The Health Economics of Macrosomia

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Abstract

High birth weight (also known as macrosomia) is a problem that has as of yet received little attention by health researchers, in particular, health economists. High birth weight is a concern mostly due to the increased difficulties it presents during birth for both the mother and the baby but there is also concern that high birth weight may continue to present negative effects later in the baby's life. Many factors have been attributed as risk factors for high birth weight including mother's age, ethnicity, parity, obesity, weight gain during pregnancy, infant gender, and gestation length. However, there is a dearth of careful analysis dedicated to determining the extent of causality of these risk factors where endogeneity concerns are present. In this thesis, I examine various issues surrounding high birth weight. I describe the situation in New Zealand (Chapter 2) to see if our experience with high birth weight reflects that found in international research. I analyse the relationship between socio-economic status and high birth weight (Chapter 3) to explore whether high socio-economic status has a unique effect on high birth weight compared to other health disorders in which it generally helps alleviate the incidence. I further investigate the relationship between obesity and high birth weight (Chapter 4) in an attempt to disentangle the causal effect of obesity on high birth weight risk from the mere correlation that has been well documented. I explore the possibility of vitamin and mineral supplements taken during pregnancy being a risk factor for high birth weight (Chapter 5), then address the potential endogeneity issues to identify a causal impact. Finally, I return to the definition of high birth weight (Chapter 6) and consider the optimal way to define the "problematic" weight threshold and whether this threshold should depend on gestation length or the ethnicity of the mother.

My findings suggest that in New Zealand, the incidence of macrosomia varies by the ethnicity and weight group of the mother and the gender of the infant. Socio-economic status

does seem to affect high birth weight risk but the nature of the relationship is complex. Obesity only appears to have a significant causal effect on high birth weight risk for women who are morbidly obese, but even for these women conventional estimation that disregards the endogeneity of obesity greatly exaggerates the effect. There does appear to be a correlation between iron supplementation and high birth weight risk but the relationship does not withstand controlling for endogeneity. My findings indicate that the currently accepted threshold used to define macrosomia is justified as it does consistently predict adverse health outcomes. However, flexible definitions which consider different grades of macrosomia or different thresholds for different ethnicities could improve on the current definition.

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Contents

List of Abbreviations

2SLS – Two-Stage Least Squares

BMI - Body Mass Index

CAU – Census Area Unit

CDC - Centre for Disease Control and Prevention

DMHDS – Dunedin Multi-Disciplinary Health and Development Study

DHB – District Health Board

ECLS - Early Childhood Longitudinal Study

HBW – High birth weight

LBW – Low birth weight

MAT - Maternity National Collection

NBW – Normal birth weight

NICU – Neonatal Intensive Care Unit

NDF – Natality Detail File

NLSY - National Longitudinal Survey of Youth

OLS – Ordinary Least Squares

PRAMS - Pregnancy Risk Assessment Monitoring System

SES – Socio-economic status

TLA – Territorial Local Authority

1. Introduction

Macrosomia is a condition where a baby is exceptionally large at the time of birth; it is also referred to as high birth weight[1]. Because of the larger size of the baby, some of the inherent risks of childbirth can become more pronounced and there is also evidence of associated health problems for the child beyond the effects of labour. Macrosomia has been the subject of increasing attention in the medical literature but has of yet received little notice from Health Economists. There also seems to be a dearth of awareness of macrosomia in New Zealand. My thesis focuses on issues relating to macrosomia. Specifically, I address five key questions: 1. What is the situation of macrosomia in New Zealand?; 2. Is socio-economic status correlated with macrosomia risk and, if so, how are they related?; 3. Is maternal obesity causally related to the incidence of macrosomia?; 4.Does taking prenatal vitamin supplements increase the risk of macrosomia?; and 5. How should macrosomia be defined? I also pose two questions as potential avenues for future research: what effect does weight gain during pregnancy have on macrosomia risk? and what are the long term effects of macrosomia?

Background Literature

Incidence and Trend

Determining the incidence of macrosomia can be a difficult task as the weight threshold at which a baby is considered macrosomic has not been universally agreed upon (discussed further in Macrosomia Definition). In the UK, approximately 9% of births have a weight of 4,000g or higher [1]. In Australia, approximately 2.2% of births weigh over 4,500g, a proportion that has risen over time [2]. To my knowledge, there has been no study in recent

[1] I use the terms ‘macrosomia’ and ‘high birth weight’ interchangeably throughout my thesis.

time specifically examining the overall incidence and trend of macrosomia in New Zealand. Data from the Ministry of Health reveals that in 2010, 2.5% of births in New Zealand had birth weights of 4,500g or more. To help rectify this apparent gap of information I conduct a descriptive overview of the macrosomia situation in New Zealand.

Risk Factors

A number of risk factors for macrosomia have been identified in the medical literature. Maternal pre-pregnancy Body Mass Index (BMI) has been found to be associated with macrosomia in a number of studies [2, 3]. However, many of these studies rely on small sample sizes and do not specifically address the degree of causality. For instance, in the presence of unobservable factors that increase the prevalence of both obesity and macrosomia, the causal effect of obesity on macrosomia may be exaggerated. I investigate this relationship, paying specific attention to teasing out the causality in one of my chapters. Weight gain during pregnancy is also commonly found to be associated with macrosomia[4] [5]. I was unable to specifically research this relationship in my thesis but expand on the possibility of how future research could be devoted to this topic.

Other factors that have been associated with increased incidence of macrosomia include: non-smoking [2, 6, 7], higher maternal age [4, 6, 7], previous macrosomic birth [2, 4, 6, 7], male infant [2, 4], higher gestational age [2, 3, 4], maternal diabetes [3, 4, 7], hypertension [3, 4, 7], higher parity [4], and caucasian ethnicity [2]. However, not all of these factors are consistently found to be associated with macrosomia. For example, Ju, Chada, Donovan and O'Rourke (2009) found no association between macrosomia and maternal age, parity or gestational diabetes.

A specific question that I am interested in addressing in one of my chapters is whether prenatal vitamin supplements have any causal effect on the incidence of macrosomia. A

number of studies have investigated the effect of different vitamin supplements on birth weight and found a positive association. However, no study to my knowledge has looked at whether the foetal growth that is aided by vitamin supplements may have the detrimental effect of increasing macrosomia incidence. In particular, iron supplementation is recommended for foetal growth and a number of studies have shown a positive correlation between iron supplementation and birth weight [8, 9, 10] but have not investigated high birth weight. However, some studies have also found no correlation between iron supplementation and birth weight [11].

There is some evidence to suggest that indicators of higher socio-economic status such as educational attainment and marital status are *positively* correlated with the risk of high birth weight [4, 7, 12]. However, one paper that specifically looked at the relationship between socio-economic status and high birth weight concluded that there was no correlation[13]. I explore in depth the relationship between socio-economic status and high birth weight.

Macrosomia Health Complications

Plentiful research suggests giving birth to babies over 4,500g carries significant risks to both the infant and the mother. For example, an increased risk of shoulder dystocia[2] has been identified by a number of studies [2, 14, 15]. Other foetal afflictions such as still birth, Erb's palsy, neonatal jaundice, and respiratory distress have been found to be more common in high birth weight babies than normal weight ones[3]. High birth weight is also associated with an increased risk of infant mortality [7, 16]. Delivering a high birth weight baby also leads to maternal complications such as vaginal, perineal and cervical tears [17].

Moreover, macrosomia has been shown to have long-lasting effects. A study of 33,413 infants born in Jerusalem found that high birth weight babies were more likely to be

[2] Shoulder dystocia is a condition when the baby's shoulder gets stuck or has significant difficult passing through the birth canal. It is identified when the baby's shoulders fail to deliver shortly after the head.

overweight in adolescence [18]. This finding is supported by research from the Nurses Health Study (a panel data set of over 120,000 women followed since the mid-70s in the US) which shows a positive relationship between high birth weight and adult body mass index [19].

Mei, Grummer-Strawn and Scanlon(2003) tracked children of low, normal, and high birth weight up to age five and found that high birth weight children had the highest proportion of overweight compared to all other categories[20]. Specifically, 31% of children who were of high birth weight and were in the 95th percentile of weight for height up to 11 months were overweight as 4-5 year olds. I was unable to obtain data that would allow me to further examine the long-term effects of high birth weight in my thesis. However, I describe potential research that could be undertaken in this line of research.

Debate around Macrosomia Definition

There is little consensus in the literature on how macrosomia should be defined. Some studies use 4,000g [3], some 4,500g [2, 14, 15], some 5,000g and some a measure of 'large for gestational age' (LGA) if the infant is in the 90th percentile of birth weight for babies of the same gestational age. The American College of Obstetricians and Gynecologists defines macrosomia as infants above 4,500g regardless of other variables such as gestational age. Berard et al. (1998) analyse the outcomes of 100 births to babies over 4,500g and conclude that elective caesareans should only be recommended for babies expected to weigh over 5,000g as the risk of complications from vaginal birth (with particular focus on shoulder dystocia) is low up to the weight of 5,000g [14]. Boulet et al. (2003) propose the use of three grades to define macrosomia for births greater than 4,000g, 4,500g, and 5,000g as grades 1, 2, and 3 respectively as they find unique risk levels associated with each grade [7].

Another relevant issue is whether or not the definition of macrosomia should be specific to an ethnic group. Differing incidences of macrosomia have been found in the United States

across ethnic groups with Native Americans being at the highest risk followed by Whites, Hispanics, Blacks and then Asian subgroups [4]. Other studies, however, have found Hispanics to have higher prevalence of macrosomic births among obese mothers [21]and amongst mothers with gestational diabetes [22]. In New Zealand, Sinclair, Rowan and Hainsworth (2007) analysed a cohort of macrosomic babies born in 2003 [15]. They found that macrosomia was less common in Asian women and more common in Polynesian women compared to their representation in overall births. Maori and European women had rates of macrosomia insignificantly different from their representation in overall births. They also found that the macrosomic infants of Asian women were more likely to suffer from shoulder dystocia and other morbidity whereas the macrosomic infants of Polynesian women were less likely to suffer these afflictions suggesting that the appropriate cut-off weight (they used 4,500g) may differ across ethnic groups. McCowan and Stewart (2004) find additional support for the findings in Sinclair, Rowan and Hainsworth (2007) within a New Zealand cohort of 10,292 births: Samoan and Tongan babies were significantly heavier and Chinese and Indian babies significantly lighter than Maori and European babies [23]. McCowan and Stewart (2004) speculate that their findings could imply that babies of ethnicities that tend to have higher average birth weights may be under-developed at weights that would be considered normal if universal cut-offs are used and therefore may not receive adequate care, however, they do not provide evidence of this. This is an important distinction, as variance in average birth weights across ethnicities does not necessarily imply that negative consequences appear at different weight cut-offs across ethnicities too. I examine the definition of high birth weight, and specifically address the desirability of ethnicity-specific definitions.

Data

In this thesis, I use data from a number of sources including both New Zealand specific and international data. The following datasets are used in at least one of my chapters:

Maternity National Collection

The Maternity National Collection (MAT) is a dataset of all births in New Zealand from July 2000 to the present. It contains data on the mother's demographics, antenatal conditions, delivery procedures, and birth outcomes. Antenatal information in MAT includes mother's BMI and smoking status; date and trimester of registration with a Lead Maternity Carer (LMC), specialist referrals, and hospital admissions. For the birth, MAT holds hospital event data including delivery type, delivery-related procedures, and delivery outcomes for the mother. MAT also collects information about live born babies including: gestation, birth weight, the Apgar score, and any perinatal conditions identified at birth.

Socio-economic status information is available in the MAT through a measure of deprivation of the meshblock area the woman resides in. This is based on the NZ deprivation index, where measures of deprivation on a scale of one to ten are assigned for meshblock areas that contain on average about 90 people. The measure captures eight dimensions of socio-economic status: household income below a defined threshold, receiving a means-tested benefit, home ownership, single parenthood, employment, education, household crowding, access to a telephone, and access to a car.

The data from the MAT can also be linked to data from the pharmaceutical dispensing dataset (PHARMS) to identify any prenatal (or other) vitamins that have been prescribed to the woman during pregnancy.

The advantages of this dataset are that it contains a large sample (all available births over an 11 year period), includes most of the variables of interest, and is specific to New Zealand. It

also has a very high linkage rate between the mother and her baby - around 97% in the last 5 years.

One drawback of this dataset is that it does not contain information on weight gain during pregnancy (BMI is only measured at one point in pregnancy) and it also misses information on individual-level socio-economic factors such as education and income. A significant drawback is that data quality and completeness varies over the sample period due to changes in reporting and funding. In particular, due to a change in funding in July 2007, DHB-funded midwives/midwifery teams no longer report primary maternity care data to the Ministry of Health. This means that there is incomplete information in MAT for women receiving their primary maternity care from DHB-funded (i.e. not self-employed/community) midwives, roughly 50% of births. This leaves little option for my research but to omit women who choose DHB-funded midwives as their LMC after 2007 from analyses as there is not information for all relevant variables. If there are systematic differences in the relationships I am investigating between women who choose DHB-funded midwives over other LMCs the results I extract from regressions using this dataset may not be generalisable to all women. However, this is unlikely to be a significant problem as there is no compelling reason to believe that such differences would exist beyond factors I am able to control for. Mean comparison (Table 1.) shows that women who choose a DHB-funded midwife are significantly younger, more likely to live in urban areas and areas with higher deprivation, less likely to be European. However, there is no significant difference in the likelihood of high birth weight between the two groups.

Natality Detail File

The Natality Detail File (NDF) is a dataset from the US that covers all births in the United States dating back to 1968 and available up to 2009. In 2003, NDF began recording mother's

pre-pregnancy weight. As this is an important risk factor for high birth weight, I use only data from 2003 to 2009.

The large population of the US and the high coverage of the data allow for an enormous sample; roughly 4 million births a year over 7 years of data allows for about 28 million potential data points. The dataset contains important variables for my research including birth weight and other birth outcome measures, pre-pregnancy weight, and demographic and socio-economic status variables such as ethnicity, marital status, and education.

Unfortunately for the purposes of my research, the NDF data has undergone many revisions over time with not all states revising at the same time. For example, in 2003, NDF changed the variable for smoking to include information on how much was smoked by the mother during each trimester as opposed to just overall, but only 24 states adopted the new definition making comparisons difficult. NDF also does not include data on height, so the information on weight cannot be accurately used to infer obesity. Finally, the dataset does not include information on vitamin supplementation.

The Pregnancy Risk Assessment Monitoring System

The Pregnancy Risk Assessment Monitoring System (PRAMS) is a survey run by the US Centre for Disease Control and Prevention (CDC) annually from 1988 to 2008 over 44 states which each sample between 1,300 and 3,400 women.

The major benefit of this dataset is that it includes information unavailable in the NDF such as height which allows for a calculation of BMI, and socio-economic variables beyond NDF such as household income.

Drawbacks of the PRAMS include: less precise information than in NDF (e.g., birth weight grouped rather than given in grams), a smaller sample, and not all variables available for all

states. There are also substantial gaps in state/year groups; most years cover fewer than 30 states.

Methods

Initially, I define macrosomia according to the most common definition, birth weight >4,500g. In the final section I scrutinise the appropriateness of this definition. It is necessary to exclude multiple births from the following analyses as firstly multiple births have unique issues of pregnancy and birth that are outside of the scope of this research and secondly as multiple births are themselves rare and tend to have markedly lower birth weights [24] the occurrence of macrosomic multiple births would be vanishingly rare. Unless otherwise stated, low birth weight (<2,500g) babies are also excluded from analyses as the counterfactual should be that of healthy-weight babies. In the datasets I use there is also a lack of information on pregnancies resulting in a still birth; often the still born baby does not have his/her weight measured. Due to the sparse information on still births I have to drop these observations from my analyses and only focus on live births.

In most of my chapters, I rely on multivariate regression analyses to establish relationships between high birth weight and other variables of interest. Multivariate analysis is an important tool to allow for the direct relationship between two variables beyond the influence of confounding variables to be uncovered. As many things have been identified as being risk factors for high birth weight it is important to control for many of these risk factors so that the relationship of interest can be precisely identified. With this in mind, for most of the regression analyses I undertake in my chapters, it is necessary to control for demographic variables such as the age, ethnicity, and the number of previous births of the mother and the gender of the infant. However, even though they are highly correlated with high birth weight,

I generally do not control for either gestation length or the incidence of gestational diabetes. The reason for this is that these variables seemingly indicate intermediate outcomes rather than inputs into infant health and would therefore encounter serious multicollinearity problems which could obfuscate the interpretation of the relationship between high birth weight and the variables of interest. Since my aim is to determine how particular variables affect the risk of high birth weight overall I want to be able to observe the effect regardless of the particular mechanism which may be driving it (e.g. gestational diabetes or gestation length). Gestational diabetes, for example, is evidenced to be correlated with high birth weight risk, but could also plausibly be highly correlated with major variables of interest such as obesity and socio-economic status indicators. Excluding gestational diabetes as a control variable allows me to determine the overall effect of these variables on high birth weight risk regardless of the presence of gestational diabetes. Gestation length is highly correlated with high birth weight; however, given that the problems associated with high birth weight are generally related to the difficulty of giving birth to such a large sized infant it does not seem highly likely that gestation length will greatly influence the negative effects of high birth weight. I revisit this assumption when looking at the definition of high birth weight.

Outline of My Thesis

My thesis addresses five topics of research: macrosomia in New Zealand, socio-economic status and macrosomia risk, obesity and macrosomia risk, prenatal vitamin supplements and macrosomia risk, and macrosomia definition and consequences. It also proposes two related prospects for future research: weight gain during pregnancy and macrosomia risk, and the long term consequences of macrosomia.

In Chapter 2, I explore New Zealand's experience with macrosomia. There appears to be a dearth of information and awareness about macrosomia in New Zealand. Hence, an overview of the situation by examining descriptive statistics from New Zealand birth data is a useful pre-cursor to a more thorough analysis pertaining to macrosomia in New Zealand. When looking at macrosomia in New Zealand, I address such specific questions as: How prevalent is macrosomia in New Zealand?; Has the rate of macrosomia been increasing over time in New Zealand?; and How does New Zealand's incidence of macrosomia compare with the existing international figures? The analysis in this chapter is purely descriptive; in the following chapters I employ more sophisticated techniques to isolate correlation and causality.

In Chapter 3, I investigate the relationship between socio-economic status and macrosomia risk. Socio-economic status is an important predictor of many health outcomes. However, due to the parabolic relationship between birth weight and pregnancy outcome (birth weight at both the low and high extremes presents risks relative to moderate weight) it is possible that the beneficial role of high socio-economic status in avoiding negative health outcomes may be less apparent or even reversed in the case of macrosomia. This could be apparent due to socio-economic status having the beneficial effect of reducing the risk of low birth weight which may present as an increase in birth weight overall, therefore increasing high birth weight risk. I explore plausible hypotheses to explain the nature of the relationship between socio-economic status and high birth weight risk. Evidence from Cesur and Kelly (2010) suggests that there may be no correlation between socio-economic status and macrosomia risk [13]. This is a remarkable finding given the strongly influential effect socio-economic status has on other health outcomes. I investigate the correlation between macrosomia and specific socio-economic variables such as household income, educational attainment, and marital status, holding other relevant variables constant.

Chapter 4 seeks to determine the extent of causality between obesity and high birth weight risk. Although there is plentiful evidence in the existing literature of a strong correlation between obesity and high birth weight, no research, to my knowledge, has addressed the seemingly pervasive endogeneity. Therefore, it is highly likely that the causality between obesity and high birth weight risk has been exaggerated. Identifying the extent of a causal relationship is important as policy or medical advice for women to obtain an ideal body mass potentially through weight loss before conception in order to avoid high birth weight may be misguided. If the correlation is merely driven by underlying factors such as genetic predisposition towards higher adiposity then weight loss before conception will not have a beneficial effect of lowering high birth weight risk. I utilise instrumental variable techniques to isolate the causal effect from the correlation.

Chapter 5 inquires as to whether the use of prenatal vitamin supplements to promote foetal growth has a partially detrimental effect by increasing macrosomia. This is a novel area of research. Many articles have examined the effect of supplements on low birth weight risk, yet none, to my knowledge, have addressed the effect it might have on birth weight at the higher end. The findings potentially have important implications for guiding prescribing practice for pregnant women and for subsidisation policy. I use multivariate analysis to establish if there is a risk and instrumental variable analysis to address potential endogeneity concerns.

Chapter 6 revisits the definitions of macrosomia. I examine the relationship between weight cut-offs and the Apgar score, birth injury, ICU admission, and assisted ventilation. I compare this to a continuous non-linear definition of birth weight to see if the relationship between birth weight and other birth outcomes can be modelled as a parabolic function. In light of evidence that ethnicity is an important predictor of high birth weight (described above) I also explore inclusions of ethnicity and birth weight interactions to see if the hazards of high birth weight vary across ethnic groups as the incidence does. Finally, I include gestation length and

high birth weight interactions to identify if high birth weight at different gestation lengths carries differing risk.

Chapter 7 presents two avenues for future research that have a potential to deliver interesting findings but which I have been unable to pursue due to limitations in data availability. The topics are: the effect of weight gain during pregnancy on high birth weight risk, and the long term effects of high birth weight. Weight gain during pregnancy has been previously identified as a risk factor for high birth weight; however, like obesity, it is highly likely to suffer from endogeneity and hence the causal effect may be much less prominent than findings from the previous literature imply. This has important implications for guiding pregnant women about appropriate levels of weight gain. Research on the long term effects of high birth weight is sparse and a careful analysis of causality would be of great value.

Chapter 8 concludes with an overview of the contributions my research has made to our understanding of high birth weight and the implications for policy.

2. Macrosomia in New Zealand

Introduction

Macrosomia or high birth weight is defined as birth weight greater than 4,500g. It is a problem that has received relatively little attention from health researchers but is gaining prominence as its incidence has risen with the obesity epidemic. Using data from the Maternity National Collection from 2001-2011, I describe the nature of the macrosomia problem in New Zealand including the incidence over time and how it compares to other countries, demographic factors that influence macrosomia risk and how birth outcomes differ for macrosomic births compared to normal weight births.

Data

As discussed in Chapter 1, MAT is a dataset of all births in New Zealand from which I am using data from 2001 to 2011. For this chapter, the data on the mother's demographics, antenatal conditions, delivery procedures and birth outcomes is the most relevant. In particular, I utilise hospital event data including delivery type, delivery-related procedures, and delivery outcomes for the mother and information about live born babies including: gestation, birth weight, the Apgar score and any perinatal conditions identified at birth.

Results

At around 2.5% of births, New Zealand's incidence of macrosomia is somewhat high in comparison to other developed nations. Ju et al. (2009) report a rate of 2.2% of births having birth weight over 4500g[2]. In 2010 1% of births in the US had a birth weight over

4500g[25]. A broad international comparison is difficult due to the sparse availability of data on birth weights and the relative lack of awareness about high birth weight as a health issue.

There has been no obvious change in macrosomia incidence in New Zealand over the past decade (Figure 2.1). In particular, there was a minor increase in incidence between years 2001 and 2008 but a reversion afterwards.

Macrosomic births are much more prevalent in women with higher body mass (Figure 2.2). Body Mass Index values are calculated using the women's weight divided by her height squared[3]. Grouping women into BMI categories of underweight (BMI<20), normal weight (BMI 20-25), overweight (BMI 25-30), obese (BMI 30-35), and morbidly obese (BMI>35) reveals that macrosomia risk increases markedly with each increase in weight group. Obese women are roughly twice as likely to have a macrosomic baby compared to normal weight women.

The incidence of macrosomia in New Zealand is much higher for Pacific women than any other ethnicity (Figure 2.3). European women have a comparatively high incidence of macrosomia whereas Asian women have an incidence less than half the national average. This is in accord with previous literature which suggests that ethnicity is an important factor in determining high birth weight risk.

Male infants in general tend to be larger so it is perhaps unsurprising that they represent a much larger proportion of macrosomic births compared to normal weight births (Figure 2.4). This is a common finding in the literature: male infants pose a greater risk of high birth weight. Controlling for infant gender is therefore very important when conducting multivariate analyses.

[3] For a more detailed discussion of BMI see Appendix A.

Macrosomia is problematic mostly for the complications it presents during childbirth (Figure 2.5). Women giving birth to macrosomic babies have a sizeably increased risk of severe tearing. Apgar scores measure the vital signs of the infant at five minutes after birth on a scale of one to ten, where any number less than seven is indicative of problems. Macrosomic babies are more likely to have an Apgar score less than seven. Caesarean births in general present more danger to the mother and baby, macrosomic births are more likely to need caesareans whether by elective decision or emergency. Surprisingly, the use of forceps or vacuum during birth is lower for macrosomic births. However, this is likely due to high risk births more likely ending with a caesarean.

Discussion

New Zealand's experience with macrosomia appears to be similar to other developed nations; however, accurate comparisons are hindered by limited availability of comparable data. Also, since ethnicity appears to have such an important impact on macrosomia incidence, cross country comparisons likely reflect in large part the demographic composition of the nations. The incidence of macrosomia in New Zealand does not appear to be increasing as the literature suggests is possibly occurring. Ethnicity, body mass, and infant gender all appear to be strongly correlated with macrosomia risk; however, more advanced techniques such as multivariate and instrumental variable analyses are required to tease out the direct and causal effects of these factors.

3. High Birth Weight and Socio-Economic Status

Introduction

Socio-economic status is highly positively correlated with a number of "good" health outcomes. In a seminal paper, Grossman (1972) modelled education as increasing health outcomes due to educated people being more informed about health inputs and using inputs more effectively [26]. As health is a normal good, health inputs such as preventative medical care that allow us to 'produce' good health are generally considered to be normal goods also; as income increases, demand for health increases, therefore derived demand for health inputs increases leading to better health outcomes. Marital status is also linked to better health outcomes, arguably because married people have more incentive and ability to invest in health inputs. Although the causality from socio-economic status indicators to generally positive health outcomes may be debateable, the correlation is irrefutable. This correlation can be seen clearly when it comes to the likelihood of giving birth to a low birth weight baby. Numerous studies in the health economics literature have found a strong negative relationship between socio-economic status and the incidence of low birth weight [27, 28, 29] . This relationship holds even when controlling for confounding factors such as age, ethnicity, and parity.

As discussed earlier, macrosomia is a serious health issue due to the increased risk of injuries during birth and the possibility of long term consequences for the infant. The propensity of high socio-economic status people to avoid negative health outcomes could, in theory, also demonstrate itself in a lower incidence of high birth weight births. However, the available evidence does not support this conjecture.

The medical literature has largely ignored the effect of socio-economic status on the likelihood of giving birth to a high birth weight baby. To my knowledge, no studies have addressed the effect of income or wealth on the risk of high birth weight. The effects of education and marital status on high birth weight have been considered by some studies and have been found to be *positively* correlated with high birth weight[4, 7, 12].

To my knowledge, Cesur and Kelly (2010) is the only economics study examining high birth weight [13]. The authors estimate the effect of high birth weight on cognitive outcomes during childhood. To rule out omitted variable bias, they examine the relationship between high birth weight and socio-economic status. They find that although socio-economic status is highly correlated with low birth weight it is not correlated with high birth weight, with or without controlling for confounding factors.

This chapter endeavours to explore the relationship (or lack thereof) between high birth weight and socio-economic status and to investigate the magnitude and robustness of any relationship to additional controls. I first propose a model where the probabilities of giving birth to a baby with low, normal or high birth weight are a monotonic function of maternal demographic characteristics and purposeful actions undertaken by the mother (e.g. stress avoidance, quitting smoking, eating adequately, etc.). Under reasonable assumptions about the effect of wealth, education, and marital status on the demand for actions, I show that increasing socio-economic status increases the amount of actions undertaken by the mother which in turn can either decrease or increase the likelihood of having a high birth weight baby. I introduce these outcomes as two competing hypotheses about the effect of socio-economic status on the incidence of high birth weight.

My empirical analysis uses data from the Pregnancy Risk Assessment Monitoring System and the Natality Detail File. As discussed in the introduction, PRAMS is a survey run by the

US Center for Disease Control and Prevention annually from 1988 to 2008 over 44 states sampling between 1,300 and 3,400 women within each cohort. The NDF covers all births in the United States and includes information on education and marital status. I use data from 2003 to 2009 (substantial revisions to the way data was collected were undertaken in 2003 and 2009 is the last year of data available).

In the empirical section below, I first examine descriptive statistics to determine whether major socio-economic indicators such as income, education and marital status appear to be correlated with the prevalence of high birth weight. I then attempt to replicate the findings of Cesur and Kelly (2010) and proceed to test the robustness of any relationship found to additional controls and explore what factors seem to be driving the relationship.

Descriptive statistics indicate that income, education and being married are *positively* correlated with the incidence of high birth weight. However, this result does not hold when controlling for other factors. Attempting to estimate Cesur and Kelly's (2010) regressions on a different dataset yields some discrepancies with their results. Contrary to their findings (but consistent with my theoretical model), my results from multivariate analysis seem to indicate that socio-economic status is negatively correlated with high birth weight risk however there may be some discrepancies in the relationship when different thresholds of defining high birth weight are considered.

Background Literature

Socio-Economic Status and Health

The relationship between socio-economic status and various health outcomes has been studied extensively by health economists. Generally, high socio-economic status in the form

of higher education, higher income, and/or being married rather than single leads to better health outcomes.

The notion that health and education are positively correlated is well supported; however, the mechanism underlying this relationship is unclear. Poor health may impede learning, therefore lowering education, education may increase health outcomes if education increases awareness about health in general, or a third factor may be related to both health and education. Grossman (1972) described how education may be causally related to health. If educated people are better informed about health inputs they will both use more health inputs and use them more effectively, increasing health outcomes. This hypothesis is supported by evidence from the US [30] and UK [31] using changes in compulsory schooling law as an instrument. Fuchs (1982) proposes another hypothesis that time preference may be a third factor driving the relationship; since education and health outcomes both require substantial investment and delayed payoffs, individuals with high discount rates will have lower investment in both health and education and vice versa.

Income or wealth are generally agreed to be correlated with good health outcomes [32]. However, similar to education, the relationship is not necessarily a direct causal one from income to health. Higher incomes of course allow for greater spending on health inputs, but it may also be the case that poor health interferes with one's earning capacity or that omitted third factors drive the relationship.

Vast evidence in the health literature supports the notion that being married improves health outcomes. Coombs (1991) reviews the literature on the effect of marital status and well-being and finds that available evidence strongly suggests that married people tend to have a lower incidence of illness and greater longevity. This relationship is suggested by some to arise from selection, sickly people are less likely to marry, but Coombs also finds extensive

support for a causal explanation, where protection and support from the spouse improves health outcomes [33].

Many studies in the health literature provide evidence that low socio-economic status is strongly related to a higher likelihood of giving birth to a low birth weight baby. Jonas, Roder, and Chan (1992) examine 12,047 births in Adelaide and find that women residing in low socio-economic areas have a higher chance of having a low birth weight baby and general poor pregnancy outcomes [28]. Pattenden, Dolk and Vrijheid (1999) examine births in England and Wales and find that 30% of low birth weight incidence can be explained by socio-economic status. [27]. Other research shows that higher education [34], income [35], and being married [29] are associated with a lower risk of low birth weight.

Socio-Economic Status and High Birth Weight

Few studies examine the effect of socio-economic status factors on the likelihood of giving birth to a macrosomic infant. To my knowledge, no studies have addressed the effect of income or wealth on the risk of high birth weight. The effect of education and marital status has received limited attention. Frank, Frisbee and Pullum (2000) find women with less than 12 years of education were about 20% less likely to have a high birth weight infant than women with more than 12 years [4]. Ourskou et. al. (2003) find women with 10 or more years of education and women who were living with a partner had higher risks of high birth weight [12]. Boulet et al (2003) also find higher rates of educational attainment and marriage for women who delivered babies weighing over 4,000g compared to women who delivered normal weight babies [7] but no multivariate analyses were used to determine if this higher proportion remained after controlling for other factors such as ethnicity.

Cesur and Kelly (2010) examine the effect of high birth weight on cognitive outcomes at a later age. To my knowledge, this is the only study on high birth weight in the health

economics literature. By using mother's pregnancy weight gain, gestational age, and mother's age as instrumental variables, they find that high birth weight had a negative effect on cognitive outcomes. They address the possibility of socio-economic status being an omitted variable that could influence both birth weight and cognitive outcomes but find that socio-economic status was a poor predictor of high birth weight (unlike low birth weight) so conclude it was unlikely to bias their results. This is a surprising finding, due to the large influence socio-economic status has on most health outcomes and, in particular, low birth weight.

Potential Confounders with Socio-Economic Status and Birth Outcomes

There are two potential avenues for socio-economic status to affect birth weight; through the effect on maternal characteristics at conception or through behaviours during pregnancy. Socio-economic status may be related (causally or not) to inherent characteristics of the mother at the time of conception and/or socio-economic status may influence the behaviours undertaken during pregnancy.

High socio-economic status women could plausibly differ in their characteristics that may also influence birth weight. For example, maternal characteristics that could potentially differ with socio-economic status include ethnicity, age, birth order, and weight. Maternal pre-pregnancy BMI has been found to be associated with the incidence of high birth weight in a number of studies [2, 3, 4] and maternal age [4, 6, 7], parity [4], and ethnicity [2] are also important predictors.

Another potential confounder is the gender of the infant. Male infants tend to be larger and evidence shows that this extends to having a higher risk of high birth weight [2, 4]. If left unaccounted for, this could conflate our results as according to Trivers and Willard (1973), the sex ratio (the proportion of male to female infants) can be increased by factors that

enhance reproductive success. Since females tend to marry men of higher socio-economic status than themselves, being born of high socio-economic status has less effect on their ability to find a partner than it does for males. Therefore, a higher socio-economic status at birth improves the reproductive success for males by a greater factor than for females and we would expect to see a positive relationship between socio-economic status and the sex ratio [36]. Almond and Edlund (2007) find evidence to support this so-called "Trivers-Willard Hypothesis"; analysing all births to white mothers between 1983-2002, they find higher education and being married both correlate with a higher likelihood of having a male infant [37].

Birth weight can also be affected by behaviours undertaken during pregnancy. Weight gain during pregnancy is commonly found to be associated with high birth weight [5]. Vitamin intake may increase the likelihood of giving birth to a high birth weight baby; in particular, iron supplementation is recommended for foetal growth and a number of studies have shown a positive correlation between iron supplementation and birth weight [8, 9, 10]. Non-smoking has been associated with an increased incidence of high birth weight [2, 6, 7]. Other factors such as stress avoidance and prenatal care could also plausibly influence high birth weight.

Model

Birth weight can be classified into three categories: low birth weight (LBW; <2,500g), normal birth weight (NBW; 2,500-4,500g), and high birth weight (HBW; >4,500g). Birth weight cannot be perfectly controlled but the actions taken by the mother can influence the probabilities of having a baby in each of the three categories. Certain actions can be undertaken by the mother to reduce the probability of low birth weight, such as quitting

smoking, consuming more calories, taking prenatal-vitamins etc. However, these actions may also have the effect of increasing the probability of high birth weight. Recognising that low, normal, and high birth weight are mutually exclusive categories that encompass all outcomes, we can state:

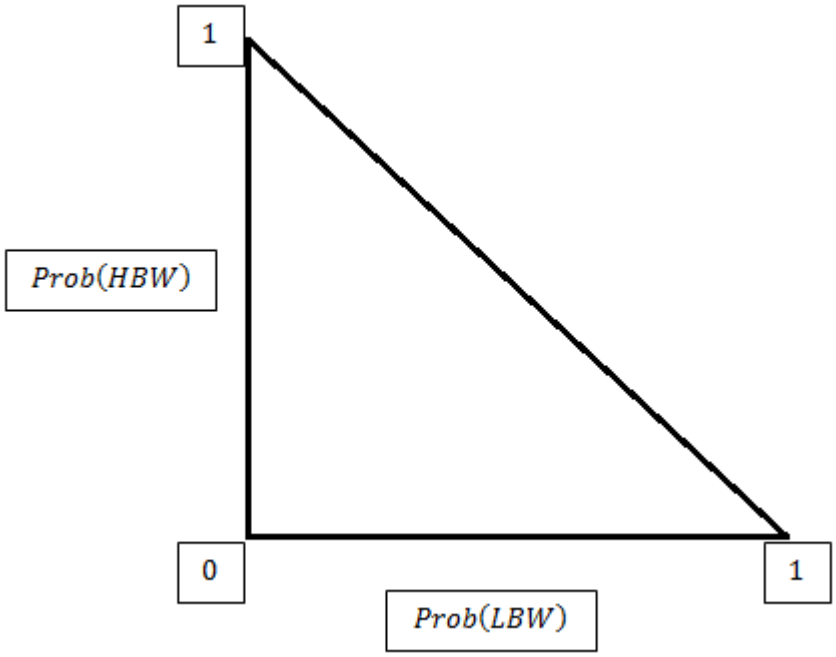
$$Prob(LBW) + Prob(NBW) + Prob(HBW) = 1$$

Expected infant's health can therefore be expressed as:

$$H_{Infant} = Prob_{LBW} \times H_{Infant}^{LBW} + Prob_{NBW} \times H_{Infant}^{NBW} + Prob_{HBW} \times H_{Infant}^{HBW}$$

where H_{Infant}^{LBW}, H_{Infant}^{NBW}, and H_{Infant}^{HBW} are constants representing the effect of low, normal, and high birth weight on infant health, respectively. As there are three different mutually exclusive outcomes with different associated probabilities, we can model the infant's health function using a Marschak triangle [38]. The Marschak triangle demonstrates the range of all possible probabilities of the three outcomes by assigning the probabilities of two of the outcomes on the axes with a line drawn between the two axes at the probability equal to one for either possible outcome. I have assigned the probabilities of low and high birth weight to the two axes, so the probability of having normal birth weight can be calculated by:

$$Prob(NBW) = 1 - Prob(LBW) - Prob(HBW)$$



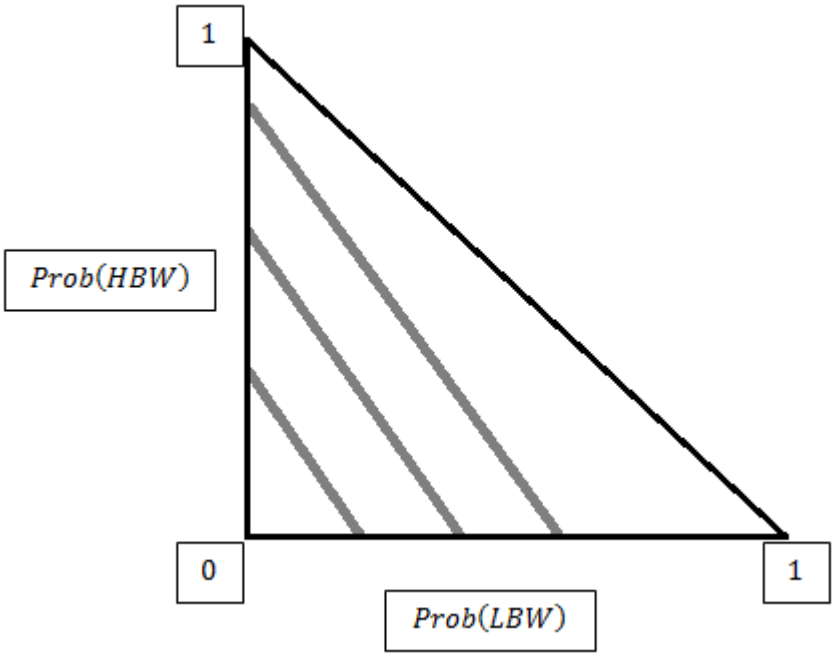
To demonstrate the trade-off between the probabilities of high and low birth weight I can show curves that reflect a constant level of infant health over different values of these probabilities. These are similar to regular indifference curves however as the optimisation function is infant health rather than utility they don't reflect the entire trade-off as low birth weight risk and high birth weight risk may affect the mother's utility function through mechanisms beyond the effect on infant health. The slope of these curves can be found using implicit function theorem:

$$\frac{\partial Prob(HBW)}{\partial Prob(LBW)} = -\frac{\frac{\partial H_{Infant}}{\partial Prob(LBW)}}{\frac{\partial H_{Infant}}{\partial Prob(HBW)}} = -\frac{H_{Infant}^{LBW} - H_{Infant}^{NBW}}{H_{Infant}^{HBW} - H_{Infant}^{NBW}}$$

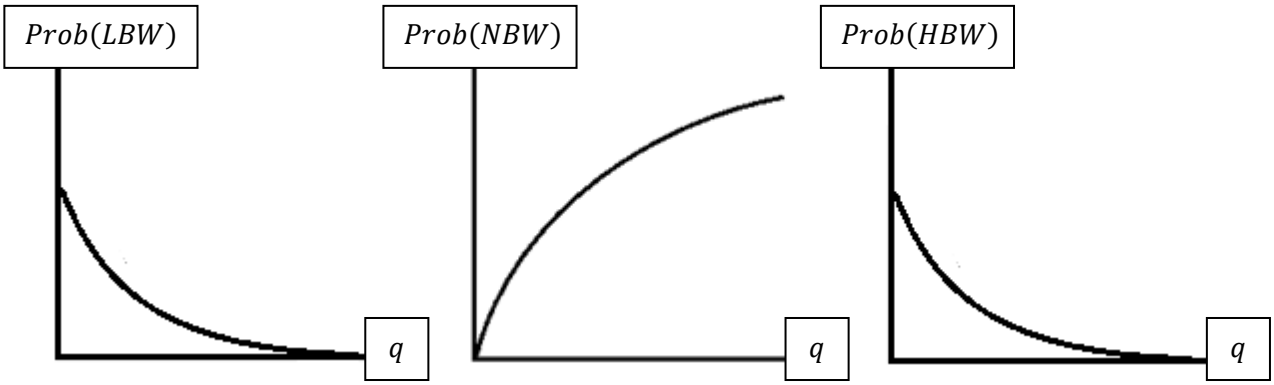
Assuming that low birth weight has a more detrimental effect on infant health than high birth weight, then:

$$\left|H_{Infant}^{LBW} - H_{Infant}^{NBW}\right| > \left|H_{Infant}^{HBW} - H_{Infant}^{NBW}\right|$$

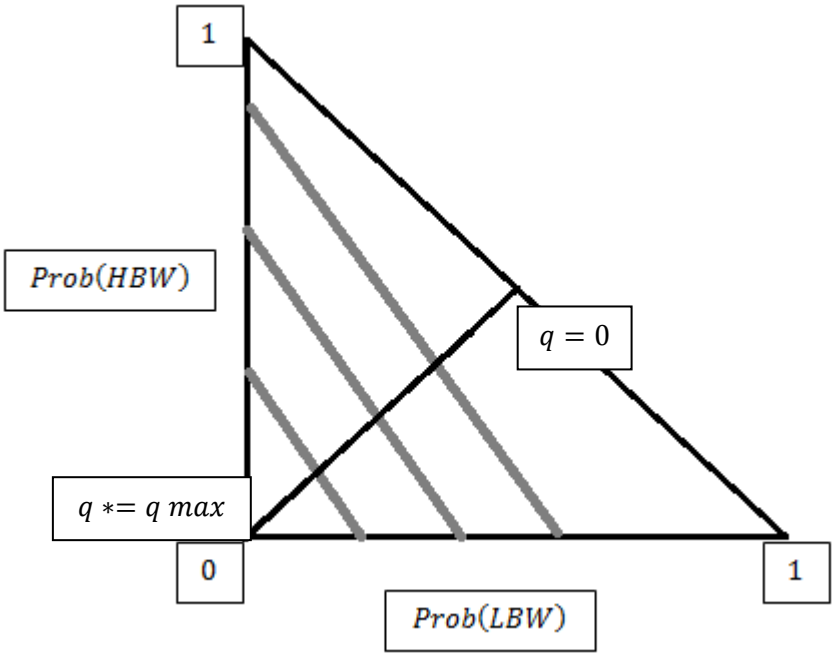
Therefore, the slope of these curves must be steeper than the hypotenuse. We can draw some of these curves on the Marschak triangle:



One possibility is that actions such as using pre-natal care, eating healthier etc. may reduce both the probability of low and high birth weight, and by induction increase the probability of normal birth weight. If we assume that mother's actions during pregnancy have a diminishing effect on the probability of low birth weight we can model the effect of actions (q) on the birth weight probabilities as:

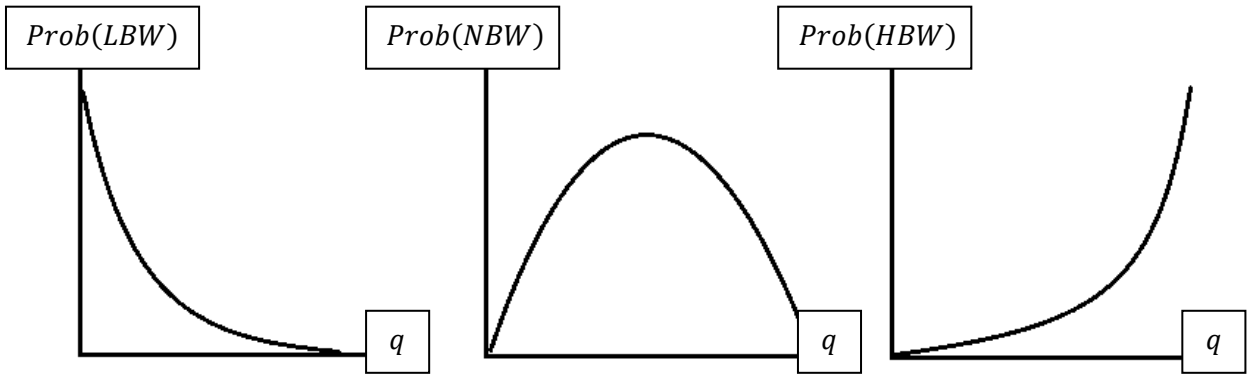


On the Marschak triangle we can find the optimal outcome:

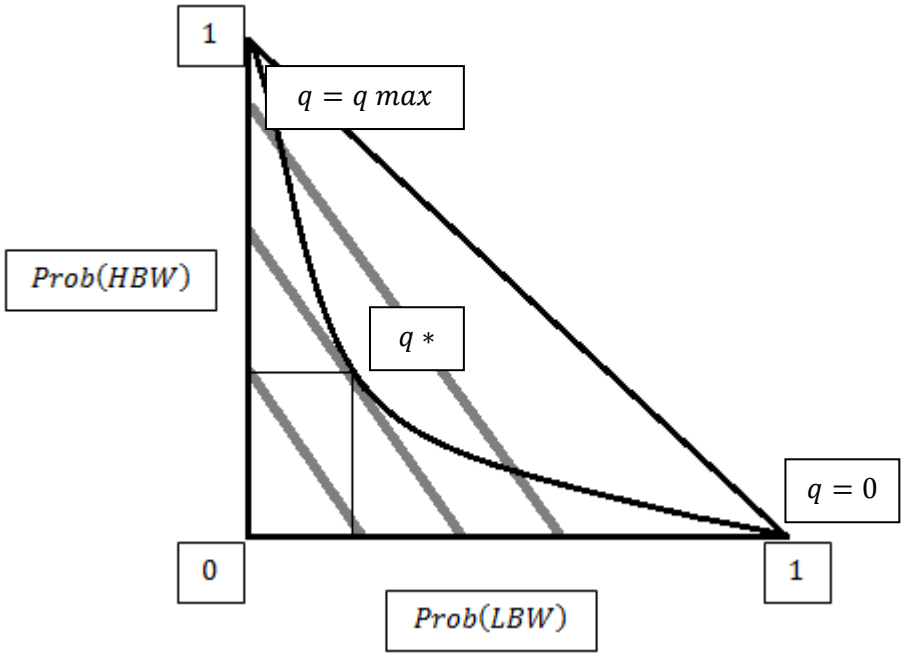


With this assumption, the optimal amount of actions is the maximum actions that can be taken to bring about certainty of normal birth weight.

An alternative assumption is that actions reduce the risk of low birth weight whilst increasing the risk of high birth weight:



This can be shown as a path of q on the Marschak triangle which allows us to find the optimal level of q, given no constraints:



Here we can see that if actions are costless, mothers will choose a level of actions that gives a higher probability of high birth weight than of low birth weight.

This analysis is missing a vital component: the constraints. Actions would not be costless, and mothers would have to decide on the best allocation of their resources across actions and general consumption to maximise their own utility.

Building on the static version of Grossman's demand for health and health capital model [26], I assume that utility of the mother is a function of the mother's own health, her infant's health, and other consumption. This allows me to define an optimisation model:

$$\max_{q,Z} U = U\left(H_{Infant}, H_{Mother}, Z\right)$$

$$s.t.\ H_{Infant} = Prob_{LBW} \times H_{Infant}^{LBW} + Prob_{NBW} \times H_{Infant}^{NBW} + Prob_{HBW} \times H_{Infant}^{HBW}$$

$$Prob_{LBW,NBW,HBW} = f(q, x)$$

$$M \geq Pq + Z$$

Where:

$$H_{Infant} = \begin{cases} H_{Infant}^{LBW}\ if\ LBW = 1 \\ H_{Infant}^{NBW}\ if\ NBW = 1 \\ H_{Infant}^{HBW}\ if\ HBW = 1 \end{cases}$$

$$LBW = \begin{cases} 1\ if\ b < 2500 \\ 0\ if\ b \geq 2500 \end{cases}$$

$$NBW = \begin{cases} 1\ if\ 2500 \leq b < 4500 \\ 0\ if\ b \geq 4500\ or\ b < 2500 \end{cases}$$

$$HBW = \begin{cases} 1\ if\ b \geq 4500 \\ 0\ if\ b < 4500 \end{cases}$$

In this series of equations , H_{Infant} denotes the health stock of the infant, H_{Mother} denotes the health stock of the mother, Z denotes a composite good representing all other consumption, $Prob_i$ represents the probability of giving birth to a baby of low (LBW), normal (NBW), or high (HBW) birth weight, the vector $\boldsymbol{x}$ denotes exogenous characteristics of the infant and the mother that affect birth weight such as ethnicity, region, age, parity etc.,

q denotes actions undertaken by the mother to influence birth weight (e.g., eating, quitting smoking, stress avoidance), M is income and P the cost of actions (q). We assume H_{Mother} is a constant.

Given that the three birth weight outcomes account for all possible outcomes and are mutually exclusive, we can rewrite the infant health function as follows:

$$H_{Infant} = Prob_{LBW} \times H_{Infant}^{LBW} + (1 - Prob_{LBW} - Prob_{HBW}) \times H_{Infant}^{NBW} + Prob_{HBW} \times H_{Infant}^{HBW}$$

$$H_{Infant} = Prob_{LBW} \times \left(H_{Infant}^{LBW} - H_{Infant}^{NBW}\right) + H_{Infant}^{NBW} + Prob_{HBW} \times \left(H_{Infant}^{HBW} - H_{Infant}^{NBW}\right)$$

The marginal utility of actions can be described as:

$$\frac{\partial U}{\partial q} = \frac{\partial U}{\partial H_{Infant}} \cdot \frac{\partial H_{Infant}}{\partial Prob_{LBW}} \cdot \frac{\partial Prob_{LBW}}{\partial q} + \frac{\partial U}{\partial H_{Infant}} \cdot \frac{\partial H_{Infant}}{\partial Prob_{HBW}} \cdot \frac{\partial Prob_{HBW}}{\partial q}$$

$$\frac{\partial U}{\partial q} = \frac{\partial U}{\partial H_{Infant}} \cdot (H_{Infant}^{LBW} - H_{Infant}^{NBW}) \cdot \frac{\partial Prob_{LBW}}{\partial q} + \frac{\partial U}{\partial H_{Infant}} \cdot (H_{Infant}^{HBW} - H_{Infant}^{NBW}) \cdot \frac{\partial Prob_{HBW}}{\partial q}$$

The tangency condition states that:

$$\frac{\partial U}{\partial q} = P \times \frac{\partial U}{\partial Z}$$

Hence $\frac{\partial U}{\partial q}$ must be strictly positive. Since low birth weight and high birth weight have negative health consequences we can say that:

$$(H_{Infant}^{LBW} - H_{Infant}^{NBW}) < 0$$

$$(H_{Infant}^{HBW} - H_{Infant}^{NBW}) < 0$$

Since q decreases the likelihood of low birth weight$\frac{\partial Prob_{LBW}}{\partial q}$ must be negative, therefore:

$$\frac{\partial U}{\partial H_{Infant}} \cdot \frac{\partial H_{Infant}}{\partial Prob_{LBW}} \cdot \frac{\partial Prob_{LBW}}{\partial q} > 0$$

If we assume that actions decrease the risk of high birth weight (i.e., the second scenario above), $\frac{\partial Prob_{HBW}}{\partial q}$ must also be negative, therefore:

$$\frac{\partial U}{\partial H_{Infant}} \cdot \frac{\partial H_{Infant}}{\partial Prob_{HBW}} \cdot \frac{\partial Prob_{HBW}}{\partial q} > 0$$

In this scenario, the level of q chosen will be chosen simply where the utility from foregone consumption is equal to the utility gained from the marginal increase in infant health. Assuming both q and Z are normal goods, increasing income will increase $\tilde{q}$ and therefore decrease the risk of high birth weight. If we assume, like Grossman (1972), that education helps people to use health inputs more effectively, we could model this as P being a decreasing function of education. Hence, with higher education, the cost of undertaking actions is lower. For instance, educated women may have less difficulty researching how to improve birth weight, and therefore the initial search cost is lower. It follows that $\tilde{q}$ would increase with education and therefore decrease the risk of high birth weight. The effect of marital status could be explained by a higher $\frac{\partial U}{\partial H_{Infant}}$. If we assume that pregnancies of married women are more likely to be planned and on average would have a higher 'wantedness' then it is not unreasonable to assume in general the utility gained from a healthy infant would be higher for married women. Therefore, being married would increase $\tilde{q}$, lowering high birth weight risk. Overall, with the assumption that actions decrease high birth weight risk, we find that socio-economic status should decrease the incidence of high birth weight.

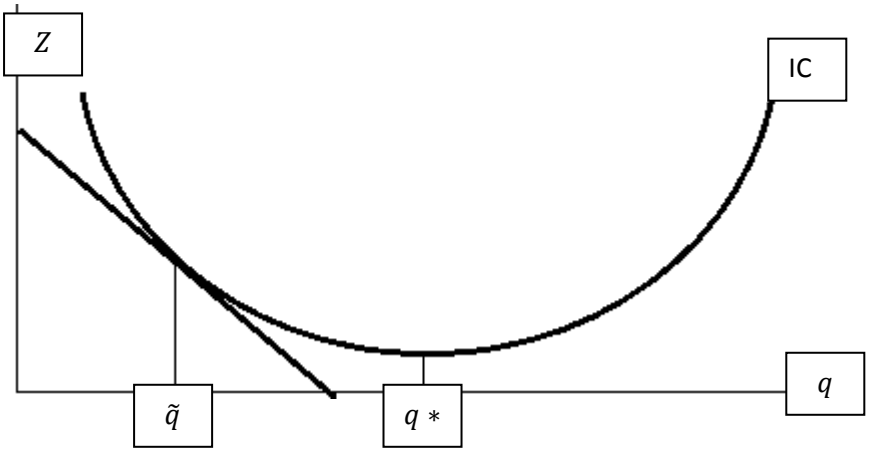
If, however, actions increase high birth weight risk then:

$$\frac{\partial U}{\partial H_{Infant}} \cdot \frac{\partial H_{Infant}}{\partial Prob_{HBW}} \cdot \frac{\partial Prob_{HBW}}{\partial q} < 0$$

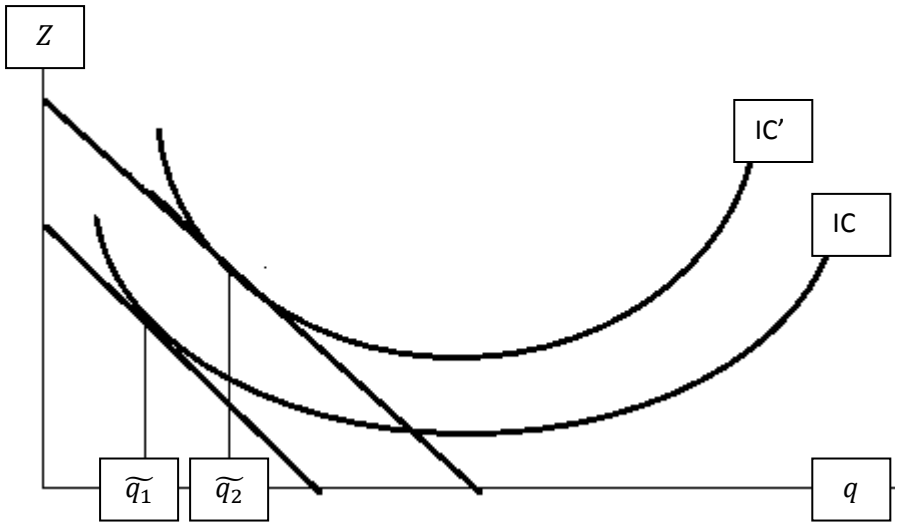
Given that the tangency condition tells us that the marginal utility from actions must be strictly positive we can conclude that:

$$\left| \frac{\partial U}{\partial H_{Infant}} \cdot \frac{\partial H_{Infant}}{\partial Prob_{LBW}} \cdot \frac{\partial Prob_{LBW}}{\partial q} \right| > \left| \frac{\partial U}{\partial H_{Infant}} \cdot \frac{\partial H_{Infant}}{\partial Prob_{HBW}} \cdot \frac{\partial Prob_{HBW}}{\partial q} \right|$$

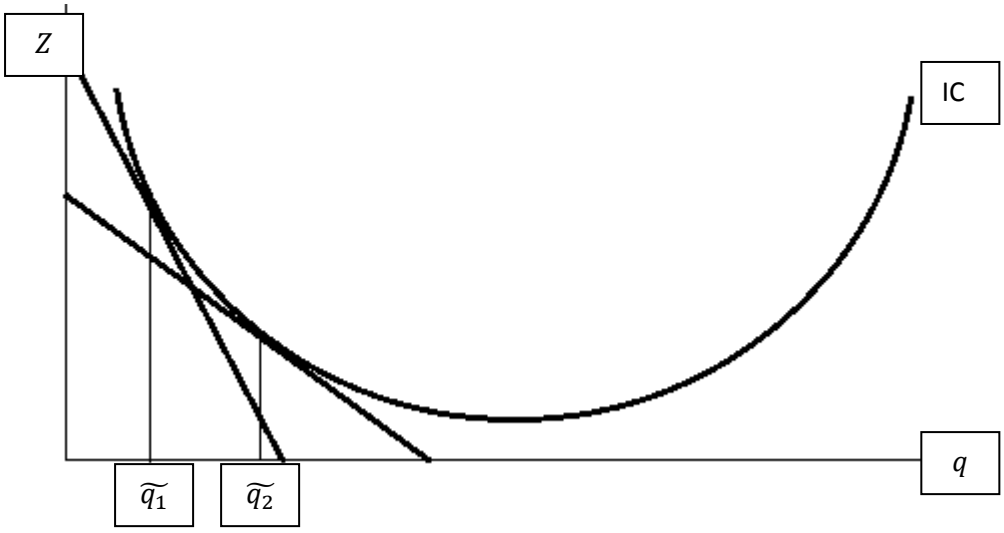
Therefore, the level of q chosen by the mother, $\tilde{q}$, will be lower than the level of q which would offset the negative consequences of low birth weight and hence maximise H_{Infant}. This can be shown with a U-shaped indifference curve, where the lowest point represents the level of actions needed to maximise H_{Infant}:



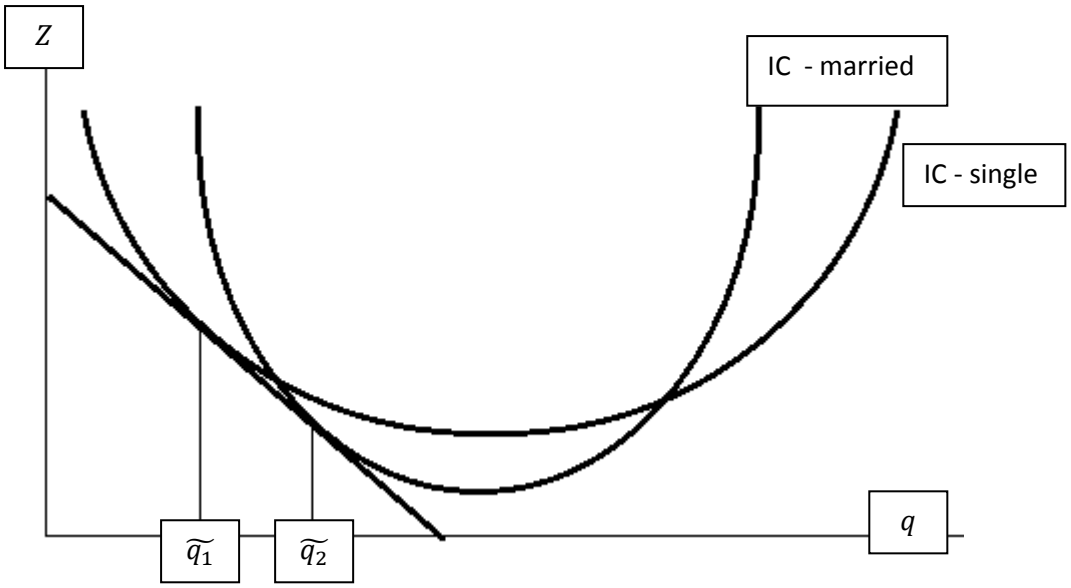
Assuming both q and Z are normal goods, increasing income will increase $\tilde{q}$:



Assuming, as explained above, that education reduces the price of actions we can demonstrate that increasing education will increase the level of actions chosen. This is shown below with the Hicksian substitution effect for a change in the price of q:



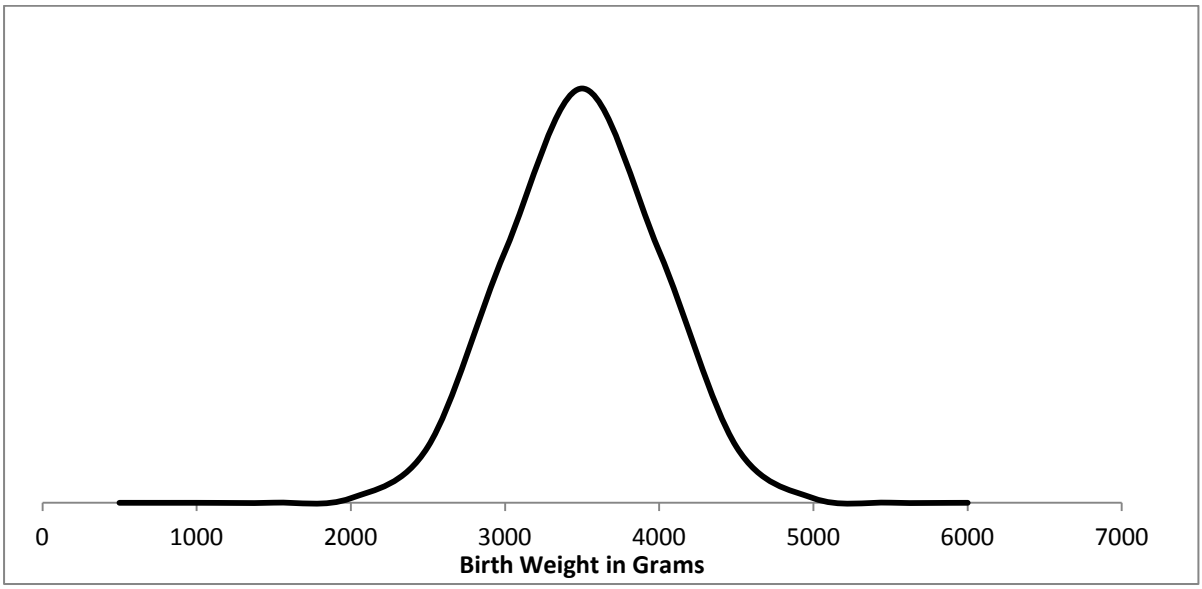
Assuming married women have a higher $\frac{\partial U}{\partial H_{Infant}}$ would produce a narrower indifference curve:



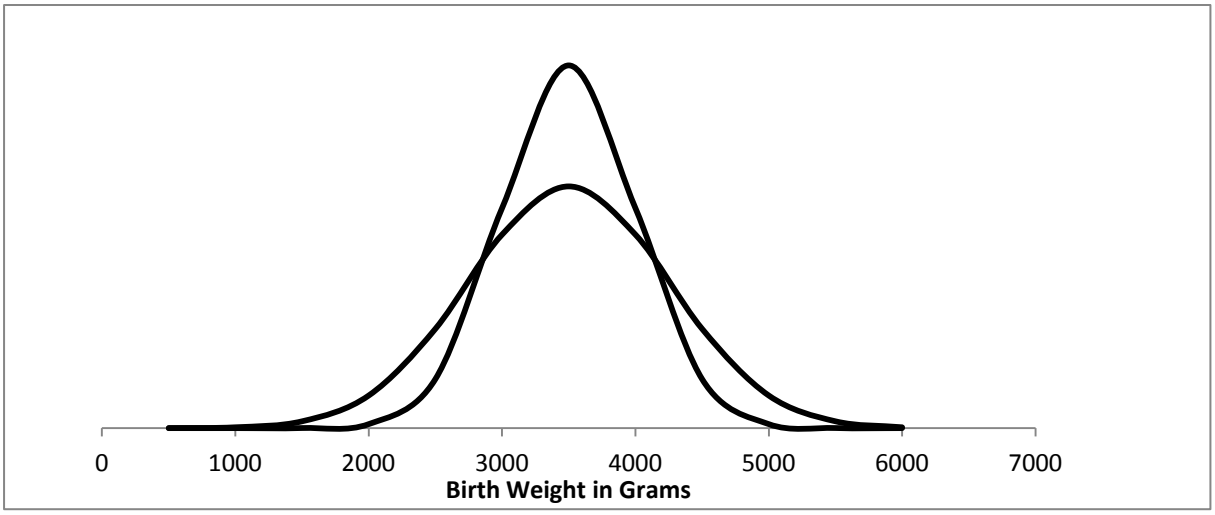
Similarly to the effect of education, being married would increase the level of actions chosen.

Under either assumption about the effect of actions on high birth weight risk, we get the conclusion that socio-economic status will increase the level of actions chosen. Therefore, the effect of socio-economic status on high birth weight risk could be either negative or positive. Either way, we would expect high socio-economic status women to have a lower risk of low birth weight and a higher likelihood of normal birth weight.

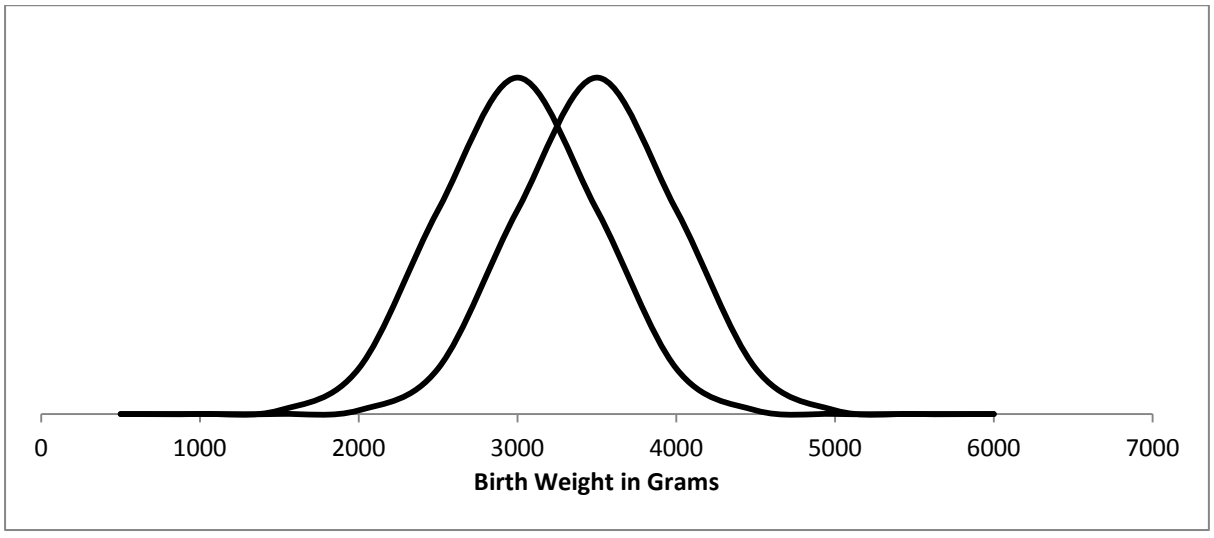
We can show the effect of socio-economic status on birth weight predicted by this model by showing the distributional shifts. Starting with a normal distribution of birth weight:



If actions reduce the risk of high birth weight, the effect of increasing socio-economic status can be shown as a tightening of the distribution, or a decrease in the variance:



Under the assumption of actions increasing high birth weight risk, my model predicts that socio-economic status increases birth weight, hence this could be shown as a shift in the distribution of birth weight:



This leaves us with two competing hypotheses to be tested empirically:

> Hypothesis 1: Mothers with a high socio-economic status are better able to avoid negative health outcomes and therefore will have a lower incidence of high birth weight.

Hypothesis 2: Mothers with a high socio-economic status have higher birth weight over all levels as they are better able to avoid low birth weight but this translates into a higher incidence of high birth weight

Data

I use two datasets to test the above hypotheses as there are differences in the sets of available socio-economic status measures and control variables between these datasets. This also allows me to check consistency across datasets used.

NDF

As discussed above, NDF is a dataset that covers all live births in the United States dating back to 1968 and available up to 2009. In 2003, substantial revisions to the way data was collected were undertaken so I am only using data from 2003 onwards.

The large population of the US and the high coverage of the data allow for an enormous sample; roughly 4 million births a year over 7 years of data provide me with about 28 million potential data points. In addition to information on birth weight, maternal pre-pregnancy weight and weight gain during pregnancy, the dataset also contains important demographic and socio-economic status variables such as ethnicity, marital status, and education. Unfortunately, no information is available on income/wealth, height, or region of residence for the majority of the dataset.

PRAMS

PRAMS is a survey run by CDC annually from 1988 to 2008 over 44 states which each sample between 1,300 and 3,400 women. The dataset does not represent a balanced panel as there are substantial gaps in state/year groups; most years cover fewer than 30 states. The sample draws from the NDF so only covers live births. The major benefit of this dataset is

that it includes information unavailable in the NDF; most importantly, it includes a measure of household income which allows for a more thorough examination of the effect of socio-economic status on high birth weight risk.

In this analysis, I focus exclusively on singleton births. Multiple births present unique pregnancy and birth issues and also tend to produce much smaller babies, so the likelihood of babies in a multiple birth being of high birth weight is very low.

To measure socio-economic status, I create a dummy variable to indicate whether the woman was married and a set of dummy variables to indicate her education level. The categories for education are elementary only, high school dropout, high school completion, some college, and a college degree holder. In both data sets, I also create variables to indicate ethnicity, infant gender, mother's age, and whether the mother smoked during pregnancy. Ethnicity is defined with five different categories: white, black, Asian/Pacific Islander, American Indian/Alaskan native, and Hispanic. NDF recode the ethnicity variables to aggregate ethnicities from a more specific categorisation into the first four categories, and separately ask for the Hispanic origin of the mother. I use the four categories but when any Hispanic origin was indicated, I classify that to be the ethnicity. I follow the same procedure with the PRAMS data.

With the NDF, I also create variables for parity, pre-pregnancy weight, and weight gain during pregnancy. Parity is expressed with a set of dummy variables indicating if this was the first birth, second birth, etc. up to a parity of eight; higher parities are included in the highest category. A set of dummy variables is used to allow for a nonlinear effect (for instance, the effect of increasing parity from one to two may be different than the effect of going from three to four).

With the PRAMS data, I can create a set of dummy variables indicating the state of residence. With these, I also create a set of dummy variables indicating the census region of residence (South, West, Northeast, and Midwest), and the census sub-regions (Mountain, Pacific, Southwest Central, Southeast Central, South Atlantic, Northwest Central, Northeast Central, Middle Atlantic, New England). Parity cannot be identified in the PRAMS data but first births can so I create a dummy variable indicating if this birth was the first for the mother. Mother's age is reported in the following groups: 17 or younger, 18-19, 20-24, 25-29, 30-34, 35-39, and 40 or older. The PRAMS dataset has a measure of mother's height and weight so a BMI measure can be calculated and women classified into categories as underweight (BMI<20), normal weight (BMI 20-25), overweight (BMI 25-30), obese (BMI 30-35) and morbidly obese (BMI>35). The PRAMS data set collects household income information from respondents for the year preceding the birth. Unfortunately, the specific question asked differs across states, so aggregating the information into common variables proved difficult. To get the closest fit for the largest possible number of states, I create five categories of annual income: <$10,000, $10,000-$20,000, $20,000-$30,000, $30,000-$40,000, >$40,000. Some states had ranges that did not match these categories. In those cases, I rounded to the nearest category. Some states refused permission to income data and individuals within states were given the option to not answer the question.

For all variables where there is missing data, I create a dummy variable indicating that the information is missing.

Methods

Initial Analysis

I first examine the descriptive relationship between socio-economic status factors and birth weight. Using both data sets separately, I compare the percentages of married women among those who had a low birth weight baby (<2,500g), a high birth weight baby (>4,500g), and a normal birth weight baby. I repeat this for each category of education, and, for the PRAMS data, income categories as well. I also report the percentage of low and high birth weight for women in each education, income, and marital status category. As I wish for the counterfactual to represent a healthy birth weight, from this point forward, all low birth weight babies are excluded from analysis. Excluding observations from analysis based on the outcome variable is generally not an acceptable method as it causes potential bias through sample selection. However, for my purposes, as there is little reason to be concerned that this bias would meaningfully affect the results, it is the best option relative to alternatives. Inclusion of low birth weight babies in the counterfactual using probit analysis would not be appropriate as the counterfactual would no longer necessarily represent a healthy baby, rendering my results useless at interpreting health outcomes. Using a continuous measure of birth weight instead of categories is highly likely to cause bias in my estimates due to the hypothesized non-monotonic relationship between socio-economic status and high birth weight; even non-linear alternatives such as taking a log of the dependent variable would not be able to capture this type of relationship. Methods such as ordered probit would be able to retain all observations and represent a non-monotonic relationship. However, these methods are substantially more complicated to implement and do not offer enough improvement to justify the extensive additional processing power required for such large data sets.

Replicating Cesur and Kelly (2010)

My next step is replicating Cesur and Kelly's (2010) regressions that led them to conclude there was no correlation between socio-economic status and high birth weight. Cesur and Kelly (2010) use two data sets; the National Longitudinal Survey of Youth (NLSY) and the Early Childhood Longitudinal Study (ECLS) Kindergarten Cohort for their regression analysis, and run separate regressions with each dataset. The data sets contain similar variables but information may be more precise in one data set. For example, NLSY contains specific information on income, education, and marital status, whereas ECLS only includes a composite measure of socio-economic status. For my analysis, I attempt to replicate the more precise measures.

I use the same dependent variable as Cesur and Kelly (2010) which is a binary variable indicating whether the baby had a birth weight greater than 4,500g. I have been able to match the socio-economic status explanatory variables used in their analysis with some minor discrepancies. For marital status, their categories include single, married, and divorced. The PRAMS does identify divorcees but only for a fraction of the dataset, so is therefore unreliable and I only include married and single as categories for marital status. For income, they have actual values whereas I only have ranges. For education, our measures are identical.

Cesur and Kelly (2010) include the same set of controls in these regressions as they do in their main analysis. As their primary focus is on the effect of high birth weight on cognitive outcomes, some of these controls seem inappropriate in a regression addressing the effect of socio-economic status on high birth weight risk and I exclude these from analysis. These variables are: the child's current age, whether the child was breast fed, the child's current height and weight, current number of children to the mother, children books at home, and the highest qualification the child expects to obtain. Clearly, as these factors do not manifest until

after birth, there is no plausible way that they could influence birth weight and therefore are not relevant for inclusion in my regression analysis.

The only other discrepancy with their variable set is that in the PRAMS data, mother's age is in ranges and only an indication of whether the baby is firstborn is available; Cesur and Kelly (2010) have mother's age in years and a variable indicating birth order. Finally, a variable for mother's BMI is included in both studies but I add its square to allow for a non-linear effect.

Cesur and Kelly (2010) report coefficients from an Ordinary Least Squares (OLS) regression analysis in their paper. However, they also repeat their analysis with a probit, results of which are unreported but I received through correspondence. I report marginal effects from a probit for consistency and as it is the more appropriate measure when using binary dependent variables.

The general form of the models I estimate using NDF and PRAMS (respectively) is as follows:

(1) $High\ Birth\ Weight_i =$

$\beta_0 + \beta_1 marital\ status_i + \beta_2 education_i + \beta_3 infant\ gender_i + \beta_4 mother's\ weight_i +$
$\beta_5 parity_i + \beta_6 ethnicity_i + \beta_7 mother's\ age_i + \beta_8 mother's\ age\ squared_i + \beta_9 year_i +$
e_i

(2) $High\ Birth\ Weight_i = \beta_0 + \beta_1 marital\ status_i + \beta_2 education_i + \beta_3 income_i +$
$\beta_4 infant\ gender_i + \beta_5 mother's\ bmi_i + \beta_6 mother's\ bmi\ squared_i + \beta_7 first\ birth_i +$
$\beta_8 ethnicity_i + \beta_9 mother's\ age_i + \beta_{10} mother's\ age\ squared_i +$
$\beta_{11} region\ of\ residence_i + \beta_{12} year_i + e$

The unit of analysis is a birth (denoted 'i') and unless otherwise specified, all explanatory variables describe the mother. '$High\ Birth\ Weight_i$' is a dummy variable with the value '1' if the birth weight is greater than 4500g ond '0' otherwise.

Further Analysis

After replicating Cesur and Kelly's (2010) regression, I subject the general form for the PRAMS dataset to a number of robustness checks such as: altering the regions of residence used to census sub-regions and states, changing the dependent variable to indicate 'very high birth weight' (>5,000g), 'somewhat high birth weight' (>4,000g), and 'large for gestational age' (birth weight in the 90th percentile or higher for the infant's gestation length),restricting the sample to only prime-age mothers between 20 and 35 years of age, using dummy variables for mother's BMI to indicate if she was underweight (BMI<20), overweight (BMI 25-30), obese (BMI 30-35), or morbidly obese(BMI>35) , and controlling for the number of people dependent on the household income.

With the NDF dataset I also perform the robustness checks of changing the dependent variable to 'very high birth weight' and 'somewhat high birth weight', and restricting the sample to only prime-age mothers.

Results

Preliminary Analysis

PRAMS

In general, women who have high birth weight babies are concentrated in higher education, higher income, and married categories (compared to those with infants of normal birth weight), whereas women who have low birth weight babies are concentrated in lower education and lower income categories (Table 3.1).

Aside from the lowest category of education, there appears to be a clear trend of a greater risk of high birth weight with increasing education (Table 3.2). Similarly, the incidence of high birth weight increases with household income with a slight drop off at the highest category (Table 3.3). Married women have a markedly higher incidence of high birth weight (Table 3.4). This is largely consistent with hypothesis 2, that high birth weight risk increases with socio-economic status.

NDF

Women who had high birth weight babies were more likely to have a high level of education and to be married than women who had normal or low birth weight babies (Table 3.5). They were also less likely to have just a high school education or be a high school drop-out, but interestingly, were more likely to have no high school education (Table 3.5). Aside from the lowest level of education this seems to support hypothesis 2; that high birth weight risk increases with socio-economic status.

Replicating Cesur and Kelly (2010)

PRAMS

My results (Table 3.6) seem to provide some support for Cesur and Kelly's conclusion; education variables are not significant, so we have no evidence that education affects high birth weight risk once other factors have been controlled for. However, my results do provide some evidence that increasing income increases high birth weight risk at least at relatively low levels of income. This is in contrast to Cesur and Kelly's finding of an insignificant relationship. My results indicate that increasing income from $10,000-$20,000 to $20,000-$30,000 increases the risk of high birth weight by 0.25 percentage points or 17% from the baseline risk. Being married was not considered as a socio-economic status variable in Cesur and Kelly's paper but here it is statistically insignificant.

The marginal effect of BMI is as expected: a higher body mass poses a higher risk of high birth weight, with a diminishing effect implied by the negative marginal effect of BMI squared. The coefficient implies that with a one point increase in BMI, the risk of high birth weight increases by roughly 0.1 percentage points or 7% from the baseline risk across conceivable BMI ranges. Male infants also have a higher risk of high birth weight as expected. The coefficient implies that a male infant has a 0.9 percentage point or 60% higher risk of high birth weight than a female infant. As expected, first births are significantly less likely to have high birth weight. Ethnicity is a highly significant determinant: the marginal effects imply that Native American babies are largest, followed by White, Hispanic, Black, and Asian babies. Age also exhibits the expected effect where older mothers experience a higher risk of high birth weight.

NDF

The NDF marginal effects can be much more precisely estimated due to the much larger sample size. With these results (Table 3.7) it appears that being married does increase high birth weight risk by roughly 0.1 percentage points or 7% from the baseline risk. Education variables with the exception of 'some college' exhibit significance but the relationship is inconsistent. High birth weight risk decreases from only elementary education to high school drop-out, then increases from high school drop-out to high school completion, and decreases for a college degree.

Further Analysis

I conduct a series of robustness checks on the main regression for the PRAMS data set (Table 3.8). In general, it seems that changes to the functional form have only minor effects on the results. For most of the checks, marriage and education effects are still statistically insignificant and income has an initially positive effect on high birth weight risk. Two outlier results are worth mentioning: When the dependent variable is either somewhat high birth weight (birth weight >4,000g) or large for gestational age, the marginal effect of marriage is positive and significant, and for both regressions, the highest level of education is positive and significant. However, when looking at very high birth weight (>5,000g), it appears that education and marital status have a negative effect on high birth weight risk and income is no longer significant.

In robustness checks on the NDF data set (Table 10), the effect of education and marital status on the risk of very high birth weight is clearly negative, whereas the effect on the risk of somewhat high birth weight is clearly positive.

The above results are generally more supportive of hypothesis 1, that socio-economic status decreases the risk of high birth weight, than hypothesis 2, that socio-economic status

increases the risk of high birth weight. If hypothesis 2 were correct, we would expect to see a positive relationship between socio-economic status and high birth weight at any threshold. In my analysis, the relationship is clearly negative at a threshold of 5,000g. The positive relationship between socio-economic status and high birth weight at the lower threshold of 4,000g is not necessarily inconsistent with hypothesis 1; if health complications associated with high birth weight do not demonstrate themselves at low levels, then 4,000g may appropriately be classified as normal birth weight – an outcome mothers with high socio-economic status should be achieving. Another possibility is that a combination of my two hypotheses is occurring: socio-economic status is shifting the birth weight distribution to the right but decreasing its variance as well, leading to higher birth weights in general but not at the extreme end where health risks are most present.

Discussion

This chapter shows through a theoretical model that socio-economic status should have an effect on high birth weight risk and provides evidence of a relationship with empirical results. My findings partly contradict Cesur and Kelly (2010) which found no relationship between high birth weight and socio-economic status. This does not necessarily refute their conclusions as the magnitudes of the coefficients in my results are similar to theirs, the major difference is in the significance tests. The difference in significance tests results can largely be attributed to the fact that I have a much larger sample size which will always lead to more precision in the estimates and higher significance. Even though preliminary analyses show that high socio-economic status women tend to have a higher incidence of high birth weight, when controlling for inherent characteristics of the mother and the child, the results refute hypothesis 2, that socio-economic status is unambiguously positively related to high birth

weight risk. Instead, my results tend to lend support to hypothesis 1, that socio-economic status improves health outcomes by reducing high birth weight risk. However, it is still possible that a combination of the two hypotheses is occurring, where socio-economic status reduces the probability of the most extreme outcomes while increasing birth weight in general.

4. Obesity and High Birth Weight

Introduction and Background Literature

As discussed in the introduction, maternal obesity has frequently been identified as a risk factor for high birth weight; however, to my knowledge, no study has specifically addressed the causality of this relationship.

The Body Mass Index (BMI) is the most commonly used measure of obesity. BMI is measured as weight (in kilograms) divided by height (in metres) squared. BMI values below 20 are classified as underweight, BMI of 20-25 as normal weight, BMI of 25-30 as overweight, BMI of 30-35 as obese, and BMI above 35 as morbidly obese.[4]

Obesity has become a significant problem in the developed world. The World Health Organisation estimates that one in ten adults globally are obese and that obesity rates have more than doubled since 1980 [39]. In New Zealand, the Ministry of Health estimates that 26.5% of adults are obese, rising from 17.0% in 1997 [40, 41]. In the United States, one in three adults are obese [42]. The rise in obesity has important implications for childbearing. Obesity reduces the ability to conceive [43, 44], increases the likelihood of a pregnant woman suffering from gestational diabetes [45] and, of particular interest to this paper, is associated with a greater likelihood of delivering a high birth weight baby.

Considerable evidence of a positive relationship between obesity and high birth weight risk is presented in the medical literature but there is a dearth of scrutiny over the causal effect. Shepard et al. (1996) conclude that maternal BMI has an independent effect on foetal growth [46]. Srofenyoh and Seffah (2006) find that women who gave birth to high birth weight

[4] For a more detailed discussion of BMI see Appendix A.

babies had significantly higher BMI values than those who gave birth to normal weight babies[47]. Berard et al. (1998) found women who gave birth to babies over 5,000g were on average 9.2kg heavier than women who gave birth to babies between 4,500 and 4,999g [14].

It seems highly plausible that difficult to observe factors such as genetic disposition to high adiposity may correlate with both obesity and high birth weight. There is substantial evidence of a strong genetic component to obesity risk [48]. In light of the high potential for endogeneity between maternal obesity and high birth weight risk, it is necessary to utilise regression techniques such as instrumental variables which can isolate the causal component of the relationship.

To explore the relationship between body mass and high birth weight risk, I use New Zealand birth data from the Ministry of Health for the period between 2007 and 2011. Probit regression analysis shows a strong positive relationship between maternal BMI category and high birth weight risk controlling for demographic and socio-economic status factors of the mother and pregnancy characteristics. The marginal effect estimates suggest that a morbidly obese woman (BMI>35) has a 2.36 percentage points higher risk of delivering a high birth weight baby compared to the baseline risk. However, this has not adequately controlled for the endogeneity of obesity. To account for endogeneity, I conduct an instrumental variable analysis using three different sets of instruments: the rurality of the mother's residence, the concentration of fast food restaurants in the mother's area of residence, and the mother's month of birth.

Biprobit results largely negate the effect of obesity on high birth weight risk except for morbidly obese women. Even for morbidly obese women, the effect is quantitatively small. In particular, my estimates imply that a morbidly obese woman has an increased risk of high birth weight of only 0.26 percentage points compared to the baseline. Instrument validity

tests suggest that the rurality variables are not sufficiently exogenous and that the mother's month of birth lacks sufficient explanatory power; however, fast-food restaurant density passes both instrumental variable tests. Moreover, the estimated effect of obesity on high birth weight is highly robust to changes in the included set of instruments. My results provide compelling evidence that ignoring the endogeneity of obesity leads to much higher estimates of its causal effect on high birth weight risk than is justified. They also suggest that only morbid obesity may be problematic for increasing high birth weight risk.

Model

High birth weight has been linked to a number of factors. A prominent finding in the medical literature is that maternal obesity increases the risk of delivering a high birth weight baby. However, little attention has been paid to causality in this relationship. If there is an independent factor which influences both the likelihood of being obese and the likelihood of giving birth to a macrosomic baby then a regression analysis which does not include the independent factor will find a strong correlation which does not necessarily reflect any causal relationship. It seems highly plausible that difficult to observe factors such as genetic disposition to high adiposity may correlate with both obesity and high birth weight. Therefore, it is necessary to utilise regression techniques such as instrumental variables which can help tease out the causal factor from the relationship.

To use an instrumental variable technique, I need variables which are highly correlated with obesity but have no independent effect on high birth weight risk. I rely on three different sets of instruments in my analysis: rurality of the mother's residence, fast-food restaurant density, and the mother's birth month. Rurality is strongly correlated with obesity risk [48], [42] and, looking at the raw data, seems to be uncorrelated with high birth weight risk, indicating it

should be a useful instrument. However, there could plausibly be factors correlated with both rurality and high birth weight risk which I have not controlled for in my regression analysis. For example, sunshine exposure and consequently vitamin D absorption which can increase birth weight [49] could be higher for women living rurally. Validity tests are required to ensure the legitimacy of rurality as an instrument.

The density of different categories of dining establishments with a particular focus on fast food restaurants within the Territorial Local Authority (TLA) area that the woman resides in comprises my next set of instruments. A significant relationship between fast food restaurant density and obesity has been a prominent finding by health researchers in recent years. Rosenheck's (2008) systematic review of 16 studies from the US and abroad concludes that there exists a significant relationship between fast food restaurant density and obesity [50] . It is generally agreed that fast food proximity lowers the notional cost of eating high caloric food and can therefore lead to higher obesity. Density of fast food outlets should not have any direct effect on high birth weight risk. However, like with rurality, there are plausible factors which could correlate with both food venue type and concentration and high birth weight risk. For instance, if unhealthy food venue options tend to concentrate in areas where people tend to be less health conscious for reasons that transcend deprivation level, ethnicity, age, rurality, or time-invariant regional characteristics (i.e., factors that I control for in my model), then food venue type and concentration may have an avenue of correlation with high birth weight risk outside of the effect on obesity. It is also possible that food venue type and concentration may be correlated with high birth weight risk through the effect of weight gain during pregnancy. Validity tests are required to check the soundness of this instrument.

The last instrument I use is the mother's birth month. Season of birth is associated with a number of health outcomes, through various suggested mechanisms. Tustin et al. (2004) find that increased sun exposure during pregnancy increases birth weight which could be caused

by increased vitamin D absorption [51]. Phillips and Young (2000) report that early exposure to cold conditions is associated with higher weight during adulthood in England [52]. Lokshin and Radyakin (2009) conclude that higher exposure to diseases during different seasons (they specifically look at the higher incidence of disease during the monsoon season in India) leads to lower anthropometric measures during childhood [53]. It is therefore plausible that the mother's birth season can affect her obesity risk later in life but it seems unlikely that there would be any non-trivial influence of mother's birth month directly on her infant's high birth weight risk. However, the link between birth month and obesity may prove to be weak.

Data

To test the relationship between body mass and high birth weight risk, I use the Maternity National Collection as described in the introduction for the period between 2007 and 2011. Information on BMI was only included in this dataset from 2007 so I am unable to use data from years prior to 2007 in this chapter. As explained in the introduction, this dataset contains demographic and health status information about the baby and mother. For this chapter, the relevant information I can identify includes the age, ethnicity, area of residence, birth date, parity, smoking status, and BMI of the mother and the birth date and birth weight of the baby as well as the length of gestation. As explained earlier, it is necessary to exclude multiple births and still birth observations. I also exclude observations with missing BMI records. Finally, I exclude non-residents as some of my variables rely on having lived in New Zealand for some time; preferably, I could select only women born in New Zealand but I do not have the available information to do this, therefore eliminating non-residents is the closest

approximation. After eliminating non-residents, multiple births, stillbirths and observations with missing BMI information, I am left with a sample of around 186,000 births.

I use the internationally recognised categories of overweight, obese, and morbidly obese for BMI values greater than 25, 30, and 35, respectively.

A significant limitation of MAT is that the measure of BMI is taken from when the mother first registers with a lead maternity carer (LMC).[5] This is concerning because the later a woman registers, the higher her BMI would be expected to be due to normal pregnancy weight gain.[6] The Institute of Medicine's most recent guidelines recommend a weight gain for normal weight women of 0.8-1lbs (0.36-0.45kg) per week in the second and third trimester after an assumed gain of 0.5-2kg in the first trimester; smaller gains are recommended for overweight and obese women [54]. Therefore, the measure is a less accurate approximation of pre-pregnancy body mass for women with later registrations. This is especially worrisome if factors that could contribute to high birth weight risk may also correlate with later registrations with a lead maternity carer. To account for this problem, I firstly eliminate all observations where the mother registered in the third trimester of pregnancy (4.7% of the sample) as these women are likely to have the least accurate approximation of pre-pregnancy BMI due to much larger cumulative weight gain in the third trimester compared to the first two. I also eliminate observations where the date of registration could not be identified (2.2% of the sample).

Focusing on women with LMC registrations in the first two trimesters, I also take some comfort in the discrete nature of the obesity measure I am using as the initially low weight gain is unlikely to push many women into higher BMI categories than they would have

[5] For more information on LMCs, see Appendix B.
[6] Similar issues affect measurement of the mother's smoking status but this presents a much less concerning problem as smoking is not a variable of major importance in this chapter.

belonged to earlier in pregnancy. For a woman of average height (1.65m), there is a 13.6kg difference between BMI categories.[7] As only 0.5-2kg is expected to be gained in the first trimester, this should have a minimal effect of pushing women into higher BMI categories and 55% of the overall sample registered in the first trimester. Weight gain in the second trimester is more rapid (about 0.36-0.45kg a week – 5.81-7.25kg total) so women who register during the second trimester (38% of the overall sample) are more likely to have an inflated measure of pre-pregnancy BMI.

To further address the BMI measurement issue, I also create a variable to control for the number of days into pregnancy when each woman registered, calculated as the number of days from the estimated date of the last menstrual period to the date of registration. I include this variable in all of my analyses.

To measure fast food density in the mother's area of residence, I use data supplied by Statistics New Zealand on the number of venues under separate classifications determined by the Australia and New Zealand Standard Industrial Classification (ANZSIC). ANZSIC was established by Statistics New Zealand and the Australian Bureau of Statistics to improve data comparability between New Zealand and Australia and other countries. It was last revised in 2006. The venue classifications I am using are: 'cafes and restaurants', 'takeaway food services', 'catering services', 'pubs, taverns, and bars', and 'clubs (hospitality)'. The number of venues of each type is aggregated by year and territorial local authority (TLA)[8].

I also create an index of the number of 'big chain' fast food restaurants in each TLA. I do this by using information available on the websites of McDonalds, KFC, Subway, Pizza Hut, Hell's Pizza, Burger Fuel, Burger Wisconsin, and Wendy's on the number of venues within

[7] BMI categories are separated by 5 units of kg/m^2. $5*1.65^2=13.6125$
[8] For an extended discussion on TLAs see Appendix C.

each TLA. As I am relying on their current websites, I am unfortunately unable to identify any time variation in the number of these restaurants open over the sample period.

TLA regions represent the areas governed by local councils which generally cover just one city or town including the rural surrounds. I use TLAs to create my variables for the availability of food venues. This level of aggregation appears to be a reasonable trade-off - although in larger cities, additional food venues on the other side of town may have no impact on food choices and therefore obesity, smaller levels of aggregation would miss the fact that many food choices are made at places of work, study, and leisure which are highly likely to be contained within the same city or town but could be considerable distance from the home and could therefore be missed by smaller aggregation levels. There are some minor differences in the TLA definition between the big chain restaurants and the ANZSIC classification variables. In November 2010, the Auckland, Manukau, Waitakere, North Shore, Papakura, Rodney, and Franklin councils were merged into one council for all of the Auckland area. For the big chain restaurants, I use the separate councils before the merger to define areas but the ANZSIC food venues data is only available for the merged area. I also create a variable for the annual population of these areas and another variable measuring the latest available median income of the region. There are 67 or 73 TLAs depending on the definition used with a median population of about 30,000.

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Methods

Using birth weight information available in the birth records, I create dummy variables to indicate whether the baby is of high birth weight (>4,500g) or low birth weight (<2,500g). With regards to the mother's demographic characteristics, I create dummy variables indicating the following ethnic categories: European, Maori, Pacific Islander, Asian, Middle

Eastern/ Latin American/ African (MELAA), and other. I also create continuous variables for the mother's age as well as age squared to account for non-linear effects of age on obesity and high birth weight risk. Finally, I create dummy variables for the number of previous births. Having parity as a dummy variable rather than a continuous measure allows for non-linear effects of increasing parity on both obesity and high birth weight risk.

I create dummy variables for each of the 21 District Health Boards (DHBs) [9] regions that the mother resides in to control for possible regional variation. DHBs are important for the governance of health services delivery in New Zealand

For the instruments, I create dummy variables to indicate whether the woman's residence is considered urban, semi-rural, rural, or remote rural defined at the Census Area Unit (CAU)[10] level, and dummy variables to indicate the mother's month of birth.

I also include dummy variables to indicate where there was missing data for the mother's age, smoking status, parity, rurality, and deprivation decile. This allows me to include these observations in some of my regression analyses without losing precision of the estimates.

I create dummy variables for the infant's gender and season of birth which I include in the second stage of the biprobit only as they should not have any impact on obesity of the mother but should have an impact on high birth weight risk.

As birth month does not represent the same birth season for women born in the northern hemisphere, and may also represent different seasonal conditions for women born in different countries within the southern hemisphere, it would be ideal to drop all women who were not born in New Zealand from the sample. However, as explained above, I cannot identify the mothers' country of birth. Dropping instead all women who are not New Zealand residents

[9] For an extended discussion on DHBs see Appendix C.
[10] For an extended discussion on CAUs see Appendix C.

should capture many of the women who were not born in New Zealand and reduce the problem.

Initially, I run probit regressions where the observations are a single birth, denoted 'i', with the following specification:

$$(1)\ High\ birth\ weight\ infant_i = \beta_0 + \beta_1\, obesity\ category_i + \boldsymbol{\beta controls_i} + e_i$$

Where 'obesity category' is alternatively specified five different ways, i.e., I repeat the probit regression five times with a different "obesity category" definition: overweight (25<BMI<30), obese (30<BMI<35), morbidly obese (BMI>35), all obese (BMI>30), or all overweight (BMI>25). I alternate obesity categories instead of including mutually exclusive categories simultaneously as bivariate probit – discussed shortly - only allows for one endogenous regressor to be intrumented for in a single regression and I want my probit results to be directly comparable. When using "overweight" or "obese", I exclude observations with a higher BMI. In other words, the counterfactual is always defined as all mothers with a lower BMI than the specified category. The controls consist of: ethnicity dummies, mother's age, mother's age squared, parity dummies, dummies to indicate the deprivation decile of the area of residence, dummies to indicate the DHB area of residence, baby's birth season dummies, a dummy variable to indicate whether the mother reported smoking at the time of registration with her LMC, infant gender, median income of the TLA of residence, year dummies, a dummy variable to indicate if the woman registered in the second trimester of pregnancy (as opposed to the first) and a variable indicating the number of days into the pregnancy when registration took place.

To account for the endogeneity of pre-pregnancy BMI, I use bivariate probit with three sets of instruments: a set of dummies indicating the mother's birth month, a set of variables indicating fast food and other relevant venues' concentration in the mother's TLA, and a set

of dummy variables indicating the rurality of the mother's CAU. The specifications are as follows:

$$(2)\ High\ birth\ weight\ infant_i = \beta_0 + \beta_1\ \widehat{obesity\ category}_\iota + \boldsymbol{\beta controls_{1,i}} + u_i$$

$$Obesity\ category_i = \gamma_0 + \boldsymbol{\gamma_1 mother's\ birth\ month_i} + \boldsymbol{\gamma_2 fast\ food\ concentration_i} + \boldsymbol{\gamma_3 rurality_i} + \boldsymbol{\gamma controls_{2,i}} + v_i$$

Where $\boldsymbol{controls_1}$ represents the same vector of controls as included in the probit regressions and $\boldsymbol{controls_2}$ excludes from the full list infant's birth season and gender variables.

However, as there is a plausible argument for the rurality instruments being correlated with the error term, I repeat the regression with only the first two sets of instruments:

$$(3)\ High\ birth\ weight\ infant_i = \beta_0 + \beta_1\ \widehat{obesity\ category}_\iota + \boldsymbol{\beta controls_{1,i}} + u_i$$

$$Obesity\ category_i = \gamma_0 + \boldsymbol{\gamma_1 mother's\ birth\ month_i} + \boldsymbol{\gamma_2 fast\ food\ concentration_i} + \boldsymbol{\gamma controls_{2,i}} + v_i$$

Finally, to allow for the possibility of fast food concentration also being correlated with the error term, I repeat the regressions with only mother's birth month, which is the least likely to have any endogeneity concerns, as a set of instruments:

$$(4)\ High\ birth\ weight\ infant_i = \beta_0 + \beta_1\ \widehat{obesity\ category}_\iota + \boldsymbol{\beta controls_{1,i}} + u_i$$

$$Obesity\ category_i = \gamma_0 + \boldsymbol{\gamma_1 mother's\ birth\ month_i} + \boldsymbol{\gamma controls_{2,i}} + v_i$$

I repeat all model specification for each of the five categories of obesity, producing fifteen sets of results.

I then perform validity tests on all fifteen of these regressions by estimating with two stage least squares and calculating the F-statistic of the first stage regression and the p-value of the Hansen's J-statistic. Higher values of the F-statistic on the first stage regression indicate that a larger proportion of the endogenous regressor's variance has been explained by the instruments. The Hansen's J-statistic is used to perform an "over-identification" test which indicates whether the excluded instruments are correlated with the error term. Low p-values indicate rejection of the null hypothesis that the instruments are uncorrelated with the error term; higher p-values are therefore desirable in this test.

Results

Simple probit estimation (first panel of Table 4.1) shows that obesity has a strongly significant effect on high birth weight risk at all thresholds. Increasing BMI into overweight, obese, and morbidly obese categories increases the risk of a high birth weight birth by 1, 1.19, and 2.36 percentage points compared to lower weight groups, respectively. Combining all overweight categories in one implies an increased risk of 1.47 percentage points and 1.73 when combing all obese categories.

Holding other factors constant, my results consistently show that Pacific women have the highest propensity to have high birth weight babies followed by European/Pakeha, Maori, Middle Eastern/Latin American/African, and Asian women. Male infants have a higher risk of high birth weight as expected. In particular, a male infant has a 1.5 percentage points higher risk of high birth weight than a female infant. Also as expected, increasing maternal age and parity increases the likelihood of high birth weight but at a diminishing rate. Women who reported smoking at their first LMC visit are roughly 1.4 percentage points less likely to give birth to a high birth weight baby.

Using all three sets of instruments in bivariate probit regressions (third panel of Table 4.2) provides the highest explanatory power in the first stage. However, the exogeneity of the instruments is questionable as the p-value when conducting the Hansen's J test drops below 0.05. When using only mother's birth month as an instrument for obesity (fourth panel of Table 4.2), exogeneity is strongly supported but the explanatory power falls dramatically. Using just mother's birth month and fast food restaurant instruments (second column of Table 4.2), the explanatory power is compromised compared to using all three but not to the same extent as when only one instrument set is used and the exogeneity tests are passed confidently. Therefore, I focus on the results with two instruments used as my main findings. These results indicate that being overweight has no significant effect on the likelihood of having a high birth weight baby, nor does being obese. There only appears to be a significant effect on high birth weight probability when the mother is morbidly obese. Morbidly obese women have a 0.26 percentage points higher chance of having a high birth weight baby (compared to all other groups holding other factors constant. Despite the changes in validity test results of the different sets of instruments, this finding is very robust to changes in the instruments used. This effect is a near magnitude smaller than what was found when using probit which strongly suggests that ignoring the endogeneity of obesity leads to highly biased estimates of its effect on high birth weight risk. The strength of this conclusion lies more in the consistency of the finding across various sets of instruments than the, admittedly lacking, strength of identification. Although the instruments only pass both instrument validity tests in two of the regressions, I contend that the consistency of the findings at the very least provides suggestive evidence to support my conclusions.

An interesting finding from the first stage results (Table 4.3) is that the density of fast food restaurants in a TLA does not have the expected effect on obesity measures. The majority of the fast food chains consistently yield negative coefficients in the first stage and particular

chains such as Hell's Pizza, Burger Wisconsin, and McDonalds frequently show a significantly negative relationship with the propensity to be overweight, obese, and morbidly obese. KFC and Burger Fuel are the only chains to have a generally consistent positive relationship with obesity risk. It is not clear what is driving these findings as both median income of the TLA and the deprivation level of the meshblock have been controlled for suggesting it is unlikely to be socio-economic status, nor could it be the effect of living in urban areas as rurality variables are also included. The overall number of fast food establishments per person in a TLA is generally insignificant so it does not appear to be driven by substitution away from less healthy options such as fish and chips either. More research into the effect of fast food on obesity in New Zealand is warranted.

Conclusion

My results suggest that although being overweight, obese, or morbidly obese is highly correlated with high birth weight risk, there is only a significant causal effect once a woman reaches the level of morbid obesity. Controlling for endogeneity also substantially reduces the size of the estimated effect. Therefore, studies on the effect of obesity on high birth weight risk which ignore the endogeneity of obesity run the risk of greatly over-estimating its role. This has important implications for public health decisions on advice given to pregnant women and for funding decisions which may depend on body mass. If the danger of obesity during pregnancy with regard to high birth weight risk has been previously overstated then advising women to try to lose weight before conception may be of less importance than previously believed.

5. Vitamin Supplements

Introduction

A key question that I am interested in addressing in this chapter is whether prenatal vitamin supplements have any causal effect on the incidence of macrosomia. A number of studies have investigated the effect of different vitamin supplements on birth weight and found a positive association[11]. However, no study to my knowledge has looked at whether the foetal growth that is aided by vitamin supplements may have the detrimental effect of increasing macrosomia incidence. In particular, iron supplementation is recommended for foetal growth and a number of studies have shown a positive correlation between iron supplementation and birth weight [8, 9, 10, 55]. However, some studies have also found no association between iron supplementation and birth weight [11]. If iron supplements have a positive relationship with birth weight then they may be beneficial for infants at risk of low birth weight but detrimental for infants at risk of high birth weight. This would present a trade-off for women in deciding whether to take them or not and would suggest that universal recommendations for iron consumption would be inappropriate as some women could be negatively affected by taking iron supplements during pregnancy.

Another point that is largely ignored in previous studies is the extent to which an association between vitamin consumption and birth weight is causal.

Supplements and Foetal Development

Iron is required for haemoglobin (Hb) synthesis; lowered haemoglobin levels due to iron deficiency result in reduced oxygen carrying capacity and cause fatigue. Iron deficiency can

also adversely affect immunity, growth, and development [55, 56]. The potential negative effects of iron intake during pregnancy are an area of emerging concern but as of yet there has been little research on the topic. Potential side-effects of taking iron supplements include nausea, constipation, and heartburn with incidence increasing with the dosage given [56, 57, 58].

There is a debate about whether supplements in general – and iron supplements in particular - should be prescribed routinely to pregnant women or whether pregnant women should have their iron status assessed individually and supplements should only be given to those in need. The optimal response depends on the general benefits of iron supplements and the possible side-effects in consideration of the financial costs of supplementation. Graves and Barker (2001) argue that taking an individualised approach based on the woman's history and health status is warranted due to the unclear general benefits and potential side-effects of iron supplementation [58]. However, Roodenburg (1995) points out that the individualised approach may be less effective at reaching all women who would benefit from iron supplementation as the difficulties associated with assessing iron status may discourage some women and/or their maternity carers from undertaking it [59].

Literature Review

Ramakrishnan et al. (1999) analyse the literature on micronutrients and pregnancy outcomes and conclude that there is strong evidence primarily from developing countries that calcium, zinc, and magnesium can increase birth weight [11]. They also find some evidence to suggest that iron supplements can increase birth weight but it is weak in part due to the difficulty of conducting randomised controlled trials due to the ethical concerns of denying iron-therapy

when it is a proven treatment for anaemia. A more recent review by Chaffee and King (2012) disputes that supplemental zinc has any effect on foetal growth [60].

Siega-Riz et al. (2006) test the effect of giving iron supplements to low income women in Raleigh, North Carolina from early pregnancy to the beginning of the third trimester. They find that women given iron supplements had mean birth weight significantly higher by 108g compared to the control group [8].

Aranda et al. (2011) examine the effect of iron supplementation on women with and without iron-deficiency before becoming pregnant. They find that iron supplementation has a positive effect on women who had pre-pregnancy iron-deficiency, but no significant effect on women who had adequate pre-pregnancy iron stores. The low number of observations (82) in this study indicates that the latter result need not be interpreted as conclusive of the effect of iron supplementation on birth weight [9].

Watson and McDonald (2010) examine the diet and supplement intake of 504 women at the fourth and seventh months of pregnancy. They find dietary intake of magnesium and vitamin D had a positive effect on birth weight and a combined supplement intake increased birth weight by 129g. Iron supplements in particular increased birth weight by 119g and were the only individual mineral supplement to have a significant effect on birth weight [61].

Overall, the evidence seems to suggest that iron supplements in particular can increase birth weight, which could plausibly indicate that they may increase the risk of high birth weight if the effect does not diminish with the growth of the foetus.

Model/Conceptual Framework

Supplements are prescribed during pregnancy for a number of reasons but a major reason is to encourage the growth and development of the foetus [62]. However, excessive growth of the foetus may lead to high birth weight which is problematic for both the baby and the mother during birth and has been shown to have longer term consequences.

Endogeneity may be present due to the fact that women are not prescribed supplements at random, and factors that researchers cannot control for could influence both the probability of being prescribed supplements and of having a high birth weight baby. Of particular concern with my data is that I am unable to determine the reason for being prescribed supplements. If, for example, the women being prescribed iron supplements are disproportionately suffering from anaemia (which is likely), I cannot identify this reason and it may be that these women would be less likely to have high birth weight in the absence of supplementation. This would hinder any causal inference about the impact of iron supplements on high birth weight risk. To address this, I employ an instrumental variable analysis, where the price of iron supplements and their availability on the Pharmaceutical Management Agency (PHARMAC) schedule (described in more detail shortly) are used as instruments for iron supplement intake. I limit my analysis to only iron supplements as opposed to the full list of supplements due to the extreme difficulty in addressing multiple endogenous binary regressors and iron seemingly having the most potential to affect high birth weight risk as it is both the most commonly prescribed and most often associated with increased birth weight in the literature.

PHARMAC is a government organisation in New Zealand that controls the subsidisation of pharmaceuticals in the country. With the official claim to reduce costs and improve health outcomes[11], PHARMAC regularly updates the schedule to take advantage of different brands

[11] See http://www.pharmac.health.nz/ (Accessed on 1/5/14)

on offer. Pharmaceuticals on the PHARMAC schedule are either partially or fully subsidised and as long as there are substitute brands available on the PHARMAC schedule doctors seldom prescribe pharmaceuticals excluded from the list. .

In the first stage of my model, I essentially estimate an imputed demand for iron:

$$Demand\ for\ iron = f(price\ and\ availability\ of\ individual\ iron\ brands)$$

As this is an imputed demand model, it does not necessarily yield negative relationships for all individual brand prices. In other words, as the brands should be close substitutes for one another, a price increase for one brand will in theory reduce the demand for that brand but increase the demand for other brands. However, the combined effect of price over all brands on the overall demand for iron should be negative.

Data

To test the relationship between supplement intake and high birth weight risk, I use New Zealand birth data (MAT) and prescription data (PHARMS) from the Ministry of Health between 2002 and 2011. The birth dataset (MAT) contains individual-level demographic and health status information about the baby and the mother. In particular, I can identify the mother's age, ethnicity, domicile of residence, birth date, parity, and smoking status, the baby's birth date and birth weight, and the length of gestation. The prescription data contains information on all prescriptions from the PHARMAC schedule classified as either a vitamin or a mineral to pregnant women during the same time frame. As women who gave birth in 2002 may have been pregnant for some of 2001, I may have missing prescription data for these women. I therefore only use observations where the date of birth is in 2003 or later.

A significant limitation of the prescription dataset is that it does not account for the potentially large quantity of supplements purchased over-the-counter rather than prescribed by a doctor. This is an unfortunate downside. However, self-administration of supplements is much more likely to suffer from endogeneity as women who take the initiative to purchase their own supplements without a prescription likely represent a subset of women with a different approach to maintaining their and their infant's health. Therefore, although the information on supplement prescriptions during pregnancy may not be a comprehensive measure of all supplements taken during pregnancy, it is likely a more exogenous measure.

I am also unable to identify if the prescriptions were taken as recommended or if some women did not adhere to their treatment. However, this is not of great concern as actual intake information once again suffers from more endogeneity than prescription data. The use of prescription information is akin to an "intent-to-treat" approach. The intent-to-treat approach serves as a way to deal with non-compliance and attrition in randomised controlled studies [63]. If non-compliers or drop-outs have different tendencies than those who continue and properly comply with a study, for example if the participants who are experiencing less improvement from a treatment are more likely to drop out than those experiencing greater improvement, then the estimated effect of the treatment will be seriously biased. It also serves the purpose of reflecting the true efficacy of a treatment by accounting for the sometimes lacking propensity for compliance with treatment advice outside of a controlled study environment [51]. The intent-to-treat approach has more broad applications as it helps to eliminate sources of endogeneity outside of randomised controlled trials. Newell (1992) gives the example of analysing the hazards of homebirths; if an intent-to-treat analysis is not used then homebirths may appear comparatively safer than hospital births because intended home births that encounter complications often end up in a hospital and as such count as a hospital birth [49]. If an intent-to-treat approach were to be used then the comparison would be

between women who intended to give birth at home and those who wanted to give birth in a hospital, regardless of where the actual birth took place.

I retrieve data on the price and availability on the PHARMAC list of the iron brands prescribed online via the PHARMAC schedule.[12] I was able to determine the out-of-pocket price of the twelve different iron brands that were available for some time over the period of my data.

I create a set of binary variables to indicate for each particular brand whether it was present on the PHARMAC list. I also create a set of variables for the out-of-pocket (i.e., full minus subsidy) prices of the iron brands. I identify prices and availability on the date when the mother enters her third trimester of pregnancy. The out-of-pocket price measure needs to be interpreted with caution though as a zero price can either indicate that the brand is effectively free (fully subsidised) or that it is not available on the PHARMAC list at that point in time, detailed further below.

The brands that were available on the PHARMAC list at some point during my sample period are as follows: Ferrograd, Healtheries Iron with Vitamin C, Ferro-tabs, Ferrosig, Ferodan, and Ferro liquid. Ferrograd and Ferro-tabs also came in varieties that contained folic acid called Ferrograd F and Ferro-F-tabs, respectively. I create separate variables for these varieties. Ferrograd and Ferodan also offered different quantity options (30 or 150 tablets and 250 or 500 tablets, respectively). I also create separate variables for these as they do not always have the same per unit price. There may be brands that were available for purchase over the counter or for prescription that never appear on the PHARMAC list during the time period of my sample. I was unable to procure price data for these brands. I discuss the implications of this below.

[12] Available from http://www.pharmac.health.nz/tools-resources/pharmaceutical-schedule/ (Accessed on *1/5/14*)

Methods

Initially, I estimate a probit model where the observations are a single birth, denoted 'i', with the following specification:

(1) $$High\ Birth\ Weight_i = \alpha + \beta_1 iron\ consumption\ trimester\ 1_i + \beta_2 iron\ consumption\ trimester\ 2_i + \beta_3 iron\ consumption\ trimester\ 3_i + \boldsymbol{\gamma other\ supplements_i} + \boldsymbol{\delta controls_i} + \varepsilon_i$$

Where iron consumption per trimester is calculated based on the date the pharmaceuticals were dispensed relative to the dates of the trimesters. The quantity prescribed and the daily doses are available for most observations which allows me to determine the number of days for which a woman will be taking each supplement. Combining that with information on potency, I am able to estimate the amount of elemental iron ingested in each trimester. For example, if a woman is prescribed 28 tablets and advised to take two per day starting one week before the end of the first trimester, then I assume that half of the total available iron in the prescription will be consumed in the first trimester and half in the second trimester. For the minority of observations where the daily dose is unavailable (16% of my sample), I estimate iron ingestion as the quantity prescribed multiplied by the potency of individual tablets in the trimester in which they were dispensed. As a robustness check, I repeat analyses which rely on the quantity of iron consumed omitting observations which did not have data on the daily dose. The findings are not sensitive to these observations being excluded. I also repeat the analysis with dummy variables indicating whether the mother was prescribed iron supplements during each trimester.

'Other supplements' is a vector of dummy variables indicating whether a woman was prescribed – at any point during her pregnancy - each of calcium, fluoride, iodine,

multivitamins, vitamin A, vitamin B, vitamin C, vitamin D, vitamin E, and zinc, respectively. 'Controls' is a vector of demographic and socio-economic variables potentially influencing high birth weight risk. For example, the model includes a set of dummy variables for the District Health Board (DHB) area that the woman resides in[13]. This is important as not only may there be effects of different areas on high birth weight risk, but also the propensity to prescribe various supplements could well be different in different DHBs. Wennberg and Gittelsohn (1973) brought attention to the issue of glaring discrepancies in medical practice between regions [64]. This 'small area variation' often appears medically unjustified and results in excessive costs and impaired health outcomes in poorly performing areas. In 1988, Wennberg started producing the Dartmouth Atlas of Health Care[14] which tracks such variations and highlights the most egregious examples of unjustified variation.

Mother's ethnicity is controlled for by a set of dummy variables indicating whether the mother is of Maori, Pacific, European, Asian, Middle-Eastern/Latin American/African, or other ethnicity. The mother's age as well as its square are included to account for a non-linear effect of age on high birth weight risk. I create a set of dummy variables to indicate the number of previous births to the mother; a set of dummies rather than a continuous measure allows for parity to have a non-linear effect on birth weight. I also create a set of dummy variables to account for the deprivation scale value of the mother's meshblock area of residence measured by the NZDEP deprivation index.. Rurality of the mother's area of residence (at the Census Area Unit level of aggregation) is controlled for with a set of dummy variables indicating whether the area is urban, semi-rural, rural, or remote rural. The infant's season of birth[15] is also controlled for with dummy variables. Rurality and season of birth are important to control for as they potentially have a strong impact on factors such as physical

[13] See Appendix C for more information on New Zealand geographic areas.
[14] Available at http://www.dartmouthatlas.org/
[15] Defined by the accepted seasons in New Zealand: Summer (December-February), Autumn (March-May), Winter (June-August), and Spring (September-November).

activity and diet during pregnancy which may influence high birth weight risk. There may also be important differences in the amount of sunshine exposure the woman has during pregnancy which through the effect of vitamin D synthesis may affect birth weight [49, 51, 63]. Infant gender is controlled for with a dummy variable.). The trimester in which the woman registered with a lead maternity carer[16], a dummy variable indicating if the woman responded that she was currently smoking at the time of registration, and weight group based on BMI[17] at registration are also included as controls.

To account for the potential endogeneity of iron prescription I also use instrumental variable methods with the price and availability of iron supplements as instruments. Where the consumption of iron supplements is expressed in the form of a dummy variable, I use a bivariate probit[18] . Where I use the quantity of iron ingested (i.e., a continuous endogenous regressor), I attempt to employ instrumental variable probit methods but encounter difficulties with estimation; when using methods which allow for calculation of the standard errors of the marginal effects the regression fails to converge and I am unable to produce estimates so can only rely on estimates of the coefficients without marginal effects.

In an attempt to rectify this, I use special regressor techniques with mother's age as the special regressor. The special regressor technique transforms the binary dependent variable using the distribution function of a continuous variable which appears additively in the model to allow effective calculation of the much simpler two-stage least squares instead of IVprobit[19]. However, the simplicity does not extend to calculations of the marginal effects. Unfortunately, I am also unable to calculate standard errors of marginal effects using this

[16] See Appendix B for more information on lead maternity carers.
[17] BMI categories in my model include: underweight (BMI<20), normal weight (20<BMI<25), overweight (25<BMI<30), obese (30<BMI<35), and morbidly obese (BMI>35).
[18] See Appendix D for more information about bivariate probit regression analysis.
[19] See Appendix D for more information about the special regressor technique.

technique due to the overwhelming computational power required. I can only do so when I draw a 1% random subsample (i.e., 4578 observations) of my data.

The general specification for instrumental variable approaches is as follows:

(2)

$$
\begin{aligned}
&High\ Birth\ Weight_i \\
&\quad = \alpha + \beta_1 iron\ consumption\ trimester\ 1_i \\
&\quad + \beta_2 iron\ consumption\ trimester\ 2_i + \beta_3 \widehat{iron\ consumption}\ trimester\ 3_i \\
&\quad + \boldsymbol{\gamma other\ supplements_i} + \boldsymbol{\delta controls_i} + \varepsilon_i
\end{aligned}
$$

$$
\begin{aligned}
&\widehat{iron\ consumption}\ trimester\ 3_i \\
&\quad = \alpha + \beta_1 iron\ consumption\ trimester\ 1_i \\
&\quad + \beta_2 iron\ consumption\ trimester\ 2_i + \beta_{3-14} Price_{1-12_i} \\
&\quad + \beta_{15-26} Availability_{1-12_i} + \boldsymbol{\gamma other\ supplements_i} + \boldsymbol{\delta controls_i} + \varepsilon_i
\end{aligned}
$$

Where the unit of observation is a single birth denoted 'i'.

As alluded to above, interpretation of the instruments' effects requires care. Price is generally an excellent instrument when the endogenous dependent variable is the consumption of a good. However, in this case, the good in question, elemental iron, is available from many different brands so the demand model has to be imputed from the demand for the individual brands. Therefore, I use the prices of all individual brands as instruments for iron consumption (both in the binary and continuous form). Aggregating the individual prices into one index would be problematic as the brands are not prescribed evenly so a simple average would overstate the importance of the prices of some brands and a weighted average based on quantity prescribed would be biased as quantity prescribed is in itself a function of price. With the inclusion of all prices in one demand equation, it is unclear

what sign to expect for each of the price coefficients. There is no reason to believe that iron is a Giffen good, so the direct effect of price on consumption should be negative. However, it is highly likely that the brands are close substitutes and so the price of one brand should have a positive relationship with the consumption of another brand. This can be represented as:

$$y = f(x_1(P_{1-n}), \dots, x_n(P_{1-n}))$$

$$\frac{dy}{dP_1} = \underset{(-)}{\frac{\partial y}{\partial x_1}\frac{\partial x_1}{\partial P_1}} + \cdots + \underset{(+)}{\frac{\partial y}{\partial x_n}\frac{\partial x_n}{\partial P_1}}$$

where y represents the demand for iron, whether in binary or continuous form, x_i represents the demand for brand_i, and P_i represents the price of brand_i. Hence, there is a negative component and (n-1) positive components to the effect of any individual price change on iron demand, making the overall sign of each price coefficient ambiguous. In theory, given that a coefficient in a regression analysis can be interpreted as the influence of the variable in question holding all other included variables constant, if the price of one brand increases when all other prices stay the same, iron has only become less affordable and we should therefore expect a reduction in overall consumption (even in light of substitution to other brands included in the model). However, there is still the possibility of substitution to brands outside of the model (such as over-the-counter brands) that I was unable to obtain data for. Hence, it is still possible to see positive coefficients on price variables.

A further complication is that price information is only available for the brands when they are included on the PHARMAC list. Therefore, without adjustment, a value of zero could either indicate an out-of-pocket price of zero or non-availability on the PHARMAC list. Hence, it is imperative to include a set of dummy variables to indicate for each brand whether it was available on the PHARMAC list at any the given point in time. Controlling for this allows the coefficients on the price variables to only reflect the effects of price changes when the drug is

available on the PHARMAC list. However, the coefficients on the availability dummies cannot be interpreted in isolation as the price variables are effectively interactions between price and availability.

Results

Probit results (Table 5.1) indicate that iron prescription in the first two trimesters does not affect high birth weight risk. Prescription in the third trimester, however, appears to significantly increase high birth weight risk. The results imply that iron prescription in the third trimester increases high birth weight risk by 0.5 percentage points and that the average quantity of iron ingested by women who were prescribed iron (3,560 milligrams) would increase the risk of high birth weight by 0.28 percentage points.[20] No other vitamin or mineral supplement seems to increase high birth weight risk and vitamin D supplements appear to significantly lower the risk.

The effect of maternal and infant characteristics on high birth weight risk closely reflects the findings from the previous chapter: ethnicity, infant gender, mother's age, and parity all exhibit strong effects on high birth weight risk.

Using bivariate probit analysis to account for the potential endogeneity of third trimester iron prescription on high birth weight risk (Table 5.2), I find that the effect is now reversed, implying that iron prescriptions during the third trimester decrease the risk of high birth weight however the effect is no longer statistically significant indicating there is no longer any evidence of iron prescription increasing high birth weight risk. An IVprobit analysis (Table 5.3) lends further support to the lack of a significant relationship.

[20] 0.000000779*3560*100=0.277

Validity tests (Table 5.4) suggest that both sets of instruments had high explanatory power and did not suffer from endogeneity.

Results using the special regressor technique (Table 5.5) on a small subsample from the dataset also support the lack of a significant relationship.

Coefficients for the instruments from the first stage of bivariate probit analyses are shown in Table 5.6.

Conclusion

These results appear to largely invalidate the hypothesis that vitamin and mineral supplements during pregnancy increase the risk of high birth weight. All but iron supplements showed no positive effect on high birth weight risk and the effect of iron supplementation did not withstand controlling for endogeneity using instrumental variable techniques.

6. Macrosomia Health Complications and Definition

In this chapter, I look at the birth outcomes of high birth weight babies to determine how the definition of high birth weight reflects the associated risks and investigate other contended definitions: a continuous measure, gradations, adjustments for gestation length, and adjustments for ethnicity.

Data and Methods

I use data from the Natality Detail File to analyse the birth complications associated with high birth weight under competing definitions. NDF is appropriate for this task due to the large number of observations allowing for precise estimates and the available information on birth outcomes. I focus on four birth outcomes: admission to the neonatal intensive care unit (NICU), requirement of assisted ventilation for the newborn, significant birth injury, and a low Apgar score.

The NICU largely deals with premature or very low birth weight babies but it also has the ability to treat conditions commonly suffered by high birth weight babies such as perinatal asphyxia [65]. I create a dummy variable indicating whether the baby was admitted into a facility or unit staffed and equipped to provide continuous mechanical ventilatory support for a newborn at any time during the baby's hospital stay following delivery.

Assisted ventilation is often required after difficult births. I define a dummy variable to indicate whether the baby was given manual breaths for any duration with bag and mask or bag and endotracheal tube within the first several minutes from birth.

A higher risk of birth injury is frequently cited as a consequence of high birth weight [66, 67]. I create a dummy variable to indicate whether the baby suffered a birth injury as defined by the NDF codebook which includes "any skeletal fractures, peripheral nerve injury, soft tissue or solid organ hemorrhage that requires intervention, including any bone fracture or weakness or loss of sensation, immediately following or soon after delivery" [66].

The Apgar score was proposed in 1953 by Virginia Apgar as a minimally intrusive and easily implementable method of evaluating the condition of the newborn baby soon after birth [68]. A rating of zero, one, or two is given on five criteria: heart rate, respiratory effort, reflex irritability, muscle tone, and colour for a total score out of ten which would indicate the best possible condition of the newborn. Even in light of significant technological advances in neonatal care and diagnostics, the Apgar score remains in use due to its ease of implementation, low level of invasiveness, and effectiveness in assessing the need of medical intervention soon after birth [69, 70]. I create a binary variable indicating if the Apgar score of the baby taken five minutes after birth was less than seven.

To analyse the health complications associated with high birth weight compared to normal birth weight, I use probit multi-variable analysis with the following specification:

(1) $$Adverse\ health\ outcome_i = \alpha + \beta_1\, high\ birth\ weight_i + \beta_2\, low\ birth\ weight_i + \boldsymbol{\gamma controls_i} + e_i$$

The adverse health outcome is a binary variable indicating whether an adverse outcome was encountered during or after birth. I repeat this specification for three adverse outcomes: NICU admission, assisted ventilation, birth injury, and Apgar score less than 7 as described above.

High birth weight is a binary variable indicating whether the infant weighed over 4,500g and low birth weight is a binary variable indicating whether the infant weighed less than 2,500g. The set of controls includes all variables available in the NDF that I have previously considered to be correlates with high birth weight risk as well as additional controls that may be more generally related to birth outcomes.

Previously featured controls include infant gender, a set of dummy variables indicating the years of education attained by the mother, a set of dummy variables indicating the number of previous births to the mother, the mother's age and its square, a set of dummy variables indicating the ethnicity of the mother (Black, White, Asian, Hispanic, Native, or Other), the BMI category of the mother (underweight, normal weight, overweight, obese, or morbidly obese), the amount of weight the mother gained during pregnancy, a dummy variable indicating whether the mother smoked at any stage during pregnancy, and dummy variables for the year of birth.

Additional controls include: a set of variables indicating gestation length - preterm (36 weeks or less), early-term (37-39 weeks), full-term (40 weeks), late-term (41 weeks), or post-term (42 weeks or more), a set of dummy variables indicating in which trimester the mother started prenatal care if at all, a set of dummy variables indicating whether the attendant at birth was a doctor, midwife, or other, and a dummy variable indicating whether the method of delivery was vaginal as opposed to caesarean, with an additional dummy variable to indicate whether it was a vaginal birth after a previous caesarean.

For all following analyses I exclude low birth weight infants as I am only interested in making comparisons between high birth weight babies and babies of a 'healthy' birth weight. This also helps to simplify the interpretations of coefficients.

To analyse the appropriateness of current birth weight cutoffs to define macrosomia, I use a probit model with the following specification:

(2) $Adverse\ health\ outcome_i =$

$$\alpha + \boldsymbol{\beta birth\ weight\ categories_i} + \boldsymbol{\gamma controls_i} + e_i$$

Where 'birth weight categories' is a vector of binary variables indicating whether the infant weighed between 4,000g and 4,500g, 4,500g and 5,000g, or over 5,000g. The counterfactual are normal weight infants; low birth weight infants are excluded from the analysis. The dependent variable is binary as described above.

I repeat the regressions with birth weight defined as a continuous variable with a quadratic relationship to adverse health outcomes:

(3) $Adverse\ health\ outcome_i = \beta_0 + \beta_1\, birth\ weight_i + \beta_2\, birth\ weight^2{}_i +$

$$\boldsymbol{\beta controls_i} + e_i$$

I expect to find a negative value for β_1 and a positive value for β_2 implying that the likelihood of having an adverse health outcome is decreasing with increasing birth weight until some optimum weight at which point it starts increasing the risk. With this information, I can find the optimal birth weights for avoiding specific adverse health outcomes.

To allow for ethnicity-specific macrosomia risks, I use interaction variables between ethnicity categories and birth weight as follows:

(4) $Adverse\ health\ outcome_i =$

$$\beta_0 + \beta_1\, high\ birth\ weight_i + \boldsymbol{\beta_2 ethnicity\ categories_i} +$$

$$\boldsymbol{\beta_3\, high\ birth\ weight_i * ethnicity\ categories_i} + \boldsymbol{\beta_4 controls_i} + e_i$$

If the interaction terms are statistically significant (and of a 'meaningful' magnitude) this will serve as evidence that ethnic groups have different risks when it comes to high birth weight.

To investigate whether high birth weight presents different risks for differing gestation lengths, I include interaction terms between gestation length and birth weight categories:

(5) $$Adverse\ health\ outcome_i = \beta_0 + \beta_1\, high\ birth\ weight_i + \boldsymbol{\beta_2 gestation\ length\ categories_i} + \boldsymbol{\beta_3\, high\ birth\ weight_i * gestation\ length\ categories_i} + \boldsymbol{\beta_4 controls_i} + e_i$$

If the interaction terms are significant and quantitatively different from zero this will imply the risk of high birth weight does vary with gestation length.

Results

High birth weight (compared to normal) increases the probability of requiring assisted ventilation and admission to NICU - by about a fifth as much as low birth weight does (Table 6.1). Specifically, high birth weight increases the probability of requiring assisted ventilation by 1.1 percentage points, an increase of 55% from the baseline risk. The risk of requiring NICU admission is increased by 4.1 percentage points or 108% from the baseline. High birth weight has a much larger effect on birth injury compared to low birth weight; low birth weight does not have a statistically significant relationship with birth injury whereas high birth weight increases the risk by 0.34 percentage points or 301% from the baseline. High birth weight increases the risk of having an Apgar score less than seven by 2.8 percentage points or 301% from the baseline. This is smaller, yet similar in magnitude to the effect of low birth weight which represents a 3.4 percentage point increase in risk of having an Apgar score less than seven.

Pre-term birth unsurprisingly appears to have the most detrimental effect on all outcome measures relative to other gestation lengths (Table 6.1). Somewhat puzzlingly, early term births appear to have better outcomes in some dimensions relative to normal term births: they are less likely to require assisted ventilation, less likely to have significant birth injury, and less likely to have Apgar scores less than seven. However, they are more likely to require an admission to NICU. Late-term and post-term babies generally appear to be more at risk of negative outcomes.

Education has a non-monotonic effect on the probability of negative outcomes: women with only elementary education appear to be able to avoid negative outcomes more successfully than those who completed high school; however, those with completed tertiary education have better outcomes than any of those with lower levels of education. First births are the most likely to encounter negative outcomes; increasing parity after the first has an inconsistent and much smaller effect on outcomes. Mother's age does not significantly affect the risk of birth injury or the probability of requiring assisted ventilation but it does increase the probability of requiring NICU admission and is associated with lower Apgar scores.

Ethnicity has important effects on birth outcomes. Babies of Hispanic and Asian women tend to have better birth outcomes compared to White women. Babies of Black women tend to have lower Apgar scores but are less likely to have birth injuries or be admitted to NICU. Babies of Native women tend to have worse outcomes than Whites. Increasing weight group increases the risk of negative outcomes significantly. Starting pre-natal care later in pregnancy is associated with worse outcomes, even worse if no pre-natal care is received. Vaginal deliveries are less likely to require assisted ventilation or NICU admission and have higher Apgar scores. However, vaginal births are associated with an increased incidence of birth injury, which is unsurprising given the greater stress of vaginal deliveries on the baby. Vaginal births after a previous caesarean are much more likely to suffer all negative

consequences compared to regular vaginal deliveries. Births overseen by a doctor as the attendant at birth tend to have worse outcomes than those overseen by a midwife – likely reflecting at least in part differences in case mix.

Necessity of assisted ventilation and NICU admission decreases with birth weight until 4,487g and 5,086g, respectively, and increases thereafter (Table 6.2). Birth injury risk increases with birth weight throughout the distribution but at a diminishing rate. The probability of having an Apgar score less than seven is minimised at 3,836g and it rises thereafter.

Using a set of alternative cutoffs for macrosomia, I find that increased risks of negative consequences are already apparent past 4,000g of birth weight. Past 4,500g, the probability of requiring assisted ventilation and having an Apgar score less than seven increases significantly as does birth injury risk. Past 5,000g of birth weight, all risks increase markedly.

Although babies of Hispanic and Asian women tend to have better birth outcomes than Whites, the pattern is reversed when the baby weighs over 4,500g (Table 6.4). Babies of Black women and Native women also tend to have relatively worse outcomes when they are of high birth weight compared to Whites.

Gestation has a complicated influence on the negative consequences of high birth weight. Among pre-term births, high birth weight appears to increase the risk of requiring assisted ventilation, NICU admission, and having an Apgar score less than seven. However, it significantly decreases the risk of birth injury amongst pre-term births (pre-term high birth weight babies are still at much higher risk of birth injury than pre-term normal birth weight babies or any other gestation length). This could perhaps be signaling that pre-term high birth weight babies are still relatively under-developed compared to high birth weight babies of longer gestation lengths thus being more likely to require assisted ventilation etc., yet since

they have less time in the womb, the pre-term high birth weight babies are more likely to be on the lower end of macrosomia relative to longer term high birth weight babies and hence might be less at risk of trauma during birth. Early term high birth weight babies are more likely to require assisted ventilation and NICU admission and to incur birth injury normal term high birth weight babies, yet they are less likely to have an Apgar score less than seven compared to normal term high birth weight babies. Late and post term high birth weight babies have relatively lower chances of requiring NICU admission and having an Apgar score less than seven compared to normal term high birth weight babies. However, post-term high birth weight babies are more likely to require assisted ventilation than normal term high birth weight babies. Even when interactions are included, high birth weight alone still shows significant, negative effects on health outcomes over all measures.

The exclusion of low birth weight babies from the regressions may be slightly problematic as it could lead to sample selection bias. In robustness checks I find that inclusion of these observations does not notably alter the results.

Conclusion

My results provide some support for the idea that the definition of high birth weight needs careful attention and should not necessarily be considered solely in binary terms. As the risks of negative health outcomes increase already past 4,000g, defining different 'grades' of high birth weight at 4,000g, 4,500g, and 5,000g may be worth considering. As the effect of high birth weight differs by ethnicity, it could be worth allowing for ethnicity-specific cut-offs. Although there do appear to be differing risks for high birth weight babies at different lengths of gestation, it is unclear that a measure of 'large for gestational age' would be most appropriate. In particular, my findings point to a negative effect of being over the 4,500g

threshold regardless of gestational age and so the “absolute” (rather than/in addition to the relative) size of the infant seems to be important.

7. Future Research

Introduction

In this chapter I visit topics that were beyond the scope of my thesis due to data availability issues in the time frame I had available to complete this thesis. I had intended to address weight gain during pregnancy and the long term effects of macrosomia as part of my thesis. However, as I was unable to obtain the necessary data I instead propose these topics here as promising paths for future research. I specify interesting hypotheses to be tested in regard to these topics and provide some potential suggestions for datasets and methods to be used for this task.

Weight Gain during Pregnancy

Background

Weight gain during pregnancy is frequently cited as a risk factor for macrosomia [46, 71]; however, no study to my knowledge has addressed to what extent this is a causal relationship independent of underlying factors and beyond a mere tautology.

Weight gain during pregnancy is unlikely to be exogenously determined as women who expect to be at risk of giving birth to a low-weight baby (for example due to family history of pregnancy complications that researchers are unaware of) may try to gain more weight during pregnancy, whereas women who expect to be at risk of giving birth to an excessively high weight baby may try to limit their weight gain during pregnancy. Also holding all else constant, a heavier baby must mean that the mother has gained more weight during pregnancy than she would have with a lighter baby since the baby's weight is included in her

measurable weight gain. It is also worth noting that weight gain during pregnancy is likely influenced by the mother's pre-pregnancy weight, as the recommendation for weight gain needed for a healthy pregnancy is inversely related to pre-pregnancy weight. Overall:

$$weight\ gain\ during\ pregnancy = g(prepregnancy\ weight, baby's\ weight, family\ history\ of\ pregnancy\ complications, other\ factors\ observable\ and\ unobservable)$$

Data

Growing up in New Zealand is a longitudinal study based in Auckland, New Zealand. The study began in 2009 and covers a nationally representative cohort of about 7,000 children. The first questionnaire was given to mothers and partners (if available) during pregnancy. It included questions on smoking and alcohol intake during pregnancy, pre-pregnancy weight, weight gain during pregnancy, diet during pregnancy, physical activity, pre-natal care uptake, and vitamin supplementation. Information on physical activity and vitamin supplementation is separated into before pregnancy, during the first trimester, and during the rest of pregnancy. It also includes demographic information such as ethnicity, age, and area of residence as well as specific socio-economic information such as education, household income, occupation, and relationship status.

Later questionnaires enquired about the health status of the infant after birth and other factors such as how long the mother breastfed for.

There are many advantages to the *Growing up in New Zealand* data. It is innovative in being a longitudinal study that starts during pregnancy rather than at birth, allowing for a more careful analysis of how factors during pregnancy affect birth outcomes and beyond. The questionnaire style allows for a larger breadth and depth of information than larger national

data collections tend to cover. As with the MAT, this data is specific to New Zealand so can be used to provide information for a New Zealand context.

On the downside, being from a single cohort, *Growing up in New Zealand* does not allow for any exploitation of variation over time. Another drawback is that relying on questionnaires runs the risk of dishonest, inaccurate, or selective disclosure of information, particularly on sensitive topics such as smoking during pregnancy. Finally, while still reasonable for exploring variation in many birth outcomes, the sample size is relatively small.

Methods

To isolate the causal effect of weight gain during pregnancy, I could exploit a factor that is highly correlated with weight gain during pregnancy but does not directly affect the probability of giving birth to a high birth weight baby. Giving up smoking whist pregnant would likely be highly correlated with weight gain during pregnancy as people tend to put on weight when they give up smoking. Although giving up smoking should not have any direct relationship with the probability of giving birth to a macrosomic baby (compared to being a non-smoker) it is possible that women who quit smoking during pregnancy may be on average more dedicated to their foetus' health than the average non-smoker as they have made a significant sacrifice for the sake of the foetus' health. Exercise during pregnancy is another factor which should be highly correlated with weight gain during pregnancy but should not have an independent effect on the likelihood of having a macrosomic baby. Paternal weight gain during pregnancy would also be a useful factor but data on this is unavailable as far as I am aware.

To account for the endogeneity of weight gain during pregnancy, a bivariate probit approach could be used. Potential instruments could include a variable indicating whether the woman gave up smoking during pregnancy and a variable accounting for physical activity during

pregnancy. Women who continued smoking throughout pregnancy should be excluded from this model as smoking throughout pregnancy has an effect on birth weight. To address the problem of weight gain during pregnancy being related to pre-pregnancy BMI, I would run the regression separately for three different samples: normal weight, overweight, and obese women. The specification could be as follows:

$$Macrosomic\ infant = \beta_0 + \beta_1 \widehat{excessive\ weight\ gain} + \boldsymbol{\beta controls} + u$$

$$excessive\ weight\ gain = \gamma_0 + \gamma_1 gave\ up\ smoking + \gamma_2 physical\ activity + \boldsymbol{\gamma controls} + v$$

Where excessive weight gain is a binary variable with a value of one if the woman gained more weight during pregnancy than the maximum recommendation for her respective BMI category.

As mentioned earlier, it is possible that women who quit smoking during pregnancy may have other differences to non-smoking women that could affect the birth outcome as quitting smoking has shown a commitment to their infant's health beyond that which is required from a non-smoker. To test whether women who quit smoking are on average more committed to their infant's health than non-smokers, I would run regressions with other health commitments such as pre-natal care and length of breastfeeding as dependent variables. If women who quit smoking are similar to non-smokers in their commitment to their infant's health then there should be no difference in their likelihood to use prenatal care or the length of breastfeeding. It would also be important to use validity tests to check that each instrument is sufficiently exogenous and has a high degree of explanatory power with regard to weight gain during pregnancy.

Unfortunately, the initially projected date for allowing external researchers access to *Growing up in New Zealand* was delayed. I was advised by one of the authors that the data would be unavailable within the time frame I had to complete my thesis. Therefore, I was unable to pursue this topic but hope to address it in future work.

Long Term Effects of Macrosomia

Introduction

With growing concerns around obesity in the western world, the problem of macrosomia is becoming increasingly apparent. Macrosomia is known to cause problems during birth for both the mother and the child (see Chapter 6 above), but effects on the long term development of the child have received as yet little attention.

In future work, I would like to examine the relationship between macrosomia and later life obesity and cognitive function. In particular, I could try to determine whether the relationship is a causal one or driven by underlying factors (e.g. genetic factors, pregnancy conditions) . It appears highly plausible that the correlation between high birth weight and later-life obesity could be driven by genetic components which lead to innate tendencies towards higher adiposity. Therefore, conclusions about high birth weight having a causal effect on obesity risk could be misguided.

I would also seek to to determine whether it is high birth weight alone that can cause long term effects or whether the relationship is different for infants of different lengths. If adiposity - rather than birth weight in itself- is the problem then the ponderal index may serve as a more useful indicator of long term effects than birth weight alone. The ponderal index (PI) is similar to BMI but is more appropriate for extreme variations of height and can therefore be used for infants. It seems plausible that long term negative health outcomes would be influenced by excess adiposity in infancy more than by excess size alone.

Background

To my knowledge, reasons for macrosomia causing long term health complications have not been explored in the literature. It would be highly valuable to find out whether macrosomia itself has longer term consequenses or whether it merely acts for a proxy for an unhealthy

environment during pregnancy or for genetic factors. Various studies have found evidence of a relationship between high birth weight and later life obesity[18, 19, 72]. However, no study to my knowledge has been able to adequately control for genetic factors or pregnancy conditions that could affect both macrosomia and later life obesity risk. Therefore, the question of causality remains unanswered.

A study by Seidman et al (1991) of 33,413 infants born in Jerusalem examines the relationship between birth weight and the likelihood of being overweight at age seventeen [18]. They control for ethnicity, paternal education, parity, maternal age, and the area of residence and find that infants weighing over 4,500g were more than twice as likely to be overweight at age seventeen than infants weighing between 3,000g and 3,500g. Unfortunately, the study does not control for parental weight so the relationship could be driven by genetic factors rather than indicating a causal link.

Research using the Nurses Health Study (a panel data set of over 120,000 women in the US followed since the mid-70s) also shows a positive relationship between high birth weight and adult BMI [19]. To control for genetic factors, the participants were asked to identify from diagrams a body mass category (out of nine different options) that most closley resembled their mother at 50 years of age. The authors find that controlling for this proxy for maternal body mass did not affect the relationship between high birth weight and being overweight later in life. The study does not control for other factors that may be related to both birth weight and obesity in adulthood such as maternal age, ethnicity, parity etc. which, combined with the imprecise measure of maternal body mass, renders the result inconclusive of a causal relationship.

Rasmussen and Johansson (1998) use data from all singleton births in Sweden between 1973 and 1976 which had available information on BMI at eighteen years of age (taken from the

military service conscription registry) [72]. Controlling for the area of residency, mother's age, educational level, parity, and marital status they calculate odds-ratios of being overweight (BMI>25) and severely overweight (BMI>30) for different categories of birth weight and length. They find that high birth weight is a risk factor for obesity for both normal and high length babies. The authors were unable to control for other important factors that can influence macrosomia and obesity risk such as smoking during pregnancy, weight gain during pregnancy, and parental obesity which again leaves the causal interpretation of a link between high birth weight and adult obesity somewhat unclear.

Cesur and Kelly (2010) examine the cognitive abilities in childhood measured by math and reading tests in relation to birth weight controlling for a host of relevant variables including socio-economic status, parity, and other pregnancy characteristics and behaviours. [13]. Importantly, they are also able to control for the mother's BMI. Overall, they find significantly negative effects of birth weight over 4,500g on cognitive outcomes. This result holds up to robustness checks including an instrumental variable regression, which provides strongly suggestive evidence that the relationship is causal.

Data

To examine the effect of macrosomia on long term health outcomes, I propose using data from the Dunedin Multidisciplinary Health and Development Study (DMHDS). The DMHDS is a longitudinal study which has followed over 1,000 subjects born in Dunedin, New Zealand, from birth into adulthood interviewing the participants on a range of health factors. The DMHDS is a useful source of information as it follows subjects over a long period of time (over 30 years) yet still boasts a high retention rate. It also features a measure of length at birth as well as weight which is important for being able to examine a ponderal index measure of the infant rather than just weight alone.

Unfortunately, the DMHDS starts at birth rather than during pregnancy so conditions in pregnancy cannot be controlled for using the core study alone. However, there are two extension studies of the DMHDS which can partially overcome this problem. The first is the Family Health Study (FHS) which asks questions of the parents of DMHDS participants about their health. This includes questions on their weight, height, physical activity, and smoking history. Although the information is taken many years after pregnancy, it can act as a proxy for BMI and behaviours during pregnancy. The other extension study is the Next Generation Study (NGS) which interviews the teenage children of participants. This interview asks questions about their health and tests their cognitive abilities, as well as asking their caregiver about pregnancy conditions such as gestation length, complications (including excessive weight gain), and smoking status.

From the DMHDS, two datasets could be requested: one examining the later life obesity and cognitive outcomes for DMHDS members using data from the FHS for controls, the other examining obesity and cognitive outcomes of the children of DMHDS members at age fifteen, with caregiver data collected in the NGS and DMHDS data to use for controls. Ideally, the controls would contain a measure of maternal age, parity, income, education, marital status, gestation length, maternal obesity during pregnancy, parental obesity, smoking behaviour during pregnancy, and weight gain during pregnancy. .

Methods

To examine the realtionship between macrosomia and obesity in later life, I could use econometric methods such as logit and probit. In a baseline specification, I would use the common definition of macrosomia as a birth weight over 4,500g. Following convention, I would define obesity as having a BMI over 30. I would use a probit model with the following specification:

$$Obese = \beta_0 + \beta_1 macrosomic\ infant + e$$

To examine whether macrosomia correlates with the probability of being obese in later life, I would calculate marginal effects from the above estimation.

The next stage of analysis would involve adding controls to the regression:

$$Obese = \beta_0 + \beta_1 macrosomic\ infant + \boldsymbol{\beta Controls} + e$$

The controls should include all previously metioned variables available from the datasets that would be related to both obesity risk and macrosomia risk. Control variables that do not affect adult obesity may serve as instruments for macrosomia in instrumental variable regression analysis. This would provide stronger evidence for a causal link. If suitable instruments can be found (instrument validity can be examined using methods such as the Hausman test) then I could conduct instrumental variable regression analysis as follows:

$$Obese = \beta_0 + \beta_1 \widehat{macrosomic}\ infant + \boldsymbol{\beta Controls} + u$$

$$macrosomic\ infant = \gamma_0 + \gamma_1 instrument + \boldsymbol{\gamma Controls} + v$$

If a significant positive relationship is found between macrosomic infants and adult obesity at all stages of analysis, this would provide suggestive evidence of a causal relationship.

To examine the effect of macrosomia on cognitive outcomes, I could repeat the three stages of analysis with results from cognitive tests taken at various stages of the DMHDS as the dependent variable. If a significant negative relationship is found at all stages of analysis this combined with previous research from Cesur and Kelly (2010) would provide compelling evidence that macrosomia has negative consequences on cognitive outcomes.

To examine whether obesity in adulthood is caused by infant size alone or foetal adiposity, I would add measures of infant's length to the analysis. Initially, I could repeat the analyses set

out above with a ponderal index value in place of the binary variable indicating whether the infant was macrosomic. If the relationship is stronger for the ponderal index this would indicate that adiposity is more important than size alone in detemining later life obesity. I could then repeat the analyses with birth weight, length and an interaction between them as separate variables. If body size independent of length is important then the coefficient on the interaction of weight and length should be positive and significant. If high adiposity is problematic but size itself is not, then the coefficient on weight should be positive, the coefficient on length should be negative, and the coefficent on the interaction should be insignificant.

Research Aims

This research could help inform us about the negative consequences of macrosomia and may help motivate public health providers to prioritise measures to reduce the rate of macrosomia. These measures may include informing women about the dangers of obesity and excessive weight gain during pregnancy and taking a more selective approach to prescribing prenatal vitamin supplements to promote foetal growth.

Unfortunately, I have been denied access to work with the highly exclusive DMDHS data so was unable to pursue this topic. However, if other datasets with information on birth weight, length, and long term outcomes become available, this could be an informative research topic.

8. Conclusion

Research Outcomes

In Chapter 2, I described the situation of macrosomia in New Zealand and found that New Zealand's experience largely reflects findings in the existing international literature. Our incidence of macrosomia appears slightly higher than in other nations but it has not been rising over recent years. Weight categories, mother's ethnicity, and infant gender appear to have strong effects on macrosomia risk.

In Chapter 3, I explored the relationship between high birth weight and socio-economic status. Generally, socio-economic status tends to improve health outcomes but in the case of birth weight the relationship is more complex as both extremes (low and high birth weight) imply negative health outcomes and socio-economic status may either aid in the avoidance of both negative outcomes or it may lower the risk of one at the expense of increasing the risk of the other. I presented theoretical models underlying these competing hypotheses and an empirical analysis to determine which one is more plausible. I found that although a descriptive analysis suggests that high socio economic status indicators of education, marital status, and income correlate with higher risk of high birth weight, when I controlled for other factors such as ethnicity, parity, and infant gender the relationship was largely reversed. This suggests that socio-economic status does lower the risk of high birth weight. However, further analysis suggested that at broader definitions of high birth weight, some socio-economic indicators become risk factors for high birth weight. This could imply that there is a combination of both of the hypothetical effects of socio-economic status present, such that the risk of moderately high birth weight is increased with higher socio-economic status but the risk of extreme high birth weight is lowered.

In Chapter 4, I dissected the causality of the relationship between obesity and high birth weight risk. Obesity is frequently cited as a risk factor for high birth weight in the literature but there is a dearth of scrutiny over the causal component in this correlation. Conclusions are often made which endorse an implied causality yet overlook obvious potential for spurious correlation; maternal body mass could well be endogenously correlated with high birth weight risk due to a possible genetic pre-disposition to high adiposity. I employed an instrumental variable analysis to explore this possibility. Specifically, I used rurality, fast-food restaurant concentration, and mother's birth month as instruments for maternal BMI category and found that when controlling for the endogeneity of obesity, the effect of obesity on high birth weight risk is no longer significant except for morbidly obese mothers (BMI>35). Even for morbid obesity, the effect on high birth weight risk falls to nearly one tenth of what is implied by an analysis which does not account for the endogenous component.

In Chapter 5, I investigated the relationship between vitamin supplement intake and high birth weight risk. While there is ample evidence in the literature demonstrating that vitamin supplements can increase birth weight, no study to my knowledge has attempted to uncover whether this relationship implies that vitamin supplement intake may increase the risk of high birth weight. Probit analysis suggests that only iron supplementation in the third trimester of pregnancy has a significant correlation with high birth weight risk. However, when I used instrumental variable analysis to account for the potential endogeneity of iron prescription, the relationship became statistically insignificant.

In Chapter 6, I investigated the negative birth consequences of high birth weight and analysed whether the most common definition of high birth weight (birth weight >4,500g) adequately reflects the threshold at which the harms of high birth weight become apparent. To do this, I analysed what risks are implied when a continuous measure of birth weight is employed as

opposed to discrete categories, what risks are implied at different thresholds of birth weight (4,000g, 4,500g, and 5,000g) and whether the risks differ for women of different ethnic groups and pregnancies at different gestation lengths. I found that while high birth weight does overall present lower risks than low birth weight it still significantly increases the risk of requiring assisted ventilation and NICU admission, suffering birth injury and having a low Apgar score. Continuous measures of birth weight relative to discrete show that increasing birth weight decreases the risks of assisted ventilation, NICU admission and having a low Apgar score initially but the relationship reverses once a certain threshold of weight is reached, whereas increasing birth weight increases the risk of suffering birth injury throughout but at a diminishing rate. By considering categories of birth weight at different thresholds, I showed that overall there do appear to be increased risks already at a lower threshold of 4,000g and that the risks are substantially larger past 5,000g. I found that high birth weight presents higher risks for Asian, Hispanic, Black, and Native women compared to White women – suggesting that optimal birth weight categories may be ethnicity-specific.

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Policy Implications

Macrosomia is becoming an increasingly important problem. My research may help increase awareness of the issue in New Zealand and could also be helpful in formulating policy guidelines with respect to weight maintenance before/during pregnancy and vitamin supplementation. It could also be relevant to targeting advice for pregnant women based on socio-economic. Finally, I demonstrate that there may be need for reconsideration of the definition of high birth weight.

An analysis of the overall situation with macrosomia in New Zealand has shown that Pacific women are most at risk of delivering a high birth weight infant relative to other ethnicities

and that women giving birth to male infants are at a higher risk. These findings were corroborated in multivariate analyses in other chapters. It is important that maternity carers be aware of those who are at higher risk so advice and care can be appropriately targeted. Similarly, the finding that socio-economic status overall appears to reduce the incidence of high birth weight could imply that women of lower socio-economic status may require extra advice and care to help them avoid macrosomia and the negative birth outcomes associated with it.

My research on socio-economic status and high birth weight could also have broader implications for studies on socio-economic status and general health outcomes where inputs may have U-shaped effects (e.g., beneficial at low values but potentially harmful at higher levels). For instance, the theoretical analysis might be applicable to efforts such as encouraging higher food intake for children (e.g., breakfast in schools programmes) that may have partial negative consequences of increasing childhood obesity.

An important finding from Chapter 4 which could have broader implications is the drastic change in the effect of BMI on high birth weight risk when controlling for endogeneity. This has strong implications for advice given to women trying to conceive. In particular, previous literature would have led one to believe that losing weight before conception was imperative for obese women to lower the risk of high birth weight but my results suggest this is of much less importance and only is appropriate for women in the morbidly obese category. This could be worthy of consideration for medical practitioners when advising overweight women who are trying to conceive; encouraging weight loss may warrant less emphasis than is currently given. This could also well imply that we need more caution when looking at the effect of BMI on other conditions when endogeneity may be present. Obesity is blamed for a number of negative health outcomes and potential policies to combat obesity are gaining greater acceptance and prominence in the public sphere. If the causal component of the effect

of obesity on negative health outcomes has been overstated in other instances as well, the benefits of weight loss policies may be overstated.

My findings on vitamin and mineral supplementation during pregnancy do not suggest that any changes are required to current prescribing or subsidisation policies. However, the analysis could be easily extended to assess the effect of vitamin supplementation on other birth outcomes which may lead to policy implications in regard to other areas of infant health.

Finally, my analysis of the consequences and definition of high birth weight strengthens the evidence of negative health outcomes associated with high birth weight and suggests that there is some basis for reconsidering how we define macrosomia. In particular, the differing effects of high birth weight on negative outcomes for different ethnic groups suggest that different thresholds for different ethnicities may be worth consideration. The risk differentials apparent for different thresholds of birth weight may warrant consideration of *grades* of high birth weight. Differences in the effect of high birth weight on outcomes for babies at different gestation lengths were relatively minor and also non-monotonic compared to the overall effect of high birth weight. This would seem to suggest that measures of 'large for gestational age' may not have high value in predicting negative outcomes.

Discussion

A broader issue I have encountered in my research is the disconnect between health research undertaken from a public health researcher's perspective and that from an economist's perspective. Most medical research relies on controlled studies, which are generally costly, and difficult to implement. Oftentimes, the health-related interventions that we are interested in analysing are not possible to implement in a controlled trial due to ethical considerations or simple impracticality. When this occurs, large-scale population data can serve to aid analysis.

However, as there is no control to use for comparison, extra care needs to be taken to address endogeneity issues so as not to make erroneous causal inference from endogenous correlations. Some public health research appears to not take as much care when analysing topics where endogeneity is highly likely to be present. For example, not a single paper I encountered addressed the possibility of maternal obesity being endogenously correlated with high birth weight risk.

Given the high culpability of genetic components influencing birth outcomes that may be blamed on behaviours during pregnancy or characteristics of the pregnant woman, it is imperative, yet often very difficult, to address endogeneity when analysing determinants of birth outcomes. A strategy to estimate the effects of pregnancy behaviours or characteristics of the pregnant woman on birth outcomes that avoids endogeneity issues caused by genetic components that has so far not been exploited (to my knowledge) would be to restrict a sample to gestational surrogate mothers, ideally where information on both the biological mother and the surrogate is available. As the surrogate is genetically unrelated to the foetus (in most circumstances, traditional surrogacy aside) her behaviours during pregnancy and her characteristics can be more cleanly interpreted as having a causal effect on birth outcomes. In the context of my research, one could analyse the effect of the biological mother's and the surrogate's BMI on the risk of high birth weight. My findings seem to suggest the relationship is driven by underlying endogenous factors (most plausibly genetic components), hence I would anticipate the results showing the biological mother's BMI having a much larger impact on high birth weight risk than the surrogate's (if the surrogate's has any discernible effect at all).

Analysing the effect of paternal characteristics on birth outcomes could also shed light on the effect of genetic components as opposed to the pregnancy environment. However, there are two major issues with this: data availability and endogeneity from mate selection. None of

the datasets I utilised in my research had any more than cursory information about the father. Obtaining extensive information about the father presents a much more difficult task than obtaining information about the mother as the father will not necessarily be present at appointments where data is collected. Fathers who are more involved in the birth may be easier to get information about but this would create sample selection issues: if absent fathers differ systematically from involved fathers then results based on readily available information may not be generalisable to all fathers. Another endogeneity problem comes from mate selection. Couples are not randomly assigned and characteristics that are being evaluated may have influenced the mother's choice of the father and may therefore be endogenously correlated with characteristics of the mother. However, it may still be a viable option to consider when attempting causal inference about the pregnancy environment on birth outcomes.

Ultimately, it appears that high birth weight deserves more attention as a serious concern for childbirth. More awareness about the risk factors would be beneficial for medical practitioners when advising pregnant women. However, once again, research into risk factors for birth outcomes requires careful attention to potential endogeneity issues. In this regard, it would be highly beneficial for health economists to pay more attention to high birth weight as an important issue.

Overall, this thesis has presented novel research on issues relating to macrosomia and has produced interesting empirical findings which may have important policy implications. It also brings light to issues which may have broader implications in general health research.

Appendix A - Obesity Measurements

The most common way to measure obesity is to use the Body Mass Index (BMI). The major advantage of BMI is that it is easy to measure. However, it has been criticised due to inaccuracies in predicting the actual level of adiposity and therefore giving an imprecise measure of healthy standards.

The Body Mass Index is calculated by the formula:

$$\frac{Weight\ in\ kilograms}{(Height\ in\ metres)^2}$$

The resulting value can then be used to determine which weight category an individual belongs. The BMI range used to determine different weight categories varies. In my thesis I have defined weight categories as follows:

BMI range	**Weight category**
BMI<20	Under weight
20<BMI<25	Normal weight
25<BMI<30	Overweight
30<BMI<35	Obese
BMI>35	Morbidly obese

A major benefit of using BMI as a measure of obesity is that it is much simpler to measure than alternatives. A more accurate measure of body fat percentage can be obtained with methods such as hydrostatic weighting which requires expensive machinery and a trained operator and hence is unfeasible to obtain for larger data sets. Other simple measures such as skin-fold body-fat percentages measured with callipers or hip-to-waist ratio do offer higher

accuracy than BMI yet the measurements can be unreliable; widely different readings can be obtained depending on where the measure is taken from, or if the instrument (tape or callipers) are not used correctly. It is much easier to get accurate height and weight measurements as most people have the ability and means to measure these themselves and so is ideal for inclusion in a survey. As BMI is the easiest measure to obtain there is a wealth of data that has utilised it making comparisons across studies easier.

BMI has been criticised for failing to account for the fact that muscle tissue is heavy so a simple measure of weight will not necessarily reflect the level of adipose tissue relative to muscle[73]. This can lead to athletic people with high muscle density and low body fat being classed as “overweight” or even “obese”. However, this should not be as much of a concern for females since females do not have the same capacity to grow muscle tissue as males. It is reasonably safe to say that inaccuracies of BMI measurements due to muscle weight are unlikely to radically skew results when solely looking at females

Appendix B – Lead Maternity Carers

Maternity care in New Zealand operates under a system where the pregnant woman nominates a ‘Lead Maternity Care’ (LMC) who is responsible for providing care during pregnancy and delivery [74]. Alternatively, women may choose to rely on DHB-funded maternity care services. The LMC may consult or contract for services required during pregnancy or delivery but remains accountable for co-ordinating the services unless another LMC is nominated. Women can choose a midwife, GP, or obstetrician as their LMC. LMCs are publically funded via a fixed fee for each pregnancy. There is no cost to the woman if they choose a Midwife or GP but obstetricians are allowed to charge an additional fee. If the woman is referred to an obstetrician due to complications, however, she would not have to pay any additional fees.

New Zealand has a very high proportion of births attended primarily by midwifes. Around 90% of women who registered with an LMC chose a midwife[75].

Appendix C – New Zealand Regional Units

Unlike Nations with a federal system of governance which allows for relatively simple division of the country into smaller areas for analysis, New Zealand has significantly less consistency in the areas commonly used. Therefore, choosing how to divide New Zealand into smaller areas for analysis is not always a simple task.

New Zealand has a number of official ways to divide the country into smaller areas.
In my thesis I exploit District Health Board (DHB) boundaries, Territorial Local Authority (TLA) boundaries, Census Area Units (CAUs) and Meshblocks.

District Health Boards

The District Health Board system was established by The New Zealand Public Health and Disability Act 2000 and has been in operation since January 1 2001. New Zealand's health system is largely government funded. Roughly three-quarters of these funds are allocated to DHBs who are responsible for planning, purchasing and providing health services, including public hospitals and the majority of public health services, within their areas. The boards are made up of appointed members and elected members. The boards' performances are monitored and held accountable to the Ministry of Health but otherwise are given free rein to make their own decisions regarding their operations. New Zealand currently has 20 DHBs after the Otago and Southland DHBs merged in May 2010 to create the Southern DHB.

[21]

Territorial Local Authorities

Territorial Local Authority areas (TLAs) are defined by the city and regional council boundaries. They are a convenient measure to exploit as they generally encapsulate cities or major towns and the surrounding rural area. Hence, they have varying populations but reflect

[21] Map sourced from www.health.govt.nz/new-zealand-health-system/key-health-sector-organisations-and-people/district-health-boards/location-boundaries-map Accessed June 2014

areas that have a genuine commonality. There are 73 TLAs in New Zealand with a median population of about 30,000.

[22]

Census Area Units

Census Area Units represent electoral boundaries for a portion of the available general electoral seats in parliament. Under New Zealand's electoral system, Mixed Member

[22] Map sourced from http://en.wikipedia.org/wiki/Territorial_authorities_of_New_Zealand#mediaviewer/File:NZTerritorialAuthorities.png Accessed June 2014

Proportional Representation (MMP), 70 of the 120 seats in parliament are elected through electorates as opposed to list seats. As of 2011, seven of these electoral seats are assigned to the Maori roll which has a separate system of defining the boundaries; the remaining 63 represent the general roll seats. Census area unit are taken as sub-regions of TLAs and generally contain 3000-5000 people.

Meshblocks

The smallest level of aggregation I use is the meshblock which is much more precise, generally covering no more than a city block in urban areas. Meshblocks are used by the New Zealand deprivation index. Meshblocks have a median population close to 90 but vary considerably[76]. Meshblocks are sub-regions of CAUs and hence can be aggregated to build CAUs or TLAs.

Appendix D – Regression Techniques

Multivariate Regression – Ordinary Least Squares

Multivariate analysis allows for estimation of the individual effects of a set of independent variables on a defined dependent variable even when these independent variables affect the dependent variable simultaneously. Starting with the model:

$$\boldsymbol{y} = \boldsymbol{X}\boldsymbol{b} + \boldsymbol{e}$$

Where $\boldsymbol{y}$ represents a vector of values of the dependent variable for every observation, $\boldsymbol{X}$ represents a matrix of values for each independent variable for every observation, $\boldsymbol{b}$ represents a vector of coefficient values which describe the effect of each independent variable on the dependent variable, and $\boldsymbol{e}$ represents a vector of values of the random error term for each observation.

Minimising the sum of squared error terms, $\boldsymbol{e}^T\boldsymbol{e}$, solves to produce the ordinary least squares estimator: $\widehat{\boldsymbol{b}} = (\boldsymbol{X}^T\boldsymbol{X})^{-1}\boldsymbol{X}^T\boldsymbol{y}$

It can be shown that the OLS estimator produces estimates of the coefficients which are efficient and unbiased if certain conditions are maintained. These conditions specify that the model be correctly specified, independent variables must not be linearly dependent, and the error term must have a constant variance, be uncorrelated with any of the independent variables, be independent between observations, and follow a normal distribution.

In my analysis, the assumption of constant variance of the error term across observations is questionable. To address this I report heteroskedasticity-robust standard errors instead of OLS standard errors. I also encounter models where the independent variables may be

correlated with the error term, and non-linear relationships between the independent and dependent variables. OLS is ill-equipped to deal with these scenarios so more sophisticated techniques are required.

IV

Instrumental variable analysis is appropriate to use when particular independent variables may be considered endogenous (correlated with the error term). Instrumental variable analysis requires variables that are uncorrelated with the error term and highly correlated with the endogenous independent variable(s) to serve as 'instruments'. A commonly used procedure to implement instrumental variables is the two-stage least squares (2SLS) method. This involves using OLS to estimate predicted values for the endogenous variable(s) using all exogenous independent variables and the instruments as regressors. These predicted values are then included in an OLS regression as independent variables along with all other exogenous independent variables but excluding the instruments. This method removes the bias implied by endogeneity in the independent variables.

$$\widehat{\boldsymbol{\delta}} = (\boldsymbol{Z}^T\boldsymbol{Z})^{-1}\boldsymbol{Z}^T\boldsymbol{X}$$

Predicted values of the endogenous variables (and the observed values of the exogenous independent variables) is given by the following equation:

$$\widehat{\boldsymbol{X}} = \boldsymbol{Z}\widehat{\boldsymbol{\delta}} = \boldsymbol{Z}(\boldsymbol{Z}^T\boldsymbol{Z})^{-1}\boldsymbol{Z}^T\boldsymbol{X} = \boldsymbol{P}_{\boldsymbol{Z}}\mathbf{X}$$

The resulting estimator solves to:

$$\widehat{\boldsymbol{b}} = (\boldsymbol{X}^T\boldsymbol{P}_{\boldsymbol{Z}}\boldsymbol{X})^{-1}\boldsymbol{X}^T\boldsymbol{P}_{\boldsymbol{Z}}\boldsymbol{y}$$

Which can be shown to be unbiased and efficient.

Validity Tests

To ensure that instruments are appropriate we need to test that the instruments are uncorrelated with the error term and strongly correlated with the endogenous independent variable. Tests of the over-identifying restrictions can be used to determine if the instruments are uncorrelated with the error term. These tests involve regressing the residuals from the 2SLS regression on the exogenous variables. If the residuals are found to be correlated with the instruments then the instruments are invalid. Strength of the explanatory power of the instruments can be determined using a test of the joint significance of the instruments from the first stage regression. A resulting F-statistic above 10 is generally accepted as evidence of a strong correlation between the instruments and the endogenous variable.

Probit

Where the dependent variable in a regression follows a binary form, OLS is not an appropriate estimator as it can produce predictions of the dependent variable below zero or above one. As predicted values of the dependent variable are most appropriately considered as probabilities of the dependent variable taking the value of one, values outside of the range zero to one should be impossible. OLS also assumes a linear relationship between independent and dependent variables which is not necessarily accurate when the dependent variable is binary. In its place, the probit estimator can be used.

The Probit estimator uses a maximum likelihood procedure as opposed to least squares which allows for a non-linear relationship between the independent and dependent variables. The conceptual framework underlying the probit procedure can be represented as a latent variable model. If we assume the dependent variable will take the value one when an unobserved variable takes a positive value and zero otherwise. Hence:

$$\boldsymbol{y}^* = \boldsymbol{Xb} + \boldsymbol{e}$$

$$\boldsymbol{y} = \begin{cases} 1 \; if \; \boldsymbol{y}^* > 0 \\ 0 \; if \; \boldsymbol{y}^* < 0 \end{cases}$$

Or:

$$\boldsymbol{y} = \begin{cases} 1 \; if \; \boldsymbol{Xb} + \boldsymbol{e} > 0 \\ 0 \; if \; \boldsymbol{Xb} + \boldsymbol{e} < 0 \end{cases}$$

The coefficients produced by the probit procedure do not have the simple interpretation as they do in OLS as they do not reflect the direct effect of the independent variable on the dependent variable, rather the effect that the independent variable has on the value of the latent variable and how this enters the probability function of $\boldsymbol{y}$. In order to interpret coefficients, marginal effects must be computed. This allows us to infer the effect a one unit increase of the independent variable would have on the probability that $\boldsymbol{y}$ takes the value one when the independent variable takes its mean value.

Bivariate Probit

Bivariate probit can be used when the dependent variable is binary and there exists an endogenous binary variable. Bivariate probit involves estimating two latent variable models using maximum likelihood methods.

IVprobit

The IVprobit method is appropriate where there is a binary dependent variable and a continuous, endogenous independent variable. The IVprobit method works by following the same ‘first-stage’ of the 2SLS estimator, regressing the endogenous variable on the set of

instruments, then takes the residuals from this regression and uses them as a variable in a probit regression. Similar to probit, marginal effects must be estimated in order to interpret the effect of the independent variables on the probability of the dependent variable taking the value one.

Special Regressor Method

The special regressor method involves transforming the binary dependent variable into a continuous variable which then allows for regular 2SLS estimation. The transformation requires a 'special regressor' which must be a continuous variable that appears additively to the error term in the model. Assuming the latent variable model can be represented with a linear specification, the additivity condition will be met by any of the independent variables [77].

If we specify the latent variable model as:

$$\boldsymbol{y}^* = \boldsymbol{V} + \boldsymbol{Xb} + \boldsymbol{e}$$

where $\boldsymbol{V}$ represents the special regressor and all other values are as described above, it follows that:

$$Y = \begin{cases} 1 \; if \; \boldsymbol{y}^* > 0 \\ 0 \; if \; \boldsymbol{y}^* < 0 \end{cases}$$

The transformation of the dependent variable is as follows:

$$T = \frac{Y - I(V \geq 0)}{f_{v|Z}(V|Z)}$$

where $I(.)$ represents an indicator variable that takes the value one when the condition inside the brackets is met, otherwise it takes the value of zero, $f_{v|Z}$ denotes the conditional

probability function of V given Z, and V has been de-meaned. It can be shown that the transformed dependent variable can reflect the latent variable [78]:

$$E(T) = E(\boldsymbol{y}^*)$$

Therefore, we can use the transformed variable as the dependent variable in a 2SLS regression.

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Tables and Figures

Table 1. Mean Comparison Between Women with DHB-Funded and Community-Funded Midwives, MAT, 2007-2011

	DHB-Funded	Community-Funded
Percent European	27.83	54.89
Percent Urban	63.99	59.88
Age	28.43	29.20
Deprivation Decile	7.02	6.01
Percent High Birth Weight	2.48	2.52

Figure 2.1 Percentage of Macrosomic Births in New Zealand, MAT, 2001-2011

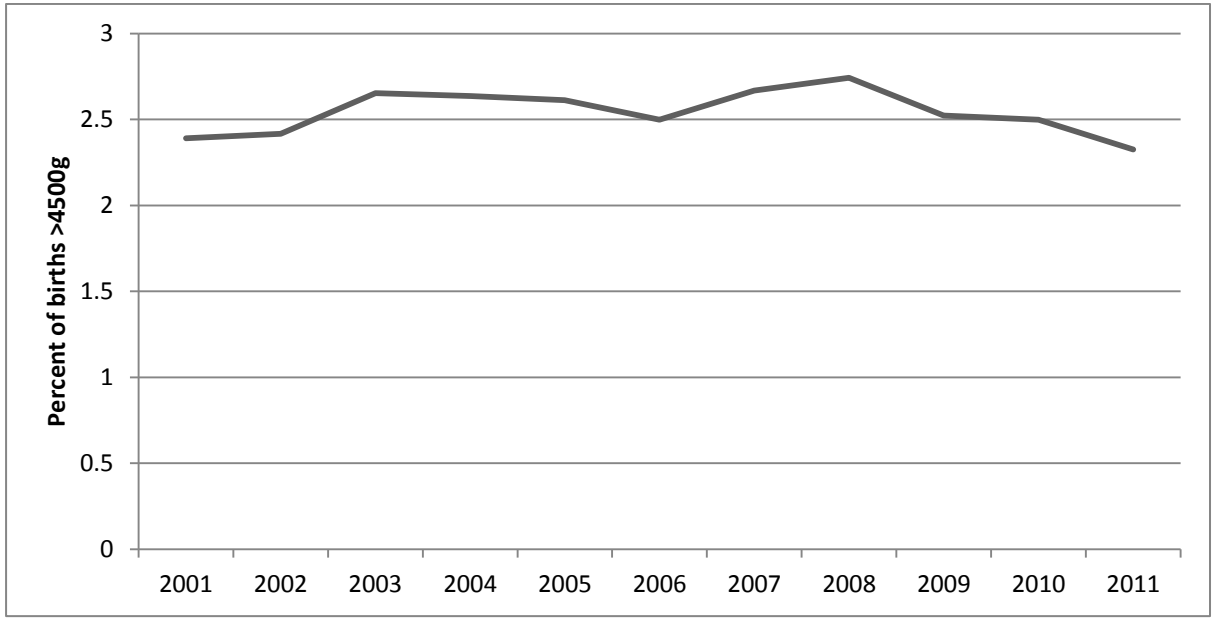

Figure 2.2 Percentage of Macrosomic Births by Weight Group in New Zealand, MAT, 2001-2011

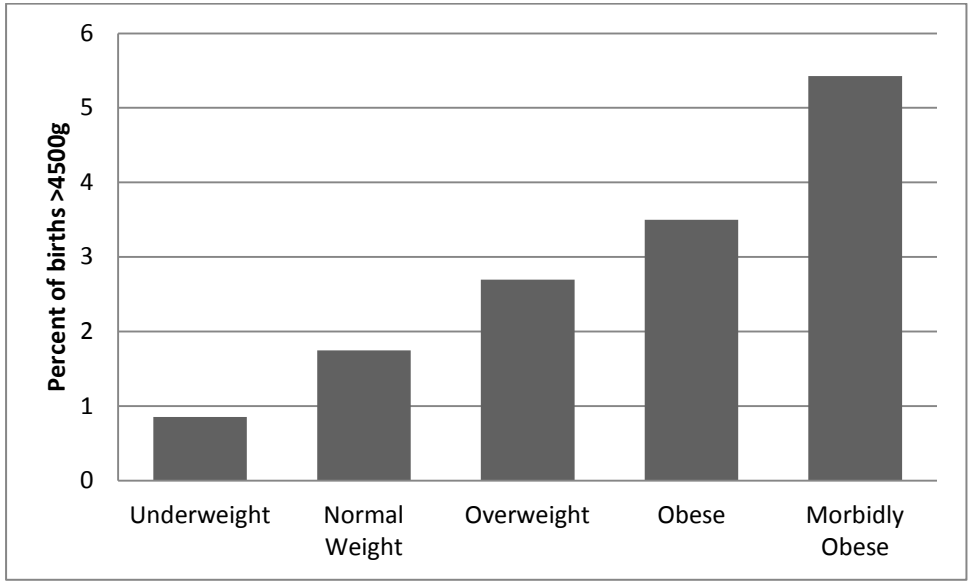

Figure 2.3 Percentage of Macrosomic Births by Ethnicity in New Zealand, MAT, 2001-2011

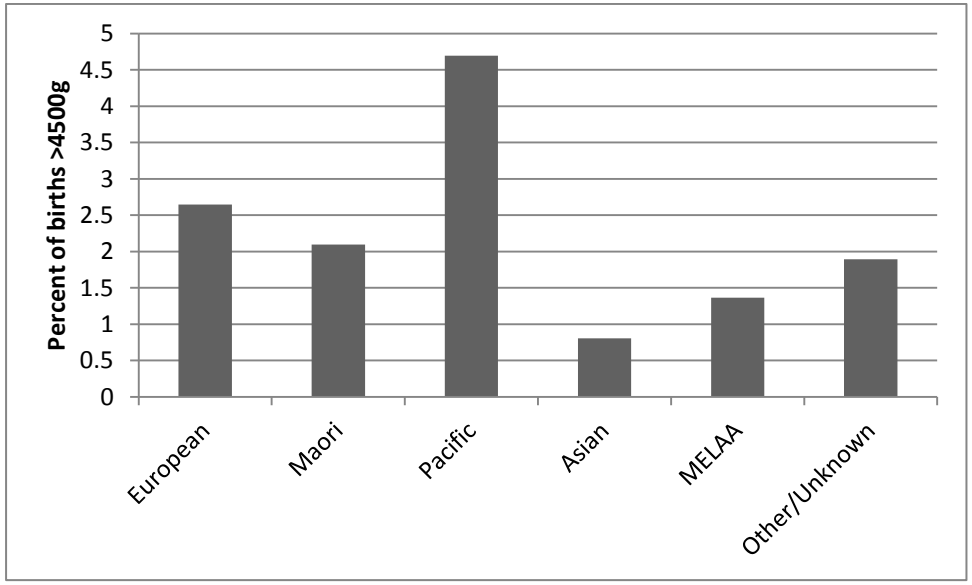

Figure 2.4 Gender Distributions of Normal Weight Compared to Macrosomic Births in New Zealand, MAT, 2001-2011

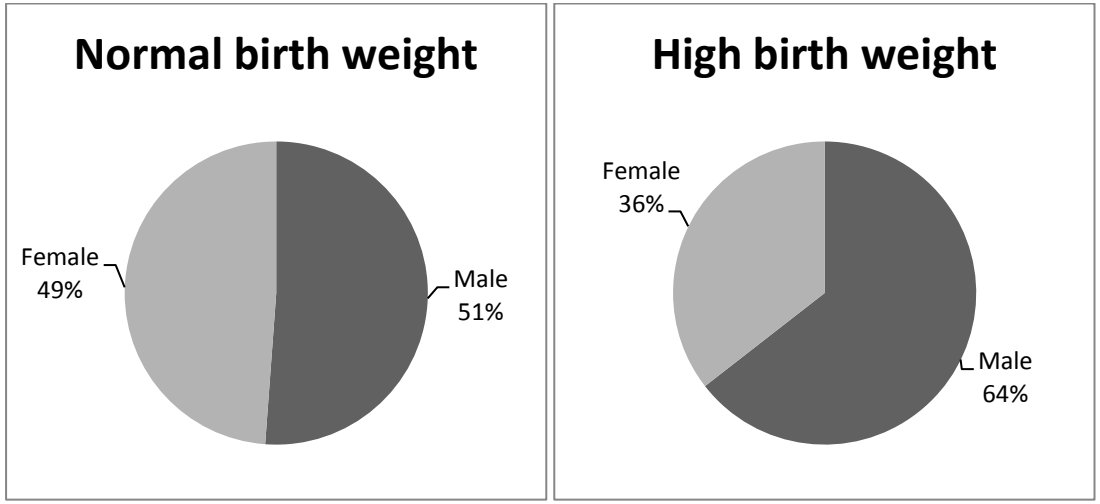

Figure 2.5 Percentage of Birth Complications by Birth Weight Group in New Zealand, MAT, 2001-2011

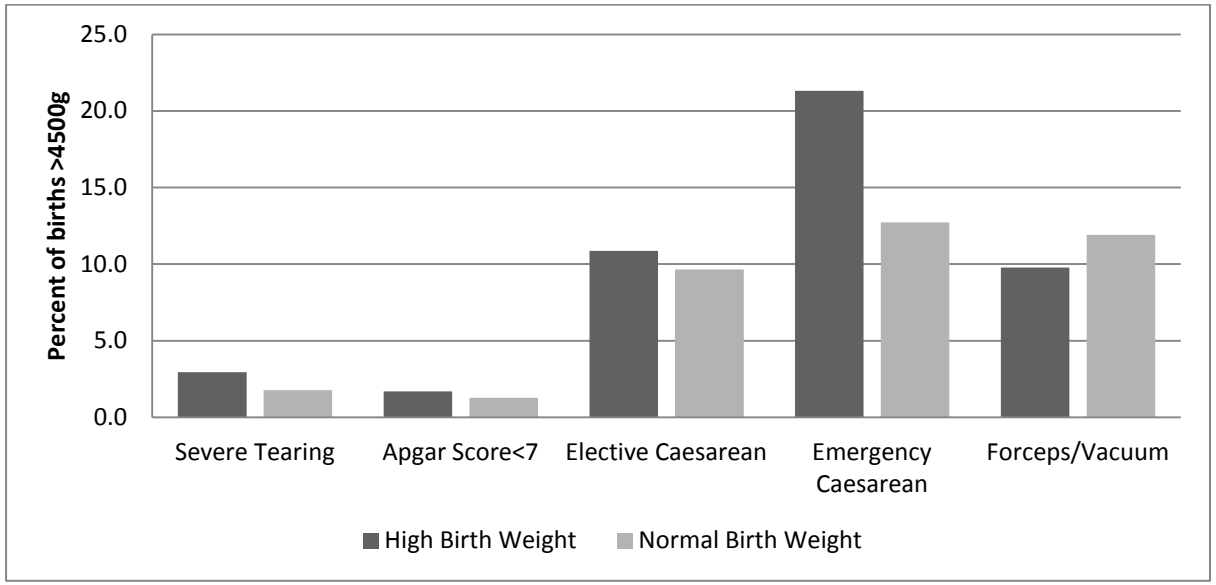

Table 3.1 Percentage of Mothers in Socio-Economic Categories by Birth Weight; PRAMS, 2003-2009

	Low Birth Weight	Normal Birth Weight	High Birth Weight
Elementary education	4.70***	4.43	4.20
High school drop out	21.04***	14.44	9.18***
High school	35.44***	31.50	31.27
Some college	20.75***	22.90	24.07
College degree	18.06***	26.73	31.28***
Married	51.01***	65.78	76.28***
Household income <$10,000	28.62***	19.47	13.09***
Household income $10,000-$20,000	20.40***	17.29	14.38***
Household income $20,000-$30,000	10.46	10.68	11.53
Household income $30,000-$40,000	9.88***	10.80	12.80**
Household income >$40,000	30.64***	41.76	48.20***

*Where *, **, *** indicate significant difference from the Normal Birth Weight percentage at the 10%, 5%, and 1% level, respectively.*

Table 3.2 Percentage of High Birth Weight Babies by Education Level; PRAMS, 2003-2009

Education	Elementary	High school drop out	High school	Some college	College degree
High Birth Weight	1.33	0.87	1.39	1.49	1.68

Table 3.3 Percentage of High Birth Weight Babies by Household Income Level; PRAMS, 2003-2009

Household Income	<$10,000	$10,000-$20,000	$20,000-$30,000	$30,000-$40,000	>$40,000
High Birth Weight	0.94	1.18	1.54	1.70	1.67

Table 3.4 Percentage of High Birth Weight Babies by Marital Status;

PRAMS, 2003-2009

Marital status	Married	Not married
High Birth Weight	1.65	0.95

Table 3.5 Percentage of Mothers in Socio-Economic Categories by Birth Weight; NDF, 2003-2009

	Low Birth Weight	Normal Birth Weight	High Birth Weight
Elementary education	2.85***	3.30	3.40***
High school drop-out	10.28***	8.24	5.84***
High school	18.70***	17.10	15.84***
Some college	12.08***	12.39	13.43***
College degree	11.14***	16.16	18.50***
Married	48.86***	62.26	71.10***

*Where *, **, *** indicate significant difference from the Normal Birth Weight percentage at the 10%, 5%, and 1% level, respectively.*

Table 3.6 Cesur and Kelly (2010) Replication; PRAMS, 2003-2009

	Marginal Effect
Married	0.0010 (0.0008)
High school drop out	-0.0032* (0.0016)
High school	-0.0021 (0.0017)
Some college	-0.0025 (0.0017)
College degree	-0.0015 (0.0018)
Household income $10,000-$20,000	0.0006 (0.0011)
Household income $20,000-$30,000	0.0031** (0.0015)
Household income $30,000-$40,000	0.0033** (0.0015)
Household income >$40,000	0.0022* (0.0012)
Male infant	0.0091*** (0.0006)
Mother's BMI	0.0023*** (0.0003)
Mother's BMI squared	-0.00002*** (0.0000)
First birth	-0.0029*** (0.0006)
Black	-0.0069*** (0.0006)
Asian	-0.0064*** (0.0007)
Hispanic	-0.0040*** (0.0008)
Native American	0.0060*** (0.0018)
Mother's age 18-19	0.0008 (0.0025)
Mother's age 20-24	0.0022 (0.0023)
Mother's age 25-29	0.0034 (0.0024)
Mother's age 30-34	0.0057** (0.0027)
Mother's age 35-39	0.0082** (0.0052)
Mother's age 40+	0.0149*** (0 .0034)

*Where *,**,*** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors are reported in parentheses.*

Table 3.7 Cesur and Kelly (2010) Replication; NDF, 2003-2009

	Marginal Effect
Married	0.0011*** (0.0001)
High school drop out	-0.0018*** (0.0001)
High school	-0.0004*** (0.0001)
Some college	0.0002 (0.0001)
College degree	-0.0007*** (0.0001)
Parity 2	0.0018*** (0.0001)
Parity 3	0.0030*** (0.0001)
Parity 4	0.0043*** (0.0001)
Parity 5	0.006*** (0.0002)
Parity 6	0.008*** (0.0003)
Parity 7	0.011*** (0.0004)
Parity 8 plus	0.017*** (0.0005)
Male infant	0.0075*** (0.0000)
Mother's age	0.0008*** (0.0000)
Mother's age squared	-0.000007*** (0.0000)
Hispanic	-0.0020*** (0.0001)
Asian	-0.0060*** (0.0001)
Black	-0.0048*** (0.0001)
Native	0.0059*** (0.0002)
Underweight	-0.0060*** (0.0002)
Overweight	0.0090*** (0.0002)
Obese	0.0170*** (0.0003)
Morbidly Obese	0.0306*** (0.0004)

*Where *,**,*** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors are reported in parentheses.*

Table 3.8 Model Specification Changes, PRAMS, 2003-2009

	Census sub-region fixed effects added	State fixed effects added	Very high birth weight (>5,000g) as dependent variable	Somewhat high birth weight (>4,000g) as dependent variable	Large for gestational age as dependent variable	BMI categories added	Sample restricted to mothers aged 20-35	Number of dependents in household variable added
Married	0.0012 (0.0008)	0.0012 (0.0007)	-0.0004* (0.0002)	0.0087*** (0.0021)	0.011*** (0.0022)	0.001 (0.0008)	0.0015* (0.0009)	0.001 (0.0008)
High school drop out	-0.003* (0.0016)	-0.003* (0.0016)	-0.0004 (0.0003)	-0.0196*** (0.0046)	-0.0092* (0.0052)	-0.004*** (0.0014)	-0.0008 (0.0025)	-0.0032* (0.0016)
High school	-0.002 (0.0017)	-0.002 (0.0017)	-0.0006* (0.0003)	-0.0026 (0.0049)	0.0059 (0.0053)	-0.0026* (0.0015)	-0.0003 (0.0024)	-0.0021 (0.0017)
Some college	-0.0024 (0.0017)	-0.0023 (0.0017)	-0.0006* (0.0003)	0.0026 (0.0052)	0.0124** (0.0056)	-0.003** (0.0015)	-0.0006 (0.0025)	-0.0024 (0.0017)
College degree	-0.0013 (0.0018)	-0.0012 (0.0018)	-0.0006* (0.0003)	0.0115** (0.0054)	0.0191*** (0.0058)	-0.0023 (0.0016)	0.0002 (0.0026)	-0.0014 (0.0018)
Household income $10,000-$20,000	0.0005 (0.0011)	0.0005 (0.0011)	0.0001 (0.0002)	0.0083*** (0.0031)	0.0071** (0.0032)	0.0009 (0.0011)	-0.0002 (0.0012)	0.0005 (0.0011)
Household income $20,000-$30,000	0.0028* (0.0015)	0.0028* (0.0015)	0.0007 (0.0005)	0.013*** (0.0037)	0.0115*** (0.0039)	0.002* (0.0015)	0.0022 (0.0016)	0.0031** (0.0015)
Household income $30,000-$40,000	0.0032** (0.0015)	0.0033** (0.0015)	0.0002 (0.0003)	0.0114*** (0.0037)	0.0126*** (0.0039)	0.0032** (0.0015)	0.0024 (0.0016)	0.0032** (0.0015)
Household income >$40,000	0.0021* (0.0012)	0.0021* (0.0012)	0.0001 (0.0003)	0.014*** (0.0031)	0.018*** (0.0032)	0.002* (0.0012)	0.0015 (0.0013)	0.0021* (0.0012)

*Where *,**,*** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors are reported in parentheses. Additional controls included but not reported.*

Table 3.9 Model Specification Changes, NDF, 2003-2009

	Very high birth weight (>5,000g) as dependent variable	Somewhat high birth weight (>4,000g) as dependent variable	Sample restricted to mothers aged 20-35
Married	-0.0002*** (0.0000)	0.0113*** (0.0001)	0.0009*** (0.0001)
High school drop out	-0.0004*** (0.0001)	-0.0098*** (0.0003)	-0.0017*** (0.0002)
High school	-0.0004*** (0.0000)	-0.0004 (0.0003)	-0.0006*** (0.0002)
Some college	-0.0004*** (0.0000)	0.0053*** (0.0004)	0.000007 (0.0002)
College degree	-0.0007*** (0.0000)	0.0054*** (0.0004)	-0.001*** (0.0001)

*Where *,**,*** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors are reported in parentheses. Additional controls included but not reported.*

Table 4.1 Marginal Effects of Obesity on High Birth Weight Risk; Probit, MAT, 2007-2011

	All Overweight		All Obese		Morbidly Obese		Obese		Overweight	
Obesity measure	0.0147***	(0.0007)	0.0173***	(0.0011)	0.0236***	(0.0017)	0.0119***	(0.0012)	0.0100***	(0.0008)
Maori	-0.0042***	(0.0009)	-0.0040***	(0.0009)	-0.0035***	(0.0010)	-0.0039***	(0.0009)	-0.0037***	(0.0010)
Pacific	0.0066***	(0.0016)	0.0059***	(0.0016)	0.0069***	(0.0016)	0.0024	(0.0016)	0.0010	(0.0017)
Asian	-0.0159***	(0.0009)	-0.0164***	(0.0009)	-0.0168***	(0.0009)	-0.0150***	(0.0009)	-0.0135***	(0.0009)
MELAA	-0.0134***	(0.0018)	-0.0131***	(0.0019)	-0.0131***	(0.0019)	-0.0112***	(0.0019)	-0.0101***	(0.0019)
Mother's age	0.0026***	(0.0005)	0.0024***	(0.0005)	0.0025***	(0.0005)	0.0019***	(0.0005)	0.0015***	(0.0005)
Mother's age squared	0.0000***	(0.0000)	0.0000***	(0.0000)	0.0000***	(0.0000)	0.0000***	(0.0000)	0.0000***	(0.0000)
Missing deprivation	-0.0020	(0.0216)	-0.0023	(0.0214)	-0.0019	(0.0217)	0.0022	(0.0238)	0.0106	(0.0305)
Deprivation 2	-0.0001	(0.0018)	-0.0001	(0.0018)	-0.0001	(0.0018)	-0.0010	(0.0017)	-0.0019	(0.0016)
Deprivation 3	-0.0024	(0.0017)	-0.0024	(0.0017)	-0.0024	(0.0017)	-0.0029*	(0.0016)	-0.0028*	(0.0015)
Deprivation 4	-0.0018	(0.0016)	-0.0017	(0.0016)	-0.0015	(0.0017)	-0.0012	(0.0016)	-0.0016	(0.0016)
Deprivation 5	-0.0017	(0.0016)	-0.0017	(0.0017)	-0.0015	(0.0017)	-0.0012	(0.0016)	-0.0018	(0.0016)
Deprivation 6	-0.0010	(0.0016)	-0.0009	(0.0017)	-0.0007	(0.0017)	-0.0009	(0.0016)	-0.0011	(0.0016)
Deprivation 7	-0.0013	(0.0017)	-0.0013	(0.0017)	-0.0009	(0.0017)	-0.0014	(0.0016)	-0.0022	(0.0016)
Deprivation 8	0.0009	(0.0017)	0.0008	(0.0017)	0.0012	(0.0017)	0.0000	(0.0016)	-0.0001	(0.0016)
Deprivation 9	-0.0006	(0.0017)	-0.0006	(0.0017)	-0.0002	(0.0017)	-0.0005	(0.0016)	-0.0016	(0.0016)
Deprivation 10	0.0002	(0.0018)	0.0001	(0.0018)	0.0007	(0.0019)	0.0003	(0.0018)	-0.0008	(0.0018)
Spring birth	0.0003	(0.0010)	0.0002	(0.0010)	0.0002	(0.0010)	0.0005	(0.0010)	0.0006	(0.0010)
Winter birth	-0.0003	(0.0010)	-0.0004	(0.0010)	-0.0003	(0.0010)	-0.0001	(0.0010)	0.0001	(0.0010)
Autumn birth	-0.0001	(0.0010)	-0.0001	(0.0010)	-0.0001	(0.0010)	-0.0001	(0.0010)	0.0002	(0.0010)
Parity 2	0.0083***	(0.0009)	0.0085***	(0.0010)	0.0087***	(0.0010)	0.0075***	(0.0009)	0.0063***	(0.0009)
Parity 3	0.0121***	(0.0014)	0.0120***	(0.0014)	0.0125***	(0.0014)	0.0115***	(0.0014)	0.0100***	(0.0014)
Parity 4	0.0165***	(0.0023)	0.0164***	(0.0023)	0.0171***	(0.0024)	0.0147***	(0.0024)	0.0146***	(0.0026)
Parity 5	0.0262***	(0.0040)	0.0253***	(0.0039)	0.0262***	(0.0040)	0.0199***	(0.0041)	0.0161***	(0.0044)
Parity 6	0.0255***	(0.0057)	0.0243***	(0.0057)	0.0249***	(0.0058)	0.0233***	(0.0065)	0.0226***	(0.0074)

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	All Overweight		All Obese		Morbidly Obese		Obese		Overweight	
Parity 7	0.0315***	(0.0090)	0.0300***	(0.0088)	0.0311***	(0.0090)	0.0198**	(0.0092)	0.0080	(0.0091)
Parity 8+	0.0330***	(0.0095)	0.0319***	(0.0094)	0.0318***	(0.0094)	0.0301***	(0.0107)	0.0321***	(0.0122)
Missing parity	0.0117	(0.0147)	0.0122	(0.0152)	0.0123	(0.0151)	0.0083	(0.0142)	0.0158	(0.0166)
Male	0.0150***	(0.0007)	0.0151***	(0.0007)	0.0152***	(0.0007)	0.0149***	(0.0007)	0.0139***	(0.0007)
Smoke	-0.0145***	(0.0008)	-0.0146***	(0.0008)	-0.0146***	(0.0008)	-0.0136***	(0.0008)	-0.0120***	(0.0008)
Registration trimester 2	0.0010	(0.0012)	0.0010	(0.0012)	0.0009	(0.0012)	0.0000	(0.0012)	0.0001	(0.0012)
Registration days	0.0000**	(0.0000)	0.0000**	(0.0000)	0.0000**	(0.0000)	0.0000*	(0.0000)	0.0000*	(0.0000)
Income	0.0000	(0.0000)	0.0000	(0.0000)	0.0000	(0.0000)	0.0000	(0.0000)	0.0000	(0.0000)
Semi-rural	-0.0022*	(0.0013)	-0.0020	(0.0013)	-0.0020	(0.0013)	-0.0014	(0.0013)	-0.0016	(0.0013)
Rural	0.0031***	(0.0010)	0.0034***	(0.0010)	0.0037***	(0.0011)	0.0037***	(0.0011)	0.0027**	(0.0011)
Remote rural	0.0009	(0.0018)	0.0012	(0.0018)	0.0015	(0.0018)	0.0016	(0.0018)	0.0004	(0.0018)
Unknown rurality	-0.0020	(0.0013)	-0.0020	(0.0013)	-0.0019	(0.0013)	-0.0009	(0.0013)	-0.0019	(0.0013)

*Where *,**,*** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported in parentheses. Year and DHB controls included but not reported.*

Table 4.2 Marginal Effects of Obesity on High Birth Weight Risk and Validity Tests; Probit and Bivariate Probit, MAT, 2007-2011

	Probit	Bivariate Probit with fast food and mother's birth month instruments	Instrument Validity Tests	Bivariate Probit with rurality, fast food, and mother's birth month instruments	Instrument Validity Tests	Bivariate Probit with mother's birth month instruments	Instrument Validity Tests
25≤BMI<30 (Overweight)	0.0100*** (0.0008)	0.0040 (0.0030)	F=5.87 P=0.884 ✓	0.0058** (0.0025)	F=7.37 P=0.828 ✓	-0.0028 (0.0054)	F=0.77 P=0.723 ✓
30≤BMI<35 (Obese)	0.0119*** (0.0012)	-0.0022 (0.0020)	F=4.31 P=0.375 ✓	-0.0003 (0.0016)	F=4.59 P=0.359 ✓	-0.0119*** (0.0025)	F=0.89 P=0.652 ✓
BMI≥35 (Morbidly Obese)	0.0236*** (0.0017)	0.0026** (0.0010)	F=4.87 P=0.241 ✓	0.0031*** (0.0005)	F=5.00 P=0.072 ✓	0.0026** (0.0011)	F=1.56 P=0.411 ✓
BMI≥30 (Obese or Morbidly Obese)	0.0173*** (0.0011)	0.0043 (0.0030)	F=7.34 P=0.247 ✓	0.0050** (0.0024)	F=7.62 P=0.122 ✓	0.0023 (0.0141)	F=1.38 P=0.413 ✓
BMI≥25 (Overweight, Obese, or Morbidly Obese)	0.0147*** (0.0007)	0.0036 (0.0121)	F=10.77 ✓ P=0.348 ✓	0.0122** (0.0055)	F=12.87 ✓ P=0.230 ✓	-0.0157 (0.0099)	F=0.94 P=0.410 ✓

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors are reported in parentheses. Additional controls included but not reported.*

Instrument validity tests consist of the first stage F statistic and the p-value from the Hansen's J statistic. Passing values are indicated with a '✓'.

Table 4.3 Marginal Effects of Instruments on Obesity; Bivariate Probit

First Stage, MAT, 2007-2011

	All Over-weight	Std Error	All Obese	Std Error	Morbidly Obese	Std. Error
Maori	0.1313***	0.0035	0.1000***	0.0031	0.0428***	0.0021
Pacific	0.2503***	0.0048	0.2565***	0.0054	0.1404***	0.0044
Asian	-0.1938***	0.0048	-0.1276***	0.0031	-0.0602***	0.0016
MELAA	0.0247**	0.0109	-0.0138	0.0089	-0.0121**	0.0055
Semirural	-0.0141**	0.0057	-0.0112**	0.0044	-0.0082***	0.0027
Rural	0.0065	0.0046	0.0008	0.0036	-0.0011	0.0023
Remote rural	0.0129*	0.0076	-0.0024	0.0059	-0.0062*	0.0036
Unknown rurality	-0.0028	0.0049	0.0020	0.0040	0.0024	0.0025
Mother's age	0.0193***	0.0018	0.0237***	0.0014	0.0145***	0.0009
Mother's age squared	-0.0003***	0.0000	-0.0004***	0.0000	-0.0002***	0.0000
Missing age	0.2565**	0.1233	0.5222***	0.1344		
Missing deprivation	0.1073	0.0788	0.0894	0.0734	0.0380	0.0516
Deprivation 2	0.0063	0.0065	0.0053	0.0058	0.0066	0.0041
Deprivation 3	0.0304***	0.0065	0.0298***	0.0060	0.0233***	0.0045
Deprivation 4	0.0429***	0.0061	0.0440***	0.0058	0.0241***	0.0042
Deprivation 5	0.0522***	0.0063	0.0428***	0.0060	0.0270***	0.0044
Deprivation 6	0.0609***	0.0060	0.0518***	0.0057	0.0312***	0.0043
Deprivation 7	0.0786***	0.0062	0.0745***	0.0060	0.0409***	0.0046
Deprivation 8	0.0837***	0.0059	0.0787***	0.0058	0.0463***	0.0045
Deprivation 9	0.0935***	0.0060	0.0859***	0.0059	0.0516***	0.0046
Deprivation 10	0.1378***	0.0063	0.1288***	0.0066	0.0712***	0.0052
Parity 2	0.0386***	0.0030	0.0324***	0.0026	0.0163***	0.0017
Parity 3	0.0536***	0.0040	0.0541***	0.0035	0.0264***	0.0024
Parity 4	0.0996***	0.0059	0.0856***	0.0054	0.0413***	0.0037
Parity 5	0.1106***	0.0089	0.1144***	0.0082	0.0558***	0.0057
Parity 6	0.1322***	0.0130	0.1476***	0.0121	0.0824***	0.0088
Parity 7	0.1298***	0.0191	0.1266***	0.0173	0.0655***	0.0121
Parity 8+	0.1002***	0.0200	0.1268***	0.0179	0.0778***	0.0130
Missing parity	0.0184	0.0384	0.0119	0.0334	-0.0123	0.0196
Smoke	0.0397***	0.0039	0.0267***	0.0031	0.0102***	0.0019
Registration days	0.0004***	0.0001	0.0002***	0.0001	0.0001**	0.0000
Registration trimester 2	-0.0085**	0.0042	-0.0079**	0.0033	-0.0020	0.0021
Income	0.0000	0.0000	0.0000	0.0000	0.0000**	0.0000
Mother's birth month - Feb	-0.0046	0.0064	0.0010	0.0051	-0.0046	0.0030

Continued…

	All Over-weight	Std Error	All Obese	Std Error	Morbidly Obese	Std. Error
Mother's birth month - Mar	-0.0023	0.0062	0.0025	0.0050	-0.0024	0.0030
Mother's birth month - Apr	-0.0070	0.0063	-0.0024	0.0050	-0.0033	0.0030
Mother's birth month - Jul	-0.0044	0.0062	-0.0008	0.0049	-0.0031	0.0030
Mother's birth month - Aug	-0.0016	0.0062	-0.0004	0.0049	-0.0025	0.0030
Mother's birth month - Sep	0.0040	0.0062	0.0017	0.0049	0.0004	0.0030
Mother's birth month - Oct	-0.0083	0.0061	-0.0072	0.0048	-0.0067**	0.0029
Mother's birth month - Nov	0.0028	0.0063	0.0008	0.0050	-0.0027	0.0030
Mother's birth month - Dec	0.0018	0.0062	0.0039	0.0050	0.0026	0.0031
Café/restaurants	-0.0028***	0.0005	-0.0015***	0.0004	-0.0014***	0.0003
Takeaways	-0.0009	0.0012	0.0003	0.0010	0.0001	0.0006
Catering	-0.0088***	0.0025	-0.0069***	0.0020	-0.0018	0.0013
Pubs	0.0031**	0.0015	0.0004	0.0012	0.0010	0.0007
Clubs	0.0019	0.0027	0.0018	0.0021	0.0022*	0.0013
Hell's pizza	-1.1180***	0.2501	-0.5555***	0.1967	-0.2312*	0.1234
Pizza Hut	0.2731	0.2260	0.2474	0.1747	-0.0185	0.1087
Burger Fuel	0.2351	0.3564	0.6767**	0.2839	0.4340**	0.1798
Burger Wisconsin	-0.7070	0.4733	-1.3557***	0.3717	-0.0737	0.2339
Wendys	-0.8436*	0.4556	-0.0294	0.3523	0.1873	0.2192
Subway	0.0072	0.0673	-0.0219	0.0512	0.0002	0.0312
KFC	0.5441***	0.1898	0.4644***	0.1499	0.4219***	0.0959
Mcdonald's	-0.3550**	0.1441	-0.1795	0.1158	-0.1611**	0.0749
Burger king	-0.2448	0.2344	-0.4674**	0.1854	-0.1163	0.1174

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported. Year and DHB controls included but not reported.*

Table 5.1 Marginal Effects of Iron on High Birth Weight; Probit, MAT, 2003-2011

	Iron quantity	Std. Error	Iron dummies	Std. Error
Trimester 1 Iron	-0.00000014	0	0.0007	0.0014
Trimester 2 Iron	0.00000004	0	0.0020**	0.0010
Trimester 3 Iron	0.00000078***	0	0.0048**	0.0023
Calcium	-0.0029	0.0021	-0.0026	0.0021
Iodine	0.0008	0.0017	0.0013	0.0017
Multivitamins	-0.0028	0.0024	-0.0024	0.0024
Vitamin b	-0.0020	0.0017	-0.0018	0.0018
Vitamin c	0.0025	0.0025	0.0040	0.0026
Vitamin d	-0.0128***	0.0025	-0.0127***	0.0025
Zinc	0.0013	0.0183	0.0007	0.0178
Maori	-0.0038***	0.0006	-0.0038***	0.0006
Pacific	0.0173***	0.0011	0.0173***	0.0011
Asian	-0.0175***	0.0006	-0.0174***	0.0006
MELAA	-0.0113***	0.0014	-0.0111***	0.0014
Semi rural	0.0004	0.0009	0.0004	0.0009
Rural	0.0030***	0.0007	0.0030***	0.0007
Remote rural	0.0014	0.0011	0.0013	0.0011
Unknown rurality	-0.0006	0.0010	-0.0005	0.0010
Mother’s age	0.0029***	0.0003	0.0028***	0.0003
Mother’s age squared	0.0000***	0.0000	0.0000***	0.0000
Missing deprivation	-0.0054	0.0048	-0.0054	0.0048
Deprivation 2	0.0019	0.0013	0.0020	0.0013
Deprivation 3	0.0013	0.0012	0.0014	0.0012
Deprivation 4	0.0011	0.0012	0.0011	0.0012
Deprivation 5	0.0006	0.0012	0.0007	0.0012
Deprivation 6	0.0015	0.0012	0.0016	0.0012
Deprivation 7	0.0012	0.0012	0.0013	0.0012
Deprivation 8	0.0025**	0.0012	0.0026**	0.0012
Deprivation 9	0.0011	0.0011	0.0012	0.0011
Deprivation 10	0.0015	0.0012	0.0016	0.0012
Spring birth	0.0012*	0.0007	0.0012*	0.0007
Winter birth	0.0002	0.0007	0.0001	0.0007
Autumn birth	0.0001	0.0006	0.0001	0.0006
Parity 2	0.0089***	0.0007	0.0090***	0.0007
Parity 3	0.0131***	0.0009	0.0132***	0.0009
Parity 4	0.0164***	0.0014	0.0165***	0.0014
Parity 5	0.0202***	0.0022	0.0204***	0.0022
Parity 6	0.0297***	0.0033	0.0300***	0.0033
Parity 7	0.0189***	0.0041	0.0191***	0.0042
Parity 8+	0.0282***	0.0048	0.0285***	0.0048
Missing parity	0.0100***	0.0014	0.0099***	0.0014
Registration trimester 2	-0.0011	0.0007	-0.0012*	0.0007

Continued…

	Iron quantity	Std. Error	Iron dummies	Std. Error
Registration trimester 3	-0.0052***	0.0011	-0.0053***	0.0011
Missing registration date	-0.0055***	0.0011	-0.0058***	0.0011
Male	0.0135***	0.0005	0.0135***	0.0005
Smoke	-0.0124***	0.0007	-0.0124***	0.0007

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported. Year and DHB controls included but not reported.*

Table 5.2 Marginal Effects of Iron on High Birth Weight; Bivariate Probit, MAT, 2003-2011

	Instrument set used			
	Free	Std. Error	Price & Availability	Std. Error
Iron trimester 1	0.0002	0.0003	0.0002	0.0003
Iron trimester 2	0.0004**	0.0002	0.0004**	0.0002
Iron trimester 3	-0.0007	0.0011	-0.0007	0.0011

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported. Marginal effects and standard errors scaled up by 1000 to give meaningful values. Additional controls included but not reported.*

Table 5.3 Effect of Iron Quantity on High Birth Weight; IVProbit, MAT, 2003-2011

	Instrument set used			
	Free	Std. Error	Price & Availability	Std. Error
Iron trimester 1	0.00006	0.00007	0.00011	0.00008
Iron trimester 2	-0.00001	0.00001	-0.00002	0.00002
Iron trimester 3	-0.00003	0.00003	-0.00005	0.00004

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Coefficient values and robust standard errors reported. Additional controls included but not reported.*

Table 5.4 Iron Validity Tests, MAT, 2003-2011

	Instrument set used			
	Free		Price & Availability	
	Dummies	Quantity	Dummies	Quantity
F-statistic	340.111✓	32.8091✓	182.439✓	22.1868✓
P-value	0.8652✓	0.5884✓	0.9818✓	0.8586✓

Instrument validity tests consist of the first stage F statistic and the p-value from the Hansen's J statistic. Passing values are indicated with a '✓'.

Table 5.5 Marginal Effects of Iron Quantity on High Birth Weight; Special Regressor Method, MAT, 2003-2011

	Instrument set used			
	Free	Std. Error	Price & Availability	Std. Error
Iron trimester 1	0.0010***	0.0003	-0.0002	0.0003
Iron trimester 2	-0.0005	0.0006	0.0000	0.0003
Iron trimester 3	0.0012	0.0018	-0.0018	0.0019

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported. Marginal effects and standard errors scaled up by 1000 to give meaningful values. Additional controls included but not reported.*

Table 5.6 Instrument Coefficients from the First Stage of Bivariate Probit Results, MAT, 2003-2011

Variable Indicator	Iron Brand	Co-efficient	Std. Error
Free	Brand 5	0.0050	0.0360
	Brand 6	0.1228	0.2026
	Brand 7	0.3357***	0.0776
	Brand 8	0.0158	0.0503
	Brand 9	-0.2082***	0.0504
	Brand 10	-0.2353***	0.0769
	Brand 11	0.1080**	0.0537
	Brand 12	-0.1103	0.1535
Availability	Brand 1	0.0393	0.2203
	Brand 2	-0.1790	0.3188
	Brand 5	-0.0262	0.0377
	Brand 6	0.1790	0.2164
	Brand 7	0.3708***	0.0784
	Brand 8	0.0385	0.0686
	Brand 9	-0.2101***	0.0743
	Brand 10	-0.2205***	0.0838
	Brand 11	0.1062**	0.0538
	Brand 12	0.0188	0.1897
Price*Availability	Brand 2	0.6479	5.3121
	Brand 4	6.3490***	1.9994
	Brand 8	0.0176	0.0449
	Brand 9	0.0606***	0.0197
	Brand 12	-0.0932	1.0523

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported.*

Table 6.1 Marginal Effects of High Birth Weight on Adverse Outcomes, NDF, 2003-2009

	Assisted Ventilation	Std. Error	NICU	Std. Error	Birth Injury	Std. Error	Apgar<7	Std. Error
LBW	4.8499***	0.0003	20.7015***	0.0006	0.0021	0.0000	3.4358***	0.0011
HBW	1.0753***	0.0004	4.0977***	0.0009	0.3351***	0.0002	2.7769***	0.0024
Male	0.2977***	0.0001	1.0030***	0.0001	0.0203***	0.0000	0.2123***	0.0003
Pre-term	2.7149***	0.0002	12.7906***	0.0005	0.0141***	0.0000	1.5008***	0.0007
Early-term	-0.0277***	0.0001	0.3987***	0.0002	-0.0055***	0.0000	-0.1358***	0.0003
Late-term	0.0655***	0.0001	0.2815***	0.0003	0.0051*	0.0000	0.0970***	0.0005
Post-term	-0.0026	0.0002	0.5285***	0.0004	0.0063*	0.0000	0.1042***	0.0006
HS dropout	0.0821*	0.0005	-		0.0064	0.0001	-0.0446***	0.0010
High school	0.0267	0.0004	-		0.0089	0.0001	-0.0279***	0.0009
Some college	0.0810*	0.0005	-		0.0072	0.0001	-0.0765	0.0010
College	-0.1363***	0.0004	-		-0.0153***	0.0001	-0.2197***	0.0010
Missing education	3.2443***	0.0004	-		-0.1205***	0.0001	0.5915***	0.0013
Parity 2	-0.4635***	0.0001	-1.1376***	0.0001	-0.0354***	0.0000	-0.4624***	0.0004
Parity 3	-0.4806***	0.0001	-1.0761***	0.0002	-0.0343***	0.0000	-0.4469***	0.0005
Parity 4	-0.4482***	0.0001	-0.9114***	0.0002	-0.0288***	0.0000	-0.3933***	0.0007
Parity 5	-0.4492***	0.0002	-0.7921***	0.0003	-0.0323***	0.0000	-0.3553***	0.0011
Parity 6	-0.4087***	0.0003	-0.7002***	0.0005	-0.0351***	0.0001	-0.2769***	0.0018
Parity 7	-0.3823***	0.0004	-0.6291***	0.0008	-0.0122	0.0001	-0.2957***	0.0027
Parity 8+	-0.4574***	0.0004	-0.6065***	0.0008	-0.0362***	0.0001	-0.2268***	0.0029
Missing Parity	-1.0047***	0.0004	-0.2720***	0.0010	-0.0681***	0.0001	-0.1431***	0.0040
Mother's age	-0.0005	0.0000	0.0272***	0.0001	-0.0001	0.0000	-0.0112	0.0001
Mother's age squared	0.0001	0.0000	0.0001	0.0000	0.0000	0.0000	0.0002***	0.0000
Hispanic	-0.6888***	0.0001	-0.7479***	0.0002	-0.0342***	0.0000	-0.2853***	0.0004
Asian	-0.7967***	0.0001	-0.6146***	0.0003	-0.0017	0.0000	-0.3543***	0.0006
Black	-0.0177*	0.0001	-0.1600***	0.0002	-0.0663***	0.0000	0.2571***	0.0005

Continued…

	Assisted Ventilation	Std. Error	NICU	Std. Error	Birth Injury	Std. Error	Apgar<7	Std. Error
Native	0.0620*	0.0004	-0.0772	0.0007	0.1281***	0.0001	0.0612***	0.0015
Underweight	-0.1261***	0.0002	-0.2250***	0.0003	-0.0178***	0.0001	-0.0540***	0.0013
Overweight	0.0467***	0.0001	0.2127***	0.0002	0.0104**	0.0001	0.0527***	0.0009
Obese	0.1443***	0.0002	0.4773***	0.0003	0.0358***	0.0001	0.1296***	0.0013
Morbidly obese	0.3074***	0.0002	0.8743***	0.0003	0.0493***	0.0001	0.1943***	0.0015
Missing weight	1.0009***	0.0020	2.1600***	0.0042	0.0909	0.0006	0.0783***	0.0008
Pre-natal care trimester 2	0.2272***	0.0001	0.2784***	0.0002	0.0094***	0.0000	0.0269***	0.0004
Pre-natal care trimester 3	0.3729***	0.0002	0.4979***	0.0003	0.0051	0.0001	-0.0126***	0.0008
No pre-natal care	0.6664***	0.0003	1.8383***	0.0006	-0.0069	0.0001	1.0337***	0.0025
Missing pre-natal Care	0.0813***	0.0002	0.9050***	0.0004	-0.0023	0.0001	0.4472***	0.0014
Weight gain	-0.0032***	0.0000	0.0010**	0.0000	0.0008***	0.0000	-0.0118***	0.0000
Smoke during Pregnancy	0.0018	0.0001	-0.1595***	0.0002	0.0089***	0.0000	0.0522***	0.0006
Vaginal	-1.4619***	0.0001	-3.2281***	0.0002	0.0678***	0.0000	-0.2067***	0.0005
Missing delivery	-1.5987***	0.0006	-1.8332***	0.0020	0.0429	0.0010	-0.0295***	0.0012
VBAC	0.7333***	0.0004	1.4738***	0.0008	0.0461***	0.0001	0.5974***	0.0022
Missing VBAC	1.3954***	0.0013	0.4336	0.0030	0.0950***	0.0002	0.5026***	0.0044
Attendant doctor	0.3558***	0.0001	0.8425***	0.0003	0.0072***	0.0000	0.0635***	0.0005
Attendant other	0.0283	0.0004	1.0635***	0.0009	-0.0140	0.0001	1.3363***	0.0036

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported. Marginal effects multiplied by 100 to give more easily observable results. Year controls included but not reported.*

Table 6.2 Marginal Effects of High Birth Weight on Adverse Outcomes; Continuous Measure, NDF, 2003-2009

	Assisted Ventilation	Std. Error	NICU	Std. Error	Birth Injury	Std. Error	Apgar <7	Std. Error
Birth Weight (KG)	-0.717***	0.000	-3.550***	0.000	0.191***	0.000	-0.821***	0.000
Birth Weight (KG) Squared	0.0001***	0.000	0.0003***	0.000	0.00001***	0.000	0.0001***	0.000

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported. Marginal effects multiplied by 100 to give more easily observable results. Additional controls included but not reported.*

Table 6.3 Marginal Effects of High Birth Weight on Adverse Outcomes; Categories, NDF, 2003-2009

Birth Weight	Assisted Ventilation	Std. Error	NICU	Std. Error	Birth Injury	Std. Error	Apgar<7	Std. Error
4000g - 4500g	0.0014***	0.0001	0.0021***	0.0002	0.0013***	0.0001	0.0010***	0.0001
4500g - 5000g	0.0063***	0.0004	0.0206***	0.0007	0.0037***	0.0002	0.0054***	0.0002
5000g+	0.0239***	0.0011	0.0945***	0.0023	0.0048***	0.0005	0.1216***	0.0016

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported. Marginal effects multiplied by 100 to give more easily observable results. Additional controls included but not reported.*

Table 6.4 Marginal Effects of High Birth Weight on Adverse Outcomes; Ethnicity Interactions, NDF, 2003-2009

	Assisted Ventilation	Std. Error	NICU	Std. Error	Birth Injury	Std. Error	Apgar<7	Std. Error
HBW	0.0068***	0.0004	0.0222***	0.0008	0.0029***	0.0002	-0.1490***	0.0026
Hispanic	-0.0052***	0.0001	-0.0050***	0.0001	-0.0004***	0.0000	0.0180***	0.0003
Asian	-0.0060***	0.0001	-0.0031***	0.0003	-0.0000	0.0000	0.0286***	0.0005
Black	0.0011***	0.0001	0.0001	0.0002	-0.0007***	0.0000	-0.0228***	0.0004
Native	0.0003	0.0004	-0.0014**	0.0007	0.0012***	0.0001	-0.0164***	0.0013
HBW*Hispanic	0.0048***	0.0007	0.0147***	0.0013	0.0006***	0.0002	-0.0721***	0.0059
HBW*Asian	0.0014	0.0016	0.0179***	0.0031	0.0001	0.0003	-0.1486***	0.0160
HBW*Black	0.0079***	0.0011	0.0473***	0.0028	0.0006**	0.0003	-0.6159***	0.0145
HBW*Native	0.0066**	0.0026	0.0113**	0.0046	0.0005	0.0004	-0.0711***	0.0174

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported. Marginal effects multiplied by 100 to give more easily observable results. Additional controls included but not reported.*

Table 6.5 Marginal Effects of High Birth Weight on Adverse Outcomes; Gestation Length Interactions, NDF, 2003-2009

	Assisted Ventilation	Std. Error	NICU	Std. Error	Birth Injury	Std. Error	Apgar<7	Std. Error
HBW	0.0067***	0.0006	0.0263***	0.0014	0.0031***	0.0003	-0.1137***	0.0035
Pre-term	0.0161***	0.0002	0.0939***	0.0005	0.0001	0.0000	-0.0978***	0.0006
Early-term	0.0005***	0.0001	0.0049***	0.0002	-0.0001**	0.0000	0.0028***	0.0003
Late-term	0.0005***	0.0001	0.0022***	0.0002	0.0001*	0.0000	-0.0100***	0.0005
Post-term	-0.0001	0.0001	0.0037***	0.0003	0.0001*	0.0000	-0.0098***	0.0006
HBW *Pre-term	0.0112***	0.0014	0.0219***	0.0022	-0.0005**	0.0001	-2.2825***	0.0291
HBW *Early-term	0.0020***	0.0007	0.0099***	0.0013	0.0002*	0.0001	0.0082**	0.0043
HBW *Late-term	-0.0005	0.0007	-0.0050***	0.0012	-0.00005	0.0001	0.0597***	0.0049
HBW *Post-term	0.0025**	0.0010	-0.0036**	0.0014	0.00000	0.0002	0.0409***	0.0062

*Where *,**, and *** indicate statistical significance at the 10%, 5%, and 1% level, respectively. Robust standard errors reported. Marginal effects multiplied by 100 to give more easily observable results. Additional controls included but not reported.*