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.

Keywords
Intergenerational mobility; gene-environment interaction; socioeconomic attainment; sibling analyses; fixed effects

Highlights

- Sensitive genotypes are not inherently positive or negative.
- Effects of sensitive genotypes on socioeconomic attainment depend on parental income.
- Sensitive genotypes increase dependence on parental income, yielding lower intergenerational economic mobility.
- Results differ when accounting for the non-random distribution of genes and environment.

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 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 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.

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 and 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 when social context accentuates the expression of positive genetic predispositions (see Shanahan and Hofer 2005 for more details about these ideal types). A combination of triggering and enhancement could explain patterns consistent with the differential susceptibility model.

Despite enjoying growing research attention (Conley et al. 2013; Belsky et al. 2013; Pluess et al. 2010; Pluess et al. 2011; Simons et al. 2011; Bakermans-Kranenburg and Ijzendoorn 2007; Belsky et al. 2007; Pluess and Belsky 2010), efforts to understand GxE interaction – and to test the differential susceptibility model – face two central limitations. Specifically, GxE research struggles to address the fact that neither environment nor genotype is randomly distributed across the general population. In terms of environment, Conley and Rauscher (2013) point out that most existing GxE research relies on environmental measures (e.g., parental maltreatment, family dinners, or peer characteristics) that could be associated with genotype (Caspi et al. 2002, 2003; Guo et al. 2008; Pescosolido et al. 2008; Shanahan et al. 2008; Daw et al. 2013). For example, using a sample of young men from New Zealand, Caspi and colleagues (2002) find that the relationship between MAOA genotype and antisocial behavior depends on whether an individual was maltreated as a child. Similarly, Guo and colleagues (2008) find an interaction between frequency of family dinners and MAOA genotype when predicting delinquent behaviors among young adult men. Although they use the Add Health sibling

sample, Guo et al. do not address between-family differences. In both of these studies, the environmental measure (likelihood of frequent family dinners or childhood maltreatment) could be associated with parental genetic characteristics. In that case, the environmental measures could be a proxy for parental genotype and the interactions found by Guo et al. (2008) and Caspi et al. (2002) could reflect genetic interaction with unmeasured parental genotype (gene-gene rather than GxE interaction).

Similarly, research finds evidence that genotype is correlated within friendship networks (Fowler et al. 2011), suggesting individuals select into peer groups (and possibly other social environments) partly on the basis of genotype. In that case, evidence of an interaction between peer characteristics and genotype (Dick et al. 2007; Daw et al. 2013) could partially reflect gene-gene interactions rather than GxE. In other words, because genetic makeup could shape aspects of environment, apparent evidence of GxE interactions could reflect an underlying interaction between genes. Although some GxE research carefully addresses non-random environmental variation (e.g., Bakermans-Kranenburg et al. 2008; Fletcher and Lehrer 2011), additional research taking methodological steps to address this issue is required to advance understanding.

In addition to environment, however, GxE research must also address the possibility that genotype is not randomly distributed. Population stratification occurs when genetic variation is correlated with environmental or genetic differences. For example, Thomas and Witte (2002:505) note that dopamine receptor D2 (DRD2) genotype is not equally distributed by ethnicity. Therefore, any apparent effects of DRD2 variation may actually reflect the social dynamics of ethnicity and not the causal effect of DRD2 genotype. Similarly, evidence of an interaction between DRD2 genotype and environmental factors (e.g., Shanahan et al. 2008) could reflect population stratification rather than GxE. Others (Gelernter and Kranzler 1999;

Abdolmaleky et al. 2004; Fan and Sklar 2005; Sabol et al. 1998) show significantly different ethnic distributions of DRD2, serotonin transporter (5-HTT), and monoamine oxidase A (MAOA) genotypes, which are frequently investigated in GxE research. While it is possible to control for ethnicity, we do not know all of the relevant characteristics we need to control based on the current research literature and, even if we did know, we would probably not have data on all of those characteristics. Without some method of randomization we cannot know whether a given allele is orthogonal to all social environments. Coupled with evidence that individuals self-select into social environments based on genotype (Fowler et al. 2011), we must conclude that analyses failing to account for the non-random distribution of genotype may be biased.

Biased estimates could result in false evidence of GxE interaction. Methodologically, therefore, GxE research must continue efforts to address the non-random distribution of both genotype and environment. Within-family analyses, including sibling comparisons, offer one methodological opportunity to address these challenges (Fletcher and Lehrer 2011).

Beyond methodological challenges, however, GxE research must also investigate broader implications of the differential susceptibility model. For example, while sensitive genotypes are often described as "plastic" in the differential susceptibility model (Belsky and Pluess 2009; Belsky et al. 2009; Barker 2005), what does plasticity entail? The term plastic suggests a high degree of malleability. In materials science, however, plasticity indicates a substance that can be shaped under pressure, but retains its new shape once the pressure is removed. A paper clip, for example, can be bent out of shape, but it retains that new shape after the pressure stops. In contrast, elastic materials such as rubber bands can be bent or shaped under pressure but return to their original shape after the pressure is removed.

If we extend this plastic analogy, then perhaps sensitive alleles are associated with greater dependence on environment, but also less economic mobility. That is, individuals with more sensitive alleles may be more dependent on parental characteristics or social background and enjoy less intergenerational mobility. Thompson (2014), for example, finds a pattern consistent with this for education, but only among men with a particular variant of the MAOA gene. The implications of the differential susceptibility model for intergenerational inequality have yet to be investigated directly.

Intergenerational Mobility

How is inequality transmitted between generations? Behavioral geneticists have argued that genes play a major role in a wide variety of psychological and behavioral characteristics, including intelligence, cognitive ability, and personality (Plomin et al. 2013, 2016; Haworth et al. 2010). In a related vein, simple genetic explanations suggest genes play a key role in mediating parent-child similarity of financial standing (Clark 2014). These genetic explanations suggest that the importance of genotype for adult economic outcomes should be unrelated to economic background. That is, if genetics simplistically determine adult financial standing, they should play the same role for everyone, regardless of the context in which one was raised.

Existing research raises considerable doubt about these simplistic genetic perspectives. We know, for example, that intergenerational mobility varies across multiple dimensions, including nation (Ermisch et al. 2012; Breen 2004; Solon 2002; Erikson and Goldthorpe 1992; DiPrete and Grusky 1990), time (Breen 2004), regional differences such as local tax expenditures (Chetty et al. 2013, 2014), occupational category (Erikson and Goldthorpe 1992; Featherman and Hauser 1978), level of education (Torche 2011; Breen and Jonsson 2005; Hout

1988), and measure, including economic, educational, cognitive, and behavioral characteristics (Ermisch et al. 2012; Duncan et al. 2005).

Intergenerational similarity clearly depends on social context. Yet, perhaps because intergenerational mobility is a population characteristic, less is known about why some individuals in a similar context experience socioeconomic mobility while others do not. Similar to studies of mobility, research on resilience (Seccombe 2002; Garmezy 1987; Werner 1993; Wexler et al. 2009) often identifies family- or community-level factors associated with greater likelihood of resilience despite disadvantaged contexts. Yet siblings who were raised in the same family and community can experience varying levels of socioeconomic attainment and intergenerational mobility. Research on individual resilience often examines developmental or psychological outcomes (Howell and Miller-Graff 2014; Lowe et al. 2015; Kassis et al. 2013), with less examination of socioeconomic attainment. At the individual level, relatively little research has documented factors that increase or decrease the likelihood of economic mobility.

If the BSC model extends to financial standing, then perhaps sensitive genotypes help explain why some individuals experience mobility while others in the same context do not. The likelihood of experiencing intergenerational mobility could partly depend on genotype – not because genotypes are inherently positive or negative, but because they relate to sensitivity to childhood environment.

Genetic Sensitivity, Environment, and Financial Outcomes

Evidence suggests that variation at the genetic locations examined in this study may be directly associated with financial outcomes (Sapra et al. 2012; Carpenter et al. 2011; Eisenegger et al. 2010). For example, recent experimental evidence (Kuhnen and Chiao 2009; Dreber et al. 2009) suggests that the long 5-HTT allele and the 7-repeat DRD4 allele are positively associated

with risk taking in financial investment decisions. Carpenter et al. (2011) find that the 7-repeat DRD4 allele is associated with risk-taking in economic experiments and predicts financial choice patterns. Zhong et al. (2009a) find an association between MAOA allele and preference for longshot financial risks. In another study, Zhong et al. (2009b) find an association between DAT1 genotype and risk tolerance in financial decisions. Although the mechanisms are not fully understood, the relationship between these genes and financial outcomes may be occurring through risky behavior or risk preferences.

The body of research linking genotype to financial decision making is growing, but tends to rely on experimental evidence (e.g., Carpenter et al. 2011; Dreber et al. 2009; Zhong et al. 2009a, 2009b). Although real life financial decision making may differ from that in the lab, the evidence that exists suggests that the genetic variants examined here may be associated with financial outcomes either directly through financial decision making or indirectly through health and behavioral measures such as ADHD (Fletcher 2014).

In addition to a potential relationship with financial outcomes, research suggests that these genotypes could also increase sensitivity to parental financial standing. For example, Taylor et al. (2006) find that short 5-HTT alleles increase sensitivity to early childrearing history and recent life events. Similarly, Retz et al. (2008) find increased sensitivity to childhood environment among those with short 5-HTT alleles. Daw et al. (2013) find evidence that individuals with more short 5-HTT alleles are more sensitive to peer behaviors. These studies examine different contexts and outcomes, including depression (Taylor et al. 2006), ADHD (Retz et al. 2008), and binge drinking (Daw et al. 2013), suggesting that the short 5-HTT alleles increase sensitivity to environment in general terms across multiple outcomes.

Similar evidence exists for the DRD4 7-repeat allele. Externalizing behavior of those with more copies of the 7-repeat DRD4 allele are more sensitive to maternal sensitivity and parenting quality (Bakermans-Kranenburg and van IJzendoorn 2006), even when an experimental intervention is used to impact parenting quality (Bakermans-Kranenburg et al. 2008). In the case of DAT1, Laucht et al. (2007) found that ADHD symptoms among those carrying the 10-repeat allele are more sensitive to psychosocial adversity than among others. Mitchell et al. (2014) found evidence that the DAT1 10-repeat allele, in combination with others, increased sensitivity to family socioeconomic status. Similarly for DRD2, evidence suggests the A1 allele increases sensitivity to maternal sensitivity (Mills-Koonce et al. 2007; Propper et al. 2008) and birth weight (Keltikangas-Jarvinen et al. 2007) when predicting child behavior, physiological reaction to maternal separation, and educational attainment.

Finally, the 2- and 3-repeat MAOA alleles also show evidence of increasing sensitivity to context. Kim-Cohen et al. (2006) find evidence that these alleles increase sensitivity to physical abuse when predicting mental health and ADHD symptoms. Research similarly finds evidence that the short MAOA alleles increase sensitivity to childhood adversity (Foley et al. 2004) and maltreatment experience (Nilsson et al. 2006) when predicting conduct disorder and criminal activity. In a paper particularly relevant for this study, Thompson (2014) documents that educational attainment of males with a short MAOA allele is more sensitive to parental income and education.

Most of the above studies investigate one genotype at a time. Consistent with the idea that these alleles all similarly increase sensitivity to context but that individual effects may be too weak to detect without huge samples, it is becoming increasingly common to create a polygenic or additive index of sensitive alleles (Mitchell et al. 2014; Pearson-Fuhrhop et al. 2013; Stice et

al. 2012). Belsky and Beaver (2011), for example, create a cumulative index of the five alleles discussed above and find that the index increases sensitivity to parenting. To build on their analysis, I use the same index in this study.

As illustrated in the review above, genetic sensitivity analyses have investigated a variety of environmental contexts and outcomes, including self-regulation (Belsky and Beaver 2011), sensation seeking (Sheese et al. 2007), and ADHD (Retz et al. 2008). The broad array of outcomes and contexts suggests that the genotypes in question increase sensitivity broadly. That is, the evidence suggests they make multiple individual outcomes more sensitive to the environment one experiences. Nevertheless, very little genetic sensitivity research investigates financial outcomes.

A growing body of GxE interaction research examines effects on educational outcomes (Thompson 2014; Conley and Rauscher 2013; Shanahan et al. 2008; Keltikangas-Jarvinen et al. 2007), which have implications for income and earnings. In a related study, Cook and Fletcher (2015) use data from the Wisconsin Longitudinal Study and sibling fixed effects to investigate GxE in relation to cognition (IQ). Although education and cognition may be more directly related to income than other outcomes investigated by GxE research, thus far I have been able to find only one study that directly examines GxE effects on financial outcomes. In addition to studying whether an index of neuroplasticity genotypes moderate the relationship between birth weight and IQ, Cook and Fletcher (2015) also examine the genotype-birth weight interaction for adult wages. Predicting both IQ and wages, and even when including sibling fixed effects, Cook and Fletcher find that an index of three genotypes moderates the effect of birth weight (a measure of environment before birth). There is some evidence, therefore, that financial outcomes may depend on an interaction between genes and environment.

Despite their careful methodological approach, Cook and Fletcher (2015) note that birth weight it not randomly distributed within sibling pairs and could differ with respect to genotype, among other things. Furthermore, birth weight is only one potential environmental factor that may moderate the relationship between genes and financial outcomes. Using a similar approach, including sibling fixed effects and data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), this study investigates whether the relationship between childhood financial standing (i.e. parental household income during childhood, which is the same within sibling pairs) and young adult financial standing varies by an index of sensitive alleles. In the process, this study investigates whether intergenerational economic mobility varies by genotype within sibling pairs.

By combining two areas of inquiry, which until now have remained largely distinct, I extend research on both GxE and intergenerational mobility to pose the following hypothesis: Under the differential susceptibility model (or the BSC hypothesis), individuals with fewer sensitive alleles should demonstrate weaker association with parental measures of financial standing (higher mobility) than those with more contextually sensitive alleles. Those with more sensitive alleles should be more sensitive to parental financial standing, yielding a greater range in outcomes and stronger association with parental socioeconomic measures (lower mobility).

Methods and Analysis

Data

Add Health follows a nationally representative sample of U.S. adolescents who were in grades 7-12 in 1994-5. Revisiting over 15,000 youth in 1996, 2001-2, and 2007-8, Add Health provides a wealth of information on adolescents and their development over time. I draw on data from Waves I and IV, when the response rates were 79% and 80.3% respectively. Selective response and attrition could reduce the generalizability of results. For example, if young adults

from low income backgrounds are less likely to appear in Wave IV, results are less generalizable to those from low income backgrounds. In the full sample analyses, I use weights constructed by Add Health to adjust for unequal likelihood of appearing in the data. The weights address concern about generalizability and allow results of the full sample to generalize to the U.S. population who were in grades 7-12 in 1994-1995 (Chen and Chantala 2014).

An additional concern for this analysis is potential bias if young adults with certain combinations of genotype and parental income are less likely to provide information on their economic standing. To test for this scenario, I compare the rate of missing socioeconomic measures among young adults below the median on both parental income and genetic sensitivity score, below the median on one of those values, and above the median on both values. In most cases, the rate of missing values is not significantly different when comparing these categories. However, in a few cases the proportion with missing values is significantly different. Specifically, young adults below median parental income and genetic sensitivity scores are more likely to be missing percent of federal poverty level; those above median parental income and genetic sensitivity score are less likely to be missing household income and percent of federal poverty level. To address potential bias induced by selective nonresponse, I conduct sensitivity analyses using multiply imputed values of the outcome measures. Results (shown in Tables S4 and S5) are consistent with those from the complete case analyses presented below.

The Adolescent Pairs Data allow detailed comparisons of 3,139 pairs of twins, singleton siblings (i.e. non-twin siblings), half siblings, step siblings, or unrelated youth who lived in the same household. Full siblings are the focus of these analyses because sibling comparisons address between-family environmental and genetic differences that could bias results from general samples.

Some GxE research uses twin comparisons to address potentially important environmental differences experienced by singleton siblings (e.g., Conley and Rauscher 2013). However, twin comparisons face their own limitations, particularly in relation to external validity. For example, twins must split parental attention and resources, particularly during crucial early years of development. Twins may face greater credit constraints when attending college as parents struggle to fund simultaneous college tuition costs for twins. Parents may also treat twins differently than singleton children, emphasizing their similarities or differences or simply providing them less attention due to stress and fatigue. I aim to balance internal and external validity by examining same sex singleton (non-twin) sibling pairs, which offer more generalizability than twins but greater internal validity than a sample of non-siblings.

I limit analyses to same sex sibling pairs because one of the sensitive genotypes examined (the short MAOA allele) is on the X chromosome, of which males have one copy and females have two copies. The number of possible sensitive genotypes therefore differs by sex, which limits meaningful comparisons of the relationship between number of sensitive genotypes and financial outcomes to same sex siblings. Furthermore, gender gaps in income and earnings are substantial, making it difficult to compare financial outcomes for opposite sex sibling pairs. To address these concerns, I compare same sex siblings: brothers to brothers and sisters to sisters. Thus, the main analyses are limited to same sex, singleton, full sibling pairs with complete information for parents and both siblings.

Some families have more than one same sex sibling pair represented in the Add Health data. In those instances, I randomly select one pair of same sex, full siblings per family and exclude the others to ensure that certain families are not over-represented. Thus, each family with any same sex sibling pairs is represented only once. Although sibling comparisons

maximize internal validity and represent the preferred method here, results from the full sample are also provided to assess the importance of accounting for the non-random distribution of genes and environment in the population.

The Wave IV DNA Data file provides information about variation at multiple genetic locations among approximately 96% of the 15,701 Wave IV respondents. Add Health collected saliva samples of respondents in the field and shipped them to the Institute for Behavioral Genetics in Boulder, CO, which extracted, quantified, and genotyped the buccal cell DNA. Of particular interest for this analysis is the genotype information for DAT1, DRD2, DRD4, MAOA, and 5-HTT genes, which previous research identifies as having a sensitive (or plastic) genetic variant (see the literature review above for details).

Measures

Parental financial standing is based on Wave I data, when the children were adolescents. Parental financial standing is measured using total household income and percent of the federal poverty level (FPL). Total household income includes income from all individuals in the household and is used as the primary measure in analyses because it offers a widely understood indicator of overall financial standing and a consistent measure for both parents and children. Results, however, are similar using parental percent of the FPL. One potential concern for the sibling analysis is that parental financial standing is measured at different ages for siblings. That is, parental financial standing may change over time. Research shows that estimates based on single-year measures of parental economic standing substantially overestimate intergenerational mobility (Solon 1992; Mazumder 2005). This analysis focuses on sibling comparisons and is not attempting to estimate mobility in the population. Nevertheless, readers should keep in mind that mobility estimates are likely overestimated due to the single-year measures of economic

standing. More importantly for this analysis, parental income could differ if measured at the same age for each sibling. I partially address this by controlling for child age, but parental financial standing is measured with error due to the single-year measure.

Child financial standing as a young adult is based on Wave IV data, when children were ages 24 to 34, with an average of about 29. Therefore, results represent effects in young adulthood rather than when individuals have reached their peak earning potential. Child financial standing is measured using total household income, percent of the FPL, individual earnings, and total household net worth (assets minus debts). Total household income is measured categorically (with 12 categories) in Wave IV. This is not ideal, because it reduces variation. However, earnings and net worth measures provide alternative continuous measures with greater variation. For total household income, I take the midpoint of each category and $15,000 above the cutoff for the highest category, which includes households with income $150,000 or above.

Nearly all Wave I interviews took place in 1995 and 98% of Wave IV interviews took place in 2008. Income questions asked about total income in the last year. Therefore, parental income from Wave I measures 1994 household income and is converted to 2007 dollars (the income year recorded in Wave IV) using the Bureau of Labor Statistics Consumer Price Index Inflation Calculator.[1] Percent of the FPL is total household income divided by the appropriate federal poverty threshold for the total number of people in the household. Poverty thresholds are based on 1994 U.S. Census Bureau Poverty Guidelines for parent measures and on 2007 Health and Human Services Poverty Guidelines for child measures in young adulthood.[2]

[1] http://data.bls.gov/cgi-bin/cpicalc.pl
[2] 1994 www.census.gov/hhes/www/poverty/data/threshld/thresh94.html; 2007 http://aspe.hhs.gov/poverty/07poverty.shtml

Parental household income and child financial outcome measures are skewed (particularly earnings and net worth). I therefore conduct regression analyses using transformed versions, which provide a more normal distribution. The log transformation does not reduce (and in nearly all cases increases) the absolute value of the skewness for both parent and child measures. The square root transformation provides a more normal distribution of the data and reduces skewness better than the log transformation. I therefore use the square root transformation for household income and individual earnings measures, although results are consistent using a log transformation. In the case of household net worth, I use the inverse hyperbolic sine (IHS) transformation, which best reduces the skew while retaining information for those with negative net worth values. Results, however, are similar using the square root transformation for consistency. To ease interpretation and because it is not highly skewed, I do not transform percent of FPL, but results using a square root transformation of this measure are similar.

Genetic sensitivity score is the same as that used in Belsky and Beaver (2011) and is calculated as the sum of the total number of sensitive alleles at polymorphisms of the DAT1, DRD2, DRD4, MAOA, and 5-HTT genes. Existing research has identified a variant at each of these polymorphisms as a sensitive (also called plastic) allele that increases sensitivity to environmental context (Belsky and Pluess 2009; Belsky and Beaver 2011; see the literature review above). Specifically, these alleles include: the long (10-repeat) DAT1 allele; the long (7-repeat) DRD4 allele; the short (2- or 3-repeat) MAOA variable number tandem repeat allele; the short 5-HTT allele (14 repeats); and the DRD2 A1 (or T) allele.

The sensitive genotypes included are related to the processing of neurotransmitters that play an important role in a variety of behaviors related to economic standing (e.g., attention,

aggression, depression) (Pearson-Fuhrhop et al. 2014; McDermott et al. 2009; Cummins et al. 2012). Furthermore, experimental research suggests variation at these genes is associated with financial decision making (e.g., Carpenter et al. 2011; Dreber et al. 2009; Zhong et al. 2009a, 2009b). Because individual alleles are not likely to have strong enough effects on complex outcomes for statistical detection (Pearson-Fuhrhop et al. 2014) and to conform to the increasingly common practice of using polygenic or additive genetic measures (Mitchell et al. 2014; Pearson-Fuhrhop et al. 2013; Stice et al. 2012; Belsky and Beaver 2011), I create a genetic sensitivity score by summing all of the sensitive alleles held by an individual. Throughout the paper, I refer to this measure as genetic sensitivity score (GSS), but an alternative term is cumulative genetic plasticity score (Belsky and Beaver 2011).

Among Add Health respondents with individual and parental income data, GSS ranges from zero to nine with a mean of 3.75. In the sibling sample used for the primary analyses, GSS ranges from zero to eight with a mean of 3.89. I create alternative GSS measures, including slightly different alleles (with alternative numbers of repeats) and including alleles at additional genetic locations with less evidence of sensitivity. Results are similar and are shown in Appendix Table S6, Panels A-E. As a sensitivity analysis to check for a potential interactive relationship among the alleles, I also create a genetic interaction sensitivity score, which interacts the number of sensitive alleles at each of the genetic locations included in the GSS. Thus, the genetic interaction score is an interaction of the number of long DAT1 alleles, long DRD4 alleles, short MAOA alleles, short 5-HTT alleles, and DRD2 A1 alleles each individual carries. These results are provided in Appendix Table S7.

Analysis

To assess whether intergenerational mobility differs by genotype, I compare the strength of parent-child economic similarity by GSS. First, I conduct analyses using the whole Add Health sample (including those with and without siblings), adjusting the standard errors for family level clustering. I regress child financial outcome on GSS, parental income, and GSS interacted with parental income, controlling for age and sex, using Add Health weights to represent the population. A second model adds controls measured during adolescence including household size, having enough money for bills, birth weight, special education status, cognitive disability status, and several parental measures, including parental education, self-rated health, alcohol use, smoking, age, employment, marital status, race, ethnicity, and seatbelt use. The goal with these controls is to address between-family differences. However, parental characteristics and genotype are not randomly distributed in the general population and, even with extensive controls, the full sample may yield biased estimates due to unobserved differences.

To better address between-family differences, I use family fixed effects models to compare same sex, full siblings. I do this by creating an indicator for same sex siblings and limiting the sample to full sibling pairs who are either both male or both female. Each sibling has an equal chance of inheriting one of two alleles at each genetic locus from each parent (Fletcher and Lehrer 2011) and sibling pairs hold constant much environmental variation, including parental income. Other environmental variation is subsequent to the genes one inherits.

Comparing results from the full sample with those from the sibling analyses will illustrate whether controlling for multiple factors in the full sample can approach sibling comparison methods. By comparing results from the full and sibling samples, this study questions the extent to which results from a general sample with controls for observed differences are similar to those

from sibling fixed effects models, which account for the nonrandom distribution of genotype and other unobserved differences between families. Different results would cast doubt on GxE research that does not address the nonrandom distribution of genotype and environment.

A potential concern of sibling comparisons is that parental assortative mating could reduce genetic variance within families and reduce the power of an analysis. Siblings share, on average, about half of their genes. If the sibling GSS correlation were higher than 0.50, it would suggest assortative mating. However, in the sample of siblings used here, the sibling GSS correlation is 0.49, which suggests assortative mating is not a concern.

In the sibling sample, as shown in Equation 1, I regress child (*i*) financial outcome on GSS and GSS interacted with parental income, controlling for age and including family (*j*) fixed effects (η_j). Parental income and sex are the same for both siblings, so the main effects drop out of the model. In both the full and sibling samples, standard errors are robust to heteroscedasticity and adjusted for family-level clustering.

$$\text{Child Income}_{ij} = \alpha_0 + \beta_1 \text{GSS}_{ij} + \beta_2 \text{GSS}_{ij} * \text{Parent Income}_j + \beta_3 \text{Age}_{ij} + \eta_j + \varepsilon_{ij} \quad (1)$$

The coefficient of primary interest is β_2, which estimates whether parent-child economic association differs by individual GSS. Under the BSC hypothesis, siblings with fewer sensitive alleles should demonstrate weaker association with parental measures than their sibling with more contextually sensitive alleles. Siblings with more sensitive alleles should be more sensitive to parental SES, yielding a greater range in outcomes and stronger association with parental income. Therefore, within same-sex sibling pairs, β_2 should be positive.

To assess robustness of the findings, the main analyses include multiple measures of child financial standing as a young adult. Sensitivity analyses using alternative GSS calculations (i.e. including alternative alleles, such as the MAOA 2-, 3-, and 5-repeat alleles, the DRD4 6-

through 10-repeat alleles, or DAT 10- through 13-repeat alleles) or controlling for age squared yield similar results. I also conduct sensitivity analyses controlling for birth weight, birth order, and parental favoritism and using parental education and percent of FPL instead of income. Results of these approaches are similar and are provided in Appendix Tables S8 and S9.

To further assess robustness or range of reaction, I conduct family fixed effects regressions separately among high and low income families and I conduct analyses using categorical measures of parental income quartile. These results are presented below and are consistent with the main analyses. As a final robustness check, I conduct family fixed effects regressions separately among white and black sibling pairs (see Appendix Table S10).

Results

Descriptive Statistics

Descriptive information for the sample of same sex singleton siblings is presented in Table 1. Mean values are presented for all siblings, but also separately for siblings from households above and below the median value of parental household income ($56,000 in 2007 dollars). Descriptive information for the full sample is provided in Appendix Table S1. Within-family differences in young adult financial standing by parental household income are presented in Appendix Table S2 and Appendix Figure S1.

Table 1: Descriptive Information – Add Health Sibling Sample

	All	Std Dev	Low Parent Hh Income	High Parent Hh Income
Young Adult Financial Standing				
Household Income	$63,666.67	$38,894.81	$54,084.97	$72,716.05
% Federal Poverty Level+	379.77	249.08	313.07	441.52
Individual Earnings†	$33,981.29	$32,176.92	$30,300.26	$37,478.28
Household Net Worth‡	$54,204.79	$160,419.00	$38,566.18	$68,772.26
Genetic Sensitivity Score	3.89	1.34	3.83	3.94
Male	0.45	0.50	0.46	0.44
Age - Child	15.71	1.71	15.67	15.75
Age - Young Adult	28.55	1.75	28.57	28.54

Parent Household Income (2007 $)	$67,013.33	$60,621.72	$30,836.60	$101,180.20
Responding Parent Age	41.01	5.04	40.31	41.68
N	630		306	324
N+	624		300	324
N†	624		304	320
N‡	564		272	292

Source: Add Health Data Waves I and IV. Includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual financial standing (Wave IV), and genetic sensitivity score (Wave IV). Low and high parent household income includes those below and above median parental income ($56,000).

As Table 1 shows, after adjusting for inflation, mean parent and child household incomes are comparable, with parent income approximately $3,300 higher than child income. This difference could reflect the relatively young age at which child household income is measured (mean 29 years, 12 years younger than mean parent age). Although mean parental income differs drastically among those above and below the median household income (a gap of approximately $70,300), the difference is much smaller in the next generation (approximately $18,600). This suggests that, on average, children experienced mobility toward the mean – upward among those from low income backgrounds and downward among those from high income backgrounds.

The family fixed effects analysis relies on sibling differences in sensitive alleles. The regression results therefore rely on sibling pairs with discordant genetic sensitivity scores. Table 2 shows the frequency distribution of individuals in the sibling sample by the absolute value of the pair difference in GSS. This table shows that approximately 30% of the sibling sample does not differ in GSS. Sibling pairs with different GSS scores typically have one or two different alleles. One potential concern in the fixed effects regressions is that if the parental characteristics of concordant siblings differ from those of discordant siblings, the generalizability of results could be limited. Appendix Table S2 provides descriptive information on a variety of measures for sibling pairs with concordant and discordant GSS values. Importantly, the

difference in means between discordant and concordant sibling pairs is not significant for any of these measures, including young adult and parental measures. Although these descriptive comparisons are not conclusive, they suggest results are generalizable to all singleton siblings, rather than just those who differ in GSS.

Table 2: Frequency Distribution of the Sibling Sample by Pair GSS Difference

Absolute Value of GSS Difference	N	% of Sample
0	188	29.84
1	268	42.54
2	138	21.9
3	32	5.08
4	2	0.32
5	2	0.32
	630	100.00

Source: Add Health Data Waves I and IV. Includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual financial standing (Wave IV), and genetic sensitivity score (Wave IV). GSS = Genetic Sensitivity Score

Regression Analyses

Table 3 shows regression results for the full sample. Models 1-4 include minimal controls (age and gender); Models 5-8 include multiple controls. Across all models, neither GSS nor its interaction with parental household income is significant. The null finding in these models could indicate no relationship, but could also reflect biased estimates due to the non-random distribution of genes and environment in the full sample.

Table 3: Regressions Predicting Young Adult Financial Outcomes: Full Sample

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth	(5) √Hh Income	(6) % FPL	(7) √Earnings	(8) IHS Hh Net Worth
Genetic Sensitivity Score	1.12	2.78	-0.05	0.23	-0.79	0.44	-4.85	0.24
	(2.84)	(8.41)	(2.42)	(0.24)	(2.81)	(9.02)	(2.73)	(0.33)
Hh Income	0.24**	0.88**	0.16**	0.01	0.14**	0.56**	0.02	0.01
	(0.04)	(0.14)	(0.04)	(0.00)	(0.04)	(0.15)	(0.04)	(0.00)
GSS x √Hh Income	-0.00	-0.00	0.00	-0.00	0.00	-0.00	0.02	-0.00
	(0.01)	(0.04)	(0.01)	(0.00)	(0.01)	(0.04)	(0.01)	(0.00)
Age	2.55**	4.76*	3.31**	0.14	4.38**	8.49**	4.51**	0.17
	(0.64)	(1.93)	(0.64)	(0.08)	(0.76)	(2.35)	(0.79)	(0.10)
Male	9.03**	40.57**	38.90**	1.99**	6.66*	33.77**	41.21**	1.56**
	(2.25)	(6.93)	(2.28)	(0.28)	(2.64)	(8.26)	(2.74)	(0.35)
Highest Parental Education					2.57**	13.65**	2.38**	-0.12
					(0.75)	(2.33)	(0.71)	(0.09)
Have Enough Money for Bills					1.31	-6.86	3.47	0.98
					(4.28)	(12.83)	(4.48)	(0.55)
Parent 1 Excellent Health					7.24*	35.81**	10.39**	0.09
					(3.14)	(10.46)	(3.56)	(0.41)
Parent 1 Alcohol Frequency					1.99	9.86*	2.80*	-0.00
					(1.42)	(4.48)	(1.42)	(0.18)
Parent 1 Smokes					-5.65	-16.92	-2.22	-0.55
					(3.20)	(9.89)	(3.54)	(0.44)
Parent 1 Age					-0.44	1.01	0.27	0.05
					(0.25)	(0.76)	(0.27)	(0.03)
Parent 1 Unemployed					-4.15	-12.26	-8.69	0.38
					(7.11)	(19.16)	(6.34)	(0.85)
Parent 1 Married					8.35	7.67	8.27	0.26
					(5.37)	(14.11)	(4.99)	(0.67)
Parent 1 is Biological Mother					8.22	43.01**	13.27*	0.44
					(5.36)	(14.96)	(5.67)	(0.69)
Parent 1 White					11.71	27.26	8.34	-0.79

					(7.06)	(21.44)	(9.67)	(0.93)
Parent 1 Latino					1.13	-16.21	8.82	-0.03
					(7.06)	(19.10)	(6.28)	(0.95)
Parent 1 Always Wears Seatbelt					4.20	7.68	4.71	0.23
					(2.88)	(9.14)	(2.98)	(0.39)
Parent 2 Employed					13.04*	19.59	-4.61	1.00
					(5.74)	(19.09)	(9.16)	(0.85)
Parent 2 Unemployed					13.29	-4.16	-11.60	0.91
					(9.84)	(26.81)	(12.45)	(1.45)
Parent 2 Alcohol Frequency					1.11	5.87	0.26	0.06
					(1.00)	(3.05)	(0.93)	(0.13)
Parent 2 White					-3.11	1.27	-7.42	1.26
					(7.02)	(21.84)	(10.10)	(0.93)
Parent 2 Latino					23.93**	64.22**	6.27	1.66
					(7.12)	(20.86)	(6.58)	(0.95)
Household Size					-0.70	-6.05*	0.65	-0.28*
					(1.07)	(3.03)	(1.11)	(0.14)
Birth Weight - log ounces					13.57	42.85	17.80*	-0.14
					(8.38)	(25.29)	(8.31)	(1.06)
Special Educ Service - past year					-25.57**	-58.92**	-11.79	-0.09
					(6.19)	(19.20)	(6.31)	(0.84)
Mentally Retarded					-37.08	-60.28	-94.70**	-4.32
					(33.55)	(48.61)	(15.03)	(3.70)
Learning Disability					-15.00**	-47.18**	-21.01**	0.62
					(4.79)	(15.42)	(4.65)	(0.68)
Constant	100.15**	12.99	12.25	-3.79	-39.21	-508.51**	-138.25**	-5.99
	(21.59)	(63.54)	(21.91)	(2.45)	(50.10)	(147.37)	(46.63)	(6.18)
Observations	10,423	10,326	10,346	9,880	6,585	6,536	6,539	6,157
R-squared	0.08	0.11	0.10	0.02	0.11	0.14	0.13	0.02

Source: Add Health Data Waves I and IV. Full sample includes all individuals with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV) and is weighted to represent the population. GSS = Genetic Sensitivity Score

Robust standard errors in parentheses, adjusted for family level clustering. * p<0.05, ** p<0.01

In Model 1, the coefficient for square root of parental household income suggests that children gain approximately 6 percent (0.24 x 0.24 = 0.06) of each additional dollar of parental income. An additional $10,000 in parental household income is associated with an additional $600 in predicted income for the child as a young adult. In Model 3 predicting individual earnings, the coefficient for parental household income suggests that children gain approximately two and a half percent (0.16 x 0.16 = 0.026) of each additional dollar of parental income. Thus, an additional $10,000 in parental household income is associated with an additional $260 of predicted child earnings as a young adult.

Table 4 shows results of models limited to the sibling sample and including family fixed effects. The main effect of GSS is negative, but the interaction between GSS and parental household income is positive and significantly different from zero when predicting income, percent of FPL, and earnings (i.e. all outcome measures except household net worth). Siblings with a higher GSS tend to have lower financial outcomes than their sibling with a lower GSS, but only in families from low income backgrounds. In each case except net worth, the predicted financial outcome for those with a higher GSS is higher among high income families. In Model 1, for example, each additional sensitive allele is associated with a higher adult household income when parental household income is approximately $70,000 or higher. In Model 2, the crossover point is approximately $50,000, and in Model 3 it is approximately $55,000. These transition points are generally centered around the overall mean ($67,000 in 2007 dollars) and the median ($56,000) of parental household income.

Table 4: Regressions Predicting Young Adult Financial Outcomes: Family Fixed Effects

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth
Genetic Sensitivity Score	-27.71**	-74.58**	-23.80*	1.50

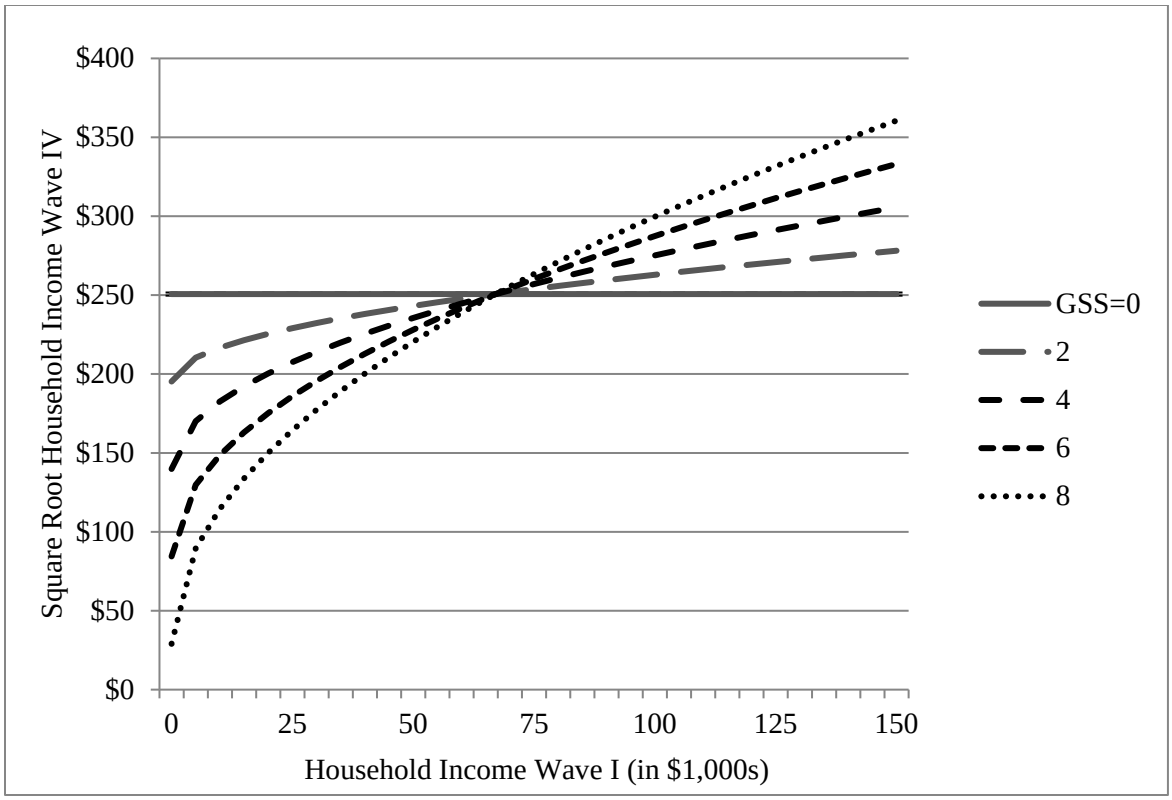
	(8.72)	(25.19)	(10.63)	(1.37)
Genetic Sensitivity Score x √Hh Income	0.11**	0.35**	0.10*	-0.00
	(0.03)	(0.10)	(0.04)	(0.01)
Age	8.17**	12.93*	8.15**	0.24
	(1.91)	(6.04)	(1.86)	(0.31)
Constant	13.63	-27.68	-71.05	-4.82
	(56.76)	(179.92)	(56.41)	(9.17)
Observations	630	624	624	564
R-squared	0.08	0.04	0.07	0.01
Number of Sibling Pairs	315	312	312	282

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering. * $p<0.05$, ** $p<0.01$

Figure 1 illustrates the relationship between parent and child household income by number of sensitive alleles. The figure clearly illustrates that, compared to their same sex sibling with fewer sensitive alleles, a sibling with more sensitive alleles tends to have lower household income in young adulthood if raised in a low income household, but higher household income if raised in a high income household. Based on Model 1 in Table 4, among those with parental income near zero, each additional sensitive allele results in approximately $770 less in annual household income. Among siblings with parental income near $150,000, in contrast, each additional sensitive allele is estimated to result in approximately $200 in additional annual household income.

Figure 1: Predicted Relationship between Parent and Child Household Income by Genetic Sensitivity Score: Sibling Fixed Effects Model, $p<0.01$



Source: Add Health Data Waves I and IV; based on Table 4, Model 1. GSS = Genetic Sensitivity Score
Compared to one's same sex full sibling, income as a young adult depends more strongly on parental income among those with a higher number of sensitive alleles. Children with a higher GSS fare more poorly than their sibling if they grew up in a lower income household, but fare better than their sibling if they grew up in a higher income household.

This pattern is similar when predicting percent of FPL and individual earnings. For those with parental income near zero, each additional sensitive allele is estimated to reduce one's standing on the federal poverty scale by 75 percentage points. For those whose parental household income was $150,000, each additional sensitive allele is estimated to increase one's standing by 60 percentage points. Based on Model 3 in Table 4, those with parental income near zero are estimated to earn approximately $570 less per year for each additional sensitive allele, while those with parental income of $150,000 are estimated to earn an additional $250 per year for each additional sensitive allele.

In Table 4 Model 1, the coefficient for the interaction between GSS and square root of parental household income suggests young adults with one sensitive allele are predicted to gain

an additional one percent (0.11 x 0.11 = 0.01) of every dollar of parental income. In other words, young adult household income is predicted to increase by about $100 if parental income increases by $10,000. However, this return to parental income increases with by the square of the number of sensitive alleles. Thus, those with two sensitive alleles are predicted to gain about five percent (0.11 x 0.11 x 2 x 2 = 0.05) of every dollar of parental income. For young adults with two sensitive alleles, their household income is predicted to increase by about $500 if parental income increases by $10,000. The return to parental income increases to 19 percent (0.11 x 0.11 x 4 x 4 = 0.19) for those with four sensitive alleles.

The null result for household net worth is intriguing. Net worth could be more strongly determined by factors beyond individual differences, such as parental net worth or financial bequests, than household income or individual earnings (see e.g., Black et al. 2015). The alleles included in the GSS have been implicated in behaviors (e.g., attention, aggression, depression) associated with labor market outcomes (Pearson-Fuhrhop et al. 2014; McDermott et al. 2009; Cummins et al. 2012). It is possible, therefore, that the increased sensitivity to context works through behaviors. In low income contexts, the sensitive alleles could encourage behaviors associated with lower earnings and income. In high income contexts, the same alleles could encourage behaviors associated with higher earnings and income. In such a scenario, these alleles could moderate labor market outcomes such as income and earnings, but remain largely unrelated to net worth. Another possible explanation is that most young adults have relatively low wealth around age 30 (Land and Russell 1996; Cagetti 2003). More time may be required for GxE effects on wealth to emerge. Results could be consistent with either pattern, but explaining the different results for net worth are beyond the scope of this paper. Future research

should investigate potential interaction effects on wealth in later waves of Add Health, when respondents are older.

Sensitivity Analyses

The above results suggest the relationship between GSS and individual financial outcomes should differ depending on parental financial standing as an adolescent. As a robustness check of this interaction, I fit separate regressions limited to sibling pairs from high and low income households. Results (discussed and presented in the Appendix, Table S11) are consistent with the main analyses. However, this approach relies on smaller sample sizes. In Table 5, I provide results from a more flexible model – an additional robustness check using categorical measures of parental household income. Specifically, I create measures of parental income quartile. Using the same regression model as that presented in the main analyses, I replace parental income with these categorical measures and their interaction with GSS. Results of this analysis are consistent with those presented above. Siblings with a higher GSS score have significantly lower household income than their sibling with a lower score, but only among siblings from the first parental income quartile ($p<0.05$). In each case except net worth, GSS predicts significantly higher financial outcomes among those from the highest parental income quartile ($p<0.05$).

Figure 2 illustrates the relationship between and GSS and young adult financial outcomes by parental income quartile. The coefficients roughly follow a linear pattern, but typically only reach significance among the highest income quartile. This pattern directly contradicts early conceptions of these genotypes as conferring risk. Rather, this pattern of significant positive effects for those from the top of the income distribution support the biological sensitivity to context hypothesis (Boyce and Ellis 2005; Ellis and Boyce 2008; Belsky 2013, 2005).

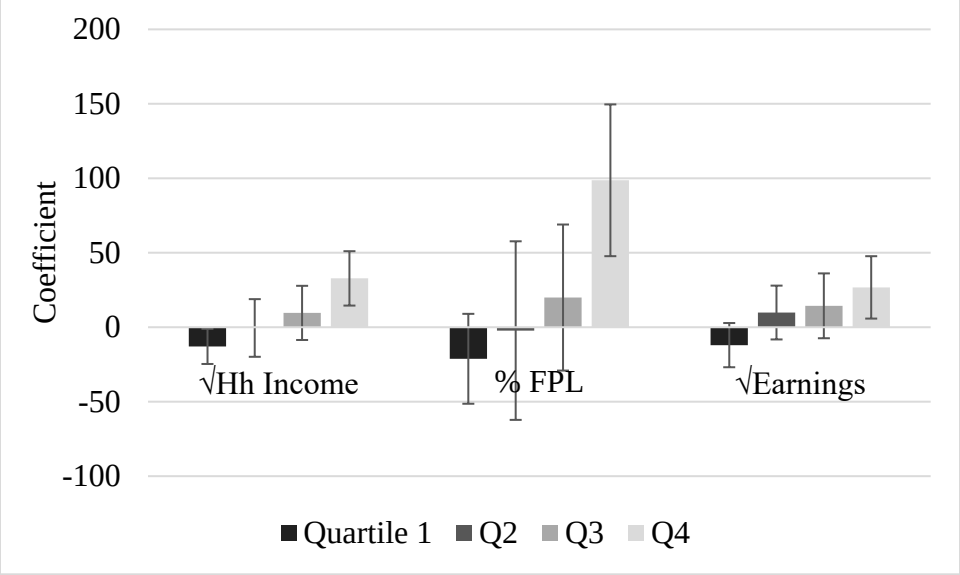
Table 5: Regressions Predicting Young Adult Financial Outcomes: Family Fixed Effects

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth
Genetic Sensitivity Score	-12.88*	-21.19	-12.02	-0.11
	(6.01)	(15.40)	(7.57)	(0.99)
Hh Income Quartile 2 x GSS	-0.49	-2.25	9.87	1.62
	(9.88)	(30.59)	(9.22)	(1.48)
Hh Income Quartile 3 x GSS	9.63	19.94	14.38	2.14
	(9.28)	(25.02)	(11.11)	(1.45)
Hh Income Quartile 4 x GSS	32.80**	98.67**	26.76*	-1.44
	(9.31)	(25.99)	(10.69)	(1.77)
Age	8.09**	12.59*	8.26**	0.26
	(1.92)	(5.95)	(1.86)	(0.31)
Constant	16.60	-14.84	-74.09	-5.87
	(57.42)	(178.73)	(56.35)	(9.10)
Observations	630	624	624	564
R-squared	0.10	0.06	0.07	0.02
Number of Sibling Pairs	315	312	312	282

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering. * p<0.05, ** p<0.01

Figure 2: Predicted Relationship between Genetic Sensitivity Score and Financial Outcomes by Quartiles of Parental Household Income: Sibling Fixed Effects Model

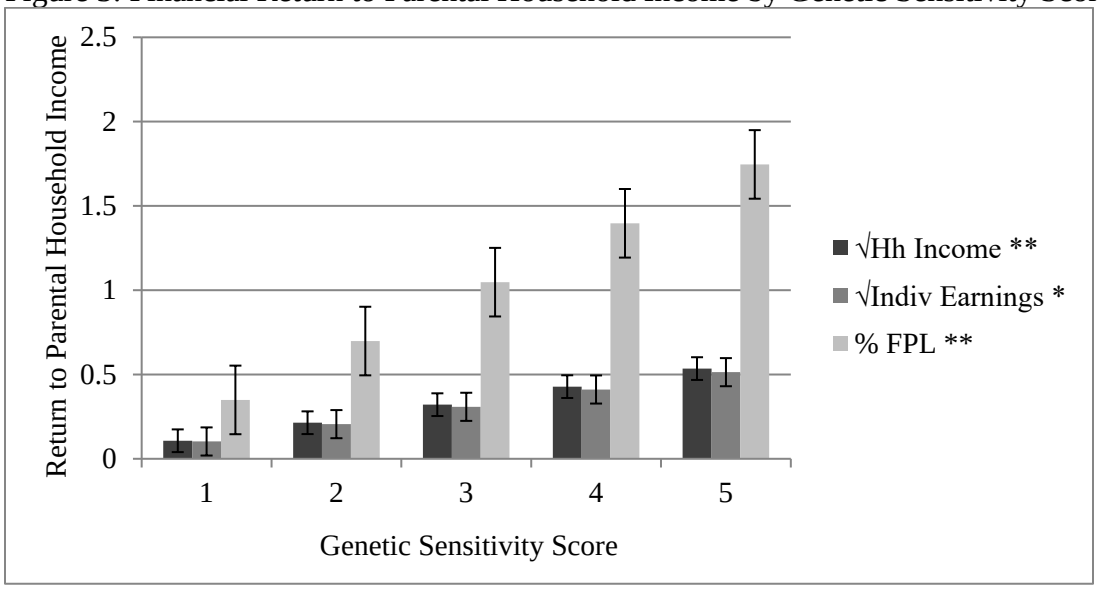


Source: Add Health Data Waves I and IV; based on Table 5, Models 1-3.

Children with higher GSS fare more poorly than their same sex full sibling if they grew up in a lower income household (Quartile 1), but fare better than their sibling if they grew up in a higher income household (Quartile 4).

In terms of intergenerational mobility, the above results suggest that individuals with a high GSS experience less financial mobility; that is, they have financial outcomes more similar to their parents than their siblings with fewer sensitive alleles. The positive interaction terms (in Table 4) between GSS and parental financial standing imply that each sensitive allele provides a greater return on parental financial standing. GSS therefore confers both greater sensitivity to adolescent financial context and less financial mobility. This positive relationship between GSS and intergenerational financial similarity is illustrated in Figure 3.

Figure 3: Financial Return to Parental Household Income by Genetic Sensitivity Score



Source: Add Health Data Waves I and IV; based on Table 4, Models 1-3.
Compared to one's same sex full sibling, financial standing as an adult depends more strongly on parental income among those with a higher number of sensitive alleles.

Solon (1992) estimated that intergenerational income elasticity (individual income return to parental income) in the U.S. is at least 0.40. This suggests that if a child's parental income was $20,000 less than the mean, the predicted child income would be $8,000 (or 40% of that difference) less than the child mean. Based on Model 1 in Table 4, each additional sensitive allele is estimated to increase the return to parental income by (0.11 x 0.11) 0.01 multiplied by

the square of the number of sensitive alleles. Those with two sensitive alleles are predicted to gain about five percent (0.11 x 0.11 x 2 x 2 = 0.05) of every additional dollar of parental income. This return increases to 19 percent (0.11 x 0.11 x 4 x 4 = 0.19) for those with four sensitive alleles. Income in this study is measured with considerably more bias than the multi-year measures Solon uses. Furthermore, I use a square root rather than a log transformation. To the extent results are comparable, however, carrying two additional sensitive alleles is estimated to confer an additional 13% (0.05/0.40) of Solon's estimated overall intergenerational income elasticity.

Sensitivity analyses using alternative GSS calculations yield similar results (presented in Appendix Table S6, Panels A-E). Results are also similar when controlling for age squared or weighting by average family weight to adjust for unequal likelihood of families appearing in the sample. In addition, results are consistent using parental education or percent of the FPL instead of income (Appendix Table S9).

Finally, results are not driven by white or black young adults alone; results for models predicting household income and percent FPL are similar when limited to either white or black sibling pairs (see Appendix Table S10). Interestingly, one difference is that the coefficient for the interaction between GSS and parental income is significant when predicting earnings among white but not black young adults. However, the opposite is true when predicting net worth. A higher GSS predicts greater net worth among black young adults from higher parental income backgrounds. The different findings for earnings and net worth between white and black young adults could suggest the interaction works through different mechanisms in these groups. For example, if discrimination limits the earning potential of young black people regardless of behaviors (Pager 2003), then GSS and its interaction may have limited potential to impact their

individual earnings. Similarly, the net worth of white young adults may depend largely on parental financial assistance (Nam et al. 2015), whereas behaviors and health may play a larger role in predicting net worth among black young adults. These potential explanations are speculative at this point and require further research.

In a further attempt to understand the mechanisms involved, I use sibling fixed effects models to investigate whether GSS predicts several parenting measures or whether the GSS-parenting relationship differs at low and high incomes. Specifically, I predict the following parent-child relationship measures: whether the child reported feeling close to their mother or father; whether the child reported that their mother or father cares; child-reported parental favoritism (a scale ranging from -2 indicating a child received a lot less love and attention from parents compared to their sibling, to 2 indicating she received a lot more); and an index of the number of activities each child reported participating in with both or either parent in the past four weeks (shopping; playing a sport; attending a religious service or church-related event; going to a movie, play, museum, concert, or sports event; working on a school project; talking about dating or a party; talking about a personal problem; talking about school work or grades; or talking about other school topics). Using the same model as Table 4, with controls for age and parental income as well as family fixed effects, I find that GSS does not significantly predict any of the parent-child relationship measures whether including the full sibling sample or when limiting the sample to siblings above or below median parental income. The null results suggest that parents do not treat their children differently by GSS and that the parenting-GSS relationship does not differ by income. These analyses are neither exhaustive nor conclusive, but the results suggest that the interaction effects between GSS and parental income (found in Tables 4 and 5) do not reflect different treatment by parents. Instead, the interaction could reflect different

treatment or interpretation of gene-related behaviors by teachers and other members of society depending on income; or it could reflect differences in the likelihood of certain income-related health conditions by genotype and environment. These are only two potential mechanisms and conclusively identifying mechanisms is beyond the scope of this paper.

Conclusion

This study investigates the relationship between an index of sensitive genotypes and intergenerational mobility, while taking methodological steps to address the non-random distribution of genotype and environment. Expanding beyond the outcomes typically examined in GxE research, this study examines financial outcomes, including household income, percent of the federal poverty level, individual earnings, and household net worth. Overall, results provide further support for the differential susceptibility model, which suggests that genetic variants previously considered to increase risk actually increase sensitivity to environmental context. Compared to their same sex sibling, individuals with more copies of sensitive genotypes do worse in low income contexts but fare better in high income contexts. These results add to the growing body of evidence suggesting the genetic variants in question increase sensitivity to context rather than risk (Belsky and Pluess 2009).

Results suggest the implications of sensitive alleles may extend further than previously thought. Beyond health and behavioral consequences, for example, results suggest young adult financial standing depends significantly on genotype as well as parental income. Contrary to traditional genetic arguments for intergenerational inequality, however, analysis of Add Health sibling data suggests the genotypes examined are not inherently positive or negative, but relate to environmental sensitivity, making some children more or less sensitive to their parents' income.

This study has a number of limitations. First, it does not identify the mechanisms involved in the GxE interaction. Health and behaviors that previous research suggests are both sensitive to genotype and environment and are associated with financial standing offer potential mechanisms; for example, ADHD depends on genotype and environment (Retz et al. 2008) and has negative effects on income (Fletcher 2014). The potential mediating role of health and behaviors is partially supported here because results generally do not hold when predicting net worth, which may be more strongly determined by other factors (Black et al. 2015). Furthermore, models predicting parenting measures suggest the GxE interaction effects do not reflect different treatment by parents.

Although identification of mechanisms is beyond the scope of this paper, the observed interaction could occur through a combination of triggering and enhancement (two types of GxE identified by Shanahan and Hofer 2005) and neurobiological responses to stress (Boyce 2012). For example, children with a sensitive genotype in a low income context could develop unhealthy responses to stress, triggered by environmental inputs such as parental stress, housing insecurity, or low quality schools. In contrast, children with the same sensitive genotype in a high income context could develop healthy responses to stress through enhanced environmental inputs, such as quality time with loving parents who teach children to rise to challenge. While this is just one possible example, it suggests one way in which sensitive genotypes could express themselves differently depending on context, promoting behaviors that are either detrimental to or supportive of economic advancement. Further research is required to identify mechanisms involved in genetic sensitivity – to parental income and more generally.

A second limitation is measurement of financial standing. Parental income is measured in a single year and child financial outcomes are measured at a relatively young age, which may

substantially underestimate intergenerational mobility (Mazumder 2005; Solon 1992). The emphasis here is on internal rather than external validity, but readers should keep in mind that results do not represent lifetime mobility and estimates of intergenerational economic similarity are likely much lower than estimates based on lifetime income or earnings.

A third potential limitation is that the analytical approach relies on the stable unit treatment value assumption (SUTVA). In this case, the assumption is that the genotype of one sibling does not impact the outcome of the other sibling. Unfortunately, sibling genotype partly proxies for parental genotype, which could influence individual outcomes. Partially mitigating this concern is the fact that parental genotype and financial standing are the same for each sibling. Nevertheless, ideally I would control for parental genotype, but it is unavailable in Add Health as well as in most other data sets with financial standing and genetic information. Future research should investigate similar interaction effects when controlling for parental genotype or investigate how such interaction effects might change when relaxing SUTVA.

Finally, family fixed effects regressions rely on siblings with different GSS scores. Therefore, if families with genetically concordant siblings differ from those with discordant siblings, the generalizability of the results could be limited. To address this concern, Appendix Table S2 compares genetically concordant and discordant sibling pairs along a wide variety of measures and finds no significant differences. Although descriptive comparisons are not conclusive, they suggest results are generalizable beyond genetically divergent siblings.

Related to the above limitation, results differ in the sibling and full samples. The different findings could reflect bias in the full sample. However, the difference could also reflect different patterns that limit generalizability of the results to within families. For example, family dynamics could amplify slight differences between siblings and, if this pattern does not occur

outside the family, result in a context-dependent form of GxE. If that is the case, results could be limited to the family context and not generalizable to other contexts. However, standard deviations of young adult household income, % FPL, and earnings are higher among genetically discordant than concordant siblings, which is consistent with the possibility that results do not merely reflect the family context.

Even if results are only generalizable to the family context, they still have implications for research on gene-environment interactions and intergenerational inequality. As typically used in GxE research, the term plastic suggests a high degree of malleability. In the context of intergenerational mobility, however, sensitivity or plasticity also entails greater dependence on social background. Results suggest that siblings with more sensitive genotypes are more dependent on parental financial standing and enjoy less intergenerational mobility. The positive interaction terms between GSS and parental income indicate sensitivity to context, but also a stronger relationship between parent and child financial standing (i.e. higher intergenerational elasticity and lower mobility). Thus, plasticity entails sensitivity, but also less mobility.

Methodologically, the evidence suggests that results can differ when looking between or within families. Comparing sibling analyses to those of the full Add Health sample suggests that this GxE pattern could be context-dependent or that between-family differences could bias results in the full sample, even when including a vast array of control measures. One possible explanation for the null findings in the full sample is genes (e.g., Plomin et al. 2016; Avinun and Knafo 2014; Trzaskowski et al. 2014; Clark 2014). Another possible explanation is that between-family differences in environment swamp any potential genetic interaction or main effects in the full sample. With its wide-reaching implications for school quality, early environment, health, and opportunities for social and cultural capital development, among other

things, parental income could matter far more for child outcomes than the sensitive genotypes included in this analysis. Alternatively, any potential impact of genotype in the full sample may only be swamped among young adults from low-income backgrounds (Guo and Stearns 2002; Turkheimer et al. 2003), but that pattern could still account for the null interaction in the full sample. Because the full sample does not account for the non-random distribution of genes or environment, this study cannot identify what explains the different results. Further research could investigate this question by examining how results change when taking methodological steps to address non-random distribution of either genes or environment. Critically, however, this study suggests that results of GxE research can differ when addressing genetic and environmental differences between families.

Both recent and classic arguments suggest genes simply mediate the intergenerational transmission of inequality (Clark 2014; Herrnstein and Murray 1994). These simple genetic explanations suggest the importance of genotype for adult economic outcomes should be unrelated to economic background. Results of this study contradict such arguments and suggest the implications of certain genotypes depend on context, promoting positive outcomes at high incomes and negative outcomes at low incomes. That is, sensitive genotypes appear to moderate rather than simply mediate intergenerational transmission. The evidence suggests financial standing depends significantly on genotype as well as parental income, but the genotypes examined are not inherently positive or negative. Rather, they relate to environmental sensitivity, making some children more or less sensitive to their parents' income. For example, results suggest that a child does not achieve higher socioeconomic outcomes than his brother because he has better genes. Rather, a child with more sensitive genotypes may be more likely

to achieve higher outcomes than his brother if his parents are high income, but lower economic outcomes if his parents are low income.

If replicated, this finding has implications for research on intergenerational mobility and could help explain the wide sibling variation in socioeconomic outcomes (Conley 2004; Plomin and Daniels 2011). Some societies may reduce the importance of sensitive genotypes for mobility more than others. Comparing estimates of mobility in children from high and low income households in multiple countries could allow informative cross-national comparisons of intergenerational mobility. If sibling genetic differences are amplified at high and low family income values, rising inequality could yield widening gaps in socioeconomic attainment within siblings and further divide families.

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Appendix
Supplementary Tables to Be Included in an Online Supplemental Document

Table S1: Descriptive Information – Add Health Full Sample

	All	Std Dev	Low Parent Hh Income	High Parent Hh Income
Young Adult Financial Standing				
Household Income	$61,920.10	39629.40	$52,285.09	$71,476.18
% Federal Poverty Level+	380.77	259.25	306.02	454.11
Individual Earnings†	$34,535.16	42462.24	$29,059.28	$39,945.74
Household Net Worth‡	$51,583.06	152240.40	$37,065.64	$65,971.10
Genetic Sensitivity Score	3.75	1.44	3.80	3.70
Male	0.51	0.50	0.50	0.52
Age - Child	15.32	1.79	15.27	15.37
Age - Young Adult	28.22	1.82	28.20	28.23
Parent Household Income (2007 $)	$64,382.40	65444.80	$29,661.44	$98,818.88
Highest Parental Education	13.48	2.28	12.47	14.47
Received AFDC Last Month	0.07	0.26	0.13	0.01
Have Enough Money for Bills	0.82	0.38	0.72	0.93
Received Food Stamps Last Month	0.12	0.33	0.24	0.01
Parent 1 Excellent Health	0.22	0.42	0.16	0.29
Parent 1 Alcohol Frequency	1.03	1.18	0.84	1.21
Parent 1 Smokes	0.31	0.46	0.40	0.22
Parent 1 Age	41.35	6.38	40.63	42.08
Parent 1 Works	0.75	0.43	0.69	0.81
Parent 1 Unemployed	0.05	0.22	0.08	0.02
Parent 1 Out of Labor Force	0.20	0.40	0.23	0.17
Parent 1 Married	0.72	0.45	0.56	0.89
Parent 1 Is Biological Mother	0.90	0.30	0.89	0.90
Parent 1 Always Wears Seatbelt	0.64	0.48	0.58	0.71
Parent 1 White	0.79	0.41	0.70	0.87
Parent 1 Black	0.14	0.35	0.21	0.07
Parent 1 Native American	0.03	0.16	0.04	0.02
Parent 1 Asian	0.03	0.17	0.03	0.03
Parent 1 Other Race	0.04	0.19	0.05	0.02
Parent 1 Latino	0.09	0.29	0.14	0.05
Parent 2 Works	0.91	0.28	0.84	0.96
Parent 2 Unemployed	0.02	0.14	0.04	0.01
Parent 2 Out of Labor Force	0.07	0.25	0.13	0.03
Parent 2 White	0.82	0.38	0.74	0.88
Parent 2 Black	0.11	0.31	0.16	0.07

Parent 2 Native American	0.02	0.14	0.03	0.02
Parent 2 Asian	0.03	0.17	0.03	0.03
Parent 2 Other	0.04	0.20	0.06	0.03
Parent 2 Latino	0.09	0.28	0.14	0.04
Parent 2 Alcohol Frequency	1.63	1.58	1.40	1.79
Household Size	4.48	1.51	4.53	4.44
Birth Weight (ounces)	119.54	19.33	117.31	121.66
Special Education Service (past year)	0.09	0.29	0.12	0.07
Mentally Retarded	0.01	0.09	0.01	0.00
Learning Disability	0.13	0.33	0.15	0.10
N	10423		5139	5284
N+	10326		5077	5249
N†	10346		5092	5254
N‡	9880		4873	5007

Source: Add Health Data Waves I and IV. Includes all individuals with complete information about parental income (Wave I), individual financial standing (Wave IV), and genetic sensitivity score (Wave IV), weighted to represent the population.

Low and high parent household income includes those below and above median parental income ($56,000).

Individuals can indicate more than one race category, so categories are not mutually exclusive.

Table S2: Characteristics of Sibling Pairs by Similarity of Genetic Sensitivity Score

	Discordant	Concordant	Difference
Young Adult Financial Standing			
Household Income	64287.33	70744.68	-6457.35
% Federal Poverty Level [a]	380.38	424.37	-43.99
Individual Earnings [b]	35980.73	35659.99	320.74
Household Net Worth [c]	57409.95	65668.54	-8258.59
Young Adult Measures			
Genetic Sensitivity Score	3.87	4.01	-0.14
Male	0.43	0.48	-0.04
Age - Child	15.59	15.88	-0.29
Age - Young Adult	28.45	28.72	-0.27
Parental Household Measures			
Parent Household Income (2007 $)	67066.97	66887.23	179.74
Parental Education - Maximum [a]	13.75	13.62	0.14
Parental Education - Average [a]	13.20	13.17	0.03
Parent % Federal Poverty Level	271.84	278.00	-6.16
Poverty	0.17	0.13	0.04
Near Poverty	0.46	0.46	0.00
Welfare Receipt [a]	0.08	0.06	0.02
AFDC Receipt	0.05	0.03	0.02
Enough Money for Bills [d]	0.79	0.86	-0.07
Foodstamps [e]	0.10	0.09	0.02
Household Size	5.08	5.19	-0.11
Responding Parent Measures			
Excellent Health [b]	0.19	0.22	-0.02
Alcohol Frequency	0.89	0.83	0.06
Smokes	0.22	0.27	-0.04
Age [a]	40.94	41.18	-0.24
Works [a]	0.75	0.76	-0.01
Unemployed [b]	0.05	0.06	-0.01
Out of the Labor Force	0.20	0.18	0.02
Married [a]	0.78	0.83	-0.05
Is Biological Mother [f]	0.94	0.96	-0.02
Always Wears Seatbelt [a]	0.67	0.66	0.01
White [a]	0.78	0.76	0.02
Black [a]	0.14	0.13	0.01
Native American [a]	0.02	0.02	0.00
Asian [a]	0.04	0.09	-0.05
Other Race [a]	0.05	0.03	0.01
Latino	0.11	0.09	0.03

Other Parent Measures			
Works [g]	0.92	0.92	0.00
Unemployed [g]	0.01	0.00	0.01
Out of the Labor Force [g]	0.06	0.08	-0.01
White [g]	0.78	0.76	0.02
Black [g]	0.11	0.09	0.02
Native American [g]	0.01	0.00	0.01
Asian [g]	0.05	0.11	-0.06
Other Race [g]	0.06	0.05	0.01
Latino [h]	0.09	0.09	0.00
Alcohol Frequency [i]	1.48	1.67	-0.19
Parent Reported Child Measures			
Birth Weight (ounces) [j]	119.42	117.18	2.24
Special Education Service - past year [k]	0.08	0.09	-0.01
Mentally Retarded [l]	0.00	0.00	0.00
Learning Disability [m]	0.10	0.16	-0.05
N - Number of Pairs	221		94
N a	220		94
N b	221		93
N c	211		89
N d	217		91
N e	220		93
N f	215		91
N g	172		76
N h	169		76
N i	171		73
N j	198		79
N k	213		91
N l	214		91
N m	212		90

Source: Add Health Data Waves I and IV. Includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

No differences between means of discordant and concordant sibling pairs are significant (at $p<0.05$ or $p<0.10$, two-tailed t-test).

Within-Family Descriptive Differences by Parental Income

To provide within-family descriptive information, I calculate sibling differences in financial outcomes. Within each sibling pair, these differences represent the financial outcome value (e.g., young adult household income) for the sibling with the higher GSS minus the value for the sibling with the lower GSS. Siblings with the same GSS are excluded from this descriptive analysis. Because individuals with more sensitive alleles are expected to fare worse in disadvantaged environments and better in supportive environments, the differential susceptibility model predicts significant negative differences among low income families, but significant positive differences among high income families.

Mean sibling differences by parental household income are presented in Table S3. For each financial outcome measure, mean sibling difference is negative among low income households and positive among high income households. For all of the measures, the difference between low and high income households is significant ($p<0.05$). Figure S1 illustrates these differences and shows that sibling difference is positive and significantly different from zero among high income households for household income and percent of the federal poverty level. Among low income households, sibling difference significantly differs from zero in the negative direction for all measures except household net worth.

These mean differences are consistent with the differential susceptibility model. The sibling with higher GSS tends to have lower financial outcomes in low income families, but higher financial outcomes in high income families. These differences, however, do not hold for household net worth.

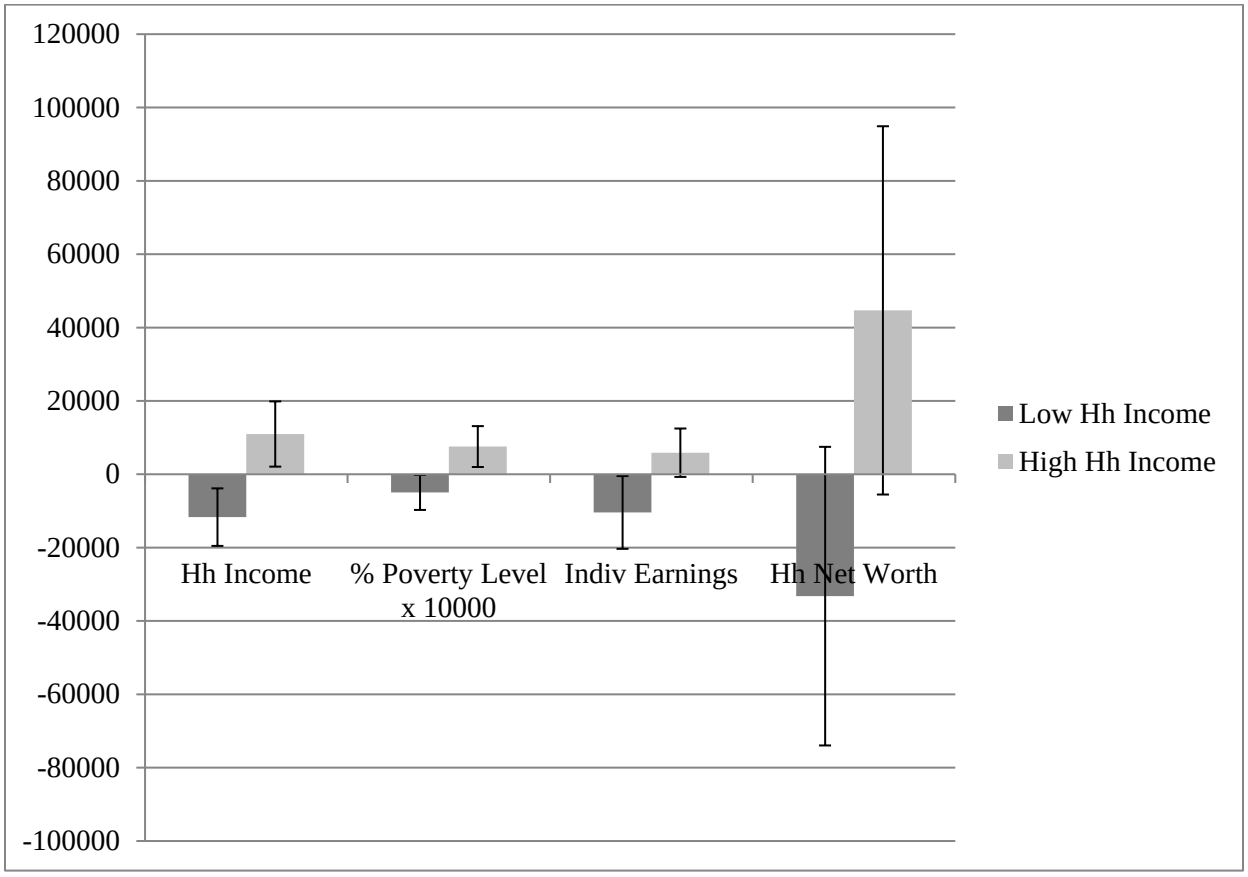
Table S3: Mean Sibling Pair Differences in Adult Financial Standing by Parental Household Income – Sibling with Higher GSS Minus Sibling with Lower GSS

	Low Parent Hh Income	N (Pairs)	High Parent Hh Income	N (Pairs)	T-Test of Difference
Hh Income	-11689.19	111	10977.27	110	**
% Poverty Level x 100	-49.56	110	75.55	110	**
Indiv Earnings	-10414.05	111	5875.45	109	**
Hh Net Worth	-33233.01	103	44683.67	98	*

Source: Add Health Data Waves I and IV; same sex singleton full sibling pairs with complete information, who differ in GSS. GSS = Genetic Sensitivity Score

Sibling pair differences are calculated as the financial outcome value for the sibling with the higher GSS minus the value for the sibling with the lower GSS. * $p<0.05$; ** $p<0.01$ (two-tailed t-test)

Figure S1: Mean Sibling Pair Differences in Adult Financial Standing by Parental Household Income



Source: Add Health Data Waves I and IV; same sex singleton full sibling pairs with complete information, who differ in Genetic Sensitivity Score (GSS).

Sibling pair differences are calculated as the financial outcome value for the sibling with the higher GSS minus the value for the sibling with the lower GSS.

Multiple Imputation Analysis

Out of concern about potential bias due to selective response, I conducted sensitivity analyses using multiply imputed measures of young adult socioeconomic standing. Multiple imputation was conducted in Stata using a multivariate normal distribution because the imputed variables approach a normal distribution and are not binary, categorical, or count data (in which case chained equation estimation would be preferable). The proportion of missing data in the sibling sample ranges from 1.7% for individual earnings to 10.6% for net worth. In the full, non-sibling sample, the proportion missing ranges from 2.3% for earnings to 12.1% for net worth. I created 10 multiply imputed data sets, using parental household income, parent age, child genetic sensitivity score, gender, and age (in Waves I and IV) to predict child socioeconomic outcome measures (in Wave IV). Results, shown in Tables S4 and S5, are consistent with those using complete case analysis. In one case, when predicting individual earnings, the interaction between GSS and household income does not reach significance in the multiple imputation estimates while it does in the complete case analyses. However, the individual earnings variable has the lowest rate of missing information, so the complete case analyses may be more valid than the multiple imputation results, given the inaccuracy of predicting missing earnings values.

Table S4: Multiple Imputation Regressions Predicting Young Adult Financial Outcomes: Family Fixed Effects

	1	2	3	4
VARIABLES	√Hh Income	% FPL	√Earnings	IHS Hh Net Worth
Genetic Sensitivity Score	-22.06*	-57.17	-17.19	1.23
	(10.20)	(32.37)	(13.09)	(1.46)
Genetic Sensitivity Score x √Hh Income	0.08*	0.27*	0.07	-0.00
	(0.04)	(0.13)	(0.05)	(0.01)
Age	6.34**	7.67	5.81**	0.50
	(2.01)	(6.28)	(2.16)	(0.29)
Constant	68.12	123.52	-3.92	-12.02
	(59.46)	(183.68)	(65.23)	(8.63)
Observations	704	704	704	704
Average Relative Variance Increase	0.06	0.08	0.02	0.14
Largest Fraction of Missing Information	0.11	0.16	0.05	0.27
Number of Sibling Pairs	352	352	352	352

Source: Add Health Data Waves I and IV. Ten multiply imputed data sets of the sibling sample, which includes same sex singleton full sibling pairs with complete information about parental income (Wave I) and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering. * $p<0.05$, ** $p<0.01$

Table S5: Multiple Imputation Regressions Predicting Young Adult Financial Outcomes: Full Sample

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth	(5) √Hh Income	(6) % FPL	(7) √Earnings	(8) IHS Hh Net Worth
Genetic Sensitivity Score	1.15	2.06	-0.01	0.22	-0.86	-0.28	-4.86	0.28
	(2.76)	(8.24)	(2.32)	(0.24)	(2.77)	(8.73)	(2.67)	(0.33)
√Hh Income	0.24**	0.91**	0.17**	0.01*	0.14**	0.58**	0.03	0.01
	(0.04)	(0.14)	(0.04)	(0.00)	(0.04)	(0.15)	(0.04)	(0.01)
GSS x √Hh Income	-0.00	-0.01	0.00	-0.00	0.00	-0.00	0.02	-0.00
	(0.01)	(0.03)	(0.01)	(0.00)	(0.01)	(0.04)	(0.01)	(0.00)
Age	2.40**	4.49*	3.06**	0.11	4.29**	8.36**	4.50**	0.18
	(0.65)	(1.98)	(0.64)	(0.08)	(0.80)	(2.40)	(0.80)	(0.10)
Male	8.85**	35.49**	38.01**	1.96**	7.00*	31.90**	40.75**	1.61**
	(2.31)	(7.11)	(2.27)	(0.28)	(2.74)	(8.53)	(2.71)	(0.35)
Constant	102.51**	10.77	15.29	-3.16	-32.02	-477.73**	-121.53**	-6.29
	(21.36)	(64.15)	(21.39)	(2.45)	(51.95)	(156.81)	(46.29)	(6.45)
Observations	11,124	11,124	11,126	11,118	6,995	6,995	6,995	6,993
Average Relative Variance Increase	0.07	0.08	0.03	0.13	0.13	0.17	0.03	0.15
Largest Fraction of Missing Information	0.11	0.09	0.05	0.12	0.40	0.61	0.18	0.38

Source: Add Health Data Waves I and IV. Ten multiply imputed data sets of the full sample, which includes all individuals with complete information about parental income (Wave I) and genetic sensitivity score (Wave IV), weighted to represent the population. GSS = Genetic Sensitivity Score

Models 5-8 include additional controls not shown: household size, Aid to Families with Dependent Children receipt, food stamp receipt, having enough money for bills, birth weight, special education status, cognitive disability status, and parental measures of education, self-rated health, alcohol use, smoking, age, employment, marital status, race, ethnicity, and seatbelt use.

Robust standard errors in parentheses, adjusted for family level clustering. * $p<0.05$, ** $p<0.01$

Table S6: Family Fixed Effects Regressions Predicting Young Adult Financial Outcomes: Alternative GSS Measures
Panel A

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth	(5) √Hh Income	(6) % FPL	(7) √Earnings	(8) IHS Hh Net Worth
GSS Alternate 1	-28.44**	-78.44**	-24.77*	1.34				
	(8.51)	(24.19)	(10.25)	(1.34)				
GSS Alternate 1 x √Hh Income	0.10**	0.35**	0.10*	-0.00				
	(0.03)	(0.10)	(0.04)	(0.01)				
GSS Alternate 2					-27.81**	-75.34**	-24.81*	1.40
					(8.72)	(25.02)	(10.64)	(1.37)
GSS Alternate 2 x √Hh Income					0.10**	0.34**	0.10*	-0.00
					(0.03)	(0.10)	(0.04)	(0.01)
Age	8.08**	12.74*	8.08**	0.25	8.17**	12.96*	8.15**	0.24
	(1.91)	(6.04)	(1.86)	(0.31)	(1.91)	(6.04)	(1.86)	(0.31)
Constant	21.31	-3.32	-64.02	-4.50	17.16	-17.07	-68.40	-4.61
	(56.61)	(179.60)	(56.66)	(9.14)	(56.77)	(179.61)	(56.55)	(9.18)
Observations	630	624	624	564	630	624	624	564
R-squared	0.08	0.04	0.07	0.01	0.08	0.04	0.07	0.01
Number of Sibling Pairs	315	312	312	282	315	312	312	282

GSS Alternate 1 is an index of 2-, 3-, or 5-repeat MAOA alleles, short 5-HTT alleles, 6- through 10-repeat DRD4 alleles, 10- through 13-repeat DAT1 alleles, and DRD2 A1 alleles.

GSS Alternate 2 is an index of 2-, 3-, or 5-repeat MAOA alleles, short 5-HTT alleles, 7-repeat DRD4 alleles, 10-repeat DAT1 alleles, and DRD2 A1 alleles.

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering.

* p<0.05, ** p<0.01

Panel B

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth	(5) √Hh Income	(6) % FPL	(7) √Earnings	(8) IHS Hh Net Worth
GSS Alternate 3	-27.33**	-74.55**	-23.20*	1.59				
	(8.38)	(23.74)	(10.24)	(1.33)				
GSS Alternate 3 x √Hh Income	0.10**	0.35**	0.10*	-0.01				
	(0.03)	(0.10)	(0.04)	(0.01)				
GSS Alternate 4					-28.95**	-78.33**	-24.60*	1.34
					(8.90)	(26.03)	(10.64)	(1.36)
GSS Alternate 4 x √Hh Income					0.11**	0.36**	0.11*	-0.00
					(0.03)	(0.11)	(0.04)	(0.01)
Age	8.11**	12.75*	8.10**	0.25	8.15**	12.90*	8.14**	0.24
	(1.91)	(6.04)	(1.86)	(0.31)	(1.91)	(6.03)	(1.86)	(0.31)
Constant	16.37	-19.42	-67.68	-4.91	15.24	-21.91	-70.07	-4.64
	(56.71)	(180.20)	(56.43)	(9.14)	(56.66)	(179.75)	(56.51)	(9.16)
Observations	630	624	624	564	630	624	624	564
R-squared	0.08	0.04	0.07	0.01	0.08	0.04	0.07	0.00
Number of Sibling Pairs	315	312	312	282	315	312	312	282

GSS Alternate 3 is an index of 2- or 3-repeat MAOA alleles, short 5-HTT alleles, 6- through 10-repeat DRD4 alleles, 10-repeat DAT1 alleles, and DRD2 A1 alleles.

GSS Alternate 4 is an index of 2- or 3-repeat MAOA alleles, short 5-HTT alleles, 7-repeat DRD4 alleles, 10- through 13-repeat DAT1 alleles, and DRD2 A1 alleles.

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering.

* $p<0.05$, ** $p<0.01$

Panel C

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth	(5) √Hh Income	(6) % FPL	(7) √Earnings	(8) IHS Hh Net Worth
GSS Alternate 5	-27.43**	-75.27**	-24.14*	1.50				
	(8.38)	(23.57)	(10.25)	(1.34)				
GSS Alternate 5 x √Hh Income	0.10**	0.34**	0.10*	-0.01				
	(0.03)	(0.10)	(0.04)	(0.01)				
GSS Alternate 6					-29.05**	-79.08**	-25.59*	1.24
					(8.90)	(25.87)	(10.66)	(1.36)
GSS Alternate 6 x √Hh Income					0.11**	0.35**	0.11*	-0.00
					(0.03)	(0.10)	(0.04)	(0.01)
Age	8.11**	12.79*	8.09**	0.25	8.14**	12.93*	8.13**	0.24
	(1.91)	(6.04)	(1.86)	(0.31)	(1.91)	(6.04)	(1.86)	(0.31)
Constant	19.77	-9.21	-65.04	-4.70	18.88	-10.83	-67.30	-4.42
	(56.69)	(179.86)	(56.56)	(9.15)	(56.67)	(179.41)	(56.66)	(9.18)
Observations	630	624	624	564	630	624	624	564
R-squared	0.08	0.04	0.07	0.01	0.08	0.04	0.07	0.00
Number of Sibling Pairs	315	312	312	282	315	312	312	282

GSS Alternate 5 is an index of 2-, 3-, or 5-repeat MAOA alleles, short 5-HTT alleles, 6- through 10-repeat DRD4 alleles, 10-repeat DAT1 alleles, and DRD2 A1 alleles.

GSS Alternate 6 is an index of 2-, 3-, or 5-repeat MAOA alleles, short 5-HTT alleles, 7-repeat DRD4 alleles, 10- through 13-repeat DAT1 alleles, and DRD2 A1 alleles.

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering.

* $p<0.05$, ** $p<0.01$

Panel D

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth	(5) √Hh Income	(6) % FPL	(7) √Earnings	(8) IHS Hh Net Worth
GSS Alternate 7	-28.36**	-77.77**	-23.84*	1.43				
	(8.52)	(24.35)	(10.24)	(1.33)				
GSS Alternate 7 x √Hh Income	0.11**	0.35**	0.10*	-0.00				
	(0.03)	(0.10)	(0.04)	(0.01)				
GSS Extended 1					-15.96*	-34.80*	-17.13**	0.83
					(6.39)	(17.03)	(6.00)	(0.82)
GSS Extended 1 x √Hh Income					0.05*	0.14	0.07**	-0.00
					(0.03)	(0.08)	(0.02)	(0.00)
Age	8.09**	12.72*	8.08**	0.25	8.30**	13.33*	8.31**	0.21
	(1.91)	(6.03)	(1.86)	(0.31)	(1.93)	(6.13)	(1.86)	(0.31)
Constant	17.82	-13.89	-66.75	-4.71	24.88	6.76	-70.02	-0.94
	(56.64)	(179.96)	(56.53)	(9.13)	(58.57)	(182.22)	(55.69)	(9.36)
Observations	630	624	624	564	630	624	624	564
R-squared	0.08	0.04	0.07	0.01	0.07	0.02	0.07	0.01
Number of Sibling Pairs	315	312	312	282	315	312	312	282

GSS Alternate 7 is an index of 2- or 3-repeat MAOA alleles, short 5-HTT alleles, 6- through 10-repeat DRD4 alleles, 10- through 13-repeat DAT1 alleles, and DRD2 A1 alleles.

GSS Extended 1 is an index of 2- or 3-repeat MAOA alleles, short 5-HTT alleles, 7-repeat DRD4 alleles, 10-repeat DAT1 alleles, DRD2 A1 alleles, COMT A alleles, DRD5 148-bp alleles, rs12945042 (between 5-HTT and BLMH) indicator for two T alleles, MAOA-CA 115-bp and above alleles

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering.

* $p<0.05$, ** $p<0.01$

Panel E

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth	(5) √Hh Income	(6) % FPL	(7) √Earnings	(8) IHS Hh Net Worth
GSS Extended 2	-15.30*	-30.18	-19.28**	1.14				
	(6.40)	(16.86)	(5.61)	(0.81)				
GSS Extended 2 x √Hh Income	0.05*	0.13	0.08**	-0.01				
	(0.03)	(0.07)	(0.02)	(0.00)				
GSS Extended 3					-16.82*	-46.10*	-14.34	1.13
					(7.11)	(19.43)	(7.85)	(1.07)
GSS Extended 3 x √Hh Income					0.06*	0.19*	0.07*	-0.01
					(0.03)	(0.09)	(0.03)	(0.00)
Age	8.26**	13.24*	8.27**	0.21	8.25**	13.16*	8.22**	0.22
	(1.93)	(6.14)	(1.86)	(0.31)	(1.93)	(6.13)	(1.87)	(0.31)
Constant	23.30	-3.06	-66.32	-1.31	18.58	2.17	-77.86	-1.29
	(58.57)	(183.22)	(55.45)	(9.35)	(58.36)	(182.37)	(55.55)	(9.37)
Observations	630	624	624	564	630	624	624	564
R-squared	0.07	0.02	0.08	0.01	0.07	0.03	0.06	0.01
Number of Sibling Pairs	315	312	312	282	315	312	312	282

GSS Extended 2 is an index of 2- or 3-repeat MAOA alleles, short 5-HTT alleles, 7-repeat DRD4 alleles, 10-repeat DAT1 alleles, DRD2 A1 alleles, COMT A alleles, DRD5 148-bp alleles, rs12945042 (between 5-HTT and BLMH) indicator for two T alleles, MAOA-CA over 115-bp alleles

GSS Extended 3 is an index of 2- or 3-repeat MAOA alleles, short 5-HTT alleles, 7-repeat DRD4 alleles, 10-repeat DAT1 alleles, DRD2 A1 alleles, COMT A alleles, DRD5 148-bp alleles, rs12945042 (between 5-HTT and BLMH) indicator for two T alleles, MAOA-CA 115-bp alleles

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering.

* $p<0.05$, ** $p<0.01$

Table S7: Family Fixed Effects Regressions Predicting Young Adult Financial Outcomes: Interaction Sensitivity Score

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth
Genetic Sensitivity Interaction Score	-38.19**	-93.00**	-42.52**	0.71
	(13.63)	(31.34)	(16.30)	(5.53)
GSIS x √Hh Income	0.16**	0.45**	0.18*	-0.01
	(0.05)	(0.11)	(0.07)	(0.02)
Age	8.23**	13.35*	8.24**	0.22
	(1.93)	(6.13)	(1.87)	(0.31)
Constant	4.17	-3.88	-70.19	-2.91
	(55.26)	(175.15)	(53.38)	(8.79)
Observations	630	624	624	564
R-squared	0.06	0.02	0.06	0.00
Number of Sibling Pairs	315	312	312	282

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

GSIS = Genetic Sensitivity Interaction Score; created by interacting the number of long DAT1 alleles, long DRD4 alleles, short MAOA alleles, short 5-HTT alleles, and DRD2 A1 alleles an individual carries.

Robust standard errors in parentheses, adjusted for family level clustering.

* p<0.05, ** p<0.01

Table S8: Family Fixed Effects Regressions Predicting Young Adult Financial Outcomes: Including Additional Controls

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth	(5) √Hh Income	(6) % FPL	(7) √Earnings	(8) IHS Hh Net Worth
Genetic Sensitivity Score	-32.48**	-92.60*	-30.05**	1.64	-33.77**	-97.50**	-32.49**	1.63
	(11.87)	(38.48)	(10.25)	(1.76)	(10.99)	(34.60)	(10.68)	(1.78)
GSS x √Hh Income	0.14**	0.45**	0.14**	-0.00	0.14**	0.47**	0.15**	-0.00
	(0.05)	(0.15)	(0.04)	(0.01)	(0.04)	(0.13)	(0.05)	(0.01)
Age	2.12	-8.73	9.77	-0.59	2.35	-6.40	9.68	-0.81
	(5.16)	(15.75)	(5.28)	(0.95)	(5.21)	(15.30)	(5.22)	(0.97)
Birth Weight (log ounces)	15.61	88.83	34.70	7.01	-39.25	-445.85	-7.29	39.53*
	(31.94)	(115.42)	(36.83)	(5.08)	(107.44)	(403.75)	(127.45)	(16.27)
Older Sibling	10.74	48.90	-6.94	2.35	-10.53	-30.29	-8.29	4.89
	(15.41)	(47.56)	(14.67)	(2.64)	(22.91)	(72.27)	(21.29)	(3.58)
Self-Reported Parental Favoritism	-12.77	-30.30	-13.45*	-2.14*	-19.19	-43.17	-32.91	-5.77
	(7.57)	(25.37)	(5.79)	(1.02)	(22.78)	(71.12)	(17.30)	(3.58)
Birth Weight x √Hh Income					0.23	2.25	0.18	-0.14*
					(0.43)	(1.79)	(0.56)	(0.07)
Older x √Hh Income					0.08	0.30	0.00	-0.01
					(0.07)	(0.26)	(0.07)	(0.01)
Favoritism x √Hh Income					0.02	0.03	0.08	0.02
					(0.09)	(0.28)	(0.07)	(0.01)
Constant	98.24	125.06	-293.91	-16.96	88.50	12.72	-292.77	-7.77
	(219.80)	(669.68)	(206.46)	(37.41)	(224.67)	(684.86)	(212.80)	(38.02)
Observations	456	450	452	411	456	450	452	411
R-squared	0.09	0.06	0.12	0.04	0.10	0.08	0.13	0.07
Number of Sibling Pairs	269	266	267	240	269	266	267	240

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering.

* p<0.05, ** p<0.01

Table S9: Family Fixed Effects Regressions Predicting Young Adult Financial Outcomes: By Parental Education and % Federal Poverty Level

VARIABLES	(1) √Hh Income	(2) % FPL	(3) √Earnings	(4) IHS Hh Net Worth	(5) √Hh Income	(6) % FPL	(7) √Earnings	(8) IHS Hh Net Worth
Genetic Sensitivity Score	-60.19**	-118.94	-50.85*	-0.75	-12.46*	-23.62	-10.11	0.83
	(22.82)	(66.74)	(23.00)	(4.17)	(6.11)	(18.65)	(7.14)	(0.77)
GSS x Average Parental Education	4.40*	9.70	3.91*	0.08				
	(1.74)	(5.20)	(1.73)	(0.32)				
GSS x % FPL					0.04*	0.12*	0.04*	-0.00
					(0.02)	(0.06)	(0.02)	(0.00)
Age	7.67**	11.44*	7.15**	0.12	8.56**	14.50*	8.70**	0.32
	(1.81)	(5.71)	(1.85)	(0.29)	(1.96)	(6.40)	(2.06)	(0.31)
Constant	29.84	19.21	-40.87	-1.63	7.44	-65.55	-89.23	-6.60
	(53.00)	(169.82)	(55.66)	(8.69)	(58.41)	(194.94)	(63.52)	(9.44)
Observations	713	705	703	639	536	532	530	482
R-squared	0.06	0.02	0.05	0.00	0.08	0.03	0.07	0.01
Number of Sibling Pairs	358	354	353	321	268	266	265	241

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV).

Robust standard errors in parentheses, adjusted for family level clustering.

* $p<0.05$, ** $p<0.01$

Table S10: Family Fixed Effects Regressions Predicting Young Adult Financial Outcomes: By Race

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	White				Black			
VARIABLES	√Hh Income	% FPL	√Earnings	HIS Hh Net Worth	√Hh Income	% FPL	√Earnings	IHS Hh Net Worth
Genetic Sensitivity Score	-28.90**	-83.24*	-26.34*	1.83	-29.68	-61.73	1.08	-4.83
	(10.60)	(32.25)	(12.99)	(1.55)	(16.39)	(34.62)	(17.33)	(2.97)
GSS x √Hh Income	0.10**	0.36**	0.12*	-0.01	0.16*	0.30	-0.06	0.03*
	(0.04)	(0.12)	(0.05)	(0.01)	(0.07)	(0.15)	(0.08)	(0.01)
Age	7.64**	11.23	6.83**	-0.16	13.43**	18.86	14.87**	2.16*
	(2.28)	(7.37)	(2.16)	(0.34)	(4.78)	(9.98)	(4.46)	(0.84)
Constant	38.21	42.16	-40.18	7.80	-171.61	-236.83	-228.75	-59.95*
	(68.69)	(220.93)	(65.38)	(10.08)	(141.79)	(293.10)	(126.91)	(25.40)
Observations	464	458	462	418	84	84	82	78
R-squared	0.07	0.03	0.06	0.01	0.22	0.11	0.27	0.23
Number of Sibling Pairs	232	229	231	209	42	42	41	39

Source: Add Health Data Waves I and IV. Sibling sample includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV), alternatively limited to white and black siblings.

Robust standard errors in parentheses, adjusted for family level clustering.

* p<0.05, ** p<0.01

Sensitivity Analyses Limited to Low- and High-Income Families

According to the differential susceptibility model, the main GSS effect should be negative among low income households and positive among high income households. Results from Table 4 suggest the crossover point is generally around median parental income. Table S11 compares GSS coefficients from family fixed effects regressions among siblings from high and low income families. The top group of coefficients in Table S11 is from models limited to those above and below median parental household income ($56,000 in 2007 dollars). However, to examine cutoffs farther from the median, such as the crossover point found in the fixed effects model predicting household income, I also compare results among sibling pairs from households above the 65th and 75th percentiles ($70,000 and $81,000) and below the 35th and 25th percentiles ($39,000 and $34,000). The regression model is the same as that presented in the main analyses, but excludes the interaction term between GSS and parental household income.

Results in Table S11 show that among low income households, the GSS coefficient is negative in all models (except those predicting net worth) and statistically different from zero in all models predicting household income ($p<0.05$). Among high income households, the GSS coefficient is positive in every model (except two of those predicting net worth) and statistically different from zero in all models predicting percent of federal poverty level ($p<0.01$). The positive GSS coefficient also reaches significance in a few other models: predicting earnings above the 65th percentile ($p<0.05$); and predicting household income above the 75th percentile ($p<0.01$). Overall, the results shown in Table S11 further support the differential susceptibility model. As in the main analyses, a higher GSS is associated with lower financial outcomes among those from low income households, but higher financial outcomes among those from high income households.

Table S11: Genetic Sensitivity Score Coefficients in Family Fixed Effects Regressions Predicting Young Adult Financial Outcomes

	Genetic Sensitivity Score Coefficient			
	Low Parent Hh Income	N	High Parent Hh Income	N
Above/Below Median Income				
√Household Income	-13.09 **	306	9.00	324
% FPL	-22.36	300	40.36 **	324
√Earnings	-7.53	304	8.93	320
IHS Hh Net Worth	0.62	272	0.06	292
Above 65th/Below 35th Percentile Income				
√Household Income	-15.21 **	214	8.96	250
% FPL	-24.58	210	47.70 **	250
√Earnings	-10.30	212	12.97 *	248
IHS Hh Net Worth	0.21	200	-0.87	222
Above 75th/Below 25th Percentile Income				
√Household Income	-14.76 *	164	20.31 **	160
% FPL	-29.77	160	78.01 **	160
√Earnings	-11.07	162	14.88	160

IHS Hh Net Worth	0.05	152	-1.64	144

Source: Add Health Data Waves I and IV. Includes same sex singleton full sibling pairs with complete information about parental income (Wave I), individual income (Wave IV), and genetic sensitivity score (Wave IV), limited to pairs with total parental income above and below the median parental income ($56,000 in 2007 dollars), above the 65th or 75th percentiles ($70,000 or $81,000), or below the 35th or 25th percentiles ($39,000 or $34,000). Robust standard errors adjusted for family level clustering are not shown. * $p<0.05$, ** $p<0.01$