Maternal Stress and Child Well-Being: Evidence from Siblings

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We study how maternal stress affects child outcomes. We find that in-utero exposure to elevated levels of the stress hormone cortisol negatively affects the cognition, health and educational attainment of offspring. These findings are based on comparisons between siblings to control for unobserved differences between mothers that could bias estimates. Our results are consistent with recent experimental results in the neuro-biological literature linking exogenous exposure to coritsol in-utero with reduced growth in the hippocampal region of the brain and declines in offspring cognitive, behavioral and motor development. Moreover, we find that the cortisol levels of low SES mothers are both higher on average and more variable, suggesting that prenatal stress may play an important role in explaining why relatively few children born into poverty are able to escape it as adults.

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I. Introduction

Intergenerational correlations in economic status in the US are high (Corak, 2004; Solon, 1999). Boys born to families with income in the bottom quintile of the income distribution have a 42 percent change of remaining there as adults and only a five percent chance of reaching the top quintile. Yet little is known about the mechanisms by which parents transmit their economic status to their children. Evidence based on twins and adoptees suggests that both nature (defined as genetic heritability of traits) and nurture (the environment provided by one's parents) play a role. But how the latter affects offspring outcomes is not well understood. In this work we focus on the role played by the parental environment and, in particular, exposure to environmental stressors in influencing offspring outcomes.

We focus on prenatal exposure to stress as a mechanism by which parents affect the economic outcomes of their children for two reasons. First, poverty is associated with greater levels of stress. The poor, on average, report a greater number of stressful events in their lives and researchers have observed higher levels of biological markers for stress in low socio-economic status adults. Second, recent evidence in neuro-biology based largely on animal experiments suggests that exogenous exposure to stress in-utero negatively affects the cognitive, behavioral and motor development of offspring. Given that these factors also determine adult economic status, greater in-utero exposure to stress among the poor has the potential to explain, in part, the intergenerational persistence of poverty in the US.

In this paper we estimate the impact of in utero exposure to stress on adult offspring educational attainment (years of schooling) using a unique dataset with detailed

information on parental characteristics including prenatal levels of the hormone cortisol (a marker for stress) and offspring outcomes. Because mothers with greater stress may differ in unobserved ways from those with lower stress, we include maternal fixed effects to address potential omitted variable bias. In this way, we limit identifying variation to temporary shocks during the prenatal period that do not appear to persist into the post natal environment.[1] When we include maternal fixed effects, we find that exposure to elevated cortisol in utero has a negative and significant impact on offspring educational attainment. To better understand why exposure to stress in-utero affects offspring educational attainment, we estimate the impact of prenatal stress on two intermediate outcomes: cognition and health at age 7.[2] Specifically, we estimate the impact of prenatal cortisol on child IQ and presence of a severe chronic health condition. We find that exposure to prenatal stress negatively affects both the health and cognition of offspring, suggesting at least two potential mechanisms behind the negative impact of cortisol on years of schooling. We follow the analysis with additional supporting evidence that the estimates reflect a causal relationship between prenatal maternal stress and offspring outcomes.

This work has a number of important implications. First, it provides further evidence that the prenatal environment has a lasting impact on offspring outcomes. This is consistent with recent work by economists in this area that has examined the impact of prenatal nutrition, as proxied by birthweight, (Black, Devereaux and Salvanjes, 2007), in

[1] Low SES mothers (defined as those with less than a HS education) are characterized by both higher average cortisol levels and greater variation in cortisol levels across births.

[2] The evidence that physical health affects schooling is limited. The most compelling evidence that child health, especially nutrition, positively affects schooling comes from developing countries. (See Alderman, et al 2001 and Miguel and Kremer, 2003.) In the US, Bleakley (2007) finds that hookworm eradication among children increased school attendance, literacy and long term income. There is more evidence that mental health affects schooling in developed countries (Currie and Stabile, 2007.)

utero exposure to the flu (Almond, 2006) and low levels of radiation (Almond, Edlund and Palme, 2008) on short and long term offspring outcomes including cognitive achievement, disability and mortality. Second, it underscores the importance of maternal stress, a difficult to measure environmental factor, in determining offspring cognition and health in humans, consistent with the growing animal literature. Our emphasis on stress represents an important advance as stress may be more relevant than nutrition or radiation in the context of the US. As a result, our estimates may generalize to a greater number of pregnant women. Finally, and perhaps most importantly, the results have implications for our understanding of the various mechanisms by which parental poverty and its corresponding greater average levels of stress and greater variability can negatively affect child outcomes and future economic status by negatively influencing the child's earliest environmental conditions.

The rest of the paper is organized as follows. In section II, we provide background information on the relationship between stress, cortisol, prenatal conditions and offspring outcomes. In sections III and IV, we describe our empirical strategy and data, respectively. In section V we present our empirical results, including robustness checks. Section VI concludes.

II. Background on Economic Status, Stress and Prenatal Programming

Related Literature on Prenatal Conditions and Offspring Outcomes

Work on the intergenerational transmission of economic status in the US has produced estimates of intergenerational income elasticities on the order of 0.5 with the highest estimate approaching 0.65 (Solon, 1999; Mazunder, 2005). Most of the existing

theoretical and empirical work that has sought to identify the mechanisms of intergenerational transmission of economic status has categorized the mechanisms as either nature (genetic inheritance) or nurture (the environment). With respect to the latter, the importance of the early childhood environment in affecting long term outcomes is now well-established (see Currie, 2001 and Heckman and Masterov, 2007 for excellent summaries). More recently, however, researchers have focused on the role of even earlier environmental conditions - those found in-utero. For example, Almond (2006) and Almond, Edlund and Palme (2008) provide convincing evidence of the importance of the in-utero environment in determining offspring outcomes by estimating negative and significant effects of in-utero exposure to the flu pandemic of 1917 and low levels of radiation, respectively, on cognitive achievement, schooling, rates of disability and welfare receipt. While this work establishes the importance of the prenatal environment in affecting long term offspring outcomes, because the shocks to the in-utero environment are random and uncorrelated with maternal economic status, it does not establish a poor in-utero environment as a mechanism by which poor mothers might transmit economic status to their offspring.

One area of research that can potentially link parental economic status with offspring outcomes through deprivation of the in-utero environment is that which has focused on the impact of birthweight (as a measure of prenatal nutrition) on offspring outcomes. Examples of this literature include Almond, Chay and Lee (2005), Royer (2006), Black, Devereaux and Salvanes (2007) and Behrman and Rosensweig (2004) who use within-twin differences in birthweight to identify the impact of fetal nutrition (assuming differences in birthweight reflect differences in nutrition only) on future

outcomes including health, mortality, education and earnings. They find relatively small short run effects but larger long run effects. Recent work by Currie and Moretti (2008) explores intergenerational correlations in birthweight. They find that mother's born low birth weight (LBW) are 50 percent more likely to give birth to LBW babies. [3] Other related studies include that of Qian and Meng (2009) and Almond, Edlund, Li and Zhang (2007) who find that in utero exposure to famine conditions in China results in diminished adult offspring outcomes (educational attainment, literacy, health and employment). While these results are attributed to reduced nutrition in-utero, they are also consistent with exposure to greater stress as a result of food insecurity.

The present study differs in important ways from previous work estimating the impact of prenatal conditions on offspring outcomes. First, we focus on exposure to in-utero stress because recent experimental evidence in the neuro-biological literature, summarized in the next section, has positively identified a biological mechanism by which exposure to in-utero stress hormones can negatively impact offspring cognition and health. We argue that because stress is relatively common among pregnant women in the US, the results are highly generalizeable. In addition, because poverty is associated with higher levels of stress, the results have important implications for our understanding of the mechanisms behind the intergenerational transmission of economic status. Specifically, our results suggest that the greater exposure to stress in-utero among the children of poor families can potentially explain, in part, the persistence of poverty in the US.

[3] Another related literature is that linking social status with longevity. This literature, which includes Marmot's Whitehall studies (Marmot, 2004) and others (eg, Rablen and Oswald, forthcoming), often hypothesizes that lower social status may increase mortality by increasing levels of stress.

SES, Stress and Prenatal Programming

Cortisol is a corticosteroid hormone or glucocorticoid produced by the adrenal cortex that is often referred to as the "stress hormone" as it is involved in the physiological response to stress and anxiety and is observed in higher than average levels in those exposed to greater stress (Wust et. al., 2000; Van Eck et al, 1996). While normal circulating levels of the hormone cortisol are necessary for a healthy adaptive response to stress, unusually high (or, in rare cases, unusually low) levels of cortisol have been linked with various pathologies.[4]

There is evidence of a socio-economic gradient in stress. Previous research has found that low income individuals report greater stressful events in their lives (Dohrenwend, 1973). Consistent with this, medical researchers have established higher than average cortisol levels among those of low economic and social status (Cohen et. al., 2006; Steptoe et al, 2003; Kunkz-Ebrecht, Kirschbaum and Steptoe, 2003).

Cortisol is considered a key agent in 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). Work based largely on animal studies has established a strong link between exogenous in-utero exposure to stress/cortisol and poor offspring outcomes. These studies generally fall into two categories: those that inject cortisol (glucocorticoids) directly into pregnant animals and those that exogenously expose pregnant animals to an environmental

[4] Elevated cortisol has been implicated in hypertension, cardiovascular disease, insulin resistance, obesity, diabetes, infection illness and depressive disorder, among others as well as the development of atypical emotional, behavioral and cognitive functioning (Ousova et al, 2004; Walker et al, 1998; Steptoe et al, 2002; McEwen, 1998; McBurnett et al 2000; Dawes et al, 1999; Van Goozen et al; 1998).

stressor. Examples of the former include Uno et al (1983) who expose fetal rheuses monkeys to high concentrations of cortisol and find that they suffer considerable damage to the hippocampus region of the brain. Welberg, Seckl and Holmes, (2001) likewise administered glucocorticoids to pregnant rats and found that the offspring of exposed rats exhibited behavioral inhibition, and impaired coping and learning in aversive situations.[5] An example of the latter type of study is Schneider (1992) who subjected pregnant rhesus monkeys to what they classify as a "mild stressor" and observed impaired motor ability and delays in learning among the offspring.[6] In a follow-up to this study, Schneider, Coe and Lubach (1992) successfully mimicked the negative impact of this mild stressor by directly injecting pregnant rhesus monkeys with stress hormones. They observed similar declines in motor and mental development among the prenatally exposed offspring.

Because these latter two studies show that even a mild stressor during the prenatal period results in diminished offspring outcomes similar to exogenous increases in prenatal cortisol, it is key to our ability to extrapolate findings based on animal experiments to humans, for whom only non-experimental research is available. In humans, researchers have related exposure to excessive amounts of cortisol in utero with impaired development of the brain and spinal cord (Yu, Lee, Lee and Son, 2004). Researchers have also linked stress and elevated cortisol in late pregnancy with poor mental and motor development of human offspring at three and eight months (Huizink et al, 2003.) But non experimental studies based on humans suffer from problems of endogeneity that experimental studies based on animals do not. In the next section we

[5] They concluded that prenatal programming of the HPA axis was responsible for the outcome based on studies of the areas of the brain affected.
[6] A mild stressor consists of removal from one's home cage to be confined to a smaller cage for ten minutes each day and subjected to three unpredictable noise bursts.

describe the threats to identification in non-experimental settings and our estimation strategy in greater detail.

III. Empirical strategy

In non-experimental settings, estimates of the impact of maternal stress on offspring outcomes likely suffer from endogeneity. This is because women with higher stress (as measured by cortisol) may differ in important, unobserved ways from women with lower stress and these unobserved factors may be responsible for the negative effects on offspring observed. For example, genes affect cortisol levels (Wust et al, 2000). If the genetic composition of women with high cortisol is correlated with other heritable traits (such as IQ) this will bias the estimated impact of cortisol on offspring cognition. To address this problem, we identify the impact of cortisol on offspring outcomes using mother fixed effects. In so doing we control for any fixed unobservable differences between mothers (such as genetics) that might bias the results.

In a fixed effect framework, identification derives from differences in stress during the prenatal period between siblings. To better understand why cortisol levels may differ across two pregnancies of the same mother, we look at corresponding changes in underlying characteristics or conditions of the family for the sample of mothers with large changes in prenatal cortisol between births (results presented in Section V). We find that for "high cortisol" births, mothers are characterized by lower income and a greater likelihood of being single during the prenatal period, but that these differences disappear by early childhood.[7] Thus identifying variation appears to come from temporary shocks to the mother (particularly to her economic status) during the prenatal period that do not

[7] In the data, we do not have another measure of income again until age 7.

persist through childhood and therefore are unlikely to be independently correlated with offspring outcomes. While this evidence is suggestive of a causal interpretation, it is not conclusive. After we present our results we discuss the possible scenarios under which the difference in cortisol across siblings would be endogenous and present evidence that is inconsistent with each scenario.

IV. Data

Description

The data are a subset of the National Collaborative Perinatal Project (NCPP). The NCPP 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 and their children were followed up through age 7.[8]

In this study we focus on a subset of 1103 children born to mothers enrolled in NCPP through either the Providence or Boston sites for whom follow-up information as adults is available. Children were selected for participation in the adult follow-up survey through a multi-stage sampling procedure which involved a core assessment interview and three component students.[9] The sampling design included an emphasis on siblings. We examine potential non-random selection of the adult follow-up sample in the following sub-section.

[8] 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. Limiting the sample in this way reduces considerably the number of women with "unwanted pregnancies." As such, we reduce any potential upward bias that could arise from unwanted pregnancies if they are associated with greater stress and fewer parental investments (Joyce, Kaestner and Korenman, 2000).

[9] 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.

Trained interviewers collected information on adult education, employment and income, disease and other characteristics between 2002 and 2004. In this work we focus on educational attainment as our measure of adult economic status primarily because educational attainment is arguably a better measure of permanent income than a single measure of annual income, but also because the offspring adult income measures in these data are heavily top-coded.[10] Of the 1103 pregnancies with adult follow-up information, 362 are siblings. Of those with multiple births, we exclude the 15 mothers with 3 or 4 births within the period 1960-1964 as their characteristics are significantly different from the rest of the sample.[11] This leaves a sample of 326 for the fixed effect estimation.

Maternal blood/serum collected during the third trimester of pregnancy (between 31 and 36 weeks of pregnancy) was analyzed for cortisol.[12] Values obtained were compared to published studies of fresh samples to assess validity after 40 years of storage (Stroud et al, 2007). The results support the overall validity of these cortisol values. We also find that the same patterns found between cortisol levels and maternal characteristics in studies based on fresh, precise measures of cortisol are present in our sample as well, providing further evidence of their validity.

Characteristics of the Sample

Table 1 presents sample means for the full NCPP Boston/Providence sample (column 2), the cortisol sample (column 3) and the sibling sub-sample (column 4). The

[10] While the educational attainment data can be externally validated (eg: the distribution of years of education is similar to that found in the CPS for a similar sample) the income data cannot.
[11] For example, the mother with 4 births had 17 years of education when the average is 11-12 years in the full sample and the 1960 census.
[12] Blood was assayed for total and free cortisol. In this analysis we focus on free cortisol, consistent with existing medical literature.

cortisol sample appears to be a representative sub-sample of the full Boston Providence NCPP sample (comparing columns 2 and 3). Mothers are similar in terms of education, income, race, age and marital status. As expected, the birth outcomes of the cortisol sample (gestation and weight at birth) are slightly better for the cortisol subsample due to the fact that they were selected based on availability of third trimester maternal secum and offspring survival to adulthood.

If we compare women in the sibling sample with those from the larger cortisol sample we see that the sibling subsample is similar in terms of cortisol levels and offspring outcomes but that the mothers in the sibling sample appear slightly less disadvantaged in terms of income, education, marital status and race compared with the full sample. In fact, the sibling sub-sample is slightly more representative of the population of women with young children as measured in the 1960 decennial census, which is more likely to be white and married, is older, more educated and less poor (column 1).[13] This is not surprising given that the recruitment for subjects in the NCPP was conducted through public clinics. To increase the generalizability of findings based on a non-random sample of mothers we apply weights to the sibling sub-sample so that the sample better reflects the income, race and educational distribution of the population of women with young children as measured by the 1960 census. The weighted means are presented in column 4.[14] The weighted sample more closely resembles the 1960 census sample in terms of socio-economic status. All analyses are based on weighted data and

[13] This comparison is based on women with children less than five years old residing in urban areas of Massachusetts and Rhode Island drawn from the 1960 census. Because of limitations of the census data, we were unable to calculate averages for Providence and Boston only.

[14] Sampling weights equal to one over the probability of being sampled were calculated for 2 racial categories, 3 education categories and 4 income categories for a total of 24 cells.

standard errors are adjusted for non i.i.d error terms within families (ie, clustered on the mother).

Intergenerational Correlations in the Sample

Intergenerational correlations in economic status are strong in this sample. Those born into poverty are nearly five times more likely to be high school drop-outs as adults than those born above the poverty line (Table 2). Similarly, the offspring of mothers (or fathers) who are high school drop-outs are four times more likely to drop out of high school themselves as adults than those with mothers (or fathers) with at least a high school degree.

Variation in Cortisol Across and Within Families

In Figure 1 we present the distribution of cortisol levels for the whole sample and the sibling subsample. They are similar, though there is less variation in the sibling subsample, as expected (μ=22.5 ng/ml and 20.6 ng/ml, respectively; σ= 22.6 and 16.3, respectively).[15] There is considerable correlation in the cortisol measures within families (ρ=0.42) which cannot be completely explained by similarities in observable characteristics. When we attempt to match unrelated children based on observable maternal characteristics (maternal age, race, education, marital status, IQ and family income), the correlation between matched pairs only reaches 0.16, underscoring a considerable correlation between cortisol levels and unobserved maternal characteristics.[16] One can see this in Figure 2 where we plot sibling cortisol levels

[15] There is one outlier in the sibling cortisol sub-sample which we remove in the regression analyses.
[16] We do this for the non-sibling sample.

against one another in the first panel. One can clearly see a strong correlation between sibling cortisol levels as evidenced by tight clustering around the 45 degree line. In the second panel we plot an individual cortisol level against its closest "match" as defined above. When we do, the points are clustered around the 45 degree line, but there is considerably more dispersion.

Despite the strong correlation in sibling cortisol levels, there is still significant within family variation. The within-family coefficient of variation for the sibling sample is 0.37, suggesting considerable variation within siblings to identify effects.

Measurement Error in Cortisol

Cortisol in this sample is measured with error. Cortisol naturally varies over the period of gestation and over the course of the day.[17] In our data, we do have information on the week of gestation the blood was drawn for which we control, but not on the time of day. If the measurement error related to time of day is random (eg there is no relationship between time of day the blood was drawn and unobserved maternal characteristics), then this would introduce classical measurement error and lead to attenuation bias of approximately 0.59 which the inclusion of family fixed effects would exacerbate. [18] In contrast, if the measurement error is non-random and covaries with

[17] With respect to variation over the period of gestation, Harville et al (2007) record average cortisol levels of .280 ug/dL for pregnant women in the 14th week of gestation increasing to .560 for those in the 31st week of gestation.

[18] We calculate $\sigma^2_c = 0.036$ and $\sigma^2_c + \sigma^2_v = 0.0625$ (where the former represents the distribution of cortisol without noise and the latter, the variance with noise) based on the following: the mean and standard deviation of 100 random draws of cortisol levels measured at 11 am are .28 and .19, respectively. If we draw 100 random observations from the pool of all measures (taken between waking an 9 pm) the mean

maternal characteristics (eg, there is a relationship between time of day of blood draw and maternal characteristics), then the inclusion of family fixed effects would reduce any bias from measurement error. We address this below.

V. Results

To explore the role of maternal stress in perpetuating the intergenerational transmission of economic status, we proceed in four stages. First, we document a negative relationship between economic status and stress (cortisol), consistent with existing bio-medical literature. Second, we estimate the impact of prenatal stress on adult educational outcomes using OLS and family fixed effect methods. Third, we explore the mechanisms by which stress affects adult educational attainment by estimating the impact of stress on intermediate outcomes: birth outcomes, cognition and health at age 7. Specifically, we estimate whether prenatal stress negatively affects weight and gestation at birth, verbal IQ and whether the child has a severe chronic condition at age 7. Finally we conduct robustness checks and provide additional evidence to support a causal interpretation of the maternal fixed effect estimates.

SES and Maternal Cortisol Levels

We document a socioeconomic gradient in maternal cortisol levels in this sample, consistent with existing literature (Cohen et al, 2006; Cohen, Doyle and Baum, 20006 ; Steptoe et al, 2003; Kunkz-Ebrecht, et al, 2003). We compare maternal/family characteristics of those with "normal prenatal cortisol" (defined as the bottom 75% of the

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and standard deviation are .30 and .25, respectively. Based on this we calculate $\sigma_c = .19$ and $\sigma_{v+} \sigma_c = .25$ and a reliability ratio of 1.7, which corresponds to an attenuation bias of 0.59.

distribution of cortisol) to those with "high prenatal cortisol" (defined as the top quartile of the distribution) in Table 3A.[19] In the cross-sectional comparisons, we find that mothers with "high prenatal cortisol" levels are characterized by higher likelihood of being single and lower income, at both pregnancy and 7 years post pregnancy. For example, women with high cortisol are characterized by $5863 less income (mean of $40,000) at pregnancy than "normal prenatal cortisol" mothers and this difference persists: these same women are characterized by $6121 less income 7 years post-natal. Mothers with elevated cortisol also have fewer years of schooling. All differences are significant except birth order and sibling number.

Not only are low SES mothers characterized by higher average cortisol levels, but low SES mothers (defined as those with less than a high school degree) are also characterized by greater variability in their cortisol levels across births (between siblings). Thirty one percent of high school drop-outs are characterized by at least a standard deviation difference in cortisol levels across births (σ=14 ug/ml), compared with 21 percent of mothers who are high school graduates. In Table 3B we explore how family characteristics vary across births in a sample of families with large changes in cortisol levels between siblings (n=80).[20]

For these within family comparison, when we compare "high prenatal cortisol" children with their "normal prenatal cortisol" siblings, the difference in real income at pregnancy of $8667 is large and significant, but the difference in income at age 7 is

[19] This method of defining "high" and "normal" levels of cortisol is generally consistent with the existing bio-medical literature. No absolute threshold by which to define "high" cortisol exists. This is especially true for cortisol measured during pregnancy since cortisol levels increase with gestation. Rather, research typically relies on relative levels (such as top quartile) for an average population (that is, one that is not known to have been exposed to unusual stressors) to define "high" values.

[20] For the sibling sample, 34 siblings are born to mothers who always have high cortisol, 202 are born to mothers who always have normal cortisol and 80 are born to mothers whose cortisol levels differ between siblings (eg – high for one child but normal for the other).

minimal and insignificant, only $608 (Table 3B). Note that the difference may have diminished prior to age 7, but the only post-natal income measures that we have in these data were collected at age 7. Likewise, for marital status, there is a large and significant difference in the probability of being single while pregnant but not so for the probability of being single when the child is age 7 which is small and insignificant (difference .09 versus .01, respectively.)[21] That a change in marital status is highly correlated with cortisol levels is consistent with previous work based on medical students showing that for a given medical student, getting married is associated with a reduction in cortisol levels, presumably through an increase in social support (Coombs and Fawzy, 1982). Unfortunately, there is no corresponding literature looking at changes over time in income and corresponding cortisol levels with which to compare our income results, as the existing literature looks only at the cross section.

These results suggest that within family differences in cortisol levels, our source of identifying variation, are correlated with short-term negative shocks during pregnancy that do not persist for long. This contrasts with cross-sectional differences in cortisol levels which are correlated with worse long term underlying conditions, as evidenced by fewer resources during the prenatal period that persists at least 7 years into the post natal period.

Relationship Between Maternal Prenatal Cortisol and Offspring Outcomes: Preliminary Evidence

[21] In Table 3, the probability of being single increases considerably between birth and age 7. This is consistent with data from the 1960 and 1970 census which shows that the probability of being single more than doubles over this period, increasing from .032 to 0.085.

Before turning to regression analysis, we provide simple non-parametric comparisons of outcomes of offspring of the same mother in Table 4. Specifically, we compare years of schooling, gestation and weight at birth, and IQ and health at age 7, of siblings exposed to different levels of prenatal cortisol. Comparing siblings, we find that those exposed to higher prenatal cortisol levels (relative to their siblings) are characterized by lower adult educational attainment and cognition and worse health at age 7, and the differences are non-negligible (columns 1-3). For example, those exposed to high cortisol have, on average, .3 years fewer education, a verbal IQ 4.3 points lower at age 7 and are 2 percentage points more likely to have a severe chronic health condition at age 7. In contrast, the relationship between prenatal cortisol and birth outcomes, though negative, is very small. This is likely due to the fact that the sample, because it was selected based on the availability of third trimester measures of cortisol and child survival to adulthood, excludes those with very poor birth outcomes. This may suggest that our results represent a lower bound if one way that in-utero exposure to cortisol affects offspring outcomes is through increased prematurity and lower birth weight, as some recent evidence suggests (see IOM, 2006).

However, because existing medical research suggests that negative effects of cortisol on mental and motor development occur when cortisol levels either exceed (or, in some rare cases, fall short of) normal circulating levels, we also present differences in offspring outcomes when one sibling is exposed to extremely high levels of prenatal cortisol (defined as in the top quartile). The results are presented in columns 4-6 of Table 4. The differences are large: siblings exposed to very high cortisol levels are

characterized by three quarters of a year of less schooling, a verbal IQ that is 4.6 points lower and nearly double the probability of a severe chronic condition at age 7.

We follow these non-parametric, unconditional comparisons with regression analyses that include multiple controls for changing prenatal and post natal circumstances in the next section.

Impact of Maternal Cortisol on Offspring Educational Attainment

In this sub-section we provide empirical evidence based on OLS and fixed effect regressions that maternal prenatal cortisol negatively affects offspring human capital accumulation as measured by years of education. In column (1) of Table 5 are OLS estimates of the impact of prenatal cortisol levels on adult years of education based on the full cortisol sample. We include as controls: maternal race (indicator for black), maternal education, marital status at birth, maternal age, maternal IQ, family income during pregnancy as a percent of the federal poverty level, child gender, number of siblings at age 7, birth order, whether the husband lives at home with the mother, the number of times the family moved between birth and age 7 (a measure of instability) and gestation at blood draw. We also include an indicator for whether there was any pregnancy complication (as noted by the attending OB) to control for the possibility that the increase in maternal cortisol observed simply reflects maternal anxiety over the health of the fetus.

We find that cortisol has a small negative and insignificant effect on adult years of education: a one standard deviation increase in prenatal cortisol levels is associated with only 0.047 fewer years of schooling. The OLS estimate based on the sibling subsample

is larger, but also insignificantly different from zero and the two estimates are statistically equivalent (column 2).

However, when we include mother fixed effects, the impact increases and is statistically significant: a one standard deviation in cortisol results in .36 years fewer schooling, or 26 percent of a standard deviation (column 3). While this effect may seem large relative to those of most educational interventions (Hanushek, 2006), it is consistent with existing evidence on the impact of prenatal conditions on offspring educational outcomes. For example, Almond (2006) estimates that in-utero exposure to the flu results in .25 fewer years of schooling – a roughly similar effect.[22] In column 4 we include a full range of time-varying controls and the results are the same, suggesting that omitted variables do not bias the FE estimates. This supports the idea that temporary reductions in resources during the pregnancy that may be correlated with temporary increases in cortisol do not independently affect long term offspring outcomes. Finally, in the last column (column 5) we also include an indicator for whether the mother worked while pregnant and the estimated impact of prenatal cortisol on offspring education is unchanged. This last finding provides evidence that maternal work does not negatively affect offspring outcomes by increasing maternal stress. In these data maternal work during pregnancy is not correlated with stress levels in either OLS or FE settings.

In the second panel of Table 5 we explore potential non-linearities in the effect by creating three dummies: bottom quartile of distribution, 25-75 percent of the distribution and the top quartile of the distribution of cortisol (with middle of the distribution,

[22] In related work, though not directly comparable due to their focus on a different outcome, Almond, Edlund and Palme (2008) find that in utero exposure to low levels of radiation reduce school test scores by six percentile points – a relatively large effect relative to most educational interventions. Bleakley (2007) also finds large effects on schooling (the probability of enrollment) associated with improvements in child health.

representing the normal range, omitted). The OLS results presented in columns 1 and 2 suggest that the effect is non-linear: in the full sample, being exposed to high prenatal cortisol results in .4 less of a year of schooling (column1). For the sibling subsample, column 2, the OLS result is similar though slightly smaller and less precise. If we look at the sibling fixed effect results (columns 3 and 4), we find that being in the top quartile of the distribution (compared with the middle of the distribution) results in three quarters of a year less schooling and the estimate is statistically significant. This represents 51 percent of a standard deviation in these data. Again, including an indicator for maternal work while pregnant (column 5) does not change the results.

Interpretation of Findings

There are three potential interpretations of our finding that the estimated impact of prenatal cortisol on adult educational attainment increases when we include family fixed effects. The first is that the OLS regressions that include a full set of controls for family resources yield estimates that are biased downwards. This would be consistent with the idea that elevated cortisol in the cross section is caused by persistent poverty and including both cortisol and multiple measures of family resources in the regression essentially amounts to "over-controlling." Indeed, when one excludes measures of family resources in an OLS regression of child outcomes on prenatal cortisol, the estimates are much larger.

A second interpretation or explanation is that the larger FE estimates might reflect a potential misspecification of the OLS regression framework. An alternative model is one in which an individual adjusts to a baseline or personal level of cortisol that may be a

function of underlying average levels of stress, genetics or some combination of factors. It is only when mothers are exposed to a new or unexpected stressor that moves them from their established equilibrium that adverse effects are found. There is some support for this model in the psychological and biological literatures. Mineka and Kihlstrom (1978) review a large body of scientific experiments, performed mostly on animals, which find that unpredictable aversive events are more stressful and have a greater negative impact on observed behavior and health than predictable aversive events which occur with either the same or greater frequency. For example, Rosenblum and Paully (1984) subject monkeys to three environments: low foraging demand (abundance of food), high foraging demand (paucity or scarcity of food) and variable foraging demand (unpredictable periods of abundance and scarcity). They find the most aversive effects on mother-infant pairs subjected to the variable foraging environments.

If this is the true model, a fixed effect strategy would be the correct specification. By including maternal fixed effects we implicitly identify the impact of deviations from mean (or personal) cortisol levels on outcomes. Given existing evidence that the range of baseline cortisol varies considerably within a non-stressed pregnant population, this alternative model seems plausible.

A final interpretation relates to measurement error in the measure of cortisol used. As noted previously, cortisol naturally varies over the course of the day and it is not known when these measures were drawn. If the time of day that the blood was drawn was not random but correlated with unobservable characteristics of the mother that might affect offspring outcomes, assuming that those unobservable characteristics remained constant across pregnancies, the inclusion of maternal fixed effects would reduce the

impact of such non-random measurement error. To explain a downward bias of OLS estimates, it would have to be the case that unobservable characteristics of mothers were correlated with both an earlier draw time and better offspring outcomes. However, if the timing of the blood draw were non-random, we would expect that maternal work would influence it. But when we control for maternal work, the results do not change. Thus, time of day of the blood draw does not appear to be systematically correlated with maternal work and if it is not correlated with work, we believe it is even less likely that it is correlated with other maternal characteristics, thus we view this interpretation as the least plausible of the three.

In the following subsections we explore the mechanisms by which maternal prenatal cortisol affect offspring educational attainment. Specifically, we focus on birth outcomes, child cognition and health as three potential intermediate outcomes affected by prenatal cortisol. We focus on these three intermediate outcomes because they have been shown in the medical literature to be adversely affected by elevated stress and cortisol in-utero and in the economics literature, to directly affect educational attainment.

Prenatal Cortisol and Birth Outcomes

There is evidence that stress in utero reduces birth weight and increases the probability of prematurity (Eskenazi, et al, 2007; Camacho, 2008). To explore this we estimate the impact of prenatal cortisol on two birth outcomes, birth weight and gestation, and present the results in Table 6. We find that in the linear specification (panel A) prenatal cortisol has no impact on birth weight and gestation in OLS models with and

without a full set of controls (columns 1 through 6). Even in the fixed effect models, there appears to be no significant, or even sizeable, effect of cortisol (in a linear specification) on either gestation or birthweight (columns 7 and 8).

However, in Panel B when we estimate the impact of very high cortisol (as measured by a value of cortisol in the top quartile of the distribution), there is some weak evidence of a negative effect on birth outcomes. The effects are statistically insignificant in all cases but one for the OLS estimates. For the one significant effect (column 5 for birthweight) the estimates imply that prenatal cortisol in the top quartile of the distribution is associated with a decline in birthweight of 171 grams which represents 36 percent of a standard deviation. When we include maternal fixed effects, however, the impact is no longer significant. In general, the estimated impact of cortisol on birth outcomes is small and insignificant in the sibling FE regressions which, as we noted previously, is likely attributable to the fact that the sample was selected based such that mothers with the worst birth outcomes are excluded from the sample. As such, the effects we estimate likely reflect a lower bound.

Prenatal Cortisol and Child IQ

Next we estimate the impact of prenatal maternal cortisol on child cognition measured at age 7.[23] Specifically we estimate the impact of cortisol on verbal IQ because of evidence that those with Cushings disease (a hormonal disorder caused by prolonged exposure of the body's tissues to high levels of the hormone cortisol) are characterized by

[23] LeWinn, et al (2008) use a subset of the NCPP sibling sample to produce maternal FE estimates of the impact of an index of free cortisol on child verbal IQ at age7 and the results are very similar.

deficits in verbal learning and other verbal functions but not non-verbal functions (Starkman et al 2001).[24]

The results suggest that cortisol has no significant effect on verbal IQ in a linear specification (Table 7). The results are small and always insignificant (Panel A). In contrast, very high cortisol (Panel B) appears to have a large negative and borderline significant (at the 10 percent level only) impact on verbal IQ in the fixed effect regressions. Specifically, exposure to cortisol in the top quartile of the distribution is associated with a 7 point lower verbal IQ (47 percent of a standard deviation). Interestingly, the effect is larger and significant only in the fixed effect model with full controls (compare columns 3 and 4 of Table 7), suggesting that in a fixed effect framework, if anything, omitted variables bias down any estimated impact.

Given the high degree of correlation between child IQ and years of education in these data (ρ=.45 for verbal IQ and ρ=.34 for performance IQ at age 7), we conclude that the adverse effect of exposure to elevated cortisol in-utero on adult years of education is likely mediated in part by the negative impact on verbal cognitive functioning. In the robustness section, we also provide estimates of the impact of cortisol on non-verbal IQ as a falsification exercise.

Prenatal Cortisol and Child Health at Age 7

We estimate the impact of exposure to elevated stress in-utero on child health as measured by the presence of any severe chronic condition at age 7 as determined by medical examination of the child (Table 8). The results differ somewhat from those for child IQ. The results with respect to the impact of a linear increase in cortisol on the

[24] The verbal IQ score is derived from the Wechsler intelligence tests.

probability of a severe chronic condition are positive and significant. In the fixed effect regressions, a standard deviation increase in cortisol is correlated with a 9.5 percent increase in the probability of a severe chronic condition (Table 8, panel A column 4). Moreover, including gestation and birthweight in the regression (column 5) does not alter the results. In contrast, we find that very high cortisol (in the top quartile of the distribution) increases the probability of a severe chronic condition by 3.8 percent, but the estimate is very imprecise. We take this as suggestive evidence that exposure to cortisol in-utero has a negative effect on offspring health that may not necessarily be evident at birth and that this may represent a second mechanism through which exposure to prenatal cortisol negatively affects completed years of schooling among offspring, though we consider the evidence slightly weaker than that for IQ.

Robustness

We perform a number of robustness checks and present the results in Table 9. Previous work has found season of birth to be correlated with offspring outcomes in humans and scientists have found seasonal patterns in cortisol levels in both animals and adult males (Laurence et al, 2008; Walker et al,1997).[25] To rule out the possibility that seasonal variation is driving our results with respect to adult education, child IQ and health, we control for season of birth in the regressions presented in Panel A of Table 9. The inclusion of three indicators for season of birth does not change the point estimates but does reduce precision.

In the second panel we explore potential gender differences in the effects of exposure to elevated cortisol in utero (Panel B, Table 9). There is no appreciable

[25] We found only small (3 ng/ml) and insignificant differences in free cortisol across seasons.

difference with respect to education or verbal IQ; there is some evidence of a reduced effect of cortisol on health for males, but it the coefficient on the interaction term is very imprecisely estimated.

We also rule out the possibility that our estimates of the impact of cortisol on offspring outcomes are driven by other circulating hormones that are often correlated with cortisol levels. Sex hormones, and testosterone in particular, are correlated with cortisol levels and have been hypothesized to affect offspring outcomes (Romano, Leoni and Saino, 2006). To identify the impact of cortisol separate from testosterone, we control for testosterone levels in the regressions presented in Panel C of Table 9. When we control for testosterone levels, the estimated impact of cortisol on education, IQ and health at age 7 is unchanged.

In Panel D of Table 9 we include controls for marital status and income at age 7 (in additional to marital status and income during pregnancy) and there is no change in the estimated effects of prenatal cortisol on educational attainment, child IQ or health.

Finally, in the last panel of Table 9 we present estimates of the impact of prenatal cortisol levels on performance IQ as a falsification exercise. Given that the existing literature based on animals points to deterioration of specific areas of the hippocampus, not general deterioration, and the literature on Cushing's disease in humans points to deficits in verbal learning only (Starkman et al, 2001), we expect an impact on verbal learning only in these data. Consistent with this, we find no significant impact of prenatal cortisol on performance IQ in either cross-sectional of fixed effect estimation (Panel E Table 9).

In the following section we discuss potential threats to identification in a fixed effect framework and present evidence inconsistent with each.

Potential Threats to Identification

We have thus far presented evidence that elevated maternal stress during the prenatal period exerts a negative and lasting effect on offspring outcomes including educational attainment, child IQ and health. To control for omitted variables correlated with maternal stress and offspring outcomes that might bias our results, we include maternal fixed effects and identify the effects off of changes in the level of maternal stress across siblings. However, it could still be the case that omitted variables that differ across two pregnancies of the same mother could bias the results. In this section we discuss three potential sources of omitted variable bias in this context.

The first source of omitted variable bias is that difficult or complicated pregnancies may lead to both higher maternal stress and worse offspring outcomes, biasing upwards our estimate of the impact of stress on offspring outcomes. However, in our both our OLS and FE estimation, we included controls for the presence of a pregnancy complication, which is very well measured in these data, and the estimated impact of maternal cortisol on offspring outcomes remained. In some regressions we also controlled for gestation at birth and birthweight (the two most common measures of the health of the fetus at birth) and again the effects were unchanged.

Second, elevated maternal cortisol during the prenatal period may mark the beginning of a decline in resources and reduction in the quality of the child's environment that extends to the post natal period. To counter this we show that in the

fixed effect setting, an increase in prenatal cortisol is associated with a transitory reduction in resources during the prenatal period that disappears by early childhood. Resources at age 7 seem to be totally uncorrelated with prenatal cortisol in the fixed effect setting.

Finally, there is the possibility that the stressor itself (and not the stress) is responsible for the negative effects on offspring outcomes that we estimate. We provide two pieces of evidence that are inconsistent with this. First, if this were the case, we would expect that as we include observed time-varying characteristics (eg, income, marital status) that are correlated with cortisol and offspring outcomes, the estimated impact of cortisol on outcomes would decline. It does not. If the inclusion of important observed characteristics does not change the estimated effects, it seems unlikely that unobserved characteristics would.

As a second piece of evidence, we argue that if the stressor were responsible for the effects we estimate, it would likely operate through a diminished childhood environment which is largely a function of parental investments in their children. To test this, we estimate whether a negative relationship exists between prenatal stress levels and parental investments in the child during the pre and post-natal periods. If we do not observe any relationship, then together, these two empirical observations provide additional evidence that we are identifying the impact of stress, not the underlying stressor, on offspring outcomes. Below we present our empirical results regarding the relationship between prenatal cortisol and multiple measures of parental investments.

Prenatal Stress and the Parental Investments

We examine whether prenatal maternal cortisol is correlated with either pre or post natal investments in the child, including: 1) the number of prenatal visits, 2) a measure of prenatal nutrition (the amount of iron in the blood 48 hours after delivery), 3) the mother's "responsiveness" to focus on her child, as assessed by a psychologist in the eighth month post-birth, 4) whether as part of the psychological examination, the interviewer concludes that the child faces an "unfavorable emotional environment" at one year of age and 5) whether the child was sent to preschool.

We present OLS and FE regression estimates for the five outcomes in a linear specification (Table 10 panel A) and for the top quartile of cortisol (Table 10 panel B). There is no significant relationship between prenatal cortisol levels and any of the five measures of parental investment in either the OLS of fixed effect regressions. Moreover, the point estimates are very small in magnitude and inconsistent in sign: there is a negative relationship between nursery school and number of prenatal visits and cortisol levels, but a positive relationship between maternal focus and the emotional environment of the child and prenatal cortisol levels. We conclude that the relationship between in-utero exposure to elevated cortisol and offspring outcomes does not reflect a lack of parental investment in the offspring either caused by or correlated with the stressor.

VI. Conclusions

In this paper we explore the role of maternal stress in affecting offspring outcomes. Specifically, we show that exposure to elevated levels of the stress hormone cortisol in-utero negatively affects offspring educational attainment. While the effects may seem large compared with many educational interventions (Hanushek, 2006), they

are comparable to the considerably larger estimates of the impact of prenatal conditions (Almond, 2006 and Almond, Edlund and Palme, 2008) on offspring educational outcomes. We also provide evidence as to the mechanisms by which prenatal stress affects educational attainment. We find that prenatal stress negatively affects both the probability of a severe chronic health condition and verbal IQ at age 7, suggesting that health and cognitive functioning are two of the mechanisms by which prenatal stress negatively affects offspring educational attainment. The results are generally non linear – the negative effects are concentrated among those with the highest cortisol levels. Cortisol levels in the normal range appear to have less of an effect on long term outcomes. These results are consistent with the emerging neuro-biology research showing that exogenous in-utero exposure to stress or cortisol impairs the developing brain of the fetus.

Our results have a number of important implications. First, consistent with recent literature suggesting an important role for prenatal conditions in determining offspring outcomes, our results imply that equality of prenatal conditions may be responsible for the often observed stronger correlations in IQ and earnings among fraternal twins relative to non-twin sibling pairs.[26] Our results also have important implications for our understanding of the causes of intergenerational income inequality. Our findings that stress hormones increase with reductions in income (in the cross section and over time for the same woman) and that maternal stress reduces offspring educational attainment (and

[26] Scarr (1997) reports correlations for MZ twins of .76-.86, DZ twins of .35-.55 and non-twin siblings of .24-.47.

hence earnings) can potentially explain the relatively low rates of intergenerational economic mobility observed for poor families in the US.

Our results suggest that poverty reduction programs can potentially have large positive intergenerational effects via reductions in stress, especially if geared toward pregnant mothers, as in the WIC program. But anti-poverty programs are costly and difficult to implement. Alternatively, policy-makers might consider programs aimed at providing women with the skills to accommodate stress more successfully. Not only have such programs proven effective at reducing perceived stress and cortisol in pregnant women, but they are easy to implement and relatively cheaper.[27]

[27] For example, Urizar et al (2004) find that a low-cost stress reduction program for low income pregnant women successfully reduced stress and morning cortisol levels.

References

Almond, Douglas (2006) “Is the 1918 Influenza Pandemic Over? Long-term Effects of In Utero Influenza Exposure in the Post-1940 U.S. Population” *Journal of Political Economy*, 114: 672-712.

Almond, Douglas, Edlund, Lena and Marten Palme (2008) “Chernobyl’s Subclinical Legacy: Prenatal Exposure to Radioactive Fallout and School Outcomes in Sweden.” NBER Working Paper 13347.

Almond, Douglas, Chay, Kenneth and David Lee (2005) “The Costs of Low Birth Weight” *Quarterly Journal of Economics*. 120(3).

Almond, Douglas, Edlund, Lena, Li, HOngbin and Jansen Zhang (2007) “Long-Term Effects of the 1959-1961 China Famine: Mainland China and Hong Kong” NBER Working Paper 13384

Behrman, Jere and Mark Rosenzweig (2004) "Returns to Birthweight," *The Review of Economics and Statistics*, Vol. 86, No. 2: 586-601.

Berenbaum, S.A. & Resnick, S.M. (1997) “Early androgen effects on aggression in children and adults with congenital adrenal hyperplasia” *Psychoneuroendocrinology*, 22, 505–515.

Black, Sandra, Devereux, Paul and Kjell Salvanes (2007) “From the Cradle to the Labor Market? The Effect of Birth Weight on Adult Outcomes” *Quarterly Journal of Economics*,

Bleakley, Hoyt (2007). “Disease and Development: Evidence from Hookworm Eradication in the American South." *Quarterly Journal of Economics*, February 2007, 122(1).

Buka, Stephen, Shenassa Edmund and Raymond Niaura (2003) “Elevated risk of tobacco dependence among offspring of mothers who smoked during pregnancy: a 30-year prospective study.” *American Journal of Psychiatry*. Vol. 160 No. 11:1978-84.

Camacho, Adriana (2008). "Stress and Birth Weight: Evidence from Terrorist Attacks", *American Economic Review-papers and procedings* 98(2), pp. 511-15.

Cohen, Sheldon, Schwartz, Joseph, Epel, Elissa, Kirschbaum, Clemens, Sidney, Steve and Teresa Seeman (2006) “Socioeconomic Status, Race and Diurnal Cortisol Decline in the Coronary Artery Risk Development in Young Adults (CARDIA) Study” *Psychosomatic Medicine* Vol 68: 41-50.

Coombs, RH and I Fawzy (1982). “The Effect of Marital Status on Stress in Medical School.” American Journal of Psychiatry. 139: 1490-1493.

Corak, Miles (2004). “Do Poor Children Become Poor Adults? Lessons for Public Policy from a Cross-Country Comparison of Earnings Mobility.” UNICEF Innocenti Research Center, Florence, Italy.

Cunha, Flavio and James Heckman (2007) “The Technology of Skill Formation” *American Economic Review* 97 (2): 31-47.

Currie, Janet (2001). “ Early Childhood Education Programs.” *Journal of Economic Perspectives 15*(2): 213–238.

Currie, Janet and Enrico Moretti (2007) "Biology as Destiny? Short and Long-Run Determinants of Intergenerational Transmission of Birth Weight" *Journal of Labor Economics* vol 25 no 2.

Currie, Janet, and Mark Stabile (2006). “Child Mental Health and Human Capital Accumulation: the Case of ADHD,” *Journal of Health Economics*, 25(6), pp.1094-1118.

DeSantis AS, Adam EK, Doane LD, Mineka S, Zinbarg RE, Craske MG.(2007) “Racial/ethnic differences in cortisol diurnal rhythms in a community sample of adolescents.” *J Adolescent Health.* 2007 Jul;41(1):3-13

Dohrenwend, Barbara S. (1973) “Social status and stressful life events” *Journal of Personality and Social Psychology*. Vol 28(2): 225-235.

Eskenazi, B (2007) “Stress Caused By September 11 Disaster Linked To Low Birth Weights.” *European Society for Human Reproduction and Embryology* (2007, October 11).

Gutteling, Barbara, deWeerth, Carolina, and Jan Buitelaar (2005) “Prenatal Stress and Children’s Cortisol Reaction to the First Day of School” *Psychoneuroendocrinology*. Vol. 30: 541-549.

Hanushek, Eric (2006). “School Resources” in The Handbook of the Economics of Education. Eric Hanushek and Finish Welch eds. Elseveier.

Heckman, James (1995) “Lessons from the Bell Curve” *Journal of Political Economy* 103(5): 1091-1120.

Heckman, James and Dimirty Masterov (2007) “The Productivity Argument for Investing in Young Children” NBER Working Paper # 13106.

Huizink, Anja, Robles de Medina, Pascale, Mulder, Eduard, Visser, Gerard and Jan Buitelaar (2003) “Stress During Pregnancy is Associated with Developmental Outcome in Infancy” *Journal of Child Psychology and Psychiatry* Vol 44 No. 6: 810-818.

Institute of Medicine Committee on Understanding Premature Birth and Assuring Healthy Outcomes, Board on Health Sciences Policy, Behrman, R.E., and Butler, A.S. (eds.) (2006). Preterm Birth: Causes, Consequences, and Prevention. Washington, DC, The National Academies Press.

Joyce, Theodore, Kaestner, Robert and Sanders Korenman (2000). "The Effect of Pregnancy Intention on Child Development." *Demography* 37(1): 83-94.

Kunz-Ebrecht, Sabine, Kirschbaum, Clemens and Andrew Steptoe (2004) "Work Stress, Socioeconomic Status and Neuroendocrine Activation Over the Working Day" *Social Science and Medicine* Vol 58: 1523-1530.

Laurence R. Gesquiere, Memuna Khan, Lili Shek, Tim L. Wango, Emmanuel O. Wango, Susan C. Alberts, Jeanne Altmann, "Coping with a challenging environment: Effects of seasonal variability and reproductive status on glucocorticoid concentrations of female baboons (Papio cynocephalus)" *Hormones and Behavior*, Volume 54, Issue 3, August 2008, Pages 410-416.

LeWinn, Kaja, Stround. Laura, Molnar, Beth, Ware, James, Koenen, and Stephen Buka "Elevated Maternal Cortisol Levels During Pregnancy are Associated with Reduced Childhood IQ" Mimeo, University of California, SF, October 2008.

Marmot, Michael (2004) Status Syndrome: How Your Social Standing Directly Affects Your Health and Life Expectancy. London: Bloomsbury.

Mazunder, Bhashkar (2005) "The apple falls even closer to the tree than we thought: new and revised estimates of the intergenerational inheritance of earnings" in Unequal Chances: family background and economic success. Russell Sage (Eds Samuel Bowles, herbert Gintis and Melissa Osboure Groves)

McEwen, Bruce (1998) "Protective and Damaging Effects of Stress Mediators" *New England Journal of Medicine* vol 338: 171-179.

Mineka, Susan and John Kihlstrom (1978) "Unpredictable and Uncontrollable Events: A New Perspective on Experimental Neurosis" *Journal of Abnormal Psychology*. 87(2): 256-271.

O'Connor, Thomas, Ben-Schlomo, Yoav, Heron, Jon, Golding, Jean, Adams, Diana and Vivette Glover (2005) "Prenatal Anxiety Predicts Individual Differences in Cortisol in Pre-Adolescent Children" Biological Psychology Vol 58: 211-217.

Qian, Nancy and Xin Meng (2009) "The Long Run Impact of Famine on Survivors: Evidence From China's Great Famine" unpublished manuscript.

Rablen, Matthew and Andrew Oswald (forthcoming) “Mortality and Immortality : The Novel Prize as an Experiment into the Effect of Status on Longevity” *Journal of Health Economics*

Romano, Maria, Leoni, Barbara nd Nicol Saino (2006) “Examination Marks of Male University Students Positively Correlate with Finger Length Ratios” *Biological Psychology* 71: 175-182.

Rosenblum, Leonard and Gayle Paully (1984) “The Effects of Varying Environmental Demans on Magternal and Infant Behavior” *Child Development*. 55: 305-314.

Royer, Heather (2006) “Separated at Girth: US Twin Etimates of the Effects of Birthweight” mimeo Case Western Reserve University

Scarr, Sandra (1997) “Behavior-Genetic and Socialization Theories of Intelligence” in Intelligence, Heredity, and Environment Robert Sternberg and Elena Grigorenko, eds. Cambridge University Press.

Sapolsky, Robert, Uno, Hideo, Rebert, Charles and Caleb Finch (1990) “Hippocampal Damage Associated with Prolonged Glucocorticoid Exposure in Primates” *The Journal of Neuroscience* Vol 10, No. 9: 2897-2902

Schneider, M. L., Coe, C. L., & Lubach, G. R. (1992). Endocrine activation mimics the adverse effects of prenatal stress on the neuromotor development of the infant primate. *Developmental Psychobiology, 25,* 427-439.

Sekl, Jonathan (1998) “Physiologic Programming of the Fetus” *Clinics in Perinatalogy* Vol 25: 939-964.

Solon, Gary (1999) “Intergenerational Mobility in the Labor Market” Handbook of Labor Economics, Vol 3, Eds Orley Ashenfelter and David Card.

Starkman, Monica, Giordani, Bruno, Berent, Stanley, Schork, Anthony and David Schteingart (2001) “Elevated Cortisol Levels in Cushing’s Disease Are Associated With Cognitive Decrements” *Psychosomatic Medicine* 63:985–993.

Steptoe, Andrew, Kunz-Ebrecht, Sabine, Owen, Natalie, Feldman, Pamela, Willemsen, Gonneke, Kirschbaum, Clemens, and Michael Marmot (2003) “Socioeconomic Status and Stress-Related Biological Responses Over the Working Day” *Psychosomatic Medicine* vol 65: 461-470.

Steptoe, Andrew and Michael Marmot (2002) “The Role of Psychobiological Pathways in Socio-Economic Inequalities in Cardiovascular Disease” *European Heart Journal* Vol 23: 13-25.

Stroud, Laura, Solomon, Catherine, Shenassa, Edmund, Papandonatos, George, Niaura, Raymond, Lipsett, Lewis, LeWinn, Kaja and Stephen Buka (2007) "Long-term stability of maternal prenatal steroid hormones from the National Collaborative Perinatal Project: still valid after all these years." *Psychoneuroendocrinology* Vol. 32 No. 2:140-50.

Uno, H, Thieme C, Kemnitz J, Farrell P (1983) "Effect of Dexamethasone on the Cerebral Coritsol Development of the Rhesus Monkey." Soc Neuroscience. Abstract 320:3.

Uno, H, Lohmiller, L, Thieme C, Kemnitz J, Engel, M, Roecker E and P Ferrell (1990) "Brain Damage Induced by Prenatal Exposure to Dexamethasone in Fetal Rhesus Macaques" Developmental Brain Research.

Urizar, Guido, Milaazo, Melina, Le, Huynh-Nhu, Delucchi, Kevin, Sotelo, Rosario, and RIchardo Munoz (2004). "Impact of Stress Reduction Instructions on Stress and Cortisol Levels During Pregnancy" *Biological Psychology* 67:275-282.

Yu, In Tag, Lee, Sang-Hun, Lee Yong-Sung, and Hyeon Son (2004) "Differential effects of corticosterone and dexamethasone on hippocampal neurogenesis in vitro." *Biochemical and biophysical research communications*. Vol 317 No. 2:484-90.

Van Eck, Marleen, Berkhof, Hans, Nicolson, Nancy and Jose Sulon (1996) "The Effect of Perceived Stress, Traits, Mood States and Stressful Daily Events on Salivary Cortisol" *Psychosomatic Medicine* Vol 85. No. 5: 447-458.

Walker, Brian, Best, Ruth, Noon, Joseph, Watt, Graham C. M. and David J. Webb (1997) "Seasonal Variation in Glucocorticoid Activity in Healthy Men" *The Journal of Clinical Endocrinology & Metabolism* Vol. 82, No. 12 4015-4019

Welberg, LAM, Sekl, Jonathan and Meghan Homes (2001) "Prenatal Glucocorticoid Programming of Brian Corticosteroid Receptors and Corticotrophin-Releasing Hormone: Possible Implications for Behaviour" *Neuroscience* Vol 104: 71-79.

Wust, Stehan, Federenko, Ilona, Hellhammer, Dirk and C Kirschbaum (2000) "Genetic Factors, Perceived Chronic Stress and the Free Cortisol Response to Awakening" *Psychoneuroendocrinology* Vol 25: 707-720.

Fig 1 : Distribution of Prenatal Cortisol

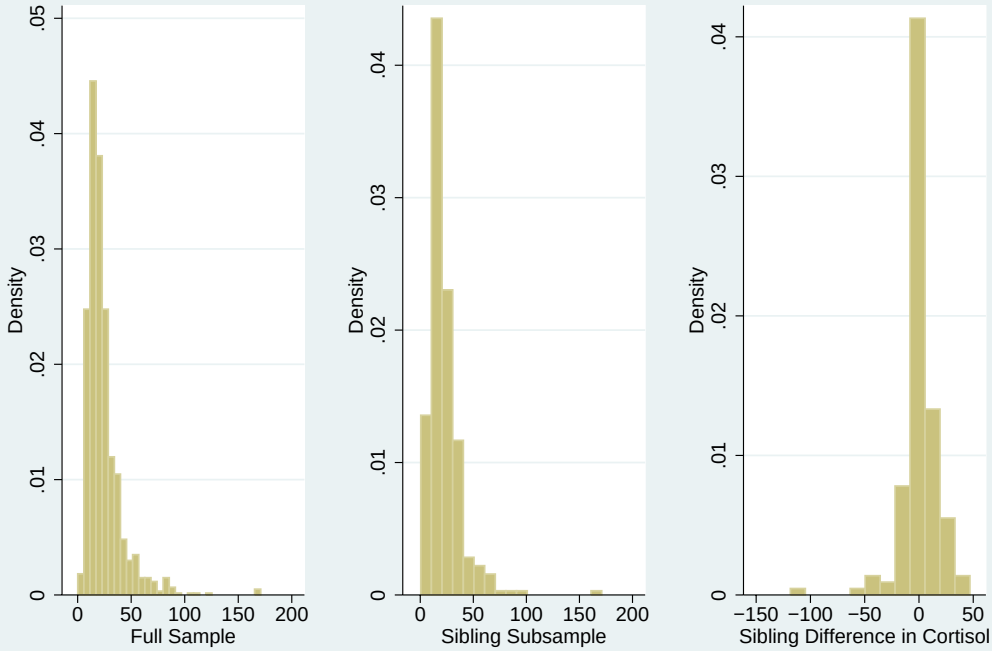

Fig 2 : Within Family Correlations in Cortisol

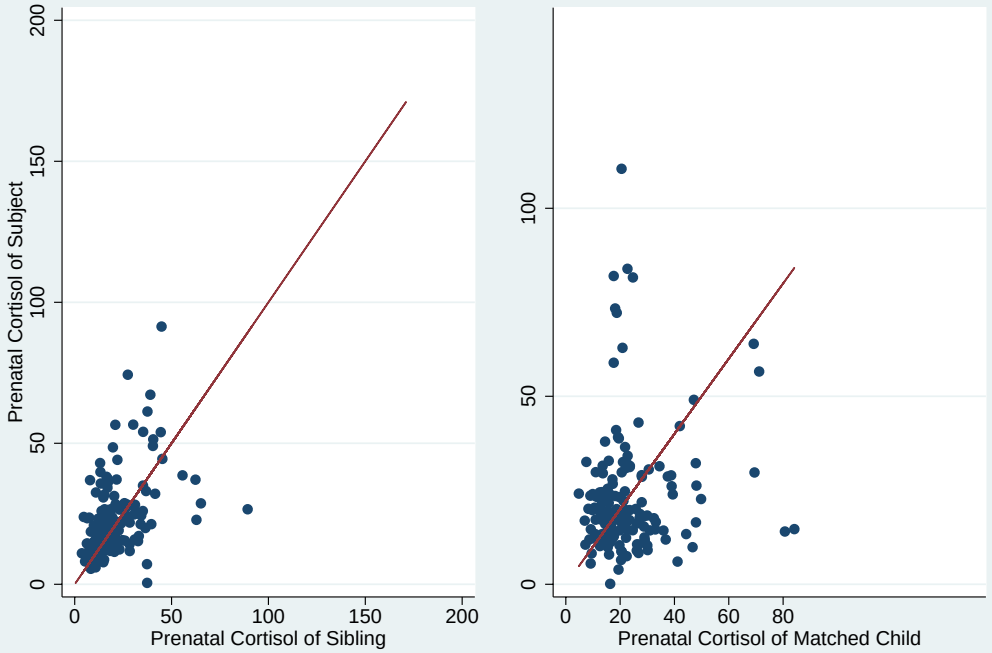

Table 1: Sample Characteristics

	1960 Census	Providence & Boston	Cortisol Sample	Sibling Sample	
	Unweighted	Unweighted	Unweighted	Unweighted	Weighted
Maternal Education	11.5	11.4	11.07	11.4	11.6
Family Income	$45,185	$30,956	$29,040	$31,217	$41,784
Mother Black	0.03	0.12	0.13	0.08	0.03
Maternal Age	30.6	25.2	24.9	24.6	25.4
Mother Single	0.03	0.11	0.09	0.056	0.024
Birthweight (grams)		3185	3307	3290	3303
Gestation (weeks)		36.8	40.1	39.9	40
Free Cortisol (ng/ml)			24	22	20.5
Verbal IQ 7 year		99.9	99.2	100.2	101.8
Adult Education			13.1	13.2	13.3
Severe Chronic Condition 7 year			0.089	0.114	0.107
Observations		17921	1058	320	320

Income in 2007 dollars

Table 2: Transmission of Poverty in the NCPP Sample

	Offspring Education	
Family Income	>=HS	<HS
>=Poverty	96%	4%
<Poverty	81%	19%
All	93%	7%
Maternal Education		
>=HS	97%	3%
<HS	88%	12%
Paternal Education		
>=HS	97%	3%
<HS	92%	8%

Table 3A: Cross Sectional Differences in Prenatal Cortisol Levels

	Normal Cortisol	High Cortisol	Difference
Single during pregnancy	0.03	0.09	0.06
Single at age 7	0.09	0.2	0.11
Income during pregnancy as % FPL	207%	168%	-39%
Real income during pregnancy	$41,720	$35,857	-$5,863
Real income at age 7	$45,739	$39,618	-$6,121
Maternal education (years)	11.6	10.8	-0.80
Sibling number	1.5	1.5	0
Birth Order	2.9	3	0.1
Male	0.38	0.44	0.06
Observations	741	223	

Note: sample includes all births; normal cortisol defined as the bottom 75% of the distribution of cortisol and high cortisol is defined as those in the top 25% of the distribution of cortisol
All differences signficant except sibling number and birth order

Tale 3B: Within Family Differences in Prenatal Cortisol Levels

	Normal Cortisol	High Cortisol	Difference
Single during pregnancy	0.02	0.11	0.09
Single at age 7	0.22	0.23	0.01
Income during pregnancy as % FPL	228%	168%	-60%
Real income during pregnancy	$41,953	$33,286	-$8,667
Real income at age 7	$46,632	$46,024	-$608
Sibling number	1.5	1.5	0
Birth Order	3.1	3.1	0
Male	0.40	0.33	-0.07
Observations	40	40	

Note: Sample limited to families in which one sibling is exposed to normal levels and the other to highly elevated levels of prenatal cortisol as defined by cortisol in the top quartile of the distribution
All differences except single at age7, real income at age7 , sibling number, birth order and male significant.

Tale 4: Within Family Differences in Prenatal Cortisol Levels and Child Outcomes

	Lower Cortisol	Higher Cortisol	Difference	Normal Cortisol	High Cortisol	Difference
Years of Education	13.46	13.17	-0.29	13.45	12.7	-0.75
Verbal IQ at 7	103.8	99.5	-4.3	100.2	95.6	-4.6
Pr. Severe Chronic Condition at 7	0.099	0.116	0.017	0.069	0.142	0.073
Gestation (weeks)	40	39.8	-0.2	39.8	40	0.2
Birth Weight (grams)	3344	3253	-91	3244	3152	-92
Observations	158	158		202	40	

Notes: Lower and Higher Cortisol (columns 1 and 2) refer to the sibling with the lower and higher levels, respectively ;
Normal and High Cortisol (columns 4 and 5) refer to cortisol in the bottom 75 percent of the distribution and top 25 percent of the distribution, respectively

Table 5: Prenatal Cortisol and Adult Education (Years of Schooling)

Panel A Linear Cortisol	(1)	(2)	(3)	(4)	(5)
Free Cortisol (ng/ml)	-0.003	-0.006	-0.025	-0.024	-0.024
	[0.003]	[0.004]	[0.012]	[0.013]	[0.013]
Inome during pregnancy as % FPL	0.189	-0.07		-0.091	-0.133
	[0.067]	[0.075]		[0.175]	[0.205]
Black	0.136	0.5			
	[0.169]	[0.391]			
Maternal education	0.162	0.14		0.346	0.345
	[0.028]	[0.044]		[0.446]	[0.447]
Mother single at birth	-0.149	-2.095		-1.9	-1.911
	[0.263]	[0.520]		[1.918]	[1.931]
Maternal age at birth	0.035	0.051		-0.007	0
	[0.013]	[0.024]		[0.193]	[0.195]
Male	-0.336	-0.063	-0.418	-0.469	-0.468
	[0.107]	[0.142]	[0.228]	[0.230]	[0.232]
Birth order	-0.011	-0.045		0.164	0.162
	[0.041]	[0.073]		[0.299]	[0.297]
Number of siblings at age 7	-0.061	-0.051			
	[0.038]	[0.050]			
Husband at home	0.368	0.269		-0.216	-0.19
	[0.164]	[0.319]		[0.592]	[0.582]
Number moves birth to age 7	-0.041	-0.073		0.022	0.023
	[0.042]	[0.069]		[0.147]	[0.148]
Providence	-0.218	-0.197			
	[0.119]	[0.174]			
Pregnancy complication	-0.024	0.322		0.443	0.437
	[0.125]	[0.170]		[0.326]	[0.327]
Mother working while pregnant					0.154
					[0.415]
Gestation at draw date	0.005	-0.003	0.055	0.059	0.057
	[0.036]	[0.057]	[0.105]	[0.101]	[0.100]
Observations	979	299	299	299	299
R-squared	0.24	0.3	0.76	0.8	0.8
Increase of std dev in cortisol on years of schooling	-0.047	-0.095	-0.395	-0.379	-0.379
Increase as a percent of standard deviation in years schooling	-4%	-7%	-30%	-29%	-29%
Panel B Quartiles of Cortisol					
Top quartile prenatal cortisol	-0.412	-0.275	-0.668	-0.76	-0.776
	[0.121]	[0.161]	[0.428]	[0.431]	[0.431]
Bottom quartile prenatal cortisol	-0.122	-0.228	0.039	-0.072	-0.092
	[0.126]	[0.172]	[0.432]	[0.427]	[0.422]
Mother working while pregnant					0.185
					[0.380]
Observations	979	299	311	299	299
R-squared	0.25	0.31	0.74	0.79	0.79
Increase as a percent of standard deviation in years schooling	-32%	-21%	-51%	-58%	-60%
Robust standard errors in brackets					
Sample	Full	Sibling	Sibling	Sibling	Sibling
Maternal Fixed Effects	N	N	Y	Y	Y

Note: All controls included in Panel A also included in Panel B

Table 6: Prenatal Cortisol and Birth Outcomes

Panel A Linear Cortisol	Birth weight	Gestation	Birth weight	Gestation	Birth weight	Gestation	Birth weight	Gestation
Free Cortisol (ng/ml)	-0.574	0.002	0.314	0	-2.833	0.002	0.071	-0.006
	[1.051]	[0.004]	[1.028]	[0.004]	[2.513]	[0.009]	[2.438]	[0.013]
Inome during pregnancy as % FPL			13.805	-0.025	24.572	0.031	-21.378	-0.198
			[25.071]	[0.094]	[38.024]	[0.120]	[72.348]	[0.277]
Black			-111.514	-0.32	-180.851	-1.074		
			[76.245]	[0.327]	[128.405]	[0.701]		
Maternal education			8.705	-0.074	-1.607	0.011	35.671	0.253
			[13.192]	[0.045]	[25.346]	[0.058]	[80.931]	[0.429]
Mother single at birth			-55.582	0.384	154.918	0.52	22.684	0.932
			[82.886]	[0.377]	[125.545]	[0.741]	[248.925]	[2.413]
Maternal age at birth			-2.798	-0.036	-3.639	-0.007	10.49	-0.063
			[5.397]	[0.020]	[10.013]	[0.029]	[57.122]	[0.297]
Male	137.115	0.076	147.804	0.145	164.914	0.139	55.819	-0.413
	[42.891]	[0.164]	[42.847]	[0.157]	[71.489]	[0.223]	[82.219]	[0.349]
Birth order			-13.712	0.006	-2.639	-0.012	-18.725	-0.239
			[17.738]	[0.072]	[33.023]	[0.107]	[102.471]	[0.522]
Husband at home			19.153	0.267	26.312	0.56	-107.632	0.346
			[60.768]	[0.272]	[93.233]	[0.390]	[238.395]	[1.048]
Number moves birth to age 7			-11.239	-0.036	0.325	0.115	14.156	0.098
			[14.551]	[0.059]	[23.141]	[0.083]	[81.094]	[0.194]
Number of siblings at age 7			10.571	-0.046	-19.835	0.041		
			[15.747]	[0.056]	[25.499]	[0.078]		
Providence			-36.501	-0.177	-109.803	-0.385		
			[50.570]	[0.175]	[89.299]	[0.259]		
Gestation at draw date	-14.224	0.04	-12.267	0.061	-29.256	-0.072	-40.224	-0.15
	[12.718]	[0.054]	[12.847]	[0.054]	[21.451]	[0.076]	[28.871]	[0.125]
Observations	1029	1028	979	978	299	299	299	299
R-squared	0.02	0	0.04	0.03	0.09	0.04	0.83	0.77

Panel B Quartiles of Cortisol	Birth weight	Gestation	Birth weight	Gestation	Birth weight	Gestation	Birth weight	Gestation
Top quartile prenatal cortisol	-76.731	-0.235	-50.828	-0.332	-171.251	-0.367	-110.69	-0.615
	[48.649]	[0.209]	[50.064]	[0.212]	[89.399]	[0.380]	[149.189]	[0.428]
Bottom quartile prenatal cortisol	6.306	-0.297	-7.405	-0.181	56.099	-0.179	-33.952	-0.004
	[49.696]	[0.194]	[49.692]	[0.190]	[69.735]	[0.261]	[109.237]	[0.579]
Observations	1029	1028	979	978	299	299	299	299
R-squared	0.02	0.01	0.04	0.03	0.11	0.05	0.83	0.78
Robust standard errors in brackets								
Sample	Full	Full	Full	Full	Sibling	Sibling	Sibling	Sibling
Mother fixed effects included	N	N	N	N	N	N	Y	Y

Note: All controls included in Panel A also included in Panel B
Birthweigt in grams, gestation in weeks

Table 7: Prenatal Cortisol and Child IQ at Age 7

Panel A Linear Cortisol	(1)	(2)	(3)	(4)	(5)
Free Cortisol (ng/ml)	-0.022	-0.036	-0.063	-0.09	-0.09
	[0.031]	[0.063]	[0.107]	[0.111]	[0.112]
Inome during pregnancy as % FPL	2.066	1.256		0.623	0.699
	[0.700]	[1.434]		[2.144]	[2.156]
Black	-8.349	-9.454			
	[1.501]	[3.054]			
Maternal education	1.757	1.126		2.595	2.599
	[0.223]	[0.501]		[3.249]	[3.268]
Mother single at birth	-0.685	-11.732		7.308	7.336
	[1.827]	[4.721]		[10.694]	[10.761]
Maternal age at birth	0.351	0.394		1.407	1.395
	[0.122]	[0.249]		[2.232]	[2.249]
Male	1.252	2.262	1.09	0.375	0.369
	[1.085]	[2.212]	[2.962]	[2.828]	[2.828]
Birth order	0.034	-0.639		-3.017	-3.02
	[0.423]	[0.897]		[4.051]	[4.067]
Number of siblings at age 7	-0.443	-0.289			
	[0.353]	[0.764]			
Husband at home	0.322	-6.299		-12.348	-12.355
	[1.708]	[4.127]		[10.889]	[11.052]
Number moves birth to age 7	-0.015	-0.473		-2.746	-2.755
	[0.323]	[0.728]		[2.286]	[2.301]
Providence	-3.758	-3.69			
	[1.227]	[2.546]			
Pregnancy complication	-0.362	1.747		4.471	4.483
	[1.122]	[2.230]		[3.039]	[3.055]
Mother working while pregnant					-0.291
					[3.777]
Gestation at draw date	0.059	0.737	0.848	1.461	1.465
	[0.306]	[0.616]	[1.079]	[1.070]	[1.082]
Observations	952	287	299	287	287
R-squared	0.28	0.24	0.79	0.83	0.83
Increase of std dev in cortisol on IQ	-0.348	-0.569	-0.995	-1.422	-1.422
Increase as a percent of standard deviation in IQ	-3%	-4%	-7%	-10%	-10%
Panel B: Quartiles of Cortisol					
Top quartile prenatal cortisol	-1.984	-1.111	-3.601	-7.375	-7.375
	[1.096]	[2.456]	[3.652]	[4.492]	[4.485]
Bottom quartile prenatal cortisol	0.134	1.312	0.028	-1.404	-1.406
	[1.323]	[2.548]	[4.947]	[4.834]	[4.912]
Mother working while pregnant					0.015
					[3.793]
Observations	952	287	299	287	287
R-squared	0.28	0.24	0.79	0.84	0.84
Increase as a percent of standard deviation in IQ	-14%	-8%	-26%	-53%	-53%
Robust standard errors in brackets					
Sample	Full	Sibling	Sibling	Sibling	Sibling
Mother fixed effects included	N	N	Y	Y	Y

Note: All controls included in Panel A also included in Panel B

Table 8: Impact of Prenatal Cortisol on Health at Age 7 -Any Severe Chronic Condition

Panel A Linear Cortisol	(1)	(2)	(3)	(4)	(5)
Free Cortisol (ng/ml)	0	0.001	0.006	0.006	0.006
	[0.001]	[0.001]	[0.003]	[0.003]	[0.004]
Inome during pregnancy as % FPL	0.001	-0.002	0.079	0.092	0.089
	[0.018]	[0.022]	[0.062]	[0.078]	[0.080]
Black	0.047	-0.027			
	[0.040]	[0.088]			
Maternal education	0.001	0.019	-0.119	-0.118	-0.115
	[0.005]	[0.012]	[0.142]	[0.143]	[0.149]
Mother single at birth	-0.022	-0.036	-0.068	-0.064	-0.07
	[0.038]	[0.049]	[0.135]	[0.136]	[0.155]
Maternal age at birth	0	-0.005	-0.015	-0.017	-0.016
	[0.003]	[0.006]	[0.066]	[0.066]	[0.068]
Male	0.017	0.077	0.17	0.169	0.184
	[0.022]	[0.047]	[0.115]	[0.115]	[0.119]
Birth order	0.003	0.006	0.043	0.043	0.044
	[0.008]	[0.024]	[0.094]	[0.094]	[0.095]
Number of siblings at age 7	-0.01	0			
	[0.008]	[0.021]			
Husband at home	0.033	0.093	0.043	0.042	0.033
	[0.028]	[0.042]	[0.254]	[0.252]	[0.256]
Number moves birth to age 7	0.001	-0.001	0.013	0.012	0.011
	[0.007]	[0.019]	[0.060]	[0.062]	[0.065]
Providence	0.045	0.076			
	[0.030]	[0.058]			
Gestation at draw date	0.005	0.003	0.002	0.002	-0.001
	[0.007]	[0.014]	[0.032]	[0.032]	[0.034]
Mother working while pregnant				-0.05	-0.042
				[0.158]	[0.156]
Gestation					0.011
					[0.042]
Birth weight kg					0
					[0.000]
Observations	952	286	286	286	286
R-squared	0.02	0.06	0.59	0.59	0.59
Impact of std dev increase in cortisol on probability of severe chronic condition	0.000	0.016	0.095	0.095	0.095

Panel B Quartiles of Cortisol	(1)	(2)	(3)	(4)	(5)
Top quartile prenatal cortisol	-0.003	0.038	0.036	0.038	0.038
	[0.023]	[0.048]	[0.148]	[0.149]	[0.148]
Bottom quartile prenatal cortisol	0.01	0.057	-0.027	-0.022	-0.028
	[0.026]	[0.044]	[0.091]	[0.092]	[0.095]
Gestation (weeks)					0.009
					[0.043]
Birth weight (grams)					0
					[0.000]
Observations	952	286	286	286	286
R-squared	0.02	0.07	0.57	0.57	0.57
Robust standard errors in brackets					
Sample	Full	Sibling	Sibling	Sibling	Sibling
Mother fixed effects included	N	N	Y	Y	Y

Note: All controls included in Panel A also included in Panel B

Table 9: Robustness

Panel A: Control for Season of Birth	Yrs Education	Verbal IQ	Health
Top quartile prenatal cortisol	-0.812	-6.805	
	[0.435]	[4.487]	
Bottom quartile prenatal cortisol	-0.078	-1.392	
	[0.435]	[4.694]	
Free cortisol			0.006
			[0.004]
Winter	-0.398	1.707	0.022
	[0.427]	[4.280]	[0.183]
Fall	-0.649	2.932	-0.125
	[0.399]	[4.755]	[0.165]
Summer	-0.439	4.096	0.008
	[0.350]	[3.716]	[0.156]
Observations	299	287	286
R-squared	0.8	0.84	0.61
Maternal FE included	Y	Y	Y

Panel B: Gender Differences	Yrs Education	Verbal IQ	Health
Top quartile prenatal cortisol	-0.736	-7.336	
	[0.493]	[5.285]	
Top quartile prenatal cortisol* Male	-0.065	-0.105	
	[0.546]	[7.116]	
Free cortisol			0.008
			[0.005]
Free cortisol*Male			-0.005
			[0.007]
Bottom quartile prenatal cortisol	-0.075	-1.409	
	[0.432]	[4.914]	
Male	-0.415	0.346	0.274
	[0.250]	[3.307]	[0.191]
Observations	299	287	286
R-squared	0.79	0.84	0.6
Maternal FE included	Y	Y	Y

Panel C: Control for Testosteron			
Top quartile prenatal cortisol	-0.767	-7.51	
	[0.432]	[4.502]	
Free cortisol			0.006
			[0.003]
Testosteron (ng/ml)	-0.137	-1.783	-0.102
	[0.338]	[3.341]	[0.106]
Bottom quartile prenatal cortisol	-0.081	-1.55	
	[0.431]	[4.826]	
Observations	299	287	286
R-squared	0.79	0.84	0.6
Maternal FE included	Y	Y	Y

Panel D: Control for Marital Status and Income at Birth and Age 7			
Top quartile prenatal cortisol	-0.749	-8.167	
	[0.448]	[4.493]	
Bottom quartile prenatal cortisol	-0.063	-1.425	
	[0.441]	[4.989]	
Free cortisol			0.006
			[0.004]
Observations	295	283	282
R-squared	0.79	0.83	0.6
Maternal FE included	Y	Y	Y

Panel E: Impact on Performance IQ	Perf IQ	Perf IQ	Perf IQ
Top quartile prenatal cortisol	-0.203	0.135	0.783
	[1.304]	[2.164]	[5.193]
Bottom quartile prenatal cortisol	-0.347	1.294	-0.663
	[1.290]	[2.307]	[5.591]
Observations	951	287	287
R-squared	0.13	0.15	0.77
Maternal FE included	N	N	Y

Note: Sample limited to sibling sample in all regressions; all regressions include full set of controls

Table 10: Prenatal Cortisol and Parental Investments

Panel A: Linear Cortisol	**Prenatal Visits**				**48 Hour Iron**				**Maternal Focus/Responsiveness**				**Unfavorable Emotional Environment**				**Nursery School**			
Free Cortisol (ng/ml)	0.006	-0.013	-0.023	-0.017	0.005	0.007	-0.001	-0.002	0	0	-0.001	-0.001	-0.001	0	0	0	0	0.001	0.002	0.002
	[0.009]	[0.018]	[0.026]	[0.027]	[0.010]	[0.014]	[0.031]	[0.033]	[0.000]	[0.001]	[0.003]	[0.003]	[0.000]	[0.000]	[0.001]	[0.001]	[0.001]	[0.001]	[0.002]	[0.002]
Gestation at draw date	-0.312	-0.361	-0.226	-0.281	-0.001	-0.043	-0.311	-0.183	0.004	-0.002	0.008	0.01	0.006	0.013	0.021	0.028	0.009	0.003	0.004	0.01
	[0.108]	[0.173]	[0.199]	[0.211]	[0.098]	[0.156]	[0.303]	[0.322]	[0.006]	[0.006]	[0.013]	[0.015]	[0.005]	[0.007]	[0.019]	[0.021]	[0.008]	[0.008]	[0.009]	[0.012]
Income/(poverty threshold) at birth	0.28	0.159		0.294	-0.129	0.104		1.179	-0.003	0.016		-0.042	-0.016	0.008		-0.016	0.012	0.009		-0.006
	[0.165]	[0.257]		[0.538]	[0.177]	[0.263]		[0.795]	[0.012]	[0.021]		[0.049]	[0.009]	[0.012]		[0.027]	[0.019]	[0.029]		[0.026]
Black	-0.171	-0.534			-0.357	1.825			-0.05	-0.103			0.077	0.024			0.153	0.168		
	[0.408]	[0.963]			[0.481]	[1.034]			[0.026]	[0.047]			[0.047]	[0.067]			[0.058]	[0.157]		
Maternal education	-0.108	-0.089		0.321	-0.075	0.091		0.076	-0.002	-0.01		-0.098	-0.009	-0.005		0.018	0.031	0.02		0.09
	[0.059]	[0.110]		[0.707]	[0.081]	[0.143]		[0.903]	[0.004]	[0.007]		[0.085]	[0.005]	[0.005]		[0.045]	[0.008]	[0.014]		[0.055]
Mother single at birth	-0.746	0.344		1.177	0.455	-0.99		2.543	-0.041	-0.051		0.01	0.259	0.168		0.064	-0.007	0.103		-0.149
	[0.632]	[0.969]		[3.233]	[0.689]	[1.901]		[0.767]	[0.037]	[0.079]		[0.108]	[0.089]	[0.108]		[0.306]	[0.061]	[0.141]		[0.150]
Maternal age at birth	0.016	0.016		-0.702	0.12	0.149		0.412	-0.002	-0.001		-0.005	0.003	0.001		0.032	0.004	0.014		0.046
	[0.033]	[0.060]		[0.467]	[0.037]	[0.049]		[0.748]	[0.001]	[0.003]		[0.035]	[0.002]	[0.002]		[0.037]	[0.004]	[0.007]		[0.032]
Male	0.107	0.326	-0.337	-0.215	-0.491	-0.921	-0.851	-0.626	0.011	0.036	-0.007	-0.017	0.016	0.007	0.004	0.001	0.002	-0.006	-0.012	-0.011
	[0.303]	[0.537]	[0.610]	[0.616]	[0.301]	[0.441]	[1.026]	[1.225]	[0.017]	[0.024]	[0.036]	[0.046]	[0.017]	[0.016]	[0.025]	[0.031]	[0.030]	[0.047]	[0.045]	[0.046]
Birth order	-0.104	-0.308		0.591	0.013	0.37		0.794	0.009	0.007		-0.037	-0.01	0.003		-0.098	-0.003	-0.044		-0.074
	[0.126]	[0.228]		[0.864]	[0.123]	[0.256]		[1.341]	[0.008]	[0.020]		[0.049]	[0.006]	[0.011]		[0.086]	[0.011]	[0.020]		[0.053]
Number of siblings at age 7	-0.301	-0.082			0.041	0.152			0.01	0.029			0.006	0.011			0	0.028		
	[0.104]	[0.167]			[0.120]	[0.180]			[0.009]	[0.013]			[0.007]	[0.011]			[0.011]	[0.016]		
Husband at home	1.445	1.134		-1.081	-0.223	0.213		2.619	-0.037	-0.035		-0.141	-0.019	-0.053		-0.044	-0.019	0.003		-0.031
	[0.385]	[0.651]		[1.451]	[0.508]	[0.544]		[1.955]	[0.032]	[0.047]		[0.139]	[0.030]	[0.054]		[0.151]	[0.041]	[0.087]		[0.088]
Number moves birth to age 7	-0.043	0.185		0.042	0.228	0.283		0.234	0.007	0.003		-0.001	0.008	0.007		-0.003	-0.006	0.011		0.012
	[0.102]	[0.189]		[0.347]	[0.113]	[0.198]		[0.457]	[0.005]	[0.007]		[0.024]	[0.006]	[0.007]		[0.021]	[0.010]	[0.014]		[0.028]
Providence	-0.906	-1.424			-0.853	-0.48			0.032	0.059			0.085	0.036			0.139	0.125		
	[0.298]	[0.447]			[0.549]	[0.513]			[0.020]	[0.035]			[0.022]	[0.022]			[0.042]	[0.066]		
Pregnancy complication	0.164	-0.4		-0.209	-0.437	0.022		-0.759	0.002	-0.016		0.008	0.024	0.008		0.026	0.001	0.044		-0.077
	[0.318]	[0.595]		[0.815]	[0.335]	[0.446]		[0.953]	[0.017]	[0.024]		[0.076]	[0.018]	[0.017]		[0.038]	[0.031]	[0.047]		[0.050]
Observations	978	298	311	298	583	194	201	194	979	299	312	299	979	299	312	299	874	282	294	282
R-squared	0.14	0.18	0.82	0.85	0.09	0.29	0.74	0.79	0.06	0.2	0.64	0.68	0.17	0.14	0.53	0.56	0.1	0.17	0.84	0.86
Panel B Quartiles of Cortisol																				
Top quartile prenatal cortisol	0.473	0.255	0.65	0.115	0.46	-0.171	-0.937	-0.846	0.008	-0.042	-0.08	-0.101	-0.034	-0.045	-0.003	-0.033	-0.013	0.048	0.105	0.131
	[0.370]	[0.541]	[1.330]	[1.652]	[0.456]	[0.773]	[0.908]	[1.024]	[0.022]	[0.034]	[0.061]	[0.082]	[0.021]	[0.028]	[0.061]	[0.066]	[0.028]	[0.036]	[0.079]	[0.091]
Bottom quartile prenatal cortisol	0.133	0.02	0.756	0.389	-0.255	0.49	-0.059	0.228	-0.002	-0.012	-0.063	-0.058	-0.02	-0.038	-0.059	-0.064	0.007	0.029	-0.001	-0.01
	[0.359]	[0.479]	[1.181]	[1.115]	[0.336]	[0.612]	[0.756]	[0.781]	[0.013]	[0.016]	[0.055]	[0.069]	[0.018]	[0.025]	[0.095]	[0.098]	[0.037]	[0.040]	[0.034]	[0.034]
Observations	583	194	201	194	978	298	311	298	979	299	312	299	979	299	312	299	874	282	294	282
R-squared	0.1	0.3	0.74	0.79	0.14	0.18	0.82	0.85	0.06	0.2	0.65	0.69	0.17	0.16	0.53	0.57	0.1	0.18	0.85	0.87
Robust standard errors in brackets																				
Sample	Full	Sibling	Sibling	Sibling	Full	Sibling	Sibling	Sibling	Full	Sibling	Sibling	Sibling	Full	Sibling	Sibling	Sibling	Full	Sibling	Sibling	Sibling
Maternal FE	N	N	Y	Y	N	N	Y	Y	N	N	Y	Y	N	N	Y	Y	N	N	Y	Y

Note : All controls included in Panel A also included in Panel B; 48 hour iron levels are missing for much of the sample