Does Access to Fast Food Lead to Super-Sized Pregnant Women and Whopper Babies?

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Citation: Lhila, Aparna 2011 "Does Access to Fast Food Lead to Super-Sized Pregnant Mothers and Whopper Babies?" Economics and Human Biology, 9(4): 364-380.
DOI: 10.1016/j.ehb.2011.07.003

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Abstract

Rise in the availability of fast-food restaurants has been blamed, at least partly, for the increasing obesity in the U.S. The existing studies of obesity have focused primarily on children, adolescents, and adults, and this paper extends the literature by raising a little-studied question and using nationally-representative data to answer it. It examines the relationship between the supply of fast-food restaurants and weight gain of pregnant women and their newborns. I study prenatal weight gain because excessive weight gain has been linked to postpartum overweight/obesity and I study *both* tails of the birthweight distribution because the origin of obesity may be traced to the prenatal period and both tail outcomes have been associated with obesity later in life. I merge the 1998 and 2004 Natality Detail Files with the Area Resource File, and County Business Patterns, which provides data on the number of fast-food restaurants in the metropolitan area where the mother resides. The empirical model includes an extensive list of MSA characteristics and MSA fixed effects to control for factors that may be correlated with both health outcomes and restaurants' location decision. Results reveal that the fast-food and weight gain relationship is robust to the inclusion of these controls but these controls greatly mitigate the fast food –infant health relationship. Greater access to fast-food restaurants is positively related to mothers' probability of excessive weight gain but it does not share a statistically significant relationship with birthweight. These relationships hold in all the socioeconomic and demographic subgroups studied.

1. Introduction

When discussing the relationship between fast-food restaurants and health, the general consensus is that increasing the supply of restaurants is likely to be accompanied by higher rates of overweight and obesity (Chou, Grossman, Saffer 2004; Dunn 2008; Brennan and Carpenter 2009). In the U.S., the incidence of obesity has been likened to an epidemic and much attention has been devoted to understanding the determinants of obesity. In this paper I concentrate on maternal and infant health and examine the relationship between an important correlate of obesity, supply of fast-food restaurants, and obesity in a specific population – pregnant women and their newborn infants. I pay attention to prenatal weight gain and *both* tails of the birthweight distribution because the origin of obesity may be traced to the prenatal period (Lederman et al 2004; Henriksen 2008) and the tail outcomes have been associated with obesity later in life.

The relationship between the availability of fast food and maternal weight gain is as follows: when the supply of fast-food restaurants increases, the time cost of obtaining it decreases and consumer theory predicts that this will lead to an increase in the demand for fast food. This increase in the demand for fast food, which tends to be more calorically dense, is likely to increase the weight gain of mothers who substitute towards fast food. This effect will be exacerbated for mothers with self-control problems; as Cutler, Glaeser, and Shapiro (2003) explain, individuals who have a harder time controlling their food intake are likely to be more responsive to a decrease in the time-cost of food. Instead of a positive relationship, the association between the supply of fast food and maternal weight gain may be zero or negative. Greater access to restaurant food will not increase weight gain among mothers who substitute unhealthy home-cooked food for fast food and it may decrease weight gain if fast food meals restrict caloric intake by limiting one's portion size.

The theoretical underpinning for this paper is drawn from Grossman (1972) who argues that individuals combine market goods and time to invest in their own health. Children, especially unborn children, rely on their parents to make investment decisions on their behalf and during the prenatal period, mothers' investment in her own health may be viewed as an investment into the health of her children. This paper examines how a change in the price of fast food (vis-à-vis lower travel and wait time costs) affects mothers' consumption decision and ultimately her and her infant's health. I do not have data on mothers' consumption directly but an increase in body weight is assumed to arise from increased caloric intake.

To the best of my knowledge, the only other study of fast food availability and maternal health is Currie et al. (2010). The authors merged 15 years of Vital Statistics data from Michigan, New Jersey, and Texas to a database of fast-food restaurants using exact geographic locations of the restaurants. They found that having a fast-food restaurant within half mile of mother's residence increased average weight gain by 0.3% and raised her probability of excessive weight gain by 2.5%. At other distances the effect on mothers' weight gain was slightly different, but the authors' concluded that the effect of fast food supply on mothers is more or less linear in distance. The authors included zip code fixed effects and limited their analysis to mothers who had at least two children in that time period which allowed them to test their model with mother fixed effects.

To conduct this study, I merge three datasets—first, the census of births in 1998 and 2004 in the U.S. from the Natality Detail Files (NDF); second, the 1998 and 2004 County Business Patterns (CBP) which yields information on the number of fast-food restaurants in the metropolitan statistical area (MSA) of mothers' residence; and third, I merge measures of socioeconomic characteristics of MSAs from the Area Resource File (ARF). I use this merged data to estimate the relationship between the number of fast-food restaurants on different measures of maternal and infant health. The estimation equation includes MSA fixed effects to control for time-invariant factors that may be correlated with both restaurants' location decision and the demand for fast food in the MSA. The MSA fixed effects purges, for instance, the taste for fast food in the MSA which may be correlated with both the restaurants' decision to locate in that MSA and the demand for such food. Further, I control for a host of time-variant MSA characteristics that may be correlated with both the demand and supply of fast-food restaurants; including these MSA characteristics in the estimation would mitigate the potential omitted-variables bias in the estimates of the fast-food effect on maternal and infant health. The results of this analysis should be interpreted with caution as the identification strategy employed does not completely rule out the potential for bias that may arise for the following reasons: First, although the model includes a great many MSA characteristics, it is plausible that some important correlates of the demand and supply of fast food, e.g. percent women of childbearing age in the labor force, are left out thereby leading to an omitted-variables bias. MSA fixed effects would not purge any time-variant MSA characteristics that are correlated with both the restaurant's location decision and demand for fast food thus producing potentially biased results. Further, in the absence of a control for mother's preconception BMI, it is possible that my results are biased.

Admittedly there are potential problems with the identification strategy, nonetheless this an exercise worth undertaking as this paper contributes to the literature in the following ways: first, prior studies have focused almost exclusively on adult and child obesity and this paper extends the literature by studying obesity outcomes in a little-studied population, pregnant women and newborn infants. In fact, to the best of my knowledge there is no literature on the effect of fast-food restaurants on birthweight. Second, the analysis is based on the census of births from the entire nation which makes the results nationally representative and provides estimates that are statistically precise. Third, Currie et al. (2010) studies the effect of having versus not having a restaurant close to mothers' residence thereby explaining the effect of a policy that would ban fast-food restaurants in residential neighborhoods. The present study adds to this knowledge by examining the effect of the number of restaurants in the entire MSA thus shedding light on the effect of reducing the number of fast-food restaurants in an MSA. An MSA is defined as an area that shares close economic and social ties with a core urbanized area through commuting and employment[1]. Examining the relationship between MSA-level restaurant availability and her demand for fast food is relevant because it is plausible that a mother's consumption decision is shaped by what is available not only around her home, but in the areas she tends to visit – on her way to work and around her place of employment.

2. Literature Review

One's local environment is found to influence one's food choice (Morland, Wing, and Diez-Roux 2002). Social scientists have examined the association between fast food availability and childhood and adult health, but very little is known about the effect of fast food supply on the health of pregnant women and their offspring. The first part of this section discusses why this is

[1] http://www.census.gov/population/www/metroareas/metroarea.html

an interesting question and describes outcomes, particularly the risk of obesity, associated with both excessive prenatal weight gain and birthweight. Second, I present a summary of the economics literature pertaining to food and obesity. Finally, I delve into the medical literature to shed light on the clinical relationship between maternal nutrition and the health of mothers and infants.

2.1 Motivation

My reasons for focusing on fast food restaurants are twofold: First, policymakers are expending considerable effort to fight the upward trend in obesity through policies targeting restaurants, especially fast-food restaurants. Examples include mandatory calorie labeling (Farley et al. 2009; Elbel et al. 2009), taxing fast food (Cawley and Liu 2008; Powell and Chaloupka 2009), limiting fast-food advertising to children (Chou, Rashad, and Grossman 2008), and outright bans on the construction of new fast-food restaurants (Sturm and Cohen 2009). These policies are based on the understanding that discouraging unhealthy eating will stem the growth in obesity. However, if the seeds of obesity are sown in the prenatal period, this paper can help explain whether policies that target adult and child obesity by limiting the supply of fast-food will also have the power to improve mothers' prenatal weight gain and the health of their infants at birth.

The second reason for focusing on fast food is the finding that the development of flavor preferences and appetite in the offspring may be influenced by mothers' food choices while pregnant. Flavors consumed by the mother are transmitted to the fetus through the amniotic fluid which programs the taste for food later in life (reviewed in Lederman et al. 2004). For instance, infants exposed to carrots in-utero consumed significantly more carrot-flavored cereal (Mennella et al 2001; Hepper 1995; Schaal, Marlier, and Soussignan 2000). Experiments using rats show that individuals who were fed low protein/high carbohydrate diets or "junk food chow" in-utero demonstrated a greater preference for high-fat and junk food later (Bellinger, Lilley, and Langley-Evans, 2004; Langley-Evans, Bellinger, and McMullen, 2005; Bayol, Farrington, and Stickland, 2007). If these results are true for humans as well, then policies that effectively reduce the consumption of fast food in the prenatal period may also be effective in reducing the taste and hence demand for fast food in subsequent generations.

Excessive prenatal weight gain has been linked to adverse health outcomes, both for the mother and the baby. In a review of the literature, Derbyshire (2008) found that women may become overweight or obese after giving birth; those with weight gain in excess of 34.5 pounds were more likely to retain the higher level of weight 15 years after birth. Excessive weight gain is associated with a higher likelihood of heart disease in the offspring (Barclay 2010) and an increase in the risk of breast cancer in mother rats (de Assis et al 2006). In terms of infant health, excessive weight gain has also been linked to lower Apgar scores[2] (Derbyshire 2008) and a greater probability of high birthweight (HBW) and obesity among offspring (Schack-Nielsen et al 2009). In turn, being born in the upper tail of the birthweight distribution has been associated with several health complications for mothers and infants. For mothers, giving birth to a large infant is associated with prolonged labor, abnormal hemorrhage, and caesarean-section delivery. For infants, in the short term HBW is associated with an increased risk of fetal hypoxia and NICU use; in the long term, large newborns are at a greater risk for diabetes, overweight, and asthma (Henriksen 2008). According to Schack- Nielsen et al (2004), HBW raises the risk of obesity in adulthood because it alters the hormone balance, increases adiposity, and decreases sensitivity to appetite signals in infants. Being born on the other end of the birthweight spectrum, i.e. small-for-gestational age (SGA), is also associated with obesity later in life (Muhlhausler et al 2008). This is because individuals born small experience catch-up growth in childhood which is associated with a higher probability of obesity in adulthood.

In the social science literature, low birthweight (LBW) has been linked to long-term health and economic wellbeing of the child, including poorer health status, lower educational attainment and more limited labor market outcomes (Behrman, Rosenzweig, and Taubman 1994, Corman 1995, Currie and Hyson 1999, Osmani and Sen 2003, Behrman and Rosenzweig 2004). Furthermore, there is increasing evidence that both LBW and HBW are negatively associated with learning and cognition (Sorenson et al 1997; Kirkegaard et al 2006; Cesur and Kelly 2010).

2.2 Food and obesity: summary of the economics literature

[2] Apgar scores are assigned one and five minutes after birth to describe the prenatal and perinatal experiences of the infant. The scores, which range from 0 to 10, rate infant health by assigning 0-2 points to each of five criteria – heart rate, respiration, muscle tone, reflex and color.

A vast body of literature has examined the causes for the rise in obesity in the U.S. Approximately 40% of the rise in obesity in the last two decades of the twentieth century has been attributed to improved agricultural technology which has led to declining food prices and hence raised food consumption (Lakdawalla and Philipson 2002; Philipson and Posner 2003). The changes in food production technology have lowered the cost of producing high-calorie food at home which in turn has led to the rise in obesity (Cutler, Glaeser, and Shapiro 2003). Another form of technological advancement, i.e., greater efficiency in the distribution technology of food such as those employed by big-box retailers like Walmart Supercenter have also served to lower food prices; this too has been found to raise BMI and obesity rates in the U.S. (Courtmanche and Carden 2010).

In addition to technology, a factor associated with obesity is the cost of preparing food at home versus the cost of eating out. Chou, Grossman, and Saffer (2004) conclude that the rise in the number of fast-food restaurants, decline in fast food prices, and to a lesser extent the rise in the number of full-service restaurants explains the rise in adult obesity between 1984 and 1999. Increasing the supply of supermarkets, reducing restaurant supply, raising fast-food prices, and lowering grocery prices have been linked to lower child BMI (Powell and Bao 2009) and healthier food choices among adults (Beydoun, Powell, and Wang 2008). On the other hand, Anderson and Matsa (2009) used the presence of interstate highways as an instrument for the supply of restaurants and concluded that reducing the number of restaurants is unlikely to substantially reduce obesity. Currie et al. (2010) also attempted to estimate the causal effect of the presence of a fast-food restaurant by including zip code and mother fixed effects and found that the presence of a fast-food restaurant within 0.5 mile of mother's residence is likely to increase weight gain by 0.3% and raise the probability of excessive weight gain by 2.5%, on average.

2.3 Relationship between maternal nutrition and maternal and infant health

This sub section discusses how nutritional intake, particularly fast food is related to mothers' prenatal weight gain and infants' birthweight. The findings from the medical literature suggest that following the adage "eating for two" is likely to lead to excessive weight gain during pregnancy. According to the Department of Health (1991), pregnant women require an additional 200 calories per day to maintain a healthy weight gain, which is equivalent to two slices of toast or a bowl of cereal (Holland et al 2000). It is not surprising then that women who reported eating “a lot more food” during pregnancy were twice as likely to gain too much weight as women who reported eating “a little more food” (Olson and Strawderman 2003). Uusitalo et al (2009) conducted a principal components study of the dietary composition of Finnish mothers and found that a diet that followed a “fast food pattern” was positively associated with the rate of weight gain during pregnancy.

While the relationship between nutrition and maternal weight gain is somewhat obvious, the relationship between nutrition and birthweight, especially in a developed country context is less established and more complicated. Low caloric intake or weight gain has been associated with intrauterine growth restriction (Kramer 1987), and the Institute of Medicine (1990) recommendations for weight gain are based on the belief that increasing weight gain has the potential to lower LBW rates in the U.S. However, this association between caloric intake and birthweight is called into question by the finding that during the Dutch famine exposure to extreme nutritional restriction lowered birthweight by a modest 400-600 grams (Artal, Lockwood, and Brown 2010). However, the famine effect varied by the trimester of exposure; first-trimester exposure had no effect on birthweight, exposure in the second trimester lowered birthweight slightly, and last-trimester exposure reduced birthweight by 10% (Stein et al 2004; Painter, Roseboom, and Bleker 2005; Kind, Moore, and Davies 2006). Further, the relationship between maternal nutrition and birthweight depends on mothers’ preconception weight and the nutritional makeup of her diet. Based on reviews of the literature, Kind, Moore, and Davies (2006) concluded that the maternal nutrition – birthweight relationship is important only for malnourished or underweight mothers, and Henriksen (2008) concluded that limiting weight gain can reduce the risk of macrosomia (birthweight > 4,000 g), particularly among obese mothers. In addition to quantity, the quality of mothers’ diet is related to infant health. The fraction of energy derived from protein is positively related to birthweight whereas high carbohydrate intake and diet consisting of saturated fats are associated with infant thinness (Moore et al 2004) and lower birthweight (Langley-Evans 1996).

3. Data

3.1 Maternal and infant health

Data on mothers' prenatal weight gain and infant health outcomes are obtained from the 1998 and 2004 Natality Detail Files (NDF), which contain the universe of live births in the United States in those years. These files are a compilation of birth certificates which provide information on birth outcomes, mothers' demographic characteristics, prenatal care use, medical risk factors, and congenital abnormalities. The advantage of using NDF is its large sample size, approximately 4 million per year; this allows me to estimate the restaurant effect on health with a great deal of statistical precision and enables analyses for a variety of subsamples (by mother's age, race, marital status and education). The analysis is restricted to singleton births because the prenatal decisions such as diet and weight gain of mothers carrying twins are likely to be markedly different. Approximately 300 MSAs with population greater than 100,000 are identified; dropping observations in MSAs with population less than 100,000, those in non-MSA areas, and restricting the sample to MSAs that appear in both 1998 and 2004 causes a loss of 23.9% of the birth records. I limit the analytical sample to births in MSAs that appear in both years because the empirical model includes MSA fixed effects. Of this sample, approximately 20% of the observations are dropped because California does not require reporting of prenatal weight gain. The sample is further reduced because of the following missing variables: previous macrosomic and LBW births are missing for 11% of the observations; in 1998, California, Indiana, South Dakota and New York (excluding New York City) did not collect information on prenatal tobacco use; in 2004, seven states (Idaho, Kentucky, New York State excluding New York City, Pennsylvania, South Carolina, Tennessee, and Washington) collected tobacco use information in a way that was not directly comparable to the information collected in 1998[3]. Applying these restrictions and dropping observations with missing values leads to a sample of 3,922,957 observations for this analysis.

[3] Since tobacco use and previous large or small infants are important controls in the model I compared the samples with and without missing values for these controls to check if dropping observations with missing data is likely to bias the analytical sample. Results (not reported) revealed that the differences in means are statistically significant although remarkably small in magnitude. The statistical significance is not surprising given the large sample sizes, thus based on the negligible differences in the magnitude of the means (generally in the third decimal place) I concluded that the two samples are similar in content. As an additional test, I estimated the empirical model with and without these controls and found that the conclusions drawn from the two sets of results (not reported) would be very similar; but because these variables theoretically belong in the empirical model, I opted to report the results from the model that includes these controls.

3.2 Supply of restaurants

Restaurant supply data were obtained from the 1998 and 2004 County Business Patterns (CBP) which is an annual county-level data on the number of firms for each industry code. I use the 6-digit North American Industry Classification System (NAICS) code to identify fast-food restaurants (722211), the primary variable of interest, full-service restaurants (722110), and grocery stores (445110) which are important controls in the empirical model. Strictly speaking, the code that I use to categorize fast food also includes carry-out pizza parlors, restaurants and delicatessens, and drive-in restaurants. This is the closest definition of "fast food" available in CBP which is arguably one of the most comprehensive and reliable sources of establishment data in the U.S. Since these restaurants are likely to be similar to fast-food restaurants in terms of convenience, price, calorie content, and palatability, it may not be too problematic for the purposes of this analysis. Further, since there may be regional variation in the usage of these types of restaurants, omitting non-chain fast-food restaurants may in fact bias the results systematically by region. The downside of using this variable is that this definition of fast-food includes establishments like Subway and the so called "fast casual" restaurants like Panera Bread, Cosi, and Quiznos, where the food is likely to be less calorically dense, and does not distinguish restaurants that also offer healthier options. This level of detail is not available in the data and thus I am unable to test if excluding these establishments from my fast-food variable will have a significant effect on the point estimates.

I sum the county-level establishment data to the MSA level to obtain the counts of fast-food restaurants, full-service restaurants, and grocery stores in the MSA of mothers' residence. I divide the supply of restaurants and grocery stores by population per thousand because doing so more accurately captures the travel and wait times at these establishments. Although each of these variables has been adjusted for population size, I simply refer to these variables as numbers of fast-food restaurants, full-service restaurants, and grocery stores in the remainder of the text.

The choice of 1998 and 2004 were driven by data availability. I use 1998 NDF and CBP data because the industry codes in the CBP changed from the 4-digit Standard Industrial Classification system to the 6-digit NAICS[4] code in 1998. The classification codes changed

[4] http://www.census.gov/eos/www/naics/ Web content accessed on December 23, 2009

enough that the CBP data pre and post 1998 were not comparable. I chose 2004 NDF and CBP because it is the last year for which sub-state geographic identifiers are available in the NDF.

3.3 Variable definitions

Institute of Medicine (1990) lays out guidelines for medically-advisable weight gain during pregnancy, which depends upon mothers' BMI at the time of conception. The guidelines are as follows: for underweight women, 28-40 pounds, for normal weight women, 25-35 pounds, and for overweight/obese women, 15-25 pounds[5]. While NDF contains information on weight gain (in pounds), it does not collect information on mothers' height or weight at any point in time. In the absence of preconception weight information, I consider excessive weight gain as weight gain in excess of 40 pounds, which would be excessive for all women, irrespective of pre-conception weight. The probability of c-section birth is not a measure of infant or maternal health, however I consider this medical procedure an additional dependent variable as it is correlated with excessive weight gain and giving birth to a heavy infant. Measures of infant health include a continuous measure of birthweight (in grams) and five other measures to distinguish between light and heavy infants which allow me to test if results are sensitive to alternate definitions. I use standard clinical cutoffs to define low birthweight (LBW, birthweight < 2500 g), macrosomia (birthweight > 4,000 g), and high birthweight (HBW, birthweight > 4,500 g). Further, since gestation length is likely correlated with birthweight, I define two additional infant health measures that account for gestation: birthweight less than the tenth percentile for gestational age is used to identify infants who experienced fetal growth restriction, termed small-for-gestation-age (SGA), and birthweight greater than the ninetieth percentile for gestation age is used to identify large-for-gestational age infants (LGA; Lu and Halfon 2003). Finally, five-minute Apgar score is included as another measure of infant health as it has been linked to excessive prenatal weight gain.

3.4 MSA characteristics

In order to control for MSA characteristics that may be correlated with both restaurants' location decision and demand for fast food, I include several MSA-level controls in the model. These

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[5] The IOM guidelines were updated in 2009 which categorized obese women separately and recommended that obese women gain 11-20 pounds. http://www.iom.edu/ Web content accessed on December 23, 2009

variables are drawn from the Area Resource File (ARF) which provides county-level information on 1998 and 2004 per-capita income, fraction of the population that is teen aged, fraction of households that are headed by a single parent, fraction of the population 25+ with a college degree or more, fraction of the population that is Black, and population per square mile[6]. I use these county-level variables to construct MSA-level measures of socioeconomic characteristics and population density in the MSA of mothers' residence. The sample women reside in MSAs that, on average, have land area of 3,776 square miles and population of 2.5 million.

Table 1 contains the means and standard deviations of the variables of interest for the two sample years. It shows that in both samples the mean birthweight is approximately 3,300 g, the mean prenatal weight gain is 30 lbs, and percent mothers with weight gain in excess of 40 lbs is 18. It is interesting to note that the likelihood of excessive weight gain and average weight gain did not decline, as the IOM guidelines would recommend, when average BMI increased from 28.83 to 30.9 and obesity rate rose from 19.5 to 26.2% between the two years[7]. Between1998 and 2004, percent macrosomic births declined from 10.4 to 8.6 and percent LBW increased from 5.9 to 6.2. Further, the mean Apgar score remained consistent at 8.9 but the percent caesarean deliveries increased from 19.5 to 27.1 between the two years, an upward trend that is consistent with the national rise in c-section rates (Menacker and Hamilton 2010). During the study period, the average number of fast-food restaurants per thousand increased from 0.65 to 0.69, but the average number of full-service restaurants and grocery stores decreased from 0.68 to 0.66 and 0.23 to 0.21, respectively. The table also shows that there is less variation in the number of fast-food restaurants than in the supply of full-service restaurants and that, on average, there are approximately one-third as many grocery stores per thousand as fast-food restaurants in the MSAs where the sample mothers reside. A closer inspection (results not reported) of the number of fast-food restaurants per 1,000 variable reveals that MSA fixed effects and the time dummy account for 23.7% of the variation in this variable and that all of the 284 MSAs experienced a change in this variable; the minimum 1998-2004 difference in the number of fast-food restaurants/1,000 is -0.23, the 25th percentile -0.06, the median -0.1, the 75th percentile 0.03, and

[6] Percent single-parent households, percent 25+ with a college degree or more, and percent teenagers are not available at the county level for 1998 and 2004; in these cases I use 1990 and 2000 information instead.
[7] Author's calculations based on the sample of non-pregnant women aged 15-44 years from the 1998 and 2004 Behavioral Risk Factor Surveillance Systems.

the maximum 0.2. That there is within-MSAs variation is important as the fully-specified model relies on this variation to identify the effect of fast-food restaurants.

<Table 1(in Excel file) about here>

4. Empirical Model

In this section I describe the econometric model I estimate and discuss the identification assumption necessary to draw a causal interpretation of the estimated effect.

I estimate reduced-form health production functions for prenatal weight gain and infant's birthweight. In order to estimate the relationship between fast-food restaurant supply and maternal and infant health, one way to proceed would be to regress my measures of maternal and infant health on the supply of restaurants in the mothers' MSA. However, the problem with such a model is that the resultant coefficient on the number of fast-food restaurants, or the supply effect, may actually be picking up the demand effect. In other words, a positive coefficient on fast-food supply could be due to restaurants' decision to locate where the demand for fast food is likely to be high, which is likely to be MSAs with higher weight gain. Another potential problem is that mothers' taste for fast food (known to her but unobserved by the researcher) may be correlated with both her health and her decision to locate in a particular MSA. For instance, it is plausible that a mother's taste for fast food, which predisposes her towards excessive weight gain, is correlated with her decision to reside in an MSA with lots of fast-food restaurants.

I attempt to estimate the causal effect of the number of fast-food restaurants on maternal and infant health by controlling for factors, including MSA fixed effects, that may be correlated with both supply of and demand for fast food in the community. The estimation equation takes the following form:

(1) $H_{im} = \alpha R_m + \beta G_m + \delta X_i + \gamma Y_m + \nu T_i + \varepsilon_m + \eta_i$

In equation (1), H represents health of mother *i* in MSA *m* which is measured in three ways – weight gain (in pounds), an indicator of excessive weight gain, and an indicator of caesarean delivery. It also represents the health of infant *i* which is quantified in several ways – birthweight (in grams), and dummy variables for SGA, LBW, LGA, macrosomia, HBW, and Apgar score. R represents the number of fast-food restaurants/1000 people in the MSA. G represents both the number of grocery stores and full-service restaurants per-thousand people in the MSA, X represents mother and child characteristics; it includes child's sex and birth order and mother's age, education, race/ethnicity, marital status, prenatal tobacco use, number of prenatal care visits, and indicators for whether the mother had given birth to a macrosomic or LBW infant prior to the birth of the i^{th} child. Y is a vector of MSA characteristics that includes the ARF variables described in the data section. Finally, T is an indicator that the child was born in 1998, ε represents MSA fixed effects and η is the idiosyncratic error term.

I turn to the marketing literature to get insight into characteristics that fast-food restaurants may take into account when making site selections. I attempt to control for as many of these characteristics as I can obtain and this comprises the vector Y in equation (1). The determinants of fast-food restaurant site selection are area demographics, particularly income, accessibility, visibility, traffic patterns, and competition or proximity to other restaurants (Simons 1992; Hurvitz et al 2009). Block, Scribner, and DeSalvo (2004) found that fast-food restaurant location is positively correlated with household income and percent Black in the population. Although I am unable to obtain information on all these characteristics, the estimation model attempts to include as many of these variables that are likely to be correlated with both the demand and supply of fast food, i.e. demographics and population density.

In the set of mother and child characteristics, tobacco use is an important control because smoking may affect one's metabolism and calorie intake (Pinkowish 1999; Chou, Rashad, and Grossman, 2008). I control for having a previous macrosomic or LBW birth in order to control for mothers' genetic predisposition towards large or small infants. The number of full-service restaurants is included in the model because other studies (Chou, Grossman, and Saffer 2004; Currie et al 2009) have found it to exert an independent effect on health. The prevalence of single-parent households in the MSA is included in the model because single parents may have less time to devote to cooking at home and hence have a higher demand for prepared foods, including fast food; this may also be correlated with a restaurant's location decision. Controlling for the availability of full-service restaurants and grocery stores in mothers' MSA is important because it is a relevant substitute in an individual's set of food choices.

4.1 Identification

Identifying the causal effect of fast-food restaurant supply on maternal and infant health relies on including controls for factors correlated with both demand and supply of fast food. This paper includes many such MSA controls in the model, nevertheless the potential for omitted variables bias remains. The MSA characteristics, Y, control for the time-variant observable factors that may be correlated with both restaurants' location decision and health outcomes in the community. Further, since I use cross-sectional data from two time periods (1998 and 2004) I am also able to include MSA fixed effects which eliminates any unobservable time-invariant MSA characteristics that may be correlated with both restaurants' location decision and maternal and infant health outcomes. The MSA fixed effects purges, for instance, the taste for fast food in the MSA which may be correlated with both the restaurants' decision to locate in that MSA and the demand for such food. Further, since a randomly-selected mother's taste for fast food is likely to be highly correlated with the average taste for fast food in the community, MSA fixed effect accounts, at least partly, for her unobserved taste for fast food.

To summarize, the fixed-effects model exploits the within-MSA variation (over time) in the number of fast-food restaurants to explain how changes in fast-food restaurant availability is correlated with changes in maternal and infant health. The identification of the fast-food restaurant effect on maternal and infant body weight rests on the assumption that after controlling for MSA fixed effects and vectors X, G, and Y in equation (1) above, changes (over time) in other determinants of maternal weight gain and infant health are uncorrelated with changes in the supply of fast-food restaurants within MSAs.

4.2 Potential biases

Four potential sources of bias that the model does not account for as follows: first, MSA fixed effects only account for the unobserved correlates of both supply and demand that are constant over time. To the extent that unobserved factors that are correlated with within-MSA changes in both demand and supply of fast food vary over time, the point estimate will be biased. For example, it may be biased upward if the taste for fast food were positively correlated with changes in both the number of fast-food restaurants and the maternal and infant health outcomes in an MSA; on the other hand, the results may be downward biased if restaurants located in areas where demand for fast food were low in an effort to capture new customers. Having information on more than one pregnancy per mother would enable the inclusion of mother fixed effects which would have served to mitigate this bias by purging unobserved mother characteristics that may be correlated with both the demand and supply of fast-food, e.g. mother's taste for fast food. Second, potential bias may arise if there were a third variable, say public transportation or residential patterns, that attract new restaurants to the area and that change coincidentally with weight gain in that community; in such a case my estimate of α would be biased by the spurious correlation between fast-food supply and weight gain when fast-food supply may have no influence on weight gain. A third source of bias is due to potential measurement error. The number of fast-food restaurants in an MSA might not adequately measure individual mother's access to fast food because it likely masks the clustering or within-MSA variation in the location of fast-food restaurants. Further, since place of residence is an individual choice, one that may be correlated with demand for fast food, it is unlikely that any measurement error is classical in nature. Having information on the supply of fast-food restaurants in the vicinity of mother's residence (as in Currie et al. 2010) and place of work would have helped reduce the magnitude of this bias. Finally, the lack of mother's preconception BMI as a control variable may be another reason for potential bias. Women's preconception BMI tend to be negatively related to their prenatal weight gain thus the sign of the potential bias will depend on the direction of correlation between women's BMI and supply of fast-food restaurants; if that correlation is positive (negative) then the bias is likely to be negative (positive). In order to take some measure of preconception BMI I use information on mother's gestational diabetes status. Approximately 3% of pregnant women experience gestational diabetes, a condition that is highly correlated with overweight and obesity (Chu et al 2007). Using diabetic status as a proxy for overweight/obese is admittedly not a perfect solution since approximately 30% of reproductive-age women in the U.S. are obese[8]. It is nonetheless a worthwhile exercise as it allows me to identify women that are very likely to be overweight or obese. Thus, I include the diabetic indicator as a control in the model and hence attempt to mitigate the potential omitted variables bias.

4.3 Estimation

[8] http://www.cdc.gov/nchs/data/hus/hus08.pdf#075

Ordinary Least Squares is used to estimate the equations both when the outcome is continuous (e.g. weight gain) and when the outcome is dichotomous (e.g. weight gain greater than 40 lbs) because of the large sample size. As a check, probit estimation is used for some of the binary dependent variables and the results (not reported) are similar to those obtained from linear probability models presented here. In addition to testing this model for the full sample, I estimate the model separately by mothers' race/ethnicity, marital status, and educational attainment to gauge whether the change in demand for fast food in response to lower access cost is different for these socioeconomic and demographic subgroups. I also separately estimate the model for the subsample of mothers diagnosed with gestational diabetes in an effort to separate women who are likely to be overweight or obese; I thus test the relationship between fast food and maternal and infant health among overweight/obese women.

5. Results and Discussion

Table 2 contains the regression results for the full analytical sample and by mother's race. I present results from five different specifications: model 1 controls for the availability of grocery stores and full-service restaurants and includes the 1998 dummy; model 2 includes the model 1 controls in addition to controlling for mother and child characteristics; model 3 adds time-variant MSA characteristics to the model 2 controls; model 4 adds MSA fixed effects to model 2 controls, and model 5 is the fully-specified model that includes all controls and MSA fixed effects. Estimating model 3 is tantamount to exploiting the between-MSA variation while controlling for time-variant MSA characteristics, whereas models 4 and 5 exploit the within-MSA variation in fast-food restaurants to estimate the relationship between fast food availability and maternal and infant health. Under each model, columns 1, 2, 3, and 4 present the results for the full, non-Hispanic White, non-Hispanic Black, and Hispanic samples, respectively. I report the coefficients on the number of fast-food restaurants, full-service restaurants, and grocery stores for some of the maternal and infant health outcomes and suppress the other coefficients and outcomes (macrosomia, HBW, and LBW) for the sake of brevity.

<Table 2 (in Excel file) about here>

5.1 Overall and by race

Maternal health (weight gain and c-section) results: The model 1 point estimate in Table 2 shows that increasing the number of fast-food restaurants by one per 1000 residents is associated with an increase in prenatal weight gain of 2.8 lbs for all mothers, 0.5 lbs for White mothers and 3.7 lbs for Hispanic mothers, but a decrease in Black mothers' weight gain by 0.8 lbs. The coefficients for the full and Hispanic samples tend to decline somewhat as controls are successively added, and the fully-specified model suggests that an increase in the supply of fast food is associated with an increase of 1.8 lbs for all women and 1.9 lbs for Hispanic women. The White results become progressively larger in magnitude as controls are added to the model and show that a unit increase in the number of fast-food restaurants is associated with a 2.2 lbs increase in weight gain, whereas the Black results become statistically insignificant when all the controls are included. Comparing the R-square associated with each model (not reported) shows that the number of fast-food restaurants, full-service restaurants, grocery stores and time fixed effects explain 0.05% of the variation in weight gain and that the addition of child and mother characteristics increases the percent explained to 4.2%. Adding MSA characteristics and MSA fixed effects has a negligible effect on R-square.

In terms of substantive significance these results are large; relative to the mean weight gain in the sample (30 lbs), results show that overall weight gain increases by 6%, White mothers' weight gain increases by 7.3% and Hispanic mothers' weight gain increases by 6.3%, on average. Another way to assess the substantive significance of these results is to consider what this weight gain means in terms of caloric intake during gestation, an exercise shown in Currie et al (2010). Since an excess of 3,500 calories leads to a pound of weight gain, these results suggest that a one-unit increase in the number of fast-food restaurants raises, on average, all mothers' caloric intake by 6,300 calories, White mothers' by 7,700 calories and Hispanic mothers' caloric intake by 6,650 during the prenatal period. Thus, for a full-term pregnancy (40 weeks), an increase in the number of fast-food restaurants is associated with a rise of 157.5 calories/week for all mothers, 192.5 calories/week for White mothers and 166.3 calories/week for Hispanic mothers. Further, the number of full-service restaurants is associated with an increase in all and White mothers' weight gain but is not statistically significantly related to the weight gain of Black and Hispanic mothers; note however that the Black and Hispanic coefficients are similar in magnitude to the White coefficient[9] and the lack of statistical significance is likely driven by the

[9] T-tests (not reported) failed to reject that the Black and Hispanic coefficients are the same as the White coefficient.

relatively smaller Black and Hispanic sample sizes. The number of grocery stores is also positively related to weight gain in the full sample and each of the subsamples.

The second set of results reveal that increasing the number of fast-food restaurants by one per-thousand residents is associated with a statistically significant increase in the probability of excessive weight gain by 7.1, 7.0, 5.6, and 9.3 percentage points in the full, White, Black, and Hispanic samples, respectively. Relative to the sample mean (18.0), this translates to a 39.4% increase for all mothers, 38.8% increase among White mothers, 31.1% for Black mothers, and 51.7% for Hispanic mothers. Full-service restaurants and grocery stores do not tend to share a statistically significant relationship with the likelihood of excessive weight gain.

The number of fast-food restaurants and the probability of caesarean delivery are positively related in the full, Black, and Hispanic samples. The coefficients show that a unit increase in fast-food supply is associated with an increase in the probability of c-section birth by 3.7 percentage points in the full and Black samples and 13.1 percentage points in the Hispanic sample. Compared to the 1998 and 2004 combined sample mean, approximately 23%, these coefficients reveal a large relationship between fast-food supply and the probability of caesarean delivery. Full-service restaurants and grocery stores are mostly statistically insignificantly associated with the likelihood of c-section births.

Birthweight and Apgar score results: The remaining rows in the table present the relationship between fast food supply and infant health measured as birthweight (in grams), probabilities of LGA and SGA, and Apgar scores[10]. The coefficient on fast-food restaurant is negatively related to birthweight when we exploit the variation between MSAs, however, the association is positive when MSA fixed effects are included. According to the results of model 5, the supply of fast food in mother's MSA is statistically significantly associated with birthweight for the overall and Black samples, but the magnitude of these associations are relatively insignificant. For instance, an increase in the number of fast-food restaurants is related to a 25g increase in birthweight in the full sample, however, this is small considering the sample mean is approximately 3,300g and

[10] I suppress the results of the HBW, LBW, and macrosomia models for the sake of brevity and because these results closely follow the results of the birthweight, LGA, and SGA models discussed here.

the standard deviation greater than 500g. In the Hispanic and White samples, the statistical relationship between fast-food restaurants and birthweight is not significant at conventional levels. The number of full-service restaurants (grocery stores) is negatively (positively) related to birthweight, and the magnitude of these associations tend to be more meaningful compared to those between fast-food restaurants and birthweight.

The relationship between fast-food restaurants and tail outcomes, LGA and SGA, are statistically significant in model 1, but the inclusion of MSA fixed effects renders the coefficients insignificant in virtually all the samples. The relationship between full-service restaurants and birthweight and that between grocery stores and birthweight are robust to alternate definitions of light and heavy infants. Given the directions of the full-service and grocery store relationships with birthweight, it is not surprising that while full-service restaurants are associated with lower LGA and higher SGA rates, grocery stores are related positively to LGA and negatively to SGA rates. The same pattern of results (not reported) holds when infant health is measured as LBW, HBW, or macrosomia. These results support the argument that mothers' nutrition does not have an appreciable effect on infant health in developed countries, except perhaps for underweight and obese mothers. Whilst I am not able to accurately identify underweight and obese mothers, I attempt to test this idea with information on gestational diabetes.

The relationship between fast-food restaurants and Apgar scores are statistically insignificant at conventional levels. The Hispanic sample is an exception; it suggests that increasing the number of fast-food restaurants by one is associated with lowering Apgar score by 0.1 points. This is equivalent to a 1.1% lower Apgar score relative to the mean, 8.9. Similar to their relationships with birthweight, the number of full-service restaurants is associated with lower Apgar score, the number of grocery stores is positively related to Apgar score, and the size of these relationships tend to be more meaningful than that between fast-food restaurants and Apgar score[11].

[11] It is possible that some of these fast-food restaurants are closer to being full-service establishments and vice versa thereby muddying the individual definitions of fast-food and full-service restaurants. Thus, following Chou, Grossman, and Saffer (2004) and Rashad (2006) I estimate the empirical model with the total number of restaurants (fast food + full service) in an alternate specification. Not surprisingly, results (not reported) suggest that the coefficient on total number of restaurants is a combination of the individual effects of fast food and full-service restaurants. For instance, number of fast-food restaurants shares a positive and statistically significant relationship with the probability of excessive weight gain whereas the effect of full-service restaurants is statistically insignificant; when total number of restaurants is used, the coefficient on restaurants is positive and significant.

<Table 3 (in Excel file) about here>

5.2 By marital status and educational attainment

Table 3 shows the relationship between the number of fast-food restaurants and maternal and infant health by mother's marital status and education obtained from the fully specified model (model 5). These results show that the fast-food and weight gain relationship is more adverse for the samples of unmarried and less educated (HS or less) women compared respectively to the samples of married and more educated (more than HS) women. For example, whereas fast food supply is associated with a 9.2 percentage point higher probability of excessive weight gain in the unmarried sample, the corresponding association in the married sample is 6.2 percentage points. Similarly, fast food is associated with 2.6 lbs weight gain in the less educated sample whereas the magnitude of the same association is 1.2 in the more educated sample. A similar pattern of relationship emerges when the outcome is the likelihood of c-section birth; among unmarried (married) women, an increase in the number of fast-food restaurants is accompanied by a 7.2 (2.1) percentage point increase in the probability of caesarean delivery and while fast-food restaurants are positively (6.0 percentage points) and statistically significantly associated with c-section birth in the less educated group, the relationship is statistically insignificant for the more educated group of women. Consistent with Table 2 findings, the relationship between fast-food restaurants and birthweight is statistically significant in the unmarried, less educated, and more educated samples, however, the magnitude of the associations are not significant. Further, fast-food supply does not share a statistically significant relationship with the tail probabilities of LGA and SGA, irrespective of marital status and educational attainment.

<Table 4 (in Excel file) about here>

5.3 Gestational diabetes sample

Table 4 presents the regression results of the effect of fast-food restaurants on maternal and infant health outcomes for the gestational diabetic sample. The story is virtually identical to the one that emerged in Table 2. Results show that fast food availability shares a statistically insignificant relationship with weight gain, but a positive (12.4 percentage points) and statistically significant relationship with the probability of excessive weight gain; the magnitude

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Further, whilst fast food is not statistically significantly related to birthweight, full-service restaurants are negatively associated with the same; in this case, total number of restaurants is statistically significant and negatively related to birthweight.

of the latter is considerably larger in the diabetic sample relative to the overall sample (Table 2). Similar to the results of Table 2, model 5 results show that the fast-food effect on birthweight is statistically insignificant in this restricted sample.

6. Discussion and Conclusion

This paper attempts to identify the causal effect of the availability of fast-food restaurants on maternal and infant health. The maternal health results are fairly robust to the inclusion of time-variant MSA characteristics and MSA fixed effects but the inclusion of the same controls tempers the relationship between fast-food and infant health outcomes, rendering them statistically insignificant. Overall, I find that the number of fast-food restaurants in mothers' MSA is substantially positively related to maternal health outcomes – average weight gain, probability of gaining excessive weight during pregnancy, and probability of a caesarean delivery. However, supply of fast-food restaurants does not share an appreciable or statistically significant relationship with infant health outcomes – birthweight measured as a continuous variable and tail probabilities, SGA, LGA, HBW, LBW, and macrosomia, and Apgar scores.

The maternal health results hold for all mothers and virtually all socioeconomic and demographic subsets considered in this paper. The results by marital status and education categories also suggest that the effect of fast food availability may be more adverse, i.e. higher weight gain and greater probabilities of excessive weight gain and c-section birth, for women in lower socioeconomic status (less educated and unmarried) than their married and more educated counterparts. Further, the magnitudes of these associations are large, which suggests that discouraging the entrance of new fast-food restaurants in the market or reducing the number of fast-food restaurants in MSAs may greatly lower prenatal weight gain, the probability of excessive weight gain, and the likelihood of caesarean delivery. The empirical model also controls for the number of full-service restaurants and grocery stores in the MSA of residence as these are relevant substitutes in an individual's set of food choices. The relationships of the availability of full-service restaurants and grocery stores with maternal health outcomes are mostly statistically insignificant, but when they are significant they tend to be positively associated.

The relationship between the availability of fast food and birthweight on the other hand is statistically and substantively insignificant, irrespective of whether birthweight is measured as a continuous variable or it is modeled as the probability of LGA, SGA, LBW, HBW, or macrosomia. It is interesting to note that while full-service restaurants and grocery stores are not significantly related to maternal health outcomes they are statistically significantly related to infant health outcomes – full-service restaurants tend to be negatively associated with birthweight and grocery stores are positively linked to birthweight.

Overall, the results of this analysis are consistent with those presented in Currie et al (2010), the study closest to this one. The most notable difference between the two sets of findings is that my estimated results are larger in magnitude than those in Currie et al (2010). This is not too alarming because our independent variables and geographic regions are measured very differently. While Currie et al (2010) examine the effect of a binary variable – exposure to fast food in the immediate vicinity of mother's residence, this paper estimates the relationship between the number of fast-food restaurants in mother's MSA of residence. Moreover, given the potential biases in the estimated coefficients, the estimates of this paper are likely upper bounds of the true effect of fast food.

The limitations of this analysis are as follows: First, to the extent that unobserved factors correlated with within-MSA changes in supply of fast-food restaurants and health outcomes vary with time, these results may be biased. I control for a host of time-variant MSA characteristics, but there may be omitted factors that are correlated with changes in both demand and supply of fast food over time. Second, I cannot control for preconception BMI, which would potentially bias my results. I try to overcome this limitation by using gestational diabetes as a proxy for overweight/obesity, however, this is an imperfect solution and the potential for bias remains. Third, I am unable to test if these results hold up to the inclusion of mother fixed effects. The advantage of including mother fixed effects would be to purge individual mothers' taste for fast food, and her expectations about her pregnancy and her infant's health, i.e. factors that may be correlated with the number of fast-food restaurants in her community and her health outcomes. I conjecture that individual mother's expectations and taste for fast food is likely to be highly correlated with the community average, but in the absence of an empirical test this remains a limitation of this paper. Finally, I argue in this paper that limiting the fast food analysis to restaurants in the immediate vicinity of mother's residence is too narrow, however, I acknowledge that including the fast-food restaurants in the entire MSA of mother's residence may be too broad and may in fact give rise to non-classical measurement error. Further research is needed to examine the effect of fast-food restaurants outside the residential zone but within the area that encompasses the choices that influence mother's dining decisions.

Bearing in mind these limitations, this paper contributes to the literature by providing evidence that policies that discourage the construction of new restaurants (as in south Los Angeles) or policies that reduce the existing number of restaurants may have a perceptible impact on the likelihood of excessive prenatal weight gain for most socioeconomic and demographic subgroups. This, in conjunction with the clinical and experimental findings discussed above, suggests that discouraging fast food consumption has the potential to improve maternal health in the short run and reduce obesity in the long run. However, the full-service restaurants and grocery store findings related to infant health also call for circumspection on the part of policymakers and cautions against a one-size-fit-all policy that is aimed solely at fast-food restaurants as a means to reducing obesity in the U.S.

Acknowledgements

I wish to thank David Bradford, Kosali Simon, Jeffrey Wenger, Jennie Wenger, four anonymous referees, and seminar participants at Central Michigan University, Indiana University-Purdue University at Fort Wayne, Lafayette College, Mills College and Food Sciences Department at University of Georgia for their insightful comments on an earlier version of this paper. I am grateful to Sangyub Ryu and Michael Koehler for their research assistance.

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