the growth of both plants. These same principles can be applied to human traits.

Misconception 2: A role for heritability in human development means no role for environmental influence

Behavioural genetic designs are one of the most powerful that we have to tell us about the role of the environment. Specifically they tell us about how variations in environmental experience relate to variation in a trait. For example, child and adolescent disruptive behaviour problems are found to be moderately heritable (e.g., Rhee & Waldman, 2002), yet there are clear demonstrations from genetically-informed studies that environmental influences are important even once genetics are accounted for (e.g., Burt, 2009; Moffitt, 2005), such as parenting (e.g., Oliver, 2015), peer relationships (e.g., Brendgen, 2014), and physical maltreatment (e.g., Jaffee, Caspi, Moffitt, &

Taylor, 2004).

Misconception 3: Highly heritable traits are immune to environmental intervention

To clarify this misconception, we highlight two examples in human development. The first example concerns height. The historical trend of an increase in average human height is well-documented (e.g., Floud, Fogel, Harris, & Hong, 2011). Notwithstanding, there is considerable variation in height within populations, for which genetic variation is extremely important: in the modern day, height is highly (~80%) heritable in developed countries (Silventoinen, et al., 2003). This high heritability is due to the relatively recent societal change (intervention) of broad availability of nutrition in these countries, such that this causal mechanism is no longer responsible for much of the variation in height within these populations. **Paradoxically, highly heritable traits are open to environmental intervention.** The second example involves a rare, genetically-transmitted disorder, phenylketonuria (PKU). Affected individuals are unable to metabolise the amino acid phenylalanine, and, left untreated, PKU causes severe intellectual disability, even mortality. This disorder is 100% heritable, yet, detected early, the effects of classic PKU can be ameliorated by an environmental intervention -- a dietary change. **The effectiveness of intervention is independent of heritability.**

The same premises are true for complex psychological traits. For example, for disruptive behaviour problems, despite their aforementioned heritable component, environmental interventions are proven to successfully reduce these difficulties for many affected children, improving life chances and cutting considerable associated costs to society (National Institute for Health and Care Excellence, 2013).

Thus, the answer to the question, 'To what extent does the heritability of known features of

development…limit the potential of policy that attempts to lessen intergenerational inequalities...” is, “not very much at all”.

But this is not the epilogue. Critically, the answer to a slightly different question, “What does the field of behavioural genetics have to offer for intervention and policy that attempts to lessen intergenerational inequality” is, we argue, “a great deal”.

Behavioural genetic studies have been used to understand “what is”, examining trait variation in a population at a given time, leaving “what could be” to intervention science. However, while interventions to lessen numerous adverse psychological symptoms have an impressive evidence base, it is common knowledge that there are differences in the extent to which individuals respond. It is in understanding the aetiological mechanisms underlying differential response that behavioural genetics may have great value. It is plausible – we would argue likely -- that variation in response is at least partly genetically influenced. Moreover, to the extent that genetic variation confers differential susceptibility to environments (Belsky, 2005; Kim-Cohen & Gold, 2009), interventions may differ in their effect as a function of genetic risk, foreshadowing the idea that the impact of interventions may differ (e.g., Brody, Beach, Philibert, Chen, & Murry, 2009; Hudson, Keers, Roberts, Coleman, Breen, Arendt, et al., 2015). With increasing recognition that genetic and environmental influences correlate and interact in their impact on development (e.g., Rutter, 2012), we maintain that without a genetically-informative component, studies targeting intervention response cannot rule out the potential for such response modifiers to capture gene-environment interplay (e.g., the transmission of both genes and environment, ‘ gene-environment correlation’). Unpicking genetic and environmental influences on response variation is arguably the first step in a logical process to understanding how to improve intervention potency, since identifying characteristics that underlie, predict or moderate response is key (Beauchaine,

Neuhaus, Brenner, & Gatze-Kopp, 2008; Moffit, 2005; Plomin & Haworth, 2010).

Through better understanding the role of genetic and environmental factors for human disease, medical science has made leaps and bounds in understanding the importance of genetics for probabilistic risk and for treatment (e.g., Stewart & Wild, 2015; Gire et al., 2014). Better understanding of such factors for psychological development may have similar potential for prevention and intervention. The better we understand what works, for whom, and why, the closer we come to individualising intervention to best target resources. Using multiple methods to understand the genetic and environmental mechanisms responsible for variations in development, environmental experience, and intervention response, will ultimately lead to more effective prevention and intervention efforts.

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