Understanding Heterogeneity in the Effects of Birth Weight on Adult Cognition and Wages[1]

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Abstract: Large literatures across the social sciences, medical sciences, and policy analysis fields have shown long term impacts of birth weight on adult outcomes, including IQ and earnings. The effects are often robust to sibling or twin fixed effects. These findings have suggested the importance of targeting interventions and policies, such as WIC and prenatal care, to increase birth weight as well as mitigate the effects of low birth weight on developmental outcomes. Advances in biological and genetic fields have shed light on the mechanisms linking low birth weight with long term outcomes, suggesting the potential that targeting resources to those at highest risk for poor outcomes may further enhance policy impacts. We combine neuroscience, genetics and social science concepts, methods and data to examine sources of heterogeneity of birth weight impacts of future outcomes. Using variation in three genes related to neuroplasticity (*APOE*, *BDNF*, and *COMT*), we find substantial heterogeneity in the effects of birth weight on adult outcomes, where a large part of the population is not affected by birth weight variation. Our results help uncover why birth weight affects adult outcomes and suggest the potential benefits of targeting interventions and policies.

1. Introduction

Large literatures across multiple disciplines have established the importance of early nutrition environments, which is often measured by birth weight, on both short and long term outcomes. Short term impacts are most straightforward to motivate, where babies born with low birth weight, from having lower levels of intrauterine nutrition or are otherwise unhealthy, have been shown to have higher infant mortality and have higher medical care costs (Almond, Chay and Lee 2005). The existence and magnitude of longer term (i.e., adult) impacts are less obvious but have been the subject of a growing body of work.[1] One motivation for this line of research is the fetal origins hypothesis by Barker (1995) who provides evidence that individuals born with low birth weight are prone to heart disease and type 2 diabetes later in life. In economics, Black et al. (2007) further test and extend this hypothesis by showing that birth weight differences between twins have lifelong impacts on IQ, labor force participation, and earnings.[2] These findings are further supported by a number of natural experiments. A long term follow up of individuals who were in utero during the Dutch Hunger Winter (Stein et al. 1975), in which fetuses were exogenously exposed to poor intrauterine nutrition during the famine of 1944-45, have been shown to have later experienced a range of negative health and economic outcomes (Stein et al. 2005, de Roij et al. 2010, Schultz et al. 2012). Additionally, individuals in utero during the month of Ramadan, which is associated with diurnal fasting, are shown to have reduced birth weights, leading to reduced mental performance later in life (Almond and Mazumder 2011).

[1] See Figlio et al. (2013) and Fletcher (2011) for evidence of "medium run" impacts of birth weight between ages 5 and 18. For "longer run" impacts see Conley and Bennett 2000, Conley et al. 2003, and Hack et al. 2002.

[2] The use of sibling fixed effects in exploring the effects of birth weight on cognitive development is first explored by Conley and Bennet (2000).

This evidence that has emerged from a variety of disciplines, research designs and contexts all point to the potential for large policy impacts from programs and interventions that reduce the likelihood of low birth weight and/or mitigate the impacts of low birth weight on future outcomes.[3] For example, Women, Infants, and Children (WIC) Food Supplementation vouchers have been shown to increase birth weight (see e.g., Bitler and Currie 2005, Joyce et al. 2008, Kennedy et al. 1982). And given the life-long effects of poor in utero nutrition, early life interventions that eliminate harmful environments are highly effective in reducing future social costs, both in terms of public health and human capital (see e.g., Doyle et al. 2009, Heckman et al. in press, Ladd et al. 2013).

In a context of reduced governmental budgets for programs that aim to improve social welfare (e.g. SNAP, WIC), a potential next step in further improving impacts under existing budget allocations is to understand the heterogeneity in the impacts of birth weight on future outcomes. Not all children born with low birth weight experience poor adult outcomes—indeed, there is much resilience to a variety of health insults experienced early in life.

Targeting programs and interventions are not new, as many programs have income or family structure eligibility requirements, with the goal of focusing resources towards those most likely to be affected by low birth weight. For example, WIC is a nutritional supplementation program for women earning less than 185 percent of the poverty level and who are pregnant or with children up to five years of age (USDA 2012). In the medical sciences, there is emerging evidence for the efficacy of targeting interventions towards individuals according to expected responses based on genetic variants (e.g. Pirmohamed et al. 2013). In some cases, such as birth weight, similar policy and interventions responses

[3] Research on low birth weight ties into life-course epidemiology, from which the accumulation of health behaviors and outcomes, starting from conception, affect later life outcomes (for review, see Glymour et al. 2009).

could, in principle, be considered to direct resources where they will be most effective.

In order to provide evidence for this direction of policy research, our focus is on biological determinants that may moderate the impact of birth weight on later-life cognitive and economic outcomes. The presence of this biological endowment, which is permanent and randomly determined at conception, has the ability to be measured at birth (as many babies provide relevant biological specimens (e.g., blood spots) following hospital delivery), and in so doing could provide information for targeting post-natal interventions.

The biological mechanisms linking poor nutrition in utero and long term effects on cognition are not fully developed, but several hypotheses have been proposed. A tenant of the fetal origins hypothesis is that nutritional insults cause the body to shift resources to the brain, leaving other organ systems prone to future deficits from this critical period of under-development. Heterogeneity in the ability to shift resources as well as differences in the potential plasticity in neurodevelopment suggests there could be varying impacts on cognition as well as health across individuals born with low birth weight.

Even with these hypothesized resource shifts to protect the brain, there is also ample evidence of long term effects on cognition. Most related to the current work is the evidence of effects of early nutrition on cognitive development, particularly IQ in early adulthood.[4] A positive and statistically significant association between birth weight and IQ has been found in a number of studies and samples (see e.g., Black et al. 2007; Conley and Bennet 2000; Conley et al. 2003; Newcombe et al. 2007). For example Newcombe et al. (2007) finds that a kilogram increase in birth weight is associated with a 3 point increase in IQ.

[4] While differential birth weight does provide a head start or lag in cognitive development, post-natal family and schooling environments are also associated with later life cognitive development (Barnett et al. 2007, Cunha et al. 2006).

Furthermore, this link between birth weight and IQ is found within MZ twin pairs, decreasing the likelihood of spurious results from unmeasured family background or genetics. This has important economic consequences, as IQ has both direct and indirect impacts on lifetime earnings, schooling decisions, and criminal and risky behavior (Heckman et al. 2006, Gensowski 2013). However, there is currently limited understanding of the mechanisms linking low birth weight with adult IQ and labor market outcomes. Physiologically, emerging evidence suggests that birth weight has a positive, linear association with regional surface area and total volume of the brain (Walhovd et al. 2012). Additional research to further uncover mechanisms could allow us to gain a better understanding of the determinants of adult IQ and productivity as well as, more speculatively, to suggest avenues to target resources at individuals most likely to be affected by low birth weight.

The main idea of the current work is to explore whether genetic variation related to neuroplasticity may uncover essential sources of heterogeneity in the impacts of low birth weight on adult outcomes. The framework follows that proposed by previous studies examining the moderating properties of particular genetic variants within varied environments.[5] The idea being that certain genetic variants moderate, or amplify, the effects of exposure to a treatment—and this variation in impact may then help us understand mechanisms linking low birth weight with adult outcomes. Furthermore, this hypothesized heterogeneity in response to a policy relevant domain—i.e., low birth weight—suggests cost-effectiveness and outcome gains from discriminant application of the intervention.

For the current work, we focus on the flexibility of neural networks within the brain. This is referred to generally as neuroplasticity.[6] Our main hypothesis

[5] One of the first, and most famous, papers on gene-environment interactions is by Caspi et al. (2003), in which the authors show that childhood abuse leads to more severe later-life depression for individuals containing a gene variant, or allele, for the serotonin transporter gene 5-HTT. For review, please see Rutter et al. (2005) and Caspi and Moffitt (2006).
[6] Neuroplasticity is discussed in detail in Section 1.1.

is that the effect of early childhood nutrition on cognitive development is moderated, or lessened, by neuroplasticity.[7] In order to measure neuroplasticity, we focus on gene variants that have both direct and interactive associations with plasticity. The use of gene variants, which are used as a proxy for neuroplasticity, allow a focus on the interaction that these genetic variants have with our environment of interest, birth weight. In other words, we will estimate the interactive effect of birth weight and neuroplasticity, measured by genetic variants, on later-life IQ and wages. Furthermore, we are able to control for common environmental conditions amongst siblings, allowing us to account for shared harmful or beneficial post-natal environments that may also influence IQ. The leveraging of the within-sibling genome also allows a quasi-experiment in genetics, as genetic variation between full biological siblings is random and has been labeled as a "genetic lottery" in prior work (Fletcher and Lehrer 2009, 2011).

1.1 Neuroplasticity

Neuroplasticity refers to the ability of the brain to maintain and strengthen neural connections as well as developing new connections between neurons (Pascual-Leon et al. 2005).[8] It is a constant process of strengthening and replacing neural connections, affecting the structure and function of axons, dendrites, and synapses (Teter and Ashford 2002). Plasticity is commonly invoked after brain injuries, or insults, such as a stroke; after which, neural networks are re-organized from damaged to undamaged areas within the brain (Frost et al. 2003, Pascual-Leon et al. 2005). Other examples of neuroplasticity are found in the increased sensitivity

[7] Using a novel data set, Figlio et al. (2013) argue that birth weight has a persistent effect on cognitive development during childhood. We, however, are interested in correction, or growth rate, not initial stunting; the idea being that in a late enough period neuroplasticity results in convergence in cognition.

[8] Neuroplasticity is not solely a positive, or favorable, condition. Continual remapping may lead to degenerative conditions (Pascual-Leon 2005, Teter and Ashford 2002).

in touching and hearing in the blind. For our purposes, plasticity represents an ability to respond to environmental shocks—i.e., poor early life nutrition. Individuals with greater plasticity, or individuals who are more able to recover from negative cognitive shocks, should be cognitively robust to a poor in utero environment. In other words, birth weight may not be a major predictor of cognitive development, or earnings, for individuals with greater neuroplasticity.

In order to measure neuroplasticity, we will use variation in genetic markers. The genes under consideration are *APOE*, *COMT*, and *BDNF*. All three genes are associated with neuroplasticity, both directly and interactively with one another.[9] The three genes were selected because they are the only ones available in the WLS dataset that have been shown to be related to neuroplasticity in the literature, as we document below.[10] Focusing only on these three genes allows us to reduce the concern of multiple hypothesis testing[11]. We also show below that the results are robust to a number of different ways of measuring neuroplasticity based on these three genes.

APOE (apolipoprotein E) is associated with the transportation of lipids, or fatty acids, within the brain. The gene variant under consideration for the current work is the E4 variant, which has strong associations with Alzheimer's disease.[12] *APOE4* is particularly poor at removing plaques within neural pathways, resulting in poor synaptic plasticity (Bu 2009, Tromer et al. 2005); therefore, the E4 variant

[9] All genetic variants under consideration are single-nucleotide polymorphisms (SNP). A SNP is a single change along a sequence of DNA. For example, "ATA" versus "ATC", where the "A" and "C" are variants for the third nucleic base in the sequence. Each SNP under consideration is assigned a reference, or "rs," number.

[10] See complete list of SNPs at http://www.ssc.wisc.edu/wlsresearch/documentation/supdoc/biomarker/cor1019b_SNPs_in_wave1_data.pdf

[11] For example, the Health and Retirement Study has recently released over 2 million genetic variants for each of the over 12,000 respondents in the genetic sample. This would allow a nearly infinite number of ways to characterize neuroplasticity, and similarly allow a very large number of regressions to be examined, leading to multiple-comparison concerns.

[12] The four variants of the APOE gene are determined by two SNPs: rs429358 and rs7412. The E4 variant is defined as having a "C" variant for each.

has a negative association with neuroplasticity.[13] When constructing our measure for neuroplasticity, we consider individuals who do not have the E4 variant for the *APOE* gene.

The Val66Met locus (rs6265) of the *BDNF* (brain-derived neutrophic factor) gene has been shown to be related to neuroplasticity.[14] *BDNF* is a protein associated with nerve growth, which is influenced by both the genotype as well as by early life stress (Roceri et al. 2002, Duman and Monteggia 2006). The presence of this protein is positively associated with hippocampal plasticity and development (Mizuno et al. 2000, Witte et al. 2012). Therefore, the genetic variant associated with greater production of this protein, and greater robustness to early stress—the Val, or "A" variant of SNP rs6265—is considered for our measure of neuroplasticity.

The *COMT* gene is associated with the production of the catechol-O-methyl transferase protein, an enzyme that breaks down catecholamines--e.g., dopamine. The Met variant of the Val158Met locus (rs4680) is associated with reduced production of *COMT* and a higher corresponding level of dopamine in the prefrontal cortex. This particular variant of the *COMT* gene has a debatable, direct impact on memory.[15] For our purposes, the Met variant of the *COMT* gene, and the corresponding increase in prefrontal dopamine, is theorized to interact with *BDNF* by increasing long-term potentiation, a key determinant of neuroplasticity.[16] This is confirmed in Witte et al. (2012), who find the Met variant of the rs4680 SNP in *COMT* has an interactive effect with the Val variant of the rs6265 SNP in the *BDNF* gene in increasing neuroplasticity.

[13] The negative association between the E4 variant and plasticity may be one reason for its correlation with Alzheimer's disease (Teter and Ashford 2002).
[14] For review, please see Cheeran et al. (2009).
[15] For review, please see Witte and Floel (2011).
[16] Long-term potentiation is the strengthening of neural pathways by a persistent increase in signal between two neurons (Bliss and Collinridge 1993).

Our baseline measure for neuroplasticity is the interaction between the number of favorable variants of each neuroplasticity gene mentioned above, where every individual has two copies for each gene—one from the mother and one from the father. For *APOE*, the favorable variant is simply not having the E4 variant; for *BDNF*, the Val allele is favorable for neuroplasticity; and for *COMT*, the Met allele is favorable. Therefore, each individual has 0, 1, or 2 favorable variants, and the score for the interaction between the three neuroplasticity genes is 0, 1, 2, 4, or 8 (see Figure 2 below for the distribution in our sample). The interaction between the individual neuroplasticity genes is intended to capture the previously found interaction between BDNF and COMT, as well as the strong negative effects associated with the E4 variant of APOE. As a check to this specification, we also explore the additive impact for the total number of favorable variants as well as an indicator for individuals who have at least one copy of each favorable gene variant. Appendix B also shows results for each gene in isolation.

In summary, neuroplasticity represents the ability of an individual to respond to cognitively damaging environments—i.e., poor in utero nutrition. To measure neuroplasticity, we explore three genes that have a biological association with neuroplasticity. We hypothesize that the neuroplasticity genes under consideration moderate the effect of birth weight, or early nutrition, on cognition and adult wages.

2. Data and Empirical Methodology

2.1 Data

The data come from the Wisconsin Longitudinal Study (WLS). The WLS is a random sample composed of one-third of 1957 high school graduates. Additional data has been collected from a selected sibling and from spouses of the graduates.

Of importance to the current work is data on both the graduates and their selected siblings, for which data were collected in 1957, 1964, 1975, 1992, and 2003 and 1977, 1993, and 2004, respectively.

Our base sample consists of 469 sibling pairs (938 individuals). As will be explained shortly, the sibling sample is needed to control for both unobserved genes and environments, which may lead to spurious estimates. The WLS contains data on IQ, our primary dependent variable of interest, for roughly 17,000 individuals: 11,000 graduates and 6,000 selected siblings.[17] A large portion of this available data is unused, however, due to the availability of birth weight and DNA data, which were collected in 2003 (2004), a time when the graduates and siblings were roughly 60 years of age. The sample of individuals who report both birth weight and DNA data is less than one-third of our 17,000 sample, from which data are used for roughly 3,800 individuals: 2,600 graduates and 1,200 siblings. Furthermore, data for complete sibling pairs reduces the sample of individuals with DNA and birth weight data from 3,800 to 938. This reduced sample constitutes our base sample and is composed of sibling pairs with complete data on DNA, birth weight, IQ, and other covariates used in estimation.

Due to the birth weight and DNA data being collected at such a late period, sample selection is a concern. It is likely that surviving until the 2003 (2004) wave is correlated with IQ and other economic and health measures. Therefore, we construct an attrition weight by first regressing an indicator for being in the sibling-pair sample on IQ, birth year, sex, and a sibling indicator.[18] Next, the inverse of this probability is used as an attrition weight in estimation, potentially correcting for issues associated with sample selection. Comparing summary statistics across sample specifications in the appendix, the sample

[17] These data are truncated slightly by the availability of other covariates used within the paper. The appendix contains summary statistics for a large number of possible samples.
[18] Siblings have greater variation in age, as well as compromising a greater proportion of individuals with birth weight and DNA data.

weights appear to correct for the differences in IQ, as well as other variables. Indeed, the (weighted) summary statistics for the analysis sample is nearly identical to the full sample of 17,000 individuals (see Table A1).[19]

Our primary dependent variable throughout the paper is IQ, which is mapped from Henmon-Nelson test scores and is representative of IQ for high school juniors. As an additional dependent variable, we also use the hourly wage rate in 1992 (1993). This variable is intended to capture economic productivity differences. The data from 1992 (1993) represent a period in which most of our sample is working (i.e, graduates are roughly 53 years of age) and has the greatest amount of coverage across waves.

As described in Section 1.1, our main independent variable of interest is neuroplasticity, which is measured in several ways, including by the interaction between 3 genes associated with neuroplasticity. These 3 genes are *APOE*, *BDNF*, and *COMT*. The plastic allele of *APOE* is defined as not being the E4 variant. The E4 variant is represented by having a "C" variant at both SNP rs429358 and SNP rs7412.[20] The plastic variant is coded as 2 if an individual has no copies of the E4 variant, as 1 if the individual contains one copy of the E4 variant, and 0 for two copies.[21] For *BDNF*, the Val variant is seen as plastic. This is defined as having a "G" variant at SNP rs6265, where having two "G" variants is coded as 2, one "G" variant is coded as 1, and having no "G" variants

[19] The Vital Statistics of 1950 provide the first national data on birth weight. Using this data as a proxy for a nationally representative birth weight cohort, we are able to assess the validity of the birth weight statistics for our base sibling sample, the majority of which were born in 1940. For 1950, the average birth weight in the U.S. is 3,310 grams, which is comparable with 3,367 grams, the average birth weight of our sibling sample. For whites in 1950, which is more representative of our base sibling sample, the average birth weight is 3,320 grams. Furthermore, 7.6% of births are less than 2,500 grams in 1950 compared to roughly 8% for our sibling sample.

[20] In our base sibling sample, the frequencies of the "C" variant for SNP rs429358 and rs7412 are 14.9% and 92%, respectively; this is comparable to frequencies in the 1000 Genomes project, which give frequencies in European populations of 14% and 93% (McVean et al. 2012).

[21] Every individual has two copies of each gene: one from the mother and one from the father.

is coded as 0.[22] For *COMT*, the Met variant is associated with neuroplasticity. The "A" variant of SNP rs4680 is associated with lower enzymatic activity and increased plasticity. The "A" variant for this SNP of *COMT* is coded in an identical manner to the "G" variant of SNP rs6265 for *BDNF*.[23]

Our main measure of neuroplasticity is the interaction across these three genes (i.e., *APOE*× *BDNF*×*COMT*), giving a minimum of 0 and maximum of 8. The interaction is used to measure neuroplasticity because the presence of all three traits is necessary for plasticity: *BDNF* and *COMT* have a previously documented interaction in plasticity, whereas the E4 variant of *APOE* is highly damaging to plasticity (Trommer et al. 2005, Witte et al. 2012). For our base sibling sample, the mean of this interaction is 2.87, with individuals containing on average 4.35 plastic alleles. For robustness, we will also use the number of plastic alleles and an indicator for the interaction being greater than zero as well as examine effects for each gene individually in Appendix B. Other covariates include sex, birth order, birth year, a sibling indicator, mother's education, father's education, and a score for socio-economic status, though several of the measures (as well as all shared family characteristics) will be subsumed in our preferred family fixed effects specification.

An additional issue in the use of our sample (and the WLS more broadly) is the lack of ethnic diversity. Our base sibling sample is composed entirely of peoples of European descent.[24] This implies that any findings may not be generalizable to a larger, more ethnically diverse population such as the United States. The ethnic homogeneity, however, is beneficial in our study of genes and

[22] In comparison to frequencies in the 1000 Genomes Project, our base sample frequency of the Val variant is 80.5%, whereas the European frequency in 1000 Genomes is 80% (McVean et al. 2012).
[23] In our base sibling sample, the frequency of the Met variant of rs4680 is 51.1%. This is roughly identical to the 52% frequency within European populations (McVean et al. 2012).
[24] Less than 1% of the WLS is composed of non-white ethnicities.

environments. Our results are unlikely to be biased by genetic, or environmental, clustering across ethnicities.[25]

2.2 Empirical Methodology

Our main empirical strategy considers the interaction between our genetic measure of neuroplasticity and exposure in early childhood, or in utero, to poor nutrition, for which birth weight is used as a proxy. Our main estimating equation is given by the following form:

$$IQ_{ij} = \beta_0 + \beta_1 NP_{ij} + \beta_2 BW_{ij} + \beta_3 NP_{ij} \times BW_{ij} + \boldsymbol{\beta}_4' \mathbf{X_{ij}} + \boldsymbol{\beta}_5' \mathbf{Z_j} + \gamma I_j + \varepsilon_{ij} \quad (1)$$

where we consider i individuals within j families. Our main outcome of interest is IQ, and the coefficient of interest is β_3, which measures the effect on the interaction between birth weight and neuroplasticity. Our hypothesis being that β_3 is negative and significant, while the main effect of birth weight, measured by β_2, is positive and significant. This finding would confirm that the early nutrition environment does play a role in cognitive development, but that this effect is lessened for plastic individuals. All estimations also include individual and family level controls—represented by $\boldsymbol{X}_{ij}$ and $\boldsymbol{Z}_j$, respectively—that include family SES and education, individual demographic characteristics, as well as within family dynamics, such as birth order, that have effects on learning and cognition (Black et al. 2005).

In addition to individual and family level controls, our extended estimating equation includes family, or sibling, fixed effects.[26] As argued in Fletcher and Lehrer (2011), Conley and Rauscher (2012), and Cook and Fletcher (2013), the inclusion of sibling fixed effects control for both unobserved genetic and environmental influences, which may bias estimation. Since siblings share roughly 50% of unique genetic variation, sibling fixed effects partially (i.e., 50%)

[25] Within family estimation should also eliminate bias associated with population stratification.
[26] In the sibling fixed effects specification, family-level controls are excluded.

control for unobserved genetic influences that may bias the gene-environment interaction by accounting for a latent gene-gene interaction.[27] In addition to controlling for potentially unobserved GG interactions, the use of sibling fixed effects also allows us to control, in part, for heritable IQ shared between siblings (Black et al. 2009). In addition, the use of sibling fixed effects allows the genetic variation in our sample to be considered quasi-exogenous, as differences in genotype of full biological siblings is the outcome of a "genetic lottery" (Fletcher and Lehrer 2009, 2011).

While controlling for unobserved genetic variation is important in verifying the effect of the GE interaction, it is also important to control for unobserved environmental differences across families. Birth weight is plausibly exogenous between siblings, but may be associated with income or education across families. We are able to control for parental income and education; however, there are many unobservables across families (e.g., parental attention, post-natal nutrition, childhood activities, quality of education, etc.) that are associated with cognitive development and high school IQ. Furthermore, across family variation is strongly associated with cognitive development (Carneiro and Heckman 2003, Cunha et al. 2006, Heckman and Masterov 2004). Therefore, the within family specification is preferred. We show descriptive statistics for our sibling sample based on discordant/concordant siblings in Appendix A.

All tables are organized as following: column (1) presents OLS estimates of the estimating equation above, excluding sibling fixed effects; column (2) weights the estimation of column (1) by the inverse of the probability of being in our base sibling-pair sample; and column (3) performs within family estimation, excluding family-level controls.

[27] Indeed, the principle of the independent assortment of genes across the genome (especially genes not in close proximity) suggests a limited role for gene-gene interactions as a possible alternative-hypothesis for our results.

3. Results

3.1 Baseline

The main effects of neuroplasticity, measured by the interaction of corresponding gene variants, and birth weight on high school IQ are examined in Table 1. All columns in Table 1 include all relevant individual and family level controls, which include birth order, birth year, a sibling indicator, sex, mother's education, father's education, and family-level SES in 1957 with standard errors clustered at the family level. The OLS estimation of column (1) shows that while birth weight does have a positive and significant effect on later-life IQ, our genetic measure for neuroplasticity has no direct association with IQ. This is to be expected; our hypothesis is that neuroplasticity is associated with correcting cognitively harmful environments, not directly improving IQ; indeed, our genetic variants of interest have not been shown to have direct effects on IQ in gene discovery exercises from the genetics literature. The estimated coefficient of birth weight in column (1) implies that a one standard deviation increase in birth weight is associated with roughly one and a half points increase in IQ. Considering the findings in Newcombe et al. (2007), our estimated effect of birth weight is not significantly different than the finding of a one kilogram increase in birth weight being associated with a 3 point increase in IQ. For our coefficient, an increase of one kilogram (or roughly 1.5 standard deviations) is predicted to increase IQ by 2.2 points, for which the 95% confidence interval contains the Newcombe et al. estimate of 3 points.

The estimates of columns (2) and (3), which weight the estimation by the inverse of the probability of being within our base sibling sample and include sibling fixed effects, respectively, do not differ substantially from the simple OLS findings of column (1). The estimates of column (2) provide evidence that the significant, positive association of birth weight on IQ and the insignificant

statistical association between our measure of neuroplasticity and IQ is not being driven by our sample of sibling pairs with both DNA and birth weight data. Furthermore, these findings are confirmed by within family estimation of column (3), reducing the possibility of a spurious relationship due to unobserved sibling-shared genetic or environmental variables. In summary, birth weight has a direct effect on IQ, while our measure of neuroplasticity has no direct association with IQ.

Table 2 regresses birth weight on our genetic measure for neuroplasticity. The purpose of Table 2 is to establish the independence of our gene and environment. Our primary focus is on the interaction between genes for neuroplasticity and birth weight, a proxy for the early nutrition environment, with the main hypothesis being that individuals with more genetic variants related to neuroplasticity are less likely to be influenced by the early nutrition environment. Therefore, in order to ensure a true interaction, not a spurious correlation, we need to ensure that our genes of interest are not influencing our environment of interest. The estimates of Table 2 provide no evidence that the genes selected to measure neuroplasticity are influencing birth weight. Additionally, genome wide association studies provide no evidence for a significant association between our proposed neuroplasticity variants and birth weight (Freathy et al. 2010). We have no reason to believe gene-environment correlation will lead to a spurious coefficient for our gene-environment interaction investigation.

3.2 Gene-Environment Interaction

Figure 1 plots the relationship between birth weight and high school IQ for those individuals with at least one plastic allele for each gene and those without. The positive association between birth weight and IQ is much more pronounced for those individuals who do not have a plastic allele for each of the three neuroplasticity genes under consideration. Conversely, plastic individuals do not

exhibit any significant association between birth weight and IQ. The findings of Figure 1 support the differential response to the early nutrition environment based upon genotype.

The estimates of Table 3 confirm the findings of Figure 1 and also provide a basis for the gene-environment estimation. Columns (1) and (2) show that birth weight has a positive and highly significant effect on IQ for individuals who have zero plastic variants for each neuroplasticity gene under consideration. The coefficient is roughly identical for both the simple OLS specification of column (1) and the within family model of column (2). For column (2), a one standard deviation increase in birth weight is associated with an increase of 3 points in IQ. The magnitude of the coefficient for non-plastic individuals is roughly twice that found in Table 1, which includes both plastic and non-plastic siblings.

Columns (3) and (4) replicate the estimation of columns (1) and (2) but restrict the sample to more plastic individuals. As a result, the coefficient of birth weight is reduced by roughly two-thirds and becomes statistically indistinguishable from zero. The findings of Table 3 support the differential impact of birth weight based upon markers for neuroplasticity: More plastic individuals gain no benefit from added birth weight, whereas less plastic individuals have a strong cognitive association to the in utero nutrition environment.

Our baseline estimating equation is explored in Table 4. The estimates of Table 4 test our main hypothesis: the effect of the early nutrition environment is moderated by individuals with flexible, or easily repaired, neural networks. A negative coefficient on the GE interaction provides support for this hypothesis by lessening the marginal effect of birth weight, for which a positive and significant association with IQ is found in Table 1. Column (1) of Table 4 estimates Equation 1 while excluding sibling fixed effects. All signs are as expected.

Importantly, the coefficient on the GE interaction is negative and significant at the 1% level. As an individual contains more and more plastic alleles, the positive marginal effect of birth weight dissipates. For the mean number of plasticity alleles, the marginal effect of birth weight is halved; while for individuals with the maximum number of plasticity alleles the marginal effect of birth weight becomes negative.

The estimates of Table 4 support our main hypothesis. Birth weight has a positive association with IQ for less plastic individuals but does not have a lasting effect for individuals with numerous plastic alleles. Importantly, this effect is consistent is across all of our estimation specifications, providing evidence that this effect is consistent within and across families and that bias due to sample selection is unlikely.

Table 5 re-estimates the findings of Table 4 with two alternative measures for neuroplasticity. Panel A considers an indicator for those who have at least plastic allele for each neuroplasticity gene under consideration. The use of the dummy is to broadly capture the more plastic individuals. As in Table 4, the interaction between this indicator and birth weight is negative, implying that those more broadly defined plastic individuals are less affected by the early nutrition environment. Specifically for our within family estimation of column (3), the effect of birth weight on IQ is reduced by two-thirds for individuals with at least one plastic allele for each gene. For Panel B, we consider an additive, instead of interactive, measure for our neuroplasticity genes. With our 3 neuroplasticity genes and 2 copies of each gene, the maximum number of favorable alleles is 6. Looking at the estimates within Panel B of Table 5, again, the interaction with birth weight is negative, while the main effect of birth weight is positive. More importantly when considering the within family estimates of column (3), the marginal effect of birth weight goes to zero as the number of plastic alleles approaches the maximum. We also show in Appendix B that the results are

qualitatively similar if we use each gene variant in isolation and also vary the measurement of neuroplasticity.

Given that neuroplasticity moderates the effects of birth weight on IQ, we should expect to see a similar effect on labor market outcomes, particularly wage. Table 6 considers wage, or productivity, in place of IQ.[28] Our wage measure comes from the 1992-3 wave of the WLS, when the graduates and siblings are on average in their mid-fifties, a time just prior to retirement when wages are likely to be at a lifetime peak. Panel A of Table 6 gives the main effect of our neuroplasticity measure and birth weight on the natural log of hourly wage in 1992-3, whereas Panel B gives estimates for our gene-environment interaction model.

For all estimations of Panel A in Table 6, our coefficients of interest, the coefficients of birth weight and neuroplasticity, are insignificantly different than zero but the sign and magnitude of the effects are as expected. In previous papers, birth weight has been shown to have a positive and significant impact on earnings; e.g., Black et al. (2007) show that a 10% increase in birth weight is associated with a 1% increase in income. For column (3) in Panel A, our sibling fixed effects model, a 10% increase in birth weight (~1/2 a standard deviation) is associated with a 5% increase in wage, with a 1% increase contained within the 95% confidence band. The insignificance of our estimates for the coefficient of birth weight may be tied to the relatively small sample for which we have data. Additionally, insignificance may be due to the absence of accounting for the differential effect of birth weight from neuroplasticity genes.

For Panel B, we estimate our proposed interaction model, replacing IQ with wages later in life. The estimated effects of neuroplasticity, birth weight, and the interaction between the two variables, however, are similar to the

[28] Appendix D replicates the findings of Table 6 with the previously specified alternative measures of neuroplasticity.

regressions with IQ. Birth weight has a positive and statistically significant association with the wage rate, but the magnitude of this effect is dependent upon our measure for neuroplasticity. When considering the sibling fixed effects estimation of column (3), a 10% increase in birth weight is associated with a 15% increase in wage for individuals with at least one plastic allele for each neuroplasticity gene. For the mean neuroplasticity score, a 10% increase in birth weight is associated with roughly a 5% increase in wage. And for the maximum neuroplasticity score, a 10% increase in birth weight is negatively associated with wage. As with IQ, neuroplasticity moderates the effects of birth weight on later life outcomes.

4. Conclusion

This research questions the ubiquity of fetal programming for cognitive and productivity outcomes and explores the potential policy implications of understanding biological and genetic explanations of heterogeneity in the impacts of birth weight. Our hypothesis is that a portion of the population is more adaptable to early exposure to environmental insults in regards to cognitive outcomes and the basis of this resiliency stems from the genetics and biology of the developmental process. This has important consequences for our understanding of the determinants of long term outcomes, such as IQ and wages as well in our understanding of the heterogeneity of theses determining processes. More speculatively, our findings may also eventually be used in designing and targeting public policies that attempt to blunt the effects of poor in utero environments. If, for example, individuals do indeed respond differently to early life environments, public policy applications that have the potential to discriminately apply interventions may (eventually) help in obtaining policy goals.

In summary, we argue that neuroplasticity, which we measure through genetic variation, moderates the impact of the early, in utero nutrition environment. Previous studies have established a strong link between birth weight and a host of economic and health outcomes. We focus on cognitive development and show that the positive association between birth weight and IQ dissipates for individuals who have genetic variants associated with neuroplasticity, where neuroplasticity represents an ability of the brain to correct, or adjust to, harmful prenatal environments. We extend this finding into a labor market outcome and show that the association between birth weight and productivity exhibits the same relationship to neuroplasticity as IQ. We also show our findings are quite robust to a variety of alternative measures of neuroplasticity.

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Tables and Figures

Table 1. Main Effects of Birth Weight and Neuroplasticity Genes on IQ

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Neuroplasticity Genes	0.2633	0.2740	-0.1799
	(0.1814)	(0.1866)	(0.2970)
Standardized Birth Weight	1.4041***	1.4510***	1.4140**
	(0.4858)	(0.5034)	(0.6672)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1333	0.1323	0.6953

Notes: (i) Neuroplasticity Genes is the interaction between the count of plastic alleles for *APOE*, *BDNF*, and *COMT* (ii) Demographic and family controls include race, sex, birth year (age), birth order, mother's education, father's education, and a score for family SES in 1957. (iii) Standard errors are clustered at the family level with *, **, and *** representing significance at the 10, 5, and 1% significance level, respectively.

Table 2. Effects of Neuroplasticity Genes on Birth Weight

Dependent Variable: Standardized Birth Weight			
	(1)	(2)	(3)
Neuroplasticity Genes	0.0114	0.0167	0.0232
	(0.0129)	(0.0135)	(0.0224)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.0450	0.0441	0.6320

Notes: (i) Neuroplasticity Genes is the interaction between the count of plastic alleles for *APOE*, *BDNF*, and *COMT* (ii) Demographic and family controls include race, sex, birth year (age), birth order, mother's education, father's education, and a score for family SES in 1957. (iii) Standard errors are clustered at the family level with *, **, and *** representing significance at the 10, 5, and 1% significance level, respectively.

Table 3. Differential Effects of Birth Weight

Dependent Variable: High School IQ				
Sample	$BDNF \times COMT \times APOE = 0$		$BDNF \times COMT \times APOE > 0$	
	(1)	(2)	(3)	(4)
Standardized Birth Weight	2.8333***	3.0411**	0.7897	0.9093
	(0.9687)	(1.5217)	(0.5482)	(0.8485)
Controls				
Demographic and Family SES	Y	Y	Y	Y
Sibling Fixed Effects	N	Y	N	Y
Test				
p-value for coeff. of col. (1) = coeff. of col. (3)	–	–	0.0002	–
p-value for coeff. of col. (2) = coeff. of col. (4)	–	–	–	0.0124
N	266	266	672	672
R Sqr.	0.1817	0.8116	0.1202	0.7397

Notes: (i) Columns (1) and (2) restrict the sample to individuals that have no plastic alleles for at least one of our neuroplasticity genes. Columns (3) and (4) restrict the sample to individuals with at least one plastic variant for each neuroplasticity gene. (ii) The estimation of Table 3 is represented graphically by Figure 1. (iii) Demographic and family controls include race, sex, birth year (age), birth order, mother's education, father's education, and a score for family SES in 1957. (iv) Standard errors are clustered at the family level with *, **, and *** representing significance at the 10, 5, and 1% significance level, respectively.

Table 4. Effect of Interaction between Birth Weight and Neuroplasticity Genes on IQ

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Neuroplasticity Genes	0.2556	0.2642	-0.1567
	(0.1804)	(0.1851)	(0.2963)
Standardized Birth Weight	2.6416***	2.7838***	2.8848***
	(0.7171)	(0.7502)	(0.9541)
G × E	-0.4232***	-0.4574***	-0.4617**
	(0.1578)	(0.1719)	(0.2009)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1390	0.1390	0.6985

Notes: (i) Neuroplasticity Genes is the interaction between the count of plastic alleles for *APOE*, *BDNF*, and *COMT*. G × E represents the interaction between our measure of neuroplasticity and standardized birth weight. (ii) Demographic and family controls include race, sex, birth year (age), birth order, mother's education, father's education, and a score for family SES in 1957. (vi) Standard errors are clustered at the family level with *, **, and *** representing significance at the 10, 5, and 1% significance level, respectively.

Table 5. G×E: Alternative Measures for Neuroplasticity

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Panel A: Neuroplasticity Genes = Indicator for $BDNF \times COMT \times APOE > 0$			
Neuroplasticity Genes	0.9378	0.8035	-3.5792**
	(1.0315)	(1.0381)	(1.5526)
Standardized Birth Weight	2.7535***	3.1386***	3.1751***
	(0.9134)	(0.9521)	(1.2084)
G × E	-1.8931*	-2.3416**	-2.2956*
	(1.0246)	(1.0728)	(1.3511)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1353	0.1357	0.7000
Panel B: Neuroplasticity Genes = BDNF + COMT + APOE			
Neuroplasticity Genes	0.4435	0.4294	-0.0497
	(0.4343)	(0.4319)	(0.7932)
Standardized Birth Weight	5.2976***	5.6284***	6.1441**
	(1.7969)	(1.8188)	(2.4866)
G × E	-0.8961**	-0.9595**	-1.0691**
	(0.3841)	(0.3933)	(0.5239)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1366	0.1360	0.6978

Notes: (i) For Panel A, neuroplasticity is measured by an indicator for having at least one plastic allele for each gene. For Panel B, the count of all plasticity genes is used to measure neuroplasticity. G × E represents the interaction between each respective measure of neuroplasticity and standardized birth weight. (ii) Demographic and family controls include race, sex, birth year (age), birth order, mother's education, father's education, and a score for family SES in 1957. (vi) Standard errors are clustered at the family level with *, **, and *** representing significance at the 10, 5, and 1% significance level, respectively.

Table 6. Effect of Interaction on Productivity

Dependent Variable: ln Wage Rate in 1992			
	(1)	(2)	(3)
Panel A: Main Effects			
Neuroplasticity Genes	-0.0141	-0.0147	-0.0187
	(0.0164)	(0.0164)	(0.0311)
Standardized Birth Weight	0.0501	0.0526	0.1008
	(0.0473)	(0.0476)	(0.0786)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	820	820	820
R Sqr.	0.0919	0.0942	0.6009
Panel B: Gene-Environment Interaction			
Neuroplasticity Genes	-0.0130	-0.0136	-0.0104
	(0.0165)	(0.0164)	(0.0311)
Standardized Birth Weight	0.1224**	0.1212**	0.3155***
	(0.0526)	(0.0525)	(0.1036)
G × E	-0.0241*	-0.0231*	-0.0664***
	(0.0129)	(0.0127)	(0.0209)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	820	820	820
R Sqr.	0.0936	0.0957	0.6069

Notes: (i) Neuroplasticity Genes is the interaction between the count of plastic alleles for *APOE*, *BDNF*, and *COMT*. G × E represents the interaction between our measure of neuroplasticity and standardized birth weight. (ii) Demographic and family controls include race, sex, birth year (age), birth order, mother's education, father's education, and a score for family SES in 1957. (vi) Standard errors are clustered at the family level with *, **, and *** representing significance at the 10, 5, and 1% significance level, respectively.

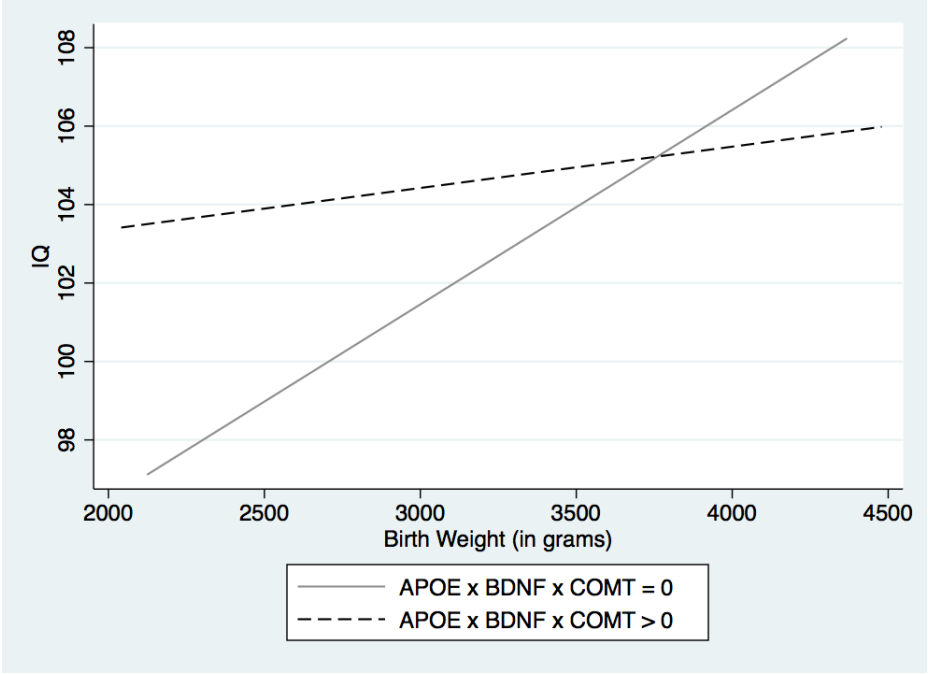

Figure 1
Differential Effects of Birth Weight by Neuroplasticicity

Notes: (i) This figure plots the effect of birth weight on IQ for two distinct populations, which are based on the number of neuroplasticity alleles. For individuals with at least one copy of a plastic allele for each neuroplasticity gene (N=672), the effect of birth weight is lessened when compared to individuals with no plastic alleles for at least one of our neuroplasticity genes (N=266). (ii) Slope coefficients, conditional on family and individual controls, are found in Table 3.

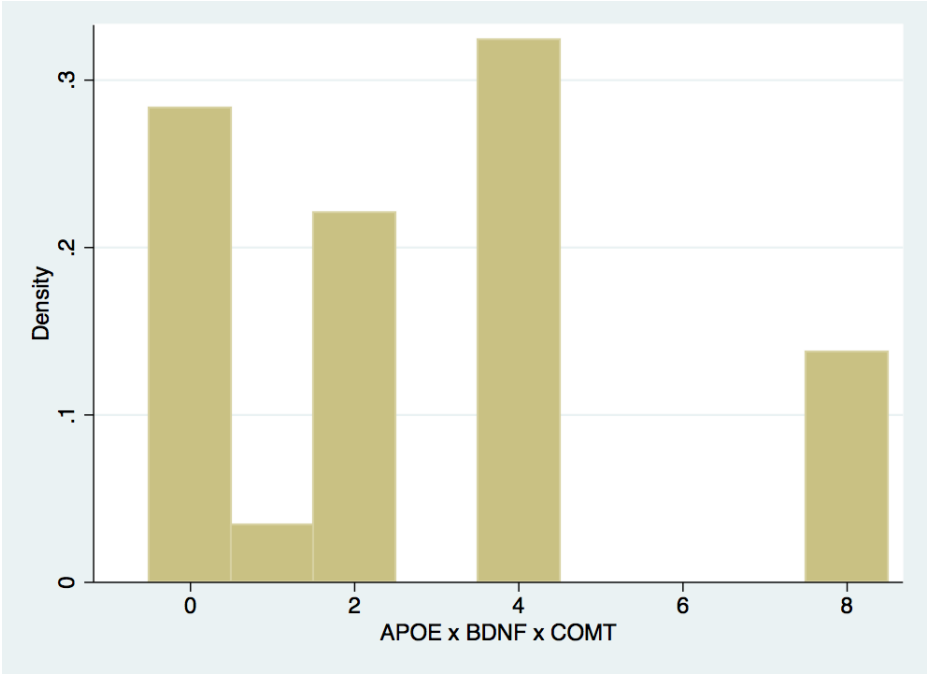

Figure 2
Distribution of Base Neuroplasticity Measure

Notes: (i) This figure gives the distribution for the interaction between BDNF, COMT, and APOE, the main measure of neuroplasticity used within the paper. Each SNP is defined by the count, or number, of plastic variants.

Appendix A: Summary Statistics and Sample Selection

Table A1. Summary Statistics for Differing Samples

	Base Sibling (Unweighted)	Base Sibling (Weighted)	All with DNA and Birth Weight	All with DNA	All with Birth Weight	Max
	(1)	(2)	(3)	(4)	(5)	(6)
IQ	104.19	101.43	103.73	103.46	102.61	101.38
	(14.82)	(14.67)	(14.80)	(14.95)	(14.85)	(15.27)
Mother's Education	10.77	10.67	10.70	10.62	10.63	10.51
	(2.78)	(2.77)	(2.76)	(2.80)	(2.74)	(2.79)
Father's Education	10.17	10.03	9.97	9.88	9.87	9.76
	(3.38)	(3.36)	(3.41)	(3.42)	(3.39)	(3.41)
Family SES in 1957	17.52	16.96	17.01	16.75	16.75	16.40
	(11.65)	(11.45)	(11.08)	(11.10)	(11.03)	(11.03)
Birth Year	1939.66	1939.24	1939.31	1939.21	1939.34	1939.24
	(4.27)	(3.75)	(3.57)	(3.65)	(3.72)	(4.39)
Female	0.55	0.51	0.56	0.52	0.58	0.52
	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)
Birth Order	2.34	2.30	2.36	2.47	2.38	2.51
	(1.42)	(1.40)	(1.67)	(1.78)	(1.68)	(1.80)
Birth Weight (in grams)	3367.55	3363.75	3378.36	–	3374.32	–
	(631.85)	(635.38)	(630.02)	–	(636.72)	–
Neuroplasticity Genes	2.87	2.88	2.94	2.95	–	–
(Interaction)	(2.58)	(2.59)	(2.56)	(2.57)	–	–
Neuroplasticity Genes	4.35	4.35	4.39	4.40	–	–
(Count)	(1.06)	(1.06)	(1.04)	(1.03)	–	–
N	938	938	3799	6097	6452	15676

Notes: Columns (1) and (2) are comprised of our base sibling pair sample. Column (3) consists of all individuals (i.e., not pairs) that contain data for the SNPs used to measure neuroplasticity and birth weight. Column (4) consists of all individuals with data for neuroplasticity SNPs and column (5) consists of all individuals with data for birth weight. Column (6) gives sample statistics for the maximum available sample. SES is an index created from father's education, mother's education, father's occupation (Duncan SEI), and family income.

Table A2. Summary Statistics for Differing Samples of Siblings

	Siblings Pairs with Data for:			
	IQ	Birth Weight	DNA	BW + DNA
	(1)	(2)	(3)	(4)
IQ	101.35	103.25	104.56	104.19
	(15.37)	(14.85)	(15.02)	14.82
Birth Weight (in grams)		3394.96		3367.55
		(636.82)		(631.85)
Neuroplasticity Genes (Count)			4.39	4.35
			(1.04)	(1.06)
Mother's Education	10.52	10.75	10.68	10.77
	(2.77)	(2.67)	(2.78)	(2.78)
Father's Education	9.74	9.98	9.97	10.17
	(3.35)	(3.37)	(3.35)	(3.38)
Family SES in 1957	16.17	16.82	16.77	17.52
	(10.93)	(11.01)	(11.26)	(11.65)
Birth Year	1939.29	1939.69	1939.49	1939.66
	(4.84)	(4.63)	(4.52)	(4.27)
Female	0.52	0.56	0.52	0.55
	(0.52)	(0.50)	(0.50)	(0.50)
Birth Order	2.54	2.35	2.50	2.34
	(1.73)	(1.48)	(1.66)	(1.42)
N	13224	2360	2246	938

Notes: Column (1) restricts the sample to sibling pairs containing data on IQ as well as our base set of controls. Column (2) gives sample statistics for sibling pairs containing data on birth weight, while column (3) restricts the sample to sibling pairs with data for our neuroplasticity SNPs. Column (4) restricts the sample to sibling pairs with both birth weight and DNA data; this represents our base sibling sample.

Table A3. Summary Statistics: Siblings with Discordant Genotypes

	Siblings Pair Sample:		
	Base	Concordant	Discordant
	(1)	(2)	(3)
IQ	104.19	104.68	103.60
	(14.82)	(15.08)	(14.50)
Birth Weight	3367.55	3370.68	3363.72
(in grams)	631.85	(636.29)	(627.12)
Neuroplasticity Genes	4.35	4.50	4.17
(Count)	(1.06)	(0.97)	(1.13)
N	938	516	422

Notes: This table separates the base sibling sample by differences in sibling genotypes. Column (1) gives our base sample; column (2) considers only siblings that are concordant in regards to our base neuroplasticity measure (i.e., the interaction between BDNF, COMT, and APOE); and column (3) gives sample statistics for siblings who differ in genotype.

Table A4. Base Estimation: Siblings with Discordant Genotypes

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Neuroplasticity Genes	0.0944	0.0742	-0.1738
	(0.2450)	(0.2519)	(0.3026)
Standardized Birth Weight	3.6768***	3.4344***	3.2309**
	(1.0546)	(1.0969)	(1.4674)
G×E	-0.4725**	-0.4075**	-0.3605
	(0.1919)	(0.2027)	(0.2883)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	516	516	516
R Sqr.	0.1664	0.1663	0.7208

Notes: This table performs our base estimation (Table 4) with sibling pairs that are discordant in regards to neuroplasticity genes.

Table A5. Summary Statistics: Siblings with Discordant Environment (Birth Weight)

	Siblings Pair Sample:		
	Base	Concordant	Discordant
	(1)	(2)	(3)
IQ	104.19	104.16	104.19
	(14.82)	(15.34)	(14.81)
Birth Weight	3367.55	3626.16	3354.82
(in grams)	631.85	(628.58)	(629.62)
Neuroplasticity Genes	4.35	4.47	4.34
(Count)	(1.06)	(0.0.85)	(1.07)
N	938	44	894

Notes: This table separates the base sibling sample by differences in sibling birth weight. Column (1) gives our base sample; column (2) considers only siblings that have identical birth weights; and column (3) gives sample statistics for siblings who differ in birth weight.

Table A6. Base Estimation: Siblings with Discordant Environment (Birth Weight)

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Neuroplasticity Genes	0.2391	0.2055	-0.1983
	(0.1870)	(0.1892)	(0.3062)
Standardized Birth Weight	2.6730***	2.7501***	2.9996***
	(0.7305)	(0.7454)	(0.9617)
G×E	-0.4410***	-0.4820***	-0.4819**
	(0.1626)	(0.1738)	(0.2021)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	894	894	894
R Sqr.	0.1414	0.1390	0.6994

Notes: This table performs our base estimation (Table 4) with sibling pairs that are discordant in regards to birth weight.

Table A7. Summary Statistics: Siblings with Discordant Gene-Environment Interaction

	Siblings Pair Sample:		
	Base	Concordant	Discordant
	(1)	(2)	(3)
IQ	104.19	102.54	104.58
	(14.82)	(14.67)	(14.84)
Birth Weight	3367.55	3349.49	3371.83
(in grams)	631.85	(639.55)	(630.36)
Neuroplasticity Genes	4.35	3.29	4.60
(Count)	(1.06)	(0.97)	(0.92)
N	938	180	758

Notes: This table separates the base sibling sample by differences in sibling birth weight. Column (1) gives our base sample; column (2) considers only siblings that have identical birth weights; and column (3) gives sample statistics for siblings who differ in birth weight.

Table A8. Base Estimation: Siblings with Discordant Gene-Environment Interaction

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Neuroplasticity Genes	0.0865	0.0745	-0.1515
	(0.2132)	(0.2226)	(0.2979)
Standardized Birth Weight	2.7858***	2.4014**	2.4742*
	(0.9148)	(0.9628)	(1.2621)
G×E	-0.4444**	-0.4000**	-0.3786
	(0.1856)	(0.1995)	(0.2464)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	758	758	758
R Sqr.	0.1411	0.1326	0.7072

Notes: This table performs our base estimation (Table 4) with sibling pairs that are discordant in regards to the interaction between birth weight and the base measure (i.e., interaction) of neuroplasticity genes.

Appendix B: Effects of Individual SNPs

Table B1. BDNF

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Panel A: BDNF = Categorical			
BDNF	0.0045	0.0708	1.4912
	(0.8455)	(0.8282)	(1.2867)
Standardized Birth Weight	2.0633	1.3300	2.7466
	(1.6560)	(1.5164)	(2.0975)
G × E	-0.3912	0.0604	-0.8219
	(0.9269)	(0.8776)	(1.1457)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1314	0.1274	0.6964
Panel B: BDNF = Indicator for at Least One Plastic Allele			
BDNF	-2.7718	-3.2794*	-1.2093
	(2.1192)	(1.9548)	(2.4292)
Standardized Birth Weight	4.9296*	4.9697*	1.3985
	(2.8294)	(2.5840)	(3.5031)
G × E	-3.5965	-3.6098	0.0130
	(2.8408)	(2.6162)	(3.5019)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1336	0.1300	0.6952
Panel C: BDNF = Indicator for Two Plastic Alleles			
BDNF	0.4237	0.5419	2.3319
	(1.0152)	(1.0186)	(1.5569)
Standardized Birth Weight	1.4989*	1.1763	2.3262*
	(0.8716)	(0.8106)	(1.1860)
G × E	-0.1269	0.3736	-1.3270
	(1.0035)	(0.9824)	(1.3111)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1314	0.1279	0.6974

Notes: (i) This table performs our base estimation (Table 4) while restricting our neuroplasticity measure solely to the SNP of BDNF. Panel A uses the count of plastic alleles (i.e., 0, 1, 2); Panel B uses an indicator for individuals who have at least one plastic variant; and Panel C uses an indicator for individuals containing two plastic variants. (ii) For our base sibling sample, 4.69% (N=44) do not carry a plastic variant of BDNF, 29.64% (N=278) carry one plastic variant, and 65.67% (N=616) carry two

Table B2. COMT

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Panel A: COMT = Categorical			
COMT	1.0489	0.9765	-1.6149
	(0.6678)	(0.6726)	(1.1147)
Standardized Birth Weight	2.6407***	2.9524***	3.3930***
	(0.8509)	(0.8691)	(1.2145)
G × E	-1.1894*	-1.5307**	-1.8397**
	(0.6351)	(0.6458)	(0.8589)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1369	0.1355	0.6997
Panel B: COMT = Indicator for at Least One Plastic Allele			
COMT	1.4789	1.5582	-3.4361*
	(1.1055)	(1.0928)	(1.7655)
Standardized Birth Weight	2.5994***	3.1344***	3.4404**
	(1.0046)	(0.9907)	(1.4300)
G × E	-1.5578	-2.3100**	-2.5871
	(1.1247)	(1.1157)	(1.5713)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1350	0.1344	0.7000
Panel C: COMT = Indicator for Two Plastic Alleles			
COMT	1.3402	1.1163	-0.3986
	(1.0634)	(1.0933)	(1.6125)
Standardized Birth Weight	1.8700***	1.8991***	2.1001***
	(0.5700)	(0.6173)	(0.7648)
G × E	-1.6594*	-1.8289*	-2.3244*
	(0.9524)	(1.0270)	(1.2367)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1351	0.1316	0.6972

Notes: (i) This table performs our base estimation (Table 4) while restricting our neuroplasticity measure solely to the SNP of COMT. Panel A uses the count of plastic alleles (i.e., 0, 1, 2); Panel B uses an indicator for individuals who have at least one plastic variant; and Panel C uses an indicator for individuals containing two plastic variants. (ii) For our base sibling sample, 24.2% (N=227) do not carry a plastic variant of COMT, 49.36% (N=463) carry one plastic variant, and 26.44% (N=248) carry two plastic variants.

Table B3. APOE

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Panel A: APOE = Categorical (Number of Non-E4 Variants)			
APOE	-0.1234	-0.4261	0.4813
	(0.9378)	(0.9589)	(1.6044)
Standardized Birth Weight	3.5779**	3.5566**	2.0827
	(1.4039)	(1.4047)	(1.6848)
G × E	-1.2753	-1.2529	-0.4037
	(0.7990)	(0.8110)	(0.9818)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1333	0.1297	0.6953
Panel B: APOE = Indicator for at Least One Plastic Allele (Non-E4)			
APOE	1.1955	0.3517	-6.2198
	(2.7048)	(2.7389)	(5.6859)
Standardized Birth Weight	4.4718**	4.4955**	6.8658***
	(2.0865)	(1.9976)	(1.7918)
G × E	-3.1892	-3.2164	-5.7101***
	(2.1399)	(2.0655)	(1.8993)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1333	0.1296	0.6977
Panel C: APOE = Indicator for Two Plastic Alleles (Non-E4)			
APOE	-0.3011	-0.5955	0.9848
	(1.1024)	(1.1229)	(1.6938)
Standardized Birth Weight	2.3399***	2.3142***	1.2145
	(0.8616)	(0.8678)	(1.0849)
G × E	-1.2527	-1.1944	0.2811
	(1.0125)	(1.0383)	(1.2910)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1326	0.1290	0.6954

Notes: (i) This table performs our base estimation (Table 4) while restricting our neuroplasticity measure solely to the SNP of APOE. For APOE, plastic variants are defined as not having the E4 variant. Panel A uses the count of plastic alleles (i.e., 0, 1, 2); Panel B uses an indicator for individuals who have at least one plastic variant; and Panel C uses an indicator for individuals containing two plastic variants. (ii) For our base sibling sample, 2.35% (N = 22) carry 2 copies of the E4 variant, 23.45% (N=220) carry one copy of the E4 variant, and 74.2% (N=696) carry no copies of the E4 variant.

Table B4. Alternative Interactions between BDNF, COMT, and APOE

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Panel A: BDNF × COMT			
BDNF × COMT	0.5856*	0.5720	-0.2741
	(0.3515)	(0.3523)	(0.5902)
Standardized Birth Weight	2.5769***	2.6819***	3.1426***
	(0.7983)	(0.8163)	(1.0899)
G × E	-0.6836**	-0.7683**	-0.9289**
	(0.3192)	(0.3322)	(0.4197)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1381	0.1356	0.6985
Panel B: BDNF × APOE			
BDNF × APOE	-0.1232	-0.1844	0.5183
	(0.3518)	(0.3538)	(0.5423)
Standardized Birth Weight	2.6487**	2.3084**	2.4166*
	(1.1133)	(1.0745)	(1.3334)
G × E	-0.4430	-0.3195	-0.3614
	(0.3534)	(0.3618)	(0.4179)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1327	0.1284	0.6962
Panel C: COMT × APOE			
COMT × APOE	0.5211	0.4510	-0.7254
	(0.3404)	(0.3458)	(0.5418)
Standardized Birth Weight	2.7811***	3.0011***	2.9384***
	(0.7541)	(0.7767)	(1.0293)
G × E	-0.7872**	-0.9427***	-0.8341**
	(0.3152)	(0.3286)	(0.4076)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1388	0.1373	0.6989

Notes: This table performs our base estimation (Table 4) with differing interactions between our 3 neuroplasticity genes. Panel A considers the interaction between BDNF and COMT, Panel B considers the interaction between BDNF and APOE, and Panel C uses the interaction between COMT and APOE.

Appendix C: Alternate Measures of Birth Weight

Table C1. Alternative Measures of Birth Weight

Dependent Variable: High School IQ			
	(1)	(2)	(3)
Panel A: Indicator for Not being Born with Low Birth Weight (i.e., Birth Weight $> 2,500$)			
Neuroplasticity Genes	0.0949	0.3197	-0.4879
	(0.5852)	(0.6064)	(0.7249)
Not Low Birth Weight	4.6784**	5.5973**	4.2233*
	(2.3587)	(2.3465)	(2.3847)
G × E	0.1747	-0.0889	0.3316
	(0.6029)	(0.6296)	(0.6979)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1340	0.1310	0.6965
Panel B: Linear Birth Weight			
Neuroplasticity Genes	2.4702***	2.6514***	2.2594**
	(0.8526)	(0.9031)	(1.1095)
Birth Weight (in grams)	0.0041***	0.0042***	0.0045***
	(0.0011)	(0.0011)	(0.0015)
G × E	-0.0007***	-0.0007***	-0.0007**
	(0.0002)	(0.0003)	(0.0003)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1390	0.1362	0.6985
Panel C: Log of Birth Weight			
Neuroplasticity Genes	13.0096*	14.9987**	12.2401
	(6.8465)	(7.1336)	(8.7894)
ln Birth Weight	10.9832***	11.1989***	11.3499**
	(3.4067)	(3.4648)	(4.4780)
G × E	-1.5738*	-1.8225**	-1.5306
	(0.8438)	(0.8795)	(1.0818)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	938	938	938
R Sqr.	0.1368	0.1338	0.6971

Notes: This table performs our base estimation (Table 4) with alternative measures of birth weight. Our base measure of birth weight is the standard score of birth weight given in grams. Panel A defines birth weight by an indicator for individuals not born of low birth weight (i.e., 2500 grams). Roughly 8% of our base sibling sample is born under 2,500 grams. Panel B uses unadjusted measure of birth weight given in grams. And Panel C uses the natural log of birth weight.

Appendix D: Recreating Table 6 with Alternate Measures of Neuroplasticity

Table D1. Effect of Interaction on Productivity: Using an Indicator for Neuroplasticity

Dependent Variable: ln Wage Rate in 1992			
	(1)	(2)	(3)
Panel A: Main Effects			
Neuroplasticity Genes	-0.2087**	-0.2181**	-0.2847
	(0.1000)	(0.1041)	(0.2507)
Standardized Birth Weight	0.0505	0.0532	0.1041
	(0.0476)	(0.0479)	(0.0795)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	820	820	820
R Sqr.	0.0954	0.0979	0.6025
Panel B: Gene-Environment Interaction			
Neuroplasticity Genes	-0.2084**	-0.2172**	-0.2728
	(0.1001)	(0.1044)	(0.2459)
Standardized Birth Weight	0.1207*	0.1182*	0.3877***
	(0.0640)	(0.0628)	(0.1308)
G × E	-0.0979	-0.0916	-0.3918***
	(0.0884)	(0.0879)	(0.1359)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	820	820	820
R Sqr.	0.0962	0.0986	0.6080

Notes: This table recreates the estimates of Table 6 using alternate measure for neuroplasticity genes. Our base measure is the interaction between BDNF, COMT, and APOE, while the estimates in Table D1 use an indicator for having at least one plastic variant for each considered gene.

Table D2. Effect of Interaction on Productivity: Using the Count of Plastic Alleles

Dependent Variable: ln Wage Rate in 1992			
	(1)	(2)	(3)
Panel A: Main Effects			
Neuroplasticity Genes	-0.0634	-0.0659*	-0.0413
	(0.0392)	(0.0397)	(0.0840)
Standardized Birth Weight	0.0507	0.0531	0.0997
	(0.0473)	(0.0476)	(0.0788)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	820	820	820
R Sqr.	0.0933	0.0957	0.6008
Panel B: Gene-Environment Interaction			
Neuroplasticity Genes	-0.0613	-0.0635	-0.0213
	(0.0394)	(0.0399)	(0.0832)
Standardized Birth Weight	0.1221**	0.1204**	0.3156***
	(0.0523)	(0.0522)	(0.1030)
G × E	-0.0237*	-0.0226*	-0.0666***
	(0.0129)	(0.0126)	(0.0207)
Controls			
Demographic and Family SES	Y	Y	Y
Sibling Fixed Effects	N	N	Y
Estimation			
Weighting by Prob. of Being in Sib Sample	N	Y	N
N	820	820	820
R Sqr.	0.0950	0.0972	0.6069

Notes: This table recreates the estimates of Table 6 using alternate measure for neuroplasticity genes. Our base measure is the interaction between BDNF, COMT, and APOE, while the estimates in Table D2 use the additive score between BDNF, COMT, and APOE.