Cesarean Sections and Later Child Health Outcomes

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10/29/2015

The rate of cesarean sections (CS) has grown substantially over the last two decades, now accounting for roughly one third of all births. The economics literature has established that a significant part of this growth is the result of physician incentives rather than medical necessity. Increasingly, however, the medical literature is raising questions about possible correlations between increased CS use and negative health outcomes for children in later life. For example, CS changes the intestinal bacteria of the infant and possibly compromises their immune system. This paper provides the first causal evidence that CS has an effect on later child health outcomes. Using variation in medical malpractice premiums as an instrument for risk-adjusted MSA-level rates of CS, I find that CS significantly increases the total rate of hospitalizations and the rate of hospitalizations that present with asthma for children up to 18 years old. These findings suggest that CS may have negative externalities on the child born via CS, which has implications for the evaluation of the socially optimal use of the CS procedure.

I. Introduction

Between 1996 and 2009, the rate of cesarean sections in the US rose from 20.7% to 32.8% (Ventura et al. 1996 and Martin et al. 2012). Since 2009, the rate has stabilized at approximately a third of all births, a number well in excess of the 2010 target rate of 15% suggested by the Centers for Disease Control (US Department of Health and Human Services 2010). Within the US, there is significant regional variation in the rates of cesarean sections (CS), ranging from 22.6% in Utah to 39.7% in Louisiana (Martin et al. 2012). Differences in CS rates can only be partially explained by differences in patient characteristics and are largely driven by nonmedical factors, with higher rates of CS reflecting less medically appropriate use of the procedure (Baicker et al. 2006). Many other nations have also experienced growth in CS rates.[1]

The potentially excessive use of CS is of concern because it has been linked to adverse health outcomes. There is a large literature on the short-term health consequences for infants, such as impaired lung function, altered metabolism, and altered feeding. More recently, an emerging medical literature has focused on long term negative health effects (Hyde and Modi 2012), including potential links between being born via CS and increased risk of asthma. These infants are not exposed to the maternal bacteria of the birth canal and have altered intestinal bacteria compared to infants born naturally, which could have a lasting impact on the immune system and other important processes, such as metabolic function (Hyde and Modi 2012). However, it is difficult to establish a causal link, both because some cesarean sections are

[1] China, in particular, has seen a very large increase in the rate of CS recently, increasing from 25.9% in 2003 to 46.2% in 2007 (Lumbiganon et al. 2010 and Gibbons et al. 2010). South American countries also have very high rates, led by Brazil, which in 2006 had a CS rate of 45.9% (Gibbons et al 2010).

medically necessary and because many studies suffer from an omitted variable problem. Indeed, the lack of causal evidence has even led some scholars to go so far as to call for a randomized controlled trial (Hyde et al. 2012).

In this paper, I provide the first evidence of a causal link between being born by CS and later health outcomes, specifically asthma. To do so, I create MSA-level rates of co-morbidities using the Nationwide Inpatient Sample Kids and MSA-level rates of CS from the National Vital Statistics System. I identify causal effects by using medical malpractice rates as an instrumental variable for MSA-level CS rates. Increases in medical malpractice premiums have been shown to increase the rates of CS (Dubay et al. 1999 and Baicker et al. 2006), which doctors may be performing defensively, as a significant proportion of malpractice suits faced by obstetricians are related to delaying or not performing a cesarean section (Minkoff 2012). Malpractice premiums are primarily driven by state level factors, such as the number of firms offering insurance and state tort laws, and are largely unrelated to patient safety (Thorpe 2005). I also show that malpractice premiums have little effect on infant health. I find a very large, robust, and causal effect on the rate of total hospitalizations and on the rate of hospitalizations that present with asthma and other chronic pulmonary disease, particularly among younger kids.

Establishing a causal effect of CS rates on the incidence of asthma is especially important given that the rates of asthma have gone up dramatically in recent years. According to the CDC (2013), the incidence of asthma increased by 28% between 2001 and 2011 and is particularly high among children. In 2011, 14% of US children had been diagnosed with asthma at some point in their lives (CDC 2013). The welfare cost to society in terms of losses to productivity for adults, losses to schooling for children, increased medical spending, and increased mortality is

potentially very high. The total cost of asthma was estimated to be $56 billion for the year 2007 alone (Barnett and Nurmagambetov 2011).

This finding could also have an important impact on doctor and patient decision making. Many CS are elective and may be chosen due to the widespread notion that the procedure is a safer for the mother and infant, despite the absence of conclusive evidence (Visco et al. 2006). Some patients and doctors may also prefer CS in order to control the timing of birth. For their part, medical providers may be increasing the rate of CS in response to the reimbursement differential that typically exists between CS and vaginal delivery (Gruber et al. 2009). Part of the increasing trend in CS may also be explained by medical providers financially compensating for falling fertility rates (Gruber and Owings 1996). More information on the possible negative effects of CS will allow doctors and patients to make a more informed and decision about whether CS is appropriate in a specific case.

The remainder of the paper is set at as follows: section II presents background information and a review of the literature concerning the potential effect of CS on asthma and other chronic pulmonary diseases and the potential mechanisms. Section III discusses the data sources and the construction of relevant variables. Section IV presents the results of a number of different specifications and includes a discussion on instrument validity, section V extends the analysis to other outcomes, and section VI concludes.

II. Background

There is a large, but inconclusive, medical literature on the link between CS and asthma and other immune deficiencies. Håkansson and Källén (2003) use linked medical birth data and hospital discharge data in Sweden to find that the procedure is associated with a significant

increase in the risk of developing asthma and gastroenteritis in children older than one. Renz-Polster et al. (2005) found that CS was correlated with allergic rhinoconjunctivitis and there was a gender specific association with asthma, with increased risk for girls. Using the Avon Longitudinal Study of Parents and Children, Maitra et al. (2004) found no increase in the risk for asthma, allergies, or wheezing. In a meta-analysis of 23 studies, Cho and Norman (2013) find that children born by CS have a 20% higher risk of developing asthma. Others have found a link between CS and other immune system deficiencies such as celiac disease (Decker et al. 2010) and a female-specific link with multiple sclerosis (Maghzi et al. 2012).

However, Lynch and Iams (2013) note that the literature is suspect because there is also a large literature that suggests an association between preterm birth and altered immune function. This may be the driving factor behind all the results because CS babies are more likely to have been born preterm because they are more likely to have health complications (Lynch and Iams 2013). There are other potential sources of endogeneity as well, as less healthy women are more likely to have cesarean sections and are also more likely to have less healthy infants. While some of the above studies restrict their sample only to elective CS, these may still be biased as there is some evidence that mothers who choose CS are more likely to be wealthier and have private insurance (Coonrod et al. 2000), which would generally reduce the infant's probability of developing asthma, which is linked with environment (Bloom et al. 2012 and Adler et al. 1994). The contribution of this paper is to address these issues of endogeneity and to establish a causal effect of CS on later health outcomes, such as asthma.

There are several potential mechanisms that may underlie the effect of CS on the immune system. The most prominent of these is related to the "Hygiene Hypothesis," in which early exposure to bacteria plays a large role in shaping the immune system and its responses later in

life (Strachan 1989). In particular, there is growing literature on the bacterial biome of the gut, which is estimated to have around 100 times as many genes in aggregate as does the human genome (Neu and Rushing 2012). Intestinal bacteria have a large role in shaping the immune system, as the bacteria stimulate the production of antibodies to pathogens by the lymphoid tissue. These gut bacteria perform a number of metabolic activities, as well as training the immune system, preventing the growth of harmful bacteria, and producing needed vitamins. However, there is a lot unknown about this diverse biome, as not all species of gut bacteria can be cultured, leading to projects such as the Human Microbiome Project, which hopes to map the bacteria of the gut and explore its effect on human health (Neu and Rushing 2012).

CS may have an effect on the makeup of intestinal bacteria by changing the first interaction an infant has with bacteria. It is unclear whether the intestinal ecosystem is generally sterile at the time of birth, though it appears to be for at least some infants (Neu and Rushing 2012). However, the species diversity is low in most infants shortly after birth and increases with environmental exposure (Magne et al. 2006). Dominguez-Bello et al. (2010) found that the method of delivery was the primary determinant of a newborn's bacterial community. Babies born naturally had a bacterial community composition that was most similar to the vaginal community of their mothers, whereas the intestines of infants delivered via CS were colonized predominantly by bacteria typically found on skin and in hospitals (Dominguez-Bello et al. 2010). Additionally, the intestinal bacteria of these infants appear to be less diverse and lacking in species generally associated with good intestinal function, such as bifidobacteria (Biasucci 2008). These differences may last for many years. A study of seven year olds found that those born through CS had significantly lower numbers of clostridia bacteria, which has been previously linked to asthma (Saliminen et al. 2004).

A possible confounder of the link between CS and gut bacteria is that CS is almost always performed after the administration of antibiotics, as dictated by standards of care (New and Rushing 2012). Few medical studies directly address this and none try to disentangle this effect. This paper will also be unable to do so. However, antibiotics are exclusively recommended for CS and for preterm premature rupture of membranes and are actively discouraged for all other births (American College of Obstetricians and Gynecologists 2011), so reducing the number of elective CS surgeries would also reduce the number of infants exposed to antibiotics in utero.

Another potential mechanism is the effect of CS on breastfeeding. Evans et al. (2003) found that babies born through CS consume significantly less breast milk in the first days of life, though the volume of milk consumed is equivalent to those born vaginally after 6 days. Since breast milk is a stimulator for intestinal flora, differences in early dietary support due to delayed lactation could also have long-term effects (Neu and Rushing 2011).

Finally, the stress of being born naturally could also be an important mechanism for healthy immune development. Contractions associated with natural birth, and indeed the whole process of natural birth, is intensely stressful for infants, who have higher levels of adrenaline after birth than adults do immediately after a heart attack (Hyde et al. 2012). This stress response may have an epigenetic impact and may modify the differentiation of a number of cell types, particularly immune cells, which have receptors for stress hormones. Hyde et al. (2012) hypothesize that there may be an important role for setting a very high hormonal threshold for this cell differentiation, so that stressful events for the mother do not trigger cell differentiation in utero. The stress of being born boosts the level of other catecholamine hormones and cortisol, which have been linked to organ development, including the gut (Cho and Norman 2013). It is

significant, then, that infants born via CS do not exhibit stress responses anywhere near those of naturally born children.

III. Data

The rates of CS were calculated using data from the National Vital Statistics System (NVSS) Birth Data, which is a 100% sample of all birth certificates in the US. I use all births from 1991-2004, which represents the intersection of publicly available data and years for which medical malpractice premium data could be obtained. Rates were calculated for cells that were at the birthyear-state -MSA-gender-race level[2], using the micro data found in the NVSS. Rates of CS were also risk-adjusted, as per Baicker et al. (2006). A regression model of the probability that an infant was delivered via CS was estimated on a number of patient-level covariates as well as state x MSA fixed effects:

$$CS_i = a + b_1 X_i + b_2(\text{State x MSA}) + e_i \qquad (1)$$

The patient-level covariates, X_i, include basic demographic variables, as well as many risk factors for CS, such as the age of the mother, birth weight, plurality of birth (e.g. twins), previous birth by CS, a range of mother morbidities, and complications of pregnancy as well as a range of possible complications that could occur during labor. Separate regressions were run for each birthyear-gender-race cohort. After adding in the group mean, the coefficients on the (state x MSA) variables, b_2, are the risk-adjusted regional rates of CS. MSAs were further subdivided by state so that, for example, the portion of the New York MSA located in New Jersey is treated separately from the rest of the greater New York City area. This is an important distinction for

[2] Interestingly, boys were significantly more likely to be born via CS in my data.

my instrumental variable strategy as medical malpractice tort law varies at the state level and is an important factor in medical malpractice premiums.

While risk-adjusting the rate of CS is not entirely necessary, given that CS will be instrumented for, I will use it in naïve OLS regressions first, which will provide a benchmark for my 2SLS results. Using the risk-adjusted rate of CS (RaCS) will provide a more meaningful benchmark as it takes into account some of the most obvious sources of endogeneity, such as infant and maternal health. Also, the RaCS is likely to be more sensitive to my instrument, since it is unlikely that changes in malpractice premiums will change the rate of medically necessary CS.

Data on child health outcomes comes from the Nationwide Inpatient Sample (NIS) Kids for years 2000, 2003, 2006, and 2009. The NIS Kids is part of a family of datasets collected and maintained by the Healthcare Cost and Utilization Project (HCUP), which is sponsored by the Agency for Healthcare Research and Quality (AHRQ). Unlike the general NIS, which includes annual inpatient data for adults, NIS Kids is only released every three years. Data from 1997, the first year this data was released, was not used because it lacked county or zip code location information at the hospital level. The target universe for the NIS Kids is all discharge data from community hospitals for inpatients younger than 21 years old (AHRQ HCUP 2005). The data contained in NIS KIDS represents a systematic 80% random sampling of a census of all community hospitals within a state designed to accurately represent the pediatric case-mix of each hospital. Data on hospital births are excluded as being out of the scope of this analysis. States that did not include hospital-level zip code data due to state privacy laws were dropped

from the analysis.[3] States that significantly limited the reported data more than the target universe were also excluded.[4] The states included in each year of the data can be seen in Table 1. Only children born between 1991 and 2004, which are the years that intersect with publicly available NVSS data and malpractice premium data, are included.

I construct two measures of child health using the NIS Kids data, the rate of hospitalization, and the rate of hospitalizations that present with asthma and other chronic pulmonary disease. There is reason to suspect that total hospitalizations may be affected by CS, as there is evidence that CS may compromise a child's immune system and make them more susceptible to a range of diseases (Hyde et al. 2012). The numerator for the rate of hospitalization is calculated using the number of observations in the NIS Kids in a birthyear-state-MSA-gender-race cell. Asthma and other chronic pulmonary disease is also a good candidate because the mechanisms by which CS could increase the risk of asthma are well documented in the science and medical literature. The numerator for the rate of hospitalizations that present with asthma and other chronic pulmonary disease is calculated using comorbidity software provided by HCUP, which creates measures of pre-existing chronic conditions based on the diagnosis coding of the ICD-9-CM.[5]

For each of these health measures, there are up to four observations for a specific birthyear by location by demographics cohort. Each observation is of the cohort at a different age over the course of the four years of NIS Kids data. So, cohorts born from 1991 to 2004 are observed in the NIS Kids data in years 2000, 2003, 2006, and 2009, which creates a sample that

[3] Notably, this excluded California and Texas, among others.
[4] After excluding states without hospital location data, this restriction also excluded Michigan, Georgia, and Virginia.
[5] The *International Classification of Diseases, Ninth Edition, Clinical Modifications.*

is up to 18 years old. The sample skews young, with an average age of seven years old, and cohorts born in earlier years are observed more often. Each observation of the number of both total hospitalizations and those presenting with asthma and other chronic pulmonary diseases within a birthyear-state-MSA-gender-race cell has the same denominator: the number of total births in that cohort as calculated from the NVSS, which is a good approximation of the total number of individuals in that cohort. Numerators were also scaled to represent a 100%. The total count of all cells, which equals the total births in the included states between 1991 and 2004, is over 30 million. This covers roughly half of all births per year, as calculated from the NVSS.

Because these rates are potentially unreliable for smaller areas, only cells that contain greater than a 500 birth count—a proxy for total individuals in that cell—are included. These constructed measures can be noisy, so the rates are Winsorized at the 99th percentile, which does not change the general result.

Data on medical malpractice insurance premiums comes from the Medical Liability Moniter (MLM), which is a national survey of insurers. I calculated average state-MSA level premiums as the premiums varied across MSAs within some states. Furthermore, the malpractice climate varies by state, which is the level regulations guiding malpractice insurance and tort reform.

In order to assure that MSAs were consistently defined, I used the Geokorr2k: Geographic Correspondence Engine program, provided online by the Missouri Census Data Center.[6] This software converts county FIPs and zip codes, in addition to other location data, to

[6] http://mcdc2.missouri.edu/websas/geocorr2k.html

MSA codes based on definitions used for the 2000 Census, ensuring that the MSAs were defined consistently across all the years of data used.

Table 2 contains weighted sample means across different age groups for some basic demographics, the rate of hospitalizations, and the rates of the co-morbidities conditional on having been hospitalized. The average of RaCS across all age groups is 16.14%; the average rate for older children is less because they were born in the early nineties. However, children who are less than one year old in my sample could have been born in any of the years for which I have NIS data, so the average rate of RaCS for this group is the average rate for the years 2000, 2003, 2006, and 2009. The rate of asthma and other chronic pulmonary diseases, conditional on hospitalization, is generally consistent with national averages reported by the CDC (2013), with rates of roughly 10% for children ages 7-13 and a slight downtick for asthma in older children as some cases of asthma become asymptomatic after puberty.

IV. Methodology and Results

a. OLS

In order to verify that the general trends in the medical literature are also found in my data, I first look at naïve OLS regressions. These results should be interpreted only as simple correlations because they do not address any endogeneity issues and are likely to suffer from omitted variable bias. I estimate the equation:

$$Y_i = \alpha + \beta_0 RaCS_i + X_i' \beta + \varepsilon_i \qquad (2)$$

where subscript *i* denotes an observation of a cohort defined by birth year and MSA-state location. Again, there are up to four observations of a cohort at different ages, though they all

have the same rate of risk-adjusted CS. Because each observation is an average rate, they are weighted by the cell count (Angrist and Pischke 2009). Y is an outcome variable that includes the rate of hospitalizations and the rates of those presenting with asthma and other chronic pulmonary diseases[7], RaCS is the risk adjusted rate of CS, and X is a vector of characteristics, including gender, age dummies[8], race, and birth year fixed effects. I include results for three different OLS specifications, which also contain state fixed effects or MSA fixed effects. Standard errors are clustered at the state-MSA level for all models.

Looking at Table 3, there is a significant correlation between RaCS and the rate of hospitalizations across all the models. While the coefficients appear to be small, they indicate large increases compared to the mean. For example, using the smallest estimate found in column (1), a 10 percentage point increase in the rate of RaCS would increase the rate of hospitalizations by roughly half of a percentage point—more than a 20% increase compared to the mean, holding other predictors constant. There is also a strong and robust correlation between RaCS and asthma and other chronic pulmonary diseases. Columns (4) through (6) suggest that a 10 percentage point increase in the rate of RaCS would increase the rate of hospitalizations that also present with asthma from 14% to 23%, depending on the model specification, compared to the sample mean, holding other predictors constant.

The OLS results in Table 3 mirror the medical literature but are misleading as they don't address any issues of endogeneity in the decision to have a cesarean section. For example, RaCS may be associated with risk factors not captured by the information in the NVSS and the risk adjustment, which could bias the estimates upwards. Alternatively, if RaCS is primarily driven

[7] Note that this is different than the conditional rates shown in table 2.
[8] The different age groupings are: less than 1 year old, 1-2 years old, 3-4 years old, 5-6 years old, 7-9 years old, 10-13 years old, and 14-18 years old.

by women from wealthier socioeconomic backgrounds choosing to undergo an elective procedure, the OLS could be downward biased. Children from wealthier backgrounds generally have lower incidences of asthma due to differences in environment, health behaviors, and incidence of illness (Bloom et al. 2012 and Adler et al. 1994). There is some evidence in the medical literature that white, privately insured women with more than a high school degree are more likely to induce labor, greatly increases the chance of CS (Coonrod et al. 2000). There is also the potential for bias due to measurement error, though the direction of this bias is less clear, because the rate of RaCS is calculated for a birth cohort and then linked with medical records for the same birthyear-state-MSA-gender-race group many years later. The degree to which the health measures cover the same individuals is not clear as families may move in and out of the area. Taken together, while the OLS estimates are clearly biased, the direction and source of the bias are unclear.

b. Instrument Validity

In order to establish a causal relationship, I use medical liability pressure, as measured by malpractice insurance premiums, as an instrument for RaCS rates. There is reason to think that CS is sensitive to medical malpractice premiums. The eight most common reasons for malpractice suits in obstetrics are all related to infant health, as opposed to maternal health, and six of these may involve an allegation of failure to perform CS or failure to perform the procedure in a timely manner (Minkoff 2012). According to Cyr (2006), these types of allegations are so common that "physicians can hardly be blamed for practicing a 'when in doubt, cut it out' philosophy."

The literature that has focused on malpractice premiums specifically has generally found a significant and positive effect of premiums on CS. Dubay et al. (1999) find that increases in malpractice premiums modestly increase the rate of CS. Baicker et al. (2006) find that differences in medical malpractice liability, including premiums as one of their two measures, account for 14.8% of the regional variation in the rate of CS[9]. The authors also show that regions with high rates of CS are performing less medically necessary procedures. There is also evidence in the medical literature suggesting that higher malpractice premiums are associated with significant, though relatively small, increases in the rate of CS (Murthy et al. 2007 and Yang et al. 2009).

There is mixed evidence of the effect of malpractice liability, measured in other ways, on the use of CS. Currie and MacLeod (2009) find that caps on noneconomic damages, which reduces the probability of a suit, actually increases the rate of CS, suggesting that doctors may be performing more procedures when liability is reduced because it is a more profitable procedure. Kim (2006) uses malpractice claims in other specialties as an instrument for malpractice claims in obstetrics and finds no impact on the rate of CS.

I find a reasonably large effect of malpractice premiums on risk-adjusted CS rates, significant at the $p<0.01$ level. I estimate that a 10% increase in premiums would increase the risk-adjusted rate of CS by about 4.1% relative to the sample mean. At 48.41, the F statistic for the first stage is also very large. These estimates are larger than those found in some of the previous literature because I use the risk-adjusted rate of CS, like Baicker et al. (2006), which is likely to be more sensitive to malpractice premiums since it already accounts for the medically necessary procedures unlikely to be induced by premium changes.

[9] Though Baicker andChandra (2005) find no significant effect of premiums on CS.

Using insurance premiums as an instrument for CS would be problematic if premiums reflected the level of obstetrician care and the probability of a medical error, which could have an impact on later health outcomes. However, premiums appear to only be weakly linked to patient safety. Due to the medical malpractice "crisis" of the early 2000s, there is a substantial amount of research on identifying the main drivers of increases in medical malpractice premiums over the years included in my data. The evidence suggests that larger market forces dictate the bulk of variation in liability premiums, as opposed to actual changes in the incidence of tort or patient safety. Malpractice premiums across specialties are highly correlated and tend to rise in tandem, indicating that they are primarily driven by system-wide, state-level factors, as opposed to the incidence of medical errors in a specific area of medicine (Baicker and Chandra 2005). Furthermore, the frequency of claims remained stable over 1991-2003, suggesting no increase in the number of medical errors despite large increases in premiums (Chandra et al. 2005). While payments for successful claims have increased over this period, increases in premiums are only weakly correlated with increases in claims payments and are not correlated with lags for claims payments (Baicker and Chandra 2005). Thus, the variation in liability premiums and the large increases seen in the early 2000s do not appear to reflect worsening medical care.

Instead, it is widely thought that these changes in premiums reflected a fluctuation in the insurance cycle, moving from a "soft market" in the 1990s to a more constrained market with higher prices and reduced insurance supply in the 2000s (Mello 2006). These cycles in insurance underwriting practices are well documented, though the exact causes leading to the increase in medical malpractice premiums in the 2000s are still not well understood. It appears that some of the largest contributors to the rapid hike in premiums include the rising price of reinsurance paired with large decreases in investment income (Thorpe 2004). Additionally, medical

malpractice has a long lag, where claims take years to be settled, introducing more uncertainty into the premium calculation (Thorpe 2004).

However, these explanations do not address the high degree of state-level variation seen in the data. The "crisis" itself was a state-level phenomenon and was limited to 10 out of 19 states in my data set (Mello et al. 2003). Thorpe (2004) notes that there was also a large reduction in the number of firms offering medical liability insurance in these states as major carriers became insolvent and exited the market and shows that increases in the Herfindahl-Hirschman Index are significantly associated with increases in premiums. Many malpractice insurers only provide coverage to one or a small number of states, so exits in the market have a differential effect on insurance supply at the state level. There are also large state-level differences in tort law and the broader laws that regulate malpractice insurance. For example, some states require all proposed premium changes to be pre-approved by state regulators on a case-by-case basis, while others let market forces work unfettered (Mello 2006). If insurers in these states were unable to adjust their prices quickly, they may have been less able to immediately offset their investment losses and their increased reinsurance costs, leading to larger future premium increases.

In order for this instrument to be valid, it must also not have an effect on later health outcomes through mechanisms other than CS. This condition could be violated if physicians perform the marginal procedures of CS much earlier than natural birth would occur, so that it is really reduced gestation time or lower birth weight driving the results. In order to test whether this occurs in my data, I use the NVSS to look at all of the births in the years and MSAs included in my analysis. I use the 5-minute Apgar score, which is a general measure of infant health and includes measures of heart rate, respiration, and neuromuscular function and is a good indicator

of infant mortality (Casey et al. 2001). There is also a very strong relationship between gestational age and Apgar score, with preterm babies averaging very low scores. I find that premiums are not correlated with the 5-minute Apgar score, confirming the findings by Dubay et al. (1999). Currie and McLeod (2009) also found that the marginal cesarean sections induced by tort reform had no impact on the 5minute Apgar score.

I do find that premiums are weakly correlated with babies considered to be very low birth weight (VLBW), defined as less than 1500 grams. However, the estimate is almost precisely zero—a 10% increase in liability premiums is associated with a 0.006% increase in the probability of being in the VLBW category. I also find a significant and larger effect on the probability of being considered low-birth weight (LBW), defined as weighing between 2500 and 1500 grams, but it is still very small—a 10% increase in premiums is associated with a 0.3% increase in low weight babies. Since the same increase in premiums is associated with a 4% increase in RaCS, it seems unlikely that the very small increase in LBW babies would be driving my results. I also find a weakly significant and nearly zero effect on the probability of being born premature, with a 10% increase in premiums increasing the probability of premature birth by 0.01%.

Using liability premiums as an instrument for CS could also be problematic if the supply of medical professionals decreases as the premiums increase. There is some evidence that, while this is unlikely to be true for other fields, it may be the case for obstetricians, who face much higher premiums than other medical specialties (Baicker and Chandra 2005, Mello et al. 2007). While it is reassuring that high premiums do not also indicate a constricted supply of doctors generally, making it unlikely that OB/Gyn premiums in the year of birth are correlated with reduced care from general practitioners later in life, it is possible that a decreased supply of

obstetricians could impact the level of prenatal care received. This, in turn, could potentially impact later child health. Dubay et al. (2001) address this issue and find that higher malpractice premiums lead to an increased prenatal care delay as well as fewer prenatal visits. I replicate these findings for the states and years included in my data. However, the coefficients, while significant, are very small. I estimate that a 10% increase in malpractice premiums reduces the probability that a woman will receive prenatal care in the first trimester by 0.176%. I also find that there are no significant increases in foregoing prenatal care entirely and I estimate a precisely zero effect on delaying care until the third trimester, conditional on seeking prenatal care. Thus, the results suggest that there is a very small percentage shift in women from receiving care in the first trimester to receiving care in the second trimester. This small effect, paired with nearly zero impact on infant health, suggest that increases in premiums are unlikely to affect later child health other than through increases in the rate of CS.

c. IV models and results

The basic IV model that I estimate through 2SLS is:

$$Y_i = \alpha + \beta_0 \widehat{RaCS}_i + X_i' \beta + \varepsilon_i \quad (3)$$

where subscript *i* denotes an observation at the birthyear-state-MSA-gender-race level and $RaCS_i$ is instrumented by medical malpractice premiums. X is a vector of characteristics, including gender, age dummies, race, and birth year fixed effects. Observations are weighted according to cell size, and standard errors are clustered at the state-MSA level for all models.

I also estimate 2SLS regressions that instrument for the interaction term between RaCS and age. For immune related diseases, we would expect the impact of CS to become less pronounced with age as factors like diet, illness, and antibiotic use also shape the gut

microbiome and immune system (Guarner and Malagelada 2003). In order to capture this effect, I also estimate:

$$Y_i = \alpha + \beta_0 \widehat{RaCS}_i + \beta_1 (\widehat{RaCS_i * I\_age_i}) + X_i' \beta + \varepsilon_i \quad (4)$$

where all variables are defined as above and the interaction terms of RaCS and the indicator variables for the different age groupings are instrumented by the interaction of medical malpractice premiums and the age group indicator variables. The omitted group is those who are less than one year old.

Table 4 presents the results for the 2SLS regressions with the rate of hospitalizations and the rate of those that presenting with asthma as the dependent variable. The coefficient on the instrumented RaCS is very significant at the $p<0.01$ level in the first specification, without the age group interaction terms, and it becomes larger, though weakly significant, when these other covariates are included. The estimates are also very large relative to the sample mean. Using the coefficient in column (1), a 10 percentage point increase in RaCS, roughly a 60% increase in the average rate of RaCS, would increase the rate of hospitalizations by about 50% across all age groups.

The effect on the rate of hospitalizations that present with asthma or other chronic lung diseases is significant across both specifications. These effects are also very large relative to the sample mean, with a 10 percentage point increase in RaCS increasing the rate of hospitalizations with asthma by about 46% to 53% across all ages, depending on the coefficient used. Looking at column (4), it appears that this effect is greater for younger children, with a large and significant coefficient on the age interaction term for kids 1-2 years old. The coefficients on the age interaction terms are jointly significant at the 1% level, so the significance on the interaction with

kids 1-2 years old is unlikely due to chance. The total effect of a 10 percentage point increase in RaCS for this age group would be roughly an 80% increase in the rate of hospitalizations that present with asthma. While these numbers seem very high, they are not unrealistic given the general trends seen in the US in recent years. Between 2001 and 2011, the rate of asthma grew by 28% (CDC 2013). Over that same period, the unadjusted rate of CS grew by about 34%, with the majority of that growth occurring in the earlier years (Martin et al. 2013 and Martin et al. 2002).

Given the above results, it is possible that increases in the rate of hospitalizations with asthma and other chronic pulmonary diseases could be driven mechanically by the increased rate of total hospitalizations if the rise in hospitalizations was unrelated to an increase in the incidence of the asthma or chronic pulmonary disease in the population. In order to demonstrate that there is a direct effect on the incidence of asthma and other chronic pulmonary disease, I restrict the analysis only to hospitalizations that list these as the primary reason for admission. This variable captures an entirely different population than the measure used thus far, which captures asthma and chronic pulmonary disease as a pre-existing chronic illness and not as the primary diagnosis or the primary reason for admission. I create two variables: one that includes only asthma-specific diagnostic codes, and another that includes all of the diagnostic codes contained in the co-morbidity measure of chronic pulmonary diseases. Increases in these variables would only be caused by an increase in the number of hospital visits caused by asthma and an increase in the number of hospital visits caused by asthma and other chronic lung disease, respectively.

The results are in Table 5. The coefficients on the instrumented RaCS are significant across specifications with and without age interaction terms. The coefficients, though small,

mask even larger effects than when using the chronic illness measure. Using the estimate from column (1), a 10 percentage point increase in RaCS would more than double the rate of hospitalizations with asthma as the primary diagnosis. Using the estimate in column (3), the same increase would increase the rate of hospitalizations primarily for asthma and other chronic lung diseases by roughly 50%. The interaction terms are also significant and suggest a similar trend to those discussed in Table 4: the effect of RaCS is diminished as children grow older. Because these variables are constructed using only primary diagnostics, as opposed to the co-morbidity measures, these increases are caused by an increase in hospitalizations caused by asthma and other chronic lung diseases, suggesting that the incidence of these illnesses has increased due to CS.

V. Robustness

In order to further verify that the increases in hospitalizations and those that present with asthma are driven by CS, I will run a placebo test, using an outcome that is a priori unrelated to CS. However, finding such an outcome is difficult, as some of the better documented pathways, such as impact on the flora of the gut or the epigenetic consequences of an extreme hormonal stress response, could have impacts on health other than just through the immune system. The influence of the gut biome is a relatively recent and rapidly expanding field of study that has linked the gut biome to many different health outcomes. There is a well-documented "bidirectional neurohumoral communication system, known as the gut–brain axis, [that] integrates the host gut and brain activities" (Collins et al. 2012). There is evidence, often coming out of animal trials, that gut bacteria can have an impact on a huge number of outcomes, such as anxiety, depression, autism, irritable bowel syndrome, memory, and liver disease (Collins et al. 2012). CS has also been correlated with other later health outcomes, such as obesity, cancers,

and skeletal diseases, though the evidence is weaker and the mechanism is less clear (Cho and Norman 2013). Furthermore, some of the more obvious candidates, like trauma and fractures, could be influenced by the activity level of the child, which could be greatly affected by asthma.

I find one class of hospitalizations, patients suffering from burns, that I expect to be completely unrelated to the rate of CS or the rate of asthma and other chronic pulmonary diseases. Table 6 contains the results from the 2SLS models with the rate of hospitalizations due to burns as the outcome variable. Reassuringly, I find no significant effect of the instrumented rate of RaCS on the rate of hospitalizations due to burns using either specification. Furthermore, the coefficients on the age interaction terms in column (2) are also jointly not significant.

VI. Conclusion

The effect of CS on the rate of total hospitalizations and the rate of hospitalizations that also present with asthma and other chronic pulmonary diseases is robust and large. This is an important finding since both the rate of CS and the rate of asthma have increased dramatically over recent years. Between 1996 and 2009, the unadjusted rate of CS rose by 11.6 percentage points (Martin et al. 1996 and Martin et al. 2009), while the rate of asthma increased by roughly 28% between 2001 and 2011 (CDC 2013). Although the coefficient estimates are small in absolute size, they are large compared to the sample means. The effect on the rate of hospitalizations that present with asthma and other chronic pulmonary disease is particularly high among younger children.

These effects are a negative externality of CS. With high malpractice premiums, doctors are further incentivized to perform more cesarean sections than are medically necessary. These excess procedures not only cost more in direct medical spending but also significantly increase

the infant's probability of being hospitalized later in life, resulting in further increases in medical costs. There are also significant economic costs associated with asthma, both in terms of direct medical spending, and from lost schooling and other impacts on the development of human capital. The total cost of asthma was estimated to be $56 billion for the year 2007 alone (Barnett and Nurmagambetov 2011).

This study is a strong, yet initial, start to understanding the causal connection between CS and later health outcomes. There is much future research to be done on this topic. There could also be a large effect of CS on other variables of economic interest, such as adult income or education, that would allow for better measurement of the social cost of medically unnecessary procedures. Additional research is also needed to understand the mechanisms behind the findings presented here, as this study is unable to distinguish between them. Understanding the mechanisms could allow doctors to mitigate the negative long term health effects of medically necessary procedures.

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Table 1: Final states from NIS Kids

NIS Kids data year	States included in analysis
2000	AZ, CO, FL, IA, KY, MD, MA, NC, NJ, NY, OR, PA, UT, WA, WI
2003	+ IL, NH, NV, VT - PA
2006	Same as 2003
2009	+ PA

Table 2: Weighted Sample Means by Age Group

Age group:	0-18	<1	1-2	3-4	5-6	7-9	10-13	14-18
Risk-adjusted CS	0.161	0.165	0.176	0.192	0.170	0.163	0.151	0.126
Rate of hospitalizations	0.024	0.052	0.023	0.026	0.024	0.023	0.020	0.025
Asthma and chronic pulmonary disease, conditional on hospitalized	0.094	0.016	0.073	0.094	0.104	0.101	0.103	0.098
Female	0.487	0.488	0.487	0.487	0.487	0.486	0.486	0.487
African American	0.156	0.154	0.158	0.158	0.146	0.158	0.154	0.163
Other race	0.036	0.040	0.043	0.039	0.036	0.039	0.031	0.027
Number of cells	7782	349	803	970	1129	1880	1591	1060
Weighted Obs	33,434,036	1,367,059	3,599,553	4,125,730	608,696	8,384,847	6,739,929	4,608,222

Table 3 : The effect of risk adjusted CS on health outcomes: OLS

	Hospitalizations			Asthma, chronic lung disease		
	(1)	(2)	(3)	(4)	(5)	(6)
RaCS	0.0535**	0.0400***	0.0365***	0.0046**	0.0031***	0.0027***
	(0.0219)	(0.0121)	(0.0100)	(0.0018)	(0.0011)	(0.0008)
Controls	yes	yes	yes	yes	yes	yes
Birthyear FE	yes	yes	yes	yes	yes	yes
State FE		yes	yes		yes	yes
MSA FE			yes			yes
Observations	7,782	7,782	7,782	7,782	7,782	7,782
R-squared	0.616	0.660	0.698	0.498	0.547	0.592

Note: The unit of observation is the birthyear-state-MSA-gender-race cell. Cells are weighted proportional to cell count. All regressions also included the covariates: female; age dummies; African American; other race. All standard errors are clustered at the state-MSA level. Stars indicate: *** $p<0.01$, ** $p<0.05$, * $p<0.1$

Table 4: The effect of risk adjusted CS on health outcomes using 2SLS

	Hospitalizations		Asthma, Chronic Lung Disease	
	(1)	(2)	(3)	(4)
RaCS	0.1230***	0.1830*	0.0104***	0.0092**
	(0.0451)	(0.1100)	(0.0033)	(0.0043)
Age 1-2 x RaCS		0.0142		0.0068**
		(0.0867)		(0.0035)
Age 3-4 x RaCS		-0.0889		0.0012
		(0.0841)		(0.0024)
Age 5-6 x RaCS		-0.103		-0.0004
		(0.0787)		(0.0026)
Age 7-9 x RaCS		-0.0982		0.0005
		(0.0882)		(0.0028)
Age 10-13 x RaCS		-0.0975		-0.0023
		(0.0770)		(0.0037)
Age 14-18 x RaCS		0.143		0.0091
		(0.1560)		(0.0101)
Controls	yes	yes	yes	yes
Birthyear FE	yes	yes	yes	yes
Observations	7,782	7,782	7,782	7,782

Note: The unit of observation is the birthyear-state-MSA-gender-race cell. Cells are weighted proportional to cell count. All regressions also included the covariates: female; age dummies; African American; other race. Less than 1 year old is the omitted age group. All standard errors are clustered at the state-MSA level. Stars indicate: *** p<0.01, ** p<0.05, * p<0.1

Table 5: 2SLS; The effect of RaCS on Primary Diagnosis

	Asthma Only		Asthma and Other Lung Disease	
	(1)	(2)	(3)	(4)
RaCS	0.0004***	0.0008**	0.0118***	0.0155***
	(0.0001)	(0.0003)	(0.0042)	(0.0047)
Age 1-2 x RaCS		0.0001		0.0104*
		(0.0003)		(0.0058)
Age 3-4 x RaCS		-0.0006**		0.0000
		(0.0003)		(0.0068)
Age 5-6 x RaCS		-0.0007**		-0.0051
		(0.0003)		(0.0032)
Age 7-9 x RaCS		-0.0005*		-0.0066*
		(0.0003)		(0.0039)
Age 10-13 x RaCS		-0.0007**		-0.0115***
		(0.0003)		(0.0034)
Age 14-18 x RaCS		-0.0002		-0.0150***
		(0.0004)		(0.0036)
Controls	yes	yes	yes	yes
Birthyear FE	yes	yes	yes	yes
Observations	7,782	7,782	7,782	7,782

Note: The unit of observation is the birthyear-state-MSA-gender-race cell. Cells are weighted proportional to cell count. All regressions also included the covariates: female; age dummies; African American; other race. Less than 1 year old is the omitted age group. All standard errors are clustered at the state-MSA level. Stars indicate: *** p<0.01, ** p<0.05, * p<0.1

Table 6: Placebo test using 2SLS

	Hospitalizations due to burns	
	(1)	(2)
RaCS	0.0005	0.0015
	(0.0004)	(0.0014)
Age 1-2 x RaCS		0.0001
		(0.0004)
Age 3-4 x RaCS		-0.0009
		(0.0009)
Age 5-6 x RaCS		-0.0010
		(0.0013)
Age 7-9 x RaCS		-0.0014
		(0.0015)
Age 10-13 x RaCS		-0.0017
		(0.0016)
Age 14-18 x RaCS		-0.0013
		(0.0013)
Controls	yes	yes
Birthyear FE	yes	yes
Observations	7,782	7,782

Note: The unit of observation is the birthyear-state-MSA-gender-race cell. Cells are weighted proportional to cell count. All regressions also included the covariates: female; age dummies; African American; other race. Less than 1 year old is the omitted age group. All standard errors are clustered at the state-MSA level. Stars indicate: *** p<0.01, ** p<0.05, * p<0.1