Peer Effects, Fast Food Consumption and Adolescent Weight Gain *

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Abstract

This paper aims at opening the black box of peer effects in adolescent weight gain. Using Add Health data on secondary schools in the U.S., we investigate whether these effects partly flow through the *eating habits* channel. Adolescents are assumed to interact through a friendship social network. We first propose a social interaction model of fast food consumption. Our approach allows to control for correlated effects at the network level and to solve the simultaneity (reflection) problem. We exploit results by Bramoullé, Djebbari and Fortin (2009) which show that if there are two agents who are separated by a link of distance 3 within a network (*i.e.*, two adolescents who are not friends but are linked by two friends), peer effects are identified. The model is estimated using maximum likelihood and generalized 2SLS strategies. We also estimate a panel dynamic weight gain production function relating an adolescent's Body Mass Index (BMI) to his current fast food consumption and his lagged BMI level. Results show that there are positive significant peer effects in fast food consumption among adolescents belonging to a same friendship school network. The estimated social multiplier is 1.59. Our results also suggest that, at the network level, an extra day of weekly fast food restaurant visits increases BMI by 2.4%, when peer effects are taken into account.

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1 Introduction

For the past few years, obesity has been one of the major concerns of health policy makers in the U.S. It has also been one of the principal sources of increased health care costs. In fact, the increasing trend in children's and adolescents' obesity has raised the annual obesity-related hospital costs for this part of the population to $127 million per year. Obesity is also associated with increased risk of reduced life expectancy as well as with serious health problems such as type 2 diabetes (Ford et al., 1997; Maggio and Pi-Sunyer, 2003), heart disease (Li et al., 2006; Calabr et al., 2009) and certain cancers (Abu-Abid et al., 2002; Calle, 2007), making obesity a real public health challenge.

Recently, a growing body of the health economics literature has tried to look into the obesity problem from a new perspective using a social interaction framework. The evidence suggests the presence of strong peer effects in weight gain. Christakis and Fowler (2007), Trogdon et al. (2008) and Renna et al. (2008) seem to be unanimously pointing to the *social multiplier* as an important element in the obesity epidemics.[1] A social multiplier may amplify, at the aggregate level, the impact of any shock that affects obesity at the individual level. This is so because the aggregate effect incorporates, in addition to the sum of the individual direct effects, positive indirect peer effects stemming from social interactions.[2]

While the presence of the social multiplier in weight gain has been widely researched, the literature on the mechanisms by which this multiplier flows is still scarce. Indeed, most of the relevant literature attempts to estimate the relationship between variables such as an individual's Body Mass Index (BMI) and his average peers' BMI, without exploring the channels at source of this potential linkage.[3] The aim of this paper is to go beyond the black box approach of peer effects in weight gain and try to identify one crucial mechanism through which peer effects in adolescence overweight may flow: *eating habits* (as

[1] While Cohen-Cole and Fletcher (2008) found that there is no evidence of peer effects in weight gain, most of the literature is consistent with the presence of these effects.

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

[3] One recent exception is Yakusheva et al. (2010) who look at peer effects in weight gain and in weight management behaviours such as eating and physical exercise, using randomly assigned pairs of roommates in freshman year.

proxied by fast food consumption).[4]

Three reasons justify our interest in eating habits in analyzing the impact of peer effects on teenage weight gain. First of all, there is important literature that points to eating habits as an important component in weight gain (*e.g.*, Levitsky et al., 2004; Niemeier et al., 2006; Rosenheck, 2008).[5] Second, one suspects that peer effects in eating habits are likely to be important in adolescence. Indeed, at this age, youngsters have increased independence in general and more freedom as far as their food choices are concerned (Rolfes and Whitney, 1996). Usually vulnerable, they often compare themselves to their friends and may alter their choices to conform to the behaviour of their peers. Therefore, unless we scientifically prove that obesity is a virus,[6] it is counter intuitive to think that one can gain weight by simply interacting with an obese person.[7] This is why we are inclined to think that the presence of real peer effects in weight gain can be estimated using behavioural channels such as eating habits. Third, our interest in peer effects in youths' eating habits is policy driven. There has been much discussion on implementing tax policies to address the problem of obesity (*e.g.*, Jacobson and Brownell, 2000; Marshall, 2000; Kim and Kawachi, 2006; Caraher and Cowburn, 2007). As long as peer effects in fast food consumption is a source of externality that may stimulate overweight among adolescents, it may be justified to introduce a consumption tax on fast food. The optimal level of this tax will depend, among other things, on the social multiplier of eating habits, and on the causal effect of fast food consumption on adolescent weight.

In order to analyze the impact of peer effects in eating habits on weight gain, we propose a two-equation model. The first equation relates the teenager's fast food consumption to his reference group's

[4] Another potentially important channel is physical activity (Trogdon et al., 2008).

[5] An indirect evidence of the relationship between eating habits and weight gain come from the literature on the (negative) effect of fast food prices on adolescent BMI (see Chou et al., 2005; Powell et al., 2007; Auld and Powell, 2008; Powell and Bao, 2009). See also Cutler et al. (2003) which relates the declining relative price of fast food and the increase in fast food restaurant availability over time to increasing obesity in the U.S.

[6] We acknowledge that some recent studies have pointed that obesity might be partially due to a virus ad-36 (see Rogers et al., 2007).

[7] Of course, having obese peers may influence a student's tolerance for being obese and therefore his weight management behaviours.

mean fast food consumption (*endogenous peer effect*), his individual characteristics, and his reference group's mean characteristics (*contextual peer effects*). This *linear-in-means* equation also provides an estimate of the social multiplier in fast-food consumption. The second equation is a panel dynamic production function that relates the teenager's BMI to his current fast food consumption and his lagged BMI level. The system of equations thus allows us to evaluate the impact of an eating habits' exogenous shock on weight gain, when peer effects on fast food consumption are taken into account.

Estimating our system of equations raises serious econometric problems. It is well known that the identification of peers effects (first equation) is a challenging task. These identification issues were pointed out by Manski (1993) and discussed among others by Moffit (2001), Brock and Durlauf (2001), and Blume et al. (2010). On one hand, (endogenous + contextual) peer effects must be identified from *correlated* (or confounding) factors. For instance, students in a same friendship group may have similar eating habits because they are alike or face a common environment. On the other hand, simultaneity between an adolescent's and his peers' behaviour (referred to as *the reflection problem* by Manski) makes it difficult to identify separately the endogenous peer effect and the contextual effects. This later task is important since the endogenous peer effect is the only source of a social multiplier.

We use a new approach based on Bramoullé, Djebbari and Fortin (2009) and Lee, Liu and Lee (2010) to address these identification problems and to estimate the peer effects equation. First, we assume that in their fast food consumption decisions, adolescents interact through a *friendship network*. Each school is assumed to form a network. School fixed effects are introduced to capture correlated factors associated with network invariant unobserved variables (e.g., similar preferences due to self-selection in schools, same school nutrition policies, distance from fastfood restaurants). The structure of friendship links within a network is allowed to be stochastic and endogenous but is strictly exogenous, conditional on the school fixed effects and observable individual and contextual variables. To solve the reflection problem, we exploit results by Bramoullé et al. (2009) who show that if there are at least two agents who are separated by a link of distance 3 within a network (*i.e.*, there are two adolescents in a school

who are not friends but are linked by two friends), both endogenous and contextual peer effects are identified. The intuition is quite simple when there are no school fixed effects. In this case, identification conditions are less restrictive: peer effects are identified when a friend of an adolescent's friend is not his friend (link of distance 2, or intransitive triad). This provides exclusion restrictions in the model. More specifically, the friends' friends mean characteristics can be used as instruments for the friends' mean fast food consumption. Finally, we exploit the similarity between the linear-in-means model and the spatial autoregressive (SAR) model. The model is estimated using a maximum likelihood (ML) approach as in Lee et al. (2010) and Lin (2010). We also estimate the model with a distribution free approach: generalized spatial two-stage least square (GS-2SLS) proposed in Kelejian and Prucha (1998) and refined in Lee (2003).

The estimation of the dynamic weight gain production function (second equation) also raises some challenging issues as fast food consumption and past BMI level are likely to be endogenous variables. In order to deal with these problems, we follow Arellano and Bond (1991) by first differencing the equation to eliminate the individual effects and by exploiting the orthogonality conditions that exist between lagged values of BMI and the current disturbances to generate instruments.

To estimate our model, we use three waves 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 behaviour is depicted through the reported frequency (in days) of fast food restaurant visits in the past week. Results suggest that there is a positive significant peer effect in fast food consumption among adolescents in general. Based on the ML approach, the estimated social multiplier is 1.59. Moreover, the production function estimates indicate that there is a positive significant impact of fast food consumption on BMI. Combining these results, we find that, at the network level, an extra day of fast food restaurant visits per week increases BMI by 2.4% on average within a year.

The remaining parts of this paper will be laid out as follows. Section 2 provides a critical survey of

the literature on peer effects in obesity as well as its decomposition into the impact of peer effects on fast food consumption and the impact of fast food consumption on obesity. Section 3 presents our two-equation model and our estimation methods. In section 4, we give a brief overview of the Add Health Survey and we provide descriptive statistics of the data we use. In section 5, we discuss estimation results. Section 6 concludes.

2 Previous literature

In recent years, a number of studies found strong "social networks effects" in weight outcomes. In a widely debated article, Christakis and Fowler (2007), using a 32-year panel dataset on adults from Framingham, Massachusetts and based on a logit specification, found that an individual's probability of becoming obese increased by 57% if he or she had a friend who became obese in a given interval. However, their analysis has been criticized for suffering from a number of limitations (see Cohen-Cole and Fletcher, 2008; Lyons, 2011; Shalizi and Thomas, 2011).[8] In particular, it ignores potential spurious correlations between two friends' BMI resulting from the fact that they are exposed to a same environment. Both Shalizi and Thomas (2011) and Lyons (2011) show that the relying on link asymmetries does not rule out shared environment as it claims. Also, the simultaneity problem between these two outcomes is not directly addressed by allowing the peer's obesity to be endogenous. Moreover, by introducing lagged obesity variables, it only partly takes into account the problem of selection that may occur as obese individuals may have a higher probability to become friends (for details see pages 217-218 in Shalizi and Thomas, 2011). Finally, by focusing on dyads over time, it introduces an upward bias resulting from the *unfriending* problem as defined by Noel and Nyhan (2011).[9] The basic idea behind this argument is that people who are alike (homophilious) are more likely to maintain social ties.[10]

[8] For a response to these criticisms and others, see Fowler and Christakis (2008), Christakis and Fowler (forthcoming) and VanderWeele (2011).

[9] Unfriending means people who stop being friends with each other.

[10] Steeg and Galstyan (2011) show that there is a test for ruling out homophily related to the use of longitudinal social networks. The intuition behind the test being that if an individual reproduces the same sequence of events as his friend, it is unlikely that homophily is a source of this replication.

Using Add health data, Trogdon et al. (2008) include school fixed effects to account for the fact that students in a same school share a same surrounding. They also estimate their BMI peer model with an instrumental variable approach. They use information on friends' parents' obesity and health and friends' birth weight as instruments for peers' BMI. They find that an one point increase in peers' average BMI increases own BMI by 0.52 point. Using a similar approach and based on Add Health dataset, Renna et al. (2008) also find positive peer effects. These effects are significant for females only (= 0.25 point). These analyses raise a number of concerns though. In particular, they assume no contextual variables reflecting peers' mean characteristics. This rules out the reflection problem by introducing non-tested restriction exclusions. As a result, the peer effect estimates may be inconsistent. Moreover, it is not clear that their instruments is truly exogenous as peers' parents obesity status or health may be correlated with unobserved variables influencing own BMI. Also, their instruments are *ad hoc* as they are not explicitly derived from the structural form of the model. In our approach, we introduce school fixed effects as well as, for each individual variable, the corresponding contextual variable at the peer level. We can thus identify both endogenous and contextual peer effects. In addition, our instruments are explicitly derived from the structural model.

Using the same dataset, Cohen-Cole and Fletcher (2008) exploit panel information (wave II in 1996 and wave III in 2001) for adolescents for whom at least one of same-sex friend is also observed over time. Compared with Christakis and Fowler's approach, their analysis introduces time invariant and time dependent environmental variables (at the school level). Friendship selection is controlled for by individual fixed effects. The authors find that peer effects are no longer significant with this specification. As in Trogdon et al. (2008) , their analysis ignores contextual variables, contrary to our approach. Moreover, the friendship network they used in estimations is incomplete, which may underestimate the endogenous peer effect (see Stinebrickner and Stinebrickner, 2006).

All the studies discussed up to this point focus on peer effects in weight outcomes without analyzing quantitatively the mechanisms by which they may occur. The general issue addressed in this paper is

whether the peer effects in weight gain among adolescents partly flow through the *eating habits* channel. This raises in turn two basic issues: a) are there peer effects in fast food consumption?, and b) is there a link between weight gain (or obesity) and fast food consumption? In this paper, we address both issues. The literature on peer effects in eating habits (first issue) is recent and quite limited. In a medical experimental context, Salvy et al. (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 a recent natural experiment, Yakusheva et al. (2010) estimate peer effects in explaining weight gain among freshman girls using a similar set up but in school dormitories. Also, they test whether some of the student's weight management behaviours (*i.e.*, eating habits, physical exercise, use of weight loss supplements) can be predicted by her randomly assigned roommate's behaviours. Their results provide evidence of the presence of *negative* peer effects in weight gain. Their results also suggest *positive* peer effects in eating habits, exercise and use of weight loss supplements.

Two caveats of these two studies are their focus on girls and their limited sample (*e.g.*, recruited participants, freshman level students). Moreover their estimates are likely to underestimate social interactions effects as co-eaters or roommates do not reflect the *true* social network influencing students' weight management behaviours (Stinebrickner and Stinebrickner, 2006). Finally, these studies do not estimate the causal links between behaviours and weight gain. Our paper finds its basis in this literature as well as the literature on peer effects and obesity discussed above. However, while both works by Salvy et al. (2008) and Yakusheva et al. (2010) rely upon experimental data, we use observational non-experimental data. Thus, peers are not limited to assigned dyads. Rather, they are considered to have social interactions within a school network. This allows for the construction of a social interaction matrix that reflects how social interaction between adolescents in schools occurs in a more realistic setting (as in Trogdon et al., 2008; Renna et al., 2008). An additional originality of our paper lies in the fact that it relies upon a structural (linear-in-means) approach when relating an adolescent's behaviour to that of

his peers. Also, the analogy between the forms of the linear-in-means model and the spatial autoregressive (SAR) model allows us to exploit the particularities of this latter model, in particular the natural instruments that are derived from its structural form.

Regarding the second issue, *i.e.*, the relationship between weight gain (or obesity) and fast food consumption, it is an empirical question that is still on the debate table.[11] 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 (see for a survey, Rosenheck, 2008).[12]

The economic literature reveals to be conservative with respect to this question. It focuses the impact of "exposure" to fast food on obesity. Dunn (2008), using an instrumental variable approach, investigates the relationship between fast food availability and obesity. He finds that an increase in the number of fast food restaurants has a positive effect on the BMI. Similarly, Currie et al. (2010) find evidence that proximity to fast food restaurants has a significant effect on obesity for 9th graders. On the other hand, Chen et al. (2009) found a small but statistically significant effect in favour of a relationship between BMI values and the density of fast food restaurants. Finally, Anderson and Matsa (2011), exploiting the placement of Interstate Highways in rural areas to obtain exogenous variation in the effective price of restaurants, did not find any causal link between restaurant consumption and obesity.

The factors underlying fast food consumption were also investigated. Jeffery and 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 BMI 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 et al. (2005) find a strong positive correlation between exposure to fast food restaurant advertising and

[11] The literature on the impact of physical activity on obesity is also inconclusive. For instance, Berentzen et al. (2008) provide evidence that decreased physical activity in adults does not lead to obesity.

[12] For instance Bowman et al. (2004) finds that children who consumed fast food consumed more total energy.

the probability that children and adolescents 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 et al., 2007). More generally, Cutler et al. (2003) and Bleich et al. (2008) argue that the increased calorie intake (*i.e.*, eating habits) plays a major role in explaining current obesity rates. Importantly, weight gain prior to adulthood set the stage for weight gain in adulthood.

While most of the economics literature analyses the relationship between adolescents' fast food consumption and their weight gain using an indirect approach (*i.e*, effect to fast food exposure), we adopt a direct approach in this paper. More precisely, we estimate a dynamic model of weight gain as a function of fast food consumption and lagged weight gain. In order to account for the endogeneity of regressors, we follow instrumental methods that were developed in the econometrics literature to estimate panel dynamic models.

3 Structural econometric model

In this section, we first propose a linear-in-means peer effects model of the adolescent's fast food consumption (first equation) and discuss the econometric methods we use to estimate it. We then present our dynamic weight gain production function which relates the adolescent's BMI level to his fast food consumption (second equation).

3.1 A structural model of peer effects in fast food consumption

Suppose that we have a set of N adolescents i that are partitioned in a set of L networks. A network is defined as a structure (*e.g.*, school) in which adolescents are potentially tied by a friendship link. Each adolescent i in his network has a set of nominated friends N_i of size n_i that constitute his reference group (or peers). We assume that i is excluded from his reference group. Since peers are defined as nominated

friends, the number of peers will not be the same for every network member. Let $\mathbf{G}_l$ ($l = 1, \ldots, L$) be the social interaction matrix for a network l. Its element g_{lij} takes a value of $\frac{1}{n_i}$ when i is friend with j, and zero otherwise.[13] We define y_{li} as the fast food consumed by adolescent i in network l, x_{li} represents the adolescent i's observable characteristics, $\mathbf{y}_l$ the vector of fast food consumption in network l, and $\mathbf{x}_l$ is the corresponding vector for individual characteristics. To simplify our presentation, we look at only one characteristic (*e.g.*, adolescent's mother education).[14] The correlated effects are captured through network fixed effects (the α_l's). They take into account unobserved factors such as preferences of school, school nutrition policies, or presence of fast food restaurants around the school. The ε_{li}'s are the idiosyncratic error terms. They capture i's unobservable characteristics that are not invariant within the network. Formally, one can write the linear-in-means model for adolescent i as follows:

$$y_{li} = \alpha_l + \beta \frac{\sum_{j \in N_i} y_{lj}}{n_i} + \gamma x_{li} + \delta \frac{\sum_{j \in N_i} x_{lj}}{n_i} + \varepsilon_{li}, \tag{1}$$

where $\frac{\sum_{j \in N_i} y_{lj}}{n_i}$ and $\frac{\sum_{j \in N_i} x_{lj}}{n_i}$ are respectively his peers' mean fast food consumed and characteristics.[15] In the context of our paper, β is the *endogenous peer effect*. It reflects how the adolescent's consumption of fast food is affected by his peers' mean fast food consumption. It is standard to assume that $|\beta| < 1$. The *contextual peer effect* is represented by the parameter δ. It captures the impact of his peers' mean characteristic on his fast food consumption. It is important to note that the matrices of $\mathbf{G}_l$'s and the vectors of $\mathbf{x}_l$'s are stochastic but assumed strictly exogenous conditional on α_l, that is, $\mathbb{E}(\varepsilon_{li}|\mathbf{x}_l, \mathbf{G}_l, \alpha_l) = 0$. This assumption is flexible enough to allow for correlation between the network's unobserved common characteristics (*e.g.*, school's cafeteria quality) and observed characteristics (*e.g.*, mother's education).[16] Nevertheless, once we condition on these common characteristics, mother's education is assumed to be independent of i's idiosyncratic unobserved characteristics. Let $\mathbf{I}_l$ be the identity matrix for a network l and $\boldsymbol{\iota}_l$ the corresponding vector of ones, the structural model (1) for network l can be rewritten in matrix

[13] Therefore, the $\mathbf{G}_l$ matrix is row normalized.

[14] Later on, in section 3.1.1, we will generalize the model to account for many characteristics.

[15] This structural model can be derived from a choice-theoretic approach where each adolescent's fast food consumption is obtained from the maximization of his quadratic utility function which depends on his individual characteristics, his own fast food consumption and his reference group's mean fast food consumption and mean characteristics. This approach also assumes that social interactions have reached a noncooperative (Nash) equilibrium (see Blume et al., 2010).

[16] In this case $\mathbb{E}(\alpha_l|\mathbf{G}_l, \mathbf{x}_l) \neq 0$.

notation as follows:

$$\mathbf{y}_l \quad = \quad \alpha_l \boldsymbol{\iota}_l + \beta \mathbf{G}_l \mathbf{y}_l + \gamma \mathbf{x}_l + \delta \mathbf{G}_l \mathbf{x}_l + \boldsymbol{\varepsilon}_l, \;\; \text{for } \; l = 1, ..., L. \tag{2}$$

Note that model (2) is similar to a SAR model (*e.g.*, Cliff and Ord, 1981) generalized to allow for contextual and fixed effects (hereinafter referred to as the GSAR model). Since $|\beta| < 1$, $(\mathbf{I}_l - \beta \mathbf{G}_l)$ is invertible. Therefore, in matrix notation, the reduced form of the model can be written as:

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

where we use the result that $(\mathbf{I} - \beta \mathbf{G}_l)^{-1} = \sum_{k=0}^{\infty} \beta^k \mathbf{G}_l^k$, so that the vector of intercepts is $\alpha_l/(1-\beta)\boldsymbol{\iota}_l$, asuming no isolated adolescents.[17]

Equation (3) allows us to evaluate the impact of a marginal shock in α_l (*i.e.*, a common exogenous change in fast food consumption within the network) on an adolescent i's fast food consumption, when the endogenous peer effect is taken into account. One has $\partial(E(y_{li}|\cdot)/\partial \alpha_l = 1/(1-\beta)$. This expression is defined as the social multiplier in our model. When $\beta > 0$ (*strategic complementarities* in fast food consumption), the social multiplier is larger than 1. In this case, the impact of the shock is amplified by social interactions.

We then perform a panel-like *within* transformation to the model. More precisely, we average equation (3) over all students in network l and subtract it from i's equation. This transformation allows us to address problems that arise from the fact adolescents are sharing the same environment or preferences. Let $\mathbf{K}_l = \mathbf{I}_l - \mathbf{H}_l$ be the matrix that obtains the deviation from network l mean with $\mathbf{H}_l = \frac{1}{n_l}(\iota_l \iota_l')$. The network within transformation will eliminate the correlated effect α_l. Pre-multiplying (3) by $\mathbf{K}_l$ yields the reduced form of the model for network l, in deviation:

$$\mathbf{K}_l \mathbf{y}_l = \mathbf{K}_l (\mathbf{I}_l - \beta \mathbf{G}_l)^{-1}(\gamma \mathbf{I}_l + \delta \mathbf{G}_l)\mathbf{x}_l + \mathbf{K}_l (\mathbf{I}_l - \beta \mathbf{G}_l)^{-1} \boldsymbol{\varepsilon}_l. \tag{4}$$

[17] When an adolescent is isolated, that is, with an empty group of friends, his intercept is α_l.

3.1.1 Identification

Our structural model raises two basic identification problems.

- Simultaneity

Simultaneity between individual and peer behaviour, also called the *reflection problem* by Manski (1993), may prevent separating contextual effects from endogenous effects. This problem has been analyzed by Bramoullé et al. (2009) when individuals interact through social networks. Let us define $\mathbf{G}$ the block-diagonal matrix with the $\mathbf{G}_l$'s on its diagonal. Assume first the absence of fixed network effects (*i.e.*, $\alpha_l = \alpha$ for all l). In this case, Bramoullé et al. (2009) show that the structural parameters of the model (2) are identified if the matrices $\mathbf{I}$, $\mathbf{G}$, $\mathbf{G}^2$ are linearly independent. This condition is satisfied there are at least two adolescents who are separated by a link of distance 2 within a network. This means that they are not friends but have a common friend (intransitive triad).The intuition is that this provides exclusion restrictions in the model. More precisely, the friends' friends mean characteristics can served as instruments for the mean friends' fast food consumption. Of course, when fixed network effects are allowed, the identification conditions are more restrictive. Bramoullé et al. (2009) show that, in this case, the structural parameters are identified if the matrices $\mathbf{I}$, $\mathbf{G}$, $\mathbf{G}^2$ *and* $\mathbf{G}^3$ are linearly independent. This condition is satisfied when at least two adolescents are separated by a link of distance 3 within a network, i.e., we can find two adolescents who are not friends but are linked by two friends. In this case, $g^3_{lij} > 0$ while $g^2_{ij} = g_{ij} = 0$. Hence, no linear relation of the form $\mathbf{G}^3 = \lambda_0\mathbf{I} + \lambda_1\mathbf{G} + \lambda_2\mathbf{G}^2$ can exist. This condition holds in most friendship networks and, in particular, in the data we use.[18]

- Endogenous network formation

Since adolescents are not randomly assigned into schools, endogenous self-selection through networks may be the source of potentially serious biases in estimating (endogenous + contextual) peer effects.

[18]Identification fails, however, for a number of non trivial networks. This is notably the case for *complete bipartite networks*. In these graphs, the population of students is divided in two groups such that all students in one group are friends with all students in the other group, and there is no friendship links within groups. These include star networks, where one student, at the centre, is friend with all other students, who are all friends only with him.

Indeed, if the variables that drive this process of selection are not fully observable, correlations between unobserved network-specific factors and the regressors are potentially important sources of bias. In our approach, we assume that network fixed effects capture these factors. This is consistent with two-step models of link formation. Each adolescent joins a school in a first step, and forms friendship links with others in his school in a second step. In the first step, adolescents self-select into different schools with selection bias due to specific school characteristics. In a second step, link formation takes place within schools randomly or based on observable individual characteristics only. As shown above, network fixed effects are cancelled out through a panel-like within transformation.

Other types of correlated effects can occur for reasons other than common network factors. For instance, one can think of other uncommon unobserved reasons for which some people might group together. To account for this possibility we allow for error terms to be autocorrelated within networks so that our model structure becomes analogous to that of a generalized spatial autoregressive model with network autoregressive disturbances (hereinafter referred to as the GSARAR model). In this case, the error terms in (2) can be written as:

$$\boldsymbol{\varepsilon}_l \quad = \quad \rho \mathbf{G}_l \boldsymbol{\varepsilon}_l + \boldsymbol{\xi}_l, \tag{5}$$

where the innovations, $\boldsymbol{\xi_l}$, are assumed to be $i.i.d.(0, \sigma^2 \mathbf{I}_l)$ and $|\rho| < 1$. Given these assumptions, we can write:

$$\boldsymbol{\varepsilon}_l = (\mathbf{I}_l - \rho \mathbf{G}_l)^{-1} \boldsymbol{\xi_l}. \tag{6}$$

Allowing for many characteristics and performing a Cochrane-Orcutt-like transformation on the structural model in deviation, the latter is given by the following structural form:

$$\mathbf{K}_l \mathbf{M}_l \mathbf{y}_l = \beta \mathbf{K}_l \mathbf{M}_l \mathbf{G}_l \mathbf{y}_l + \mathbf{K}_l \mathbf{M}_l \mathbf{X}_l \boldsymbol{\gamma} + \mathbf{K}_l \mathbf{M}_l \mathbf{G}_l \mathbf{X}_l \boldsymbol{\delta} + \boldsymbol{\nu}_l, \tag{7}$$

where $\mathbf{X}_l$ is the matrix of adolescents' characteristics in the lth network, $\mathbf{M}_l = (\mathbf{I} - \rho \mathbf{G}_l)$ and $\boldsymbol{\nu}_l = \mathbf{K}_l \boldsymbol{\xi}_l$.

The elimination of fixed network effects using a *within* transformation leads to a singular variance matrix such that $E(\boldsymbol{\nu}_l\boldsymbol{\nu}_l' \mid \mathbf{X}_l, \mathbf{G}_l) = \mathbf{K}_l\mathbf{K}_l'\sigma^2 = \mathbf{K}_l\sigma^2$. To resolve this problem of linear dependency between observations, we follow a suggestion by Lee et al. (2010) and applied by Lin (2010). Let $[\mathbf{Q}_l \quad \mathbf{C}_l]$ be the orthonormal matrix of $\mathbf{K}_l$, where $\mathbf{Q}_l$ corresponds to the eigenvalues of 1 and $\mathbf{C}_l$ to the eigenvalues of 0. The matrix $\mathbf{Q}_l$ has the following properties: $\mathbf{Q}_l'\mathbf{Q}_l = \mathbf{I}_{n_l^*}$, $\mathbf{Q}_l\mathbf{Q}_l' = \mathbf{K}_l$ and $\mathbf{Q}_l'\iota = 0$, where $n_l^* = n_l - 1$ with n_l being the number of adolescents in the lth network. Pre-multiplying (7) by $\mathbf{Q}_l'$, the structural model can now be written as follows:

$$\mathbf{M}_l^*\mathbf{y}_l^* = \beta\mathbf{M}_l^*\mathbf{G}_l^*\mathbf{y}_l^* + \mathbf{M}_l^*\mathbf{X}_l^*\gamma + \mathbf{M}_l^*\mathbf{G}_l^*\mathbf{X}_l^*\delta + \boldsymbol{\nu}_l^*, \tag{8}$$

where $\mathbf{M}_l^* = \mathbf{Q}_l'\mathbf{M}_l\mathbf{Q}_l$, $\mathbf{y}_l^* = \mathbf{Q}_l'\mathbf{y}_l$, $\mathbf{G}_l^* = \mathbf{Q}_l'\mathbf{G}_l\mathbf{Q}_l$, $\mathbf{X}_l^* = \mathbf{Q}_l'\mathbf{X}_l$, and $\boldsymbol{\nu}_l^* = \mathbf{Q}_l'\boldsymbol{\xi}_l$. With this transformation, our problem of dependency between the observations is solved, since we have $E(\boldsymbol{\nu}_l^*\boldsymbol{\nu}_l^{*\prime} \mid \mathbf{X}_l, \mathbf{G}_l) = \sigma^2\mathbf{I}_{n_l^*}$.

Following Lee et al. (2010), we propose two approaches to estimate the peer effects model (8): a maximum likelihood approach (ML) and a generalized spatial two stage least squares (GS-2SLS) approach. The ML approach imposes more structure (normality) than GS-2SLS. Therefore, under some regularity conditions, ML estimators are more asymptotically efficient than GS-2SLS ones when the restrictions it imposes are valid.

3.1.2 Maximum Likelihood (ML)

Assuming that $\boldsymbol{\nu}_l^*$ is a n_l^*-dimensional normally distributed disturbance vector, the log-likelihood function is given by:

$$\ln \mathbb{L} \;=\; \frac{-n^*}{2}\ln\left(2\pi\sigma^2\right) \;+\; \sum_{l=1}^{L}\ln|\mathbf{I}_{n_l^*} - \beta\mathbf{G}_l^*| \;+\; \sum_{l=1}^{L}\ln|\mathbf{I}_{n_l^*} - \rho\mathbf{M}_l^*| \;-\; \frac{1}{2\sigma^2}\sum_{l=1}^{L}\boldsymbol{\nu}_l^{*\prime}\boldsymbol{\nu}_l^*, \tag{9}$$

where $n^* = \sum_{l=1}^{L} n_l^* = N - L$, and, from (8), $\boldsymbol{\nu}_l^* = \mathbf{M}_l^*(\mathbf{y}_l^* - \beta\mathbf{G}_l^*\mathbf{y}_l^* - \mathbf{X}_l^*\gamma - \mathbf{G}_l^*\mathbf{X}_l^*\delta)$. Maximizing (9) with respect to $(\beta, \boldsymbol{\gamma}', \boldsymbol{\delta}', \rho, \sigma)$ yields the maximum likelihood estimators of the model.[19] Interestingly, the

[19] For computational simplicity, one can concentrate the log-likelihood function (9) and maximize the concentrated log-likelihood function. See Lee et al. (2010) for more details.

ML method is implemented after the elimination of the network fixed effects. Therefore, the estimators are not subject to the incidental parameters problem that may arise since the number of fixed effects increases with the the size of the networks sample.

3.1.3 Generalized spatial two stage least squares (GS-2SLS)

To estimate the model (8), we also adopt a generalized spatial two-stage least squares procedure presented in Lee et al. (2010). This approach provides a simple and tractable numerical method to obtain asymptotically efficient IV estimators within the class of IV estimators. In the case of our paper this method will consist of a two-step estimation.[20] To simply the notation, Let $\mathbf{X}^*$ be a block-diagonal matrix with $\mathbf{X}^*{}_l$ on its diagonal, $\mathbf{G}^*$ be a block-diagonal matrix with $\mathbf{G}^*{}_l$ on its diagonal, and $\mathbf{y}^*$ the concatenated vector of the y_l^*'s over all networks.

Now, 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 $\mathbf{S} = \begin{bmatrix}\mathbf{X}^* & \mathbf{G}^*\mathbf{X}^* & \mathbf{G}^{*2}\mathbf{X}^*\end{bmatrix}$. In the first step, we estimate the following 2SLS estimator:

$$\hat{\boldsymbol{\theta}}_1 = (\tilde{\mathbf{X}}^{*\prime}\mathbf{P}\tilde{\mathbf{X}}^*)^{-1}\tilde{\mathbf{X}}^{*\prime}\mathbf{P}\mathbf{y}^*,$$

where $\hat{\boldsymbol{\theta}}_1$ is the first-step 2SLS vector of estimated parameters $(\hat{\boldsymbol{\gamma}}_1', \hat{\boldsymbol{\delta}}_1', \hat{\boldsymbol{\beta}}_1)$ of the structural model. This estimator is consistent but not asymptotically efficient within the class of IV estimators.

Now, in the second step, we estimate a 2SLS using a new matrix of instruments $\hat{\mathbf{Z}}$ given by:

$$\hat{\mathbf{Z}} = \left[\mathbf{G}^*\hat{\mathbf{y}}^* \quad \mathbf{X}^* \quad \mathbf{G}^*\mathbf{X}^*\right],$$

where $\mathbf{G}^*\hat{\mathbf{y}}^*$ is computed from the first-step 2SLS reduced form (pre-multiplied by $\mathbf{G}^*$):

$$\mathbf{G}^*\hat{\mathbf{y}}^* = \mathbf{G}^*(\mathbf{I} - \hat{\boldsymbol{\beta}}_1\mathbf{G}^*)^{-1}(\mathbf{X}^*\hat{\boldsymbol{\gamma}}_\mathbf{1} + \mathbf{G}^*\mathbf{X}^*\hat{\boldsymbol{\delta}}_\mathbf{1}).$$

[20] Note that for this particular case we impose $\rho = 0$ and thus $M_l = I_l$.

We then estimate:

$$\hat{\boldsymbol{\theta}}_2 = (\hat{\mathbf{Z}}'\tilde{\mathbf{X}}^*)^{-1}\hat{\mathbf{Z}}\mathbf{y}^*.$$

This estimator can be shown to be consistent and asymptotically best IV estimator. Its asymptotic variance matrix is given by $N[\mathbf{Z}'\tilde{\mathbf{X}}^*\mathbf{R}^{-1}\tilde{\mathbf{X}}^*\mathbf{Z}]^{-1}$. 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 and Prucha (1998), we assume that errors are homoscedastic. The estimation theory developed by Kelejian and Prucha (1998) under the assumption of homoscedastic errors does not apply if we assume heteroscedastic errors (Kelejian and Prucha, 2010).

3.2 A weight gain production function

In this section, we propose a weight gain production function that relates an adolescent's BMI in time t to his lagged BMI, his fast food consumption as well as his own characteristics in period t. Let y_{it}^b be an adolescent i's BMI level at time t, and y_{it}^f be the adolescent's fast food consumption. Then, for a given vector of characteristics $\tilde{\mathbf{x}}_{it}$, the weight gain production function can be formally expressed as follows (for notational simplicity we suppress l):

$$y_{it}^b = \pi_0 + \pi_1 y_{i,t-1}^b + \pi_2 y_{it}^f + \boldsymbol{\pi}_3\tilde{\mathbf{x}}_{it} + \eta_{it}, \tag{10}$$

where

$$\eta_{it} = \mu_i + \zeta_{it},$$

with μ_i representing the individual i's time-invariant error component (unobserved heterogeneity) and ζ_{it}, his idiosyncratic error that may change across t. We consider that μ_i is a fixed effect, $|\pi_1| < 1$ and the error ζ_{it} is serially uncorrelated. (A test of this latter assumption is provided in the empirical section). As discussed earlier, our interest in this production function goes beyond a mere association between fast food consumption and weight gain. We are particularly interested to analyze the magnitude of a change in BMI resulting from a common exogenous shock on fast food consumption within the network, when

peer effects are taken into account. Our two equation model allows us to compute this result. Partially differentiating (10) with respect to y_{it}^f and using the social multiplier $[= 1/(1-\beta)]$ yields the magnitude of a short run change in BMI (*i.e.*, for $y_{i,t-1}^b$ given) resulting from a common marginal shock on fast food consumption: $\partial E(y_{it}^b|\cdot)/\partial \alpha_l = \frac{\pi_2}{1-\beta}$. This expression entails two components: the impact of the fast food consumption on the BMI $(= \pi_2)$ and the multiplier effect $(= \frac{1}{1-\beta})$.

At this point it is important to mention that OLS estimates of (10) will not be consistent for two reasons. First, the adolescent's fast food consumption is not exogenously determined and may be affected by his own BMI. In addition, there may exist a correlation between lagged dependent variable and the error term due to the presence of a time-invariant error component. One way to resolve this issue is to apply a first difference. While such a transformation wipes out all individual time-invariant characteristics, it has the advantage of making the correlation between right hand side regressors and the error term easier to handle. Formally, assuming that all characteristics (except age) are time-invariant, the transformed model can be written as follows:

$$\Delta y_{it}^b = \tilde{\pi_0} + \pi_1 \Delta y_{i,t-1}^b + \pi_2 \Delta y_{it}^f + \Delta \zeta_{it}, \tag{11}$$

where Δ is the first difference operator. To resolve the problem of correlation between the right hand side variables and the error term one can instrument for $\Delta y_{i,t-1}^b$ and Δy_{it}^f. To instrument the lagged dependent variable we can either use $\Delta y_{i,t-2}^b$ or $y_{i,t-2}^b$ (Hsiao, 1981). Following the suggestion of Arellano and Bond (1991), we instrument it using y_{t-2}^b. We also use birth weight x_{bw}. High birth weight is associated overweight in adolescence (Gillman et al., 2003; Sorensen et al., 1997) it is therefore expected that it would affect y_{it}^b only through it impact on $y_{i,t-1}^b$. As for the fast food consumption variable Δy_{it}^f, valid instruments could be $y_{i1}^f, y_{i2}^f, y_{i3}^f, \ldots, y_{i,s-1}^f$ for an equation differenced at $t = s$. Unfortunately such information is not available in our dataset, we thus instrument it using birthweight and the strictly exogenous variables that we have used in our peer effects model.

4 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, neighbourhood, 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.[21] 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 (about 3000 students). Every student attending these selected schools answered the detailed questionnaire. 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 adolescents and is located in a rural area. Consequently, fast food consumption may be subject to downward bias if one accepts the argument that the fast food consumption among white adolescents is usually lower than that of black adolescents.[22]

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.[23] 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 drinks, desserts, *etc.*). This allows us to depict food consumption patterns of each adolescent and relate

[21] Adolescents were asked to nominate either 1 female friend and 1 male friend or 5 female friends and 5 male friends.

[22] It is unclear whether the black population consumes more fast food than the white population. Block et al. (2004) provides evidence that predominantly black neighbourhoods have relatively more fast food restaurants than predominantly white neighbourhoods.

[23] It includes all meals that are consumed at a fast food restaurant such as McDonald's, Burger King, Pizza Hut, Tacco Bell.

it to that of his peer group. In addition, the availability of friend nomination allows us to retrace school friends and thus construct friendship networks.[24] To estimate the weight gain production function, we considered information from wave I, wave II and wave III.

We exploit friends nominations to construct the network of friends. Thus, 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.[25]

4.1 Descriptive statistics

In our peer effects model, the dependent variable of interest is fast food consumption, as approximated by the reported frequency (in days) of fast food restaurant visits in the past 7days. Table 1 reports respectively the mean and the standard deviation of the endogenous variable, the covariates used 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 et al., 1996). 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.[26] Evening meals are the only time parents have the power to check on the quality/quantity of the food consumed. This coupled with the freedom to choose food leaves the door wide open for peer influence in fast food consumption.

The covariates of the fast food peer effect equation include the adolescent's personal characteristics, family characteristics as well as the corresponding contextual social effects.The personal characteristics

[24] In the saturation sample, all students are asked to name their friends.
[25] It is important to note that we do not impose symmetry in the network.
[26] These figures are not reported in the paper but can be provided upon request.

are gender, age, ethnicity (white or other) 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 for mother and father education. We observe that around 45% of mothers and fathers have at least some college education. To control further 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 in our the sample have a weekly allowance. At this point, it is important to highlight that since we use cross section data, we do not have to control for fast food prices as they are taken into account by network fixed effects. As for the weight gain production function, the dependent variable that we use is the variation in the BMI between waves two and three. The covariates are the fast food consumption, the lagged BMI (Table 2), age, and all time-invariant variables appearing in Table 1.

4.2 The Construction of the Graph Matrix

We construct a sub-matrix of graph for each school separately (matrice $\mathbf{G}_l$) and then we include all these sub-matrices in the block-diagonal matrix $\mathbf{G}$.[27] As we have no prior information about how social interaction takes place, we assume, as in most studies, that an adolescent is equally influenced by his nominated friends. Further, we assume this influence decreases with the number of friends. In each school we eliminate adolescents for which we have missing values. We allow the sub-matrices to contain adolescents who are isolated. Since these latter may be friends with other adolescents in the network, they may affect the network even if they claim not to have any friends at all. They also introduce variability that helps the identification of the model. We also do not impose symmetry on the $\mathbf{G}$ matrix. In a more general sense we allow for a "Twitter" rather than "Facebook" type of networking. This imposes less restriction on the social interaction and mimics better the social interaction.

As mentioned earlier, Bramoullé et al. (2009) show that, the structural parameters are identified if

[27] Following the previous literature and given the lack of information on this matter, we assume that there can be social interactions within each school but no interactions across schools.

the matrices **I**, **G**, $\mathbf{G}^2$ *and* $\mathbf{G}^3$ are linearly independent. One way check whether these four matrices are linearly independent as follows. First, vectorize each matrix, that is, stack its columns on top of each other to form a matrix L. Second, verify whether the matrix formed by concatenating these stacked vectors has rank four. An alternative more general diagnosis check for linear independence is to do the first step as above and compute $L'L$ and scale it into a correlation form. Then compute the Belsley, Kuh, and Welsch *condition index* by finding the square root of the ratio of the largest eigen value to the smallest eigen value. If this ratio is below 30, then collinearity is said not to be a problem and linear independance of the four matrices is verified. In our data, the reflection problem is solved since **I, G,** $\mathbf{G}^2$ and $\mathbf{G}^3$ are linearly independent and the condition index value is 2.21.

5 Results

5.1 Baseline: OLS peer effects estimates

We first estimate a naive OLS of the peer effects model where we regress the fast food consumption of an adolescent on the average fast food consumption of his peers, his individual characteristics as well as the average characteristics of his peers. We then apply a panel-like *within* transformation to account for correlated effects (OLS_w). It is clear that the estimates of naive OLS and OLS_w are inconsistent. The former ignores both correlated effects and simultaneity problems while the latter ignores simultaneity problems. However, they are reported to provide a baseline for this study.

Estimation results reported in Table 3 show that there is a positive significant peer influence in fast food consumption. According to the naive OLS estimates, an adolescent would increase his weekly frequency (in days) of fast food restaurant visits by 0.21 in response to an extra day of fast food restaurant visits by his friends. On average, this corresponds to an increase of 9% (= 0.21/2.33). OLS_w estimate is slightly lower (= 0.15, or 6.6%). This reduction in the estimated effect may partly be explained by the fact that adolescents in the same reference group tend to choose a similar level of fast food consumption

partly because they are alike or face a common environment. How can we compare these results to those obtained previously in the related literature? Although there are few studies that investigated the presence of peer effects in fast food consumption, a richer body of literature has investigated a tangent issue : obesity. In their paper Trogdon et al. (2008) show OLS results for peer effects in obesity of 0.30. Also, Renna et al. (2008) reports endogenous effects of 0.16 for OLS estimates. This makes our OLS estimates comparable to those obtained in the literature on obesity.

As for the individual characteristics they seem to be increasing in age, father education and weekly allowance. Turning our attention to the contextual peer effects, we notice that fast food consumption decreases with mean peers' mother's education and increases with mean peers' father's education. The former result indicates that friends' mother education negatively affects an adolescent's fast food consumption.

5.2 ML and GS-2SLS peer effects estimates

Next, we estimate our linear-in-means (or GSAR) model with school fixed effects and using ML. We then estimate a more general version of this model by allowing network autoregressive disturbances (GSARAR model). Also, given that ML approach imposes normality on the error term, we relax this assumption and estimate the model using a distribution free approach : GS-2SLS.

Estimation results displayed in Table 4 show a positive and statistically significant endogenous effect of 0.13 (or 5.5%) for the GSAR model. This effect is slightly smaller than the ones obtained in the previous section. However, based on the more general GSARAR model, the estimated autocorrelation coefficient is significant and negative. Since it is significant, this indicates that the GSAR model is rejected as a particular case of the GSARAR model. Also, the fact that it is negative may provide evidence that friendship might be for other purposes than having a common preference in the formation of friends for fast food

consumption.[28]

With the GSARAR specification, the endogenous peer effect remains statistically significant but increases to 0.37, suggesting that an adolescent would increase his weekly frequency (in days) of fast food restaurant visits by 0.37 (or 15.9%) in response to an extra day per week of fast food restaurant visits by his friends. The social multiplier associated with an exogenous increase in an adolescent fast food consumption is 1.59 ($= \frac{1}{1-0.37}$), which reflects a fairly strong endogenous peer effect.[29] The increase in the endogenous effect coefficient as a result of a GSARAR specification on the error term is comparable to the one obtained by Lin (2010) in an empirical application of such a model on peer effects in academic achievement. As for the magnitude of the endogenous effects, it remains lower than ones obtained in the literature on peer effects in obesity.[30] Recall that Trogdon et al. (2008) estimate for the endogenous effect is 0.52 using an instrumental approach.

When we relax the normality assumption, the endogenous effect resulting from GS-2SLS estimation reveals to be smaller than the one obtained by ML (0.11 instead of 0.13). However, it is no longer significant. This does not come as a surprise, as less structure is not without a cost in precision. This makes the GSARAR specification preferable to others, at least as long as the normality assumption is an appropriate assumption. To sum up, we can say that results in general are consistent with the hypothesis that fast food consumption is linked to issues of identity and friends (Story et al., 2002).

As for individual effects, they follow fairly the baseline model. Fast food consumption is positively associated with age and father's education as well as positively associated with weekly allowance.[31] Mother's education seems to have a negative but non significant impact on fast food consumption. It is important to note that while the general perception is that fast food is an *inferior* good, the empirical

[28] Lin (2010) also obtains a negative estimated autocorrelation parameter when her model takes endogenous and contextual effects into account.

[29] As suggested by Glaeser et al. (2003), large social multipliers tend to occur when the endogenous effect is 0.33 or more.

[30] One possible explanation is that we are estimating peer effects using one potential behavioural channel.

[31] McLellan et al. (1999) found results pointing in a similar direction and suggest that limiting pocket money may be a good way to promote healthy adolescent behaviour.

evidence suggests that there is a positive income elasticity (McCracken and Brandt, 1987; Jekanowski et al., 2001; Aguiar and Hurst, 2005). Thus, the positive relation between fast food consumption and allowance is therefore in line with the positive relation between income and fast food consumption.

Turning our attention to the contextual social effects, fast food consumption increases with mean peers' father's education and decreases with mean peers' mother's education. This suggests that adolescents are perhaps more influenced by their friends' mothers than their own.

5.3 Weight gain production function estimates

Estimation results presented in the earlier sections are consistent with the presence of peer effects in fast food consumption. Nevertheless, we still need to provide evidence of the presence of a relationship between fast food consumption and weight gain. In this section we report estimates of the weight gain production function presented earlier. As noted above, the variables we used to instrument the fast food consumption variable Δy_{it}^{f} are the instruments previously used in the fast food consumption model.

Results from the Arellano and Bond (1991) estimator are reported in Table 5. Note that since the model is estimated using a first differencing (fixed effects) approach, parameters associated with time-invariant covariates are not identified and therefore do not appear in the table (the constant is an estimate of age parameter). In line with our expectations, results reveal a positive significant impact of a change in fast food consumption on the BMI level. An extra day of fast food restaurant visits per week increases weight by 0.36 BMI points (or by 1.54%) within a year.[32]

The presence of a causal link between fast food consumption and BMI does not come as a surprise since previous findings have been pointing in this direction (Levitsky et al., 2004; Niemeier et al., 2006; Rosenheck, 2008). Somewhat surprisingly, lagged BMI level has a negative effect on current BMI level

[32]We investigated the possibility of an omitted variable bias by introducing three (endogenous) physical exercise variables from Add Health in the weight gain equation: exercising activities, active sports, and recreational sports. None of these variables were significant and the fast food estimated effect (= 0.34), while a little smaller, was quite robust to this modification.

($= -0.727$). This suggests that an exogenous shock on weight gain has a stronger effect on BMI in the short term than in the long term. This may partly be explained by the fact that given a past increase in his BMI an adolescent may be induced to adopt more healthy eating habits.

The consistency of our estimates rely heavily on the validity of the instruments that are used. In order to test the validity of the instruments a Sargan test is computed. The test statistic reported in Table 5 indicates that we do not reject the joint null hypothesis that the instruments are valid and that the over-identification restrictions are satisfied. Observe that the Sargan test provides an (indirect) test for serial autocorrelation of the idiosyncratic error ζ_{it}. Indeed, the variable y^b_{t-2} would not be a valid instrument under serial autocorrelation. Arellano and Bond (1991) suggest to use this test when the length of the panel is too short to perform a direct serial autocorrelation test (*e.g.*, when $T = 3$ as in our case).

Combining the impact of fast food on weight gain with the social multiplier, our results suggest that, within a network, an extra day of fast food restaurant visits per week lead to a BMI increase of 0.57 points ($\frac{0.36}{1-0.37}$), or 2.4% on average, within a year. These results highlight the role of peer effects in fast food consumption as one of the transmission mechanisms through which weight gain is amplified.

6 Conclusion

This paper investigates whether peer effects in adolescent weight gain partly flow through the eating habits channel. We first attempt to study the presence of significant endogenous peer effects in fast food consumption. New methods based on spatial econometric analysis are used to identify and estimate our model, under the assumption that individuals interact through a friendship social network. Our results indicate that an increase in his friends' mean fast food consumption induces an adolescent to increase his own fast food consumption. This peer effect amplifies through a social multiplier the impact of any exogenous shock on fast food consumption. Our estimated social multiplier is 1.59.

We also estimate a dynamic weight gain production function which relates the adolescent's Body Mass Index to his fast food consumption. Results are in line with our expectations; they reveal a positive significant impact of a change in fast food consumption on the change in BMI. Specifically, a one-unit increase in the weekly frequency (in days) of fast food consumption produces an increase in BMI by 1.5% within a year. This effect reaches 2.4% when the social multiplier is taken into account. Coupled with the reduction in the relative price of fast food and the increasing availability of fast food restaurants over time, the social multiplier could exacerbate the prevalence of obesity in the years to come. Conversely, this multiplier may contribute to the decline of the spread of obesity and the decrease in health care costs, as long as it is exploited by policy makers through tax and subsidy reforms encouraging adequate eating habits among adolescents.

There are many possible extensions to this paper. From a policy perspective, it would be interesting to investigate the presence of peer effects in physical activity of adolescents. A recent study by Charness and Gneezy (2009) finds that there is room for intervention in peoples' decisions to perform physical exercise through financial incentives. It would be thus valuable to investigate whether there is a social multiplier that can be exploited to amplify these effects. Furthermore, in the same way, it would be interesting to study the presence of peer effects weight perceptions. So far, most of the peer effects work has focused mainly on outcomes (BMI). At the methodological level, a possible extension would be to relax the normality assumption and to assume a Poisson or a Negative Binomial distribution to account for the count nature of the consumption data at hand. As far as we know, no work has been carried out in this area. Finally, it would be most useful to develop a general approach that would allow same sex and opposite sex peer effects to be different for both males and females.

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Table 1: Descriptive Statistics

Variable	Mean	S.D
Fast Food Consumption[a]	2.33	1.74
Female	.50	.50
Age	16.36	1.44
White	.57	.49
Black	.15	.34
Asian	.01	.09
Native	.13	.33
Other	.14	.35
Mother Present	.85	.35
Mother Education		
No high school degree	.15	.35
High school/GED/Vocational Instead of high school	.36	.48
Some College/Vocational After high school	.21	.39
College	.18	.38
Advanced Degree	.06	.24
Don't Know	.04	.20
Father Education		
No high school degree	.16	.36
High school/GED/Vocational Instead of high school	.33	.47
Some College/Vocational After high school	.17	.37
College	.18	.38
Advanced Degree	.08	.26
Don't Know	.06	.24
Missing	.02	.16
Grade 7-8	.11	.32
Grade 9-10	.27	.44
Grade 11-12	.62	.48
Allowance per week	8.28	11.65
Observations:		2355

[a]Frequency (in days) of fast food restaurant visits in the past week.

Table 2: Body Mass Index

Variable	Mean	S.D
Bmi (wave1)	22.81	4.59
Bmi (wave2)	23.24	4.76
Bmi (wave3)	26.78	6.31
Birthweight	7.40	1.38

Table 3: Peer effects in fast food consumption

	OLS			OLSw		
	Coef.		**S.E**	**Coef.**		**S.E**
Endogenous Peer Effects	0.2078	***	0.0331	0.1548	***	0.0344
Individual Characteristics						
Female	-0.0721		0.0787	-0.0847		0.0789
Age	0.1559	***	0.0434	0.1315	***	0.0461
White	-0.1076		0.0940	-0.0602		0.1127
Mother Present	-0.0152		0.0997	-0.0358		0.0989
Mother No High School (Omitted)						
Mother High School	-0.0848		0.1195	-0.0455		0.1202
Mother Some College	-0.0377		0.1335	-0.0210		0.1340
Mother College	0.0214		0.1421	-0.0137		0.1425
Mother Advanced	-0.0259		0.1875	-0.0353		0.1877
Mother Don't Know	-0.1714		0.2067	-0.2124		0.2059
Father No High School (Omitted)						
Father High School	0.2743	**	0.2067	0.2682	**	0.1167
Father Some College	0.2117		0.2067	0.1971		0.1338
Father College	0.3115	**	0.1375	0.2592	*	0.1381
Father Advanced	0.1732		0.1752	0.1294		0.1760
Father Don't Know	0.2778		0.1756	0.2393		0.1750
Father Missing	0.0908		0.2338	0.0477		0.2331
Grade 7-8 (Omitted)						
Grade 9-10	0.0883		0.1931	-0.0776		0.2183
Grade 11-12	0.3164		0.2265	0.1269		0.2526
Allowance per week	0.0093	***	0.0031	0.0074	**	0.0031

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Table 3: *Continued*

	OLS			OLSw		
	Coef.		**S.E**	**Coef.**		**S.E**
Contextual Peer Effects						
Female	-0.0898		0.1245	-0.1071		0.1285
Age	-0.0321		0.0215	0.0316		0.0718
White	0.0111		0.1244	-0.0055		0.1694
Mother Present	0.0773		0.1668	0.1008		0.1707
Mother No High School (Omitted)						
Mother High School	-0.3878	**	0.1868	-0.2977		0.1913
Mother Some College	-0.3947	*	0.2127	-0.3825	*	0.2168
Mother College	-0.2531		0.2180	-0.2935		0.2213
Mother Advanced	-0.7011	**	0.3089	-0.5954	*	0.3112
Mother Don't Know	-0.4337		0.3598	-0.4150		0.3610
Father No High School (Omitted)						
Father High School	0.2060		0.1943	0.2999		0.1914
Father Some College	0.3639	*	0.2128	0.3890	*	0.2139
Father College	0.2850		0.2238	0.3068		0.2263
Father Advanced	0.2760		0.2891	0.2171		0.2953
Father Don't Know	0.4737		0.2995	0.5358	*	0.3001
Father Missing	0.6931		0.4619	0.7692	*	0.4640
Grade 7-8 (Omitted)						
Grade 9-10	-0.0769		0.2383	0.0104		0.2773
Grade 11-12	-0.0094		0.2630	-0.0396		0.3388
Allowance per week	0.0056	**	0.0053	0.0043		0.0054
Constant	-0.5199		0.6618			
N=2239						

*** Significant at 1% level ** Significant at 5% level * Significant at 10% level

Table 4: Peer effects in fast food consumption GSAR, GSARAR and GS-2SLS

	MLE						GS-2SLS		
	GSAR		**S.E**	**GSARAR**		**S.E**	**GSAR**		**S.E**
Endogenous Peer Effects	0.1292	***	0.0292	0.3656	***	0.0657 *	0.1102		0.3929
ρ				-0.2577	***	0.0732			
Individual Characteristics									
Female	-0.0783		0.0782	-0.0726		0.0780	-0.0838		0.0793
Age	0.1401	***	0.0440	0.1426	***	0.0426	0.1345	**	0.0531
White	-0.0618		0.1111	-0.0511		0.1113	-0.0618		0.1137
Mother Present	-0.0319		0.0986	-0.0334		0.0982	-0.0375		0.1000
Mother No High School (Omitted)									
Mother High School	-0.0329		0.1190	-0.0420		0.1190	-0.0436		0.1214
Mother Some College	-0.0102		0.1327	-0.0075		0.1327	-0.0161		0.1409
Mother College	0.0045		0.1410	0.0084		0.1410	-0.0142		0.1426
Mother Advanced	-0.0156		0.1862	-0.0243		0.1860	-0.0365		0.1880
Mother Don't Know	-0.2190		0.2042	-0.2137		0.2029	-0.2137		0.2062
Father No High School (Omitted)									
Father High School	0.2777	**	0.1157	0.2514	**	0.1156	0.2689	**	0.1169
Father Some College	0.2031		0.1326	0.1735		0.1324	0.1956		0.1344
Father College	0.2777	**	0.1372	0.2646	*	0.1367	0.2577	*	0.1388
Father Advanced	0.1340		0.1746	0.1297		0.1743	0.1275		0.1769
Father Don't Know	0.2514		0.1735	0.2307		0.1725	0.2419		0.1766
Father Missing	0.0547		0.2308	0.0334		0.2291	0.0515		0.2355
Grade 7-8 (Omitted)									
Grade 9-10	-0.1457		0.2138	-0.1398		0.2131	-0.0789		0.2186
Grade 11-12	0.0268		0.2439	0.0374		0.2430	0.1249		0.2533
Allowance per week	0.0076	**	0.0031	0.0074	***	0.0031	0.0075	**	0.0032

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Table 4: *Continued*

	MLE				GS-2SLS	
	GSAR	**S.E**	**GSARAR**	**S.E**	**GSAR**	**S.E**
Contextual Peer Effects						
Female	-0.1571	0.1255	-0.1290	0.1212	-0.1108	0.1325
Age	-0.0369 *	0.0216	-0.0727 ***	0.0221	0.0359	0.0811
White	0.0068	0.1372	0.0245	0.1279	-0.0159	0.1922
Mother Present	0.0623	0.1660	0.0818	0.1621	0.1077	0.1811
Mother No High School (Omitted)						
Mother High School	-0.3200 *	0.1867	-0.2248	0.1833	-0.3000	0.1924
Mother Some College	-0.4276 **	0.2116	-0.3745 *	0.2065	-0.3881 *	0.2224
Mother College	-0.3464	0.2166	-0.3179	0.2130	-0.3080	0.2549
Mother Advanced	-0.6582 **	0.3066	-0.5436 *	0.2971	-0.5774 *	0.3490
Mother Don't Know	-0.4701	0.3560	-0.3063	0.3539	-0.4038	0.3741
Father No High School (Omitted)						
Father High School	0.3177 *	0.1921	0.2887	0.1871	0.3299	0.3219
Father Some College	0.3873 *	0.2103	0.3895 *	0.2060	0.4051	0.2561
Father College	0.3194	0.2223	0.2638	0.2171	0.3298	0.3028
Father Advanced	0.1744	0.2897	0.1365	0.2817	0.2340	0.3305
Father Don't Know	0.5532 *	0.2959	0.4869 *	0.2899	0.5683	0.4140
Father Missing	0.7748 *	0.4606	0.7048	0.4500	0.7769 *	0.4690
Grade 7-8 (Omitted)						
Grade 9-10	0.1923	0.2668	0.1816	0.2549	0.0057	0.2804
Grade 11-12	0.3177	0.2849	0.2531	0.2691	-0.0340	0.3422
Allowance per week	0.0025	0.0053	0.0010	0.0051	0.0048	0.0069
Constant						
N=2239						

*** Significant at 1% level ** Significant at 5% level * Significant at 10% level

Log likelihood for the GSAR and the GSARAR are respectivley -4488.84623 and GSARAR -4486.3837

Table 5: Weight gain Production Function, Arellano and Bond

	Coefficient	S.E	
Constant	3.7206	0.13991	***
BMI_{t-1}	-0.72737	0.19108	***
Fast $food_t$	0.35761	0.17936	**
Sargan test Chi2(57)		61.955	
N		1445	