The effects of asymmetric and symmetric fetal growth restriction on 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 relationship between health and education; the causal pathway—whether health causes education, education causes health, or health and education are jointly determined by a third factor—being the primary focus. 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).

A related, but divergent, set of literature has emerged which does not focus on 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. Without understanding the "black box" mechanism by which fetal programming occurs, policy interventions are little more than a shot in the dark.

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: symmetrical and asymmetrical. 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 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.

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 result 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 (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

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

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 restrictions that presents. 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

In asymmetrically growth restricted fetuses, the insult to fetal growth typically onsets late in the pregnancy. This subtype 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 the restrictive effects of poor nutrition may not present until the fetus becomes more

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

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 are 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).

To understand the potential cognitive differences between symmetrically growth restricted children and those asymmetrically growth restricted and normal birthweight, refer to Figure 1. 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.

2.2 Economics 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. Particular emphasis will be placed on studies that are concerned with cognitive development (of which there are few).

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 human capital development. In the decade that followed, many of papers revisited the question. Following the literature on labor market outcomes, the human capital development literature began controlling for genetic and family endowments, and using samples of siblings or twins with mother fixed effects to control for unobserved genetic factors became the preferred method of identification. Behrman and Rosenzweig (2004) are the first in the economics literature to use twin fixed effects. Using a measure of fetal growth (birth weight divided by gestational age), they find a significant impact on the length of schooling.[3]

Another innovation—this time in functional form—is made by Almond, Chay, and Lee (2005), who determine the effects of birth weight on infant health are non-linear using a series of dummy variables to categorically identify birth weight in 200 gram increments. This

[3]Royer (2009) later showed the fetal growth measure to be problematic. Since gestational age is measured with error, coefficients estimated using fetal growth are biased.

implies that if birth weight impacts human capital development through decreased health, the effect of birth weight on human capital development is also non-linear. Additionally, they find that the effect of birth weight on childhood health is much smaller when utilizing twin fixed effects, which supports the notion of biases resulting from the exclusion of family and genetic characteristics. This hypothesis is examined by Oreopoulos et al. (2008) using Canadian administrative data with twin and sibling fixed effects and Royer (2009) using administrative data from California with twin fixed effects only. The twin effects estimates of Oreopoulos et al. (2008) are generally not significant when using categorical dummies like Almond, Chay, and Lee (2005); however it is unclear whether this is due to a lack of true effect or a lack of power due to the small sample size. When estimates are obtained using a larger sample size including sibling fixed effects, the birth weight categories less than 3500 grams show a negative effect on the probability of reaching grade 12 by age 17 when compared to infants weighing more than 3500 grams. These effects also show significant non-linearities, which is consistent with Almond, Chay, and Lee (2005).

Royer (2009) tests for non-linearity by splitting her sample at a birth weight of 2500 grams. She finds that birth weight has a significantly larger effect on educational attainment at ranges above 2500 grams, which provides evidence that the impact of birth weight is indeed non-linear. However, a larger effect at the heavier portion of the distribution runs contrary to hypothesis that the mechanism through which birth weight affects educational attainment is purely through health—since Almond, Chay, and Lee (2005) found the lower end of the birth weight distribution caused the largest negative effect on health. This relationship does not persist when categorical dummies are used; estimates are essentially constant for all birth weight categories compared to infants born weighing greater than 2500 grams.[4]

The largest twin and sibling study to date is Black, Devereux, and Salvanes (2007). Like Oreopoulos et al. (2008) and Royer (2009), they estimate the effects of birth weight

[4]Very few of these estimates are statistically significant.

on both infant health and education and labor market outcomes. Instead of using birth weight dummies to account for non-linearities, they use logarithmic functional form. They find that a 10 percent increase in birth weight increases IQ score by approximately 6 points and increases the probability of graduating high school by nearly 1 percentage point. One of the more interesting observations of this paper is that the bias of the OLS estimates (observed by comparing the OLS estimates to the twin or sibling fixed effects estimates) is much larger for short-run (infant health) outcomes than for long-run (education and labor market) outcomes. This phenomenon is also true for Oreopoulos et al. (2008) and Royer (2009).

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 (Forthcoming)). 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. 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.

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 below.

Pathway from Birth weight $\Longrightarrow$ Education

$$(A) \quad LBW = f(\text{Behavior}, X)$$

$$(B) \quad H_i = f(LBW, X)$$

$$(C) \quad H_{i+1} = f(H_i(LBW), X)$$

$$(D) \quad \text{EDUC} = f(H_{i+1}(H_i(LBW)), X)$$

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 research 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 fails to answer two key questions: Can birth weight be considered an exogenous physical health endowment? And can changes in the fetal environment be used to explain whether the observed effect on human capital occurs via decreased

physical 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. 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 biased estimator of the effects of physical health on education. The bias can be thought of two ways. First, 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. Second, 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 under-stated, 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 (see 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. Results that show larger cognitive effects for insults to the early fetal environment (such as Almond, Edlund, and Palme (2009) and Almond and Mazumder (Forthcoming)) can be explained by the fact that symmetric growth restriction is early onset and results in large cognitive deficits even in early life. Royer's (2009) finding that the effects of birth weight on education are strongest for higher birth weights could be explained by a different mixture of asymmetric and symmetric IUGR infants. Symmetric IUGR infants are more severely growth restricted and tend to be lower birth weight than asymmetric IUGR infants. Thus, the higher birth weight group likely has a larger percentage of asymmetrically growth restricted infants than the lower birth weight group, and therefore is more likely to show positive cognitive benefits. Another anomaly that could potentially be explained by recognizing the heterogeneity in low birth weight infants is the difference in bias between short-run and long-run outcomes. Both Black, Devereux, and Salvanes (2007) and Oreopoulos et al. (2008) find a large bias in the short-run effects of low birth weight, but no such bias for long-run outcomes.[5] The lack of apparent bias on education and labor market outcomes could partially be due to failing to decompose the differing effects of

[5]Bias is measured by comparing OLS estimates to estimates that include twin fixed-effects.

asymmetric and symmetric IUGR. Since asymmetric IUGR has little or no effect on cognitive ability, it likely pulls down estimates for the average effect of low birth weight for long-run outcomes that are highly correlative with cognitive ability. On the contrary, both subtypes of IUGR exhibit similar poor health outcomes; thus short-run outcomes—which are typically measured in terms of physical health—are not affected by failing to decompose the estimates. This is discussed further in Section 6.

2.3 Medical Literature

The medical literature generally agrees that infants affected by IUGR are at greater risk for health and developmental problems into early childhood. Newborns that experienced growth restriction in utero are at increased risk of perinatal suffocation, are 20 times more likely to have congenital malformations, are nine times more likely to develop infections, and are more likely to be have hypoglycemia, low serum calcium levels in the blood, difficulty regulating body temperature, and respiratory distress. As children and adults, individual who experienced growth restriction in utero are at risk for permanently stunted growth, particularly if they were born preterm. There is also increased risk of developmental, behavioral, and cognitive problems (Levene, Tudehope, and Thearle, 2000; Martin, Fanaroff, and Walsh, 2005). The fetal origins hypothesis, or Barker hypothesis, famously linked asymmetric growth restriction to coronary heart disease in adulthood. Further studies have shown associations between poor fetal growth and adult hypertension and diabetes, although the academic debate continues over the reliability of these studies (Cunningham et al., 2009). Most medical literature centers around the collection of clinical data of infants with similar socioeconomic and demographic characteristics. The sample sizes are usually quite small, but closer to a controlled experiment.

Of particular interest is the current research on the cognitive effects of IUGR. Weisglas-Kuperus et al. (2009) examine the relationship between growth restriction and cognitive

function, as measured by IQ scores at age 19. This study is unique in that is recognizes potential difference for asymmetric and symmetric growth restriction, as well as neonatal growth restraint. They define IUGR as birth weight or length below less than 2 standard deviations below the mean, adjusted for gestational age and gender. A growth restricted infant is considered of the asymmetric type if its head circumference is not 2 standard deviations below the mean. Neonatal growth restraint is defined as being normal size at birth, but having weight or length less than 2 standard deviations below the mean at 3 months of age. Controlling for maternal age, parental education, gender, and race they find that symmetric growth restriction has the largest effect on IQ (nearly a 6 point decrease), followed by neonatal growth restraint (4.1 point decrease), and asymmetric growth restriction still reduces IQ by 3.7 points compared to the non-growth restricted group. From the confidence intervals provided, these values do not appear to be statistically different, however. They also find evidence that being preterm affects IQ. However, this study has a small sample size (n=556) and few control variables.

Another study that tests the effect of birth outcomes on IQ is Breslau et al. (1994). Controlling for maternal education, maternal IQ, and race, they find a decrease in IQ at age six of nearly 5 points for low birth weight infants relative to those of normal birth weight. Although the authors do not explore differences in symmetric and asymmetric growth restriction, they do observe a gradient relationship between birth weight and IQ—those with the lowest birth weight had lower IQs. A follow up study examining math and reading achievement scores at age 11 found this cognition shock to be persistent. The difference in test scores at age 11 is mostly explained by IQ score at age 6, which suggests the cognitive deficit is a lasting effect from early childhood, but not a compounded effect (Breslau, Johnson, and Lucia, 2001). This provides evidence that negative effects to cognitive ability in early life may explain differences in outcomes in later life.

Ekeus et al. (2010) examine the impact of gestational age rather than birth weight. They

use a large sample of Swedish birth records matched with cognitive test scores from military service. They find that gestational age predicts lower test scores in a gradient fashion—the largest effects are on those infants born very preterm (24-32 weeks gestation). According to another study, this effect may be due to decrease grey matter and white matter in the brain of the pre-term infant. Soria-Pastor et al. (2009) perform MRI scans on pre-term children that were born between 30 to 34 weeks of gestational age and compared them to a matched control sample. They find decreased volumes of grey and white mater in the preterm infants brains. They also show that grey matter reductions in certain regions of the brain are highly correlated with decreased IQ scores. Northam et al. (2011) confirm these results, finding that preterm infants have both lower white matter volume and IQ scores. These results are consistent with the hypothesis of symmetric growth restriction reducing the total cell number due to early onset growth injury.

My paper improves on this literature in several ways. First, I show the first empirical evidence of the "brain sparing" effect. That is, I show that there is statistically significant difference between the effect of symmetric growth restriction and the effect of asymmetric growth restriction on cognitive ability. Second, I test the robustness of these results to different definitions of asymmetric and symmetric growth restriction, and I show the results are also robust to using mother fixed-effects. Furthermore, my paper shows that the most important metric for determining cognitive ability is not birth weight or gestational age, but rather head circumference alone is a better anthropometric measure for predicting IQ.

3 Methodology: Testing the Brain Sparing Hypothesis

To evaluate the differential impact of asymmetric and symmetric growth restriction on cognitive ability, the following equation is estimated using OLS:

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

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. Cognitive ability is measured by 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 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, the number of prenatal visits (as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.[6] If IQ at age 7 is the dependent variable, X_i also includes a socioeconomic status score for the family when the child is 7 years old. When estimating Equation 1, the implicit assumption is that this set of controls is sufficiently correlated with unobserved genetic and home environment characteristics to act as a sufficient proxy.

Despite controlling for an extensive set of parental and socioeconomic characteristics, there remains the possibility of unobserved genetic characteristics or characteristics of the

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

home environment biasing coefficients if this unobserved heterogeneity is correlated with size at birth and cognitive ability. In the economics literature, this particular endogeneity problem is typically dealt with by using twin or sibling difference estimators. 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. Despite containing data on nearly 60,000 births, the CPP contains approximately 700 twin pairs and less than 10,000 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 involve with the study when having another child. If any of these family characteristics are correlated with anthropometric measures or IQ, which they likely are, then we have 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 appropriately sized child 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 mothers 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. Another difference in this specification is the removal of subjects with any congenital malformations. Malformations are not a major concern with the OLS estimates due to the large overall sample size.[7] 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. 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 difference in 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 cen-

[7] OLS estimates are unaffected by removing observations with major congenital malformations.

ters 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 the 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 42 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, length at birth, or gestational age are removed.[8] 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. 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. Although the medical literature provides some guidance, much of the literature concerns identifying growth restriction in utero using ultrasonography. Furthermore, data sets on neonates generally do not contain all the clinical data that the physician uses to assess a newborn infant. 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 differential between the symmetric and asymmetric subtypes.

There is some academic debate in the medical literature concerning the definition and

[8]When definitions of growth restriction do not include length, those with missing values for length at birth are not removed.

characteristics of asymmetric and symmetric growth restriction. The controversy includes debates about the proportion of asymmetric versus symmetric growth restriction, the causes of each subtype, which subtypes has worse health outcome, and whether there are truly two distinct subtypes. Since Campbell and Thoms (1977) published their study on growth restriction, a proportion of 70 percent asymmetric and 30 percent symmetric has been widely cited as the prevalence of each subtype of IUGR. However, several studies find half of all IUGR infants are asymmetrically restricted and half are symmetrically restricted (Martikainen, 1992; Delpisheh et al., 2008), a 40 percent asymmetric and 60 percent symmetric division is seen in another study (Salafia et al., 1995), and a 20 percent asymmetric and 80 percent symmetric ratio is found in two studies (Dashe et al., 2000; Nikkila, Kallen, and Marsal, 2007). It should be noted that most of these studies use different methodologies and cutoffs for differentiating between the subtypes of IUGR.

Although typically asymmetric growth is thought to be accompanied by a better prognosis than symmetric growth restriction, Salafia et al. (1995), Dashe et al. (2000) and Nikkila, Kallen, and Marsal (2007) all find asymmetrically growth restricted infants to have more health problems and health anomalies than symmetrically growth restricted infants. Martikainen (1992) finds little or no evidence of differences between the two subtypes with regard to developmental delays. Finally, despite the fact that the vascular mechanism for "brain sparing" has been clinically observed in both animal and human subjects (Uerpairojkit et al., 1996), there are potential challenges to the sparing hypothesis. Geva et al. (2006) find that infants that demonstrate growth impairment via ultrasound in the late second or early third trimesters, which is typical of asymmetric growth restriction, show signs of impaired memory function, and Roza et al. (2008) find that infants that exhibited the kind of vascular redirection in utero that is typical of asymmetric growth restriction showed signs of behavioral problems. Finally, Vik et al. (1997) finds no evidence of early or late onset of growth restriction using ultrasound diagnosis, and they find no evidence of larger head circumference

among asymmetrically growth restricted infants.

Many of the studies employ the ponderal index (=birth weight/length^3) to distinguish between the asymmetric and symmetric subtypes (Martikainen, 1992; Delpisheh et al., 2008; Vik et al., 1997). However, this measure being shown to be a worse predictor of IUGR than birth weight alone (Haggarty et al., 2004). Still others use a ratio of head circumference to abdominal circumference (Dashe et al., 2000; Nikkila, Kallen, and Marsal, 2007). However, it is unclear if this measure is appropriate since information about the absolute size of the head and abdomen is lost by using the ratio. Other common distinctions are head circumference or length below the 10th percentile or 2 standard deviations for symmetric IUGR.

Obviously 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.

Since definitive classifications are hard to come by, and there is no large-sample study that successfully demonstrates infants categorized by its method exhibit the expected characteristics from the literature, this paper adopts a "kitchen sink" approach. That is, I employ dozens of different classifications and show that the expected characteristics are exhibited by most of them, and I show that my results are consistent across most of the different classifications. I make no attempt to match a specific ratio of asymmetrically to symmetrically growth restricted infants due to a lack of agreement on such a ratio in the medical literature. However the different classifications employed have a good deal of variation in the ratio of asymmetric and symmetric, and this does little to affect the results. This paper's decompositions of restricted growth can be broken down into two main types: in-sample definition and out-of-sample definition. In-sample definitions are generated using percentile cutoffs created from the CPP data set. Out-of-sample definitions are generated using published standards

of birth anthropometry in the medical literature.

4.1.1 In-sample classification

Since the data set this paper employs is very large, it is reasonable to use in-sample measurements to create cutoff values between the general population and growth restricted infants and between asymmetrically and symmetrically growth restricted newborns. It is common in both the economics and the medical literature to define IUGR using only the neonate's birth weight. Typical cutoffs include low birth weight (LBW), which is medically defined as a birth weight less than 2500 grams, very low birth weight (VLBW), which is medically defined as a birth weight less than 1500 grams, and minus two standard deviations from the mean, which due to the normality of birth weight, typically includes those below the 3rd percentile. The most common medical definition for IUGR is birth weight below the 10th percentile for gestational age, which is the definition I employ in this paper. Infants are labeled as IUGR when their birth weight is below the 10th percentile of the sample controlling for race, for gender, and for one of four calculated gestational age categories.[9] However, since approximately half of the sample smoked during pregnancy—widely documented as a major cause of fetal growth restriction—it is likely that a much greater proportion than 10 percent of the sample experienced some form of growth restriction. Therefore an alternative definition of birth weight below the 20th percentile for gestational age is also tested.

Asymmetric growth restriction is characterized by the brain sparing effect, which leaves brain growth—and thus head growth—largely intact. Thus I define asymmetric growth restriction as being IUGR yet having a head circumference at birth at or above the 10th percentile (controlling for race, gender, and gestational age). I also experiment with using the 5th percentile as the cutoff. Symmetrically growth restricted infants are the remaining

[9]The categories are gestational age less than 32 weeks, from 32 weeks to 36 weeks, from 37 weeks to 40 weeks, and greater than 40 weeks. The main results of this paper are unchanged if values are instead calculated by actual gestation week. However, the values are slightly less precise due the small number of observations at some early gestational ages.

IUGR infants, with both birth weight and head circumference below the 10th (5th) percentile. Since symmetric growth restriction also affects skeletal growth–and thus body length–I also create definitions incorporating crown-heel length at birth. Symmetric growth restriction is defined as having IUGR and having crown-heel length in lowest 10th (5th) percentile as well as head circumference below the 10th (5th) percentile.

The preferred in-sample definition of asymmetric growth restriction is having birth weight below the 10th percentile for gestational age, gender, and race and having a head circumference at or above the 10th percentile for gestational age, gender, and race. Symmetric growth restriction has the same birth weight standard and a head circumference below the 10th percentile cutoff. The preferred in-sample definition that utilizes crown-heel length is having birth weight below the 10th percentile for gestational age, gender, and race; having a head circumference at or above the 10th percentile for gestational age, gender, and race; and having a crown-heel length at or above above the 10th percentile for gestational age, gender, and race for asymmetric growth restriction. Symmetric is complimentarily defined as head circumference and crown-heel length below the 10th percentile cutoff. The 10th percentile cutoff for birth weight is preferred because it is by far the most commonly used standard, and the common alternative—birth weight more than two standard deviations below the mean—is far too restrictive, particularly when defining growth restriction from within the sample.

4.1.2 Out-of-sample classification

Using within-sample growth standards to define IUGR and for decomposing IUGR into its subtypes could be problematic. The CPP data all come from urban areas. Thus, the black population and those of low socioeconomic status are over sampled. Furthermore, nearly half of the mothers in the CPP data smoked during pregnancy. Since smoking during pregnancy is linked to decreased birth weight, the CPP sample may be smaller than the

general population. To remedy any potential problems arising from in-sample classification, I use well known growth standard publications from 1960s and 1970s to calculate a second set of IUGR variables.

The preferred period birth weight data come from a 50 percent sample of all US births from 1968, reported by Hoffman et al. (1974). These data are preferred due to the large sample size, nearly 1.23 million births, the large variation in gestational ages, and the ability to get percentile data broken down by both gender and race. The second set of data are from the famous Colorado birth studies (Lubchenco et al., 1963; Lubchenco, Hansman, and Boyd, 1966). These data contain percentiles on birth weight, head circumference, and length collected from approximated 5,000 births from 1948 to 1961. However, these data are limited to caucasian infants. The third reference is Usher and McLean (1969). These data are collected for 300 caucasian new borns from 1959 to 1963 in Montreal, Canada. Although these data are somewhat limited, they have three distinct advantages. First, the data come from some of the exact years the CPP is collected. Second it contains data on birth weight, head circumference, and length broken down by gestational age. And third, the data can be used for robustness checks because it contains anthropometric measures broken down across birth weight categories in addition to gestational age. The final data used come Miller and Hassanein (1971). These data include information on head circumference and length by percentile collected from 1,692 new borns born in the University of Kansas Medical Center. Even though the sample size for these data is large, it is not as large as the Colorado birth data. However, the measurements collected from the Colorado study have been shown to be significantly smaller than those taken in later studies. This is likely due to the high altitude of Denver, which, as previously mentioned, can significantly impact growth. The Kansas data is noted to contain larger infants, on average, than the Colorado data, and is therefore preferred to the more widely used Colorado data.

The preferred definitions from out-of-sample sources utilizes the birth weight data from

Hoffman et al. (1974) and head circumference and crown-heel length standards from Miller and Hassanein (1971). These standards are chosen as preferred simply because they are formed using the largest samples (excluding the non-representative Colorado data).

For all of data from outside sources, symmetric IUGR is defined as having birth weight and head circumference (or birth weight, head circumference, and crown-heel length) below the 10th percentile for gestational age.[10] For all of the data except for the Montreal births, this can be done directly from the percentile information published in the respective papers. For the Montreal data, however, percentile breakdowns are not included, only mean and standard deviation by gestational age. Since birth weight is approximately normally distributed, the desired value is computed by subtracting the product of the standard deviation and the appropriate z-score from the mean to find the desired percentile for all three anthropometric measures.

One of the primary advances of the way in which I have defined both in-sample and out-sample classifications is that birth weight cutoffs are standardized 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.

When results from in-sample definitions conflict with those constructed using published standards, more weight will be given to estimates resulting from using published standards. This is done because the goal is to make the results as generalizable as possible, and the CPP is clearly not a nationally representative sample. Summary statistics for the preferred

[10]For these definitions the actual week of gestation is use since there are no sample size issue when using and outside data to define the cutoffs.

classifications are found in Table 2.

5 Results: Growth Restriction and Cognitive Ability

5.1 OLS Results

Results from estimating Equation 1 by OLS are found in Table 3. The top of Table 3 displays results from estimating within-sample definitions, and the bottom shows results from using outside sources to define the subtypes of growth restriction. Each table shows estimates using both IQ at age 4 and at age 7. Additionally, due to concerns about measurement error in crown-heel length, coefficients are estimated using categorizations defined without using this variable as well. The sample size, R-squared, and the p-value of the F test for equality of the asymmetric and symmetric coefficients are found below each set of results.

The first thing to notice about these results is the consistency across different models and definitions of the variables of interest. This speaks to the strength of the empirical relationship estimated by these equations. 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 and 5 points, which is approximately a third of a standard deviation. Although the coefficients estimated for asymmetric restriction are negative and sometimes statistically different from zero, the magnitude is typically much smaller than for symmetric restriction, ranging from less than 1 point to less than 1.4 points. The estimates of the effects of symmetric growth restriction are always nearly 3 to 4 times larger than those of asymmetric growth restriction. Each table contains the p-value for the F-test of equal slopes for symmetric and asymmetric growth restriction. The estimates are statistically different across all specifications. This evidence supports the hypothesis that in certain settings fetuses have the ability to compensate for a poor in utero environment and at least partially spare cognitive development. These

results lead us to two major conclusions: 1.) cognitive ability is unambiguously negatively affected by 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".

Reestimating these results using different cutoffs for the in-sample classifications or different references for the out-of-sample classifications does nothing to affect the results. Magnitudes remain relatively constant, and significance almost never changes. Tables of results for all the classifications can be found in the Tables 4 and 5, and a description of all alternative definitions can found in Figure 2.

5.2 Fixed Effects Results

Results from estimating the fixed effect model of Equation 2 are displayed in Table 6. The arrangement of the table is identical to the table of OLS results. These results corroborate the OLS results. Estimates for symmetric growth restriction are large (although somewhat reduced in magnitude from the OLS results) and statistically significant across both specifications for both IQ at age 4 and at age 7. Coefficients for asymmetric growth restriction are insignificant in all specifications. Asymmetric estimates are also of similar magnitude to the OLS results. The main difference between the OLS and fixed effects results is that the standard errors on the coefficients triple in size. This makes statistically distinguishing between the asymmetric and symmetric estimates via F-statistic much more difficult, which is reflected by only 2 out of 8 specifications showing statistical difference at a 90 percent confidence level.[11] However, both OLS and fixed effect specifications yield estimates of similar magnitudes and significance, despite many different potential identification issues, which provides support for the brain-sparing hypothesis. Like the OLS results, the results from

[11]We could also think about the one-tailed test, where the alternative hypothesis is that the magnitude of the coefficient of symmetric growth restriction is larger than the coefficient of asymmetric growth restriction by dividing the provided P-values by two. In this case, 5 of the 8 specifications are statistically different at the 90 percent confidence level.

the fixed effect estimation are robust to using several alternative definitions. Results from these estimations are in Tables 7 and 8.

5.3 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, asymmetrically growth restricted infants are low birth weight, and symmetrically growth restricted infant are very low birth weight. 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 232 grams for the definitions that do not include length and a difference of 318 grams for definitions that do incorporate length. For definitions constructed using published sources, asymmetric IUGR infants outweigh symmetric IUGR infants by 340 grams (for both definitions).[12] Furthermore, for all but one definition 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 all the analysis of this paper has done is to restate 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 the contribution of decomposing the effect of IUGR using head circumfer-

[12]It is worth pointing out that the differences in birth weight could be completely accounted for by the difference in head size. A 1 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)

ence, 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, the new definition of asymmetric growth restriction is having a head circumference and length greater than the 75th quantile for birth weight.[13] So instead of defining asymmetric growth restriction by the absence of small features (head circumference and length $>$ 10th percentile) for gestational age, it is now defined as having large features for birth weight.

For in-sample definitions, the 75th percentile is calculated from the CPP data using 10 birth weight categories.[14] Definitions are also created based on the proportionality standards for birth weight categories published in Miller and Hassanein (1971). 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. Based on the new definitions, symmetrically growth restricted infants now have slightly higher birth weights and gestational ages that asymmetrically growth restricted infants. Summary statistics of these definitions are in Table 9.

The results from Sections 5.1 are recalculated using these new definitions, and the results are displayed in Table 10. Despite the new definition, estimates for the IQ regressions are largely unchanged. The estimated impacts of asymmetric growth restriction are slightly larger in magnitude than before. However, they are still half the size of the effects of symmetric growth restriction, and the slopes remain statistically different with 95 percent confidence.

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,

[13]Because of the discrete nature of the head circumference data, identification of higher quantiles to use as cutoffs was not consistently possible for every birth weight category

[14]The categories begin at a <600 grams category and increasing in 200 gram increments (i.e. 600-800 grams, 800-1000 grams, etc.). This method is identical to that utilized by Miller and Hassanein (1971).

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.

5.4 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 is 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 are found in Figure 3. The first pair of graphs depict the relationship between IQ at ages 4 and 7 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:

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

where IQ_i is child i's IQ score at ages 4 or 7, 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, and is displayed in Figure 3.

The graphs show a gradual, and nearly constant, increase in IQ as head circumference increases. The second pair of graphs, which display the marginal effects from the graphs directly above them, provide further evidence of a linear effect. At age 4, the marginal effect is just below 1 (that is, an increase of 1 centimeter in head circumference increases IQ at age 4 by nearly 1 point) and remains constant across the entire distribution of head circumference. The marginal effect at age 7 tells a similar story, albeit with a slightly smaller magnitude and some evidence of a reduce effect on the tails of the head circumference distribution.

Figure 4 shows several graphs of the effect of head circumference on IQ across the distribution 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.[15] At both ages 4 and 7, 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 any 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 5 and 6. Graphs in Figure 5 are constructed in the same fashion as Figure 3, except in these graphs birth weight is the variable of interest. After

[15]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.

controlling for the standard set of covariates and head circumference, 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, Figure 6 shows 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.

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 cognition. Furthermore, the effect of head circumference on IQ appears to be approximately linear. These results also clearly show the 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.

5.5 Discussion of IQ & IUGR

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 "clean" 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 impair-

ment on labor market outcomes and education. Furthermore, these results provide evidence 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 make large strides in the direction of identifying the mechanisms behind the fetal origins hypothesis.

These results may also 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 the effect on childhood health and the effects on adult education are inconsistent with a mechanism of health alone. 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 weight above 2500 grams (Royer, 2009). That is, the portion of the birth weight distribution with the greatest improvement in health is the same as the area of the distribution with the more modest gains in education. Part of this paradox could be explained by the differential impacts of asymmetric and symmetric growth restriction. The effect of both subtypes on childhood physical health are similar (as I will show in Section 6); thus estimation of the effect of birth weight on these outcomes is largely unbiased. This explains why Almond, Chay, and Lee (2005) find the expected results. The results of Royer (2009) are puzzling, however, particularly given the results of Almond, Chay, and Lee (2005), unless one considers the difference between 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 5.3 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 2200 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 (this will be shown in detail in Section 6). 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 (Forthcoming)), 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 verified 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.

6 Physical Health and 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 3) are not the result 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 education in later life.

6.1 Methodology

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

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

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. Results are estimated using a logistic regression.

For measures of physical health, I employ data on congenital malformations at age 7. 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. Binary variables are constructed for whether any congenital malformation is present at age 7, whether any major congenital malformation is present at age 7, whether any cardiothoracic malformation is present at age 7, and whether any malformation known to be associated with IUGR is present at age 7. This variable is coded as 1 if there is a cardiothoracic defect, a musculoskeletal defect, a defect of the alimentary tract, or a defect of the liver, bile duct, or spleen.[16]

For a second set of health measures, I utilize data on vision, hearing, and speech evaluations, as well as data on the presence of seizures. These more specific measures are chosen because they are more clearly related to educational and labor market success. Outcome variables for the first three outcomes are binary indicators or abnormal evaluations for vision,

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

hearing, or speech.

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, 5 indicator variables for gestational age, as well as indicators for race, gender, year of birth, and location of birth.

One potential problem with using logistic regression in this context is the relative rarity of health problems present in the dataset. It has been shown that logit models do not preform well with rare events, which can lead to underestimated probabilities even with large samples (King and Zeng, 2001). This might lead a false conclusion that IUGR or one of its subtypes has no effect on childhood health when, in fact, it does. To mitigate this problem, all results are also estimated using rare events logit estimation from King and Zeng (2001).

Finally, 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 my 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.

6.2 Empirical Results

Equation 4 is estimated using logistic regression on the binary outcomes described in Section 6.1. The estimated odds ratios β_1 and β_2 are presented in Tables 11 and 12.[17] Table 11 shows results from using the congenital malformations, and Table 12 shows results using presence of vision, hearing, and speech abnormalities and seizures. In both tables, results utilizing the within-sample definitions of asymmetric and symmetric growth restriction are displayed above the definitions constructed from published birth standards. Below both sets of estimates 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 being either asymmetrically or symmetrically growth restricted increases the likelihood of childhood health problems.

Evidence from Table 11 shows that both subtypes of IUGR infants are significantly associated with having congenital malformations. The first two columns show estimates for the presence of any congenital malformation, the second two columns show results for the presence of any major malformation, the third pair of columns contain results for cardiothoracic malformations, and the final two columns show results for congenital malformations commonly associated with IUGR. As with the results from Section 3, the first column of each pair contains results for classifying asymmetric and symmetric growth restriction without the use of crown-heel length, and the second column of each pair shows results for classifying the IUGR subtypes using crown-heel length.

The in-sample results, displayed at the top of the table, show that both subtypes are significantly associated with having any congenital malformation, having any major malformation, having a cardiothoracic malformation, and having a malformation known to be concurrent with IUGR. However, these results indicate infants with symmetric growth restric-

[17] Odds ratios are presented rather than marginal effects because congenital malformations are relatively rare events; so the odds ratios are easier to interpret in this case.

tion have a greater likelihood of also having a congenital malformation in every specification. Furthermore, the F-test for the equality of coefficients of asymmetric and symmetric growth restriction show that the magnitude of the coefficient for symmetric is statistically different than asymmetric in almost every specification.

The preferred results, utilizing classifications constructed from published birth standards, tell a slightly different story. Both asymmetric and symmetric growth restriction are still significantly associated with having a congenital malformation. However, the magnitude of the coefficients is now statistically indistinguishable. These results show that having either subtype of IUGR is associated with an increased likelihood of having any congenital malformation by 1.238 to 1.237 times. The likelihood of having any major malformation increases approximately 1.5 times when IUGR is present. Cardiothoracic malformations are 1.68 to 2.16 times more likely for infants with IUGR. And IUGR infants are approximated 1.4 times more likely to have a malformation that has been previously associated with IUGR.

These results demonstrate that IUGR is a serious physical health threat to a child regardless 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, these results show that IUGR may be associated with more major health complications than previously attributed to it, since both subtypes are more highly associated with any type of major malformation than just those malformations previously attributed to IUGR. However, investigation of this question is left for future research.

Tables 12 tells a very similar story with more specific health conditions. The first pair of columns show results for an abnormal visual screening, the second pair of columns show results for an abnormal hearing screening, the third pair of columns show results for an abnormal speech evaluation, and the final pair of columns are estimates for the presence of non-febrile seizures. Estimates for asymmetric and symmetric growth restriction are statis-

tically indistinguishable in nearly all specifications. As with the malformation results, the magnitudes of the coefficients for symmetric growth restriction are noticeably larger in magnitude for the in-sample results (although only one specification show statistical difference). However, magnitudes are much closer in the preferred, out-sample classifications (with the noticeable exception of the hearing evaluation results).

As with the results of IQ on IUGR in Section 5.3, estimates for physical health are re-estimated using the definitions with matched birth weight. These results are displayed in Tables 13 and 14. The congenital malformation results (Table 13), show that when using classifications of asymmetric and symmetric growth restriction with matched birth weight, both subtypes show similar associations with congenital malformations. More notably, the out-sample classifications actually show a stronger association between malformations and asymmetric growth restriction than for symmetric growth restriction. Recall from Section 5.3 that this is despite there being a statistically larger cognitive effect for symmetric growth restricted infants over asymmetric growth restricted infants when using these same classifications. Results for vision, hearing, speech, and seizures in Table 14 also show an absence of any statistical difference between the coefficients for asymmetric and symmetric growth restriction. The conclusion from this set of results is that even after removing any statistical differences in health outcomes, there is still a persistent difference between asymmetric and symmetric growth restriction regarding cognitive outcomes.

As with the results on cognitive outcomes, using a series of different in-sample and out-of-sample classifications does little to affect the above results. Signs and magnitudes remain vary similar regardless of the classification use. However, due to the rarity of the dependent variable, some specifications are less robust to the statistical noise that results from using much larger cutoffs for IUGR. Tables with these results are available upon request.

The results shown here demonstrate that there is strong evidence that asymmetric growth restriction is significantly associated with poor physical health, and that there is some evi-

dence to suggest that the health shock may even be as severe as 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 effects 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, it is important that the classifications chosen for asymmetric and symmetric growth restriction conform to the characteristics of the medical narrative; this provides evidence that the cutoffs chosen accurately discriminate between IUGR neonates and non-growth restricted neonates, as well as accurately differentiating between those IUGR neonates with asymmetric growth restriction and those with symmetric growth restriction. Secondly, understanding the effects conditions in utero have on IUGR can inform potential policy interventions, as well as future research.

To this end, multinomial regression models are estimated for the presence of the subtypes of IUGR. Variables of interest include indicators of infections in early or late pregnancy and the smoking habits of the mother.

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. The CPP allows for a test of the differential impact of two 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 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. 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.

Table 15 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 1.24 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 of growth restriction by 2-3 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.

Table 16 shows the relative risk of becoming symmetrically growth restricted versus asym-

metrically growth restricted given the growth restriction factors of interest. As expected early growth insults (measured by infections) increase the probability of becoming symmetrically growth restricted, and a late growth insult increases the probability of an infant becoming asymmetrically growth restricted relative to symmetric growth restriction.[18] However, these measures are not statistically significant. The lack of precision is likely due to the small number of women who contracted major infections during pregnancy (5,152), which is only approximately 10 percent of those mothers with surviving children. There is some evidence that smoking is more probable to cause symmetric growth restriction rather than asymmetric growth restriction, particularly if the mother is a moderate or heavy smoker.

8 Discussion & Conclusion

This paper reexamines the underlying causes of the relationship between birth weight and education. 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 (measure by childhood IQ score), while the asymmetric type typically is shown to leave cognitive faculties mostly unchanged. Furthermore, there is evidence to suggest asymmetric and symmetric growth restriction have some different underlying causes and onset at different points in pregnancy. These results have broad implications for future research in economics, medicine, and public policy concerning infant health and pregnancy interventions.

Potentially one of the most important contributions of this paper is 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

[18] Taking the reciprocal of the estimates in the table shows a late insults make asymmetric restriction 1.235 to 1.5 times more probable.

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.

Finally, these innovations have implications for early childhood intervention programs. Programs like Head Start are designed to improve later life human capital development through early childhood investment in cognitive ability and knowledge. However, if we consider investment in human capital to behave as suggested by Cunha and Heckman (2007),

then these early childhood investment could be complimentary to investments made while the child was in utero. If complimentarity is high between in utero investments (or lack there of) and childhood investments—as may be the case with decreased brain size due to symmetric growth restriction—these early childhood investments will not be as valuable for those born with symmetric growth restriction. The welfare implications of this is, of course, an empirical question that involves the estimation of the degree of complimentarity between in utero and childhood investments, accurate estimation of the prevalence of symmetric growth restriction in specific populations, as well as the resulting cost-benefit analysis. All of these are beyond the scope of this paper; however, this could be a focus of future work.

One could argue that the current economics literature simply estimates a reduced form effect of birth weight on education. That is, the distinction between asymmetric and symmetric growth restriction can be ignored as long as we consider the currently estimated results in the literature as a reduce form effect. Whether the mechanism at work is through changes in IQ or health does not change the fact that increasing birth weight improves adult outcomes, one might say. However the subtypes are associated with different causes and different timing of onset, which calls into question the value of reduced form estimation. 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.

8.1 Future research

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). Therefore, separating a growth restricted sample by the timing of the potential cause is a promising avenue to explore.

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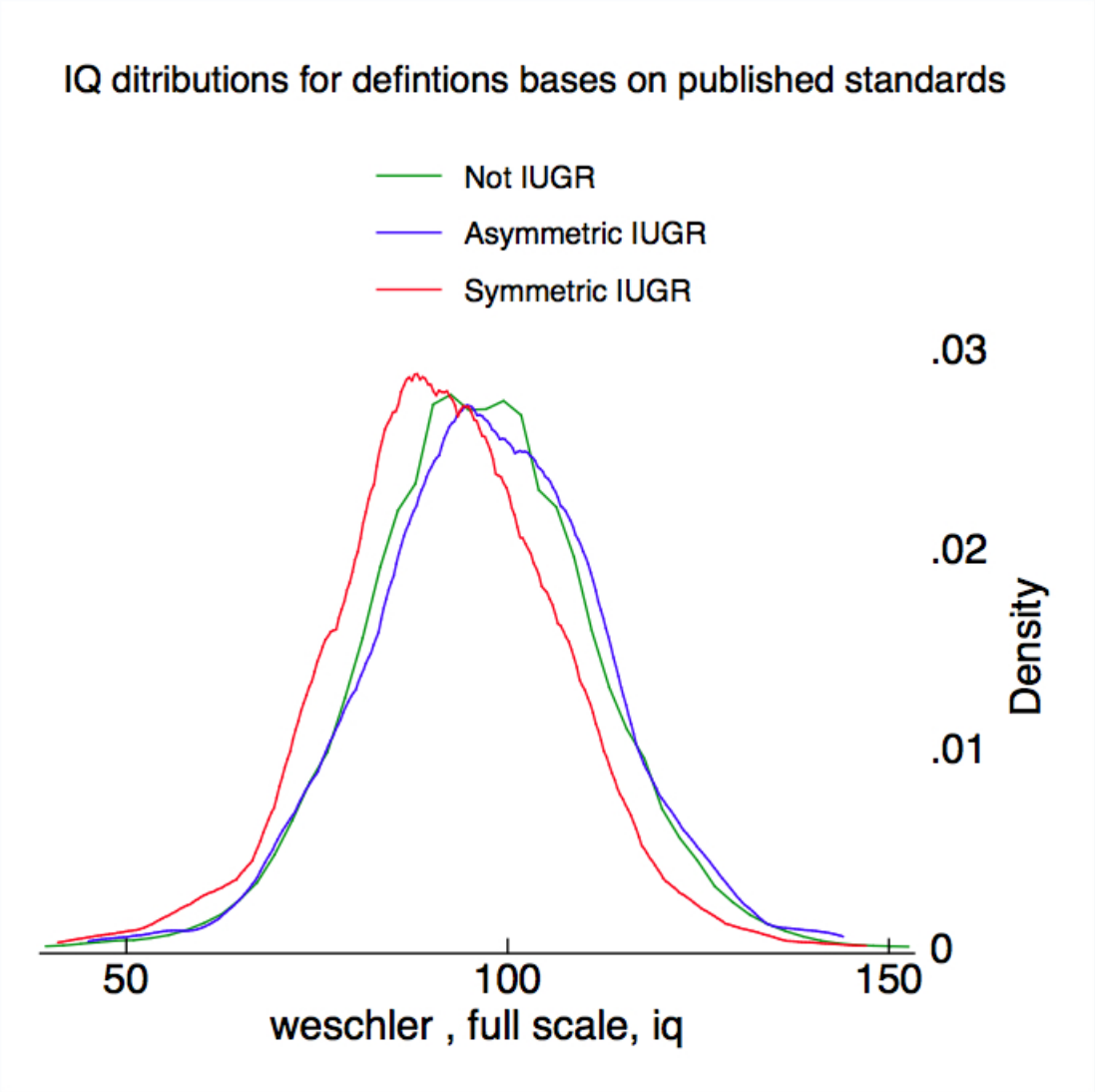

Figure 1: IQ distributions broken down by growth type

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
Cardiothoracic malformation*	0.012	—	0	1	47,006
Malformaion related to IUGR*	0.112	—	0	1	47,006
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 birth weight	mean head circum.
In-Sample Definitions					
Asymmetric	0.031	1480	47019	2493	33.23
Symmetric	0.073	3422	47019	2263	31.05
Asymmetic (using C-H length)	0.036	1691	46799	2512	33.32
Symmetric (using C-H length)	0.029	1342	46799	2202	30.89
Out-Sample Definitions					
Asymmetric	0.024	991	42009	2480	33.34
Symmetric	0.081	3391	42009	2296	31.27
Asymmetric (using C-H length)	0.013	532	41823	2564	33.55
Symmetric (using C-H length)	0.059	2475	41823	2223	31.10

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 begin 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 and crown-heel at or above the adjusted 10th percentile when denoted "using C-H length". Symmetric growth restriction is complimentarily defined as being IUGR with head circumference (or head circumference and crown-heel length) 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 (or head circumference and crown-heel length) adjusted for gestational age and gender according to Miller and Hassanein (1971).

Table 3: OLS Results for IQ

	IQ at Age 4		IQ at Age 7	
	No Length	C-H Length	No Length	C-H Length
In-sample Results				
Asymmetric	-1.348***	-1.206**	-1.120***	-0.946*
	(0.43)	(0.60)	(0.39)	(0.56)
Symmetric	-4.382***	-4.780***	-3.706***	-4.117***
	(0.33)	(0.39)	(0.27)	(0.32)
N	34641	34503	37003	36857
R^2	0.31	0.31	0.34	0.34
P-value for Equal β	0.000	0.000	0.000	0.000
Out-sample Results				
Asymmetric	-1.137**	-1.014	-1.097**	-0.700
	(0.54)	(0.73)	(0.48)	(0.64)
Symmetric	-4.307***	-4.927***	-3.601***	-4.009***
	(0.32)	(0.38)	(0.26)	(0.31)
N	31093	30967	33108	32979
R^2	0.31	0.31	0.34	0.34
P-value for Equal β	0.000	0.000	0.000	0.000

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, the number of prenatal visits (as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.

Figure 2: Summary of definitions for different categorizations of the of IUGR

In-Sample Classifications

Label	IUGR Definition	HC	C-H	Subtype Cutoff
sym	BW <10%tile or ponderal index <10%tile		X	<10%tile
sym10_10*	BW <10%tile for race, gender, & gest. age	X		<10%tile for race, gender, & gest. age
sym20_5	BW <20%tile for race, gender, & gest. age	X		<5%tile for race, gender, & gest. age
sym10_5	BW <10%tile for race, gender, & gest. age	X		<5%tile for race, gender, & gest. age
sym20_10	BW <20%tile for race, gender, & gest. age	X		<10%tile for race, gender, & gest. age
sym20_10_10	BW <20%tile for race, gender, & gest. age	X	X	<10%tile for race, gender, & gest. age
sym10_5_5	BW <10%tile for race, gender, & gest. age	X	X	<5%tile for race, gender, & gest. age
sym10_10_10*	BW <10%tile for race, gender, & gest. age	X	X	<10%tile for race, gender, & gest. age

Out-Sample Classifications

Label	IUGR Definition	HC	C-H	Subtype Cutoff
sym2	Hoffman et al (1974) by race, gender, & gest. age	X		Lubchenco et al (1963) by gest. age
sym3	Hoffman et al (1974) by race, gender, & gest. age	X		Usher & McLean (1969) by gest. age
sym4*	Hoffman et al (1974) by race, gender, & gest. age	X		Miller & Hassanein (1971) by gender & gest. age
sym2ch	Hoffman et al (1974) by race, gender, & gest. age	X	X	Lubchenco et al (1963) by gest. age
sym3ch	Hoffman et al (1974) by race, gender, & gest. age	X	X	Usher & McLean (1969) by gest. age
sym4ch*	Hoffman et al (1974) by race, gender, & gest. age	X	X	Miller & Hassanein (1971) by gender & gest. age
m_sym10	US Vital Statistics 2006-2008	X		CDC growth curves

* Indicates preferred definition, HC indicates "head circumference", C-H indicates "Crown-heel length".
All Out-sample classifications use the 10 percentile cutoff. Fields indicate the source of anthropometric standard

Table 4: OLS results for IQ at ages 4 and 7 using multiple in-sample def.

	sym	sym10_10	sym20_5	sym10_5	sym20_10	sym20_10_10	sym10_5_5	sym10_10_10
At Age 4								
Asymmetric	-2.175***	-1.348***	-1.809***	-2.012***	-1.636***	-1.687***	-1.720***	-1.206**
	(0.48)	(0.43)	(0.22)	(0.35)	(0.26)	(0.31)	(0.40)	(0.60)
Symmetric	-4.341***	-4.382***	-4.218***	-4.944***	-3.495***	-4.358***	-6.124***	-4.780***
	(0.37)	(0.33)	(0.33)	(0.40)	(0.26)	(0.34)	(0.53)	(0.39)
N	36656	34641	34641	34641	34641	34503	34503	34503
R^2	0.31	0.31	0.31	0.31	0.31	0.31	0.31	0.31
P-value for Equal β	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
At Age 7								
Asymmetric	-1.752***	-1.147***	-1.416***	-1.600***	-1.108***	-1.009***	-1.240***	-0.967*
	(0.41)	(0.39)	(0.19)	(0.30)	(0.22)	(0.26)	(0.36)	(0.56)
Symmetric	-3.013***	-3.699***	-3.767***	-4.264***	-3.190***	-3.730***	-5.332***	-4.109***
	(0.31)	(0.27)	(0.28)	(0.33)	(0.22)	(0.28)	(0.44)	(0.32)
N	38394	37003	37003	37003	37003	36857	36857	36857
R^2	0.33	0.34	0.34	0.34	0.34	0.34	0.34	0.34
P-value for Equal β	0.010	0.000	0.000	0.000	0.000	0.000	0.000	0.000

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, the number of prenatal visits (as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.

Table 5: OLS results for IQ at ages 4 and 7 using multiple outs sample def.

	fgrsym2	fgrsym3	fgrsym4	sym2ch	sym3ch	sym4ch	m_sym10
At Age 4							
Asymmetric	-1.862***	-1.499**	-1.014	-2.024***	1.175***	-1.137**	-0.240
	(0.35)	(0.72)	(0.73)	(0.33)	(0.44)	(0.54)	(0.45)
Symmetric	-7.015***	-4.582***	-4.927***	-5.793***	-4.463***	-4.307***	-2.816***
	(0.77)	(0.35)	(0.38)	(0.47)	(0.40)	(0.32)	(0.23)
N	31910	34503	30967	32043	32043	31093	36799
R^2	0.31	0.31	0.31	0.31	0.31	0.31	0.31
P-value for Equal β	0.000	0.000	0.000	0.000	0.000	0.000	0.000
At Age 7							
Asymmetric	-1.710***	-1.230**	-0.724	-1.966***	0.616*	-1.116**	-0.010
	(0.30)	(0.62)	(0.64)	(0.28)	(0.36)	(0.48)	(0.40)
Symmetric	-5.502***	-3.655***	-4.021***	-4.541***	-3.575***	-3.603***	-2.625***
	(0.66)	(0.29)	(0.31)	(0.40)	(0.33)	(0.26)	(0.19)
N	34092	36857	32979	34232	34232	33108	38542
R^2	0.34	0.34	0.34	0.34	0.34	0.34	0.33
P-value for Equal β	0.000	0.000	0.000	0.000	0.000	0.000	0.000

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, the number of prenatal visits (as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.

Table 6: Fixed Effects Results for IQ

	IQ at Age 4		IQ at Age 7	
	No Length	C-H Length	No Length	C-H Length
In-sample Results				
Asymmetric	-1.236	-2.017	-0.740	-0.880
	(1.49)	(1.95)	(1.25)	(1.91)
Symmetric	-3.712***	-3.434***	-3.402***	-3.929***
	(1.07)	(1.23)	(0.88)	(0.97)
N	8646	8616	9018	8987
R^2	0.84	0.84	0.84	0.84
P-value for Equal β	0.142	0.520	0.063	0.145
Out-sample Results				
Asymmetric	0.809	-1.313	-1.032	-0.389
	(1.92)	(2.37)	(1.53)	(1.84)
Symmetric	-3.099***	-3.455***	-2.977***	-3.766***
	(1.08)	(1.23)	(0.97)	(1.06)
N	7785	7759	8097	8070
R^2	0.86	0.86	0.85	0.85
P-value for Equal β	0.058	0.426	0.253	0.108

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, the number of prenatal visits (as a quadratic function), and indicators for gestational age, race, gender, and year of birth.

Table 7: Fixed effects results for IQ at ages 4 and 7 using multiple in-sample def.

	sym	sym10_10	sym20_5	sym10_5	sym20_10	sym20_10_10	sym10_5_5	sym10_10_10
At Age 4								
Asymmetric	-2.832	-1.236	-0.651	-1.852	-0.255	0.256	-2.292*	-2.017
	(1.73)	(1.49)	(0.77)	(1.19)	(0.86)	(0.97)	(1.30)	(1.95)
Symmetric	-3.209**	-3.712***	-4.515***	-4.143***	-3.535***	-3.185***	-4.132**	-3.434***
	(1.25)	(1.07)	(1.09)	(1.24)	(0.91)	(1.11)	(1.66)	(1.23)
N	8908	8646	8646	8646	8646	8616	8616	8616
R^2	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84
P-value for Equal β	0.840	0.142	0.001	0.145	0.003	0.013	0.361	0.520
At Age 7								
Asymmetric	-1.462	-0.740	-1.090*	-2.042**	-0.871	-0.607	-1.219	-0.880
	(1.32)	(1.25)	(0.64)	(0.99)	(0.71)	(0.82)	(1.14)	(1.91)
Symmetric	-2.300**	-3.402***	-3.086***	-3.099***	-2.605***	-3.212***	-3.653***	-3.929***
	(1.00)	(0.88)	(0.90)	(1.02)	(0.76)	(0.90)	(1.32)	(0.97)
N	9214	9018	9018	9018	9018	8987	8987	8987
R^2	0.84	0.84	0.84	0.84	0.84	0.84	0.84	0.84
P-value for Equal β	0.552	0.063	0.037	0.414	0.055	0.023	0.147	0.145

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, the number of prenatal visits (as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.

Table 8: Fixed effects results for IQ at ages 4 and 7 using multiple out-sample def.

	fgrsym2	fgrsym3	fgrsym4	sym2ch	sym3ch	sym4ch	m_sym10
At Age 4							
Asymmetric	-0.649	-0.676	-1.313	-0.902	2.388	0.809	-0.116
	(1.26)	(2.43)	(2.37)	(1.17)	(1.49)	(1.92)	(1.54)
Symmetric	-7.257***	-3.629***	-3.455***	-5.448***	-4.729***	-3.099***	-3.244***
	(2.47)	(1.09)	(1.23)	(1.54)	(1.30)	(1.08)	(0.80)
N	8008	8616	7759	8038	8038	7785	8939
R^2	0.85	0.84	0.86	0.85	0.85	0.86	0.84
P-value for Equal β	0.014	0.264	0.426	0.014	0.005	0.058	0.053
At Age 7							
Asymmetric	-1.698*	-1.604	-0.389	-1.897**	0.016	-1.032	0.323
	(1.01)	(1.91)	(1.84)	(0.96)	(1.16)	(1.53)	(1.24)
Symmetric	-4.127**	-2.750***	-3.766***	-3.206***	-2.719***	-2.977***	-2.144***
	(1.96)	(0.91)	(1.06)	(1.21)	(1.04)	(0.97)	(0.67)
N	8349	8987	8070	8380	8380	8097	9246
R^2	0.85	0.84	0.85	0.85	0.85	0.85	0.84
P-value for Equal β	0.255	0.579	0.108	0.361	0.160	0.253	0.059

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, the number of prenatal visits (as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.

Table 9: Summary of IUGR variables using matched birth weight

	mean	count	N	mean birth weight	mean head circum.
In-Sample Definitions					
Asymmetric	0.017	794	47019	2374	33.36
Symmetric	0.087	4108	47019	2325	31.48
Asymmetic (using C-H length)	0.006	285	46799	2368	33.40
Symmetric (using C-H length)	0.068	3203	46799	2314	31.34
Out of Sample Definitions					
Asymmetric	0.078	2551	46660	2346	32.17
Symmetric	0.025	2249	46660	2381	30.66
Asymmetric (using C-H length)	0.009	1611	45658	2326	32.13
Symmetric (using C-H length)	0.061	1053	45658	2371	30.62

All variables binary (0/1).

For all variables, IUGR is defined as having a birth weight below the 10th percentile adjusted for gestational age, race, and gender. For the in-sample variables, the 10th percentile cutoff is defined using the CPP sample, and for the out-sample variables, the cutoff is defined using Hoffman et al. (1974). Asymmetric growth restriction is defined as having a head circumference (or head circumference and length) in the fourth quartile for one of the 200 gram birth weight categories. Symmetric growth restriction is complimentarily defined as having a head circumference (or head circumference and crown-heel length in the first three quartiles for one of the 200 gram birth weight categories. For in-sample variables, quartile cutoffs are calculated using the CPP data, and for out-sample variables, quartiles are determined according to Usher and McLean (1969).

Table 10: OLS results for IQ using matched birth weight

	IQ at Age 4		IQ at Age 7	
	No Length	C-H Length	No Length	C-H Length
In-sample Results				
Asymmetric	-1.842***	-2.054**	-1.190**	-0.299
	(0.61)	(0.95)	(0.55)	(0.81)
Symmetric	-3.758***	-4.054***	-3.259***	-3.352***
	(0.30)	(0.33)	(0.25)	(0.27)
N	34641	34503	37003	36857
R^2	0.31	0.31	0.34	0.34
P-value for Equal β	0.004	0.045	0.000	0.000
Out-sample Results				
Asymmetric	-2.864***	-2.968***	-2.503***	-2.305***
	(0.30)	(0.34)	(0.25)	(0.29)
Symmetric	-5.455***	-5.384***	-4.660***	-4.954***
	(0.56)	(0.90)	(0.47)	(0.79)
N	34373	33664	36719	35962
R^2	0.31	0.31	0.34	0.34
P-value for Equal β	0.000	0.011	0.000	0.001

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, the number of prenatal visits (as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.

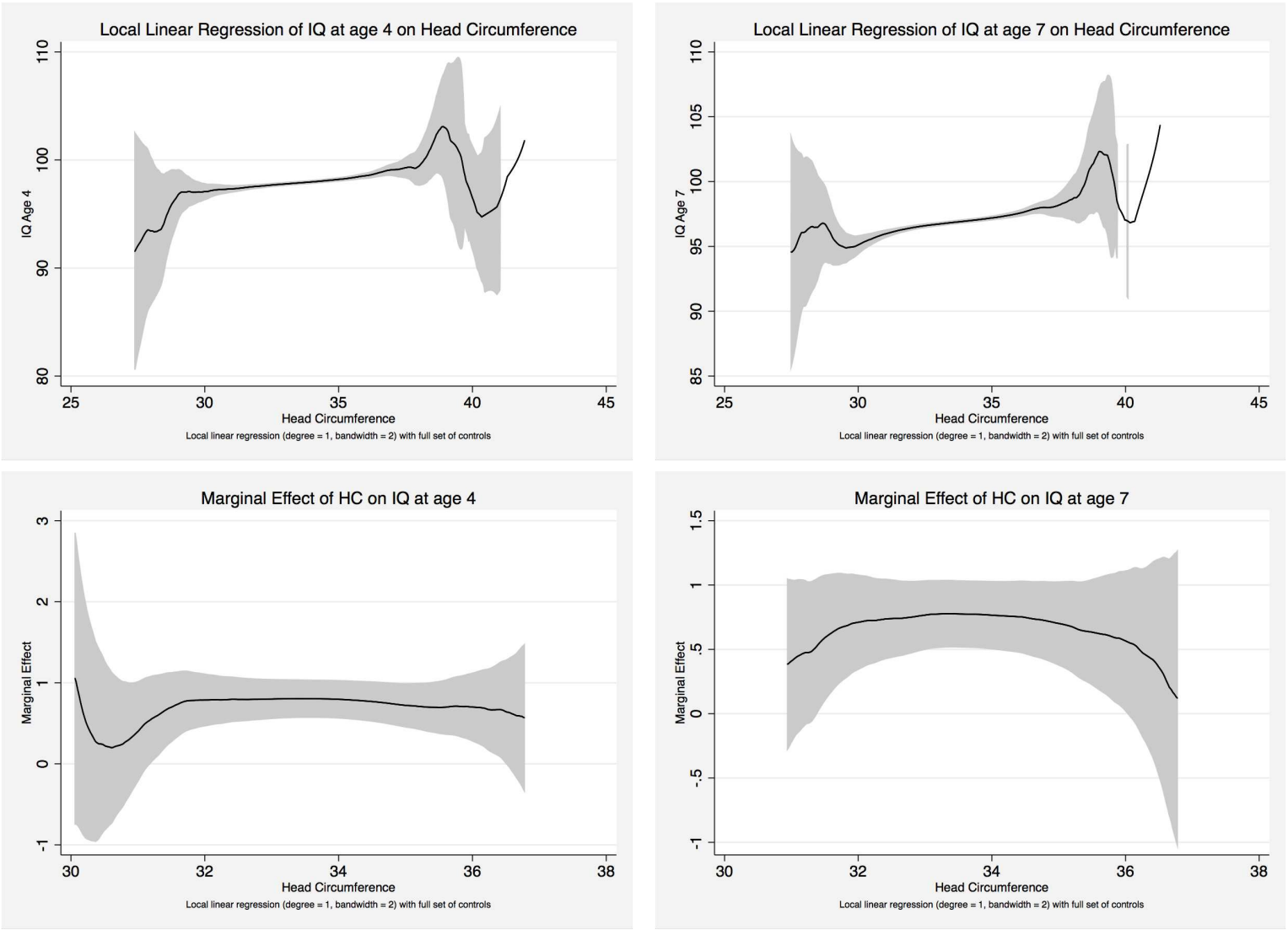

Figure 3: Local Linear Regression for the Effects of Head Circumference IQ

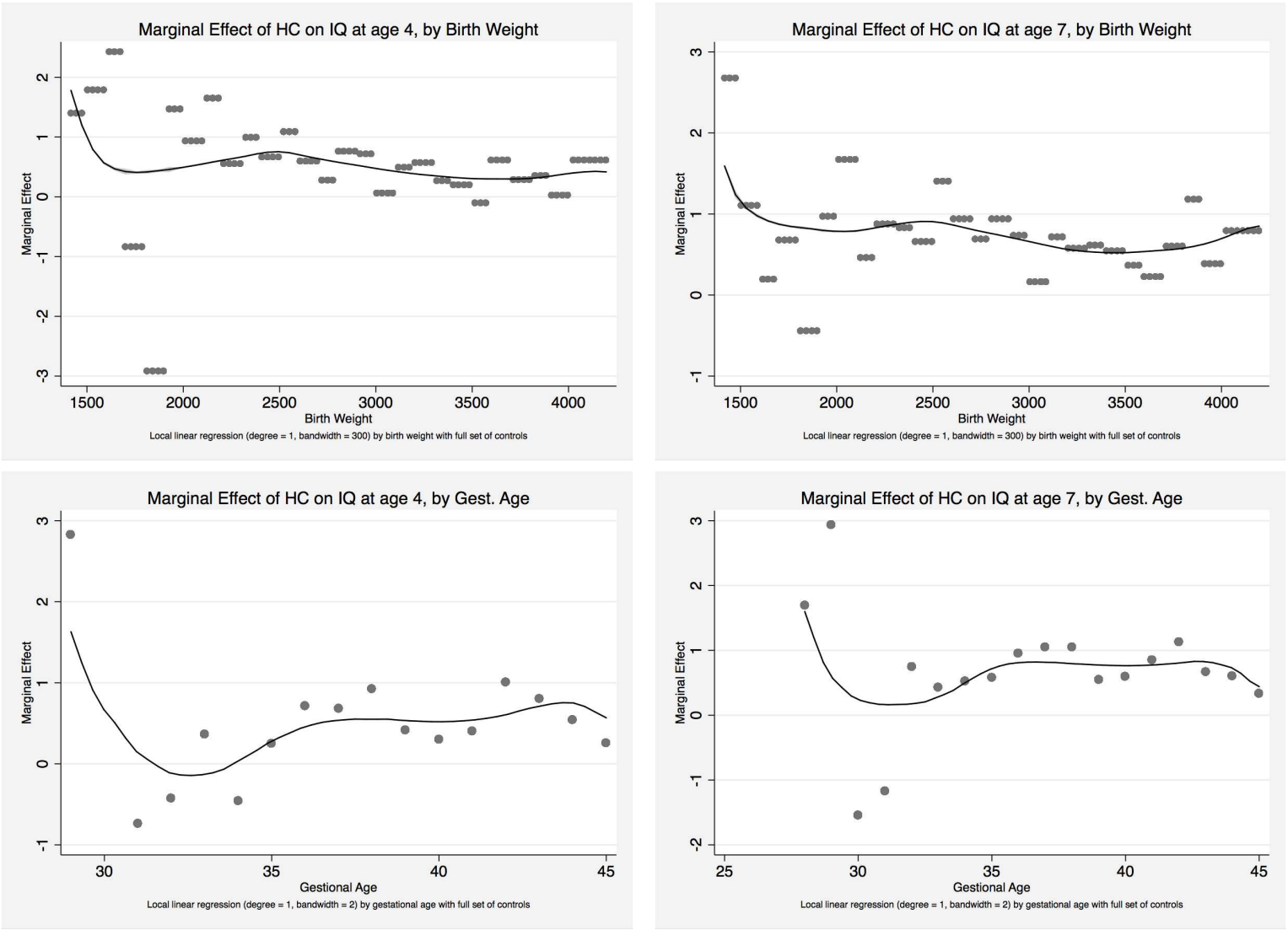

Figure 4: The Effects of Head Circumference IQ by Gestational Age and birth weight

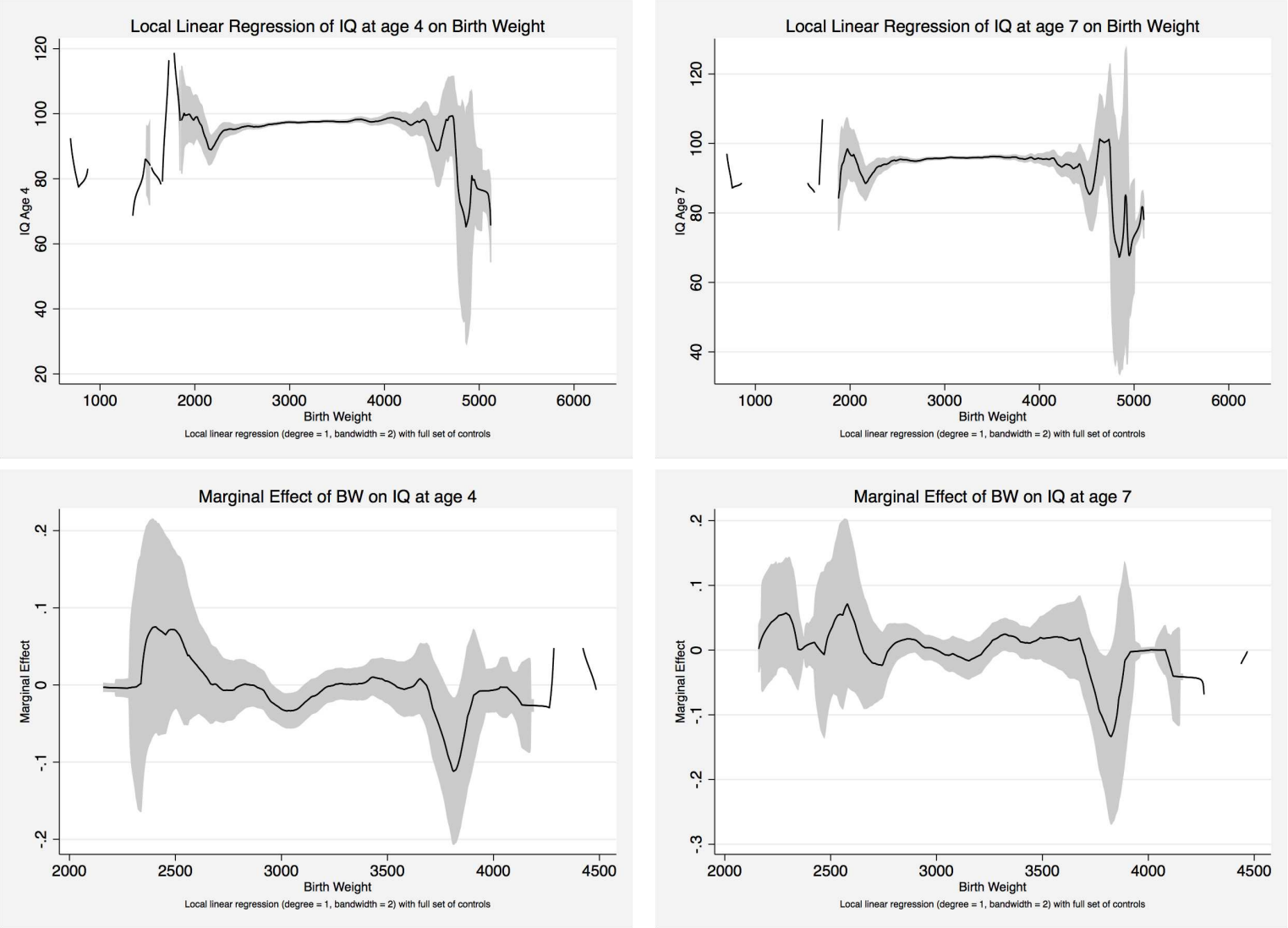

Figure 5: Local Linear Regression for the Effects of Birth Weight IQ

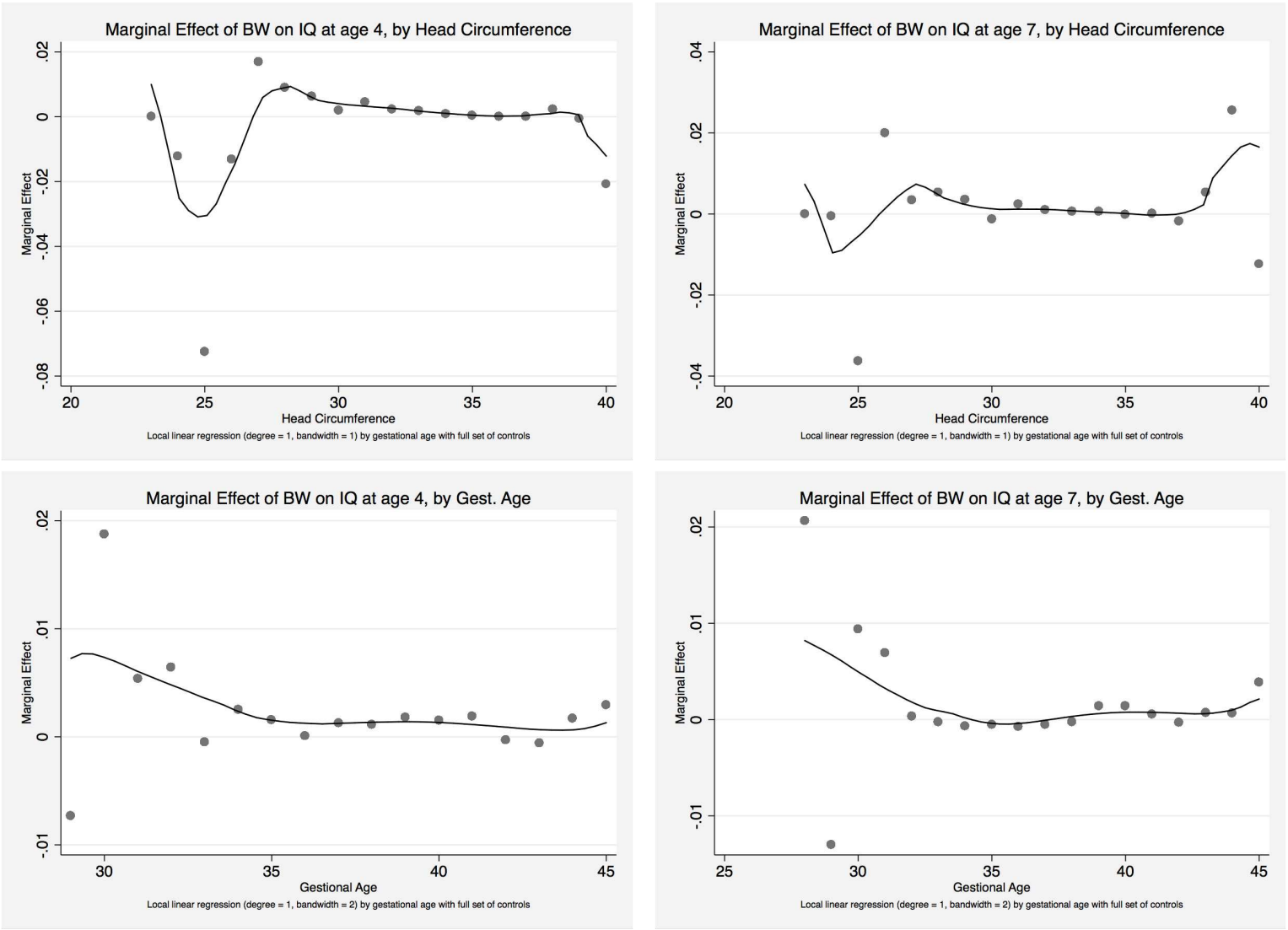

Figure 6: The Effects of Birth Weight on IQ by Gestational Age and Head Circumference

Table 11: Rare events logit: congenital malformations

	Any Con. Malf.		Major Con. Malf.		Cardiothor. C.M.		IUGR C.M.	
	No Length	C-H Length	No Length	C-H Length	No Length	C-H Length	No Length	C-H Length
In-sample Results								
Asymmetric	1.243***	1.282***	1.352***	1.436***	0.986***	1.320***	1.085***	1.142***
	(0.09)	(0.08)	(0.12)	(0.11)	(0.32)	(0.35)	(0.13)	(0.13)
Symmetric	1.471***	1.569***	1.621***	1.755***	1.766***	1.934***	1.474***	1.686***
	(0.07)	(0.11)	(0.09)	(0.14)	(0.30)	(0.47)	(0.11)	(0.18)
N	43127	42962	43127	42962	46288	46112	43122	42957
P-value for Equal β	0.030	0.043	0.050	0.091	0.093	0.358	0.017	0.029
Out-sample Results								
Asymmetric	1.351***	1.284***	1.511***	1.438***	1.677***	1.927***	1.399***	1.462***
	(0.11)	(0.14)	(0.15)	(0.19)	(0.50)	(0.74)	(0.18)	(0.25)
Symmetric	1.360***	1.371***	1.500***	1.533***	1.723***	2.163***	1.344***	1.409***
	(0.06)	(0.07)	(0.08)	(0.10)	(0.30)	(0.39)	(0.10)	(0.12)
N	38592	38445	38592	38445	41417	41260	38587	38440
P-value for Equal β	0.942	0.576	0.944	0.645	0.934	0.758	0.786	0.849

Exponentiated Coefficients (Odds Ratios). 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 12: Rare events logit: hearing, speech, vision, and seizures

	Visual		Hearing		Speech		Seizure	
	No Length	C-H Length	No Length	C-H Length	No Length	C-H Length	No Length	C-H Length
In-sample Results								
Asymmetric	1.212***	1.176***	1.225***	1.143***	1.240***	1.137***	1.369***	1.477***
	(0.08)	(0.08)	(0.19)	(0.17)	(0.27)	(0.22)	(0.28)	(0.27)
Symmetric	1.382***	1.507***	1.456***	1.779***	1.813***	1.822***	1.625***	2.287***
	(0.06)	(0.10)	(0.15)	(0.26)	(0.22)	(0.35)	(0.20)	(0.38)
N	46298	46122	46298	46122	46298	46122	46298	46122
P-value for Equal β	0.084	0.014	0.313	0.079	0.093	0.165	0.430	0.157
Out-sample Results								
Asymmetric	1.243***	1.238***	0.912***	0.801***	1.317***	1.462***	1.162***	1.562***
	(0.10)	(0.14)	(0.19)	(0.24)	(0.32)	(0.46)	(0.30)	(0.48)
Symmetric	1.355***	1.360***	1.507***	1.577***	1.612***	1.575***	1.516***	1.613***
	(0.06)	(0.07)	(0.15)	(0.18)	(0.21)	(0.23)	(0.20)	(0.23)
N	41426	41269	41426	41269	41426	41269	41426	41269
P-value for Equal β	0.319	0.407	0.013	0.010	0.406	0.816	0.282	0.922

Exponentiated Coefficients (Odds Ratios). 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 13: Matched birth weight rare events logit: congenital malformations

	Any Con. Malf.		Major Con. Malf.		Cardiothor. C.M.		IUGR C.M.	
	No Length	C-H Length	No Length	C-H Length	No Length	C-H Length	No Length	C-H Length
In-sample Results								
Asymmetric	1.368***	1.227***	1.404***	1.160***	0.624*	0.850	1.105***	0.837***
	(0.13)	(0.19)	(0.16)	(0.24)	(0.36)	(0.85)	(0.18)	(0.26)
Symmetric	1.407***	1.417***	1.565***	1.569***	1.702***	1.724***	1.405***	1.416***
	(0.06)	(0.07)	(0.08)	(0.09)	(0.27)	(0.30)	(0.10)	(0.11)
N	43127	42962	43127	42962	46288	46112	43122	42957
P-value for Equal β	0.776	0.315	0.331	0.060	0.023	0.203	0.104	0.015
Out-sample Results								
Asymmetric	1.409***	1.314***	1.505***	1.402***	1.643***	1.577***	1.355***	1.272***
	(0.06)	(0.07)	(0.08)	(0.09)	(0.27)	(0.30)	(0.10)	(0.11)
Symmetric	1.269***	1.087***	1.448***	1.440***	1.600***	2.360**	1.311***	1.493***
	(0.10)	(0.15)	(0.14)	(0.23)	(0.48)	(0.98)	(0.17)	(0.31)
N	42794	41918	42794	41918	45937	44986	42789	41913
P-value for Equal β	0.218	0.140	0.712	0.879	0.935	0.581	0.817	0.531

Exponentiated Coefficients (Odds Ratios). 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 14: Matched birth weight rare events logit: hearing, speech, vision, and seizures

	Visual		Hearing		Speech		Seizure	
	No Length	C-H Length	No Length	C-H Length	No Length	C-H Length	No Length	C-H Length
In-sample Results								
Asymmetric	1.294***	1.126***	1.324***	0.928**	1.261***	1.323**	2.054***	1.409**
	(0.12)	(0.18)	(0.27)	(0.36)	(0.34)	(0.57)	(0.46)	(0.64)
Symmetric	1.336***	1.314***	1.393***	1.388***	1.736***	1.520***	1.452***	1.496***
	(0.06)	(0.06)	(0.13)	(0.15)	(0.20)	(0.21)	(0.18)	(0.20)
N	46298	46122	46298	46122	46298	46122	46298	46122
P-value for Equal β	0.734	0.274	0.809	0.169	0.186	0.722	0.334	0.892
Out-sample Results								
Asymmetric	1.275***	1.255***	1.405***	1.264***	1.414***	1.349***	1.376***	1.263***
	(0.06)	(0.06)	(0.14)	(0.14)	(0.18)	(0.20)	(0.18)	(0.19)
Symmetric	1.504***	1.484***	1.254***	1.457***	2.171***	2.029***	1.628***	1.979***
	(0.11)	(0.19)	(0.23)	(0.42)	(0.44)	(0.70)	(0.34)	(0.64)
N	45947	44996	45947	44996	45947	44996	45947	44996
P-value for Equal β	0.079	0.286	0.553	0.689	0.226	0.486	0.538	0.425

Exponentiated Coefficients (Odds Ratios). 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 15: Multinomial Logit on the Causes of IUGR

	In Sample Def.		Outside Sample Def.	
	No Length	C-H Length	No Length	C-H Length
Symmetric				
Early Infection	1.165**	1.239*	1.235**	1.201**
Late Infection	0.960	0.969	0.999	0.947
Light Smoker	1.750***	1.845***	1.736***	1.750***
Moderate Smoker	2.739***	2.924***	2.740***	2.788***
Heavy Smoker	2.816***	2.971***	2.602***	2.596***
Asymmetric				
Early Infection	0.926	0.940	1.064	1.023
Late Infection	1.043	1.094	1.246	1.145
Light Smoker	1.644***	1.547***	1.580***	1.571***
Moderate Smoker	2.397***	2.160***	1.948***	2.332***
Heavy Smoker	2.898***	2.357***	1.855***	2.587***
N	46118	45943	41111	41267

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Exp. Coefficients (Relative Risk Ratios). 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 prenatal visits(as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.

Table 16: Multinomial Logit on the Causes of IUGR

	In Sample Def.		Outside Sample Def.	
	No Length	C-H Length	No Length	C-H Length
Symmetric				
Early Infection	1.245	1.654**	1.100	1.174
Late Infection	0.919	0.821	0.787	0.797
Light Smoker	1.059	1.076	1.263*	1.115
Moderate Smoker	1.176*	1.473***	1.531***	1.185
Heavy Smoker	1.049	1.323	1.578**	1.016
N	4695	2990	2895	4226

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Exp. Coefficients (Relative Risk Ratios). 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 prenatal visits(as a quadratic function), and indicators for gestational age, race, gender, year of birth, and location of birth.