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Plastic and immobile: Unequal intergenerational mobility by genetic sensitivity score within sibling pairs



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ABSTRACT

Contrary to traditional biological arguments, the differential susceptibility model suggests genotype may moderate rather than mediate parent-child economic similarity. Using family fixed effects models of Add Health sibling data, I investigate the relationship between an index of sensitive genotypes and intergenerational mobility. Full, same sex sibling comparisons hold constant parental characteristics and address the non-random distribution of genotype that reduces internal validity in nationally representative samples. Across multiple measures of young adult financial standing, those with more copies of sensitive genotypes achieve lower economic outcomes than their sibling if they are from a low income context but fare better from a high income context. This genetic sensitivity to parental income entails lower intergenerational mobility. Results support the differential susceptibility model and contradict simplistic genetic explanations for intergenerational inequality, suggesting sensitive genotypes are not inherently positive or negative but rather increase dependence on parental income and reduce mobility.

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1. Introduction

A recent *New York Times* article – “The Mixed-Up Brothers of Bogotá” by Susan Dominus (2015) – chronicles the lives of two pairs of identical twins in Colombia who were switched shortly after birth and raised as fraternal twins. One pair grew up in a rural setting, with poor parents and limited opportunity for schooling, while the other pair grew up in an urban setting with a struggling single mother but more opportunity for mobility. The two boys raised in the city now have professional occupations (accountant and engineer), while the two boys raised in the country are butchers. The identical twins share common behaviors, even after 27 years of living apart, but the story illustrates how social context can trump genes and determine financial standing as a young adult.

Contrary to this emphasis on social context, traditional biological explanations for intergenerational transmission of financial standing emphasize the importance of genes in mediating parent-child similarity (Clark et al, 2014). That is, a simplistic genetic argument would suggest that parents pass financial standing on to their children through their genes. In support of this perspective, behavioral geneticists have argued that genes play a major role in a wide variety of psychological and behavioral characteristics, including intelligence, cognitive ability, autism, hyperactivity, personality, schizophrenia, political beliefs, altruism, and food preferences (Plomin et al., 2013, 2016; Haworth et al., 2010).

Failure to identify specific genes that account for these high heritability estimates (e.g., Visscher, 2008) has raised doubt about these behavioral genetic claims. At the same time, there is growing recognition that the distinction between genes and

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environment is a false dichotomy (Kendler and Karkowski-Shuman, 1997; Kendler and Baker, 2007; Traynor and Singleton, 2010) and that genes and environment work together to influence individual outcomes (Belsky and Pluess, 2009). For example, work by the recently formed Social Science Genetic Association Consortium (e.g., Chabris et al., 2015) emphasizes that individual outcomes reflect complex interactions among multiple genes, behaviors, and environmental factors. The relationship between genes and child outcomes, in other words, is much more complex than simple genetic determinism (Turkheimer, 2011; Conley, 2011a).

Gene-environment interactions are one aspect of this complex relationship. Rather than determining one's financial standing or educational attainment, for example, the importance of the genes one inherits may depend on social context or environment (Guo and Stearns, 2002; Turkheimer et al., 2003). As Conley (2011b:231) describes it, "A gene for aggression lands you in prison if you are from the ghetto, but in the board room if you are manor born."

To investigate these competing claims, this paper asks whether environment – specifically parental income – moderates the relationship between genes and financial standing. There are two difficulties of testing this type of interaction. First, genotype and social environment are not randomly distributed throughout the population. Second, given this non-random distribution, unobserved confounders – such as parental behaviors, education, ethnicity, or social capital – could influence both parent and child financial standing. I use sibling comparisons to address both of these challenges.

Family fixed effects models allow sibling comparisons, controlling for all stable characteristics shared by siblings from the same family, including parental characteristics. In addition, within full sibling pairs each sibling has an equal chance of inheriting one of two alleles (genetic variants) at a particular genetic location from each parent (Fletcher and Lehrer, 2011). Thus, genotype is randomly distributed within full sibling pairs. Capitalizing on these methodological advantages of siblings, and using a genetic index previously found to increase sensitivity (Belsky and Beaver, 2011), I use family fixed effects models of sibling data from the National Longitudinal Survey of Adolescent Health to Adult (Add Health; Harris, 2009) to investigate whether an index of sensitive genotypes moderates parent-child economic similarity.

In the process, this paper also addresses a new question related to intergenerational mobility: Why do some children follow in their parents' financial footsteps, reproducing inequality between generations, while others experience greater mobility? At the individual level, relatively little research has documented factors that increase or decrease the likelihood of economic mobility or why some individuals in a similar context experience socioeconomic mobility while others do not.

In the following sections, I review literature on gene-environment interaction research, intergenerational mobility, and the relationship between genes, environment, and financial standing. I then provide details about data and methods, followed by results and a conclusion including limitations and implications.

2. Gene-environment interaction

Rising assortative mating (Schwartz and Mare, 2005), increasing inequality (Piketty and Saez, 2014), and evidence of gene-environment interaction (GxE) and genetic selection into various environments (Belsky and Pluess, 2009; Fowler et al., 2011) all raise concerns about efforts to distinguish genetic and environmental contributions to complex traits. The missing heritability problem or "genetic dark matter" – the difference between twin heritability estimates and the variation in traits explained by genome-wide data – suggests that the relationship between genes and child outcomes is much more complex than simple genetic determinism (Turkheimer, 2011:600; Conley, 2011a). GxE interactions could partially account for missing heritability.

Over the last two decades, research has found evidence of GxE interaction, suggesting genetic effects on a variety of measures (e.g., depression, college attendance) depend on our environment (Caspi et al., 2003; Guo et al., 2008; Shanahan et al., 2008; Thapar et al., 2007; Caspi et al., 2010). Replication of GxE effects have often proved elusive (see Dick, 2011; Manuck and McCaffery, 2014 for reviews). Part of the failure to replicate could stem from the tendency in early GxE research to limit analysis to potential negative effects (e.g., Caspi et al. 2002; 2003).

More recent advances in GxE research suggest that certain genotypes increase sensitivity to environments, increasing *variation* in outcomes rather than simply *risk* of negative outcomes (Belsky, 2013, 2005; Belsky and Pluess, 2009; Belsky et al., 2007). Called the differential susceptibility model or the biological sensitivity to context (BSC) hypothesis (Boyce and Ellis, 2005; Ellis and Boyce, 2008; Belsky, 2013, 2005), this model suggests genotypes previously considered risky may confer risk or benefit, depending on the environment. This differential susceptibility model is appealing because it could help explain the survival of these genotypes in human populations (Belsky, 2005). Though potentially risky in a negative environment, in a particularly *supportive* environment carriers of sensitive genotypes could achieve even more *positive* outcomes than those with more stable genotypes. Sensitive genotypes, in other words, may make individuals carrying them more dependent on the characteristics of their environment than others who carry more stable genotypes.

The mechanisms accounting for this heightened sensitivity are not fully understood, but possibilities include neurobiological responses to stress (cortisol and fight-or-flight response) and epigenetics (Boyce, 2012). Shanahan and Hofer (2005) identify four ways in which environment could moderate gene expression: triggering; compensation; social control; and enhancement. Triggering occurs when an individual has a genetic predisposition for an outcome and some adverse social context (e.g., a stressful life event or childhood maltreatment; Caspi et al. 2002, 2003) triggers the expression of the adverse outcome. In contrast to adverse social context, a highly supportive context can compensate for a genetic predisposition by preventing the expression of an adverse outcome. Social control can limit genetic expression, not by providing a rich environment as in compensation, but by constricting behavior through social norms or institutions. Finally, enhancement occurs

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