

Peer Effects, Fast Food Consumption and Obesity*

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Abstract

Recently, the economic burden of obesity costs has been one of the major concerns of health policy makers in the United States. A growing body of the health economics literature has tried to look into the obesity problem from a social network perspective. Empirical evidence seem to be pointing at a social multiplier effect as a responsible for the obesity epidemics. In this paper, we take this evidence a step ahead and try to identify one of the behavioral channels through which the identified peer effect may flow, namely eating habits. To do so, we use a spatial autoregressive approach and adopt the identification methods developed by Bramoullé, Djebbari & Fortin (2008) and Lee, Liu & Lin (2008). We use the Add health survey in which information about the adolescent fast food consumption reflects eating habits. Peer groups are defined using nominated friends. The model is estimated using a Generalized 2SLS strategy proposed in Kelejian and Prucha (1998) and refined in Lee (2003). Preliminary results show that there is a positive non-significant peer effect in fast food consumption among adolescents in general. Results become larger and significant once we focus on girls with same sex friends. We find no evidence of peer effects in eating habits for boys with same sex friends.

Keywords: Obesity, peer effects, fast food, spacial models.

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1 Introduction

...“If the prevalence of obesity could be reduced (and along with it, chronic disease), health spending could be cut by \$100 billion to \$150 billion per year, trimming up to 18.7 percent off the nation’s total health-care budget” (Thorpe’s team estimates)

For the past few years obesity has been one of the major concerns of health policy makers in the US. It has also been one of the principal sources of the increased health care costs. Obesity is associated with serious health problems such as type 2 diabetes, heart disease and certain cancers. Also, obesity has often been related to maternal employment. It may be considered as one of the consequences of the increased female labor participation and the decreased time allocated to household tasks. Anderson, Butcher & Levine (2003) find that the increase in the hours worked by the mother explains part of the increase in child obesity. In fact women allocate less time to the preparation of meals and consequently purchase more fast food (Cawley & Liu 2007). Furthermore, from a labor market perspective, obesity seems to have a negative impact on employment of both women and men (Morris 2007) as well as on wages (Cawley 2004). This latter effect on wages was attributed to increased health insurance costs for these workers rather than discrimination (Bhattacharya & Bundorf 2005). One can therefore argue that the obesity epidemics is not a mere health issue: it is a serious public concern. Its underlying causes and impacts go beyond the medical field. It is partly triggered by changes in the patterns of labor market participation and has a non-negligible impact on both government expenditures and labor market outcomes. In recession, the burden of obesity is likely to become heavier. The impact of the economic downturn usually translates into an increased consumption of inferior goods (such as fast food) and a decreased spending on normal goods (such as gym memberships).¹

A growing body of the health economics literature has tried to look into this problem from a new perspective using a social interaction framework. Christakis & Fowler (2007), Trogdon, Nonnemaker & Pais (2008), and Renna, Grafova & Thakur (2008) seem to be unanimously pointing at

¹At the start of the 2008 recession, “the share prices of some fast food companies outperformed the Standard & Poor’s 500 Index in the stock market” (Ludwig & Pollack 2009).

the *social multiplier* as a responsible for the obesity epidemics. As a consequence, an estimated aggregate elasticity on obesity is stronger than the true individual effect since it incorporates, in addition to this effect, positive peer effects stemming from social interactions.² In this paper, we take these evidence a step ahead and try to identify the channels through which peer effect in adolescence obesity may flow. Adolescents are usually vulnerable. They often compare themselves to their friends and may alter their choices to conform to the behavior of their peers. Unless we scientifically prove that obesity is a virus,³ it is counter intuitive to think that one can gain weight by simply interacting with an obese person. Therefore, we are inclined to think that the presence of real peer effects in obesity can be estimated using behavioral channels such as *eating habits*. Eating often occur in a social context. Therefore people in general and adolescents in particular may get influenced by each other when it comes to eating habits including fast food consumption.

Ones' interest in peer effects in general is mainly policy driven. In this spirit, we try to pinpoint a variable that may at the same time explain (partly) the obesity epidemics and serve as a target for policy intervention. The current peer effect literature on obesity considers exclusively BMI measures. Information on the presence of peer effects in BMI measures is valuable but it implicitly points at the BMI as an externality. While taxing obesity might raise discriminatory issues, taxing fast food comes in line with consumption tax, which, to the best of our knowledge does not raise such issues. Thus informing policy makers about peer effects in fast food consumption allows for the use of the identified multiplier effect to internalize this externality.

Unfortunately, highlighting the presence of peer effects in fast food consumption presents difficult identification as well as estimation challenges. Identification issues were pointed by Manski (1993) and discussed among others in Moffit (2001), Brock & Durlauf (2001), and Durlauf (2003). It is difficult to separate the global effect of peers, often referred to as *social effects*, from the *correlated effects* i.e., effects resulting from the fact that in-

²Researchers also found that friends had a significant effect in adolescent smoking, drinking, illicit drug use (Clark & Loheac 2007) and risky sex (Jaccard, Blanton & Dodge 2005).

³We acknowledge that some recent studies have pointed that obesity might be partially due to a virus ad-36 (see Rogers, Fusinski, Rathod, Loiler, Pasarica, Shaw, Kilroy, Sutton, McAllister, Mashtalir et al. (2007)).

dividuals from the same group tend to behave similarly because they are alike and/or share a common environment. Also, the *Reflection Problem* (see Manski 1993) makes it difficult to identify *endogenous social effect* (peer actions) from the *exogenous social effect* (peer characteristics). To address these problems, we use a spatial autoregressive approach and adopt the identification methods developed by Bramoullé et al. (2008) and Lee et al. (2008). To account for the presence of a right-hand side endogenous variable, the model is estimated using a generalized two-stage least square proposed in Kelejian & Prucha (1998) and refined in Lee (2003). We partially address the problem of correlated effects by considering them as fixed effects at the network (school) level. We then get rid of these effects through a panel-like *within* transformation.

To estimate our model of fast food consumption with peer effects, we use the second wave of the National Longitudinal Study of Adolescent Health (Add Health). We define peers as the nominated group of individuals reported as friends within the same school. The consumption behavior is depicted through the reported frequency (in days) of fast food restaurant visits in the past week.⁴ Preliminary results show that there is a positive non-significant peer effect in fast food consumption among adolescent in general. Results become larger and significant once we focus our attention to female with same sex friends. We found no evidence of peer effects in eating habits for boys with same sex friends.

The remaining parts of this paper will be laid as follows. Section 2 provides a survey of the literature on the impact of fast food consumption on obesity and on the impact of peer effects on fast food consumption. Section 3 presents the structural model of social interaction, the identification problem it raises and the adopted solution. Section 4 introduces the econometric strategy that we intend to adopt. In section 5 we give a brief overview of the Add Health Survey and we provide descriptive statistics of the data we use. In section 6 we discuss estimation results. Finally section 7 concludes.

⁴To be conservative, we assume that the student goes once a day.

2 Survey of the literature

The general issue addressed in this paper is whether the peer effect in obesity partly flows through the *eating behavior* channel. This raises two more basic issues: a) is there a link between fast food consumption and obesity? and b) are there peer effects in fast food consumption? In this paper, we focus on the second issue assuming that there is a link between fast food consumption and obesity. In fact, the relationship between fast food consumption and obesity is an empirical question that is still on the debate table.⁵ There is no clear evidence in support of a causal link between fast food consumption and obesity. Nevertheless, most of the literature in epidemiology find evidence of a positive correlation between fast food consumption and obesity (Rosenheck 2008).⁶

The economic literature reveals to be conservative with respect to this question. It focuses on the impact of “exposure” to fast food on obesity. Dunn (2008) investigates the relationship between fast food availability and obesity.⁷ He finds that an increase in the number of fast food restaurants has a positive effect on the Body Mass Index (BMI). Similarly, Currie, DellaVigna, Moretti & Pathania (2009) find evidence that proximity to fast food restaurants has a significant effect on obesity for 9th graders. The factors underlying fast food consumption were also investigated. Jeffrey & French (1998) show that hours of TV viewing per day and the frequency of meals eaten at fast food restaurants are both positively associated with increase in the body mass index of women. One drawback of this study is that it uses a non representative sample (i.e., individuals who volunteered for the study of weight gain prevention). Chou, Rashad & Grossman (2005) find a strong positive correlation between exposure to fast food restaurant advertising and the probability that children and adolescent are overweight. This effect seems to be stronger and more significant for girls (Chou et al. 2005). In fact, this influence can be clearly seen as children are more likely to pick up items that are in “Mac Donald’s” packaging (Robinson, Borzekowski, Matheson & Kraemer 2007). More generally, Cut-

⁵The literature on the impact of physical activity on obesity is also inconclusive. Petersen, Schnohr and Sorensen (2004) show that decreased physical activity in adults does not lead to obesity.

⁶For instance Bowman, Gortmaker, Ebbeling, Pereira & Ludwig (2004) finds that children who consumed fast food consumed more total energy (187 Calories).

⁷He uses an instrumental variable approach.

ler, Glaeser & Shapiro (2003) and Bleich, Cutler, Murray & Adams (2008) argue that the increased calorie intake (i.e., eating habits) plays a major role in explaining current obesity rates.

As far as the peer effects on eating habits is concerned, the literature is much more limited. In a medical experimental context, Salvy, Vartanian, Coelho, Jarrin & Pliner (2008) assess the presence of “peer effect” in pre-adolescent girls’ snack intake as a function of the co-eaters’ weight status. They show that overweight girls eating with an overweight peer consumed more calories than overweight participants eating with normal weight peers. In their study, they estimate a mixed-effect model using an experimental dataset where peers are dyads who do not know each other. Our approach is related with their work. We both are relating food intake and peers. Nevertheless, it diverges on many counts, namely the dataset type, the peer definition, the model estimated and estimation method.

3 Structural Model of Social Interaction

3.1 The Basic Model

To have a better understanding of the identification problems that social interaction models suffer from, we provide a brief overview of the *linear-in-means* model (Manski 1993). This model relates the outcome of an individual to his own characteristics, the corresponding mean characteristics of his peers and their mean outcome. Unlike most of the literature on obesity, the use of social interactions at the network level will allow us to identify endogenous peer effects (fast food consumption of peers) and exogenous peer effects (characteristics of peers).⁸

Suppose a social network with N adolescents. Each adolescent i has a set of friends P_i , the number of which is n_i . Then, the structural model is given by:

⁸It is important to highlight the fact that most of the literature on peer effect on obesity focused on peer effects on the output level *i.e.*, the BMI. In this paper, we consider peer effects on the input level, *i.e.*, fast food consumption.

$$y_i = \alpha + \beta \frac{\sum_{j \in P_i} y_j}{n_i} + \gamma x_i + \delta \frac{\sum_{j \in P_i} x_j}{n_i} + \varepsilon_i, \quad (1)$$

where y_i represents fast food consumed by individual i per unit of time, x_i represents the individual i 's observable characteristic. To simplify our presentation, we look at only one characteristic (e.g., adolescent pocket money).⁹ The ε_i 's are the error terms. The terms $\frac{\sum_{j \in P_i} y_j}{n_i}$ and $\frac{\sum_{j \in P_i} x_j}{n_i}$ are respectively his peers' mean fast food consumed and characteristics. In the context of our paper, β is what we call *endogenous social effect*. It reflects how the individual's consumption of fast food is affected by his peers' mean fast food consumption. One usually assumes that $|\beta| < 1$. The *exogenous social effect* is represented by the parameter δ . It captures the impact of his peers' mean characteristic on his fast food consumption. In this baseline formulation, we assume that the vector of the adolescents' characteristic in the network, \mathbf{x} , is strictly exogenous, that is, $E(\varepsilon_i | \mathbf{x}) = 0$. Thus, we assume no correlated effects.

3.2 Group Interactions and the Identification Problem

Following the formulation of Bramoullé et al. (2008), let us denote \mathbf{y} the y_i vector, \mathbf{I} the identity matrix and $\mathbf{1}$ a vector of ones. We can therefore rewrite (1) in matrix notation as follows:

$$\mathbf{y} = \alpha \mathbf{1} + \beta \mathbf{G} \mathbf{y} + \gamma \mathbf{x} + \delta \mathbf{G} \mathbf{x} + \boldsymbol{\varepsilon}, \quad (2)$$

where $E(\boldsymbol{\varepsilon} | \mathbf{x}) = 0$, $E(\boldsymbol{\varepsilon} \boldsymbol{\varepsilon}' | \mathbf{x}) = \boldsymbol{\Sigma}$. The variance matrix $\boldsymbol{\Sigma}$ is positive definite and \mathbf{G} is the interaction matrix, where G_{ij} takes a value $\frac{1}{n_i}$ for i 's friends and zero otherwise.¹⁰ Note that the model (2) is similar to a spatial autoregressive (SAR) model (e.g., Cliff & Ord (1981)) extended to allow for exogenous effects.

⁹The model can be easily generalized using more than one characteristic.

¹⁰The \mathbf{G} matrix is row normalized.

Since $(\mathbf{I} - \beta\mathbf{G})$ is invertible, the corresponding reduced form model can be written as follows:

$$\mathbf{y} = \alpha(\mathbf{I} - \beta\mathbf{G})^{-1}\boldsymbol{\iota} + (\mathbf{I} - \beta\mathbf{G})^{-1}(\gamma\mathbf{I} + \delta\mathbf{G})\mathbf{x} + (\mathbf{I} - \beta\mathbf{G})^{-1}\boldsymbol{\varepsilon} \quad (3)$$

Bramoullé et al. (2008) have shown that this model is identified (that is, one can recover the structural parameters $(\alpha, \beta, \gamma, \delta)$) if and only if the matrices \mathbf{I} , \mathbf{G} and \mathbf{G}^2 are linearly independent. This condition will be satisfied for instance if an individual has a friend's friend who is not his friend, *i.e.*, the network has an *intransitive* triad.

Let us first suppose that adolescents interact in groups. This is the case when one can partition the network in subsets (*e.g.*, schools or classes) such that each adolescent belonging to a subset interacts with all the others in the same subset but with no others in the other subsets. Assume also that the individual i is included in his group ($i \in P_i$) and that groups have the same size, then it is easy to show that $\mathbf{G} = \mathbf{G}^2$. This means that all friends' friends of an adolescent are also his friend. In this case, it is clear that the matrices \mathbf{I} , \mathbf{G} and \mathbf{G}^2 are linearly dependent and the model is underidentified. In fact, the only parameters that we would be able to recover are the reduced form parameters $\alpha(1 - \beta)^{-1}$, γ and $(1 - \beta)^{-1}(\gamma\beta + \delta)$, a composite parameter for global social effects.¹¹ With such information at hand, one cannot disentangle the *endogenous social effect*, β from the *exogenous social effect*, δ , our prime interest from estimating an equation of type (2). Moreover, from a policy perspective, the reduced form parameters do not identify the channel through which the obesity flows and thus, do not allow policy makers to take an enlightened decision as far as the control for the spread of obesity is concerned. In fact, disentangling these two effects provides valuable information for, if obesity flows through eating behavior (endogenous social effect), then a tax on fast food can lead to a decrease in obesity through a social multiplier effect.¹²

¹¹On the other hand if at least two groups are of different sizes, the structural parameters are identified.

¹²Similarly if we identify the channel to be physical exercise then a subsidy that helps people exercise can through a multiplier effect lead to a decreased spread of obesity.

3.3 Network Interactions with correlated effects

Now, let us suppose that individuals interact in networks (rather than in groups), and that individual i is excluded from his reference group ($i \notin N_i$). Suppose also that the correlated effects be present. To understand how this works, assume that we observe an i.i.d sample $(y_l, \mathbf{x}_l, \mathbf{G}_l)$ with $l = 1, \dots, L$ from a population of networks. At the individual level, the model (1) can thus be written as:

$$y_{il} = \alpha_l + \beta \frac{\sum_{j \in N_i} y_{lj}}{n_i} + \gamma x_{li} + \delta \frac{\sum_{j \in N_i} x_{lj}}{n_i} + \varepsilon_{li}, \quad (4)$$

where α_l represents a correlated effect, modeled here as an unobserved fixed effect common to all adolescents in the same network. In the context of our study this would refer for instance to recreational facilities available in school, fast food restaurants around the school or school snack policies and other school policy regarding health and eating habits.¹³ One assumes that $E(\varepsilon_{li} | \mathbf{x}_l, \mathbf{G}_l, \alpha_l) = 0$, i.e., \mathbf{x}_l and \mathbf{G}_l are strictly exogenous, given α_l . To eliminate the correlated effects, one can perform a panel-like *within* transformation to the model at the network (or global) level. More precisely, we average (4) over all students of network l and subtract it from i 's equation.¹⁴ To write the full structural model in matrix notation, let us first define the matrix $\mathbf{I}_l - \mathbf{H}_l$, with $\mathbf{H}_l = \frac{1}{n_l} (\mathbf{1}_l \mathbf{1}_l')$. This matrix obtains the deviation from network means. Vertically concatenating all variables of all networks, the structural model in matrix can be written as:

$$(\mathbf{I} - \mathbf{H})\mathbf{y} = \beta(\mathbf{I} - \mathbf{H})\mathbf{G}\mathbf{y} + \gamma(\mathbf{I} - \mathbf{H})\mathbf{x} + \delta(\mathbf{I} - \mathbf{H})\mathbf{G}\mathbf{x} + (\mathbf{I} - \mathbf{H})\varepsilon, \quad (5)$$

where \mathbf{H} is the block-diagonal matrix of all \mathbf{H}_l . One sees from (5) that the network within transformation has eliminated the α_l 's, that is, the correlated effects.

¹³These effects might be observed in real life but are not observed in the dataset.

¹⁴Alternatively, one could perform a *local* transformation where the averaging is performed over all student i 's friends. Bramoullé et al. (2008) has shown that if the model is identified with a global transformation, it will also be identified with a local transformation.

The resulting reduced form equation is given by:

$$(\mathbf{I} - \mathbf{H})\mathbf{y} = (\mathbf{I} - \mathbf{H})(\mathbf{I} - \beta\mathbf{G})^{-1}(\gamma\mathbf{I} + \delta\mathbf{G})\mathbf{x} + (\mathbf{I} - \mathbf{H})(\mathbf{I} - \beta\mathbf{G})^{-1}\varepsilon.$$

The identification of the model with correlated effects is more demanding. Bramoullé et al. (2008) has shown that it is identified if the matrices \mathbf{I} , \mathbf{G} , \mathbf{G}^2 and \mathbf{G}^3 are linearly independent. This condition will be satisfied for example when the diameter of the network (*i.e.*, maximal friendship distance) is greater than or equal to 3.¹⁵

4 Empirical Model and Estimation strategy

Having laid the intuition behind the identification method we used, we will now turn to the empirical model we estimate. For a given vector of inputs \mathbf{y} , a given matrix of individuals' characteristics \mathbf{X} , and a block diagonal matrix \mathbf{G} where each block represents a school's network, the transformed model is given by:

$$(\mathbf{I} - \mathbf{H})\mathbf{y} = \beta(\mathbf{I} - \mathbf{H})\mathbf{G}(\mathbf{I} - \mathbf{H})\mathbf{y} + (\mathbf{I} - \mathbf{H})\mathbf{X}\gamma + (\mathbf{I} - \mathbf{H})\mathbf{G}(\mathbf{I} - \mathbf{H})\mathbf{X}\delta + \boldsymbol{\nu}, \quad (6)$$

with $\boldsymbol{\nu} = (\mathbf{I} - \mathbf{H})\varepsilon$. As mentioned in the previous section, in the presence of correlated effects, the identification is achieved whenever we have linear independence between \mathbf{I} , \mathbf{G} , \mathbf{G}^2 and \mathbf{G}^3 , a condition that will be checked with the data we use.

Also, we impose some structure to the error terms, that is, $E(\varepsilon | X, G) = 0$ and $E(\varepsilon\varepsilon' | X, G) = \sigma^2\mathbf{I}$.¹⁶ Note that the variance-covariance matrix of $\boldsymbol{\nu}$ is singular and has the following form:

¹⁵The diameter of the network is defined as the maximal distance that can relate any two individuals in a network. Where the distance is the minimal segment that links any two individuals.

¹⁶In this preliminary version of the paper, we ignore the possibility of spatial correlation in the error terms.

$$E(\nu\nu' | \mathbf{X}) = (\mathbf{I} - \mathbf{H})(\mathbf{I} - \mathbf{H})'\sigma^2 = (\mathbf{I} - \mathbf{H})\sigma^2. \quad (7)$$

To resolve the problem associated with the singularity of the variance-covariance matrix, we follow Lee et al. (2008). Consider the orthonormal base of $(\mathbf{I} - \mathbf{H})_l$ and let \mathbf{Q}'_l the sub-matrix corresponding to the eigenvalues of 1. \mathbf{Q}'_l has the following properties : $\mathbf{Q}'_l\mathbf{Q}_l = \mathbf{I}_{n_l^*}$, $\mathbf{Q}_l\mathbf{Q}'_l = (\mathbf{I} - \mathbf{H})_l$ and $\mathbf{Q}'_l\mathbf{v} = 0$ where $n_l^* = n_l - 1$.

Let us denote by \mathbf{Q} the bloc diagonal matrix, where \mathbf{Q}'_l are the matrices on the diagonal (with $l = 1, \dots, L$). Premultiplying (6) by \mathbf{Q}' , the model can now be written as follows:

$$\mathbf{y}^* = \beta\mathbf{G}^*\mathbf{y}^* + \mathbf{X}^*\gamma + \mathbf{G}^*\mathbf{X}^*\delta + \nu^*, \quad (8)$$

where $\mathbf{y}^* = \mathbf{Q}'\mathbf{y}$, $\mathbf{X}^* = \mathbf{Q}'\mathbf{X}$, $\mathbf{G}^* = \mathbf{Q}'\mathbf{G}\mathbf{Q}$ and $\nu^* = \mathbf{Q}'\nu$. Note that with this transformation $E(\nu^*\nu^{*'} | \mathbf{X}) = \sigma^2\mathbf{I}_{n_l^*}$. Note also that while the bloc diagonal matrix \mathbf{G} is composed of row normalized matrices that have zero diagonals (\mathbf{G}_l), \mathbf{G}^* will not preserve these properties. This come without a cost as far as the consistency of the estimates is concerned.

To estimate the model in (8) we adopt a generalized two-stage least squares procedure (G2LS) developed by Kelejian & Prucha (1998) and refined in Lee (2003) and Lee et al. (2008). This approach provides a simple and tractable numerical method to estimate an asymptotically optimal IV estimator. In the case of our paper this method will consist of a two-step two-stage estimation. To simplify the notation, let us denote by $\tilde{\mathbf{X}}^*$ the matrix of explanatory variables such that $\tilde{\mathbf{X}}^* = [\mathbf{G}^*\mathbf{y}^* \quad \mathbf{X}^* \quad \mathbf{G}^*\mathbf{X}^*]$. Let \mathbf{P} be the weighting matrix such that $\mathbf{P} = \mathbf{S}(\mathbf{S}'\mathbf{S})^{-1}\mathbf{S}'$, and \mathbf{S} a matrix of instruments such that $\mathbf{S} = [\mathbf{X}^* \quad \mathbf{G}^*\mathbf{X}^* \quad \mathbf{G}^{*2}\mathbf{X}^*]$. In the first step, we estimate the following 2SLS estimator:

$$\hat{\theta}_{2SLS} = (\tilde{\mathbf{X}}^{*'}\mathbf{P}\tilde{\mathbf{X}}^*)^{-1}\tilde{\mathbf{X}}^{*'}\mathbf{P}\mathbf{y}^*,$$

where θ is the vector of parameters of the structural model (6). This step allows us to obtain consistent estimates of θ . However, they are not asymptotically efficient. Using a suggestion by Kelejian & Prucha (1998), Lee (2003) shows that $E[\tilde{\mathbf{X}}^*]$ is an ideal matrix of instruments and that, using estimates of the parameters from the first step, one can construct a matrix of instruments which converges to this matrix.

To avoid any confusion with the first step, let us denote this new matrix of instruments $\hat{\mathbf{Z}}$ such that $\hat{\mathbf{Z}} = \mathbf{Z}(\hat{\theta}_{2SLS})$ with:

$$\mathbf{Z}(\theta) = [\mathbf{E}[\mathbf{G}^* \mathbf{y}^*(\theta) | \mathbf{X}^*, \mathbf{G}^*] \quad \mathbf{X}^* \quad \mathbf{G}^* \mathbf{X}^*],$$

and where

$$E[\mathbf{G}^* \mathbf{y}^*(\theta) | \mathbf{X}^*, \mathbf{G}^*] = \mathbf{G}^* (\mathbf{I} - \beta \mathbf{G}^*)^{-1} [(\mathbf{X}^* \gamma + \mathbf{G}^* \mathbf{X}^* \delta)].$$

We then estimate:

$$\theta^{LEE} = (\hat{\mathbf{Z}}' \tilde{\mathbf{X}}^*)^{-1} \hat{\mathbf{Z}} \mathbf{y}^*.$$

This estimator can be shown to be asymptotically best IV estimator. The variance matrix is given by:

$$\sqrt{N}(\hat{\theta}_{Lee} - \theta) \sim N \left(0, \left[\mathbf{Z}' \tilde{\mathbf{X}}^* \mathbf{R}^{-1} \tilde{\mathbf{X}}^* \mathbf{Z} \right]^{-1} \right).$$

The matrix \mathbf{R} is consistently estimated by

$$\hat{\mathbf{R}} = s^2 \frac{\hat{\mathbf{Z}}' \hat{\mathbf{Z}}}{N},$$

where $s^2 = N^{-1} \sum_{i=1}^N \hat{u}_i^2$ and \hat{u}_i are the residuals from the second step. It is important to note that, as in Kelejian & Prucha (1998), we assume that errors are homoscedastic. The estimation theory developed by Kelejian & Prucha (1998) under the assumption of homoscedastic errors does not apply if we assume heteroscedastic errors (Kelejian & Prucha Forthcoming 2008).

5 Data and Descriptive Statistics

The Add Health survey is a longitudinal study that is nationally representative of American adolescents in grades 7 through 12. It is one of the most comprehensive health surveys that contains fairly exhaustive social, economic, psychological and physical well-being variables along with contextual data on the family, neighborhood, community, school, friendships, peer groups, romantic relationships, etc. In the first wave (September 1994 to April 1995), all students (around 90 000) attending the randomly selected high schools were asked to answer a short questionnaire. An In-home sample (core sample) of approximately 20 000 students was then randomly drawn from each school. These adolescents were asked to participate in a more extensive questionnaire where detailed questions were asked. Information on (but not limited to) health, nutrition, expectations, parents' health, parent adolescent relationship and friends nomination was gathered.¹⁷ This cohort was then followed in-home in the subsequent waves in 1996 (wave II) and 2001 (wave III). The extensive questionnaire was also used to construct the saturation sample that focuses on 16 selected schools. Every student attending these selected schools answered the detailed questionnaire. The saturation sample includes 16 selected schools. There are two large schools and 14 other small schools. All schools are racially mixed and are located in major metropolitan areas, except one large school that has a high concentration of white and is located in a rural area. Consequently, the fast food consumption may be subject to downward bias if one accepts the argument that the fast food consumption among the White adolescents is usually lower than that of the Black adolescents.¹⁸

In this paper we use the saturation sample of wave II in-home survey to investigate the presence of peer effects in fast food consumption. One of the innovative aspects of this wave is the introduction of the nutrition section. It reports among other things food consumption variables (e.g., fast food, soft drink, desserts, etc.). This allows us to depict food consumption patterns of each adolescent and relate it to that of his peer group. In addition, the availability of friend nomination allows us to retrace school friends and

¹⁷ Adolescents were asked to nominate either 1 female friend and 1 male friend or 5 female friends and 5 male friends.

¹⁸ It is unclear whether black population consumes more fast food than the white population. Block, Scribner & DeSalvo (2004) shows that predominantly Black neighborhoods have relatively more fast food restaurants than predominantly White neighborhoods.

thus construct friendship networks. Our analysis focuses on the saturation sample.¹⁹

We use friends nominations to construct the network of friends. First, we consider all nominated friends as network members regardless of the reciprocity of the nomination. If an adolescent nominates a friend then a link is assigned between these two adolescents. Following Renna et al. (2008), we also present results with networks stratified by gender.

5.1 Descriptive statistics

The dependent variable of interest is fast food consumption. In the survey, adolescents are asked to report the frequency of their fast food consumption during the past 7 days. Table 1 reports respectively the mean and the standard deviation of the endogenous variable, the covariates and other relevant characteristics. We note that on average, adolescents' fast food consumption is fairly within the range of 2.33 times/week. This is consistent with the frequency reported by the Economic Research Service of the United States Department of Agriculture (Lin, Guthrie & Blaylock 1996). Around around 62% of the adolescents consumed fast food twice or more in the past week and 44% of the adolescents who had consumed fast food did so 3 times in the past week. We also inspected the data to check for the presence of parental intervention in eating habits. We note that parents are absent 38% of the times during the consumption of evening meals and that 83 % of children have the freedom to chose their meals. Evening meals is the only time when parents have the power to check on the quality/quantity of the food consumed. This coupled with the freedom to chose food leaves the door wide open for peer influence in fast food consumption.²⁰

The covariates include the adolescent's personal characteristics, family characteristics as well as the corresponding exogenous social effects. The personal characteristics are gender, age, ethnicity (white or not) and grade. We observe that 50% of the sample are females, that the mean age is 16.3 years and that 57% are White. Family characteristics are dummies

¹⁹In the saturation sample all students are asked to name their friends.

²⁰These figures are not reported in the paper but can me provided upon request.

for mother and father education. We observe that around 45% of mothers and fathers have at least some college education. To control for parents' income we use child allowance as a proxy. An adolescent's allowance is on average 8.28 per week, around 50 % of the adolescents our the sample have a weekly allowance. Evidence in the literature has shown a positive correlation between allowance and fast food consumption.

5.2 The Construction of the Graph Matrix

We construct a sub-matrix of graph for each school separately and then we include all these sub-matrices in a block-diagonal matrix \mathbf{G} .²¹ In each school we eliminate individuals for which we have missing values. We allow the sub-matrices to contain individuals that are isolated.²² Since these latter may be friends with other individuals in the network, they may affect the network even if they declare having no friends at all. They also introduce variability that helps the identification of the model. To check whether identification of the structural parameters holds, we provide evidence that \mathbf{I} , \mathbf{G} , \mathbf{G}^2 and \mathbf{G}^3 are linearly independent. We first vectorize the identity matrix \mathbf{I} , as well as the matrix \mathbf{G} , \mathbf{G}^2 and \mathbf{G}^3 . We then concatenate $vec(\mathbf{I})$, $vec(\mathbf{G})$, $vec(\mathbf{G}^2)$ and $vec(\mathbf{G}^3)$ in one matrix and check that the rank of the matrix formed had a rank of four.

6 Results

This section presents our preliminary results of the peer effects in fast food consumption. To provide a baseline, section 6.1 discusses the results of the linear-in-means model as presented in section 3.1 (no correlated effects). We compare the OLS estimates with the 2SLS estimates of the baseline model. In section 6.2 , we present the results of the model in global transformation as presented in section 3.3. We provide both the OLS as well

²¹Following previous literature, we assume that there is interaction within each school but not interaction between schools.

²²An isolated individual is an individual with an empty group of friends. We do not observe schools where all individuals are isolated

as the G2SLS estimates. We finally present results stratified at the gender level.

6.1 Baseline: Linear-in-Means Model without correlated effects

To start our inspection of peer effects in fast food consumption it is useful to look at the simplest form of the model through a naive OLS method. To account for the presence of an endogenous variable on the right-hand side, we also use a 2SLS approach. The elements of the instrument matrix are similar to the ones that are presented earlier in the \mathbf{S} matrix. The only difference is that the elements are not expressed in deviation.

In tables 2 and 3, the parameter estimates from the OLS and standard errors are presented in the first two columns while the parameter estimates and standard errors from the 2SLS are presented in the last two columns. We begin by comparing the endogenous social effects β across the OLS case and the 2SLS case. We note that the endogenous social effect estimated by OLS is 0.21 where as the one estimated by 2SLS is 0.22.²³ This latter estimate is significant suggesting that an adolescent would increase his fast food consumption by 0.22 in response to a one point increase in his friends' mean fast food consumption.

As for individual characteristics, fast food consumption seems to be increasing with age, father's education, and the allowance per week. Turning our attention to the exogenous social effects, we observe that fast food consumption decreases with mean mother's education. It increases with mean father's education of peers and allowance. These results must be interpreted with caution though, since they do not take correlated effects into account.

²³The naive estimates seem to be slightly biased downward in comparison with the 2SLS estimates suggesting a negative correlation between the structural error term and the endogenous variable.

6.2 Linear-in-Means Model with correlated effects

The baseline model does not take into account the presence of correlated effects. As mentioned earlier, correlated effects may arise when we have endogenous group formation (homophily is an example) or when we have common environmental factors. In this section we attempt to control for these effects by performing a panel-like *within* transformation at the network level.

An inspection of tables 4 and 5 reveals that estimates of the endogenous effects 0.15 in the OLS case and 0.11 in the G2SLS case. Endogenous effects are lower once we take into account the endogeneity and control for correlated effects, they also become statistically insignificant when using the G2SLS method. Despite our believe that fast food consumption is linked to issues of identity and friends (Story, Neumark-Sztainer & French 2002), empirical results do not come in line with our expectations. We are therefore inclined to infer that peer effects in fast food consumption may not be the adequate channel that we should be looking at in the quest of assessing peer effects and obesity. As for individual specific effects, they follow fairly the basic linear-in-means model. Fast food consumption is positively associated with age and father's education and positively associated with allowance. Turning our attention to the exogenous social effects, fast food consumption decreases with peers' mother's education.

Given that interactions within-gender are stronger (Kooreman 2007), we try to assess whether the adolescents are more affected by the consumption habits of their same gender friends. This interest is motivated by the inconclusive evidence in the obesity literature. Christakis & Fowler (2007) found that same sex friends had relatively greater influence than opposite sex friends. Also, Renna et al. (2008) finds that females' IV estimates are larger and statistically more significant than non-IV estimates while males' IV estimates are statistically insignificant. On the other hand, Trogdon et al. (2008) does not find evidence that same sex friends have more influence than friends of other gender. To address this issue, we estimate a stratified specification using the G2SLS method.

Tables 6 and 7 show results for endogenous social effects stratified by same sex friends. We first compare the endogenous effects across both genders. We notice that endogenous social effects for females with same

sex friends are positive and significant (.57) while endogenous effects for males with same sex friends are negative but not significant (-.14). Females seem to be more influenced by their same sex friends than boys. This result is consistent with some previous finding in the literature on obesity (Christakis & Fowler 2007) and (Chou et al. 2005). The social multiplier associated with an exogenous increase in females' fast food consumption is 2.32 ($= 1/(1 - 0.57)$), which reflects a strong endogenous peer effect.

Looking at individual characteristics we note that females' fast food consumption increases with father's education. Males' fast food consumption show different patterns, it is increasing with age and allowance and father's education. As for the exogenous social effects, females' fast food consumption decreases with the mean father's education of peers. Males' fast food consumption seems to deviate from female's patterns once again. It decreases with mean mother's education of peers and increases with father's education of peers. This suggests peer effects in fast food consumption may be gender sensitive.

7 Conclusion

In this paper we study peer effect in obesity through peer effects in fast food consumption in an attempt to highlight the presence of an *eating habit* channel. Preliminary results show that there are no significant endogenous effects in fast food consumption at the global level. Nevertheless, when stratifying by gender, we find evidence of a significant positive endogenous effect on females with same sex friends and negative but non significant effect on males. Our results are consistent with results previously obtained in the literature. The absence of peer effects in fast food consumption at the global level comes in line with previous literature, namely Cohen-Cole & Fletcher (2008b) and Cohen-Cole & Fletcher (2008a). These authors show that the estimated peer effects are in reality over-estimated and suggest that caution should be taken when tackling peer effects. On the other hand, the presence of positive and significant peer effects for females with peers of the same sex converges with a larger body of the literature on peer effects and obesity. For instance, Renna et al. (2008) found that these effects are positive and significant for females with peers of the same sex but insignificant for males.

Basically, our results are consistent with the hypothesis that if there are peer effects in obesity, a non-negligible part of it flows through *eating habits*. In the case of females with peers of the same sex, our estimated social multiplier is as high as 2.32. This social multiplier coupled with the positive impact of the ongoing economic downturn on fast food consumption can exacerbate the prevalence of obesity in the years to come. On the other hand, this multiplier may contribute to the reduction of the spread of obesity and the decrease in health care costs, as long as it is exploited by policy makers through adequate tax and subsidy reforms.

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Variable	Mean	S.D
Fast Food Consumption (past 7 days)	2.33	1.74
Female	.50	.50
Age	16.36	1.44
Mother Present	.85	.35
Mother Education		
High school/GED/Vocational Instead of high school	.36	.48
Some College/Vocational After high school	.20	.39
College	.18	.38
Advanced Degree	.06	.24
Don't Know	.04	.20
Father Education		
High school/GED/Vocational Instead of high school	.32	.46
Some College/Vocational After high school	.17	.37
College	.18	.38
Advanced Degree	.08	.26
Don't Know	.03	.15
Missing	0.02	.16
Grade 9-10	.26	.44
Grade 11-12	.61	.48
Allowance	8.28	11.65
Observations:		2355

Table 1: Descriptive Statistics

Variable	OLS		S.E	2SLS		S.E
endogenous social effect	0.2077	***	0.0331	0.2192	***	0.0334
Individual Specific Characteristics						
female	-0.0721		0.0786	-0.1468	*	0.0793
age	0.1559	***	0.0434	-0.1003	**	0.0438
white	-0.1076		0.0939	-0.1753	*	0.0947
Mother present	-0.0151		0.0996	-0.1416		0.1005
<i>Mother no HS (Omitted)</i>						
Mother HS	-0.0848		0.1195	-0.1713		0.1205
Mother Some College	-0.0376		0.1335	-0.1373		0.1346
Mother college	0.0213		0.1421	-0.0819		0.1433
Mother Advanced	-0.0258		0.1875	-0.1247		0.1890
Mother Don't know	-0.1714		0.2066	-0.3122		0.2084
<i>Father no HS (Omitted)</i>						
Father HS	0.2742	**	0.1162	0.2193	*	0.1172
Father Some College	0.2116		0.1336	0.1601		0.1347
Father College	0.3115	**	0.1374	0.2755	**	0.1386
Father Advanced	0.1731		0.1752	0.1273		0.1767
Father Don't know	0.2778		0.1756	0.2291		0.1771
Father Missing	0.0907		0.2338	0.0658		0.2357
<i>Grade 7-10 (Omitted)</i>						
grade9-10	0.0882		0.1931	0.2651		0.1947
grade11-12	0.3164		0.2265	0.8655	***	0.2284
Allowance	0.0092	***	0.0031	0.0078	**	0.0032

Table 2: Peer effects in fast food consumption without correlated effects

*** significant at 1% ** significant at 5% * significant at 10%

Variable	OLS	S.E	2SLS	S.E
Exogenous social Effects				
female	-0.0897	0.1246	-0.0847	0.1255
age	-0.0320	0.0214	-0.0440	** 0.0216
white	0.0110	0.1244	0.0602	0.1254
Mother present	0.0773	0.1667	-0.0449	0.1681
<i>Mother no HS (Omitted)</i>				
Mother HS	-0.3877	** 0.1868	-0.4059	** 0.1883
Mother Some College	-0.3947	* 0.2127	-0.4525	** 0.2145
Mother college	-0.2531	0.2179	-0.2961	0.2198
Mother Advanced	-0.7011	** 0.3089	-0.7387	** 0.3115
Mother Don't know	-0.4336	0.3598	-0.4546	0.3628
<i>Father no HS (Omitted)</i>				
Father HS	0.2060	0.1942	0.2298	0.1959
Father Some College	0.3638	* 0.2727	0.4023	* 0.2145
Father College	0.2850	0.2238	0.3186	0.2257
Father Advanced	0.2760	0.2890	0.2841	0.2914
Father Don't know	0.4737	0.2994	0.4946	0.3020
Father Missing	0.6931	0.4618	0.5744	0.4657
<i>Grade 7-10 (Omitted)</i>				
grade9-10	-0.0769	0.2383	0.1436	0.2403
grade11-12	-0.0093	0.2629	0.3214	0.2652
Allowance	0.0056	** 0.0053	0.0026	0.0053
Constant	-0.5198	0.6617	3.6270	** 0.6673
Observations:				2355

Table 3: Peer effects in fast food consumption without correlated effects (Continued)

*** significant at 1% ** significant at 5% * significant at 10%

Variable	OLS	S.E	G2SLS	S.E
endogenous social effect	0.1548 ***	0.0344	0.1102	0.3929
Individual Specific Characteristics				
female	-0.0847	0.0789	-0.0838	0.0793
age	0.1315 ***	0.0461	0.1345 **	0.0531
white	-0.0602	0.1127	-0.0618	0.1137
Mother present	-0.0358	0.0989	-0.0375	0.1000
<i>Mother no HS (Omitted)</i>				
Mother HS	-0.0455	0.1202	-0.0436	0.1214
Mother Some College	-0.0210	0.1340	-0.0161	0.1409
Mother college	-0.0137	0.1425	-0.0142	0.1426
Mother Advanced	-0.0353	0.1877	-0.0365	0.1880
Mother Don't know	-0.2124	0.2059	-0.2137	0.2062
<i>Father no HS (Omitted)</i>				
Father HS	0.2682 **	0.1167	0.2689 **	0.1169
Father Some College	0.1971	0.1338	0.1956	0.1344
Father College	0.2592 *	0.1381	0.2577 *	0.1388
Father Advanced	0.1294	0.1760	0.1275	0.1769
Father Don't know	0.2393	0.1750	0.2419	0.1766
Father Missing	0.0477	0.2331	0.0515	0.2355
<i>Grade 7-10 (Omitted)</i>				
grade9-10	-0.0776	0.2183	-0.0789	0.2186
grade11-12	0.1269	0.2526	0.1249	0.2533
Allowance	0.0074 **	0.0031	0.0075 **	0.0032

Table 4: Peer effects in fast food consumption with correlated effects

*** significant at 1% ** significant at 5% * significant at 10%

Variable	OLS	S.E	G2SLS	S.E
Exogenous social Effects				
female	-0.1071	0.1285	-0.1108	0.1325
age	0.0316	0.0718	0.0359	0.0811
white	-0.0055	0.1694	-0.0159	0.1922
Mother present	0.1008	0.1707	0.1077	0.1811
<i>Mother no HS (Omitted)</i>				
Mother HS	-0.2977	0.1913	-0.3000	0.1924
Mother Some College	-0.3825 *	0.2168	-0.3881 *	0.2224
Mother college	-0.2935	0.2213	-0.3080	0.2549
Mother Advanced	-0.5954 *	0.3112	-0.5774 *	0.3490
Mother Don't know	-0.4150	0.3610	-0.4038	0.3741
<i>Father no HS (Omitted)</i>				
Father HS	0.2999	0.1964	0.3299	0.3219
Father Some College	0.3890 *	0.2139	0.4051	0.2561
Father College	.3068	0.2263	0.3298	0.3028
Father Advanced	0.2171	0.2953	0.2340	0.3305
Father Don't know	0.5358 *	0.3001	0.5683	0.4140
Father Missing	0.7692 *	0.4640	0.7769 *	0.4690
<i>Grade 7-10 (Omitted)</i>				
grade9-10	0.0104	0.2773	0.0057	0.2804
grade11-12	-0.0396	0.3388	-0.034	0.3422
Allowance	0.0043	0.0054	0.0048	0.0069
Observations:				2339

Table 5: Peer effects in fast food consumption with correlated effects (Continued)

*** significant at 1% ** significant at 5% * significant at 10%

Variable	Female G2SLS	S.E	Male G2SLS	S.E
endogenous social effect	0.5761 *	0.3262	-0.1398	0.3051
Individual Specific Characteristics				
age	0.0749	0.0698	0.2030 **	0.0678
white	-0.0426	0.1665	-0.0419	0.1667
Mother present	-0.1192	0.1547	0.0199	0.1403
<i>Mother no HS (Omitted)</i>				
Mother HS	0.1714	0.1728	-0.2456	0.1908
Mother Some College	0.0701	0.1960	-0.1491	0.2182
Mother college	0.0010	0.2085	-0.0935	0.2088
Mother Advanced	-0.0842	0.2828	-0.0633	0.2714
Mother Don't know	-0.4552	0.3408	-0.1900	0.2911
<i>Father no HS (Omitted)</i>				
Father HS	0.3554 **	0.1708	0.1780	0.1729
Father Some College	0.2089	0.1987	0.2385	0.1996
Father College	0.6048 **	0.2134	0.0223	0.2008
Father Advanced	0.4624 *	0.2699	0.0004	0.2520
Father Don't know	0.6665 **	0.2613	-0.1427	0.2528
Father Missing	0.2621	0.3442	-0.3995	0.3696
<i>Grade 7-10 (Omitted)</i>				
grade9-10	-0.2377	0.3143	0.2124	0.3112
grade11-12	-0.0395	0.3707	0.4060	0.3592
Allowance	-0.0006	0.0045	0.0159 ***	0.0047

Table 6: Peer effects in fast food consumption by gender and with correlated effects (Continued)

*** significant at 1% ** significant at 5% * significant at 10%

Variable	Female G2SLS	S.E	Male G2SLS	S.E
Exogenous social Effects				
age	-0.0503	0.1349	0.0243	0.1034
white	-0.0443	0.2596	0.2181	0.2985
Mother present	0.0985	0.2760	-0.2433	0.2231
<i>Mother no HS (Omitted)</i>				
Mother HS	-0.1721	0.2594	-0.6395 **	0.2956
Mother Some College	0.1042	0.2839	-0.6187 **	0.3414
Mother college	-0.3932	0.3244	-0.4798	0.3321
Mother Advanced	0.0952	0.4207	-0.9986 **	0.4750
Mother Don't know	0.5527	0.6216	-1.155 **	0.5014
<i>Father no HS (Omitted)</i>				
Father HS	-0.3243	0.3856	0.5118 **	0.2850
Father Some College	-0.4134	0.3346	0.4923	0.3054
Father College	-0.4585	0.4043	0.4819	0.3162
Father Advanced	-0.8134 *	0.4928	0.4229	0.3928
Father Don't know	-0.0651	0.5776	0.5545	0.4728
Father Missing	-0.5949	0.7093	0.3824	0.7807
<i>Grade 7-10 (Omitted)</i>				
grade9-10	0.3697	0.4554	-0.5347	0.4027
grade11-12	0.3280	0.5456	-0.6166	0.5124
Allowance	0.0048	0.0076	0.0004	0.0086
Observations:		1164		1159

Table 7: Peer effects in fast food consumption by gender and with correlated effects (Continued)

*** significant at 1% ** significant at 5% * significant at 10%