Obesity and high birth weight

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Abstract

High birth weight (HBW) increases the risk of many negative health consequences. Maternal obesity has frequently been identified as a risk factor for HBW. However, the causal connection is uncertain due to obesity being likely endogenously related to HBW. To account for the endogeneity of obesity, I conduct instrumental variable analysis using the mother's birth month, fast-food restaurant density, and rurality as instruments. Biprobit results largely negate the effect of obesity on HBW risk except for morbidly obese women. A surprising finding from my results is that some fast food establishments appeared to decrease obesity risk.

Introduction and Background Literature

High birth weight (A.K.A. macrosomia) defined as birth weight greater than 4500g is an issue which has become increasingly apparent over recent years. Recent research suggests giving birth to babies over 4,500g carries significant risks to both the infant and the mother. An increased risk of shoulder dystocia has been identified by a number of studies to be associated with high birth weight ([1];[2];[3]). 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 [4]. High birth weight is also associated with an increased risk of infant mortality [5, 6]. Delivering a high birth weight baby also leads to maternal complications such as vaginal, perineal and cervical tears [7]. Long term consequences of high birth weight have been found including impaired cognitive performance in childhood [8] and higher prevalence of obesity later in life [9, 10]. Maternal obesity has frequently been identified as risk factors for high birth weight however, to my knowledge no paper has specifically addressed whether this relationship is causal or merely correlated.

Obesity is a big problem in the developed world and appears to be growing. The World Health Organisation (WHO) estimates that one in ten adults globally are obese and that obesity rates have more than doubled since 1980[11]. In New Zealand, the ministry of health estimates that 26.5% of adults are obese, rising from 17% in 1997[12, 13]. In the United States one in three adults are obese[14]. The rise in obesity has important implications for childbearing. Obesity reduces the ability to conceive [15, 16], increases the likelihood of a pregnant woman suffering from gestational diabetes[17] and, of particular interest to this paper, increases the likelihood of delivering a high birth weight baby. Due to the problems of obesity for childbearing, in New Zealand women with a body mass index [BMI] value of 32 or over are ineligible for publicly funded fertility treatment.

Considerable evidence of a positive relationship between obesity and high birth weight risk is present 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[18]. Srofenyoh & Seffah (2006) found that women who gave birth to high birth weight babies had significantly higher BMI values than those who gave birth to normal weight babies[4]. Berard et. Al. (1998) found women who gave birth to babies over 5000g were on average 9.2kg heavier than women who gave birth to babies between 4500 and 4999g[2].

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 utilize regression techniques such as instrumental variables which can tease out the causal factor from the relationship.

To test the relationship between body mass and high birth weight risk I use New Zealand birth data from the Ministry of Health between 2007 and 2011. This data set contains demographic and health status information about the baby and mother. From this data set I can identify the age, ethnicity, domicile of residence, birth date, parity, smoking status, and Body Mass Index (BMI) of the mother and the birth date and birth weight of the baby as well as the length of gestation. After eliminating non-residents, multiple births, stillbirths and missing BMI information I am left with 186,000 birth observations.

Probit regression analysis shows a strong positive relationship between increasing 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 point higher risk of high birth weight compared to the baseline risk. However, this has not adequately controlled for the endogeneity of obesity. To account for the endogeneity I conduct instrumental variable analysis using three different sets of instruments.

Biprobit results largely negate the effect of obesity on HBW risk except for morbidly obese women. The estimated effect drops by an order of magnitude and is only significance for morbidly obese women. The marginal effect 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 the estimated coefficient is highly robust to changes in the included set of instruments. My results provide compelling evidence that at the very least ignoring the endogeneity of obesity will lead to a much higher estimate of its causal effect on high birth weight risk than is justified. They also suggest that maternal weight may only be problematic for increasing high birth weight risk for morbidly obese women.

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 high birth weight. However, little attention has been paid to examining to what extent this link is causal. 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 regression analysis which does not include the independent factor will show 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 utilize regression techniques such as instrumental variables which can help tease out the causal factor from the relationship.

To use instrumental variables I need variables which are correlated with obesity risk but have no independent effect on high birth weight risk to use as instruments. I rely on three different instruments in my analysis. The first instrument I use is rurality of residence. Rurality is strongly correlated with obesity risk [19], [14] and at first glance seems to be uncorrelated with high birth weight risk, which indicates it should be a useful instrument. However, there could plausibly be factors that would correlate with both rurality and high birth weight risk which I have not controlled for in my regression analysis, which could invalidate rurality as an instrument. Sunshine exposure and consequently vitamin D absorption which can increase birth weight [20] and arguably 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 over recent times. Rosenheck's (2008) systematic review of 16 studies concludes there exists a significant relationship between fast food restaurant density and obesity[21]. It is generally agreed that fast food proximity lowers the notional cost of eating high caloric food and can therefore lead to higher obesity risk though the causality of the relationship is disputed [22]. It 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 wider region then food venue type and concentration may have an avenue of correlation with high birth weight risk outside of the effect on obesity that I am unable to control for and could invalidate its use as an instrument. 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) found increased sun exposure during pregnancy increased birth weight which could be caused by the increased vitamin D absorption[20]. Phillips and Young (2000) report that early exposure to cold conditions is associated with higher weight during adulthood in England[23]. Lokshin and Radyakin (2009) conclude that higher exposure to diseases in the monsoon season led to lower anthropometric measures during childhood[24]. It is therefore plausible that the mother's birth season can affect her obesity risk later in life but it seems entirely implausible that there would be any non-trivial influence of mother's birth month on high birth weight risk other than the effect it has on the mother's body mass, which indicates this instrument is potentially a highly reliable one. However, the link between birth month and obesity may prove to be unsubstantial.

Data

To test the relationship between body mass and high birth weight risk I use New Zealand birth data from the Ministry of Health between 2007 and 2011. This data set contains demographic and health status information about the baby and mother. From this data set I can identify the age, ethnicity, domicile 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. After eliminating non-residents, multiple births, stillbirths and missing BMI information I am left with 186,000 birth observations.

 A significant limitation of this data is that the measure of BMI is taken from when the mother first registers with a lead maternity carer. This is concerning because the later a women registers the higher her BMI would be expected to be due to normal pregnancy weight gain, therefore the measure is a less accurate approximation for pre-pregnancy body mass for 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 likely represent the most exceptional cases of 'slackness' and also because they are

most likely to have the most skewed measure of BMI due to much larger 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). I also create a variable to control for the number of days into her pregnancy the women registered, taken from the estimated date of her last menstrual period to the date of registration. Due to the discrete nature of the obesity measure I am using it is less likely to be problematic as weight gain in the first two trimesters is unlikely to push many women into higher categories than they otherwise would have been had they registered earlier. For a woman of average height (1.65m) there is a 13.6kg difference between categories, only 1.5kg is expected to be gained in the first trimester which should have very little effect on pushing women into higher weight categories, 55% of my sample registered in the first trimester. Weight gain in the second trimester is more rapid (about 0.5kg a week) and the women who register during the second trimester are more likely to have a skewed measure of their BMI, 38% of my sample registered in the second trimester. The same also applies for smoking status but this presents a much less concerning problem as it is not a variable of major importance.

To measure the fast food density in the mother's residence I use data supplied by Statistics New Zealand on the number of venues under the ANZSIC classifications of respectively; cafes and restaurants, takeaway food services, catering services, 'pubs, taverns, and bars', clubs (hospitality) by year and territorial local authority. I also create an index of the number of 'big chain' restaurants in each TLA. I do this by using information available on the websites for McDonalds, KFC, Subway, Pizza Hut, Hell's Pizza, Burger Fuel, Burger Wisconsin, and Wendy's of the number of venues within each TLA. As I am relying on their websites, I am unable to distinguish any time variation in the number of these restaurants open over the sample period.

Method

From my data set I create variables to be used in regression analysis. Using the birthweight data I create a dummy variables to indicate if the baby is high birth weight (>4500g) and if the baby is low birth weight (<2500g). I create dummy variables to indicate the ethnicity of the mother from the following categories; European, Maori, Pacific Islander, Asian, Middle Eastern/ Latin American/ African (MELAA), and other. I create a continuous variable to describe the mother's age and also a variable of the mother's age squared to account for non-linear effects of age on obesity and high birth weight risk. I create dummy variables to describe the number of previous births to the woman up to 8 births at which I include any women with higher parity counts in this category. 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.

For the instuments I create dummy variables to indicate whether the woman's residence is considered urban, semi-rural, rural, or remote rural, 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 the regression analysis without losing precision of the estimates.

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

New Zealand has many different ways of distinguishing area boundaries, I utilise three different levels of aggregation of area boundaries. The largest level of aggregation I use is the areas defined by the District Health Boards. This separates the nation into 21 different areas. I create dummy variables for each of the district health board regions that the mother resides in. The next level of aggregation is the Territorial Local Authority. These regions represent the areas covered by local councils which generally will cover just one city or town including the rural surrounds. I use these areas to create my variables for the food venues as 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 will miss the fact that many food choices will be made at places of work, study, and leisure which are highly likely to be contained within the same city or town and seldom outside it 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 merge to define areas but the ANZSIC food venues data was given with only the merged area which I have used. I also create a variable for the population of these areas by year and median income of the region from the latest available. There are 67 or 73 TLAs depending on the definitions used with a median population of roughly 30,000. Lastly, 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. I use this level of aggregation to create variables for the deprivation level of the meshblock that the woman resides in. The deprivation level is measured by the deprivation index which assigns a score from 1 to 10 based on measures of material and social deprivation, in areas such as income, employment, communication, transport, support, qualifications, owned home and living space for residents within the meshblock.

As birth month may does not represent the same birth season for women born in the northern hemisphere, and will 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. Unfortunately, I cannot identify the country of birth for women in the sample the best I can do is to drop all women who are not New Zealand residents from the sample. This should capture many of the women who were not born in New Zealand but not all.

Results

Simple probit estimation as shown in the first column on Table 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 high birth weight by 1, 1.19, and 2.36 percentage points respectively.

Table 1. Marginal effects of obesity on high birth weight risk after probit and bivariate probit

	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 *,**,*** indicates significance at the 10%, 5%, and 1% level respectively. Robust standard errors in parentheses.

Instrument validity tests consist of the first stage F statistic and the p-value from the Hansen's J statistic.

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, respectively. Male infants were shown to have a higher risk of high birth weight as expected. The results imply that a male infant has a 1.5 percentage point higher risk of high birth weight than female infants. Also as expected, increasing maternal age and parity increased the likelihood of high birth weight but at a diminishing rate.

Using all three sets of instruments in bivariate probit regression provides the highest explanatory power in the first stage results. 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 the exogeneity test is passed without question, however the explanatory power falls dramatically. Using just mother's birth month and fast food restaurant instruments 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 a woman is morbidly obese. Morbidly obese women have a 0.26 percentage point higher chance of having a high birth weight baby compared to all other groups, holding other factors constant. Despite the changes is validity test results against 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 will lead to highly biased estimates of its effect on high birth weight risk.

A curious finding from the first stage results was that the fast food restaurants density in a TLA did not have the expected effect on obesity measures. The majority of the fast food chains showed consistently negative coefficients in the first stage and particular chains such as Hell's Pizza, Burger Wisconsin, and McDonalds frequently showed a significant negative relationship with the propensity to be overweight, obese, and morbidly obese. KFC and Pizza Hut were 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 affect of living in urban areas as rurality variables are also included. The overall number of fast food establishments per person in a TLA was generally insignificant so it doesn't 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 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 the causal effect also substantially reduces the size of the estimated effect. Therefore, studies which look at the effect of obesity on high birth weight risk without treating the endogeneity of obesity run the risk of greatly over-estimating the effect.

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