Parental Behaviors, Genes, and the Intergenerational Transmission of Poverty

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Extended Abstract

Introduction

How is poverty transmitted between generations? Cultural explanations suggest parental values and behaviors, though developed in response to structural marginalization, are passed on to children, preventing them from escaping poverty (Lewis 1966, 1969). If poverty is culturally transmitted, limiting parental exposure should weaken the intergenerational transmission process, reducing the likelihood that children who experienced poverty will end up there as adults. Recent research, however, provides contradictory evidence about the potential consequences of childhood exposure to behaviors associated with poverty, suggesting minimal (Lauen and Gaddis 2013) or negative effects (Wildeman and Turney 2014).

Genetic explanations suggest economic standing is inherited at birth and is therefore even less amenable to policy changes than culture (Clark 2014; Herrnstein & Murray 1994). If poverty is genetically determined, adult poverty status should be largely immune to parental behaviors. Further, this traditional view suggests any genetic risk of poverty is fixed and therefore independent of environment. In the last decade, however, research has found evidence of gene-environment interactions, suggesting that genetic effects on a variety of behaviors (ranging from depression to college attendance) depend on our environment (Caspi et al. 2002, 2003; Guo, Roettger, and Cai 2008; Shanahan et al. 2008; Pescosolido et al. 2008).

More recently, a new hypothesis has emerged, suggesting that gene-environment interactions are more complex than previously thought. The biological sensitivity to context hypothesis (Boyce and Ellis 2005; Ellis and Boyce 2008; Obradovic et al. 2010) suggests certain

genotypes make individuals more sensitive to environments than others, increasing variation in outcomes. In other words, rather than the idea that certain "risky" genotypes simply yield more negative outcomes (simple genetic effects perspective), or more negative average outcomes in poor environments (traditional gene-environment interaction perspective), the sensitivity argument suggests these genotypes are not necessarily negative, but rather increase variation in outcomes depending on environment. Thus, in a deprived environment, those with a "risky" genotype could experience more negative outcomes (such as a greater risk of depression, delinquency, or violent behavior or a lower likelihood of college attendance), but in a particularly *supportive* environment they could achieve even *more positive* outcomes than those with more stable, alternative genotypes.

Using family fixed effect models of sibling data from the National Longitudinal Study of Adolescent Health, I investigate two questions. First, do parent-child activities (e.g., talking about school, going to a play) moderate parent-child economic similarity? Second, consistent with the Biological Sensitivity to Context hypothesis, does an index of risky genotypes (random within full sibling pairs) moderate rather than mediate parent-child economic similarity?

Methods

To improve on existing research, this study uses a novel approach, asking whether intergenerational similarity differs by genotype within full sibling pairs. Intergenerational transmission is often conceived as constant within a family, yet we know that siblings can experience different environments and inherit different genes. In fact, because each sibling has an equal chance of inheriting one of two alleles at each gene from each parent, genotype within full sibling pairs is random. I improve on existing studies by exploiting this random genetic variation within full biological siblings. Furthermore, while I cannot fully randomize

environment, siblings hold constant much environmental variation that is difficult to account for among unrelated individuals.

Using data from the National Longitudinal Study of Adolescent Health (Add Health), I investigate whether intergenerational transmission of financial standing depends on interactions with parents and genetic risk score. Genetic risk score (GRS) is an index of alleles which have previously been associated with an increased risk of negative outcomes, such as ADHD, drug use, and depression. Measured in Wave IV, GRS counts the number of short (2/3R) MAOA alleles, short 5-HTT alleles, long (6-10R) DRD4 alleles, and long (10R) DAT alleles. These candidate genes are related to the dopamine system, which plays an important role in a variety of behaviors related to academic, health, and financial outcomes (e.g., attention, sitting still, sensation seeking). These genes are therefore ideal for investigating intergenerational transmission of financial standing, in addition to a variety of other outcomes.

Parent activities is an index measuring the frequency with which a child does the following with either parent: shop; play a sport; go to a religious service; talk about someone you're dating; go to a movie, play, or other event; talk about a personal problem; talk about school work or grades; work on a project for school; talk about other things you're doing in school. Culture of poverty arguments suggest that poverty is transmitted when parents pass their beliefs and values on to their children. This index of parent activities is a proxy for child exposure to parental beliefs and values. Spending more time and participating more frequently in activities with parents should strengthen the transmission process according to the culture of poverty perspective.

Parental financial standing is based on Wave I, when children were ages 10 to 19. Child financial standing is measured in Wave IV, when children from Wave I have become adults ages

24 to 34. I use two approaches to investigate differences in the intergenerational relationship by parental interactions and genotype. First, using family fixed effects, I regress financial standing as an adult on parental interactions or genetic risk score and its interaction with parental financial standing. The main effect of parental financial standing drops out because it is the same within sibling pairs. I exclude any pairs with different parental financial standing in Wave I as well as sibling pairs of opposite sex.

In equation 1 below, ß$_2$ estimates whether parent-child financial similarity for child *i* in family *j* varies by genetic risk score. Including family fixed effects, if ß$_2$ is significant, it would suggest that the candidate genes moderate intergenerational transmission. If genetic risk score increases the association between parent and child income relative to one's sibling, it would support arguments that these genotypes increase sensitivity to context.

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

Second, I run regressions separately by Wave I poverty status. In this second analysis, I include all siblings (both same and opposite sex) to ensure sufficient sample sizes in both poverty categories. Equation 2 below predicts child financial standing with the index of parent activities. With family fixed effects, ß$_1$ estimates the relationship between interactions with parents and adult financial standing compared to one's full sibling. By limiting the sample to siblings above and below the poverty line as children, the model compares the relationship between parent activities and child financial outcomes by childhood poverty status. X includes controls for age and gender differences between siblings.

$$\text{Child Income}_{ij} = \alpha_0 + \beta_1 \text{Parent Activities}_{ij} + \beta_2 \text{X} + \eta_j + \varepsilon_{ij}$$

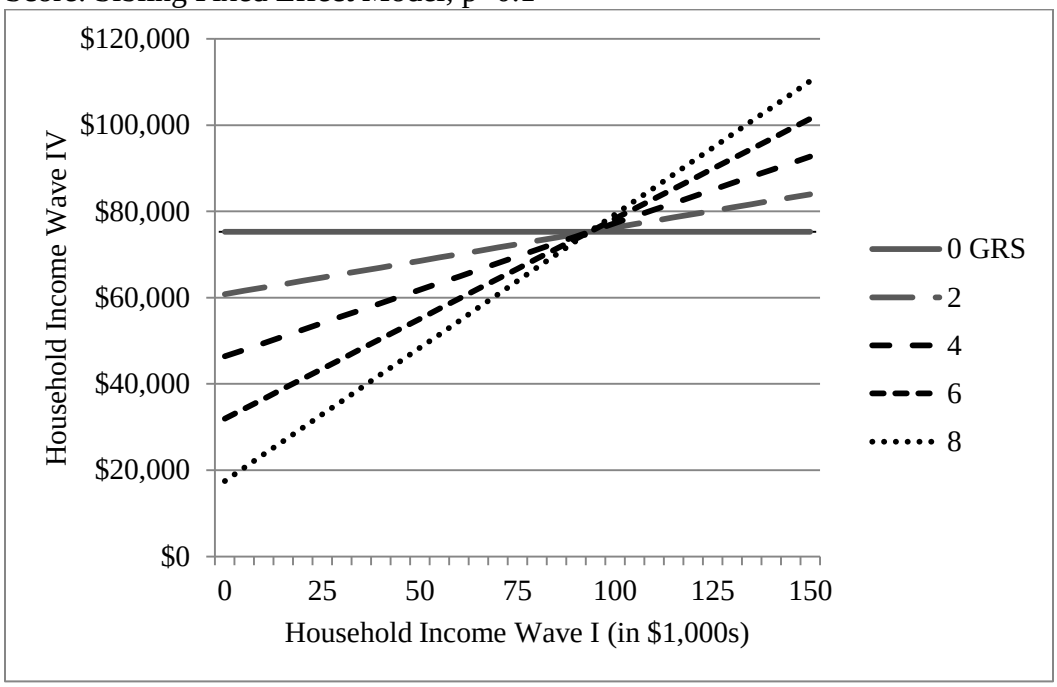
Preliminary Results

Preliminary results are consistent with the Biological Sensitivity to Context Hypothesis. As Figure 1 shows, compared to one's full sibling, income as an adult depends more strongly on parental income as the number of risky alleles increases. Furthermore, within full sibling pairs, children with a higher genetic risk score fare more poorly than their sibling if they grow up in a lower income household, but fare better than their sibling if they grow up in a higher income household. These results suggest the consequences of particular alleles depend on context. While traditionally portrayed as conveying risk, the alleles investigated here can convey increased potential in supportive environments. Policies which provide more supportive environments, including household income subsidies for families with children, may improve the likelihood of positive adult outcomes for all children, particularly those with sensitive alleles.

Preliminary results shown in Figure 2 suggest parent-child activities significantly reduce the likelihood of adult poverty for those in poverty as a child, but are unrelated to poverty for others. Contradicting arguments that culture mediates transmission, this finding suggests policies could weaken the cycle of poverty by subsidizing parent-child activities and providing poor parents more time to spend with their children. While they may have other important benefits, "welfare-to-work" programs could perversely strengthen the intergenerational transmission of poverty by reducing time parents can spend with children.

Preliminary Results

Figure 1: Predicted Relationship between Parent and Child Household Income by Genetic Risk Score: Sibling Fixed Effect Model, $p<0.1$



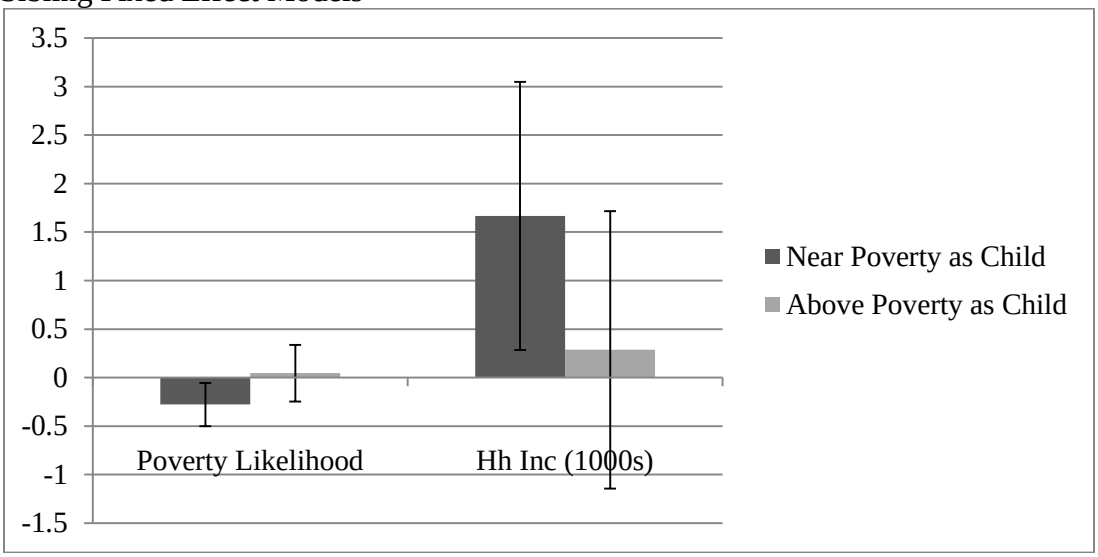
Source: Add Health Data Waves I and IV. N = 932 siblings.

GRS = Genetic Risk Score – an index of the number of short (2/3R) MAOA alleles, short 5-HTT alleles, long (6-10R) DRD4 alleles, and long (10R) DAT alleles, measured in Wave IV.

Sibling fixed effect models include same sex full siblings and fraternal twins, but exclude identical twins who share the same genetic risk score.

Compared to one's full sibling, children's income as an adult depends more strongly on the income of their parents when they have a greater number of risky alleles. In support of the Biological Sensitivity to Context hypothesis, children with a higher genetic risk score 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.

Figure 2: Predicted Effect of Frequency of Parent-Child Activities on Adult Economic Standing: Sibling Fixed Effect Models



Source: Add Health Data Waves I and IV.

Sibling fixed effect models are run separately for families near or above poverty at Wave I.

Parent activities measure the frequency with which a child does the following with either parent: shop; play a sport; go to a religious service; talk about someone you're dating; go to a movie, play, or other event; talk about a personal problem; talk about school work or grades; work on a project for school; talk about other things you're doing in school.

Controls include age and gender. Sample sizes: xtlogit poverty N=162; xtreg household income N=1736.

Among children who grew up near poverty, and compared to one's full sibling, children who participate in more activities with their parents have higher household incomes and are less likely to be in poverty as an adult. Among children who grew up above poverty, parent-child activities are unrelated to adult economic standing.

References

Boyce, W.T. and B.J. Ellis. 2005. "Biological Sensitivity to Context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity." *Development and Psychopathology* 17: 271-301.

Caspi, Avshalom, Joseph McClay, Terrie E. Moffitt, Jonathan Mill, Judy Martin, Ian W. Craig, Alan Taylor, and Richie Poulton. 2002. "Role of Genotype in the Cycle of Violence in Maltreated Children." *Science* 297:851-54.

Caspi, Avshalom, Karen Sugden, Terrie E. Moffitt, Alan Taylor, Ian W. Craig, HonaLee Harrington, Joseph McClay, Jonathan Mill, Judy Martin, Antony Braithwaite, and Richie Poulton. 2003. "Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene." *Science* 301:386-89.

Clark, Gregory with Neil Cummins, Yu Hao, and Daniel Diaz Vidal. 2014. *The Son Also Rises: Surnames and the History of Social Mobility*. Princeton, NJ: Princeton University Press.

Ellis, B. J. and W. T. Boyce. 2008. Biological Sensitivity to Context. *Current Directions in Psychological Science* 17: 183-7.

Guo, Guang, Michael E. Roettger, and Tianji Cai. 2008. "The Integration of Genetic Propensities into Social-Control Models of Delinquency and Violence among Male Youths." *American Sociological Review* 73(4):543-68.

Herrnstein, Richard J. and Charles Murray. 1994. *The Bell Curve: Intelligence and Class Structure in American Life*. New York: Free Press.

Lauen, Douglas and S. Michael Gaddis. 2013. "Exposure to Classroom Poverty and Test Score Achievement: Contextual Effects of Selection?" *American Journal of Sociology* 118(4):943-979.

Lewis, Oscar. 1969. "The Culture of Poverty." In *On Understanding Poverty: Perspectives from the Social Sciences*, D.P. Moynihan (ed.). New York: Basic Books.

Lewis, Oscar. 1966. *La Vida; A Puerto Rican Family In The Culture Of Poverty*. New York: Random House.

Obradovic, J., N.R. Bush, J. Stamperdahl, Nancy E. Adler, W. Thomas Boyce. 2010. "Biological Sensitivity to Context: The Interactive Effects of Stress Reactivity and Family Adversity on Socioemotional Behavior and School Readiness." *Child Development* 81:270-89.

Pescosolido, Bernice A., Brea L. Perry, J. Scott Long, Jack K. Martin, John I. Nurnberger, Jr., and Victor Hesselbrock. 2008. "Under the Influence of Genetics: How Transdisciplinarity Leads Us to Rethink Social Pathways to Illness." *American Journal of Sociology* 114(S1):S171–S201.

Shanahan, Michael J., Stephen Vaisey, Lance D. Erickson, and Andrew Smolen. 2008. "Environmental Contingencies and Genetic Propensities: Social Capital, Educational Continuation, and Dopamine Receptor Gene DRD2." *American Journal of Sociology* 114(S1):S260-S86.

Wildeman, Christopher and Kristen Turney. 2014. Positive, Negative, or Null? The Effects of Maternal Incarceration on Children's Behavioral Problems. *Demography*. Retrieved April 25, 2014 from http://link.springer.com/article/10.1007%2Fs13524-014-0291-z#page-1.