Preliminary & incomplete, please do not cite or circulate

Biology, Stress and the Intergenerational Transmission of Economic Status

Anna Aizer
Brown University

Laura Stroud
Brown Medical School

Stephen Buka
Brown University

April, 2007

The children of poor black families are twice as likely as their white counterparts to remain poor as adults. We explore whether prenatal conditions can explain this difference. But unlike previous work that has focused on birthweight, we focus on the impact of maternal prenatal stress hormones (cortisol) on offspring outcomes. Medical research has established a key role for maternal stress hormones in prenatal programming, linking stress hormones in utero with declines in offspring cognition, behavior and health**.** We find that mothers of low SES have elevated cortisol and that elevated prenatal cortisol negatively affects IQ and educational attainment of blacks but not whites. We provide two possible explanations for the greater effects for blacks that are consistent with medical models. The first is based on biological differences between blacks and whites in the production of vitamin D that can influence the impact of cortisol on the brain. The second is based on environmental differences: the negative effect of cortisol is greatest for those in stressful environments and blacks may be subject to more stress. Our findings suggest an important role for prenatal conditions in explaining why the children of poor blacks are less likely to escape poverty than their white counterparts and underscore the importance of incorporating biological processes in explanations of economic phenomenon.

I. Introduction

The US is characterized by especially low levels of intergenerational economic mobility among black families: while 17 percent of white children born in the bottom decile of the income distribution remain there as adults, 42 percent of black children do (Hertz, 2005). Previous research has generally found that intergenerational correlations in cognition and educational attainment are responsible for three fifths of the intergenerational transmission of economic status (Bowles and Gintis, 1997). In turn, intergenerational correlations in cognition and education have been largely attributed to either genetic inheritance or the greater investment of high income parents in their children's human capital (Becker and Tomes, 1979). These mechanisms, in addition to being difficult to separately identify, also do little to explain black-white differences in intergenerational mobility.[1]

In this paper we examine the role of another mechanism in perpetuating intergenerational inequality and specifically in explaining black-white differences in intergenerational economic advancement – prenatal programming. Prenatal programming refers to "the action of a factor during a sensitive period or window of fetal development that exerts organizational effects that persist throughout life" (Seckl, 1998). Programming agents include growth factors, hormones, and nutrients that can produce permanent adaptations in a developing fetus. In particular, we focus on in-utero exposure to high levels of stress hormones and the programming of the offspring's stress response system which can result in diminished economic status later in life via reduced cognitive functioning, behavioral deficits or poor health.

[1] Other factors that can potentially explain black-white differences in intergenerational correlations in economic status are discrimination and segregation, a point to which we return.

A well-functioning stress response system is key to one's ability to successfully respond to stressors and medical evidence has shown that an impaired system negatively affects behavior, cognition and health (Bartels et al 2003; McEwen, 2000). Stress hormones or glucocorticoids (cortisol in humans) are an important component of the stress response system and are key to our ability to successfully adapt to stressors. Cortisol is often used as an index of stress response system functioning and it has been shown that 1) poverty is associated with elevated cortisol levels, and 2) exogenous exposure to elevated stress hormones in utero (in animal experiments) results in offspring with an impaired stress response system and diminished cognitive functioning, consistent with a prenatal programming hypothesis.[2]

In this paper, we examine whether exposure to cortisol in-utero can explain higher rates of persistent poverty observed among blacks. We focus on race for three reasons. First, previous medical evidence suggests that blacks have higher cortisol than whites. Second, there is reason to believe that even conditional on cortisol levels blacks may suffer more from an impaired stress response system. Boyce and Ellis (2005) present a theory whereby stress reactivity can have negative effects under adversity and positive effects under "conditions of support and protection." This heterogeneity combined with mounting evidence that blacks experience greater psychological stress (Lepore et al, 2006; Steffen et al, 2004), suggests that an impaired stress response system may have particularly negative effects for blacks. Another reason why blacks may be more likely to suffer negative consequences from elevated cortisol is related to biological differences in vitamin D accumulation across races. Evidence based on animal models suggests that

[2] According to Seckl (2004) "It is now axiomatic that early-life environmental factors influence prenatal development and may cause structural and functional changes which persist for the lifespan."

vitamin D can mitigate the negative impact of glucocorticoids on the brain (the hippocampus in particular) and it is well established that blacks are much more likely to suffer from vitamin D deficiency (Obradavoic et al, 2005 and Harris, 2007). A third reason why we focus on blacks is that previous work has failed to explain why the children of poor blacks are more likely to remain poor than their white counterparts. Hertz (2005) explores whether racial differences in parental education can explain these differences, and finds that they do not, suggesting other factors must mediate the relationship.

To explore the role of prenatal programming and cortisol in explaining the intergenerational transmission of economic status, we use a unique dataset (a subset of the National Collaborative Perinatal Project or NCPP) that contains information on family characteristics at birth, prenatal measures of stress hormones, and offspring outcomes – child IQ and adult education and income for a sample of 1103 children born to low income women in the early 1960s. Using instrumental variable (IV) techniques to overcome the potential endogeneity of cortisol, we find that exposure to high levels of cortisol in utero is of greater consequence for blacks – resulting in lower IQ and educational attainment as adults, consistent with a prenatal programming hypothesis. Among blacks, a one standard deviation increase in prenatal maternal cortisol results in 8-13 point drop in child IQ. This impact persists through adulthood: blacks in this sample gained, on average, three years of school relative to their mothers, but those with a standard deviation increase in cortisol achieved only a two year gain. These results can help explain the extraordinary persistence of poverty across generations of black families

- black children are both more likely to inherit maladaptive stress response systems from their parents and suffer greater negative consequences because of it.

Our findings contribute to a large literature that attempts to distinguish between the roles of nature and nurture in perpetuating economic status across generations. We contribute to this literature in three ways. First, we expand the notion of "nature" to include not only genetics but also the in-utero conditions of the biological mothers. Second, we use a novel identification technique to identify nature from nurture in this context: by instrumenting for in-utero cortisol we can determine whether maternal stress hormones affect child outcomes via prenatal programming of the offspring stress response system by eliminating the effects of potential confounders (ie, a stressful environment shared by mother and child that might increase the stress hormones of both.) And third, we explore the interaction between nature and nurture in this context. Our finding of significant interactive effects between race and prenatal cortisol in the production of offspring human capital is consistent with medical evidence that suggests that there may be considerable heterogeneity in the effect.

Our findings also relate to a growing strain of economic inquiry that seeks to incorporate biological processes into economic models, as the growing field of neuroeconomics has done (Camerer, Lowenstein, and Prelec, 2005). An example of the latter includes David Laibson's work on hyperbolic discounting which draws on scientific evidence that separate neural systems value immediate and delayed monetary rewards to develop a model to explain the dynamically inconsistent choices that are often observed (Laibson, 1997).

The rest of the paper is organized as follows. In section II we summarize existing theoretical and empirical research on the intergenerational transmission of economic status. In section III, we provide background information on the workings of the stress response system and summarize existing medical evidence on the associations between SES, stress, cortisol and outcomes such as as cognition/disease. In section IV, we describe the data. In section V we present our empirical results. Section VI concludes.

II. Intergenerational Transmission of Economic Status: Previous Work

Becker and Tomes (1979) develop a model to explain the process by which the economic status of parents is transmitted to their children. In their model, nicely summarized by Solon (1999), a child's earnings are a function of parental investments in the child's human capital and the child's endowment, which includes genetic or cultural traits passed from the parent to the child, and market luck. The child's endowment is positively correlated with parental endowment which also affects the level of investment in the child's human capital. One of the key implications of this model regards the interpretation of empirical estimates of the impact of parental endowments on child earnings. If parental endowments are correlated with both child endowments and human capital investments it's not clear which mechanism (genetic inheritance, inheritance of cultural traits or greater human capital investment) is behind intergenerational correlations in earnings, hindering identification of the mechanism.

In this paper we implement an empirical strategy that enables us to identify a mechanism by which parental conditions at birth affect adult offspring outcomes. The mechanism we identify is that of prenatal programming – in utero conditions associated

with a family's lower economic status that can program important biological systems of the fetus, resulting in diminished economic status later in life. Almond (2006) presents empirical evidence of the importance of in-utero conditions in explaining adult mortality, education and disability in the context of the 1918 flu pandemic, consistent with a prenatal programming hypothesis. Our paper differs from Almond (2006) in that the in utero conditions we examine are not random (as the 1918 flu pandemic was) but are correlated with low socio economic status. In this context, prenatal programming can potentially explain not only reduced economic status later in life but also the intergenerational transmission of economic status. Furthermore, we not only explore the role of prenatal programming in explaining the transmission of economic status but also how it interacts with one's environment, underscoring the importance of examining not only the main effects of nature and nurture separately, but also their interactive effects.

Empirical Estimates of Intergenerational Transmission of Economic Status

Most of the existing empirical work has sought to quantify the amount of intergenerational mobility and/or determine the mechanism(s) behind intergenerational correlations in economic status. The first studies found generally low correlations between the earnings of fathers and sons; however, these studies were based on single year estimates of income and earnings of parents and children. Estimates based on only a single year suffer considerable measurement error which tends to bias down estimates of intergenerational earnings correlations.[3] More recent work based on longer term measures of income has found considerably higher estimates of intergenerational

[3] In addition, the earnings of sons in earlier data were based on early (younger) years which are less representative of permanent earnings.

earnings correlations on the order of 0.5, with the highest estimate of 0.65 (Solon, 1999; Mazumder, 2005).

However, there appears to be considerable heterogeneity in intergenerational transmission of economic status. Hertz (2005) estimates intergenerational correlations in earnings for black and whites separately using the PSID. He finds that blacks in the bottom of the income distribution experience especially low rates of upward mobility: 17 percent of whites in the bottom decile of family income remain their as adults compared with 42 percent of black children. Hertz explores whether lower levels of parental education among black families can explain these differences and finds that they cannot, concluding that other mechanisms must be responsible.[4]

Identifying the Mechanisms behind Intergenerational Transmission of Economic Status

Most of the existing empirical work that has sought to identify mechanisms of intergenerational transmission of economic status has categorized the mechanisms as either nature or nurture. Nature typically refers to genetic inheritance, though we argue here that it should include in-utero conditions as well. Nurture refers to the environment in which the child is raised. Separating the effects of nature and nurture is difficult given correlations between the two. The most convincing identification strategies rely on samples of twins and adoptees to separately identify the effects of nature and nurture. Taubman (1989), Behrman and Taubman (1989) and Behrman Rosenzweig and Taubman (1994) analyze data on identical and fraternal twins in a structural framework based on

[4] Loury (1981) extends the model of intergenerational transmission of economic status to include credit constraints. In his model, if and when parents are credit constrained, they may not be able to invest the efficient amount in their children's human capital. This model could explain black-white differences in intergenerational mobility if blacks are more credit constrained than whites as evidence suggests (Perraudin and Sorensen, 1992).

established behavioral-genetic models. In twin studies, the implicit assumption is that stronger within identical twin correlations are attributable to genetic similarities (as identical twins share all their genes and fraternal twins only half their genes). These studies generally conclude that both environment and genetic heritance matters in the transmission of such outcomes as educational attainment, income and obesity. However, Goldberger (1978) discusses limitations to the behavioral-genetic model which include, for example, the fact that parents may treat identical twins more similarly than they do fraternal twins so that not only are their genes the same, but so is their environment.

Research based on samples of adoptees generally finds that characteristics of both adoptive parents and biological parents contribute to the education and earnings of their children (Sacerdote, forthcoming; Bjorklund, Lindahl and Plug, 2006). By including the characteristics of both adoptive parents and biological parents, Bjorklund, Lindahl and Plug (2006) estimate what portion of the transmission is due to nature (birth parents) versus nurture (adoptive parents). They also find interactive effects between the two (nature and nurture).

But this work makes no attempt to distinguish genetic inheritance from prenatal conditions or a prenatal programming hypothesis. It could be, for instance, that poor birth mothers provide poor in-utero conditions. A prenatal programming hypothesis would imply that this is why their children earn less as adults, not genetic inheritance of certain traits. Recent work by Currie and Moretti (forthcoming) on intergenerational correlations in birthweight, Royer (2006) Black (2006) and Behrman and Rosensweig (2004) on the impact of birthweight on future economic status are mindful of the distinction between genetic inheritance and in-utero conditions. Currie and Moretti

(forthcoming) find that mother's born low birth weight (lbw) are 50 percent more likely to give birth to lbw babies and that the intergenerational transmission of lbw is stronger for poor mothers than others, suggesting important interactive effects between nature and environment. In an attempt to control for genetic differences, Currie and Moretti analyze between sister differences and find that the effects remain. Royer (2006) Black (2006) and Behrman and Rosensweig (2004) use within-twin differences to identify the impact of fetal nutrition (assuming differences in birthweight reflect differences in nutrition only) separate from genetics on future outcomes including education and earnings.

There are two main innovations of the present study. First, we focus not on birthweight (which previous economic literature views as a proxy for in utero nutrition) in part because the estimated impact of birthweight on future economic status has generally been found to be quite small, which may be due to the fact that the vast majority of pregnant women in the US and other developed nations do not suffer from malnutrition. Instead, the prenatal conditions on which we focus relate to stress as measured by elevated stress hormones, a condition arguably more relevant in the US than is malnutrition. Second, we use instrumental variable methods to identify the effects of prenatal conditions separate from genetics on the intergenerational transmission of economic status. And finally, like Currie and Moretti (forthcoming) we focus on how in-utero stress and the environment of the offspring interact to affect economic status later in life.

III. SES, Cortisol and the Stress Response

In this section we describe the psysiology of the stress response system, the role of cortisol in regulating the response, and the impact of elevated cortisol on the body. This is followed by a review of evidence on the relationship between economic status, race, stress, and cortisol. We end with a description of how elevated maternal cortisol may affect an offspring's stress response system via prenatal programming, thereby reducing economic status later in life.

Physiology of the Stress Response

Individuals are constantly challenged by intrinsic or extrinsic adverse forces referred to as stressors. When challenged by a stressor, one's stress response system is activated. The stress response system enables individuals to adapt to the stressor: attention is enhanced and the brain focuses on the specific challenge or threat. Metabolism, cardiac output and respiration accelerate and blood flow is redirected to the aroused brain, heart and muscles (Tsigos and Chrousos, 2006). Two main biological systems coordinate the adaptive response of the individual to the stressor - the autonomic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis.

The HPA axis is a central part of this effort. The HPA comprises the hypothalamus which secretes the two peptides vasopressin and CRH which stimulate the secretion of ACTH in the pituitary (the second component of the HPA axis). ACTH acts on the adrenal glands (the third component of the HPA axis) which produce the golucocorticoid cortisol in response to stimulation by ACTH. Cortisol acts back on the hypothalamus and the pituitary glands to suppress the production of CRH and ACTH in a negative feedback cycle, modulating the stress response and helping the body to return to

a stable state, referred to as homeostasis, a crucial component of the cycle. However, the systems that protect an individual in the short run cause damage when activated for long periods of time. Over the long term, the normal feedback system may break down and the body has trouble returning to homeostasis. This is referred to as allostatic load.[5] Allostatic load may result from either 1) an excessive number of stressful events leading to over-exposure to stress hormones or 2) the body's failure to manage the hormonal stress response system (McEwen, 2000). During allostatic load, stress hormones remain elevated for prolonged periods of time.

The Impact of Elevated Cortisol on the Body

When stress hormones have been elevated for a prolonged period of time, they have a backlash effect – suppressing the immune system, causing brain nerve cells to shrink, damaging the hippocampus and impairing memory (McEwen, 2000). Chronically elevated glucocorticoids (cortisol) can impede the action of insulin, promoting the deposition of body fat and hardening of the arteries (Brindley and Rolland, 1989). Elevated cortisol has been implicated in a range of pathologies including hypertension, cardiovascular disease, insulin resistance, obesity, diabetes, infection illness and depressive disorder (Ousova et al, 2004; Walker et al, 1998; Steptoe et al, 2002).

Elevated cortisol also leads to the development of atypical emotional, behavioral and cognitive functioning (McEwen, 1998; McBurnett et al, 2000; Dawes et al, 1999; Van Goozen et al, 1998). Animal studies in which experimenters administer

[5] McEwen (2000) defines allostatic load as "the price the body pays for being forced to adapt to adverse psychological or physical situations, and it represents either the presence of too much stress or the inefficient operation of the stress hormone response system, which must be turned on and then turned off again after the stressful situation is over."

glucocorticoids (cortisol in humans) to animal subjects have documented damage to the hippocampus (Sapolsky et al, 1990). Adults with memory problems are characterized by atypical cortisol cycles (Fiocco, Wan, Weekes, Pim and Lupien, 2006). And researchers have found that high cortisol levels in young adults with Cushing's syndrome (a hormonal disorder caused by prolonged exposure to high levels of cortisol, a relatively rare condition) are associated with cognitive deficits (Starkman et al 2001).

However, recent evidence suggests that the effects of a maladaptive stress response system are heterogeneous, depending on one's environment. Boyce and Ellis (2005) present a theory whereby the effect of stress reactivity can be either risk-augmenting or risk-protective depending on the context: those in high-stress environments suffer negative consequences and those in low-stress environments thrive. They cite as corroborating evidence non-experimental studies that find that highly reactive children in high-stress environments sustained higher rates of respiratory illness, while highly reactive children in low stress environments were healthier than all other children (Boyce et al, 1995). Experimental studies also support their theory. In a 1998 study of monkeys subjected to "confinement stress," Boyce et al (1998) found that monkeys with higher baseline reactivity were healthier in low stress environments but much less healthy in high stress environments than those with low or moderate reactivity.[6] This suggests that the impact of elevated prenatal cortisol on offspring cognition may be heterogeneous – with negative effects for those in more stressful environments and potentially protective effects for those in supportive environments.

Cortisol, Stress, and SES

[6] Health was measured as incidence of violent injuries.

Medical studies have documented a link between stress and cortisol. Wust et. al. (2000) found that cortisol response was increased under chronic stress conditions, defined as social stress, worries and lack of social recognition. Van Eck et al (1996) found that stressful daily events were associated with increased cortisol secretion and that mood played a mediating role. More recently, researchers have sought to explain the relationship between SES and cortisol and have hypothesized that individuals of low SES have higher levels of cortisol because they face more stress and have less social support. For example, Cohen et. al. (2006) found that lower education, lower income and being black were each independently associated with higher levels of cortisol. For whites, the association between lower SES and cortisol was explained by higher levels of depressive symptoms, poorer social networks/support, feelings of helplessness, and smoking. However, these factors could not explain higher cortisol levels among blacks. Another study based on the Whitehall II epidemiological cohort examined the relationships between SES, job demands/job control and cortisol levels. In particular, the authors sought to explain the negative relationship between SES and cortisol levels previously found among men (but not women) in Whitehall II. Measuring cortisol over the course of the day, the authors found that cortisol levels increased among lower SES individuals as they experienced high job demands, but that the cortisol levels of high SES individuals did not increase in response to high job demand (Steptoe et al, 2003; Kunkz-Ebrecht, Kirschbaum and Steptoe, 2003). The findings of both higher levels of cortisol among low SES individuals on average and a greater increase in cortisol levels in response to high job demands among low SES men is consistent with theories of the heterogeneous effects of stress reactivity put forth by Boyce and colleagues. We explore the possible

interactive effect between stress reactivity and environmental stressors in the context of race in the next section.

Stress, Stress Reactivity, Cortisol and Race

The higher levels of allostatic load and cortisol observed among blacks is not well understood. Geronimus et al (2006) find that blacks have higher allostatic loads than whiles at all ages and that these differences cannot be explained by poverty. And while Cohen et. al. (2006) found that the usual social and behavioral variables that explained the association between lower SES and cortisol among whites, they could not explain the relationship among blacks suggesting that some unmeasured factors might be responsible. Evidence from the medical literature on black-white differences in cardiovascular disease (CVD) may shed some light on observed racial differences in cortisol levels. To explain racial differences in CVD, researchers have hypothesized that social stress, and in particular exposure to racism, may be responsible for the difference. This research has generally found that acute exposure to racism is associated with increases in cardiovascular activation among blacks and also that past exposure to racism can sensitize individuals to future stressors, increasing cardio-vascular response to both race-related and non race-related stressors (see Brondolo, et al 2003 for a review).[7] These studies suggest that perceived racism may represent an acute but poorly measured stressor faced by blacks that causes a significant negative physiological response. This can potentially explain both higher levels of cortisol observed among blacks as well as

[7] Laboratory studies expose participants to racist stimuli in a lab (racist videos, harassment from a white experimenter) and measure cardio-vascular response; observational studies monitor cardiovascular activity throughout the day and ask the participant to keep a time diary of daily events.

greater negative effects of maladaptive stress reactivity for blacks based on the hypothesis put forth by Boyce and colleagues.

Yet another reason why cortisol might have a greater negative impact on blacks relates to biological differences in vitamin D acquisition. Very recent work based on animal experiments shows that while vitamin D alone appears to have no significant impact on neurite growth in the brain (the hippocampus), it does act to reduce the negative impact of glucocorticoids on neurite growth (Obradovic et al, 2005). Neurite growth is a major mechanism whereby neural networks increase in size which is strongly related to cognitive ability.

Blacks have lower levels of circulating vitamin D than whites. This is a purely physiological phenomenon: vitamin D is obtained from sun exposure and diet and the lower levels of vitamin D observed among blacks results from the fact that pigmentation reduces vitamin D production in the skin. In a 1998 study of young black and white Boston women age 20-40, the authors found that vitamin D concentrations for the black women were less than half the concentrations for white women (Harris and Dawson-Hughes, 1998).[8] If blacks are more deficient in vitamin D then they may be less able to mitigate the negative impact of cortisol on the brain and, as a result, suffer greater consequences of elevated cortisol in terms of diminished IQ and future economic status.

Thus there are two potential mechanisms which may produce greater negative effects of cortisol for blacks. The first relates to environmental differences: blacks face more stress due in part to racism and this stress interacts with cortisol to produce negative effects on cognition and future economic status. The second relates to biological differences: blacks are biologically predisposed to vitamin D deficiencies due to their

[8] Not only were their levels lower but the increase over the summer months was also smaller.

skin pigmentation which in turn reduces their ability to offset the negative effects of cortisol on brain development and cognition. In this work we are unable to determine which of these two mechanisms is responsible for the greater effects of maternal cortisol on cognition and economic status that we find for blacks.

Intergenerational Transmission of Cortisol

A number of studies have documented correlations between maternal cortisol levels and/or stress and child cortisol levels. Gitau et al (2001) and Gitau et al (1998) compared cortisol levels in matched maternal-fetal pairs and found that fetal concentrations were linearly related to maternal concentrations (though the latter were more than ten times as great). O'Conner et al (2005) find that the ten year old children of mothers with higher levels of prenatal anxiety have higher levels of cortisol, controlling for a wide range of family characteristics. Gutteling, de Weerth and Buitelaar (2005) found that in a sample of Dutch children, those whose mothers had higher levels of morning cortisol during pregnancy showed higher levels of cortisol on the first day of school at age five. Finally Huizink et al (2003) find that stress and elevated cortisol in late pregnancy were negatively related to both mental and motor development at three and eight months. Though the authors do not have measure of cortisol levels for the children, they infer that the mechanism is the child's impaired stress response system.

While the above studies document correlations between maternal and child cortisol levels, they do not establish transmission, but could simply reflect a shared stressful environment. There are three mechanisms by which mothers may transmit

elevated cortisol and a dysregulated HPA axis to their offspring. The first is genetic heritability and the second is prenatal programming. We discuss each below.

Studies based on twins have shown that genetics are an important determinant of an individual's cortisol level. Bartels et al (2003) analyzed cortisol levels in a sample of 180 twin pairs and used the different degree of genetic relatedness between monozygotic (MZ) and dizygotic (DZ) twin pairs to estimate the contribution of genetics vs. environment to cortisol levels. Genetic heritability was generally high and highest for early morning cortisol samples (60%). Wust et al (2000) also using data on twins concluded that cortisol levels at waking are under genetic control but that fluctuations over the course of the day are not.

The second mechanism by which mothers may transmit a dysregulated HPA axis to offspring is via fetal programming. Prenatal maternal cortisol levels can affect offspring via the transmission of stress hormones (including cortisol) across the placenta. The most convincing evidence of prenatal programming consists of experiments on animals that have shown that exogenous exposure to glucocorticoids in the prenatal period affects fetal programming of the HPA axis. Welberg, Seckl and Holmes, (2001) administered glucocorticoids to pregnant rats and found that the offspring of exposed rats exhibited behavioral inhibition, and impaired coping and learning in aversive situations. They concluded that prenatal programming of the HPA axis was responsible for the outcome based on studies of the areas of the brain affected. Other studies on primates have successfully mimicked the negative impact of a mild stressor during pregnancy on motor and mental development by exogenously exposing mothers to stress hormones (Schneider, 1992). Among humans, non-experimental research has shown that exposure

to excessive amounts of cortisol in utero can affect the developing brain and spinal cord (Yu, Lee, Lee and Son, 2004).

But since repeated stress increases cortisol, correlations in cortisol levels between mothers and children may simply reflect a shared stressful environment. In this analysis we focus on fetal programming as the mechanism and we use instrumental variable methods to isolate this mechanism separate from either genetics or environment, though we allow the effect to vary with offspring's environment.

IV. Data

The data are a subset of the National Collaborative Perinatal Project (NCPP). The NCPP was a collaborative multi-site study in the late 1950s and early 1960s that sought to identify the prenatal and early childhood determinants of subsequent child health and well-being. Factors investigated fell into three categories: abnormal conditions of the pregnancy, environmental factors (social and economic conditions) and biological factors in parents (Gordon et al 1972). The study comprised a prospective survey of 55,908 pregnancies between 1959 and 1965 across 12 cities. Women were enrolled primarily though public clinics where they sought prenatal care.[9] Extensive data were collected at each prenatal visit, during labor and delivery and during five follow-up periods: 4 months, 8 months, 1 year, 4 years and 7 years. During follow-up periods, children were examined by physicians and psychologists who evaluated them along a wide range of neurological, development, behavioral and cognitive measures.

[9] Unwed women who planned to put their children up for adoption and women who arrived at the hospital for delivery without any prenatal care were excluded from the study.

In this study we focus on a subset of children born to mothers enrolled in NCPP through either the Providence or Boston sites for whom follow-up information for these children as adults is available. In 2000, a small subset of children of pregnant women enrolled in the Providence and Boston NCPP sites were contacted and interviewed as adults.[10] The subjects were approximately 35-40 years old at the time of follow-up. Trained interviewers collected information on adult SES (education, income, employment), disease and other characteristics (Buka, Shenassa and Niaura, 2003). The final sample with adult follow up information includes 1103 subjects, 543 from Boston and 560 from Providence. We subsequently drop all children born to immigrants because previous work has documented different rates of intergenerational mobility for the children of immigrants in the US.

Maternal blood/serum collected during the third trimester of pregnancy (between 31 and 36 weeks of pregnancy) was analyzed for these 1103 mothers. The samples were assayed for cortisol, testosterone, cortisol binging globulins (CBG) and sex hormone binding globulin (SHBG). Values obtained were compared to published studies of fresh samples to assess validity after 40 years of storage (Stroud et al, forthcoming). The results support the overall validity of these cortisol and testosterone values. However, they are still measured with error. Cortisol naturally varies over the course of the day. Current studies of cortisol levels follow relatively standard methods of collection – taking measurements immediately upon waking and at specific intervals throughout the day.

[10] The original cohort from the Providence NCPP site included 4,140 pregnancies, of which 3,138 subjects were assessed at age 7. The cohort from the Boston NCPP site included 13,737 pregnancies of which 8,931 were assessed at age 7. The subset of those followed up in 1996 was selected from among those who had completed the 7 year assessment for a study investigating the consequences of maternal smoking. The subset of the NCPP for which adult follow-up data are available comprise a subset of those for whom maternal smoking data are available.

The cortisol measures in this study are spot measures and it is not known when during the day they were obtained, introducing measurement error and attenuation bias. To overcome the attenuation bias associated with measurement error, we use instrumental variables, as discussed later.

Table 1 presents sample means for the entire NCPP cohort (all 12 cities), the NCPP Boston/Providence cohort, and the analysis sub-sample. The subset of individuals selected from the Providence and Boston NCPP for adult follow-up is slightly less educated and of slightly lower SES than those in the general Boston/Providence cohort, but the differences, while statistically significant, are small. There are also significant differences with respect to birthweight and gestation which we attribute to the following: 1) the analysis sample does not contain very premature or low birth weight babies that subsequently died within the first seven years of life (as the greater NCPP sample does) and 2) because cortisol measures were based on blood collected in the third trimester, all births in the analysis subsample reached at least 31 weeks gestation and usually longer, thereby excluding the most premature births.

In the second to last column of the table are descriptive statistics from the 1960 census of women with children less than five years old residing in Massachusetts and Rhode Island.[11] As is evident from the table, women in our sample are more likely to be black, younger, less educated, single and poorer. This is not surprising given that the NCPP sample is urban and that recruitment for subjects in the NCPP was conducted through public clinics. The demographic composition of the NCPP aids our investigation

[11] Because of limitations of the census data, we were unable to calculate averages for Providence and Boston only.

of the intergenerational transmission of income (poverty) and differences in transmission by race.

V. Results

Estimation of the role of stress, maternal cortisol and prenatal programming in explaining higher rates of intergenerational transmission of economic status among blacks proceeds in three stages. First we establish a correlation between maternal economic status and the economic status of her adult offspring in these data and document higher rates of persistent poverty for blacks. Next we explore the role of prenatal cortisol in mediating this relationship. To do so, we first document higher levels of cortisol among women of lower socio-economic status. Second, given the medical evidence linking in-utero cortisol with future cognitive ability, we explore IQ as a mechanism by which maternal cortisol and prenatal programming may "transmit" economic status from mother to child with a particular focus on differences by race. For this we estimate the impact of prenatal cortisol levels on child IQ using OLS and IV techniques. Finally, we link prenatal maternal cortisol with adult economic status by providing causal estimates of the impact of maternal prenatal cortisol on intergenerational gains in education.

Our results suggest that cortisol which is elevated in low-income mothers has a negative causal impact on cognitive ability and the educational attainment of the children of black mothers and, to a lesser extent, single mothers. The fact that we find large and significant effects for these groups but not others is consistent with recent scientific theories that the effect of prenatal cortisol on offspring outcomes is moderated by the

amount of stress in the offspring's environment. Single mothers and black mothers are arguably subject to greater stress than other groups.[12] Our results help to illuminate how conditions at birth and prenatal programming can interact with a child's environment to explain why the children of poor blacks are less likely to escape poverty than their white counterparts as are the children of single mothers.

A. Persistent Poverty Across Generations

Intergenerational correlations in education and income are present in this sample and comparable to correlations reported in existing literature based on other data. The raw correlation between maternal education (highest grade completed) and the education level of her adult offspring is 0.285 and the correlation between family income during the prenatal period and the personal income of adult offspring is 0.156.[13]

In Table 2 we present evidence of greater persistence of poverty among blacks in these data, consistent with previous findings based on the PSID (Hertz, 2005). In panel A of the table are the share of children born into the bottom 25% of the income distribution who remain in the bottom 25% of the income distribution as adults (column 1) and the share who grow up to be high school drop-outs (column 2), by race.[14] While 31 percent of poor white children remain in the bottom 25 percent of the income distribution as adults, 43 percent of black children do and while 8.5 percent of poor white children never

[12] This is also generally consistent with the findings of Currie and Moretti (forthcoming) that the intergenerational transmission of LBW is stronger for poor mothers.

[13] The correlation of 0.156 is on the lower end of the spectrum but reasonable given that the income measures are based on single years and the fact that roughly 25% of the adult personal income reports are top coded, introducing considerable measurement error, a point to which we return.

[14] We do not utilize the full spectrum of adult income due to top-coding in the data: 25 percent of the responses to the personal income question are top-coded and 53 percent of the responses to the household income question are top-coded. Instead, we construct an indicator for whether the responded reported income in the bottom 25 percent of the income distribution in these data.

complete their high school degree (an important determinant of permanent income), 23 percent of black children never do. In panel B are similar numbers for those children born to mothers without a high school degree: 28 percent of white children in this category grow up to be in the bottom 25 percent of the income distribution and seven percent never complete high school, while 58 percent of black children in this category end up in the bottom part of the income distribution and 16 percent never receive their high school degrees.

B. The Role of Prenatal Maternal Cortisol in Explaining Persistent Intergenerational Poverty

Maternal SES and Prenatal Cortisol

In these data, low social and economic status is associated with elevated cortisol levels, consistent with existing literature linking socio-economic status and cortisol in non-pregnant adults. Figure 1A presents mean maternal cortisol levels by race (white/black), marital status (single/married), maternal education (<HS/>=HS), poverty (below/above), and SES index (low/high). The SES index is a composite index adapted from the US Census Bureau that averaged centiles derived from the education and occupation of the head of the household and family income. It ranges from 0 to 9.9 with higher scores representing higher SES. As is evident from the figure, Blacks, less educated mothers, poor mothers and those of low SES have higher levels of cortisol. The differences are statistically significant except for differences by race. In Figure 1B we present distributions of cortisol by socio-economic group. The distribution is roughly

normal for all groups. The relationship between maternal prenatal cortisol and maternal SES in this sample is explored in greater detail in Stroud et al (???)

Prenatal Programming of IQ

In this section we explore IQ as the mechanism by which prenatal maternal cortisol affects offspring economic status. We focus on prenatal programming of child cognitive ability as a mechanism given 1) medical evidence linking elevated cortisol to the prenatal programming of cognition in animal and other studies, and 2) evidence that the transmission of cognitive ability and education explains three fifths of the intergenerational transmission of economic status (Bowlus and Gintis, 1997) as well as work by Neal and Johnson (1996) showing that pre-market differences in cognitive ability explain much of the black-white wage difference.

We begin by documenting both the correlation between family background and child IQ and the importance of child IQ in determining adult economic status (especially among blacks) in this sample. This is followed by estimation of the causal impact of prenatal maternal cortisol on child IQ with particular attention to differences by race.

Family Background, Child IQ, and Adult Earnings

Family background is highly correlated with child IQ in this sample. Figures 2A and 3A display this. IQ at age 4 (Figure 2A) and age 7 (Figure 3A) is higher for those children born to white mothers, married mothers, mothers with at least a high school degree, non-poor mothers, non-teen mothers and high SES mothers. These differences range from 7 points to 16 points (one half to one standard deviation) and all differences

are statistically significant. The only family background measure with which IQ does not appear to be correlated is whether the mother was on welfare. Child IQ is also highly correlated with later economic status. Figures 2B and 3B illustrate this point: children with low IQs are more likely to grow up to be high school drop outs and poor relative to those with high IQs.

In Table 3, we explore how much of the black white differences in economic status in this sample can be explained by differences in IQ at age 7 (12 point difference) and other factors. In the first 6 columns of Table 3 we estimate the impact of various individual and family characteristics on the probability of being in the bottom 25% of the income distribution. In columns 7-12 we do the same for the probability of being a high school drop out. In the first and seventh columns we control only for age, gender and being black. Being black is associated with a 6.7 percent increase in the likelihood of being in the bottom 25 percent of the income distribution and a 6.4 percent increase in the likelihood of not receiving a high school degree as an adult. In the remaining columns we control for maternal score on the SRA (Science Research Association Test or SRA-AH which is a test of cognitive ability), maternal education, whether it was a teen pregnancy, child IQ at age 7 and finally parental income at birth. As is evident from the table, including maternal score on the SRA, whether she was a teen mother, or maternal education do little to diminish racial differences in adult socio economic status. When we control for child IQ (columns 5 and 11), however, black-white differences in both adult income and education decline to zero. Controlling for family income at birth also reduces black-white differences in the probability of being in the bottom 25% of the

income distribution as an adult, but not does not diminish black-white differences in high school drop–out rates.

The Impact of Maternal Cortisol on Offspring IQ – OLS Estimates

In this section we provide empirical evidence that maternal prenatal cortisol negatively affects offspring cognitive ability, but the effect is concentrated among those born into families of the lowest socio economic status. In Table 4 are OLS estimates of the impact of cortisol on child IQ (measured at age 4) stratified by poverty status at birth (columns 1 and 2), stratified by race (columns 3-6) and stratified by marital status at birth (columns 7-10). As is evident from the table, the negative impact of prenatal maternal cortisol on child IQ is considerably larger for those born into families of the lowest social economic status: the poor, blacks, and single mothers. For example, for those above poverty, a one standard deviation increase in cortisol results in a 1 point decrease in IQ, while for those below poverty, the same increase results in a 3 point decrease in IQ (one fifth of a standard deviation). These results are consistent with medical evidence that stressful environments exacerbate the negative impact of cortisol and stress reactivity. What is also evident from the table is that once we include cortisol as a regressor, income becomes less protective for low ses groups. For example, in all regressions income has a positive and significant impact on child IQ: each $1000 increase in family income at birth is associated with a 2 point rise in IQ. This implies that moving from the 20^{th} percentile of the income distribution for families in 1960 to the 80^{th} is associated with a 12 point rise in IQ (fourth fifths of a standard deviation). However, when we control for cortisol, income appears considerably less "protective" for the low SES groups in the sense that

the association between income and IQ falls by roughly 30 percent. This suggests that for low SES groups a substantial portion of the positive impact of income on child outcomes may be working through reduced cortisol levels and/or mitigation of the negative impact of cortisol on child outcomes.

We follow this with OLS and IV regressions of child IQ on maternal prenatal cortisol including a full set of controls for family characteristics at birth to control for the fact that the estimated impact of cortisol on IQ may be confounded with other family characteristics. We also control for birthweight as elevated cortisol in utero has been associated with reduced birth weight. The following equation is estimated:

$$IQ = \alpha + \beta_1 Cortisol + \beta_2 \mathbf{X} + \beta_3 Birthweight + \varepsilon \qquad (1)$$

Where **X** is a vector of family and child characteristics at birth including gender, maternal race, age and education, parity (number of living siblings), family income at birth, marital status, maternal score on the SRA, whether there was any pregnancy complication and whether the family resided in Providence or Boston at the time of the birth.

In Table 5 are OLS estimates of the impact of maternal prenatal cortisol levels on offspring IQ measured at age 4 (equation 1) including a full set of controls. The results when we include the full set of controls are remarkably similar to those presented in Table 4 that include only controls for income and gender. The heterogeneity in the effect of cortisol on IQ remains – children of black mothers with high cortisol do score significantly lower on IQ tests at age 4 (column 2). And the magnitude of the effect is similar. It is also the case that cortisol still has a greater negative impact on the children

of poor mothers and the children of single mothers even after the full set of controls is included (columns 3 and 4).

We explore whether the negative and significant coefficient on the interaction term between race and cortisol reflects other factors that may be correlated with cortisol and that have a greater impact on blacks than whites. For example, since blacks are more likely to be poor and the poverty*cortisol interaction is negative and significant, the coefficient on the interaction term cortisol*black may simply reflect this relationship. In column (5) of Table 5 we include both interactions (poverty*cortisol and black*cortisol) and in column (6) we include a third interaction between race and poverty. When we do, the coefficient on the interaction term cortisol*black remains, suggesting that the negative relationship between maternal cortisol and child IQ that we see for blacks is not merely reflecting an interaction between race and SES.[15] In Table 6 are the same regressions for IQ score at age 7. The results are similar but the estimated negative effect of cortisol on IQ slightly smaller.

IV Estimates

While medical evidence based in part on animal experiments shows that high maternal cortisol can negatively affect offspring economic status by negatively affecting cognition, it is also the case that exposure to repeated stress elevates cortisol levels. As such, in a non-experimental setting it is not clear whether elevated maternal cortisol levels affects offspring outcomes or merely reflects high levels of stress associated with poverty and race and for which we cannot control.

[15] We argue that the coefficient on this interaction term is not due to the fact that blacks have higher levels of testosterone which is correlated with cortisol and we provide evidence of this in the appendix.

In order to determine whether it is elevated maternal cortisol itself (and not the stress that it may reflect) that affects offspring outcomes, we instrument for maternal cortisol. Instrumental variables methods enable us to determine whether maternal cortisol affects offspring outcomes via the inheritance of a maladaptive stress response system by eliminating the effects of potential confounders (ie, stress in the family environment.) The second reason to instrument for maternal cortisol is measurement error. Prenatal maternal cortisol is measured with error in this sample for two reasons. First, the blood was drawn forty years ago and stored at temperatures not considered ideal by today's standards. Second, there is no record of the time of day the blood was drawn. Cortisol levels vary significantly over the course of the day – rising immediately after waking and then declining throughout the day. This classical measurement error leads to attenuation bias for which an instrumental variable strategy may correct.

The instrument for maternal cortisol must be correlated with cortisol levels but not independently affect economic status. We include two instruments for maternal cortisol. The first is drugs administered during pregnancy that have been shown in the medical literature to increase cortisol levels. These drugs include DES, estrogen and progesterone supplements. Pregnant women were administered these drugs when they had pregnancy complications (which we include as a control) but not all women with pregnancy complications receive these drugs. In this analysis we control for pregnancy complications. Even if we control for pregnancy complications, it is still possible that these drugs may affect child outcomes independent of their impact on cortisol by, for example, affecting the health of the child as it develops in utero (ie – fail the exclusion restriction). To limit this possibility, we also run the regressions controlling for

birthweight – the most common measure of health at birth. We refer to instrumental variable regressions in which the only instrument is whether a woman received one of these drugs as IV1. A second instrument is testosterone which plays a role in regulation of the HPA axis and is highly correlated with cortisol levels but does not increase with exposure to stress as cortisol does. It is well documented that testosterone levels increase during pregnancy. The exact cause is not known, though evidence suggests that the increase is due largely to an decrease in the rate at which testosterone is metabolized and less an increase in the production of testosterone (Bammann, Coulam and Jiang, 1980; Kerlan, Nahoul, Le Martelot and Bercovici, 1994).[16] Instrumental variable regressions based on both instruments (drugs administered during pregnancy and testosterone) are referred to as IV2. The first stage equation is:

$$\text{Cortisol} = \alpha + \tau_1 \text{Drugs} + \tau_2 \text{Testosterone} + \tau_3 \mathbf{X} + \tau_4 \text{Birthweight} + \upsilon \qquad (2)$$

In Table 7A are results from the first stage in which we estimate prenatal cortisol levels as a function of the instruments listed above and the exogenous characteristics of the family and child. Administration of any one of these drugs during pregnancy increases the level of maternal cortisol by 30 percent of a standard deviation. Testosterone levels also have a positive and significant effect on cortisol levels – a one standard deviation increase in testosterone is associated with an increase in cortisol of 10 percent of a standard deviation. In addition, the administration of these drugs does not appear to be correlated with SES. When we regress an indicator for whether a mother

[16] In non-pregnant women, testosterone levels have been found to be associated with age, BMI and smoking, but not alcohol consumption, physical exercise or nutritional intake (Sowers et al, 2001).

received any of these drugs on maternal education, marital status, income, race, parity, child gender and pregnancy complication, the only coefficient that is statistically significant (at the 10% level) is pregnancy complication (Table 8). All other coefficient estimates are small in magnitude and imprecisely estimated and an F statistic of all family characteristics is only 1.39. It does not appear that receiving these drugs is correlated with SES, nor is testosterone (with the exception of race, which is well established in the medical literature and a small negative association with the number of siblings).

To instrument for the interaction cortisol*black, we follow Wooldridge (2002) and construct an additional variable $\widehat{\text{cortisol}}$*black based on predicted cortisol from the above first stage (equation 2) interacted with black. This variable serves as an instrument for cortisol*black. The results from this regression are in Table 7B. Not surprisingly $\widehat{\text{cortisol}}$*black is a strong instrument for cortisol*black.

In Table 9A are IV estimates of the impact of maternal prenatal cortisol on offspring IQ at age 4. We focus on the interaction between (instrumented) cortisol and race in the IV regressions. The coefficient estimate on the interaction term (cortisol*black) is negative and significant implying that a standard deviation increase in maternal cortisol leads to a 7 point reduction in IQ (half a standard deviation decline) among black children. The impact is slightly larger if we use estrogen/progesterone/DES and testosterone as instruments (IV2 vs IV1). In Table 10 are IV estimates of the impact of maternal prenatal cortisol on 7 year IQ which are roughly one third smaller (and insignificant for IV2).

We also explore whether the impact of cortisol might be greater for other groups whom we believe face more stressful environments: poor families and single mothers. In Table 9B are IV estimates of the impact of prenatal cortisol on child IQ that include interactions between cortisol and poverty (columns1 and 2) and cortisol and single motherhood (columns 3 and 4). The coefficient on the interaction term between cortisol and poverty is small and insignificant, but the coefficient on the interaction between cortisol and single mother is negative and significant. A one standard deviation in maternal prenatal cortisol decreases child IQ among the children of single mothers by 8 points or 55% of a standard deviation. This evidence is somewhat consistent with the hypothesis put forth by Boyce et al (1995) that the negative impact of dysregulated stress response system is greater for those in stressful environments.

Impact of Prenatal Maternal Cortisol on the Transmission of Adult Economic Status

Finally, we estimate the impact of maternal prenatal cortisol on the transmission of adult economic status. Because of measurement error in the adult income measures collected, we rely instead on intergenerational transmission of education. In addition, education is arguably a better measure of permanent income than a single year of earnings in one's early 30's. We generate three outcome measures to capture intergenerational transmission of education: 1) the natural log of the ratio of adult education to maternal education, ln(adult education/maternal education), the linear difference (adult education-maternal education), and the percent change in education (adult education-maternal education)/maternal education. Nearly all children (95%) are

more educated than their mothers in this sample. The median gain in years of education between generations was two years, the average three (for whites the average gain was 3.2 years of education and for blacks 2.7 years).

In all three OLS regressions (columns 1, 4, 7 of Table 11), both cortisol and the interaction black*cortisol have negative but insignificant effects on the intergenerational transmission of education. In the IV regressions, the estimated impact of the interaction between black and cortisol increases in (absolute) magnitude and is now significant. For black children, increasing prenatal cortisol by one standard deviation results in 30% less gain in educational attainment (one year less) relative to one's mother. In regressions not shown, the impact of cortisol on adult educational gains does not appear to be greater for single mothers once we instrument for cortisol. The fact that once we instrument we only find effects for blacks but not other groups suggests that either blacks face significantly more stress in their environments than other low SES groups or that blacks' deficiencies in vitamin D are responsible for the greater impact of cortisol that we find.

VI. Conclusions

In this paper we explore whether stress hormones and prenatal programming can explain why rates of persistent poverty are higher for black families. Stress hormones are higher in low socio-economic status mothers and medical evidence has shown that elevated prenatal cortisol can affect offspring cognition, consistent with a prenatal programming hypothesis. In addition, recent theoretical and empirical evidence suggests that these effects can vary with the child's environment: negative effects are found for

those children in stressful environments and positive effects for those in protective environments, suggesting an important interaction between nature and nurture.

We find that elevated prenatal cortisol levels results in lower cognitive ability and educational attainment of offspring, but the effect is concentrated on blacks. Given the higher levels of stress black American face and their greater stress reactivity documented in the literature, these findings are consistent with existing theory regarding heterogeneous effects based on the stress of the child's environment. But they are also consistent with documented biological differences between the races in vitamin D acquisition and recent evidence that vitamin D can serve to mitigate the deleterious effects of cortisol on the brain. The fact that we do not find persistent significant effects for other groups that are also subject to more stressful environments (poor mothers and single mothers), suggests that physiological differences in vitamin D production may be responsible, though we cannot rule out alternative explanations.

These results help us to understand the role of both nature and nurture in explaining why poor black children are less likely to escape poverty than their white counterparts and underscore the importance of in-utero conditions, and not only nutrition, in explaining the intergenerational transmission of economic status. They also underscore the ways in which discrimination may affect the children of black mothers not only through overt discrimination in the job market but also indirectly by elevating the level of stress in their lives, thereby negatively affecting their human capital accumulation and future economic status.

Appendix II: Testosterone and the Impact of Cortisol on Blacks

We argue that the impact of cortisol on blacks that we find is not attributable to higher rates of testosterone among blacks. We provide as evidence estimates of the impact of testosterone and testosterone interacted with cortisol on IQ score at age 4 (column 2 appendix table 1). We find no significant relationship. Finally we add testosterone and its interaction with black in a regression that includes the interaction between black and cortisol (column 3 appendix table 1) and find that the coefficient on the interaction black*cortisol is not diminished when we include controls for testosterone and testosterone*cortisol. We conclude that the interactive effect between cortisol and race that we find is not driven by other factors correlated with race.

References

Becker, Gary and Nigel Tomes (1979) “An Equilibrium Theory of the Distribution of Income and Intergenerational Mobility", *Journal of Political Economy*

Loury, Glenn (1981) “Intergenerational Transfers and the Distribution of Earnings,” *Econometrica,* Vol. 49, No. 4: 843-867.

Perraudin, William and Bent E. Sørensen (1992) “The Credit-Constrained Consumer: An Empirical Study of Demand and Supply in the Loan Market” *Journal of Business & Economic Statistics*, Vol. 10, No. 2: 179-192.

Table 1 Characteristics of NCPP and Analysis Sample

	Entire NCPP	Bos/Pvd NCPP	Analysis Sample	1960 Census
Maternal Characteristics				
Share White	0.47	0.86	0.87	0.97
Share Black	0.46	0.12	0.12	0.03
Share Hispanic	0.06	0.002	0.00	
Maternal Age	24.3	25.2	25.0	30.62
Single	0.23	0.11	0.09	0.00
Maternal Education	10.62	11.40	11.12	11.42
Share <HS	0.55	0.39	0.43	0.12
Share HS	0.36	0.45	0.50	0.70
Share College	0.04	0.08	0.05	0.18
SES index	47	58	56	
Income	4030	4903	4784	6475
Immigrant	0.11	0.10	0.06	0.06
Maternal work	0.15	0.19	0.17	0.16
Share in Providence	0.07	0.23	0.51	0.14
Child Characteristics				
male	0.48	0.47	0.41	
birthweight (gms)	3096	3185	3307	
birthweight (gms)- male		3239	3384	
birthweight (gms)- female		3109	3258	
observations (# children)	59392	17921	1103	4080

1960 census includes all women with a child <5 living in Massachusetts or Rhode Island in 1960.

Table 2 Persistence of Economic Status by Race

	Adult Household income Bottom 25%	Adult HS Drop-Out
a. Children born into bottom 25% of Household Income distribution		
White	0.313	0.085
Black	0.426	0.227
b. Children born to Mothers without a HS degree		
White	0.284	0.072
Black	0.576	0.164

Table 3 Determinants of Adult Economic Status

	Adult Income in Bottom 25 Percent						Adult HS Drop Out					
black	0.067	0.065	0.064	0.054	-0.006	0.01	0.064	0.057	0.065	0.059	0.016	0.058
	[0.039]	[0.039]	[0.039]	[0.038]	[0.040]	[0.040]	[0.021]	[0.021]	[0.021]	[0.021]	[0.021]	[0.022]
male	-0.219	-0.219	-0.217	-0.212	-0.21	-0.215	0.018	0.017	0.017	0.021	0.024	0.02
	[0.026]	[0.026]	[0.026]	[0.026]	[0.026]	[0.026]	[0.014]	[0.014]	[0.014]	[0.014]	[0.013]	[0.014]
age	0	0	-0.001	0.002	0.002	0.003	-0.005	-0.004	-0.004	-0.004	-0.003	-0.002
	[0.007]	[0.007]	[0.007]	[0.007]	[0.007]	[0.007]	[0.004]	[0.004]	[0.004]	[0.004]	[0.004]	[0.004]
maternal score SRA		-0.017						-0.042				
		[0.041]						[0.022]				
teenage mother			0.05						-0.024			
			[0.043]						[0.023]			
maternal highest grade completed				-0.039						-0.015		
				[0.006]						[0.003]		
child IQ					-0.006						-0.004	
					[0.001]						[0.001]	
annual family income at birth						-0.049						-0.028
						[0.011]						[0.006]
Observations	1049	1049	1049	1049	1049	1007	1043	1043	1043	1043	1043	1001
R-squared	0.07	0.07	0.07	0.1	0.1	0.08	0.01	0.02	0.01	0.03	0.06	0.03

Standard errors in brackets

Table 4 Impact of Cortisol on Child IQ - Stratified by Parental Poverty, Race and Marital Status

	Above Poverty	Below Poverty	White	Black	White	Black	Married	Single	Married	Single
cortisol (ng/ml)	-0.012	-0.045			-0.009	-0.036			-0.008	-0.042
	[0.006]	[0.009]			[0.006]	[0.013]			[0.006]	[0.014]
income at birth in 1000s			2.667	2.664	2.568	1.9	2.429	2.158	2.336	1.486
			[0.394]	[1.168]	[0.400]	[1.173]	[0.405]	[1.290]	[0.410]	[1.261]
Observations	860	228	961	135	952	135	1006	91	998	90
R-squared	0.02	0.11	0.07	0.04	0.07	0.1	0.05	0.03	0.05	0.12

Standard errors in brackets
all regressions control for gender

Table 5 Impact of Maternal Prenatal Cortisol on 4 Year IQ - OLS

	(1)	(2)	(3)	(4)	(5)	(6)
cortisol (ng/ml)	-0.009	-0.001	-0.003	0	0.003	-0.001
	[0.005]	[0.006]	[0.006]	[0.006]	[0.007]	[0.006]
black*cortisol		-0.038			-0.03	-0.037
		[0.013]			[0.013]	[0.013]
black*poverty						-0.682
						[2.724]
cortisol*single			-0.041			
			[0.016]			
poverty*cortisol				-0.027	-0.019	
				[0.011]	[0.010]	
below poverty (at birth)				6.277	4.127	
				[3.311]	[3.110]	
maternal score SRA	0.059	0.057	4.496	0.058	0.056	0.057
	[0.014]	[0.014]	[1.397]	[0.014]	[0.014]	[0.014]
maternal education <HS	-11.386	-11.833	-11.134	-11.31	-11.663	-11.809
	[1.535]	[1.488]	[1.508]	[1.498]	[1.511]	[1.492]
maternal education HS	-7.845	-7.966	-7.889	-7.83	-7.926	-7.97
	[1.456]	[1.492]	[1.515]	[1.493]	[1.498]	[1.492]
black	-5.509	5.51	-5.373	-5.409	3.332	5.595
	[1.413]	[4.290]	[1.542]	[1.519]	[4.247]	[4.337]
single	-6.88	-6.356	5.823	-5.996	-5.795	-6.322
	[1.711]	[1.583]	[5.619]	[1.664]	[1.636]	[1.587]
age of mother, years	0.209	0.222	0.211	0.22	0.227	0.222
	[0.091]	[0.088]	[0.087]	[0.089]	[0.088]	[0.088]
ln(income at birth)	0.855	0.782	0.632	0.601	0.571	0.754
	[0.646]	[0.504]	[0.415]	[0.545]	[0.544]	[0.512]
number of siblings	-0.209	-0.232	-0.176	-0.137	-0.164	-0.227
	[0.273]	[0.278]	[0.270]	[0.288]	[0.289]	[0.277]
male	-3.476	-3.608	-3.493	-3.537	-3.624	-3.612
	[0.867]	[0.880]	[0.873]	[0.879]	[0.881]	[0.882]
Providence	-4.082	-3.997	-3.842	-3.938	-3.885	-4.007
	[0.955]	[0.988]	[0.993]	[1.024]	[1.026]	[0.989]
pregnancy complication	-2.981	-3.106	-3.107	-2.933	-3.041	-3.107
	[0.885]	[0.856]	[0.851]	[0.860]	[0.856]	[0.856]
Observations	1007	1007	993	1007	1007	1007
R-squared	0.24	0.25	0.24	0.25	0.25	0.25

Standard errors in brackets

Table 6 Impact of Maternal Prenatal Cortisol on 7 Year IQ - OLS

	(1)	(2)	(3)	(4)	(5)
cortisol (ng/ml)	-0.008	-0.003	-0.002	0	-0.003
	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]
black*cortisol		-0.025		-0.019	-0.024
		[0.011]		[0.010]	[0.011]
black*poverty					-1.204
					[2.402]
poverty*cortisol			-0.019	-0.014	
			[0.009]	[0.009]	
below poverty (at birth)			3.351	2.055	
			[2.748]	[2.699]	
maternal score	0.052	0.051	0.05	0.049	0.051
	[0.012]	[0.012]	[0.012]	[0.012]	[0.012]
Maternal education <HS	-8.898	-9.193	-8.705	-8.923	-9.154
	[1.297]	[1.308]	[1.317]	[1.320]	[1.311]
Maternal education HS	-5.043	-5.127	-5.025	-5.085	-5.131
	[1.248]	[1.249]	[1.254]	[1.253]	[1.251]
black	-6.386	0.998	-6.332	-0.908	1.106
	[1.314]	[3.286]	[1.306]	[3.202]	[3.292]
single	-5.639	-5.309	-4.7	-4.588	-5.243
	[1.626]	[1.612]	[1.649]	[1.629]	[1.624]
age of mother, years	0.138	0.147	0.144	0.149	0.147
	[0.084]	[0.083]	[0.085]	[0.084]	[0.083]
ln(income at birth)	0.532	0.474	0.075	0.054	0.428
	[0.426]	[0.422]	[0.421]	[0.421]	[0.419]
number of siblings	-0.259	-0.273	-0.137	-0.153	-0.265
	[0.233]	[0.233]	[0.239]	[0.240]	[0.231]
male	1.35	1.259	1.293	1.238	1.254
	[0.778]	[0.781]	[0.778]	[0.782]	[0.783]
Providence	-4.417	-4.36	-4.155	-4.123	-4.379
	[0.843]	[0.844]	[0.879]	[0.880]	[0.843]
pregnancy complication	-1.011	-1.104	-0.941	-1.013	-1.103
	[0.774]	[0.770]	[0.771]	[0.768]	[0.770]
Observations	988	988	988	988	988
R-squared	0.26	0.26	0.26	0.26	0.26

Robust standard errors in brackets

Table 7 Determinants of Maternal Prenatal Cortisol - First Stage

	(1)	(2)
estrogen/progesterone/DES	25.47	21.589
	[13.858]	[14.957]
testosteron (ng/ml)		17.142
		[5.387]
Gestation at blood draw	2.917	2.546
	[1.715]	[1.709]
maternal score	-0.108	-0.105
	[0.084]	[0.086]
maternal education <HS	8.831	7.641
	[9.376]	[9.394]
maternal education HS	11.6	11.571
	[8.901]	[8.902]
black	6.015	1.188
	[9.824]	[8.776]
single	32.45	33.206
	[11.673]	[10.422]
age of mother, years	-1.996	-1.773
	[0.549]	[0.561]
ln(income at birth)	-8.046	-7.97
	[3.580]	[3.953]
number of siblings	-5.303	-4.793
	[1.658]	[1.672]
male	1.869	1.318
	[5.208]	[5.309]
Providence	4.457	4.765
	[5.932]	[5.854]
pregnancy complication	8.296	8.246
	[5.576]	[5.454]
Observations	1007	1007
R-squared	0.1	0.11

Robust standard errors in brackets

Table 8 Determinants of Prenatal Drug Administration

	(1) estrogen/progesterone/DES	(2) testosterone
maternal score	0	0
	[0.000]	[0.000]
maternal education <HS	-0.023	0.064
	[0.025]	[0.054]
maternal education HS	-0.028	-0.003
	[0.024]	[0.045]
black	-0.015	0.277
	[0.016]	[0.082]
single	-0.02	-0.049
	[0.018]	[0.059]
age of mother, years	0	-0.012
	[0.001]	[0.004]
ln(income at birth)	-0.007	-0.003
	[0.009]	[0.017]
number of siblings	0	-0.032
	[0.004]	[0.010]
male	-0.015	0.028
	[0.011]	[0.033]
Providence	-0.017	-0.017
	[0.012]	[0.041]
pregnancy complication	0.046	0.009
	[0.014]	[0.031]
gestational age(wks) at draw_date	0	0
	[0.000]	[0.000]
Observations	1014	1007
R-squared	0.02	0.09
Robust standard errors in brackets		
F statistic	1.39	14.02
p value	0.1708	0

Table 9A Impact of Maternal Prenatal Cortisol on IQ 4 Years - IV

	OLS	IV1	IV2
cortisol (ng/ml)	-0.001	0.141	-0.002
	[0.006]	[0.082]	[0.045]
black*cortisol	-0.038	-0.155	-0.154
	[0.013]	[0.064]	[0.052]
maternal score	0.057	0.075	0.059
	[0.014]	[0.020]	[0.015]
maternal education <HS	-11.833	-12.88	-11.643
	[1.488]	[1.993]	[1.639]
maternal education HS	-7.966	-9.417	-7.876
	[1.492]	[2.084]	[1.699]
black	5.51	39.666	40.293
	[4.290]	[19.089]	[15.308]
single	-6.356	-9.047	-4.741
	[1.583]	[3.140]	[2.219]
age of mother, years	0.222	0.467	0.19
	[0.088]	[0.193]	[0.126]
ln(income at birth)	0.782	1.948	0.781
	[0.504]	[0.857]	[0.598]
number of siblings	-0.232	0.536	-0.231
	[0.278]	[0.549]	[0.359]
male	-3.608	-3.829	-3.564
	[0.880]	[1.120]	[0.927]
Providence	-3.997	-4.794	-4.167
	[0.988]	[1.297]	[1.053]
pregnancy complication	-3.106	-4.22	-2.915
	[0.856]	[1.365]	[0.986]
Observations	1007	1014	1007
R-squared	0.25	0.25	0.25

Robust standard errors in brackets

Table 9B IV Estimates of the Impact of Cortisol on Child IQ (age 4) - Interactions with Poverty and Marital Status

	IV1	IV2	IV1	IV2
	(1)	(2)	(3)	(4)
below poverty at birth*cortisol	0.002	-0.002		
	[0.033]	[0.030]		
single*cortisol			-0.097	-0.092
			[0.043]	[0.045]
cortisol	0.115	-0.04	0.14	-0.03
	[0.072]	[0.046]	[0.068]	[0.044]
below poverty at birth	-1.478	-0.269		
	[9.260]	[8.647]		
black	-6.774	-5.828	-6.535	-5.545
	[1.589]	[1.556]	[1.588]	[1.564]
maternal education	1.661	1.481	1.812	1.61
	[0.225]	[0.218]	[0.234]	[0.228]
maternal score	6.739	5.218	6.282	4.686
	[1.542]	[1.453]	[1.568]	[1.495]
single	-9.631	-4.813	21.168	24.463
	[2.509]	[2.089]	[13.742]	[14.319]
age of mother, years	0.487	0.19	0.486	0.163
	[0.167]	[0.127]	[0.161]	[0.126]
ln(income at birth)	1.702	0.465	1.779	0.463
	[0.756]	[0.638]	[0.709]	[0.611]
number of siblings	0.472	-0.388	0.635	-0.311
	[0.459]	[0.359]	[0.459]	[0.362]
male	-3.907	-3.539	-4.175	-3.752
	[0.903]	[0.901]	[0.912]	[0.913]
Providence	-4.234	-3.78	-4.566	-4.07
	[1.019]	[1.006]	[0.980]	[0.969]
pregnancy complication	-3.637	-2.389	-3.955	-2.594
	[1.080]	[0.942]	[1.074]	[0.946]
birth weight kg	2.179	1.107	1.985	0.841
	[0.941]	[0.898]	[0.957]	[0.917]
Observations	1007	1007	1007	1007
R-squared	0.24	0.24	0.24	0.24

Robust standard errors in brackets

Table 10 Impact of Maternal Prenatal Cortisol on IQ 7 Years - IV Estimates

	OLS	IV1	IV2
cortisol (ng/ml)	-0.003	0.096	0.078
	[0.005]	[0.075]	[0.043]
black*cortisol	-0.025	-0.093	-0.058
	[0.011]	[0.053]	[0.046]
maternal score	0.051	0.063	0.061
	[0.012]	[0.016]	[0.015]
maternal education <HS	-9.193	-9.955	-9.675
	[1.308]	[1.642]	[1.508]
maternal education HS	-5.127	-6.195	-5.972
	[1.249]	[1.670]	[1.478]
black	0.998	20.638	10.481
	[3.286]	[15.646]	[13.829]
single	-5.309	-7.539	-7.493
	[1.612]	[3.149]	[2.413]
age of mother, years	0.147	0.31	0.29
	[0.083]	[0.167]	[0.123]
ln(income at birth)	0.474	1.28	1.154
	[0.422]	[0.796]	[0.577]
number of siblings	-0.273	0.297	0.195
	[0.233]	[0.486]	[0.343]
male	1.259	1.207	1.205
	[0.781]	[0.918]	[0.885]
Providence	-4.36	-4.875	-4.831
	[0.844]	[1.038]	[0.980]
pregnancy complication	-1.104	-1.838	-1.714
	[0.770]	[1.133]	[0.946]
Observations	988	995	988
R-squared	0.26	0.26	0.26

Robust standard errors in brackets

Table 11 OLS and IV Regressions of the Impact of Cortisol on Intergenerational Transmission of Education

	ln(education/maternal education)			**education - maternal education**			**% change in education level**		
	OLS	IV1	IV2	OLS	IV1	IV2	OLS	IV1	IV2
black*cortisol	-0.00016	-0.00151	-0.0014	-0.00107	-0.01445	-0.01335	-0.00029	-0.00245	-0.00236
	[0.00021]	[0.00066]	[0.00075]	[0.00259]	[0.00722]	[0.00813]	[0.00028]	[0.00114]	[0.00131]
cortisol (ng/ml)	-0.00009	0.00788	0.00418	-0.00163	0.07852	0.04289	-0.00011	0.01166	0.00603
	[0.00009]	[0.00109]	[0.00072]	[0.00110]	[0.01250]	[0.00875]	[0.00015]	[0.00180]	[0.00120]
black	-0.02957	0.30611	0.3012	-0.46766	2.866	2.81572	-0.03679	0.50679	0.52318
	[0.06707]	[0.19258]	[0.22080]	[0.81428]	[2.11754]	[2.41515]	[0.09559]	[0.32027]	[0.37584]
single	0.04288	-0.20807	-0.08949	0.14423	-2.44661	-1.3081	0.09824	-0.26829	-0.08734
	[0.03373]	[0.04747]	[0.04061]	[0.36657]	[0.53165]	[0.45030]	[0.06515]	[0.07823]	[0.07134]
age of mother, years	0.00066	0.01514	0.0083	-0.00035	0.14345	0.07768	0.00377	0.02507	0.0147
	[0.00174]	[0.00287]	[0.00228]	[0.02016]	[0.03121]	[0.02553]	[0.00304]	[0.00534]	[0.00436]
income at birth in 1000s	-0.01136	0.02574	0.00915	-0.05826	0.32906	0.16957	-0.01984	0.03433	0.00911
	[0.00683]	[0.00804]	[0.00765]	[0.08501]	[0.09981]	[0.09494]	[0.01114]	[0.01242]	[0.01181]
number of siblings	0.00148	0.04305	0.02312	-0.01445	0.40965	0.21819	-0.00036	0.06087	0.03052
	[0.00496]	[0.00720]	[0.00618]	[0.05682]	[0.08367]	[0.07237]	[0.00842]	[0.01101]	[0.00951]
male	-0.05385	-0.06589	-0.05942	-0.7988	-0.90315	-0.84064	-0.06259	-0.07953	-0.06992
	[0.01402]	[0.01416]	[0.01443]	[0.17329]	[0.17872]	[0.18034]	[0.02127]	[0.02093]	[0.02132]
Providence	0.02927	-0.00888	0.00857	0.05099	-0.32863	-0.16107	0.0537	-0.00185	0.02471
	[0.01477]	[0.01477]	[0.01495]	[0.18893]	[0.19622]	[0.19469]	[0.02148]	[0.02085]	[0.02154]
pregnancy complication	0.03274	-0.04441	-0.0104	0.42222	-0.36667	-0.04019	0.05575	-0.0575	-0.00591
	[0.01447]	[0.01632]	[0.01603]	[0.17902]	[0.20712]	[0.20079]	[0.02263]	[0.02367]	[0.02378]
Observations	1093	1093	1093	1093	1093	1093	1093	1093	1093
R-squared	0.03	0.07	0.04	0.03	0.05	0.03	0.04	0.06	0.03

Robust standard errors in brackets

Figure 2A
4 Year IQ and Parental SES at Birth

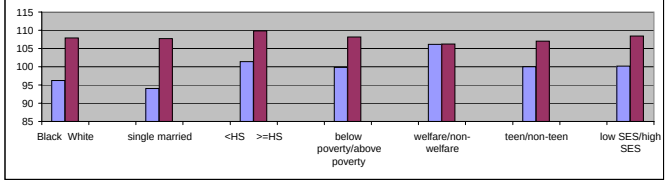


Figure 2B
4 year IQ and Adult SES

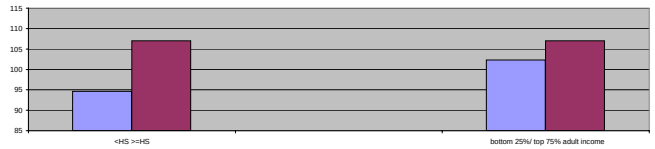


Figure 3A
7 year IQ and Parental SES at Birth

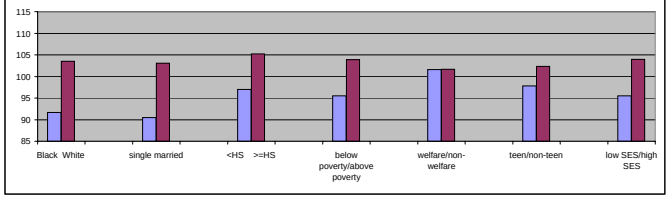
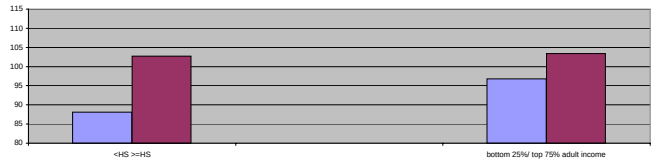


Figure 3B
7 year IQ and Adult SES



Appendix Table I Is Testoerone Driving the Results for Blacks?

	4 year IQ	7 year IQ	4 year IQ	7 year IQ
cortisol (ng/ml)	-0.002	-0.003	-0.002	-0.003
	[0.012]	[0.009]	[0.011]	[0.009]
testosterone*cortisol	-0.006	-0.006	0.003	-0.001
	[0.012]	[0.009]	[0.012]	[0.009]
testosterone	-0.073	2.866	-2.504	1.464
	[3.253]	[2.736]	[3.217]	[2.761]
black*cortisol			-0.04	-0.024
			[0.014]	[0.012]
maternal score	0.059	0.052	0.057	0.051
	[0.014]	[0.012]	[0.014]	[0.012]
maternal education <HS	-11.298	-8.965	-11.756	-9.24
	[1.482]	[1.291]	[1.509]	[1.308]
maternal education HS	-7.836	-5.002	-8.005	-5.106
	[1.487]	[1.246]	[1.509]	[1.250]
black	-5.094	-6.717	6.753	0.33
	[1.555]	[1.319]	[4.438]	[3.507]
single	-6.953	-5.543	-6.46	-5.259
	[1.622]	[1.622]	[1.577]	[1.607]
age of mother, years	0.188	0.15	0.205	0.16
	[0.089]	[0.085]	[0.087]	[0.084]
ln(income at birth)	0.863	0.525	0.778	0.468
	[0.511]	[0.428]	[0.507]	[0.424]
number of siblings	-0.241	-0.208	-0.29	-0.235
	[0.276]	[0.236]	[0.275]	[0.238]
male	-3.435	1.304	-3.569	1.223
	[0.880]	[0.779]	[0.880]	[0.783]
Providence	-4.116	-4.386	-4.026	-4.333
	[0.986]	[0.841]	[0.987]	[0.845]
pregnancy complication	-2.977	-1.024	-3.109	-1.107
	[0.860]	[0.774]	[0.853]	[0.770]
gestational age(wks) at draw_date	0	0	0	0
	[0.000]	[0.000]	[0.000]	[0.000]
Observations	1007	988	1007	988
R-squared	0.25	0.26	0.25	0.26

Robust standard errors in brackets