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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 propose a two-equation model. The first equation provides a social interaction model of fast food consumption. To estimate this equation we use a quasi maximum likelihood approach that allows us to control for common environment at the network level and to solve the simultaneity (reflection) problem. Our second equation is a panel dynamic weight production function relating an individual's Body Mass Index z-score (zBMI) to his fast food consumption and his lagged zBMI, and allowing for irregular intervals in the data. Results show that there are positive but small peer effects in fast food consumption among adolescents belonging to a same friendship school network. Based on our preferred specification, the estimated social multiplier is 1.15. Our results also suggest that, in the long run, an extra day of weekly fast food restaurant visits increases zBMI by 4.45% when ignoring peer effects and by 5.11%, when they are taken into account.

Keywords: Obesity, overweight, peer effects, social interactions, fast food, spatial models.
JEL Codes: C31 I10, I12

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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 (Ogden et al., 2012) has raised the annual obesity-related medical costs to $147 billion per year (Finkelstein et al., 2009). Obesity is also associated with increased risk of reduced life expectancy as well as with serious health problems such as type 2 diabetes (Maggio and Pi-Sunyer, 2003), heart disease (Calabr et al., 2009) and certain cancers (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. An important part of the evidence suggests the presence of peer effects in weight gain. On one hand, Christakis and Fowler (2007), Trogdon et al. (2008), Renna et al. (2008) and Yakusheva et al. (2014) are pointing to the *social multiplier* as an important element in the obesity epidemics. As long as it is strictly larger than one, a social multiplier amplifies, at the aggregate level, the impact of any shock (such as the availability and the reduction in relative price of junk food) that may affect 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. On the other hand, Cohen-Cole and Fletcher (2008*b*) found that there is no evidence of peer effects in weight gain. Also, results from a placebo test performed by the same authors (Cohen-Cole and Fletcher, 2008*a*) indicate that there are peer effects in acne (!) in the Add Health data when one applies the Christakis and Fowler (2007) method discussed later on.

While the presence (or not) of the social multiplier in weight has been widely researched,[1] the literature on the mechanisms by which this multiplier flows is still scarce. Indeed, most of the relevant

[1]For a complete review see Fletcher et al. (2011) who conducted a systematic review of literature that shows that school friends are similar as far as body weight and weight related behaviours.

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.[2] The aim of this paper is to go beyond the black box approach of peer effects in weight gain and try to identify one potentially important mechanism through which peer effects in adolescence overweight may flow: *eating habits* (as proxied by fast food consumption).

Three reasons justify our interest in eating habits in analyzing the impact of peer effects on teenage weight. First of all, there is important literature that points to eating habits as an important component in weight gain (*e.g.*, Niemeier et al., 2006; Rosenheck, 2008).[3] 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. 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, it is counter intuitive to think that one can gain weight by simply interacting with an obese person.[4] 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.*, Caraher and Cowburn, 2007; Powell et al., 2013). 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.

[2] One recent exception is Yakusheva et al. (2011) and Yakusheva et al. (2014) who look at peer effects in overweight and in weight management behaviours such as eating and physical exercise, using randomly assigned pairs of roommates in freshman year.

[3] 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 adolescents' BMI (see Auld and Powell, 2009; Powell, 2009; 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.

[4] Of course, having obese peers may influence an individual's tolerance for being obese and therefore his weight management behaviours.

In order to analyze the impact of peer effects in eating habits on weight gain, we propose a two-equation model. The first *linear-in-means* equation relates an individual's fast food consumption to his individual characteristics, his reference group's mean fast food consumption (*endogenous peer effect*), and his reference group's mean characteristics (*contextual peer effects*). The endogenous peer effect reflects the possibility that eating behaviour of his friends influences a teenager's own behaviour. For instance, one reason why an adolescent may want to go to a fast food restaurant is to be with his friends during the lunch. Contextual effects, such as the average level of education of his friends' mother, may also affect a teenager's eating habits. Thus, mothers with higher education may encourage not only their children but also their children's friends to develop accurate eating bebaviour.

The second equation is a panel dynamic production function that relates an individual's BMI adjusted for age (z-score BMI, or zBMI) to his fast food consumption, his lagged zBMI and other control variables. The system of equations thus allows us to evaluate the impact of an eating habits' exogenous shock on an adolescent's weight, when peer effects on fast food consumption are taken into account. To estimate our two-equation model, we use three waves of the National Longitudinal Study of Adolescent Health (Add Health), that is, Wave II (1996), Wave III (2001) and Wave IV (2008).[5] 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.

Estimating our system of equations raises serious econometric problems. It is well known that the identification of peer effects (first equation) is a challenging task. These identification issues were first pointed out by Manski (1993) and discussed among others by Bramoullé, Djebbari and Fortin (2009) and Blume et al. (2013). 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 share similar characteristics (*i.e.*., homophily) or face a common environment (*e.g.*, same school). On the other hand, simultaneity between an adolescent's and his peers' behaviour (re-

[5]Note that for the first equation we use wave II and for the second equation we use the three waves.

ferred to as *the reflection problem* by Manski) may make it difficult to identify separately the endogenous peer effect and the contextual effects.

We use a new approach based on Bramoullé et al. (2009) and Lee et al. (2010), and extended by Blume et al. (2013) 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, conditional on the school fixed effects and observable individual and contextual variables, is strictly exogenous. The possibility that friends select each other using unobservable traits that may be correlated with their fast food consumption decisions is an important issue and is discussed later on.

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. Finally, we exploit the similarity between the linear-in-means model and the spatial autoregressive (SAR) model with or without autoregressive spatial errors.[6] The model is estimated using a quasi maximum likelihood (QML) approach as in Lee et al. (2010). The QML is appropriate when the estimator is derived from a normal likelihood but the error terms in the model are not truly normally distributed. We also estimate the model using generalized spatial two-stage least square proposed in Kelejian and Prucha (1998) and refined in Lee (2003), which is less efficient than QML.

The estimation of the production function (second equation) also raises serious econometric issues. First, fast food consumption is likely to be an endogenous variable correlated with the individual error

[6]Our approach is more general than the SAR model as the latter usually ignores contextual effects and spatial fixed effects.

term. Moreover, the short and the long term impacts of fast food consumption on zBMI may be different, suggesting the introduction of lagged zBMI as an explanatory variable. Finally, Add Health data waves are collected at irregular intervals. As a consequence, estimators obtained from standard dynamic panel data models are inconsistent. In order to deal with these problems, we use a nonlinear instrumental approach developed by Millimet and McDonough (2013).

Results suggest that there is a positive but small endogenous peer effect in fast food consumption among adolescents in general. Based on our QML specification, the estimated social multiplier is 1.15. Moreover, the production function estimates indicate that there is a positive significant impact of fast food consumption on zBMI. Combining these results, we find that, in the long run, an extra day of weekly fast food restaurant visits increases zBMI by 4.45% when ignoring peer effects and by 5.11%, when they are taken into account.

The remaining parts of this paper will be laid out as follows. Section 2 provides a 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 the specification of our fast food equation with peer effects. Section 4 is devoted to our weight production function. In section 5, we give an overview of the Add Health Survey and we provide descriptive statistics of the data we use. In section 6, we discuss estimation results. Section 7 concludes.

2 Previous Literature

In recent years, a number of studies found strong "social network effects" in weight outcomes. In a widely debated article, Christakis and Fowler (2007) 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.[7] However, their analysis has been criticized for suffering from a number of limitations (see Cohen-Cole and Fletcher,

[7]They used a 32-year panel dataset on adults from Framingham, Massachusetts and a logit specification.

2008*b*; 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 the same environment. Both Shalizi and Thomas (2011) and Lyons (2011) show that 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.

In the same spirit, Trogdon et al. (2008) investigate the presence of peer effects in obesity using Add Health data. They include school fixed effects to account for the fact that students in a same school share a same surrounding. The authors also estimate their BMI peer model with an instrumental variable approach using information on friends' parents' obesity and health and friends' birth weight as instruments for peers' BMI. They find that a one point increase in peers' average BMI increases own BMI by 0.52 point. Based on a similar approach and using Add Health dataset, Renna et al. (2008) also find positive peer effects. These effects are significant for females only (= 0.25 point).[9] 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. In our approach, we introduce school fixed effects and, for each individual variable, the corresponding contextual variable at the reference group level.

Using the same dataset as Trogdon et al. (2008) and Renna et al. (2008), Cohen-Cole and Fletcher (2008*b*) 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.

[8] For a response to these criticisms and others, see Fowler and Christakis (2008), VanderWeele (2011) and Christakis and Fowler (2013).

[9] Also Ali, Amialchuk and Heiland (2011) and Ali, Amialchuk and Renna (2011) provide evidence that there are peer correlations in weight related behaviours and peer influence in weight misperception respectively.

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 recent paper, and one of the most careful studies that truly randomizes the network formation to date, Yakusheva et al. (2011) estimate peer effects in explaining weight gain among freshman girls using a similar set up but in school dormitories. In their paper, 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. In a subsequent paper, Yakusheva, Kapinos and Eisenberg (2014) investigate the presence of peer effects in weight gain exploiting random assignment of roommates during first year of college. The authors find evidence that suggests that peer effects in weight gain are predominantly significant among females.[10]

Our paper finds its basis in this literature as well as the literature on peer effects and obesity discussed above. However, while works by Yakusheva et al. (2011) and Yakusheva et al. (2014) rely upon experimental data, we use observational non-experimental data. Peers 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 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

[10]In contrast, De la Haye et al. (2010) provide evidence that close adolescent male friends tend to be similar in their consumption of high-calorie food.

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 (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 finds evidence of a positive correlation between fast food consumption and obesity (see, Anderson et al., 2011; Rosenheck, 2008).

The economic literature tends to be conservative with respect to this question. It focuses the impact of "exposure" to fast food on obesity. Dunn et al. (2012), using an instrumental variable approach, investigates the relationship between fast food availability and obesity. They finds that an increase in the number of fast food restaurants has a positive effect on the BMI among non-whites. Alviola IV et al. (2014), using a similar approach, provides evidence that the number of fast food restaurants has a significant impact on school obesity rates. Similarly, Currie et al. (2010) find evidence that proximity to fast food restaurants has a significant effect on obesity for 9th graders. Also, Anderson and Matsa (2011), exploiting the placement of Interstate Highways in rural areas to obtain exogenous variations in the effective price of restaurants, did not find any causal link between restaurant consumption and obesity. 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 prior to adulthood sets the stage for weight in adulthood. While most of the economics literature analyses the relationship between adolescents' fast food consumption and their weight using an indirect approach (*i.e*, effect to fast food exposure), we adopt a direct approach linking weight as a function of fast food consumption, lagged weight and control variables.

In the next two sections, we present our two-equation model of weight with peer effects in fast food

[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.

consumption. We first propose a linear-in-means social interaction equation of fast food consumption (first equation) and discuss the econometric methods we use to estimate it. We then present our econometric weight production function which relates the adolescent's zBMI level to his fast food consumption (second equation).

3 Social Interactions Equation of Fast Food Consumption

We assume 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. Therefore, the $\mathbf{G}_l$ matrix is row normalized. 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).[12] The network invariant unobservable variables are captured through fixed network 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 equation 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. In the context of our paper, β is the *endogenous peer effect*. It reflects how the adolescent's consump-

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

tion 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.[13] It captures the impact of his peers' mean characteristic on his fast food consumption. It is important to note that the $\mathbf{G}_l$ matrix and the $\mathbf{x}_l$' vector are allowed to be stochastic but are 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). Nevertheless, once we condition on these common characteristics, mother's education is assumed to be independent of the idiosyncratic error terms. Let $\mathbf{I}_l$ be the identity matrix for a network l and $\boldsymbol{\iota}_l$ the corresponding vector of ones, the equation (1) for network l can be rewritten in matrix notation as follows:

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

Note that equation (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 equation (2) can be written as:

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

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.[14]

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 complementarity* in fast food consumption), the social multiplier is larger than 1. In this case, the impact of the shock is amplified by

[13] It is standard to assume the presence of a contextual effect for each individual characteristic influencing the outcome. Otherwise, the model may impose *ad hoc* exclusion restrictions which generate invalid instruments and inconsistent estimators.

[14] When an adolescent is isolated, his intercept is α_l.

social interactions as more fast food consumption by his peers induces an adolescent to adopt a similar behaviour.

We then perform a panel-like *within* transformation. 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 that 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 network fixed effect α_l. Pre-multiplying (3) by $\mathbf{K}_l$ yields the reduced form of the model for network l, in deviation:

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

3.0.1 Identification

Our peer effects structural equation (2) raises two basic identification problems.

- Simultaneity

Simultaneity between individual and peer behaviour (the *reflection problem*) may prevent separating contextual effects from endogenous effects. This problem has been analyzed by Bramoullé et al. (2009) when individuals interact through social networks. They show that the conditions of identification depend on both the values of parameters and the structure of the network. More explicitly, let us first assume throughout that $\beta\gamma + \delta \neq 0$. Then 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 equation (2) are identified if the matrices $\mathbf{I}$, $\mathbf{G}$, $\mathbf{G}^2$ are linearly independent. This condition is satisfied when 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.[15] The intuition is that this provides exclusion restrictions in the model. More precisely, the friends' friends

[15] More generally, equation (2) is identified when individuals do not interact in groups or interact in groups with at least three different sizes (see Bramoullé et al., 2009).

mean characteristic can served as instrument 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.[16]

- Correlated effects

The presence of confounding unobservable variables affecting fast food consumption and correlated with the explanatory variables raises difficult identification problems. First, 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. 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. Recall also that network fixed effects take into account common unobservable variables at the school level that may influence fast food consumption (*e.g.*, availability of fast food restaurants).

Of course, one limitation of using network fixed effects is that it ignores the possibility that the

[16]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.

links formation within a network depend on omitted variables. The matrix $\mathbf{G}$ may be endogenous even when controlling for the network fixed effects and observable characteristics. Thus friends may select each other using unobservable traits that may be correlated with fast food consumption (*e.g.*, impulsivity, a specific taste for sugar- and fat-rich food, *etc*). Recently, some researchers (*e.g.*, Hsieh and Lee, 2011; Goldsmith-Pinkham and Imbens, 2013; Liu et al., 2013; Badev, 2013) have made attempts to develop econometric models allowing for the joint estimation of network formation and network interactions. However, empirical results using Add Health data and focusing on outcomes such as smoking, sleeping behaviour, and scholar performance, do not seem to detect much difference in peer effects when networks are assumed exogenous and when they are allowed to be endogenous.[17]

One specification of our peer effects equation also allows the error terms to be (first-order) autocorrelated within networks. Therefore its structure becomes analogous to that of a generalized spatial autoregressive model with network autoregressive disturbances (denoted as the GSARAR model). This model implies that in addition to the endogenous and contextual effects, some unobserved characteristics of the friends are also interdependent. 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 equation (4) in deviation, the latter is given by the following structural form:

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

[17] One reason may be that this data base includes a very large number of observable characteristics, some of them being used in the regressions. Another explanation is that, although statistically significant, the explanatory power of the individual characteristics on the probability that two individuals are friends is extremely small (Boucher, 2014)

where $\mathbf{X}_l$ is the matrix of adolescents' characteristics[18] in the lth network, $\mathbf{M}_l = (\mathbf{I} - \rho\mathbf{G}_l)$ and $\boldsymbol{\nu}_l = \mathbf{K}_l\mathbf{M}_l\boldsymbol{\xi}_l$.

Following Lee et al. (2010), we propose two approaches to estimate the peer effects equation (7): a Quasi maximum likelihood approach (QML) and a generalized spatial two stage least squares (GS-2SLS) approach. The QML estimators are estimated assuming that they are normally distributed. However, we do allow the log-likelihood function to be *partially misspecified*, as standard errors are computed to be robust to non-normal disturbances (using a sandwich formula). Assuming that the error terms are *i.i.d.* and under a number of regularity assumptions (see p.152, Lee et al., 2010), QML estimators are consistent but not asymptotically efficient. On the other hand, GS-2SLS estimators also assume that the error terms are *i.i.d* but impose less regularity conditions than QML estimators. QML estimators are asymptotically more efficient than GS-2SLS estimators.[19]

4 Weight Production Function

In this section, we propose a dynamic (AR(1)) weight production function that relates an individual's zBMI in period t (assumed a year) to his lagged zBMI, his fast food consumption and his own characteristics in period t. Let y_{it}^b be an individual i's zBMI level in period t, and y_{it}^f be the individual's fast food consumption in period t. Then, for a given vector of characteristics $\tilde{\mathbf{x}}_{it}$, the data generating process (DGP) of the weight production function can be formally expressed as follows (for notational simplicity we suppress l):

$$y_{it}^b = \pi_1 y_{i,t-1}^b + \pi_2 y_{it}^f + \boldsymbol{\pi}_3'\tilde{\mathbf{x}}_{it} + \mu_i + \zeta_{it}, \tag{8}$$

where π_1 is the autoregressive parameter ($|\pi_1| < 1$), μ_i is the individual i's time-invariant error component (fixed effect) and ζ_{it}, his idiosyncratic error that may change across t. One difficult problem with (8)

[18] Following the linear-in-means model, we allow the peers' mean characteristic corresponding to each individual's characteristic to have a potential effect on his fast food consumption. Therefore, we do not impose *ad hoc* (identifying) exclusion restrictions to the structural peer effects equation.

[19] The derivation of the QML and GS-2SLS estimators are presented in the Appendix.

is that the Add Health data set, the waves are *irregularly spaced*. This means that the successive periods of observed data (that is, for 1996, 2001 and 2008) do not conform to successive (yearly) periods as defined by our underlying DGP. In that case, standard methods to estimate a dynamic panel model with endogenous variables (*e.g.*, Anderson and Hsiao, 1981; Arellano and Bond, 1991) yield inconsistent estimators. To address this point, we follow Millimet and McDonough (2013) (hereinafter MM) approach. From repeated substitution in eq. (8) we rewrite equation (8) defined over the *observed* periods $m = 1,\ 2,\ 3$, one obtains:

$$y_{im}^{b} = \pi_1^{g_m} y_{i,m-1}^{b} + \pi_2 y_{im}^{f} + \boldsymbol{\pi}_3' \tilde{\mathbf{x}}_{im} + \theta\mu_i + \tilde{\zeta}_{im}, \tag{9}$$

where g_m is the gap size or the number of years between observed period m and $m-1$, (which, in our case, are equal to $g_1 = 1, g_2 = 5, g_3 = 7$)[20]; $\theta = \frac{1-\pi_1^{g_m}}{1-\pi_1}$, and

$$\tilde{\zeta}_{im} = \sum_{j=1}^{g_m-1} (\pi_2 y_{i,t(m)-j}^{f} \ + \ \boldsymbol{\pi}_3' \tilde{\mathbf{x}}_{i,t(m)-j})\pi_1^{j} \ + \ \sum_{j=0}^{g_m-1} \pi_1^{j} \zeta_{i,t(m)-j}, \tag{10}$$

where $t(m)$ is the actual period reflected by the observed period m: $t(1) = 1;\ t(2) = 6;\ t(3) = 13$.

Equation (9) shows that when data are irregularly spaced, 1) the coefficient on the lagged dependent variable is not constant but equal to $\pi_1^{g_m}$; 2) the error term $\tilde{\zeta}_{im}$ contains the covariates and the idiosyncratic errors from the missing periods between m and $m-1$, and the current error; 3) the unobserved fixed effect has a period-specific factor loading, θ. The first point raises the following difficulty: the equation is now nonlinear in π_1, which suggests the use of a nonlinear in parameters approach. More importantly, unequally spaced data relegate missing covariates into the error term (point 2). This is a serious source of concerns as long as some contemporary covariates are serially correlated and therefore become mechanically endogenous. Finally, one cannot eliminate the fixed effect μ_i using standard first-differencing or mean-differencing transformations since the factor loading parameter θ varies from one observed period to another (point 3).

[20] One has $g(1) = 1$ since Wave I from Add Health data (which corresponds to $m = 0$) was collected in 1995.

To estimate such an equation, MM suggests the use of a nonlinear instrumental approach extending Everaert (2013) technique for estimating dynamic panel data models. It consists first in instrumenting the lagged zBMI ($= y^b_{i,m-1}$) with the OLS residual of $y^b_{i,m-1}$ regressed on its backward mean $\bar{y}^b_{i,m-1}$, where $\bar{y}^b_{i,m-1} = \frac{1}{m}\sum_{s=0}^{m-1} y^b_{i,s}$. The intuition here is that the residual (which reflects the part of the lagged zBMI not explained by its backward mean) is likely to be highly correlated with the lagged zBMI. However, it should be uncorrelated with the fixed effect reflecting the time-invariant unobserved part of the individual's zBMI. Also it should be uncorrelated with the contemporary idiosyncratic error term as long as the latter is i.i.d.$(0, \sigma_\epsilon)$. Therefore, the residual is a good candidate as an instrument for the lagged zBMI. More explicitly, MM shows that a nonlinear IV version using of Everaert (2013) technique (referred to as E-NLS-IV) to account for irregular spacing yields consistent estimators, when T$\rightarrow \infty$ and the covariates are strictly exogenous and serially uncorrelated. Also, while the estimators are inconsistent when T is fixed and N$\rightarrow \infty$, Monte Carlo simulations by MM suggests that this approach has superior small sample properties compared to other dynamic panel data estimators.

Second, some covariates (in particular, the individual's fast food consumption, y^f) are likely to be correlated with the unobserved effect and/or to be serially correlated. In the first case, Everaert (2013) suggests to use Hausman and Taylor (1981) type instruments for these covariates, that is, deviations from individual sample means (*e.g.*, $\ddot{y}^f$). Also, in the presence of serially correlated covariates, one solution suggested by MM is to impute data for the missing periods. For instance, we can use current value of covariates to approximate missing covariates between periods m and $m-1$[21]. Therefore, in eq. (10), we can write:

$$\sum_{j=1}^{g_m-1} (\pi_2 y^f_{i,t(m)-j} \; + \; \boldsymbol{\pi}_3' \tilde{\mathbf{x}}_{i,t(m)-j})\pi_1^j \approx (\pi_2 y^f_{i,m} + \boldsymbol{\pi}_3' \tilde{\mathbf{x}}_{i,m}) \frac{\pi_1 - \pi_1^{g_m}}{1-\pi_1}. \tag{11}$$

In this paper, we estimate the weight production function given by eqs. (9) to (11) using a nonlinear

[21]No approximation is needed for variables such age, for which we have perfect information at each period.

instrumental approach and based on current values of covariates to approximate missing data for the missing periods. Following MM, we denote this estimator: E-NLS-IV-C. We also present a GMM version of this estimator using a two-step approach to obtain an optimal weighting matrix (clustered at the individual level).

As discussed earlier, our interest in this production function goes beyond a mere association between fast food consumption and weight. We are particularly interested to analyze the magnitude of a change in zBMI 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 (8) with respect to $y^f_{i,t-1}$ and using the social multiplier $[= 1/(1-\beta)]$ yields the magnitude of a short run change in zBMI (*i.e.*, for $y^b_{i,t-1}$ given) resulting from a common marginal shock on fast food consumption: $\partial E(y^b_{it}|\cdot)/\partial \alpha_l = \frac{\pi_2}{1-\beta}$. This expression entails two components: the impact of the fast food consumption on zBMI ($= \pi_2$) and the multiplier effect ($= \frac{1}{1-\beta}$). In the long run, at the new stationary state, the impact of the shock on zBMI is given by $\frac{\pi_2}{(1-\beta)(1-\pi_1)}$.

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, neighbourhood, community, school, friendships, peer groups, romantic relationships, *etc.* In wave I (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.[22] This cohort was then followed in-home in the subsequent waves in 1996 (wave II), 2001 (wave III) and 2008-2009 (wave IV). 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.

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 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. To estimate the weight production function, we considered information from wave I, wave II, wave III and wave IV.

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 (directed network with non symmetric links).

[22] Adolescents were asked to nominate up to five female friends and five male friends.

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

5.1 Descriptive statistics

In our social interactions equation, 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 7 days. 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 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. 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.

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 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 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 production function, the dependent variable that we use is zBMI in waves III and IV. [24] The zBMI variables for each wave, the fast food variables for wave II, III and IV are detailed in Table 4.[25]

[24] To compute the backward mean we used all four waves.

[25] It is important to note that information on fast food consumption was not collected in wave I.

5.2 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 $\mathbf{G}$.[26] 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 the peer effects equation. As mentioned earlier, Bramoullé et al. (2009) show that 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 verified with our data. We also compute the Belsley, Kuh, and Welsch *condition index* to check for the presence of collinearity between these matrices. If this index is below 30, then collinearity is said not to be a problem and linear independence of the four matrices is verified. In our data, the reflection problem is clearly solved since $\mathbf{I}$, $\mathbf{G}$, $\mathbf{G}^2$ and $\mathbf{G}^3$ are linearly independent and the condition index value is 2.21.

6 Results

6.1 Baseline: OLS peer effects estimates

We first estimate a naive OLS of the peer effects equation 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

[26] 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.

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 2 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 face a common environment or because adolescents with similar characteristics tend to attend the same school (homophily). As for the individual characteristics, age, father education and weekly allowance positively affect fast food consumption. Turning our attention to the contextual peer effects, we notice that the latter variable 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.

6.2 GS-2SLS and QML peer effects estimates

Next, we estimate our peer effects equation with school fixed effects using GS-2SLS (with *i.i.d.* error terms and without imposing autoregressive disturbances: $\rho = 0$). We then estimate this equation using a QML approach with *i.i.d.* error terms . Also, we estimate another plausible version of this model by allowing network autoregressive disturbances (GSARAR model).

Estimation results are displayed in Table 3. The GS-2SLS approach (see last two columns) assumes that the instrument for $\mathbf{G}^*\mathbf{y}^*$ is given by $\mathbf{G}^*\hat{\mathbf{y}}^*$ (see eq. 14).[27] One can check whether this instrument is weak by regressing $\mathbf{G}^*\mathbf{y}^*$ on $\mathbf{G}^*\hat{\mathbf{y}}^*$, $\mathbf{X}^*$ and $\mathbf{G}^*\mathbf{X}^*$ and performing a Stock-Yogo test (see Table 3).

[27] The star superscript indicates that the original variable has been transformed to eliminate the problem of singular variance matrix generated by the use of the within transformation to eliminate fixed network effects. See Appendix.

It consists in comparing the Cragg-Donald F statistic associated with the estimated coefficient of $\mathbf{G}^*\hat{\mathbf{y}}^*$ ($= 17.80$) with its critical value when one assumes a 10% tolerance[28] for the size distortion of the 5% Wald test (= 16.38). Based on this test, we reject that the instrument is weak. The endogenous effect resulting from GS-2SLS estimation is positive (=0.11 or 4.73%) but non significant. When using the GSAR QML approach, estimation results show a positive endogenous effect of 0.129 (or 5.3%). This estimate reveals to be very close to the one obtained by GS-2SLS and is slightly smaller than the OLS ones obtained in the previous sub-section. It is statistically significant at the 5% level if we perform a one tail test (one-tail p value = 0,039) but at 10% if we consider a two tail test (two tail p value= 0.0785). While this result suggests that one has to be quite cautious when accepting the estimate, it can be argued that one should perform a one-tail test since one expects the endogenous peer effect to be either positive or zero. In that case, the social multiplier associated with an exogenous increase in an adolescent fast food consumption is 1.15 ($= \frac{1}{1-0.129}$) and is significantly different from 1, based on a one-tail test (its standard error is 0.084 using the delta method, with a one tail p-value of 0.037). This reflects a relatively low endogenous peer effect.

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 using the *linear-in-means* equation , a richer body of literature has investigated a tangent issue : obesity. As compared with endogenous effects obtained in the literature on obesity, our peer effect is intermediate between studies that obtain no peer effects (Cohen-Cole and Fletcher, 2008*b*) and the literature that provides evidence that there are peer effects are strong, for instance, generating a social multiplier larger than 1.5 (e.g., Christakis and Fowler, 2007; Trogdon et al., 2008).[29]

To check the sensitivity of these results to the presence of SAR disturbances, we also estimate our model using a GSARAR QML specification. The estimated spatial autocorrelation coefficient is negative

[28]This level of tolerance is the smallest one that can be computed given that there is only one excluded instrument.

[29]More specifically, Cohen-Cole and Fletcher (2008*b*) finds a statistically insignificant social multiplier of 1.03, Christakis and Fowler (2007) find a statistically significant social multiplier of 2.63 and Trogdon et al. (2008) find a statistically significant social multiplier of 2.08.

but not significant at the 5% level. Moreover the endogenous peer effect is large (=0.3655) but no longer significant even at the 10% level (one-tail test). Also a likelihood test does not reject the GSAR QML specification. Therefore we consider the latter as our preferred one. This suggests a much lower endogenous peer effect (= 0.13), which can be interpreted as a lower bound to this parameter, at least when assuming that selection on unobservables is not an important source of biases, after controlling for network fixed effects and observable characteristics (see our discussion above).

To sum up, we can say that results in general are consistent with the hypothesis that fast food consumption is linked to issues of interactions with friends. However, our social multiplier estimate does not appear to be very strong (as the endogenous effects are less than 0.3), at least when we consider a specification which seems reasonable. This result, despite its small magnitude, addresses the puzzle around the behavioural channels through which peer effects in weight gain flows. Indeed, while Yakusheva et al. (2014) in their attempt to uncover the channels through which these effects flow have tested for two behavioural channels exercise and eating disorders (*e.g.*, anorexia), they could not test for the presence of peer effects in eating habits due to data limitations.

As for estimated individual effects and focusing on the GSAR QML specification, 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. 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 evidence suggests a positive income elasticity (Aguiar and Hurst, 2005). The positive relation between fast food consumption and allowance is thus in line with the positive relation between income and fast food consumption.

One advantage of our spatial approach is that it allows to identify both endogenous and contextual peer effects. Turning our attention to the latter, we note in particular that an adolescent's fast food consumption decreases with peers' mother's education but increases with mean peers' father's educa-

tion. While the former causal effect seems natural as mothers with higher education may (directly and indirectly) encourage both their children and their friends to have better eating habits, the latter effect is rather puzzling. One partial explanation is that fathers with higher education are more likely to be absent from home. Therefore they have less positive influence on their children's and friends' eating habits.

6.3 Weight 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 production function presented earlier.

Results from the estimation of the production function are reported in Table 5. Specification (1) shows baseline OLS estimates of equation (8) (where t is replaced by m), specification (2) shows the NLS estimates of equation (9), specification (3) shows E-NL-IV-C estimation results for equation (9) and finally specification (4) shows GMM version the previous estimator using a two step with an optimal weighting matrix. All specifications are estimated using wave 3 and wave 4, but where information from wave 1 and wave 2 are used to construct the instruments.

In line with our expectations, the general results indicate that lagged zBMI and current fast food consumption have positive significant effect (which is between 0 and 1, in the case of the lagged zBMI) on current zBMI. These results seem to be robust across different specifications with some differences that can be explained by the differences in the assumptions made on the DGP. More specifically results in specification (1), our baseline specification, are comparable to previous findings by Niemeier et al. (2006) who used the same data set. The impact of lagged zBMI is 0.7591 (compared to 0.7600 for Niemeier et al. (2006)). As for the impact of fast food consumption, it is 0.014 (compared to 0.020 for Niemeier et al.

(2006)). [30]

Comparing specification (1) with specification (2), we notice that the estimate of the fast food consumption marginal effect is much higher in the former than in the latter case (0.0145 vs. 0.0039). The basic explanation is that when estimating the parameter π_2 in the specification (1), we are not accounting for missing data on y_{it}^f in time intervals between m and $m-1$. In fact, we are estimating $\pi_2(\frac{1-\pi_1^{g_m}}{1-\pi_1})$, with $(\frac{1-\pi_1^{g_m}}{1-\pi_1}) > 1$, instead of estimating π_2. Also the estimate associated with the lagged endogenous variable is smaller in specification (1) than in specification (2) (*i.e.*, 0.7591 vs. 0.9545). The explanation is that in the OLS specification we are implicitly estimating $\pi_1^{g_m}$ with $|\pi_1| < 1$ as we are ignoring the irregularly time intervals.

While specification (2) accounts for the unequally spaced intervals, it does not account for the correlation between lagged zBMI and the time-invariant unobserved effect and the idiosyncratic error term of zBMI. Further it does take into account the fact that fast food consumption may be correlated with the time-invariant unobserved effect. To account for these possibilities, we follow MM and instrument zBMI using the OLS residuals of the regression lagged zBMI on its backward mean. As for the fast food consumption, it is instrumented using Hausman and Taylor (1981) type of instruments, that is, by taking the deviations of fast food consumption with respect to individual sample mean as an instrument.

The estimation of specification (3) shows results that are consistent with previous results, with some differences in the magnitude of the parameters. The estimated parameter associated with zBMI is smaller than the one obtained in the second specification (0.8461 vs. 0.9545). As for the estimated parameter for fast food consumption it is marginally higher when using E-NL-IV-C (0.0040 vs. 0.0039). All results remain statistically significant. To complement results from the E-NL-IV-C we estimate an optimal GMM version (with a weighted matrix clustered at the the individual level) of the same model. Estimation results are again within the expected lines and consistent with previous estimates with some variation

[30] It is important to note that Niemeier et al. (2006) used a different wave and a different approach.

in the estimates of the lagged zBMI (0.8447 vs. 0.8461) and fast food consumption parameters (0.0039 vs. 0.0031).

We retain specification (4) as our preferred one as it provide optimal and robust estimators. Results from this specification show that lagged zBMI has a positive significant effect on current zBMI level (= 0.8447). This suggests that an exogenous shock on weight has a stronger effect in the long term than in the short term. Based on specification (4), an extra day of fast food restaurant visit per week increases zBMI by 0.02 ($= \frac{0.0031}{1-0.8447}$) zBMI points or 4.45% in the long term. The presence of a causal link between fast food consumption and zBMI does not come as a surprise since previous findings have been pointing in this direction (*e.g.*, Levitsky et al., 2004; Niemeier et al., 2006; Rosenheck, 2008). Combining the impact of fast food on weight gain with the social multiplier, our results suggest that an extra day of fast food restaurant visits per week leads to a zBMI increase of 0.0351 zBMI points, or 5.11% ($= 4.45\% \times 1.15$) on average, as compared with 4.45% with no peer effects. These results highlight a role for peer effects in fast food consumption as one transmission mechanism through which weight gain is amplified.

As for the other covariates, age reveals to have a positive significant effect on zBMI, an additional year increasing zBMI by 0.0072 zBMI points. Also being female and white have a negative effect on zBMI (respectively 0.0176 and 0.0153). To test for the relevance of the instruments we use Kleibergen-Paap rank test (which is used here to take into account the fact that the estimated standard errors are panel clustered). The test rejects the null hypothesis that the instruments are weak.[31]

7 Conclusion

This paper investigates whether peer effects in adolescent weight 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

[31] As the model is exactly identified, it is not possible to conduct an over identification test.

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. However, our estimated social multiplier based on our preferred (conservative) specification is small as it is equal to 1.15.

We also estimate a dynamic weight production function which relates the individual's Body Mass Index to his fast food consumption. Our results reveal a positive significant impact of a change in fast food consumption on the change in zBMI. Specifically, in the long run, a one-unit increase in the weekly frequency (in days) of fast food consumption produces an increase in zBMI by 4.45%. This effect reaches 5.11% when the social multiplier is taken into account. This suggests the presence of a positive but low endogenous peer effect. In short, our results are intermediate between studies on overweight or obesity that report no peer effects (*e.g.*, Cohen-Cole and Fletcher, 2008*a*) and others that provide evidence of strong peer effects (e.g., Trogdon et al., 2008; Christakis and Fowler, 2007)

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 somewhat increase 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, or used to implement network based interventions to promote healthy eating behaviours (Fletcher et al., 2011).

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 BMI outcomes. At the methodological level, a possible extension would be 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: 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 2: *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=2339						

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

Table 3: Peer Effects in fast food consumption GSAR, GSARAR and GS-2SLS

	Quasi Maximum Likelihood						GS-2SLS		
	GSAR		**S.E.**	**GSARAR**		**S.E.**	**GSAR**		**S.E.**
Endogenous Peer Effects	0.1293	* †	0.0729	0.3511		3.3869	0.1102		0.3945
ρ				-0.2342		3.6954			
Individual Characteristics									
Female	-0.0787		0.0589	-0.0861		0.0832	-0.0839		0.078
Age	0.1401	***	0.0479	0.1477		0.1386	0.1346	**	0.0531
White	-0.0622		0.0795	-0.0582		0.2083	-0.0619		0.1169
Mother Present	-0.0319		0.076	-0.0278		0.1097	-0.0375		0.0973
Mother No High School (Omitted)									
Mother High School	-0.0333		0.051	-0.0549		0.3647	-0.0437		0.1212
Mother Some College	-0.0106		0.1156	-0.0258		0.2193	-0.0161		0.1401
Mother College	0.004		0.1043	-0.0025		0.1384	-0.0143		0.1449
Mother Advanced	-0.0159		0.1737	-0.0555		0.5745	-0.0366		0.1873
Mother Don't Know	-0.2193	***	0.0813	-0.2327	**	0.1101	-0.2138		0.2098
Father No High School (Omitted)									
Father High School	0.2774	**	0.1165	0.268		0.4137	0.2689	**	0.1179
Father Some College	0.2027	**	0.0889	0.1923		0.4408	0.1957		0.137
Father College	0.2775	***	0.0837	0.28	*	0.154	0.2577	*	0.1364
Father Advanced	0.1336		0.1821	0.1418		0.1918	0.1275		0.1785
Father Don't Know	0.2512	***	0.0917	0.2519		0.2648	0.242		0.1742
Father Missing	0.0548		0.1302	0.0721		0.1264	0.0515		0.2308
Grade 7-8 (Omitted)									
Grade 9-10	-0.1499		0.2569	-0.1444		0.2677	-0.0789		0.1957
Grade 11-12	0.0225		0.2706	0.0329		0.3268	0.1249		0.23
Allowance per week	0.0076	***	0.0016	0.0076	***	0.0016	0.0075	**	0.0032

One tail test: † Significant at 5% level.

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Table 3: Peer Effects in fast food consumption GSAR, GSARAR and GS-2SLS *continued*

	Quasi Maximum Likelihood						GS-2SLS		
	GSAR		**S.E.**	**GSARAR**		**S.E.**	**GSAR**		**S.E.**
Contextual Peer Effects									
Female	-0.1563		0.1049	-0.1721		0.1178	-0.1108		0.1323
Age	-0.0379	**	0.0152	0.0158		0.5929	0.0359		0.0813
White	0.0079		0.1064	0.0246		0.4373	-0.0159		0.1865
Mother Present	0.0651		0.2354	0.0955		0.6757	0.1078		0.1794
Mother No High School (Omitted)									
Mother High School	-0.3181	**	0.1327	-0.4149		0.6549	-0.3001		0.1858
Mother Some College	-0.4254	***	0.1534	-0.4448		0.2741	-0.3882	*	0.2213
Mother College	-0.3443		0.2255	-0.3462		0.3121	-0.308		0.2496
Mother Advanced	-0.6565	**	0.3032	-0.6871		0.9187	-0.5775	*	0.3407
Mother Don't Know	-0.4664	*	0.2508	-0.5488	*	0.2901	-0.4038		0.3604
Father No High School (Omitted)									
Father High School	0.3197	***	0.1211	0.3074		0.4624	0.329		0.3128
Father Some College	0.389	***	0.1161	0.3476		0.5849	0.4051	*	0.244
Father College	0.3214		0.2128	0.3354		0.2238	0.3298		0.3035
Father Advanced	0.1765		0.2179	0.233		0.5672	0.2341		0.3299
Father Don't Know	0.5544	***	0.1172	0.5478		0.7167	0.5684		0.3985
Father Missing	0.777	**	0.3058	0.7783		0.5355	0.7769	*	0.4461
Grade 7-8 (Omitted)									
Grade 9-10	0.1998		0.2873	0.19		0.4396	0.0058		0.252
Grade 11-12	0.3272		0.2311	0.384		0.3932	-0.0341		0.3195
Allowance per week	0.0025		0.0033	0.0047		0.0237	0.0048		0.0072
Log Likelihood	-4488.847			-4487.55					
N=2339									
Stock-Yogo Test									17.80
Critical value (r=0.10)									
at sign. level of 0.05%									16.38

Two tail test: *** Significant at 1%;** Significant at 5%; * Significant at 10% level.

Table 4: Descriptive Statistics-Production function

Variable	Mean	S.D.
zBMI wave I	0.4017	1.0178
zBMI wave II	0.4485	1.0210
zBMI wave III	0.7279	1.0903
zBMI wave IV	1.0173	0.9712
Fast food wave II	2.3869	1.7810
Fast food wave III	2.6206	2.0685
Fast food wave IV	2.2602	2.0790
Age wave II	16.5741	1.5674
Age wave III	22.0200	1.5613
Age wave IV	29.1677	1.5417
female	0.5156	0.4999
White	0.6250	0.4842
Obs.		1848

Table 5: Weight Production Function

	Spec 1 OLS	Spec 2 NLS	Spec 3 E-NL-IV-C	Spec 4 GMM (Adj. W)
Fast food consumption	0.0145***	0.0039***	0.0040**	0.0031**
	(0.0050)	(0.0009)	(0.0020)	(0.0014)
zBMI (lagged)	0.7591***	0.9545***	0.8461***	0.8447***
	(0.0115)	(0.0024)	(0.0186)	(0.0146)
Age	0.0164***	0.0031***	0.0072***	0.0072***
	(0 .0013)	(0.0001)	(0.0008)	(0.0006)
Female=1	-0.0189	-0.0022	-0.0188*	-0.0176**
	(0.0187)	(0.0034)	(0.0108)	(0 .0077)
White=1	-0.0317*	-0.0030	-0.0158	-.0153**
	(0.0190)	(0.0034)	(0.0107)	(.0077)
Obs.	3696	3696	3696	3696
Kleibergen Paap rk Wald F statistic			210.67	210.67
Stock-Yogo test *Critical value (r=0.10)*				
at sign. level of 0.05%			7.03	7.03
Excluded Instruments :				
Residual	No	No	Yes	Yes
Fast food de-meaned	No	No	Yes	Yes

*** Significant at 1% level ** Significant at 5% level * Significant at 10% level.
All S.E are robust and clustered at individual level.
GMM weighted matrix clustered at the individual level.
1848 observations per wave.

APPENDIX

A.Quasi Maximum Likelihood (QML) of the Peer Effects Model

Let us rewrite equation (7) for convenience:

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

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:

$$\mathbf{M}_l^*\mathbf{y}_l^* = \beta\mathbf{M}_l^*\mathbf{G}_l^*\mathbf{y}_l^* + \mathbf{M}_l^*\mathbf{X}_l^*\gamma + \mathbf{M}_l^*\mathbf{G}_l^*\mathbf{X}_l^*\delta + \boldsymbol{\nu}_l^*, \tag{12}$$

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

Assuming that $\boldsymbol{\nu}_l^*$ is a n_l^*-dimensional *i.i.d* distributed disturbance vector, the log-likelihood function of (12) is given by:

$$\ln \mathbb{L} \quad = \quad \frac{-n^*}{2}\ln\left(2\pi\sigma^2\right) \; + \; \sum_{l=1}^{L}\ln|\mathbf{I}_{n_l^*} - \beta\mathbf{G}_l^*| \; + \; \sum_{l=1}^{L}\ln|\mathbf{I}_{n_l^*} - \rho\mathbf{M}_l^*| \; - \; \frac{1}{2\sigma^2}\sum_{l=1}^{L}\boldsymbol{\nu}_l^{*\prime}\boldsymbol{\nu}_l^*, \tag{13}$$

where $n^* = \sum_{l=1}^{L} n_l^* = N - L$, and, from (12), $\boldsymbol{\nu}_l^* = \mathbf{M}_l^*(\mathbf{y}_l^* \; - \; \beta\mathbf{G}_l^*\mathbf{y}_l^* \; - \; \mathbf{X}_l^*\gamma \; - \; \mathbf{G}_l^*\mathbf{X}_l^*\delta)$. Maximizing (13) with respect to $(\beta, \boldsymbol{\gamma}', \boldsymbol{\delta}', \rho, \sigma)$ yields the maximum likelihood estimators of the model. Interestingly, the QML 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. To compute robust standard errors, we use use a sandwich form $A^{-1}BA^{-1}$, where A is minus the expectation of the Hessian matrix and B is the expectation of the outer product of the gradient matrix. An advantage of this approach is that it allows us to obtain robust standard errors that are not driven by the normality assumption that ML imposes on the error term.

B. Generalized Spatial Two Stage Least Squares (GS-2SLS) of the Peer Effects Model

To estimate the model (12), 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.[32] 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 $\mathbf{S} = \left[\mathbf{X}^* \quad \mathbf{G}^*\mathbf{X}^* \quad \mathbf{G}^{*2}\mathbf{X}^*\right]$. 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 $\mathbf{G}^*$):

$$\mathbf{G}^*\hat{\mathbf{y}}^* = \mathbf{G}^*(\mathbf{I} - \hat{\beta}_1\mathbf{G}^*)^{-1}(\mathbf{X}^*\hat{\boldsymbol{\gamma}}_\mathbf{1} + \mathbf{G}^*\mathbf{X}^*\hat{\boldsymbol{\delta}}_\mathbf{1}). \tag{14}$$

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).

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