Sound Body, Sound Mind? Asymmetric and Symmetric Fetal Growth Restriction and Human Capital Development

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Abstract

This paper explores the causal pathway by which poor fetal health translates into reducing educational attainment and earnings as an adult. Using insights from the medical literature, I decompose low birth weight infants into two distinct subtypes: a symmetric type, which is characterized by cognitive deficits, and an asymmetric type, which exhibits little to no cognitive problems. Using data from a longitudinal survey of newborns, I establish three results: First, there is empirical evidence of brain sparing in the asymmetric subtype, but not in the symmetric subtype. Second, despite differences in cognitive impairment, both subtypes exhibit similar impairment to physical health. And finally, there is evidence that the causes and timing of onset during pregnancy are different for asymmetric and symmetric growth restriction. The results indicate that differentiating between these subtypes may offer new opportunities to identify the underlying casual relationships between health and human capital development, as well as uncovering the "black box" mechanism behind the fetal origins hypothesis. These results also have broad implications for the timing of policy interventions aimed at pregnant women.

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1 Introduction

One of the most ubiquitous topics of research in areas of labor and health economics is human capital development. Early literature focused on the causal relationship between health and education, and the hypothesis that poor physical health reduces educational attainment is supported by many studies. The primary method of identification is to use birth weight as an exogenous measure of health endowment (see Grossman [2008] for a summary of the history of this research). In a related, but divergent, set of literature the focus is not identifying the causal link between health and education, per se. This literature, summarized by Almond and Currie (2011), instead focuses on the effect of in utero conditions on adult outcomes as a research question. Papers cite the Fetal Origins Hypothesis (or Barker Hypothesis) as the mechanism that translates in utero health to adult education and earnings, and identification generally relies on "natural experiments", where there is a sharp change in the environment of the fetus for some specific population (e.g. Almond [2006]) or sibling/twin difference models (e.g. Royer [2009]). However, fetal programming occurs through some unknown biological mechanism, which makes causality about the relationship between health and education difficult to determine.

This paper seeks to close the gap between the health and education literature and the fetal origins literature by exploring the causal pathway by which poor fetal health translates into reduced educational attainment and earnings as an adult. Using insights from the medical literature, I decompose low birth weight infants into two distinct subtypes: symmetric and asymmetric. According to medical theory, the symmetric type exhibits proportional growth restriction in all major organs, including the brain. The asymmetric type, while also growth restricted, exhibits spared brain growth and development. By recognizing this heterogeneity, I establish three results: First, there is empirical evidence of lower IQ scores in the symmetric subtype but not in the asymmetric subtype. Second, despite differences in cognitive impairment, both subtypes exhibit similar impairment to physical health. And finally, there is evidence that the causes and timing of onset during pregnancy are different for asymmetric and symmetric growth restriction.

Figure 1 shows the importance recognizing the heterogeneity in growth restricted infants. It shows the distributions for IQ broken down by symmetric growth restriction, asymmetric growth restriction, and non-IUGR. There appears to be little or no difference between the distributions for asymmetric growth restriction and non-IUGR. However, there is a clear negative shift in the IQ distribution for symmetric growth restriction.

These results inform the economics literature in several ways. Previous studies that use

low birth weight as an indicator of the health endowment may inappropriately attribute poor educational and labor market outcomes to low birth weight per se, rather than to the poor cognitive development that occurs in some babies. As a result, combining asymmetric and symmetric births can lead to invalid inference. Second, differentiating between the subtypes offers a potential mechanism for the fetal origins hypothesis: human capital is affected through decreased cognitive function caused by brain growth restriction in utero. Thus, more focused estimates of fetal programming on education and earnings can be obtained by focusing only on the subset of growth restricted infants for which brain development is compromised. Third, because this decomposition shows one group with impaired cognitive function and physical health and another group in which only physical health is affected, we can conclude that using birth weight alone to empirically test the causal effects of physical health on education is inappropriate. However, an unbiased test may be possible using only the asymmetric subtype, for which only physical health is affected. Furthermore, these results may also help inform some of the inconsistencies in the current economics literature. Lastly, since these subtypes are shown to have different causes and timing during pregnancy, these results can help inform more effective policy interventions.

This paper proceeds as follows: Section 2 provides information about intrauterine growth restriction and its subtypes, as well as reviews of relevant literature in medicine and economics. Section 3 describes the empirical strategy for testing the effect of growth restriction on cognitive function. Section 4 describes the data used and definitions constructed to differentiate between the types of growth restriction. Section 5 discusses the results for testing the association between growth restriction and cognitive ability. Section 6 details the relationship between growth restriction and physical health. The causes and timing of growth restriction is explored in Section 7. And Section 8 discusses the relevance of the results, possibilities for future research, and concludes.

2 Background

Intrauterine growth restriction (IURG) (also known as fetal growth restriction or FGR) is a condition of decreased development and growth prior to birth. IUGR is the result of some abnormal circumstance during pregnancy that reduces placental function. The source of the problem can be a placental, maternal, or fetal abnormality. Examples of common placental disorders that affect its function are multiple gestations, placental tumors, infection, chronic separation, and abnormal insertion. Maternal abnormalities that contribute to or are associated with growth restriction are maternal size, nutrition, socioeconomic status, chronic

disease, and the use of certain illegal and prescription drugs. Diseases that have the largest negative impact on fetal growth are ones that cause narrowed blood vessels or low oxygen levels in the blood, both of which reduce the ability of the placenta to deliver nutrients and oxygen to the fetus. Use of certain drugs can also do damage by narrowing blood vessels or reducing blood-oxygen levels[1]; however, the main effect of drugs like methadone, heroine, and alcohol on birth weight is through a toxicity that directly impedes cellular replication and growth. Environmental factors such as exposure to toxic chemicals and high altitude are also known or believed to cause IUGR. Fetal factors that contribute to growth restriction include chromosomal abnormalities, metabolic disorders, various syndromes, and congenital infection (Martin, Fanaroff, and Walsh 2005).

Intrauterine cell growth occurs in three phases. The first stage lasts from conception to 16 weeks of gestation and is characterized by a rapid increase in cell number (hyperplasia). In the second phase, hyperplasia continues and is accompanied by rapid increase in cell size (hypertrophy). This phase lasts until 32 weeks of gestation. In the final stage, the fetus grows only by increase in cellular size. This is the part of fetal development in which the fetus develops most of its fat and muscle weight (Cunningham et al. 2009). Because of the difference in biological processes occurring during different stages of fetal development, the timing—not just the severity—of the shock to fetal growth is crucial to the pattern of growth restriction. It is common in medical contexts to classified IUGR into two categories: symmetric growth and asymmetric growth (Martin, Fanaroff, and Walsh 2005).

2.1 Causes of Fetal Growth Restriction

Typically occurring late in pregnancy, asymmetric growth restriction is characterized by preservation of blood flow to the carotid vessels (responsible for supplying blood to the brain) in utero (Kliegman et al. 2007). That is, the fetal brain continues to get adequate nutrition and oxygen, despite other organs suffering.[2] This is known in the medical literature as a the "brain sparing" effect, and it is thought to be the result of the fetus adapting to poor intrauterine condition by redistributing its own cardiac output mainly to essential organs like the brain (Martin, Fanaroff, and Walsh 2005).

Asymmetric growth restriction can be caused by poor maternal nutrition, especially late in pregnancy. Nutrition demands of the embryo and fetus in early pregnancy are small; thus

[1]This is suggested as a mechanism for the effect of cigarettes on growth restriction (Martin, Fanaroff, and Walsh 2005).

[2]The spleen, liver, adrenal, thymus, and fat tissues are the most compromised by late onset growth restriction (Martin, Fanaroff, and Walsh 2005).

poor nutrition may not cause restricted growth until the fetus becomes more calorically and nutritionally demanding in the second and third trimesters (Martin, Fanaroff, and Walsh 2005). Other common causes of asymmetric restriction are the worsening of a maternal vascular disease, such as preeclampsia or chronic hypertension, in the later stages of pregnancy (Kliegman et al. 2007).

Catch-up growth can occur once the infant is placed in a more favorable environment after birth. The final stage of growth is only hypertrophic, only cell size–not cell number–increases. Since asymmetric growth restriction is typically late onset, infants of this subtype tend to have a better prognosis with regard to catching-up to the normal growth curve during the perinatal stage (Martin, Fanaroff, and Walsh 2005).

Symmetric growth restriction typically has an earlier onset. This type of growth restriction is considered symmetric because birth weight, length, and head circumference are equally affected. Despite the early insult to growth, these fetuses may continue to grow at a normal rate throughout pregnancy; however the gross size is permanently reduced due to a disruption of early cellular replication. Insults to the fetal environment in the first 16 weeks of pregnancy impair fetal cells from replicating, reducing the total number of cells and, thus, the potential for growth. Common causes are chromosomal abnormalities, genetic factors, severe malnutrition, birth defects, infection early the early stages of pregnancy, or severe maternal hypertension (Kliegman et al. 2007). Early growth delays are also reported for fetuses of many diabetic mothers. The use of illegal drugs and medication not approved for pregnancy is often associated with symmetric growth restriction due to its ability to affect cellular replication. Due to its early onset, symmetric IUGR is known to restrict growth in all major organs including the brain and skeleton (Martin, Fanaroff, and Walsh 2005).

2.2 Economic Literature

The economic literature on human capital development and its relationship to the fetal environment and early childhood is quite extensive, albeit a relatively new area of focus. I refer the reader to Almond and Currie (2010) for an all-inclusive literature review. The literature reviewed here is only focused on recent literature concerning the effect of low birth weight or poor in utero conditions on human capital development in childhood or adulthood.

The idea that low birth weight—considered a poor health endowment—might affect human capital in adulthood was first proposed by Currie and Hyson (1999). They found that individuals that were of low birth weight were less likely to pass standardized test and less likely to be employed. The implication here is that poor health causes a reduction in hu-

man capital development. Many studies have confirmed this relationship utilizing samples of sibling or twin difference models (Behrman and Rosenzweig 2004; Almond, Chay, and Lee 2005; Oreopoulos et al. 2008; Royer 2009; Black, Devereux, and Salvanes 2007).

Other literature concerning the fetal origins hypothesis focuses on using "natural experiments" of sharp changes in the fetal environment rather than differences in birth weight (e.g. Almond [2006] and Almond and Mazumder [2011]). Estimates obtained using this empirical strategy have the advantage of eliminating socioeconomic bias inherent with this type of estimation without sacrificing generalizability like twin-effects estimation does. The disadvantage of this methodology is it only reveals the effect of changes in the fetal environment on human capital development; the causal pathway, whether through reduced physical health or impaired cognitive ability, is impossible to determine. [3]

The implied mechanism translating poor fetal health into poor human capital can be summarized as follows: poor conditions experienced by the fetus in utero cause poor health in childhood; poor health in childhood causes poor health in adulthood; and poor health in childhood and adulthood causes decreased educational attainment, lower income, and lower socioeconomic status. A summary of empirical equations that are typically estimated to show the pathway between birth weight and schooling are found in Figure 2.

Equation (A) is a birth weight production function. The variable of interest is the behavior of the mother, specifically modifiable behavior that can be influenced by policy. Equation (B) describes the relationship between poor fetal health and poor infant health, H_i. Equations (B) and (C) taken together describe what is call the *Fetal Origins Hypothesis* (or Barker Hypothesis). It suggests that the same poor in utero conditions that produce low birth weight "program" a fetus to have health problems as an adult. Finally, the Equation (4) is the question that started this line of research: how does health effect education? Since estimating Equation (D) using adult health, H_{i+1}, is endogenous, researchers typically estimate the reduced form model—considering low birth weight as an exogenous measure of health endowment. Estimation then proceeds via family fixed effects or by quasi-natural experiments of exogenous changes in the fetal environment.

However, the proposed mechanism does not fully address two key questions: Can birth weight serve as a valid proxy for physical health? And can changes in the fetal environment be used to explain whether the observed effect on human capital occurs via decreased phys-

[3]One exception in this literature is Almond, Edlund, and Palme (2009). By focusing on early pregnancy, the authors are able to show that exposure to the Chernobyl fallout in utero has a significant impact on schooling outcomes, but not physical health. However, the rationale provided for focusing on early pregnancy is specific to radiation exposure. Thus, it is unclear if the link between cognitive ability and early pregnancy problems generalizes to additional insults to the fetal environment.

ical health, decreased cognitive ability, or some combination of the two? Reexamining the pathway from birth weight to schooling with the assumption that there are two different subtypes of fetal growth restriction may help answer these questions. In this paper, I estimate versions of Equations (B) and (C), adding the decomposition of low birth weight, as well as decomposing health into cognitive health and physical health. I also estimate Equation (A) allowing asymmetric and symmetric growth restriction to potentially have different causes and timing of onset.

From estimating Equations (B) and (C) I find severe cognitive impairment in the symmetric group but not the asymmetric group, as measured by IQ scores in early childhood. This makes interpreting effects of birth weight as a causal effect of physical health on education or labor market outcomes inappropriate. More specifically, this implies that Equation (D) is a misleading estimator of the effects of physical health on education. Estimating Equation (D) combines the effects of asymmetric and symmetric growth restriction, and the cause of decreased achievement in education and in the labor market is likely due to cognitive impairment for infants suffering from symmetric growth restriction, not necessarily physical health. Thus the effect of health on education may be over-stated. We could also think of the true impact of growth restriction on education and labor market outcomes as being driven by symmetric growth restriction. In this case, combining the symmetrically growth restricted infants with the asymmetrically growth restricted infants, for whom little or no cognitive effect is present, under-states the potential gains from policy intervention. Furthermore, estimating the value of interventions in the fetal environment (Equation (A)) is problematic because in this paper asymmetric and symmetric growth restriction are shown to have different timing of onset. Symmetric growth restriction onsets early in pregnancy, whereas asymmetric growth restriction onsets late. This—coupled with the differences in cognitive outcomes—means that the intended impact of a policy may be over- or understated, depending on the type of growth restriction most reduced.

The idea that the effect of a poor fetal environment may affect human capital through cognitive ability rather than through physical health is not a new one (this is recognized as a possibility by both Royer [2009] and Black, Devereux, and Salvanes [2007], for example). However, the results of this paper not only provide a mechanism for how this takes place, but also allows for the separation of the cognitive effects from the physical health effects. The advantage of this is that it may be possible to perform an unbiased test of the effects of physical health on education by utilizing asymmetric growth restriction alone. Furthermore, focusing on symmetric growth restriction alone may show that the costs of early pregnancy complications (measured in reduced human capital) are much larger than

are currently attributed to them.

On a narrower scope, the results found in this paper may offer some rationale for common unexplained findings in the literature and provide a mechanism that may help reconcile seemingly contradictory results. These are discussed in detail in Section 8.

3 Methodology: The Brain-Sparing Hypothesis

To evaluate the differential impact of asymmetric and symmetric growth restriction on cognitive ability, I estimate the following equation:

$$C_i = \alpha_0 + \beta_1 I_{asym} + \beta_2 I_{sym} + \gamma X_i + \epsilon_i \tag{1}$$

where C_i is a measure of cognition, I_{asym} and I_{sym} are indicator variables for whether a child was born asymmetrically or symmetrically growth restricted, and X_i is a vector of controls. The measures for cognitive ability are Welsher IQ scores at ages 4 and 7. As noted by Cunha and Heckman (2007), IQ scores are a better measure of pure cognitive ability, as opposed to scores on performance test, which were not designed to measure cognition.

Given the developmental story concerning asymmetric and symmetric growth restriction, the hypothesis is for symmetric growth restriction to have a large, negative effect on IQ scores compared to non-growth restricted children due to disrupted brain development in utero. On the contrary, β_1, the coefficient on asymmetric IUGR, is expected to be small and possibly not significantly different from zero due to the "brain sparing" that characterizes asymmetric growth restriction.

The problem with estimating Equation 1 is that both cognitive ability and the incidence of growth restriction in utero are likely jointly determined by socioeconomic and genetic traits of the child's parents. To avoid a downward bias in the estimates β_1 and β_2 that would result from this endogeneity, X_i must contain sufficient covariates to remove any conditional correlation between growth restriction and the error term. I include in X_i the mother's age (as a quadratic function), the mother's height, indicators for marital status, indicators for the mother's and the father's education attainment, indicators for family income at birth, a socioeconomic status score for the family when the child is 7 years old, the number of prenatal visits (as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.[4] When estimating Equation 1, the implicit assumption is that

[4]Prenatal visits are included quadratically because a high number of visits may indicate a problem pregnancy.

this set of controls is sufficiently correlated with unobserved genetic and home environment characteristics to act as a proxy.

Despite controlling for an extensive set of parental and socioeconomic characteristics, there remains the possibility of unobserved genetic characteristics or characteristics of the home environment biasing coefficients if this unobserved heterogeneity is correlated with size at birth and cognitive ability. In the economics literature, researchers typically use twin or sibling difference estimators to deal with this particular endogeneity problem. The assumption is that a mother fixed effect controls for heterogeneity in the home environment while also removing some endogeneity from genetic factors.

There are, however, several identification issues with using a mother fixed effect. One of the largest limitation is data availability. The data used for this paper, despite containing nearly 60,000 births, include less than 700 twin pairs and fewer than 9,000 subjects with siblings in the sample that can be used for estimation. Another issue is the generalizability of results. Children living in an environment with siblings—especially twins—may not share patterns of cognitive development with other groups in the population.

These issues are amplified when using prospective survey data. Subjects with a sibling recorded in the sample must have parents who not only made the decision to have more children, but also chose to have more children within the time frame of the data collection of the study, did not move, and chose to be involved with the study when having another child. If any of these family characteristics are correlated with anthropometric measures or IQ, then there is a selection problem. Furthermore, when using the empirical method employed in the paper (categorical dummies), identification of the coefficients in the fixed effect model is driven only by families who have at least one IUGR child and one child that grew normally in utero for comparison. This occurs only occasionally, and there is a significant reduction in statistical power to draw valid inferences, given the already small sub-sample size. Finally, fixed effect identification implicitly assumes that a mother's behavior does not change after having an IUGR child.

Despite these issues, controlling for family environment (and possibly some genetic traits) is an interesting avenue to explore. Therefore, I also estimate the following equation in addition to Equation 1

$$C_i = \alpha_0 + \beta_1 I_{asym} + \beta_2 I_{sym} + \gamma W_i + M + \epsilon_i \tag{2}$$

where M is a mother fixed effect, and W_i is a subset of the control set X_i that is not perfectly correlated with M. Additionally, since birth order may affect unobserved parental

investment, I also include an indictor for being the first born child, the interaction between this indicator and an indicator for being IUGR, and the number of children in the family. The same results are expected for the fixed effect specifications as the OLS specifications, despite the fact that estimates will likely be noisier due to the issues stated above.

One issue the above estimations cannot solve is whether the improvements in cognitive ability merely reflect differences in physical health (which may affect education). To answer this question, in Section 6 I estimate the difference in physical health outcomes associated with asymmetric and symmetric growth restriction. If there is no discernible difference in physical health between the two subtypes, then the above estimation can safely be considered a test of brain sparing. In Section 7, I further explore the necessity of differentiating between the asymmetric and symmetric growth restriction by testing whether the subtypes have different causes or timing of onset.

4 Data

The data are from the Collaborative Perinatal Project (CPP). The CPP is a multi-hospital study of pregnancy and early childhood conducted from 1959 to 1974. The study consists of 59,391 pregnancies to women randomly recruited to participate in the study at medical centers in one of 12 major U.S. cities from 1959 to 1966. Data were collected on the mother and father's medical history and demographic characteristics. Information about the pregnancy was recorded at each prenatal visit. Data was collected on the surviving children at 4, 8 and 12 months of age, as well as at 4, 7 and 8 years of age. The entire CPP dataset contains 6,783 variables broken into 52 data files. The computerized version of this data used in this paper is available from John's Hopkins University (Lawlor et al. 2005).

This data has several distinct advantages. First and foremost, to the author's knowledge, this is the only prospective study on children that includes anthropometric measurements at birth—like head circumference and crown-heel length—in addition to birth weight and gestational age. These anthropometric measures are critical for identifying asymmetric and symmetric IUGR in newborns. Furthermore, this dataset contains information about the child's intelligence, as well as measures of health. This not only allows for the potential differentiation between the subtypes of IUGR infants, but also allows for statistical testing of the effects these conditions have on early childhood metrics of intelligence and health.

Since the data were collected in metropolitan areas, black families and families of low socioeconomic status are over-sampled. Over 80 percent of those sampled for the CPP earned less than the mean family income in 1960, and nearly 70 percent of families earned

below the median family income. Furthermore, African American families make up nearly 47 percent of the original sample. To put this in perspective, nearly 89 percent of United States population was white in 1960; so the CPP was obviously not representative of the population at the time. However, since this paper is particularly concerned with poor fetal health, this is actually an advantage because growth restricted infants are more common among black and low-income parents.

Not all of the nearly 60,000 observations are used in this study. Measurement error is a concern with this dataset. Specifically the accuracy of the gestational age and birth weight combinations could be problematic. This is because the date of the last normal menstrual period is often reported with poor accuracy, especially for unplanned pregnancy. This is particularly true before wide spread use of ultrasonography to estimate and verify gestational age. To mitigate this problem, infants reported as born at a gestational age less than 26 weeks or greater than 45 weeks are dropped. Additionally, implausible combinations of gestational age and birth weight are removed according to criteria developed by Alexander et al. (1996). Observations whose race is not defined as black or white are also dropped. The small number of observations that were not black or white and the lack of published growth standard for other races made it difficult to classify these infants by anthropometric measurements. Finally, since this paper attempts to identify subtypes of growth restricted infants by anthropometry for gestational age, observations with missing values for birth weight, head circumference at birth, or gestational age are removed. This leaves 47,019 observations for analysis. The number of observations in each regression varies depending on the number of missing values in the dependent variable or independent variables of interest. For example, IQ scores at age 4 are only available for 34,641 children.[5] Table 1 contains summary statistics for the variables utilized in this paper.

4.1 Classification of asymmetric and symmetric growth restriction

One of the primary challenges of this project is identifying the subtypes of growth restricted infants. Large, public-use data—such as the National Natality files—do not contain the necessary anthropometric data to make this distinction. Although the medical literature provides some guidance, much of the literature concerns identifying growth restriction in

[5]For regressions with mother fixed effects subjects with any congenital malformations are also removed. Malformations are not a major concern with the OLS estimates due to the large overall sample size(OLS estimates are unaffected by removing observations with major congenital malformations). However, the estimates of the fixed effect model are driven by the comparison of two observations, which increases the possibility that a large outlier could affect the results.

utero using ultrasonography. Medical studies on the subject generally use some combination of birth weight, head circumference, and crown-heel length to both determine whether a neonate is growth restricted and to differentiate between the symmetric and asymmetric subtypes. A review of the medical literature's treatment on the identification and classification of asymmetric and symmetric fetal growth restriction is delegated to Appendix 3.

In short, there is a of lack academic consensus in the medical literature regarding the definition of asymmetric and symmetric growth restriction and—perhaps consequently—conflicting empirical evidence concerning the theory behind the classification. Therefore a major contribution of this paper is a large scale, statistical test of the brain-sparing hypothesis using multiple definitions for distinguishing between the subtypes of IUGR. This paper's decompositions of restricted growth can be broken down into two main types: in-sample definition and out-sample definition. In-sample definitions are generated using percentile cutoffs created from the CPP data set. Out-sample definitions are generated using published standards of birth anthropometry in the medical literature. A breakdown of the pros and cons of each definition and the methodologies behind them is found in Appendix 1. This section describes only the preferred definitions and datasets used to construct variables of interest. Appendix 1 contains further discussion of alternative definitions and datasets.

Neonates are defined as IUGR if their birth weight is below the 10th percentile for gender, race, and gestational age. In addition to be being the textbook definition for IUGR, the 10th percentile cutoff is also the most commonly used in the medical literature. Since asymmetric growth restriction is characterized by relatively normal brain growth (and consequently normal head growth) despite impaired overall growth, neonates that are classified IUGR with a head circumference above the 10th percentile for gender, race, and gestational age are defined as asymmetrically growth restricted. Symmetric growth restriction is likewise defined as an IUGR neonate with a head circumference below the 10th percentile for gender, race, and gestational age, since it is characterized by proportionally retarded growth.

For the out-sample classifications, these cutoffs are determined by published birth standards from approximately the same time period from which the data is collected. The preferred datasets were chosen by sample size and representativeness of the population (see Appendix 1). The advantage of using the published birth standards is that the samples are designed to be representative. For example, the preferred birth weight data come from a 50 percent sample of all U.S. births in 1968, and percentiles are available by gender, gestational age, and race (Hoffman et al. 1974). The disadvantage is that the head circumference and birth length standards come from significantly smaller samples and percentiles are not available for white and black newborns separately.

Since the CPP is such a large dataset of birth records, I also define classifications for IUGR and its subtypes from within this sample. The greatest advantage of using the CPP to create percentile cutoffs is the head circumference data; the CPP is a much larger dataset, and it is possible to define percentiles by race in addition to gestational age and gender. The disadvantage of using in-sample definitions is that the CPP is not a representative sample. Thus the 10th percentile cutoff created from the CPP may not reflect where this value lies for the general population.

Summary statistics for the preferred definitions can be found in Table 2 under the headings "In-Sample Definitions" and "Out-Sample Definitions". To the authors knowledge, this is the first paper to standardize birth weight cutoffs by gestational age, race, and gender. Thus, by construction, both groups defined as IUGR and as normal birth weight have a cross section of all gestational ages, as well as a representative balance of each gender and race. As Behrman and Rosenzweig (2004) point out, using birth weight alone is likely measuring differences in gestational age. However, this is also true for gender and race, which are also highly correlated with birth weight. Standardizing birth weight by gestational age, gender, and race ensures that the effects being measured in this paper are that of IUGR and not that of other variables highly correlated with birth weight.

5 Results: IUGR & Cognitive Ability

5.1 Main Results

Results from estimating Equation 1 and Equation 2 are found in Table 3. The top of the table displays results from estimating within-sample definitions, and the middle shows results from using outside sources to define the subtypes of growth restriction. The two columns on the left of the table show OLS estimates using both IQ at age 4 and at age 7; the two columns on the right of the table show fixed effects estimates of the same outcomes. The sample size, R-squared,[6] and the p-value of the one-sided F-test that the symmetric coefficients are less than the asymmetric coefficients are found below each set of results.

Estimates of the marginal impact of symmetric growth restriction are large and highly significant across all specifications and definitions. At the mean, the presence of symmetric IUGR reduces a child's IQ by somewhere between 3.7 and 4.7 percentage points for the OLS models and 3.2 to 4.7 percentage points for the fixed effects models, which is approximately a quarter to a third of a standard deviation. Although the coefficients estimated for asym-

[6] R-squared for fixed effects regression measures the explained *within group* variation.

metric restriction are also mostly negative and sometimes statistically different from zero, the magnitude is typically much smaller than for symmetric restriction, ranging from less than a point to 1.9 points. The estimates of the effects of symmetric growth restriction are approximately 2 to 5 times larger than those of asymmetric growth restriction. Each table contains the p-value for the F-test that the symmetric growth restriction has a larger negative impact than asymmetric growth restriction. The estimates are statistically different at the $\alpha = 0.001$ level across all OLS specifications and statistically different at the $\alpha = 0.05$ level in all but one fixed effects specification.

Both OLS and fixed effects specifications yield estimates of similar magnitudes and significance, despite the models having different identification issues, which provides strong support for the hypothesis being tested. The main difference between the OLS and fixed effects results is that the standard errors are significantly larger in the fixed effects results due to the smaller sample size.[7] This problem is exacerbated by the reality that many families do not have at least one IUGR child and one non-IUGR child, which is necessary for identification. This makes statistically distinguishing between the asymmetric and symmetric estimates via the F-statistic much more difficult, which is reflected by larger p-values. However the consistency between specifications speaks to the strength of the empirical relationship estimated by these equations.

Reestimating these results using different cutoffs for the in-sample classifications or different published standards for the out-sample classifications does little to affect the results. Magnitudes remain relatively consistent, and significance almost never changes. In Appendix 2, the OLS results for all the classifications can be found in the Tables 2 and 3, and a description of all alternative definitions can found in Figure 1. Like the OLS results, the results from the fixed effect estimation are robust to using several alternative definitions. Results from these estimations are in Tables 4 and 5 also in Appendix 2.

Taken together, these results lead us to two major conclusions: 1.) cognitive ability is unambiguously negatively affected by symmetric growth restriction in utero, and 2.) there is strong evidence that asymmetrically growth restricted fetuses are at least partially shielded from brain growth restriction through "brain sparing". This implies that lumping these two groups together as *low birth weight* or implicitly grouping them together by identifying the marginal effect of birth weight or a change in the "fetal environment" on some human capital outcome is likely yielding misleading estimates.

[7] One may also be concerned that the sample of children who have a sibling also sampled by the CPP is fundamentally different from the sample as a whole. However, OLS results are nearly identical to those found here when the OLS sample is limited to individuals with siblings.

5.2 Standardizing Birth Weight Across Sub-Types

One concern with the analysis in the previous sections is that the decomposition of IUGR into asymmetric and symmetric subtypes is simply another way to state differences in birth weight. That is, it may be that symmetrically growth restricted infants are significantly smaller than asymmetrically growth restricted infants. Simple difference of means tests show there may be some truth to this hypothesis. For in-sample definitions, there is a statistically significant difference in birth weight of about 230 grams. For definitions constructed using published sources, asymmetric IUGR infants outweigh symmetric IUGR infants by 340 grams.[8] Furthermore, there is a statistically significant difference in gestational age at birth of 0.46 to 0.83 weeks.

The problem here is two-fold. First the gradient relationship between birth weight and both health and education is well documented in the economics literature. If the analysis of this paper is simply restating the common conclusion that there is a negative relationship between birth weight and health and ability in a different manner, then it has little of value to add to the literature. Furthermore, since lower birth weight infants are known to be in poorer health, the difference in cognitive ability may still be a result of physical health affecting schooling—since low IQ could be a reflection of poor schooling.

To investigate this, I construct new measures of the IUGR subtypes such that birth weight is forced to be comparable between asymmetric and symmetric growth restriction. IUGR continues to be defined in same manner as previously described in Section 4.1. However, instead of defining asymmetric growth restriction by the absence of small features (head circumference $>$ 10th percentile) for gestational age, it is now defined as having large features for birth weight. The new in-sample definition of asymmetric growth restriction is having a head circumference greater than the 90th percentile for birth weight calculated from the CPP data using 17 birth weight categories.[9]

Another reason this methodology is useful as a robustness check is that Yogman et al. (1989) shows that birth weight is a better standard for head circumference comparisons than gestational age. Summary statistics of these definitions are in Table 2 under the "Matched BW Definitions" heading. As designed, the birth weights of the subtypes are within 70

[8]It is worth pointing out that the differences in birth weight could be completely accounted for by the difference in head size. A one centimeter change in head circumference causes an increase in birth weight of approximately 250 grams, and the mean difference in head circumference between asymmetrically and symmetrically growth restricted newborns is approximately 2.5 cm (a predicted difference of more than 600 grams)

[9]The categories begin at a $<$1000 grams category and increasing in 200 gram increments (1000-1200 grams, 1200-1400 grams, etc.). This method is identical to that utilized by Usher and McLean (1969).

grams (less than 2.5 ounces) of each other using the new definitions, while preserving the differences in head circumference.

The results from Sections 5.1 are recalculated using these new definitions, and the results are displayed in bottom section of Table 3. Despite the new definition, estimates for the IQ regressions are largely unchanged. However, the estimates for asymmetric growth restriction are notably less precise. This likely is due to the count of identified asymmetric IUGR infants being less than half that of the standard in-sample definition (see Table 2).

In summary, these results show that the estimates found in Sections 5.1 are not purely a result of difference in birth weight. Redesigned cutoffs to discriminate between the subtypes of IUGR standardized by birth weight show little differences from the main results. That is, the same statistical pattern persists despite no longer being able to discriminate between the subtypes by severity of growth restriction (proxied by birth weight and health differences). This provides strong evidence that the etiology of poor health and cognitive outcomes for growth restricted infants goes beyond birth weight alone.

5.3 Other Identification & Selection Issues

Because of the non-random nature of the CPP sample and the sample attrition problem that arises with the follow-up data on IQ, sample selection may be a concern. In this section, I explore four different areas that may cause selection bias: the over-sampling of black and low-income families, maternal smoking, fetal mortality, and sample attrition.

The oversampling of low-income and black families is of little concern for the analysis of this paper. This is due to how the variables of interest are identified. The over-sample of low-income families is likely to result in a lower mean level of ability for the CPP sample. Since I use dummy variables to identify the effect of asymmetric and symmetric growth restriction on cognitive ability, the estimates are identified on the difference in conditional means between the IUGR group and the normal birth weight group. Thus, a lower average ability in the group as a whole means I am less likely to identify a difference between the IUGR children and the rest of the group, giving the magnitude of the estimates a lower-bound interpretation.[10]

The CPP data were collected in a dynamic period regarding public knowledge and attitude about the dangers of smoking. Thus one may worry that the results are influenced by changes in smoking behavior, particularly if this change affects people of different socioeco-

[10]This is not likely a concern for the asymmetric estimates either, since they are still found to be statistically distinguishable from the symmetric estimates.

nomic statuses differently. However, all results hold when controlling for maternal smoking behavior.

Another concern is that IUGR babies have a higher rate of neonatal death than babies that grew normally in utero. As pointed out by Almond (2006), increased mortality among IUGR babies creates an upward bias since survivors presumably have more favorable genetics characteristics. This is not a problem for estimates of symmetric growth restriction—estimated coefficients will simply represent an upper bound for the negative effects (or a lower-bound in magnitude). This upward bias may present a problem for the asymmetric estimates, which are expected to be near zero. However, this potential bias is unlikely to affect the estimates of this paper. The primary reason for this is that a very small portion of the total sample suffers from neonatal mortality. Only 107 of more than 4800 IUGR infants die in the first 3 months of life. Of these, only 15 are asymmetrically growth restricted. Thus any noticeable bias will likely only affect the coefficients on symmetric growth restriction, and the bias will be toward zero. Furthermore neonatal mortality rates are not statistically different between asymmetrically growth restricted neonate and non-IUGR neonates.

A larger concern is sample attrition. Approximately 13,000 children are lost to the sample between birth and ages 4 and 7 (when the IQ tests are performed), which represents about one-quarter of the usable sample at birth. If children not returning for the 4 or 7 year follow-up is a random event, then this will simply make the estimates of this paper less precise. Indeed this may be the case, since there is no difference in observable characteristics between the two groups. However, I test the possibility further by running a Heckman selection model. There are three problematic reasons why a child may not be present in the 4 or 7 year old sample: the family has moved away from the area of the participating hospital, death, and inability to bring the child in for testing because of single-parent household. I construct an exclusion restriction to account for each of these factors. To account for the possibility of moving, I construct a dummy variable for whether the mother is still living in the same region as she was born—the assumption is that if the mother has made a major move before, then she is more likely to do so again; also a mother who still live the same reason as she was born is reasonably less like to move after having children. To proxy for the likelihood of fetal or neonatal mortality I use the mother's number of prior fetal deaths. Finally to accord for unfavorable family dynamics I include an indicator variable for whether or not the father lived in the home at the time of birth. The results of the Heckman specification are nearly identical to the OLS and fixed effects models.

5.4 Head Circumference and Birth Weight as a Continuous Measure

The definitions used in the main part of the analysis are consistent with the medical literature. However from a statistical perspective, it is interesting to investigate what data artifacts can be hidden by creating a categorical dummy by conditioning on two variables. This is of particular concern since no other paper in this literature has utilized head circumference nor used birth weight standardized by gestational age and gender as regressors in a health production function. In this section, I use kernel-weighted local linear smoothing techniques to further explore the relationships between IQ and head circumference, birth weight, and gestational age. I find that the relationship between IQ and size at birth appears to be driven completely by head circumference across the distribution of the explanatory variables. Birth weight has little or no impact on IQ when controlling for head circumference.

The first set of results is found in Figure 3. The top graph depicts the relationship between IQ at age 4 and head circumference at birth. These graphs are constructed by local linear smoothing after an orthogonal, linear projection off of the standard set of controls and birth weight. That is, I estimate the following equation:

$$IQ_i = \alpha + g(HC_i) + \beta BW_i + \gamma X_i + \epsilon_i \tag{3}$$

where IQ_i is child i's IQ score at ages 4, BW_i is the birth weight, and X_i is the standard set of controls discussed in Section 3. The function $g(HC_i)$ is estimated using local linear smoothing.

At smaller head circumferences, the graph shows a gradual, and nearly constant, increase in IQ as head circumference increases. The effect tapers off at the mean IQ level after head circumference increases beyond the mean for asymmetrically growth restricted infants. That is, at both age four, the marginal effect (slope of the estimated graph) of a one centimeter increase in head circumference is high at small head circumferences and gradually decreases reaching zero around the mean head circumference. There is some evidence that the marginal effect is negative for very large head circumferences. This may reflect complications that are associated with macrocephaly (abnormally large head) or megalencephaly (abnormally large brain) such as chromosomal abnormalities or mental retardation. The OLS regression line (represented by the dashed line with 95% confidence interval shown as the shorter dashed lines) is displayed for comparison.

Figure 3 also shows two graphs of the effect of head circumference on IQ across the dis-

tribution of birth weight and gestational age. The goal of this analysis is to see if the effect of head circumference changes based on other characteristics of the child at birth that have been considered important in previous literature. The graphs are constructed by estimating the coefficient of a linear regression of IQ on head circumference with a standard set of controls separately for different categories of birth weight and gestational age.[11] The estimated marginal effect of head circumference on IQ is positive and relatively constant across the distribution of both birth weight and gestational age (the few large outliers on the left portion of the distribution are from highly imprecise estimates due to small sample size). This implies that the effect of head circumference is likely independent of other anthropometric factors at birth. This quells concern about misspecification of earlier models. It also shows evidence that the effect of birth size on cognitive ability is driven by head (brain) size and not birth weight, per se.

Further evidence that the relationship between birth size and cognition is driven by head circumference is found in Figures 4. The top graph is constructed in the same fashion as Figure 3, except in these graphs birth weight is the variable of interest. After controlling for the standard set of covariates and head circumference,[12] birth weight seems to have no affect on IQ at any part of its distribution. The marginal effect oscillates around zero without ever becoming significantly different. Furthermore, the bottom two graphs show that this remains true across the distribution of head circumference, as well as the distribution of gestational age. This evidence suggests that prior literature that finds an impact of birth weight on educational outcomes may simply be picking up bias from the correlation between birth weight and the omitted variable, head circumference. In fact a simple regression of birth weight on head circumference shows that a 1 cm increase in head circumference increases birth weight by more than 250 grams, and the raw correlation coefficient between the variables is 0.77. However, even after controlling for head circumference, it is clear from the top left graph of Figures 4 that the OLS regression estimate (again represented by the dashed line) is not representative of true empirical relationship.

In conclusion, looking at the distributional effects of birth weight and head circumference reveals that head circumference, and not birth weight, appears to be the most important variable in determining childhood cognitive ability. However, the effect only seems relevant for the lower tail of the head circumference distribution. These results also clearly show the

[11] For birth weight, a separate regression is run for 100 gram categories of birth weight starting at 600 grams. For gestational age, separate estimates are obtained for each week of gestation starting at 26 weeks. The gestational age regressions include birth weight as a control variable.

[12] Given the shape of the top graph in Figure 3, head circumference is included as a piece wise function that allows for different slope after 33 cm.

need for more widespread collection of head circumference measures in birth records. Further exploration of these distributional effects could be a promising avenue for future research.

6 Physical Health & IUGR

All current literature—from both economics and medicine—points to a strong relationship between IUGR and health outcomes in later life. However due to differences in fetal growth patterns, asymmetric growth restriction and symmetric growth restriction may result in very different outcomes with regard to physical health. It may be the case that the less severe insult to growth in asymmetrically growth restricted fetuses results in a rapid return to normal health as neonates. On the other hand, asymmetric growth restriction could also result in a lasting negative impact on health. This distinction is important for two reasons: First, showing that there is little difference in physical health between asymmetric and symmetric growth restriction provides evidence that differences in cognitive ability (shown in Section 5) are not a reflection of differences in physical health. Second, providing evidence that the asymmetric subtype is similarly deficient in physical health means that asymmetric growth restriction has the potential of providing a bias free estimate of the effects of physical health on human capital development.

6.1 Methodology

I test the possibility that symmetrically and asymmetrically growth restricted infants have different physical health outcomes using the following empirical model:

$$H_i = \alpha_0 + \beta_1 I_{asym} + \beta_2 I_{sym} + \gamma X_i + \epsilon_i \tag{4}$$

Where H_i is a measure of physical health (or health problems) during childhood, I_{asym} and I_{sym} are as described in Equation 1, and X_i is a vector of controls.

For measures of physical health at birth, I utilize data on 5 minute APGAR scores and congenital malformations detected by age 7. First I employ an indicator for an APGAR score below 7 out of 10 (a low to marginal score).[13] In these data, a congenital malformation is defined as a "gross physical or anatomic developmental anomaly" that was either present at birth or was detected by age 7. Using this, I construct binary variables for whether any congenital malformation is present at age 7 and whether any major congenital malformation

[13]Similar results are found when the indicator is for a low score (below 4) or using multinomial logit.

is present at age 7.[14] Concerning outcomes of childhood health, the CPP contains data on vision, hearing, and speech evaluations, as well as data on the presence of seizures. For vision, hearing, and speech I use a binary indicator for an abnormal screening. These measures are also useful because they are more clearly related to educational and labor market success.

To control for demographic and socioeconomic differences that could affect both the onset of growth restriction and childhood health, logistic regressions include control variables for the mother's age and the mother's age squared, the mother's height, 6 indicator variables for the mother's education level, an indicator for whether the mother works, family income, the number of prenatal visits and the number of prenatal visits squared, indicator variables for gestational age, as well as indicators for race, gender, year of birth, and location of birth. For continuity with Section 5, all observations with missing IQ scores are excluded from this analysis.

It has been shown that logit models do not preform well with rare events (like congenital malformations), which can lead to underestimated probabilities even with large samples (King and Zeng 2001). To mitigate this problem, results are estimated using rare events logit estimation from King and Zeng (2001). No significant differences are found when using alternative methodologies.

There is significant medical evidence that there is reverse causality between health and IUGR. As stated in Section 2, IUGR may be the result of insults that originate from the mother, placenta, or fetus. Fetal insults may include a congenital malformation or birth defect that was present from conception. If this is the case, then coefficients estimated from Equation 4 are biased due to the fact that growth restriction may be the result of a major birth defect. This fact has been almost completely ignored in the empirical literature to date. This is probably due to the difficulty of dealing with endogeneity with regard to growth restriction. Instrumental variables are not particularly promising because there are almost no variables that predict IUGR that do not also predict congenital malformations. This is particularly problematic when the desired goal is to decompose IUGR into its subtypes for the analysis. The results of these models should, therefore, be viewed as associations or correlations rather than causal estimates. The main value of these results is to infer if differences in physical health may account for the difference in cognitive ability.

Equation 4 is estimated using rare events logistic regression on the binary outcomes described above. The estimated odds ratios β_1 and β_2 are presented in Table 4.[15] Each

[14] Whether a malformation is considered major or minor was determined by the authors of the CPP.

[15] Odds ratios are presented rather than marginal effects because the health conditions studied are relatively rare events; so the odds ratios are easier to interpret in this case.

column shows the increased odds of being diagnosed with a particular health problem if the child was asymmetrically growth restricted or symmetrically growth restricted compared to children who did not suffer from fetal growth restriction. The top rows of the table show results for in-sample definitions, and the bottom rows show results for out-sample definitions. The first column shows estimates for when the dependent variable is the presence of any a low or moderate APGAR score; the second column shows estimates for the presence congenital malformation; the third column shows estimates for the presence of a major congenital malformation; the fourth column show results for an abnormal visual screening; the fifth column shows results for an abnormal hearing screening; the sixth column shows results for an abnormal speech evaluation; and in the final column are estimates for the presence of non-febrile seizures. Below the estimates (both in-sample and out-sample) are the number of observations and the p-value from the F-test for equal coefficients of asymmetric and symmetric growth restriction.

The estimates show that both asymmetric and symmetric growth restriction increase the likelihood of each of the poor health indicators, and the magnitudes of the coefficients are statistically indistinguishable. These results show that having either subtype of IUGR is associated with a 2 to 3 fold increase in the likelihood of having a low APGAR score. Both subtypes increase likelihood of having any congenital malformation by over 1.23 to 1.37 times. The likelihood of having any major malformation is approximately 1.5 times higher when IUGR is present than when it is not. Results for abnormal vision, hearing, and speech screening and the presence of non-febrile seizures show a similar pattern of both subtypes being associated with increased risk of poor health. The distance between the coefficients for asymmetric and symmetric restriction is more visible in these estimates, but remains statistically indistinguishable in nearly ever specification. Asymmetric coefficients are marginally statistically distinguishable from symmetric coefficients in 3 specifications: the in-sample estimates of having a major congenital malformation (at the 95 percent level), the in-sample estimates of having an abnormal speech evaluation (90 percent level), and the out-sample estimates of an abnormal hearing evaluation (90 percent level), which actually shows that children that suffer asymmetric growth restriction are *less* likely to have an abnormal hearing screening. However, in each instance the corresponding in-sample or out-sample estimates are not statistically distinguishable. As with the results on cognitive outcomes, using a series of different in-sample and out-sample classifications does little to affect the above results. Signs and magnitudes remain vary similar regardless of the classification use. Tables with these results are available upon request.

These results demonstrate that IUGR is a serious physical health threat to a child re-

gardless of the possibility of brain sparing. The strong effect of both subtypes lends credibility to the fetal origins hypothesis, as these conditions have a high probability of causing health complications when these children become adults. Furthermore, the results shown here show strong evidence that asymmetric growth restriction is significantly associated with poor physical health, and there is no convincing evidence to suggests that the health shock is less severe than with symmetric growth restriction. Combining these results with those in Section 5, it appears that asymmetric growth restriction is a severe health shock at birth that leaves cognitive faculties largely in tact; whereas symmetric growth restriction severely affects both physical health and cognitive ability. That is, because asymmetric growth restriction shows strong pattern of decreased physical health and little evidence of decreased cognitive function, it is a reasonable candidate for an unbiased test of the effect of physical health on educational attainment.

7 The Causes of Asymmetric and Symmetric Growth Restriction

The final goal of this study is to investigate the factors that are potentially responsible for asymmetric and symmetric growth restriction. This analysis is important for two reasons. First, understanding the effects conditions in utero have on IUGR can inform potential policy interventions, as well as future research. Second, the availability of data that allows researchers to decompose IUGR into its subtypes using anthropometry is currently quite limited. Therefore, finding other, more commonly collected, variables that can be used to discriminate between asymmetric and symmetric growth restriction is valuable for future research. To this end, multinomial regression models are estimated for the presence of the subtypes of IUGR.

The CPP allows for a test of the differential impact of three major causes of growth restriction. The first test is the association between the timing of the growth insult (whether early or late onset) using data on the stages of pregnancy during which major infections were present in the mother. An early growth insult is defined as contracting a major viral, bacterial, or fungal infection during the first or second trimester, and a late growth insult is defined as contracting a major infection during the third trimester only. Theory suggests that infections early in pregnancy should cause symmetric growth restriction and infections late in pregnancy may be responsible for asymmetric growth restriction.

The second factor tested is the effect of the smoking behaviors of the mother. Since

the majority of CPP data were collected before the Surgeon General's report on the health risks of smoking in 1964, a significant number of mothers (approximately half) smoked while pregnant. Smoking behavior is grouped into three categories: light smokers, moderate smokers, and heavy smokers. Light smokers are defined as consuming 10 or fewer cigarette per day; moderate smokers consume greater than 10 cigarettes but less than 20 cigarettes per day, and heavy smokers consume more than 20 cigarettes (the number in a standard pack) per day. It is unknown if smoking should differentially affect the subtypes of growth restriction since there are several proposed mechanisms concerning how smoking affects fetal growth (e.g. low blood-oxygen levels, vasoconstriction, toxicity, etc.).

The third factor is whether the child's mother was diabetic. The constructed binary variable equals one if the mother was diabetic before becoming pregnant. I also include a variable that indicates if the mother had gestational diabetes only. It is hypothesized that diabetic mothers are more likely to have symmetrically growth restricted babies, but less likely to have asymmetrically growth restriction babies since the growth insult is present from conception.

Table 5 shows the relative risk ratios of becoming symmetrically or asymmetrically growth restricted—using absence of growth restriction as the base outcome—given the potential growth insults listed above. Contracting an infection early in pregnancy makes symmetric growth restriction 1.17 to 1.2 times more probable than normal growth. An infection in the third trimester appears to decrease the probability of symmetric growth restriction and increase the probability of asymmetric growth restriction (both relative to normal growth), although all of these coefficients are estimated imprecisely. Coefficients on smoking are estimated with high precision and show that consuming cigarettes while pregnant increases the probability of growth restriction 2 to 3 fold. To put this in perspective, if IUGR occurs in approximately 10 percent of all pregnancies, then these results suggest that 20 to 30 percent of mothers that smoke will have a growth restricted baby. There is little evidence that smoking is associated with one subtype more than the other. This may be due to the inability to know if the mother changed her smoking habits during pregnancy. A symmetrically growth restricted child is approximately 1.9 times more probable with a diabetic mother (although the coefficient is small and insignificant for the out-sample definition). Interestingly, a diabetic mother is less than a third as likely to have an asymmetrically growth restricted baby than a non-IUGR baby. This means that symmetric growth restriction is about 6.5 times more probable than asymmetric growth restriction for a diabetic mother. Gestational diabetes does not appear to influence fetal growth, thus the estimated coefficient is not reported.

These results are interesting for two reasons. First, the evidence suggests that the vari-

ables I created to capture symmetric and asymmetric growth are consistent with growth patterns suggested by the medical literature. Second, since the timing of a growth insult and maternal diabetes are much more likely to be available in a dataset than head circumference, these measures may be useful in distinguishing between the subtypes in future work.

8 Discussion & Conclusion

This paper reexamines the underlying causes of the relationship between birth weight and human capital development. Using information from the medical literature, two distinct classifications of low birth weight infants are identified: asymmetric and symmetric growth restriction. Both types of growth restriction are shown to negatively impact childhood health. However, the symmetric type is shown to also have a severe negative effect on cognitive ability (measured by childhood IQ score), while the asymmetric type typically is shown to leave cognitive faculties mostly unchanged. Although the notion that children who experience asymmetric growth restriction have decreased cognitive function cannot be completely dismissed by these results, we can conclude that the effect is small. Thus, utilizing this difference in cognitive ability between the subtypes of IUGR infants has the potential of providing unattenuated estimates of the effect of early life health on education and labor market outcomes. At a minimum, these results demonstrate that estimates of the effect of birth weight on educational and labor market outcomes currently found in the economics literature are not measuring the true effect of health on adult labor market outcomes, but rather they likely measure the effect of cognitive impairment on labor market outcomes and education. Likewise, "quasi-experimental" studies that implicitly treat all shocks to the fetal environment homogeneously could be misleading. For example, evidence suggests that interventions that only focus on improving health and nutrition in later pregnancy are unlikely to be sufficient to overcome the achievement gap. Finally, these results have broad implications for future research in economics, medicine, and public policy concerning infant health and pregnancy interventions.

8.1 Implications for Economics

These results may help explain several inconsistencies in the economic literature on neonatal health and human capital development. One of the anomalies associated with the current research on birth weight and human capital development is that research shows that gains to

childhood health from increased birth weight are largest at the lower end of the birth weight distribution (Almond, Chay, and Lee 2005), whereas the effects of birth weight on educational attainment are largest at birth weights above 2500 grams (Royer 2009). These facts are inconsistent with the idea that birth weight is a measure of physical health, which drives educational attainment. However part of this paradox could be explained by the differential impacts of asymmetric and symmetric growth restriction. It is possible that the results found in Royer (2009) are really picking up a crude difference between symmetrically growth restricted infants and normal birth weight infants (and asymmetrically restricted infants). In Section 5.2 we found a slight difference in mean birth weight between symmetric IUGR and asymmetric IUGR infants. Specifically the mean birth weight for symmetric growth restriction tends to be around 2300 grams; whereas the mean birth weight for asymmetric growth restriction is generally around 2500 grams (both depending on the exact classification used). That is, if the sample is split in two using a 2500 gram cutoff, it is likely that the majority of those below 2500 grams are symmetrically growth restricted, and those above 2500 grams are asymmetrically growth restricted and normal birth weight. Therefore, it is plausible that the finding of Royer (2009) is not due to a difference in birth weight per se, but a difference in infants with brain growth restriction and those without brain growth restriction.

Another paradox in the literature that may be partially explained by the recognition of the difference between asymmetric and symmetric growth restriction is the fact that the omitted variable bias that results from leaving out family fixed effects is large for childhood outcomes but disappears for adult outcomes (Oreopoulos et al. 2008; Black, Devereux, and Salvanes 2007). If the mechanism by which adult outcomes are affected by birth weight is through childhood health, why do the results not present with the same bias? To understand why this change occurs, one must first understand the difference in data on childhood outcomes and adult outcomes. Data on childhood outcomes are almost entirely related to physical health; whereas data on adult outcomes used in economics literature (such as income or education) are highly correlated with the cognitive ability of the individual. Given the results of this paper, we know that the effect of birth weight on an outcome correlated with cognitive ability is attenuated due to the mixture of the effects of asymmetric and symmetric growth restriction. Furthermore, this bias moves in the opposite direction of the bias due to omitting family factors. Thus, the bias from omitting family factors seems to disappear; when, in fact, it is merely being mixed with a countervailing bias. Childhood outcomes do not exhibit this problem because the data used for childhood outcomes are typically measures of physical health, for which the effects of asymmetric and symmetric growth restriction are

quite similar. Thus the coefficients are not biased in the same fashion by failing to distinguish between the subtypes.

Finally, these results may help explain why largest negative effects to human capital are generally the result of a poor fetal environment in early pregnancy (see Almond, Edlund, and Palme [2009] and Almond and Mazumder [2011]), whereas the largest improvement to birth weight occurs with interventions in the third trimester (Almond, Hoynes, and Schanzenbach 2011). This is inconsistent with the idea that birth weight measures important changes in the fetal environment. However, this can be explained by the different timing of onset of symmetric growth restriction and asymmetric growth restriction, which is shown in Section 7. Changes in the early pregnancy environment are likely to influence the occurrence of symmetric growth restriction. Thus, factors that negatively affect the fetal environment in early pregnancy will be the most likely to decrease cognitive function and have greater effect on human capital measures. Meanwhile changes in the late pregnancy environment are likely to only affect the outcome asymmetric growth restriction, even if it results in large changes in birth weight.

8.2 Implications for Public Policy

It is important to consider how public health policy can be reevaluated in light of its results. The decomposition of growth restricted newborns into asymmetric and symmetric subtypes reveals that not only do these groups have large differences in cognitive function, but also that they may be caused by different factors at different points in the pregnancy. An example of why this distinction matters is policy intervention, like the Food Stamp Program (FSP). The FSP has recently been linked to improved birth outcomes, as measured by birth weight improvements in the third trimester of pregnancy (Almond, Hoynes, and Schanzenbach 2011). Given the current literature on birth weight and human capital development, improvements to birth weight are likely evaluated as improving future educational attainment and earnings as well. However, given the results of this paper, this supposition seems problematic. While increasing birth weight in the third trimester may indeed improve the physical health of the child, damage done to brain growth due to symmetric growth restriction has already begun before the third trimester begins. Thus the social gains from increasing birth weight through the FSP are likely overstated.

On the other hand, programs that naturally lend themselves to earlier intervention in pregnancy may currently be undervalued. Medicaid, for example, encourages women to get early prenatal care by lowering the cost of doing so (Currie and Grogger 2002). Early

prenatal care can increase the likelihood of proper nutrition throughout pregnancy and allows for early detection of illnesses that have detrimental effects on the growing fetus, such as anemia, pregnancy induced diabetes, and preeclampsia. Interventions in the early stages of pregnancy are more likely to prevent symmetric growth restriction, and thus have larger impacts on cognitive-based human capital development in the population. However, since there are currently no studies showing the impact of Medicaid's introduction on educational attainment, the effects of this program could currently be undervalued. Similar comments could be said about the evaluation of the WIC program.

8.3 Future Research

One could argue that the current literature simply estimates a reduced form effect of birth weight on education and the distinction between asymmetric and symmetric growth restriction can be ignored since head circumference and birth weight are highly correlated. Indeed, whether the mechanism at work is through changes in IQ or health does not change the fact that increasing birth weight improves adult outcomes. However the subtypes are associated with different causes and different timing of onset, which calls into question the value of reduced form estimation and increases the value of understanding the biological mechanism behind the change. As the example scenarios described above illustrate, ignoring the heterogeneity that exists in low birth weight infants could result in expensive and ineffective policy interventions.

Future research should focus on further exploration and utilization of data. First, and perhaps most importantly, is the possibility of using asymmetric growth restriction to estimate the effect of physical health on education. This paper shows that asymmetric growth restriction is associated with a significant decrease in childhood health at age 7, and that these effects are similar to the effects of symmetric growth restriction (the sum of these effects is what has typically been estimated in the literature). Since brain development and growth are generally spared in the case of asymmetric growth restriction—as evident by the results contained in the above analysis—this presents the opportunity for the unbiased estimation of the effects of health on education and labor market outcomes in later life. This paper's reach is limited because in the data utilized subjects are only followed until age 8. Obtaining estimates of the effects of asymmetric and symmetric growth restriction using a dataset that contains information about completed education as an adult or any labor market outcomes could be valuable in explaining the mechanisms of human capital development further. Therefore, one important implication of this paper this that head circumference

needs to become a standard measurement collected with birth data. However, since the current reality is that very few datasets contain multiple anthropometric measurements, the results of Section 7 could be useful. I find support for the hypothesis that symmetric growth restriction onsets early in pregnancy (first two trimesters) and that asymmetric growth restriction onsets late in pregnancy (third trimester). Additionally maternal diabetes appears to be much more likely to cause symmetric growth restriction than asymmetric growth restriction. Therefore, separating a growth restricted sample by the timing of the potential cause or by whether the mother is a diabetic are promising avenues to explore.

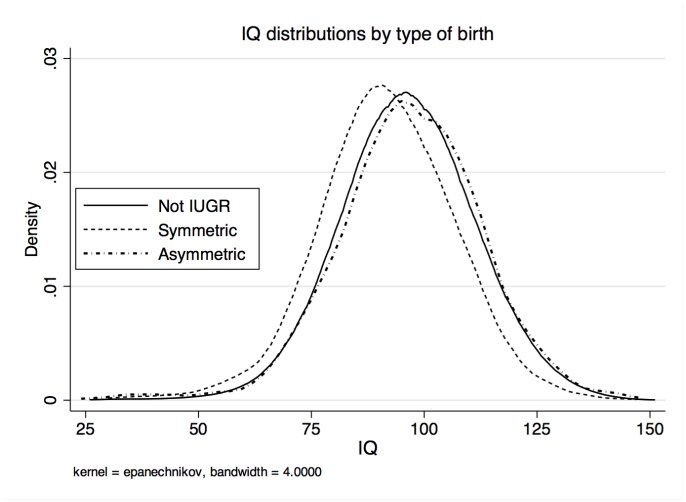

Figure 1: IQ Distributions by Growth Type

$$
\begin{aligned}
(A) \quad LBW &= f(\text{Behavior}, X) \\
(B) \quad H_i &= f(LBW, X) \\
(C) \quad H_{i+1} &= f(H_i(LBW), X) \\
(D) \quad \text{EDUC} &= f(H_{i+1}(H_i(LBW)), X)
\end{aligned}
$$

Figure 2: Pathway from Birth Weight to Education

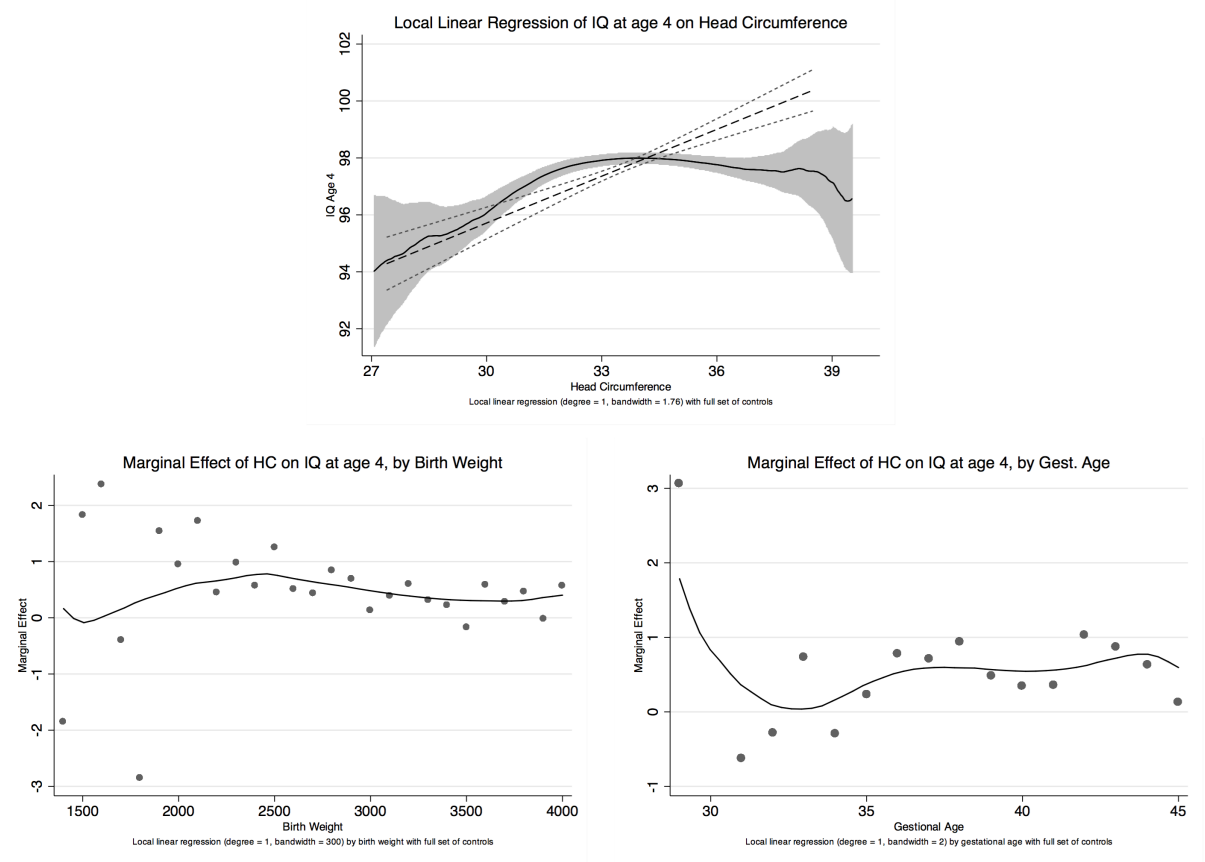

Figure 3: The Relationship Between Head Circumference and IQ

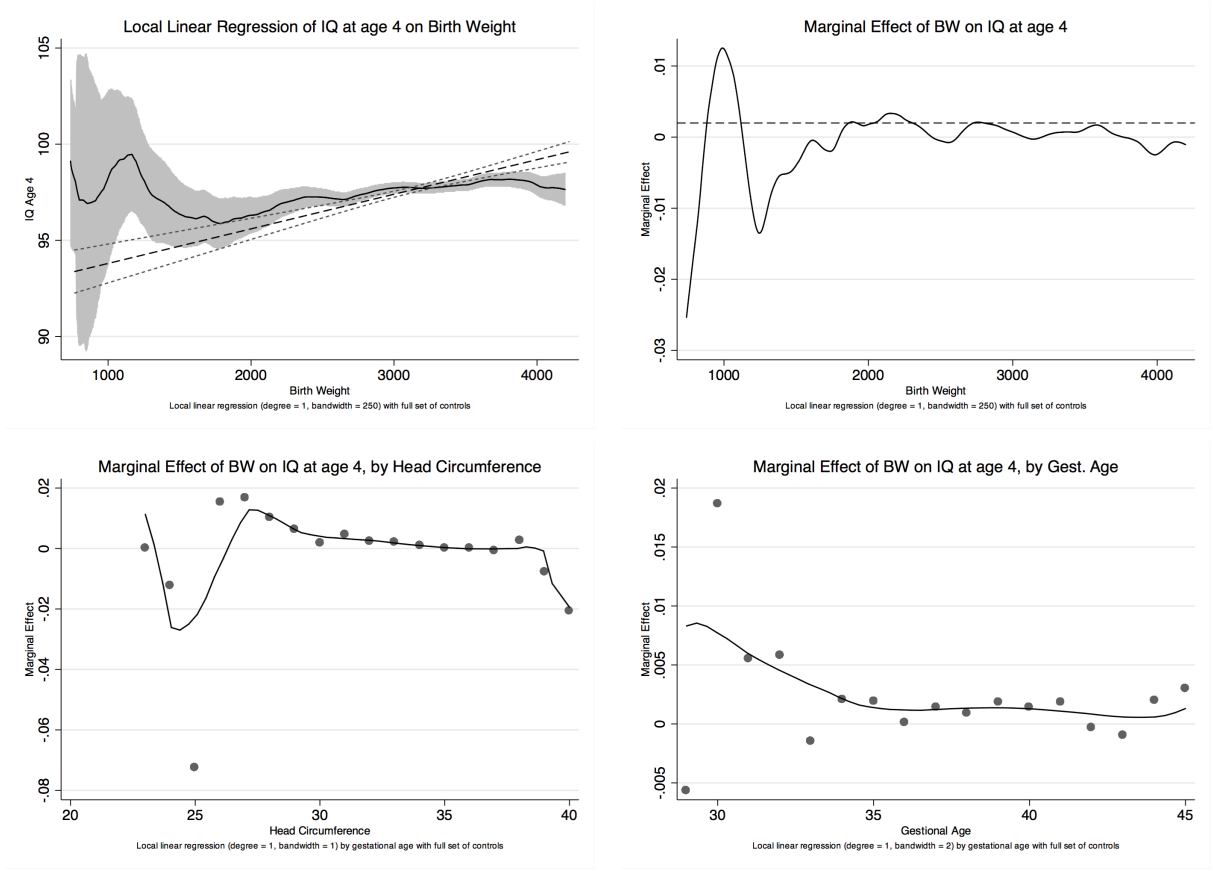

Figure 4: The Relationship Between Birth Weight and IQ

Table 1: Summary Statistics

	mean	sd	min	max	N
Outcome Variables					
IQ at age 4	97.72	16.62	25	172	34,641
IQ at age 7	95.94	14.96	26	153	37,003
Congenital malformation*	0.277	—	0	1	47,011
Major congenital malformation*	0.158	—	0	1	47,011
Visual Screening Abnormal*	0.218	—	0	1	37,570
Speech Screening Abnormal*	0.018	—	0	1	37,421
Hearing Screening Abnormal*	0.032	—	0	1	37758
Seizures*	0.018	—	0	1	38190
Infant Characteristics					
Birth weight (g)	3152	554	482	5613	47,019
Gestational age (weeks)	39.27	2.71	26	45	47,019
Head circumference (cm)	33.64	1.68	16	46	47,019
C-H length (cm)	49.79	2.87	20	63	46,799
Black*	0.466	—	0	1	47,019
Female*	0.493	—	0	1	47,019
Mother Characterstics					
Mother's age	24.22	6.02	11	49	47,019
Mother's height	63.54	2.69	40	80	47,019
Prenatal visits	8.77	4.06	1	35	47,019
Mother smokes*	0.478	—	0	1	46,661
Mother diabetic*	0.012	—	0	1	46,774
Preeclampsia*	0.155	—	0	1	47,019
Mother work*	0.144	—	0	1	47,019
Mother married*	0.773	—	0	1	47,019
Mother single*	0.146	—	0	1	47,019
Mother's Education					
$\leq$ 7 yrs*	0.093	—	0	1	47,019
Grade school*	0.079	—	0	1	47,019
Some high school*	0.388	—	0	1	47,019
HS graduate*	0.302	—	0	1	47,019
Some college*	0.073	—	0	1	47,019
College grad. or higher*	0.045	—	0	1	47,019
Family Income					
No income*	0.003	—	0	1	47,019
1,999 or less*	0.133	—	0	1	47,019
2,000-3,999*	0.408	—	0	1	47,019
4,000-5,999*	0.226	—	0	1	47,019
6,000-7,999*	0.099	—	0	1	47,019
8,000-9,999*	0.035	—	0	1	47,019
10,000 or more*	0.024	—	0	1	47,019

* Binary variables (0/1).

Table 2: Summary of IUGR Variables

	Mean	Count	N	Mean BW (g)	Mean HC (cm)
In-Sample Definitions					
Asymmetric	0.031	1480	47019	2493	33.23
Symmetric	0.073	3422	47019	2263	31.05
Out-Sample Definitions					
Asymmetric	0.024	991	42009	2480	33.34
Symmetric	0.081	3391	42009	2296	31.27
Matched BW Definitions					
Asymmetric	0.012	602	47019	2393	33.48
Symmetric	0.091	4300	47019	2324	31.46

All variables binary (0/1). For in-sample variables, intrauterine growth restriction is defined as having a birth weight below the 10th percentile of the CPP data adjusted for gestational age, race, and gender. Asymmetric growth restriction is defined as being IUGR and having a head circumference at or above the 10th percentile of the CPP data adjusted for gestational age, race, and gender or head circumference. Symmetric growth restriction is complimentarily defined as being IUGR with head circumference below the adjusted 10th percentile. For out-sample variables, IUGR is defined as birth weight below the 10th percentile adjusted for gestational age, race, and gender according to Hoffman et al. (1974). The difference symmetric and symmetric growth restriction is determined by 10th percentile of head circumference adjusted for gestational age and gender according to Miller and Hassanein (1971). For matched birth weight, IUGR is defined as above. Asymmetric growth restriction is defined as having a head circumference at or above 90th percentile for one of the 200 gram birth weight categories. Symmetric growth restriction is complimentarily defined as having a head circumference below the 90th percentile for one of the 200 gram birth weight categories. Quartile cutoffs are calculated using the CPP data

Table 3: Regression Results of Fetal Growth Restriction on IQ

	OLS		Fixed Effects	
	Age 4	Age 7	Age 4	Age 7
In-sample				
Asymmetric	-1.079**	-0.838**	-1.271	-1.684
	(0.45)	(0.40)	(1.36)	(1.21)
Symmetric	-4.649***	-3.705***	-4.089***	-4.708***
	(0.34)	(0.28)	(1.03)	(0.92)
N	31378	31378	6915	6915
R^2	0.31	0.33	0.04	0.03
P-value for $\beta_S < \beta_A$	0.000	0.000	0.026	0.010
Out-sample				
Asymmetric	-0.900	-0.932*	1.175	-1.936
	(0.56)	(0.48)	(1.68)	(1.33)
Symmetric	-4.416***	-3.771***	-3.221***	-3.396***
	(0.33)	(0.28)	(1.03)	(0.94)
N	28142	28142	6232	6232
R^2	0.31	0.33	0.05	0.03
P-value for $\beta_S < \beta_A$	0.000	0.000	0.004	0.155
Matched BW				
Asymmetric	-1.430**	-0.922	-0.035	-1.401
	(0.72)	(0.65)	(2.12)	(1.91)
Symmetric	-3.859***	-3.098***	-3.605***	-4.061***
	(0.30)	(0.25)	(0.95)	(0.84)
N	31378	31378	6915	6915
R^2	0.31	0.33	0.16	0.10
P-value for $\beta_S < \beta_A$	0.001	0.001	0.043	0.083

Robust standard errors in parentheses. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Controls for mother's age (as a quadratic function), the mother's height, indicators for marital status, indicators for the mother's and the father's education attainment, indicators for family income, socioeconomic status, the number of prenatal visits (as a quadratic function and indicators for gestational age, race, gender, year of birth, and location of birth. For fixed effects regressions, dummy variables control for the first born child and whether the first born child was IUGR, standard errors are clustered by family, and R-squared values are measure of explained within group variation.

Table 4: Rare Events Logit of Childhood Health on IUGR

	APGAR	Cong. Malf	Major C.M.	Vision	Hearing	Speech	Seizure
In-sample							
Asymmetric	3.272***	1.227***	1.351***	1.203***	1.170***	1.084***	1.542***
	(0.39)	(0.13)	(0.14)	(0.09)	(0.17)	(0.19)	(0.35)
Symmetric	2.421***	1.413***	1.598***	1.333***	1.489***	1.814***	1.714***
	(0.23)	(0.03)	(0.07)	(0.09)	(0.22)	(0.31)	(0.27)
N	31378	30940	30940	30952	31086	30818	31378
P-value for Equal β	0.114	0.118	0.048	0.134	0.279	0.092	0.665
Out-sample							
Asymmetric	2.607***	1.302***	1.495***	1.176***	0.963***	1.170***	1.188***
	(0.44)	(0.13)	(0.15)	(0.09)	(0.18)	(0.24)	(0.38)
Symmetric	2.150***	1.365***	1.521***	1.335***	1.634***	1.548***	1.671***
	(0.16)	(0.06)	(0.12)	(0.08)	(0.22)	(0.23)	(0.29)
N	28142	27748	27748	27760	27877	27635	28142
P-value for Equal β	0.373	0.638	0.888	0.237	0.070	0.179	0.319

Exponentiated Coefficients. Standard errors in parentheses. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Controls for mother's age (as a quadratic function), the mother's height, indicators for the mother's education attainment, indicators for family income, the number of prenatalvisits (as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.

Table 5: Multinomial Logit on the Causes of IUGR

	In-Sample	Out-Sample
Symmetric		
Early Infection	1.167**	1.204**
Late Infection	0.921	0.902
Light Smoker	1.798***	1.800***
Moderate Smoker	2.881***	2.952***
Heavy Smoker	2.833***	2.595***
Gest Diabetes	0.770	0.508
Diabetes	1.878***	0.980
Asymmetric		
Early Infection	0.931	1.032
Late Infection	1.025	1.119
Light Smoker	1.677***	1.595***
Moderate Smoker	2.497***	2.439***
Heavy Smoker	2.938***	2.626***
Gest Diabetes	0.514	0.680
Diabetes	0.302**	0.207**
N	45900	41075

Exponentiated coefficients (relative risk ratios). Controls for multiple gestation, maternal diabetes, eclampsia, mother's age (as a quadratic function), the mother's height, indicators for the mother's education attainment, indicators for family income, the number of prenatal visits(as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.
* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

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