Heterogeneous peer effects and gender-based interventions for teenage obesity*

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Abstract

This paper explores the role of gender heterogeneity in the social diffusion of obesity among adolescents and its policy implications. We propose a generalized linear social interaction model which allows for gender-dependent heterogeneity in peer effects through the channel of social synergy. We estimate the model using data on adolescent Body Mass Index and network-based interactions. Our results show that peer effects are gender-dependent, and male students are particularly responsive to the weight of their female friends. Our simulations indicate that female-tailored interventions are likely to be more effective than a gender-neutral approach to fighting obesity in schools.

JEL codes: L12, C31, Z13, D85
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1 Introduction

Obesity has reached epidemic proportions in children and adolescents in the United States, increasing from 5% in 1980 to over 19% in 2018 (Skinner et al. 2019; Fryar, Carroll, and Afful 2020). Mounting evidence suggests that the extra pounds often start children on the path to health problems such as cardiovascular diseases, diabetes, and cancer (Bendor et al. 2020) raising the medical cost due to obesity among adults in the U.S. to $260.6 billion in 2016 (Cawley et al. 2021). To explain such an alarming phenomenon, a large number of studies have focused on socio-economic factors such as growing unhealthy eating habits and the decline in time spent doing physical exercise (Papoutsi, Drichoutis, and Nayga 2013).

Complementary to these views, health economists have also attempted to investigate the obesity epidemic from the perspective of social interactions (Christakis and Fowler 2007; Halliday and Kwak 2009; Trogdon, Nonnemaker, and Pais 2008; Yakusheva, Kapinos, and Eisenberg 2014; Cohen-Cole and Fletcher 2008; Fortin and Yazbeck 2015). Most of these studies document the presence of positive and significant peer effects which could increase the prevalence of obesity by changing reference norms for body image and/or by boosting the social transmission of unhealthy habits related to diet and physical activity.[1] Our paper follows the second strand of the literature by exploring the role of gender heterogeneity in the social diffusion of Body Mass Index (BMI) outcomes among teenagers,[2] and its consequences in terms of anti-obesity interventions.

Most studies on peer effects assume social interactions to be *homogeneous* (Manski 1993; Bramoullé, Djebbari, and Fortin 2009; Blume et al. 2015; De Paula 2017). This means that the effects of all peers are equal regardless of the particular type, such as race or gender. However, this assumption is restrictive and may not be realistic when speaking of the weight of adolescent students, arguably a period in life in which social interactions are important to structure an individual's body. In this context, heterogeneity in peer effects along gender lines could operate through different channels. One channel relates to the activities teenagers do together: for example, students may go to fast-food restaurants with peers of the same gender, or conversely, they may practice sport together with same-gender friends.[3] However, adolescents are also responsive to the BMI of opposite-gender peers for a variety of reasons related for instance to the influence of good eating habits and maturity of these peers.

The aims of this paper are threefold. First, we propose an econometric model which allows for *heterogeneous* peer effects along gender lines. Second, building on our identifica-

[1] Despite some studies have pointed to a virus (Rogers et al. 2007), the standard channel of social propagation of obesity is thought to be related to complementarities in behavior and/or self-image.

[2] Although various methods exist to measure excess body fat, BMI (kg/m2) is the most widely utilized measure of excess adiposity and risk for related diseases.

[3] Rees and Sabia (2010) document the heterogeneity in sport participation along gender lines, using the same Add Health data we use.

tion analysis, we estimate this model using detailed network data on teenagers' friendship from the Add Health dataset. Third, based on simulations using our model results, we show that ignoring gender-based heterogeneity of peer effects may lead to inefficient health interventions to curb obesity. Let us focus on each of these goals in turn.

Firstly, to our knowledge, we are the first to explore gender heterogeneity in peer effects on teenage body size. While the literature on dietary choices and weight outcomes of adolescents is sizable (Kapinos and Yakusheva 2011; Mora and Gil 2013; Corrado, Distante, and Joxhe 2019; Fortin and Yazbeck 2015; Angelucci et al. 2019), studies focusing on the heterogeneity of peer effects are rare. Some contributions suggest that female adolescents are more responsive than male ones to their peers' weight-related outcomes (Arduini, Iorio, and Patacchini 2019; Renna, Grafova, and Thakur 2008; Yakusheva, Kapinos, and Eisenberg 2014). However, none of these studies allow heterogeneity in between-gender peer effects, as we do. In our model, two types of individuals (*i.e.*, male *vs.* female students) interact within the same network (*i.e.*, a school). This defines an (augmented) 'heterogeneous' model with two *within-gender* and two *between-gender* peer effects, with respect to the 'homogeneous' setting with one peer effect term. We characterize our model econometrically and theoretically. We first derive sufficient identification conditions for the instrumental variable strategy, and we propose an efficient GMM estimator with additional quadratic moments.

Our methodological approach is closely related to the ones developed by Hsieh and Lin (2017) and Arduini, Patacchini, and Rainone (2020), but with important differences. Hsieh and Lin (2017) model peer effects *via* Bayesian methods, and estimate them through Markov Chain Monte Carlo sampling techniques. Similarly to us, Arduini, Patacchini, and Rainone (2020) derive a set of identification conditions that generalize the standard linear model of Bramoullé, Djebbari, and Fortin (2009) to allow for heterogeneous peer effects. Their paper puts a strong accent on the asymptotic and finite-sample properties of the 2SLS estimator. In contrast, as we are interested in analyzing the impact of policy interventions on outcomes, our paper puts more emphasis on the micro-foundation of our econometric model. In particular, we show that our approach is consistent with the best response functions of a Nash non-cooperative model when social interactions display 'social synergy'. We show that our micro-theoretic model is identifiable as long our econometric model is identified and we have some information (proxy) on an individual's effort to influence his or her BMI outcome.

Secondly, we illustrate our econometric model using the 1996's saturation sample of the National Longitudinal Study of Adolescent Health (Add Health) which provides census data on 16 selected schools. Respondents from the sample reported their height and weight (which we use to compute the BMI), and they were also asked to *name up to five male friends and up to five female friends within their school*, which allows us to map the friendship networks. We first provide test-based evidence that homophily in link formation is not a concern in our setting: once we control for school-level fixed effects, the network appears to be conditionally exogenous for BMI determination. Our main results show that

that peers' outcomes (and characteristics) affect BMI in a way that is gender-specific. In particular, we find that the 'male-female' endogenous peer effect (that is, the effect on male students' BMI of the BMI of their female friends) is significantly larger than the other estimated peer effects (for male-male, female-male, female-female interactions respectively). This result is in line with Kooreman (2007) and Hsieh and Lin (2017) who find that the influence of female students on male students is generally larger than the reverse for a number of documented adolescent behaviors. This effect could be due to the fact that girls are more mature and presumably more influential than boys at the same age during childhood and adolescence. This hypothesis is consistent with recent studies in neuroscience (*e.g.,* Gong et al. 2009; Lenroot and Giedd 2010; Lim et al. 2015; Goyal et al. 2019) suggesting that girls tend to optimize brain connections earlier than boys.

Thirdly, we present a simulation exercise to show that acknowledging gender heterogeneity in the social diffusion of obesity could help design efficient interventions. We simulate the impact of an intervention proposing one balanced meal per week in replacement of one fast-food type serving. On the basis of our most conservative findings, we conclude that the social spillovers of offering meal replacement to female students on the BMI of others are 50% higher than the spillovers of males. This suggests that returns from (resources spent on treating) females are 9% larger than returns from males in terms of overall BMI decrease in the student population. If we further assume that females are more responsive to the intervention, we conclude that the social spillovers of females are twice the spillovers of males, which translates into a 55% gain in terms of aggregate BMI decrease from reaching out to female students. Our analysis indicates that ignoring gender-based heterogeneity of peer effects may lead to severe biases in the analysis of the impact of anti-obesity policies. More generally, while ex-ante evaluations which rely on structural models are common in other fields of economics (*e.g.*, Wolpin 2007), they are novel in the context of social interactions. By providing the infrastructure to evaluate how interventions interplay with heterogeneous social diffusion, we think that our paper brings an important contribution to policy making and can be of considerable interest in a wide variety of contexts where peer effects differ along individual dimensions (*e.g.*, race, education).

The rest of the paper is organized as follows. In section 2, we describe our econometric model. In section 3 we introduce the data and provide test-based evidence of network exogeneity. Section 4 presents our main results. Section 5 describes a simulation exercise to evaluate anti-obesity policies in presence of heterogeneous social spillovers along gender lines. Section 6 concludes. Appendix A shows how the heterogeneous model is micro-founded into an identifiable non-cooperative game of social synergy. Appendix B formalizes the identification conditions and illustrates the estimation techniques.

2 The Model

2.1 Setting

We study a setting where n agents (*e.g.*, students) are distributed across R social networks (*e.g.*, schools), with $r = 1, \ldots, R$. In a given network r of size n_r there are n_r^f female agents and n_r^m male agents ($n_r^f + n_r^m = n_r$).[4] These agents interact with both own-gender and other-gender peers and their outcome (*e.g.*, the BMI) can be influenced by their behavior.

For each network we define four fixed and known adjacency matrices: $\mathbb{A}_{z,r}(z = 1, \cdots, 4)$. The matrix $\mathbb{A}_{1,r}$ is such that $a_{1,r,ij} = 1$ if in network r the male student i is influenced by the male student j, and 0 otherwise.[5] The matrix $\mathbb{A}_{2,r}$ is such that $a_{2,r,ij} = 1$ if in network r the male student i is influenced by the female student j, and 0 otherwise. The matrices $\mathbb{A}_{3,r}$ and $\mathbb{A}_{4,r}$ are similarly defined for female students, that is, $\mathbb{A}_{3,r}$ represents the impact of female friends on female students, and $\mathbb{A}_{4,r}$ the impact of male friends on female students in network r. These matrices are directed: the fact that i influences j does not necessarily imply that j influences i (*e.g.*, we could have $a_{1,r,ij} \neq a_{1,r,ji}$).[6]

Let us call $n_{i,r}^m$ and $n_{i,r}^f$ the number of male and female individuals influencing i in the network r respectively. The social interaction matrix $\mathbb{G}_{z,r}$ is the weighted version of matrix $\mathbb{A}_{z,r}$ such that one has $g_{1,r,ij} = 1/(n_{i,r}^m + n_{i,r}^f)$ if i is a male student in network r and is influenced by the male student j, and 0 otherwise. Since we allow for individuals to be 'isolated', that is, not influenced by anyone in their network (*i.e.*, $n_{i,r}^m = n_{i,r}^f = 0$), the $\mathbb{G}_{z,r}$'s matrices are not row-normalized (*i.e.*, not all matrix's rows sum up to one). Thus, the social interaction matrix for the whole population in network r could be written as $\mathbb{G}_r = \mathbb{G}_{1,r} + \mathbb{G}_{2,r} + \mathbb{G}_{3,r} + \mathbb{G}_{4,r}$. Our heterogeneous peer effect model for the network r writes as

$$\begin{aligned}\mathbf{y_r} = \quad & \boldsymbol{\iota}_{n_r}\alpha_r + \beta_{mm}\mathbb{G}_{1,r}\mathbf{y}_r \; + \; \beta_{mf}\mathbb{G}_{2,r}\mathbf{y}_r \; + \; \beta_{ff}\mathbb{G}_{3,r}\mathbf{y}_r \; + \; \beta_{fm}\mathbb{G}_{4,r}\mathbf{y}_r \; + \\ & \gamma\ \mathbf{x}_r + \delta_{mm}\mathbb{G}_{1,r}\ \mathbf{x}_r \; + \; \delta_{mf}\mathbb{G}_{2,r}\ \mathbf{x}_r \; + \; \delta_{ff}\mathbb{G}_{3,r}\mathbf{x}_r \; + \; \delta_{fm}\mathbb{G}_{4,r}\ \mathbf{x}_r \; + \boldsymbol{\epsilon}_r, \qquad (1)\end{aligned}$$

where $\mathbf{y}_r$ is the BMI vector and $\boldsymbol{\iota}_{n_r}$ is a $n_r \times 1$ vector of ones. α_r stands for a fixed effect specific to network r, which takes into account the unobserved factors which commonly influence the BMI of all students within a school. The βs coefficients represent the 'endogenous' peer effects (*i.e.*, the effect of peers' outcomes) which are heterogeneous. For instance, β_{mm} measures the effect of the outcome of male peers on (the BMI of) male students. In the same way, β_{mf} stands for the effect of the outcomes of female peers on male students, β_{ff} of female peers on female students, and β_{fm} of male peers on female

[4] In what follows, we order all vector and matrices so that the first n_r^f rows correspond to female agents of network r, and the remaining n_r^m rows are for male agents in network r.

[5] The student i is excluded from his/her own reference group.

[6] This is because in our illustration we choose to use information on social links as declared by respondents and the two reports may not coincide within a dyad. Nevertheless, our estimation strategy is also compatible with undirected network data.

students. Without loss of generality and for notational simplicity, we assume a unique individual characteristic.[7] We also allow for heterogeneous contextual effects δs that account for the effect of the characteristic of peers on student's outcomes that reads the same way (*e.g.*, δ_{mm} measures the effect of the characteristic of male peers on the outcome of male students).

Finally, if we observe $R > 1$ distinct networks, we can stack up the data and write the heterogeneous model succinctly as

$$\mathbf{y} = \bar{\mathbb{G}}(\boldsymbol{\beta})\mathbf{y} + \gamma\mathbf{x} + \bar{\mathbb{G}}(\boldsymbol{\delta})\mathbf{x} + \boldsymbol{\iota}\boldsymbol{\alpha} + \boldsymbol{\epsilon} \tag{2}$$

under the assumption that $\mathbb{E}(\boldsymbol{\epsilon}|\mathbf{x}, \iota\alpha, \bar{\mathbb{G}}_z, z = 1, \cdots, 4) = 0$, and where $\mathbf{y} = (\mathbf{y}_1^{'}, ..., \mathbf{y}_R^{'})'$, $\mathbf{x} = (\mathbf{x}_1^{'}, ..., \mathbf{x}_R^{'})'$, $\boldsymbol{\iota} = D(\boldsymbol{\iota}_{n_1}, ..., \boldsymbol{\iota}_{n_R})$ where D indicates a block diagonal matrix, $\boldsymbol{\alpha} = (\alpha_1, ..., \alpha_R)'$, $\boldsymbol{\epsilon} = (\boldsymbol{\epsilon}_1^{'}, ..., \boldsymbol{\epsilon}_R^{'})'$, $\boldsymbol{\beta} = (\beta_{mm}, \beta_{mf}, \beta_{ff}, \beta_{fm})'$, $\boldsymbol{\delta} = (\delta_{mm}, \delta_{mf}, \delta_{ff}, \delta_{fm})'$, $\bar{\mathbb{G}}_z = D(\mathbb{G}_{z,1}, ..., \mathbb{G}_{z,R})(z = 1, \cdots, 4)$, $\bar{\mathbb{G}}(\boldsymbol{\beta}) = \beta_{mm}\bar{\mathbb{G}}_1 + \beta_{mf}\bar{\mathbb{G}}_2 + \beta_{ff}\bar{\mathbb{G}}_3 + \beta_{fm}\bar{\mathbb{G}}_4$ and $\bar{\mathbb{G}}(\boldsymbol{\delta}) = \delta_{mm}\bar{\mathbb{G}}_1 + \delta_{mf}\bar{\mathbb{G}}_2 + \delta_{ff}\bar{\mathbb{G}}_3 + \delta_{fm}\bar{\mathbb{G}}_4$.

The conditional exogeneity assumption $\mathbb{E}(\boldsymbol{\epsilon}|\mathbf{x}, \iota\alpha, \bar{\mathbb{G}}_z, z = 1, \cdots, 4) = 0$ means that the network represented by the social interaction matrices $\bar{\mathbb{G}}_z$, for $z = 1, \cdots, 4$ is taken as exogenous relative to the random term $\boldsymbol{\epsilon}$, once we control for individual characteristics $\mathbf{x}$ and school-level fixed effects. This issue will be discussed in Subsection 3.3.

Note that if we impose $\beta_{mm} = \beta_{mf} = \beta_{ff} = \beta_{fm} = \beta_h$ and $\delta_{mm} = \delta_{mf} = \delta_{ff} = \delta_{fm} = \delta_h$ in equation (2), we obtain the so-called 'homogeneous' model

$$\mathbf{y} = \beta_h\bar{\mathbb{G}}\mathbf{y} + \gamma\mathbf{x} + \delta_h\bar{\mathbb{G}}\mathbf{x} + \boldsymbol{\iota}\boldsymbol{\alpha} + \boldsymbol{\epsilon}. \tag{3}$$

This corresponds to the specification by Bramoullé, Djebbari, and Fortin (2009) with fixed effects and will be used as benchmark for our empirical analysis in Section 4.

In Appendix A we micro-found Equation 2 in a non-cooperative model where peer effects work through the channel of strategic complementarity ('social synergy') in BMI within the social network. The assumption of social synergy is plausible in our context, because body size can only be indirectly chosen through effort, that is, healthy life habits (*e.g.*, good dietary behavior, physical exercise). We show that we can identify all parameters of the utility function, provided that we have a good proxy for individuals' effort, and discuss the relevance of that in terms of policy evaluation.

2.2 GMM Estimation Procedure

This section describes the details of our estimation procedure. We first derive the conditions under which the parameters of equation (2) are identified. We then discuss the efficient GMM estimator with additional quadratic moment restrictions. The methods we detail here will be the basis for our empirical exercise in Section 4. All proofs are relegated to Appendix B.

[7] In our empirical analysis (section 4), we allow for many individual characteristics.

Assuming that the matrix $\mathbf{S}(\boldsymbol{\beta}) = (\mathbf{I} - \bar{\mathbb{G}}(\boldsymbol{\beta}))$, where $\mathbf{I}$ is the identity matrix, is invertible,[8] we can write the reduced form of equation (2) as

$$\mathbf{y} = \mathbf{S}(\boldsymbol{\beta})^{-1}\left[\gamma\mathbf{x} + \bar{\mathbb{G}}(\boldsymbol{\delta})\mathbf{x} + \boldsymbol{\iota}\boldsymbol{\alpha}\right] + \mathbf{S}(\boldsymbol{\beta})^{-1}\boldsymbol{\epsilon}. \tag{4}$$

Equation (4) allows us to rewrite

$$\bar{\mathbb{G}}_z\mathbf{y} = \mathbf{W}_z(\boldsymbol{\beta})\left[\gamma\mathbf{x} + \bar{\mathbb{G}}(\boldsymbol{\delta})\mathbf{x} + \boldsymbol{\iota}\boldsymbol{\alpha}\right] + \mathbf{W}_z(\boldsymbol{\beta})\boldsymbol{\epsilon},$$

where $\mathbf{W}_z(\boldsymbol{\beta}) = \bar{\mathbb{G}}_z\mathbf{S}(\boldsymbol{\beta})^{-1}$ and $z = 1, \cdots, 4$. This illustrates that the right-hand side terms in equation (2) are endogenous ($\mathbb{E}\left[(\mathbf{W}_z(\boldsymbol{\beta})\boldsymbol{\epsilon})'\boldsymbol{\epsilon}\right] \neq 0$), so that the model cannot be consistently estimated by OLS. However, it can be estimated with instrumental variables techniques, as we explain in what follows. Let us rewrite equation (2) more succinctly as

$$\mathbf{y} = \mathbf{Z}\boldsymbol{\theta} + \boldsymbol{\iota}\boldsymbol{\alpha} + \boldsymbol{\epsilon}, \tag{5}$$

with $\mathbf{Z} = \left[\bar{\mathbb{G}}_1\mathbf{y}, \bar{\mathbb{G}}_2\mathbf{y}, \bar{\mathbb{G}}_3\mathbf{y}, \bar{\mathbb{G}}_4\mathbf{y}, \mathbf{X}\right]$, $\mathbf{X} = \left[\mathbf{x}, \bar{\mathbb{G}}_1\mathbf{x}, \bar{\mathbb{G}}_2\mathbf{x}, \bar{\mathbb{G}}_3\mathbf{x}, \bar{\mathbb{G}}_4\mathbf{x}\right]$, $\boldsymbol{\theta} = (\boldsymbol{\beta}, \gamma, \boldsymbol{\delta})'$. In order to eliminate the fixed effects $\boldsymbol{\iota}\boldsymbol{\alpha}$ avoiding the incidental parameters problem, we perform a global transformation on equation (5).[9] For that purpose we define the global transformation matrix $\mathbf{J} = D(\mathbf{J}_1, ..., \mathbf{J}_R)$ where $\mathbf{J}_r = (\mathbf{I}_r - \frac{\boldsymbol{\iota}_r\boldsymbol{\iota}_r'}{n_r})\ \forall\ r \in \{1, ..., R\}$, such that $\mathbf{J}\boldsymbol{\iota}\boldsymbol{\alpha} = \mathbf{0}$.[10] The transformed model reads as

$$\mathbf{J}\mathbf{y} = \mathbf{J}\mathbf{Z}\boldsymbol{\theta} + \mathbf{J}\boldsymbol{\epsilon}. \tag{6}$$

Following Liu and Lee (2010), one can write

$$\mathbf{Q}_\infty = \mathbf{J}\left[\{\mathbf{Q}_{z,\infty}\}_{\{z=1,2,3,4\}}, \mathbf{X}\right] \tag{7}$$

where $\mathbf{Q}_{z,\infty} = [\mathbf{Q}^0_{z,\infty}\mathbf{x}, \mathbf{Q}^0_{z,\infty}\boldsymbol{\iota}]$ and $\mathbf{Q}^0_{z,\infty}$ contains all products of the social interaction matrices of arbitrary order[11]

$$\begin{aligned}\mathbf{Q}^0_{z,\infty} \quad = \quad & [\bar{\mathbb{G}}_z\bar{\mathbb{G}}_1, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_2, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_3, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_4, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_1^2, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_2\bar{\mathbb{G}}_4,\\ & \bar{\mathbb{G}}_z\bar{\mathbb{G}}_3^2, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_3\bar{\mathbb{G}}_4, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_4\bar{\mathbb{G}}_1, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_4\bar{\mathbb{G}}_2, \bar{\mathbb{G}}_z\bar{\mathbb{G}}_1^3, ...]\end{aligned}$$

[8] A sufficient condition for this assumption to hold is that $|\beta_{mm}| < 1$, $|\beta_{mf}| < 1$, $|\beta_{ff}| < 1$ and $|\beta_{fm}| < 1$. This condition also implies that the matrix $\mathbf{S}(\boldsymbol{\beta})$ is uniformly bounded in absolute value. See a discussion on this point in Arduini, Patacchini, and Rainone (2020).

[9] The incidental parameters problem, as it was defined by **incidental**, and discussed at length in Lancaster (2000), occurs whenever the data available for each group or network are finite.

[10] Note that this transformation only captures the selection bias stemming from the fact that individuals in the same network face a common environment. It does not address the problem of network endogeneity, which is discussed in section 3.3.

[11] Recall that the matrix ordering leads by construction to the following identities: $\bar{\mathbb{G}}_1\bar{\mathbb{G}}_4 = 0_{n_r}$, $\bar{\mathbb{G}}_3\bar{\mathbb{G}}_2 = 0_{n_r}$, $\bar{\mathbb{G}}_1\bar{\mathbb{G}}_3 = 0_{n_r}$, $\bar{\mathbb{G}}_3\bar{\mathbb{G}}_1 = 0_{n_r}\bar{\mathbb{G}}_2\bar{\mathbb{G}}_1 = 0_{n_r}$, $\bar{\mathbb{G}}_4\bar{\mathbb{G}}_3 = 0_{n_r}$,$\bar{\mathbb{G}}_2^2 = 0_{n_r}$,$\bar{\mathbb{G}}_4^2 = 0_{n_r}$.

Defining $\mathbf{Q}_K^z$ as a subset of $\mathbf{Q}_{z,\infty}$ of size K and $\mathbf{Q}_K = \mathbf{J}\left[\mathbf{Q}_K^1, \mathbf{Q}_K^2, \mathbf{Q}_K^3, \mathbf{Q}_K^4, \mathbf{X}\right]$, the 2SLS estimator of model (2) is

$$\hat{\boldsymbol{\theta}}_{2sls} = (\mathbf{Z}'\mathbf{P}_K\mathbf{Z})^{-1}\mathbf{Z}'\mathbf{P}_K\mathbf{y},$$

where $\mathbf{P}_K = \mathbf{Q}_K(\mathbf{Q}'_K\mathbf{Q}_K)^{-}\mathbf{Q}'_K$ and where

$$\mathbf{Q_K} = \mathbf{J}\left[\bar{\mathbb{G}}_1^2\mathbf{x}, \bar{\mathbb{G}}_3^2\mathbf{x}, \bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\mathbf{x}, \bar{\mathbb{G}}_2\bar{\mathbb{G}}_3\mathbf{x}, \bar{\mathbb{G}}_2\bar{\mathbb{G}}_4\mathbf{x}, \bar{\mathbb{G}}_3\bar{\mathbb{G}}_4\mathbf{x}, \bar{\mathbb{G}}_4\bar{\mathbb{G}}_1\mathbf{x}, \bar{\mathbb{G}}_4\bar{\mathbb{G}}_2\mathbf{x}\right].$$

$\mathbf{Q}_K$ includes matricial products up to the second order only, which is the set of instruments we use in Section 4. This is a particular case of the lagged-friend instrumental strategy which has been widely used in presence of network data (Calvo-Armengol, Patacchini, and Zenou 2009; Kelejian and Prucha 1998; Patacchini and Zenou 2012). Under plausible regularity conditions (Liu and Lee 2010), this approach provides a consistent estimator, provided that the model is identified. The identification conditions for equation (2) are formalized in Proposition 1 below, which extends the conditions derived by Bramoullé, Djebbari, and Fortin (2009) to the case of peer effects heterogeneity. For a detailed discussion, we remand to the proof in Appendix B.

Proposition 1 *Suppose model (2) holds with no correlated effects. Suppose that $\bar{\mathbb{G}}(\boldsymbol{\beta})$ is invertible and that $(\delta_{mm} + \gamma\beta_{mm}) \neq 0$, $(\delta_{ff} + \gamma\beta_{ff}) \neq 0$, $(\delta_{mf} + \gamma\beta_{mf}) \neq 0$ and $(\delta_{fm} + \gamma\beta_{fm}) \neq 0$. If vector columns of matrix $\mathbf{Q}_K$ are linearly independent, then social effects are identified.*

Following Liu and Lee (2010) in the context of homogeneous best-response functions, we propose a GMM estimator of our heterogeneous model with additional quadratic moment equations. These quadratic moments exploit the existing correlations between the error term of the reduced form model, thus provide more precision compared to the traditional 2SLS estimators.[12]

Let the IV moments be given by the expression $g_1(\boldsymbol{\theta}) = \mathbf{Q}'_K\boldsymbol{\epsilon}(\boldsymbol{\theta})$ where $\boldsymbol{\epsilon}(\boldsymbol{\theta}) = \mathbf{J}(\mathbf{y} - \mathbf{Z}\boldsymbol{\theta} - \boldsymbol{\iota\alpha})$. The additional quadratic moments are given by the expression $g_2(\boldsymbol{\theta}) = \left[\mathbf{U}'_1\boldsymbol{\epsilon}(\boldsymbol{\theta}), \mathbf{U}'_2\boldsymbol{\epsilon}(\boldsymbol{\theta}), ..., \mathbf{U}'_q\boldsymbol{\epsilon}(\boldsymbol{\theta})\right]'\boldsymbol{\epsilon}(\boldsymbol{\theta})$, where $\mathbf{U}_j$ is such that $tr(\mathbf{J}\mathbf{U}_j) = 0$.[13] In addition, let the combined vector of linear and quadratic empirical moments be given in $g(\boldsymbol{\theta}) = [g'_1(\boldsymbol{\theta}), g'_2(\boldsymbol{\theta})]$. Finally, let $\widetilde{\Omega} = \widetilde{\Omega}(\tilde{\sigma}^2, \tilde{\mu}_3, \tilde{\mu}_4)$ where $\tilde{\sigma}^2$, $\tilde{\mu}_3$ and $\tilde{\mu}_4$ are initial estimators of the second, third and fourth moments of the error term of our model. Following Liu, Patacchini, and Rainone (2017), extented to the case of heterogeneous peer effects, the optimal weighting matrix associated with our GMM estimation strategy is given by

$$\Omega = Var\left[g(\boldsymbol{\theta})\right] = \begin{bmatrix} \tilde{\sigma}^2\mathbf{Q}'_K\mathbf{Q}_K & \mu_3\mathbf{Q}'_K\omega \\ \mu_3\omega'\mathbf{Q}_K & (\mu_4 - 3\sigma^4)\omega'\omega + \sigma^4\Upsilon \end{bmatrix},$$

[12] An additional advantage is that the objective function of the GMM estimator uses the optimal weighting matrix that increases efficiency with respect to the 2SLS case.

[13] Following Liu and Lee (2010), we use $\mathbf{U}_k = \bar{\mathbb{G}}_k - tr(\mathbf{J}\mathbf{G}_k)\mathbf{I}/tr(\mathbf{J})$ for $k = 1, ..., 4$.

where $\omega = [vec_D(\mathbf{U}_1), vec_D(\mathbf{U}_2), ..., vec_D(\mathbf{U}_q)]$, $\mathbf{E}^s = \mathbf{E} + \mathbf{E}'$, $\forall$ square matrix $\mathbf{E}$ of size n, $vec_D(\mathbf{A}) = (a_{11}, a_{22}, ..., a_{nn})$ and $\Upsilon = \frac{1}{2}\left[vec(\mathbf{U}_1^s), vec_D(\mathbf{U}_2^s), ..., vec_D(\mathbf{U}_q^s)\right]$. The feasible optimal GMM estimator is given by

$$\hat{\boldsymbol{\theta}}_{gmm} = argmin\ _{\boldsymbol{\theta}\in\Theta} g'(\boldsymbol{\theta})\widetilde{\Omega}^{-1} g(\boldsymbol{\theta})$$

which will be implemented in our estimates of Section 4.

3 Data

3.1 Add Health

In this section we use data from the National Longitudinal Study of Adolescent Health (Add Health) to study the heterogeneous influences of peers on the body weight of adolescents. Add Health is a panel study of a nationally representative sample of adolescents in grades 7-12 in the United States, conducted by the Carolina Population Center. It combines information on respondents' social, economic, psychological and physical well-being with data on family, neighborhood, community, school, friendships, peer groups, and romantic relationships. The richness of this information puts Add Health among the largest and most comprehensive longitudinal surveys of adolescents ever undertaken.

Wave I of Add Health consists of an In-school questionnaire that was filled out by 90,118 students in 145 schools and 80 communities during the 1994-1995 school year. A subset of these students was then chosen for an in-depth survey: Wave II, which was held in 1996, includes a detailed In-Home questionnaire that was completed overall by 14,738 students out of the original Wave I pupils. Students who were selected for the In-Home survey were asked for information on their height and weight, which can be used to compute body mass indices (BMI). Wave II also provides information on social interactions, because respondents are asked to *name up to five male friends and up to five of their female friends within their school.* Other notable covariates include age, racial background, grade variables, parents' education and health status.

For the purpose of our analysis, we use the saturated sample of Wave II that focuses on 16 selected schools. Every student attending these 16 schools answered the In-Home questionnaire, thus providing information on BMI and social links. We construct student BMI according to the formula: BMI = (weight in kilograms)/(height in meters)^2.[14] Having a census of the schools' population (rather than a random sample of students within a given school) is crucial for our estimation strategy, because we can fully reconstruct friendship

[14]We do not use self-declared body mass indices, although declared BMIs are shown to reflect real variables in the context of Add Health.

interaction matrices. In fact, our estimation strategy crucially relies on the whole network topology (because friends of friends could have an indirect influence on one's behavior).[15]

3.2 Descriptive statistics

The saturation sample comprises 2220 students in all 16 schools of the Wave II In-Home survey. For the purpose of our estimation, we retain 1914 students (so 87% of the whole sample) for which we can trace some social interaction. This means that these 1914 students *either* mention some friends within the school *or* they are mentioned as friends by someone within the school (or both). Note, however, that our interaction matrices represent *directed* links (*e.g.*, $g_{ij} > 0$ if student i is influenced by student j, but not necessarily *vice versa*). Thus we still retain in our sample 203 students that are 'isolated' because they are not influenced by any peer within the boundaries of their school.[16]

Table (1) provides descriptive statistics of our estimation sample of 1914 students. Average BMI is 23.1 with a standard deviation of 4.79. This reveals that on average, the population considered is *normal* in terms of weight. In terms of individual characteristics, we can see that the male-female population is equally distributed, and that mean age is about 16. White students are more represented (64%) than the other racial communities. The percentage of Black, Asian and Hispanic students is 14%, 14% and 16% respectively. 61% of students in our sample attend grade 11 or 12 and 27% are in grade 9 or 10. Most of the parents hold at least a high school degree and 19% of mothers hold a college degree compared to 16% for fathers of the students in our sample. 92% of mothers report being in good health compared to 78% of fathers.

Statistics about the directed network point to more links with same-gender friends: males have on average 1.72 links with males and 0.84 with females, while females have 1.63 links with females and 0.91 with males. The total number of friends is similarly distributed between male and female friends. This also suggests that the constraint put in the number of friends by the Add Health study is not binding, as individuals actually report having fewer friends than the number of allowed nominations during the survey.[17]

3.3 Network endogeneity

In everyday life individuals with common preferences or characteristics tend to associate together. This well-documented tendency which is named 'homophily' could be a source

[15] Thanks to census data, we avoid making strong distributional assumptions to deal with sampled dyadic observations (Chandrasekhar and Lewis 2016).

[16] Following Lin (2010), we exclude those students who do not cite anyone *and* are not cited by anyone (13% of the sample). These observations could in fact disguise measurement errors in social links or differences in underlying preferences.

[17] This alleviates the concern that the network may be only partially observed. Also, it is worth noting that censoring leads to an underestimation of the magnitude of peer effects, as shown by Griffith (Forthcoming) using Add Health data. This is reassuring in our context where peer-effect estimates are significantly positive.

of estimation bias when individuals self-select into social links. In particular, endogeneity arises whenever individual-level unobservables simultaneously determine link formation and the outcome of interest. As in equations (2) or (3) we assume that the social network is exogenous, it is essential to provide evidence that this assumption is not rejected in the context of our analysis.

The issue of network endogeneity has recently been addressed by a number of papers. One strategy uses a control approach and adopts a Bayesian method based on a full parametric specification of the network formation and the outcome equation (*e.g.*, Goldsmith-Pinkham and Imbens 2013; Patacchini and Rainone 2017; Hsieh and Lee 2016). Another approach also controls for the endogeneity of the social links using a control function but adopting a frequentist approach based on a nonparametric specification of the network formation (*e.g.* Johnsson and Moon 2021). Our approach is test-based, in the sense that we implement two different testing strategies that indicate that network endogeneity is not a concern in our setting. More precisely, our results suggest that once we control for school-level effects, our network appears to be *conditionally* exogenous. We discuss the results for the homogeneous peer-effect model, which should reassure the reader for the heterogeneous model too.

The first strategy follows Liu, Patacchini, and Rainone (2017), Goldsmith-Pinkham and Imbens (2013), and Boucher and Fortin (2016). The intuition goes as follows. Let us focus on equation (3) for a given network r, and suppose that $\boldsymbol{\epsilon}_r = \pi \mathbf{v}_r + \mathbf{e}_r$, where $\mathbf{v}_r$ is an unobserved attribute at the individual level and $\mathbf{e}_r$ is a vector of random disturbances. Let us further assume that the link formation process between any two individuals depends on distances in observed and unobserved characteristics as in equation (8) below:

$$g_{ij,r} = \kappa + \sum_{k=1}^{K} \zeta_m |x_{i,r}^k - x_{j,r}^k| + \phi |v_{i,r} - v_{j,r}| + \kappa_r + u_{ij,r}. \tag{8}$$

If there is homophily along unobserved characteristics, then we must observe that $\phi < 0$, *i.e.*, the closer two individuals are in terms of unobservables, the higher the probability that they become friends. If, in addition, $\pi \neq 0$, these unobservables have a direct effect on $\mathbf{y}_r$ as well. Thus, if data reveal a positive and statistically significant correlation between the predicted probability to observe a link between the two individuals ($\hat{g}_{ij,r}$) and the difference between their residuals in the outcome equation ($|\hat{\epsilon}_{i,r} - \hat{\epsilon}_{j,r}|$), conditional on whether the link is observed or not ($g_{ij,r} = 1$ vs. $g_{ij,r} = 0$), then we cannot reject the presence of endogeneity in network formation. Following this idea, we first perform a naive regression of the predicted probability to observe a link ($\hat{g}_{ij,r}$) and differences in residuals for the entire sample ($|\hat{\epsilon}_i - \hat{\epsilon}_j|$). We also include a binary variable g_{ij} indicating whether the link is formed, and we differentiate between cases where school-level fixed effects are included and cases where there are no fixed effects. Our results reported in Table (2) suggest that, in the absence of fixed effects, there is a negative and significant effect of differences in residuals and the predicted probability to observe a link. However, whenever fixed effects are accounted for, the effect is no longer significant. This suggests that these fixed effects capture all unobserved heterogeneity that is relevant for BMI determination and common to all students within the same school.

We then refine the testing strategy by focusing on the whole distribution of predicted probabilities to observe a link.[18] Our test is based on a visual observation strategy aiming at detecting the presence of endogeneity in network formation. The idea is that if the estimated Kernel densities are visually similar for both $g_{ij,r} = 1$ and $g_{ij,r} = 0$, then there is no evidence of network endogeneity. Figure (1) summarizes the results of our nonparametric estimation without fixed effects. We can see that the two Kernel density estimates are not similar without the inclusion of school fixed effects. However, once we control for school fixed effects (Figure 2), densities get visually similar.

Overall, based on the results above we conclude that the fixed-effect strategy is rather efficient in reducing the selection bias associated with confounding correlates, and there is no evidence of the residual endogeneity as related to students' BMI in our data. This is in line with several recent papers concluding against a severe assortativity bias in Add Health data (Goldsmith-Pinkham and Imbens 2013; Boucher 2016; Badev 2021).[19]

4 Results

4.1 Homogenous peer effects and BMI

Table (3) presents the GMM estimates from the homogeneous peer effects model of equation (3), with fixed effects at the level of the school. The set of characteristics $\mathbf{x}$ comprises: student attributes (age, race, grade),[20] education level and health status of mother and father respectively.[21] We use as instruments for $\bar{\mathbb{G}}\mathbf{y}$ all lagged-friends characteristics of the second degree, that is, the (average) attributes of friends of friends $\bar{\mathbb{G}}^2\mathbf{x}$. The weak instrument test statistic is reported at the bottom of the table.

Results indicate that the coefficient associated with the endogenous peer effect ($\bar{\mathbb{G}}\mathbf{y}$) is significant at 1%. Its estimated magnitude suggests that, *ceteris paribus*, a 1-unit increase in the average BMI of peers induces an increase of 0.208 units in the student's BMI. This is aligned with the recent literature reporting evidence of positive but small endogenous peer effects on weight. Under social synergy preferences, this implies a so-called social multiplier

[18]This follows Liu, Patacchini, and Rainone (2017), who split the overall sample into buckets according to the thresholds of values of the predicted probabilities to observe a link. We concentrate on the whole distribution to avoid defining arbitrary thresholds.

[19]Boucher and Fortin (2016) suggest that with a rich set of control variables as those that can be used in our data set, the impact of homophily may be small. Other studies using different data sets reach the opposite conclusion (*e.g.*, Carrell, Sacerdote, and West (2013) and Hsieh et al. (2020)).

[20]The omitted category for race includes Hispanic, Asian and American Indian respondents, while the omitted category for grade is "7 or 8".

[21]The reference category for parents' education is "*I don't know*". The variable representing parents' health status (as reported by the student) is a dummy.

equal to 1.23, *i.e.*, the total impact of a common shock on the aggregate outcome in a network is 1.23 times the sum of its direct effects at the individual level.[22]

We also remark that several individual and peer attributes appear to influence one's BMI. The first two columns report the estimates and standard errors of individual own characteristics $\mathbf{x}$, and columns 3 and 4 refer to the contextual effects, that is, effects of friends' characteristics $\bar{\mathbb{G}}\mathbf{x}$. We notice that white and black students have a lower BMI (relative to their Hispanic, Asian or American Indian friends), and the same holds for students in lower grades and whose father is in good health. Regarding contextual effects, several estimates appear statistically significant, including peers' age and parents' education. In particular, having friends whose mother has a college or an advanced level of education strongly reduces a student's BMI, which may indicate transmission of information *via* learning good health habit.

4.2 Gender heterogeneity and BMI

In this subsection, we present the estimates from the model allowing for within- and between-gender heterogeneity in peer effects. We also provide Wald statistics to test the homogeneous model against the heterogeneous model.

Table (4) provides the results from the GMM estimation of equation (2), with fixed effects at the level of the school. The set of instruments now includes all exogenous attributes of friends at distance 2, per category,that is: $\bar{\mathbb{G}}_1^2\mathbf{x}$ and $\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\mathbf{x}$ (the attributes of males/female friends of males friends of male students); $\bar{\mathbb{G}}_2\bar{\mathbb{G}}_4\mathbf{x}$ and $\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3\mathbf{x}$ (the attributes of males/female friends of females friends of male students); $\bar{\mathbb{G}}_4\bar{\mathbb{G}}_1\mathbf{x}$ and $\bar{\mathbb{G}}_4\bar{\mathbb{G}}_2\mathbf{x}$ (the attributes of males/female friends of males friends of female students), $\bar{\mathbb{G}}_3\bar{\mathbb{G}}_4\mathbf{x}$ and $\bar{\mathbb{G}}_3^2\mathbf{x}$ (the attributes of males/female friends of females friends of female students). The weak instrument test statistic is reported at the bottom of the table. The upper panel provides the four endogenous peer effects coefficients (standard errors of the estimates are reported in the adjacent columns), namely: the effects of male peers' BMI on the BMI of male students ($m-m$, columns 3 and 4), the effects of female peers' BMI on the BMI of male students ($m-f$, columns 5 and 6), the effects of female peers' BMI on the BMI of female students ($f-f$, columns 7 and 8) and the effects of male peers' BMI on the BMI of female students ($f-m$, columns 9 and 10).

As in the case of the homogeneous model, the endogenous peer effect estimates are positive and highly significant, suggesting that interaction with peers of all types influences a student's BMI. The within-gender point estimates are similar (the $m-m$ and $f-f$ coefficients are 0.214 and 0.225 respectively), and they are slightly larger but comparable for magnitude to the $f-m$ coefficient (0.184) which represents the effect of the average BMI of male peers on female student's BMI. On the other hand, the estimated coefficient for

[22]This is computed as $\frac{1}{1-0.208}\times 0.89 + 1\times 0.11$ because for isolated students (11%) the social multiplier is 1. This is in line with the results of Fortin and Yazbeck (2015) who estimate the social multiplier to 1.15 in a model where peer effects are homogeneous across gender and limited to fast food consumption.

the between-gender effect from females to males is noticeably larger (0.392). This suggests that males respond more to the average BMI of their female friends that the reverse, a result which is also obtained by Kooreman (2007) and Hsieh and Lin (2017) for several adolescent behaviors. As mentioned in the introduction, in our context this may be partly due to the fact that girls become mature and their brain reaches their peak volume earlier than boys in the adolescence.[23]

We report the estimates and standard errors related to individual characteristics in columns 1-2, and the ones for contextual effects (within- and between- gender) in columns 3 to 9. Grade 11-12 students are the ones who report a higher BMI (in line with the results from the homogeneous model), together with those whose father has no high school education (effect significant at 10% only). The other coefficients for the individual effects do not appear significant. Our results also reveal an important number of differences in the estimates of contextual effects depending on the nature (within- or between-gender) of social interactions. However, some regularities emerge in line with the results of the homogeneous model. For instance, the educational level of male-peer parents has a negative impact on a male student's BMI. Again, this may reflect the transmission of information on the benefits of good health habits. Furthermore, having male peers whose father holds some college degree negatively affects male students' BMI.[24]

Table (6) reports the results from a Wald test which rejects joint homogeneity in contextual and endogenous effects (statistics =120.71 as compared with a critical χ^2 value of 81.06 at the 1% significance level), as well as homogeneity in endogenous effects only (statistics =107.77 as compared with a critical χ^2 value of 16.81 at the 1% significance level). One thus concludes that gender heterogeneity is the appropriate hypothesis in our context. This result has potentially important consequences in terms of public policy evaluation, which we illustrate in the next section through a simulation exercise.

5 Gender-based Policy Evaluation

Interventions to curb obesity among teenagers may take various forms, aiming at improving health habits through action (*i.e.*, by changing the cafeteria menu, subsidizing gym access, *etc.*) or information (*i.e.*, educational campaigns about nutrition and healthy lifestyle). In what follows, we present a simulation exercise to illustrate the point that efficient interventions should account for gender heterogeneity in peer effects. We first show how to estimate the total treatment effect of an intervention when peer effects are heterogeneous along gender lines. We then describe the simulation procedure and discuss the results

[23] According to a neuroscience study by Lim et al. (2015), the optimizing of brain connectivity usually occurs during ages 10-12 in girls and 15-20 in boys. Girls also mature faster than boys on a physical level: girls undergo puberty earlier than boys by about 1-2 years and generally finish the stages of puberty quicker than males.

[24] We also perform a robustness analysis of our results when using the zBMI instead of absolute BMI, and the GMM estimation strategies reveals similar patterns.

under different hypotheses regarding the design and the response to the intervention.

5.1 Treatment effect

Let us assume we observe one population of teenage students with social synergy preferences connected in a social network where endogenous peer effects are at work. We want to assess the effect of an intervention designed to curb the prevalence of obesity among the target population, whose allocation is represented by the intent-to-treat vector $\boldsymbol{itt}$ ($itt_i = 1$ if student i is offered the intervention). We now assume that the intervention induces a gender-dependent shift in the BMI intercept, and look at the model:

$$\mathbf{y} = \boldsymbol{\iota\alpha} + \gamma \boldsymbol{itt} + \bar{\mathbb{G}}(\boldsymbol{\beta})\mathbf{y} + \boldsymbol{\epsilon} \tag{9}$$

where the coefficients $\boldsymbol{\gamma} = (\gamma_f, \gamma_m)$ represent the response to the intervention of (male, female) students.[25] In a linear intent-to-treat model without peer effects ($\mathbb{G}_z = 0_{n_r}$ for $z = 1, ..., 4$), the total treatment effect would be given by the coefficients $\boldsymbol{\gamma}$. In models with social lags in the dependent variable, the interpretation of the estimated parameters is complicated by the fact that the treatment status of an individual affects not only his own outcome (the *direct* effect), but also the outcome of others (the *indirect* effect). To define a measure of the treatment effect in our context, we start from the reduced form of equation (9):

$$\mathbf{y} = \mathbf{S}(\boldsymbol{\beta})^{-1}[\boldsymbol{\iota\alpha} + \gamma \boldsymbol{itt}] + \mathbf{S}(\boldsymbol{\beta})^{-1}\boldsymbol{\epsilon} \tag{10}$$

where $\mathbf{S}(\boldsymbol{\beta}) = [\boldsymbol{I} - \bar{\mathbb{G}}(\boldsymbol{\beta})]$, and derive the closed-form of the $N \times N$ matrix of partial derivatives with respect to the intervention, which we call $\frac{\partial E(\mathbf{y}|\boldsymbol{itt})}{\partial \boldsymbol{itt}}$. The k^{th} column of $\frac{\partial E(\mathbf{y}|\boldsymbol{itt})}{\partial \boldsymbol{itt}}$ is an $N \times 1$ vector that represents the effect of the treatment of individual $k = 1, ..., N$ on the outcomes of all other individuals and writes

$$\frac{\partial E\left(\mathbf{y}|\boldsymbol{itt}\right)}{\partial \boldsymbol{itt}_k} = \mathbf{S}(\boldsymbol{\beta})^{-1}[\gamma \mathbf{e}_k] \tag{11}$$

where $\mathbf{e}_k$ is an $N \times 1$ vector with 1 at the k^{th} element and 0 elsewhere. Following the practice in spatial and network econometrics (Hsieh and Lee 2016; LeSage and Page 2009; Comola and Prina 2021), we compute the treatment effect of the intervention as follows: the *direct* treatment effect is the average of the diagonal elements in $\frac{\partial E(\mathbf{y}|\boldsymbol{itt})}{\partial \boldsymbol{itt}}$. The *indirect* treatment effect, which operates through the change in the treatment status of peers, is the average of the column (or row) sums of the non-diagonal elements of $\frac{\partial E(\mathbf{y}|\boldsymbol{itt})}{\partial \boldsymbol{itt}}$.[26] The

[25] For the sake of simplicity, the only individual attribute we include is the treatment status, and we rule out contextual peer effects. This latter condition implies that the treatment status of peers only impacts own BMI through the changes in peers' BMI. Imposing positive contextual peer effects would increase the estimates of social spillovers in Section 5.3.

[26] The row sum represents the impact of changing the treatment status of all other individuals on the outcome of one particular individual, while the column sum represents the impact of changing the treatment status of one particular individual on the outcome of all other individuals. These two quantities coincide.

total treatment effect is then calculated as the sum of the direct and the indirect effects.[27] Note that the formula of equation (11) also applies to the homogeneous peer effect model of equation (3), once we replace $\beta_{mm} = \beta_{mf} = \beta_{ff} = \beta_{fm} = \beta_h$ in $\mathbf{S}(\boldsymbol{\beta})$.

5.2 Simulation Procedure

We now describe the details of our simulation exercise.

Routine

For given values of $\boldsymbol{\gamma}, \boldsymbol{\beta}$ we implement the following procedure:

1. Generate a dataset with N nodes, equally distributed between males and females, and multiple intent-to-treat vectors $\boldsymbol{itt^k}$ for $k = 1, ..., K$;

2. generate the interaction matrices as follows: first, we draw the binary matrices $\mathbb{A}_z$ as random graphs where each link exists independently with a probability p_z (Erdös and Rényi 1959). We then row-standardized $\mathbb{A}_z$ to obtain $\mathbb{G}_z$ for $z = 1, ..., 4$;[28]

3. compute the (direct, indirect, total) treatment effect from equation (11) for the homogeneous and heterogeneous model respectively, for: all students, males, females;

4. compute the aggregate decrease in BMI associated to each treatment vector itt_k;

5. repeat the procedure of steps (1) to (3) for $s = 1, ..., 500$ times.

Calibration

In order to calibrate γ we think of an intervention that replaces one fast-food type serving option with one balanced meal, following an experimented tradition of school-level interventions (*e.g.*, a weekly vegetarian menu in the cafeteria). We assume that the response to the intervention is given by

$$\gamma_g = impact_g * compliance_g \quad for\ g = m, f \tag{12}$$

where *impact* represents the gender-specific impact of the intervention (*e.g.*, one less fast-food meal could induce different changes on females' body size for reasons related to nutrition and biology), and *compliance* represents the propensity of students to comply with

[27] Note that the estimates of both the direct and indirect effects result from complex interactions between the parameters and the social-interaction structure. For instance, an arbitrary diagonal element does *not* necessarily equal the estimated coefficient γ, because the former also includes feedback loops (where observation i affects observation j, and observation j also affects observation i) and longer paths that might go from observation i to j to k and back to i. This is because the series expansion of $\mathbf{S}(\beta)^{-1}$ contains terms $(\mathbb{G})^k$ that, for $k \geqslant 2$, have non-zero elements on the diagonal.

[28] Note that the randomness of the network structure is the source of variation for the quantities of interest (points 3 and 4 below).

the intervention which may also depend on gender (*e.g.*, females could be more or less likely to choose the healthy meal rather than looking for fast-food options within or outside the cafeteria).[29]

We rely on the estimates of the weight production function related to eating habits by Fortin and Yazbeck (2015), which are computed using longitudinal data from Add Health. In our context, their estimate suggest that (in absence of social interactions) if a student eats one fast-food meal less per week, his/her BMI decreases by 0.85 in the long term.[30] Our first set of results assumes that the impact of the intervention is the same for males and females, and all individuals comply with the intervention, which gives $\gamma_f = \gamma_m = -0.85$. In our second set of results, we assume that $\gamma_f > \gamma_m$, which could be rationalized either with a differential impact or with differential compliance by gender.

The remaining population parameters are calibrated in line with our Add Health sample and estimation results, as follows:

- we fix $N = 120$, $p_1 = p_3 = 0.03$ and $p_2 = p_4 = 0.015$. This gives an expected number of within- and between-gender links of 1,8 and 0.9 respectively, as in our sample;
- we set the peer effect estimates for the heterogeneous model to $\beta_{mm} = 0.21$, $\beta_{mf} = 0.39$, $\beta_{ff} = 0.23$, $\beta_{fm} = 0.18$ in line with the results of Table (4), and we pick $\beta_h = 0.24$ accordingly.[31]

5.3 Simulation Results

5.3.1 Gender-neutral response

Panel A of Table (6) reports the results from the simulation exercise assuming $\gamma_f = \gamma_m = \gamma = -0.85$, *i.e.*, full compliance and same impact across gender. The upper part of the panel reports the average value of the treatment effect (direct, indirect, total) for all students together and by gender for the homogeneous and heterogeneous model respectively (mean and standard deviation over 500 draws).

The estimate of the direct effect is -0.85 throughout, meaning that one less fast-food meal per week has a long-term 'direct' effect of decreasing student's own BMI by 0.85 units. This is the same as the response parameter γ in absence of the intervention (although these

[29]For the sake of simplicity, we are ruling out complications related to non-random attrition.

[30]In their preferred estimates of the BMI production function controlling for lagged BMI, Fortin and Yazbeck (2015) find that an extra day of fast food restaurant visit per week increases zBMI (that is, the BMI standardized for gender and age) by 0.02 points in the long term. This result is estimated by pooling together female and male students. This is also consistent with the results by Niemeier et al. 2006. Since the average zBMI in our sample is 0.55, we have transposed their result in our metric as $(23.1*0.02)/0.55 \approx 0.85$.

[31]β_h is the weighted average of the four values above, which ensures internal consistency (i.e. the two models deliver comparable outcome vectors y for any arbitrary α). This does not need to coincide with the peer-effect coefficient from Table (3) because the estimation on Add Health data includes covariates and fixed effects.

two quantities do not need to coincide precisely, as explained in footnote 27, they often do). This estimate is stable across models (homogeneous and heterogeneous) and across gender (males and females) as it is expected to be.

The indirect treatment effect represents the social spillover through network lines. The estimate of the indirect treatment effect for the homogeneous model is -0.25 for all students confounded, males and females. This means that treating a randomly chosen student has on average an indirect effect of -0.25 units on the BMI of the others, given the existing social synergies. This indirect effect is sizable, as it represents approximately a 30% increase with respect to the direct effect. That is, on the basis of the evidence from Add Health, we conclude that social interactions amplify the impact of the intervention by about 30% with respect to the benchmark scenario of no interpersonal links and/or no social synergies among students. When we turn to the heterogeneous model (columns 4-6) we notice that the overall indirect coefficient is still -0.25, but this is actually a weighted average of an estimated effect of -0.20 for males vs. -0.29 for females. This suggests that, once gender-based heterogeneity is accounted for, the social spillovers (in term of BMI decrease among peers) of the intervention on female students are 50% higher than the corresponding social spillovers from males.

The bottom part of panel A reports the aggregate effect of the intervention on BMI for three intent-to-treat vectors representing different partial-intervention designs. $\boldsymbol{itt^1}$ depicts a scenario where 50% of students were randomly selected for the obesity-curbing intervention, regardless of their gender. $\boldsymbol{itt^2}$ represents a scenario where only female students were selected for the intervention, while $\boldsymbol{itt^3}$ represents a case where only male students were selected. In all three scenarios, the expected number of treated students stays the same (i.e. 60 out of 120). The aggregate effect reported in Column 4 ('without PE') does not take into account the social spillovers driven by peer effects.[32] Columns 5 and 6 ('with PE') report the aggregate effect on BMI accounting for social spillovers. Since the intent-to-treat vectors are drawn independently for each simulated network, we report the average BMI decrease accounting for social spillovers (column 5) and its standard deviation (column 6) over the 500 simulations.

The estimated decrease in BMI without social spillovers is the same across all treatment vectors (51 BMI points throughout column 4). Once we account for social spillovers, results from $\boldsymbol{itt^1}$ suggest that treating 50% of students at random (i.e. regardless of gender) decreases aggregate BMI by 65.7 points.[33] This corresponds to a decrease of 0.55 BMI points per student, or 11.5% of BMI standard deviation in Add Health. However, the magnitude of the impact is larger (-68.5 BMI points) when we treat female students only in $\boldsymbol{itt^2}$. Conversely, the magnitude of the impact is smaller (-63.1 BMI points) when we treat male students only in $\boldsymbol{itt^3}$. These numbers represent a 'natural' metric of efficiency in the context of our policy evaluation exercise: returns from treating females are 9%

[32] This boils down to summing up the direct effect over treated individuals.

[33] This statistics is by construction the same for the heterogeneous peer-effect model under $\boldsymbol{itt^1}$ and for the homogeneous peer-effect model under all three intent-to-treat vectors.

larger than returns from treating males. This means that monetary resources spent on females generate a decrease in BMI over the student population which is 9% above the one generated by males.

To summarize, even in the 'neutral' setting of Panel A where all students are affected by the intervention to the same extent, we find that social spillovers from females are about 50% larger than the ones from males, which results in an additional 9% returns from treating females in terms of aggregate decrease in BMI. This result is entirely driven by the heterogeneity of peer effects along gender lines: all students respond to the intervention in the same way, but females generate more social spillovers.

5.3.2 Gender-heterogeneous response

Panel B of Table (6) explores a scenario where females are more responsive to the intervention, that is, $\gamma_f > \gamma_m$. This could be due to the fact that the intervention is more effective on female compliers, or to the fact that compliance is higher among females – the practical relevance of the issue will be discussed below. In particular, we have calibrated a mean-preserving spread of $\gamma_f = 1; \gamma_m = 0.7$ so that the resulting BMI vector is comparable to panel A.

Results from Panel B for Column (1) (homogeneous peer effect model, males and females confounded) are comparable to Column (1) in Panel A, as expected. Columns (2) and (3) report the estimates of the homogeneous peer effect model for females and males respectively: the estimated direct effects are -1 and -0.7, in line with the γ_f, γ_m from the data-generating process. The indirect effects are now -0.29 and -0.2 respectively for females and males: even if peer effects are homogeneous within and across gender, females now have a larger impact on their peers because they experience a larger BMI decrease following the intervention. As before, the estimated effect of -0.25 in Column (1) is a weighted average of the gender-specific effects in columns (2) and (3) respectively.

When we turn to the heterogeneous model (columns 4 to 6) we see that all three estimates of the direct effect are comparable to the ones for the homogeneous model, as expected. However, we can see that the difference in the estimated indirect effects across gender lines becomes even wider. The indirect effect for females is now double that the one for males, -0.34 in Column (5) versus -0.17 in Column (6). This is due to the fact that when $\gamma_f > \gamma_m$ and peer effects are allowed to be heterogeneous across gender, females loose more weight *and also* influence more their peers so that the estimated spillovers are sensibly larger. The weighted average of these estimates is still 0.25 (as in Column 1), meaning that if we assume gender-homogeneous peer effects and consider a random sample of students regardless of their gender, we expect an indirect effect of 0.25 on average. However, this overall estimate hides a large disparity across gender lines, as the expected social spillovers from females are double the ones from males.

The bottom part of Panel B reports the effect of the intervention on aggregate BMI. Results show that treating 50% of students at random induces an aggregate decrease of

-66.1 BMI points under $\boldsymbol{itt^1}$, which hinders a large disparity between the aggregate BMI decrease from treating females only (-80.6 under $\boldsymbol{itt^2}$) and the corresponding value from treating males only (-51.9 under $\boldsymbol{itt^3}$). This suggests that, because of social synergies, keeping the budget constant, the returns from treating females only are 55% larger than returns from treating males only (from -51.9 to -80.6 BMI points).

To summarize, we had seen in Panel A that the heterogeneity of peer effects along gender lines has tangible consequences even in a setting where all students respond in the same way to the intervention, regardless of their gender. If we further assume that female students respond more to the intervention under scrutiny (Panel B), the estimated social spillovers from females are double the size of male-generated spillovers. This translate into a 55% gains in terms of aggregate BMI decrease from reaching out to female students.

5.4 Discussion

The simulation results above suggest that acknowledging peer-effect heterogeneity along gender lines could increase the efficiency of policy interventions. In fact, by assuming homogeneity we are failing to incorporate some important information that could be used to optimize the allocation process whenever resources are scarce.

The last two decades have witnessed the implementation of a large variety of policy instruments aimed at curbing obesity among teenagers in western countries. Those include interventions administered remotely (*e.g.*, online nutrition education program, email nudges with tailored dieting advice or steps/day goal) and offline (*e.g.*, face-to-face discussion groups, interactive action planning, supply of fruits and vegetables, supply of wearable sport activity trackers). Evidence from the literature on nutrition science suggests that young adults respond differently to interventions depending on their gender (Poobalan et al. 2010; Sharkey et al. 2020).[34] In particular, females appear more motivated to undertake dietary changes, while males are generally more responsive to incentives related to physical activity. Since interventions are often constrained in terms of resources, one way to allocate resources efficiently could be to design policy instruments implicitly tailored to address the motivation and barrier of one specific gender. On the basis of our results above, it is *ceteris paribus* preferable to invest in interventions aimed at educating teenagers towards better dietary patterns, because the higher direct impact on the female population could in turn spills over more effectively to their male peers. Such policy instruments are easy to implement, and they do not aim at impacting the structure of social interactions directly.[35]

Finally, it is worth noting that, throughout the exercise above, we have modeled the response to the intervention as a shift in the BMI. This benchmark assumption allows us

[34] In an extensive meta-analysis, Sharkey et al. 2020 find that gender-targeted programs are more effective to tackle youth obesity, but the results are not statistically significant due to the small sample size.

[35] According to our results, an increase in the frequency of between-gender links could also magnify the effect of the anti-obesity campaign. However, interventions aimed at manipulating directly social links (Goette, Huffman, and Meier 2012; Fafchamps and Quinn 2018) are widely seen as difficult to implement and scale up.

to be relatively agnostic with respect to the precise mechanism at work. However, policy makers may have alternative assumptions, based on their knowledge of the policy under scrutiny: for instance, they can hypothesize that the intervention affects the way peers influence the marginal utility of own BMI. In order to do a policy-evaluation exercise on the basis of alternative assumptions, one could rely on the theoretical framework developed in Appendix A.

6 Conclusion

This paper explores gender heterogeneity in the social transmission of BMI among teenagers, and its policy consequences. We propose a model where social interactions allow for between- and within-gender heterogeneity, and the Body Mass Index (BMI) results from social synergy among peers in a way that is micro-founded in a non-cooperative manner. We characterize the model econometrically, showing how identification conditions generalize those of the homogeneous model by Bramoullé, Djebbari, and Fortin (2009).

We illustrate the model using BMI and detailed social interactions of adolescents in the Add Health dataset. Comparing the GMM estimates of a standard homogeneous model of peer effects with our heterogeneous model, we show that Add Health data display significant gender-dependent heterogeneity in peer effects. In particular, results suggest that male students are more affected by the average BMI of their female friends that the reverse. We argue that these results are not driven by the endogeneity of declared links, as long as we control for fixed effects absorbing all unobserved heterogeneity which is common to all students within a given school.

Designing interventions on the basis of recipients' social interactions is a promising avenue ahead. One interest of our approach is to introduce observable gender-dependent heterogeneity in the model. This may help policy makers to evaluate the impact of reforms on adolescent obesity and to find the most appropriate tracking of students to reach the optimal outcome level, a point that we illustrate with a simulation exercise to evaluate an intervention replacing one fast-food type serving with one balanced meal per week. Results from our simulations show that, in the most conservative scenario where all students are affected by the intervention to the same extent, the social spillovers stemming from female students are 50% higher than the spillovers from males. This result is entirely driven by the heterogeneity of peer effects along gender lines, and it translates into an 9% gain in terms of aggregate BMI decrease from reaching out to females rather than males. If we further assume that female students respond more to the kind of intervention under scrutiny (as the literature on nutrition science seems to suggest), we conclude that social spillovers from females are double the size of male-generated spillovers and that resources spent on females generate a decrease of aggregate BMI which is 55% above the one generated by resources spent on males.

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Tables and Figures

Table 1: Descriptive statistics

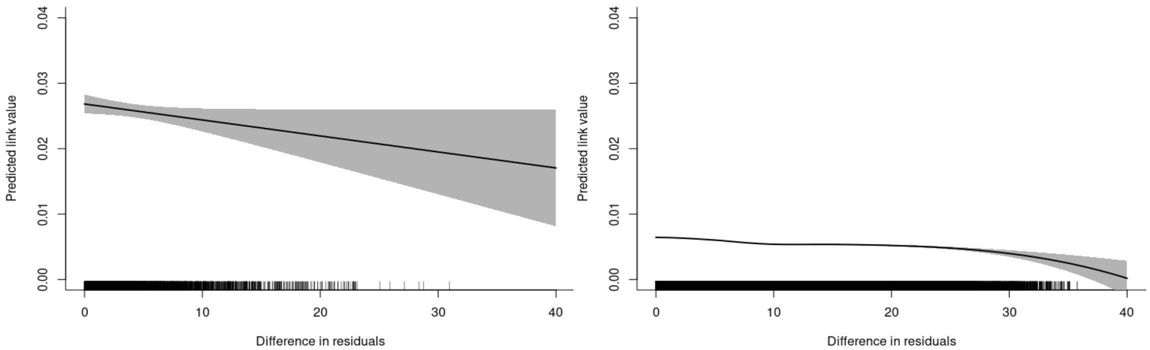
	Mean	s.d.	Min	Max
Weight status				
BMI	23.1	4.79	13	46
Males' BMI	23.4	4.72	15	45
Females' BMI	22.8	4.83	13	46
Individual characteristics				
Age	16.34	1.43	13	20
Female	0.50	0.50	0	1
White	0.64	0.48	0	1
Black	0.14	0.35	0	1
American Indian	0.03	0.17	0	1
Asian Pacific	0.14	0.34	0	1
Hispanic origin	0.16	0.37	0	1
Grades 7-8	0.11	0.32	0	1
Grades 9-10	0.27	0.45	0	1
Grades 11-12	0.61	0.49	0	1
Mother education and health status				
No high school	0.12	0.33	0	1
High school	0.36	0.48	0	1
Some college	0.19	0.39	0	1
College	0.18	0.38	0	1
Advanced	0.07	0.25	0	1
Don't know	0.04	0.19	0	1
Good health status	0.92	0.27	0	1
Father education and health status				
No high school	0.11	0.31	0	1
High school	0.26	0.44	0	1
Some college	0.15	0.35	0	1
College	0.16	0.37	0	1
Advanced	0.07	0.25	0	1
Don't know	0.05	0.22	0	1
Good health status	0.78	0.42	0	1
Network statistics				
Average number of friends	2.55	1.96	0	10
Males: Number of male friends	1.72	3.80	0	5
Males: Number of female friends	0.84	1.33	0	5
Females: Number of male friends	0.91	1.21	0	5
Females: Number of female friends	1.63	1.49	0	5
N=1914				

Table 2: Endogenous network - Dep. var.: predicted link probability

	(1)	(2)
	Coefficient	**Coefficient**
$\lvert\hat{\epsilon}_i - \hat{\epsilon}_j\rvert$	-0.00008 ***	-0.0000002
	(0.0000024)	(0.00000025)
g_{ij}	0.01972 ***	0.00015 ***
	(0.00016)	(0.000016)
Intercept	0.00643 ***	0.09544 ***
	(0.000019)	(0.000025
Network fixed effects	No	Yes
Observations	790,524	790,524

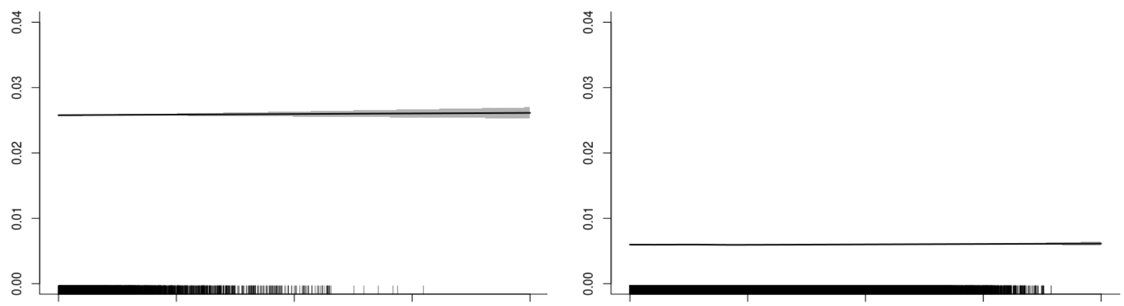
*** p<0.01, ** p<0.05, * p<0.1

Figure 1: Kernel densities without school fixed effects



$g_{ij,r} = 1$ $\qquad$ $g_{ij,r} = 0$

Figure 2: Kernel densities with school fixed effects



$g_{ij,r} = 1$ $\qquad$ $g_{ij,r} = 0$

Table 3: GMM estimation of homogeneous peer effects

	(1)	(2)	(3)	(4)
	Individual effects		**Contextual effects**	
	Coef.	**SE.**	**Coef.**	**SE.**
Endogenous peer effect	**0.208*** **	**0.022**	—	—
Personal characteristics				
Age	0.145	0.098	-0.301***	0.058
White=1	-0.456*	0.274	0.680**	0.326
Black=1	-0.661*	0.358	0.830*	0.439
Grade 9-10	0.557	0.574	0.764	0.651
Grade 11-12	1.306**	0.644	0.341	0.719
Mother education				
No high school	-0.419	0.371	-1.979***	0.579
High school	-0.081	0.314	-1.300***	0.509
Some college	0.117	0.336	-1.043*	0.550
College	0.311	0.344	-2.177*	0.542
Advanced	-0.132	0.428	-3.182***	0.701
Father education				
No high school	0.749	0.321	0.000	0.478
High school	0.133	0.252	-0.645	0.384
Some college	0.321	0.284	-1.270***	0.414
College	0.108	0.287	0.301	0.445
Advanced	0.400	0.372	-0.388	0.578
Parents health status				
Mother Good health	-0.315	0.307	0.950*	0.485
Father Good health	-0.572**	0.231	-0.263	0.344
Weak instruments test (Kleibergen-Paap/Cragg-Donald statistics): 6.931				
N = 1914				

Table 4: GMM estimation of heterogeneous peer effects

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Individual effects		**Contextual effects**							
			$m-m$		$m-f$		$f-f$		$f-m$	
	Coef.	***SE***	***Coef.***	***SE***	***Coef.***	***SE***	***Coef.***	***SE***	***Coef.***	***SE***
Endogenous peer effects			**0.214*****	**0.040**	**0.392*****	**0.132**	**0.225*****	**0.030**	**0.184****	**0.089**
Personal characteristics										
Age	0.095	0.098	-0.239***	0.087	-0.655***	0.217	-0.473***	0.097	-0.239	0.163
White=1	-0.441	0.275	0.017	0.481	-0.379	0.880	1.822***	0.491	0.185	0.872
Black=1	-0.511	0.362	-1.065	0.736	1.367	1.084	1.420***	0.601	1.081	1.112
Grade 9-10	0.529	0.583	1.723**	0.823	-1.706	1.322	2.054**	0.979	0.981	1.175
Grade 11-12	1.282**	0.651	1.137	0.910	0.085	1.446	1.656	1.082	-0.479	1.241
Mother education										
No high school	-0.464	0.371	-3.577***	0.911	-1.163	1.803	0.339	1.040	-2.644	1.676
High school	-0.093	0.314	-2.514***	0.753	0.107	1.648	-0.050	0.986	-0.706	1.292
Some college	0.155	0.338	-1.569*	0.887	0.004	1.704	-0.164	1.006	0.020	1.469
College	0.335	0.344	-2.985***	0.808	-2.213	1.851	-1.805*	1.031	-1.245	1.364
Advanced	-0.062	0.428	-3.747***	1.117	-6.355***	2.371	-1.053	1.223	-3.964**	1.782
Father education										
No high school	0.635*	0.321	-1.245	0.790	-0.234	1.496	-0.169	0.754	2.874**	1.349
High school	0.066	0.252	-2.062***	0.639	2.916**	1.232	-0.305	0.625	-0.220	1.027
Some college	0.330	0.285	-2.520***	0.667	0.999	1.291	-0.698	0.687	-2.033*	1.190
College	0.010	0.289	-1.500**	0.712	1.985	1.583	0.649	0.740	2.884**	1.219
Advanced	0.392	0.373	-1.825**	0.910	4.019*	2.065	0.507	0.935	-0.276	1.485
Parents health status										
Mother Good health	-0.252	0.308	1.704**	0.780	2.319*	1.272	0.122	0.811	0.189	1.432
Father Good health	-0.471	0.233	0.113**	0.572	-0.278	1.035	-1.110**	0.560	-0.300	0.941
Weak instruments test (Cragg-Donald statistic): 12.3										
N = 1914										

Table 5: Wald Statistics for Gender Homogeneity in Peer Effects

GMM - BMI	
Full Homogeneity: $\beta_{mm} = \beta_{mf} = \beta_{ff} = \beta_{fm} / \delta_{mm} = \delta_{mf} = \delta_{ff} = \delta_{fm}$	120.71
Only-Endogenous Homogeneity: $\beta_{mm} = \beta_{mf} = \beta_{ff} = \beta_{fm}$	107.77

Full-gender model: 54 d.o.f.. threshold values respectively 81.06, 72.15 and 67.67 for 0.01%. 0.05% and 10% significance. Only-endogenous model: 6 d.o.f.. threshold values respectively 16.81, 12.59 and 10.64 for 0.01%. 0.05% and 0.10% significance.

Table 6: Simulation results

Panel A: $\gamma_f = \gamma_m = -0.85$						
model:	homogeneous PE			heterogeneous PE		
mean y	23.21			23.23		
	(1)	(2)	(3)	(4)	(5)	(6)
	all	females	males	all	females	males
TE: direct	-0.85	-0.85	-0.85	-0.85	-0.85	-0.85
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
TE: indirect	-0.25	-0.25	-0.24	-0.25	-0.29	-0.20
	(0.01)	(0.02)	(0.02)	(0.01)	(0.02)	(0.01)
TE: total	-1.10	-1.10	-1.09	-1.10	-1.14	-1.05
	(0.01)	(0.02)	(0.02)	(0.01)	(0.02)	(0.01)
Aggregate effect on BMI						
				without PE	with PE	
$\boldsymbol{itt^1}$: 50% students at random				-51	-65.7	(5.98)
$\boldsymbol{itt^2}$: 50% students, females only				-51	-68.5	(1.36)
$\boldsymbol{itt^3}$: 50% students, males only				-51	-63.1	(0.88)

Panel B: $\gamma_f = -1, \gamma_m = -0.7$						
model:	homogeneous PE			heterogeneous PE		
mean y	23.21			23.23		
	(1)	(2)	(3)	(4)	(5)	(6)
	all	females	males	all	females	males
TE: direct	-0.85	-1	-0.7	-0.85	-1	-0.7
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
TE: indirect	-0.25	-0.29	-0.20	-0.25	-0.34	-0.17
	(0.01)	(0.02)	(0.02)	(0.01)	(0.03)	(0.01)
TE: total	-1.10	-1.29	-0.90	-1.10	-1.34	-0.87
	(0.01)	(0.02)	(0.02)	(0.01)	(0.03)	(0.01)
Aggregate effect on BMI						
				without PE	with PE	
$\boldsymbol{itt^1}$: 50% students at random				-51	-66.1	(6.13)
$\boldsymbol{itt^2}$: 50% students, females only				-60	-80.6	(1.60)
$\boldsymbol{itt^3}$: 50% students, males only				-42	-51.9	(0.72)

Note: average values computed over 500 draws. Standard errors in parenthesis.

Appendix A

We now develop a non-cooperative model to micro-found our estimating equation through the channel of strategic complementarity ('social synergy') in BMI within the social network. We develop the theoretical model for one network of non-isolated students where heterogeneous peer effects only work through the 'endogenous' channel (*i.e.*, δs are set to zero). This is done to simplify the notation, and is aligned with the simulation exercise of section 5. However, the discussion can be trivially extended to the most general case.

Let us consider one population of students ($n^m + n^f = n$), interacting among them. The student i's reference group is non-empty: $n_{i,m} + n_{i,f} > 0$ for each i.[36] The friendship network is defined by four fixed and known binary adjacency matrices $\mathbb{A}_z (z = 1, \cdots, 4)$, and their weighted version $\mathbb{G}_z (z = 1, \cdots, 4)$, defined as above. Every individual maximizes a gender-dependent quadratic utility function which is separable in private and social sub-utilities, subject to a linear production function for the BMI. The maximization program of a type-m individual i is:

$$\max_{y_{i,m}, e_{i,m}} \quad U_{i,m}(e_{i,m}, \mathbf{y}) = -y_{i,m} - \frac{e_{i,m}^2}{2} + \psi_{mm} y_{i,m} \mathbf{g}'_{1i} \mathbf{y}_m + \psi_{mf} y_{i,m} \mathbf{g}'_{2i} \mathbf{y}_f, \tag{13}$$

$$s.t. \quad y_{i,m} = \alpha_0 - \alpha_1 e_{i,m} + \alpha_2 x_{i,m} + \eta_{i,m}, \tag{14}$$

where $y_{i,m}$ is the outcome (*i.e.*, BMI) of individual i in category m, $\mathbf{y}_m$ is the vector of outcomes in m category, $\mathbf{y}_f$ is the vector of outcomes in f category, $\mathbf{y}$ is the concatenated vector of outcomes in f and m categories, e_i stands for the effort of i, $\mathbf{g}'_{zi}$ is the i^{th} row of the social interaction matrix $\mathbb{G}_z$, and x_i and $\eta_{i,m}$ denote observable and unobservable individual characteristics. The first two terms in the utility function describe the private sub-utility: the first term assumes that an increase in BMI reduces the individual i's utility.[37] The second term $\frac{e_{i,m}^2}{2}$ represents the cost of effort to reduce weight and assumes that the marginal cost of effort is increasing with effort. The social sub-utility corresponds to the two last terms in the utility function: we assume that social interactions influence preferences through the channel of social synergy in BMI between a student and his reference group of each type (Fortin and Yazbeck 2015).[38]

[36]Note that the empirical illustration relaxes this assumption, allowing for isolated students.

[37]We are ignoring here a situation where very low weight negatively affects health (*e.g.*, anorexia).

[38]This framework is also consistent with a mechanism of *pure conformity* in social interactions. In that case, an individual's utility is positively affected by the degree to which he conforms with his peers' outcome or characteristics due for instance to the presence of social norms. However, while in a model such as ours, the channels of social synergy and pure conformity are observationally equivalent (Blume et al. 2015; Boucher and Fortin 2016; Boucher et al. 2022), it seems plausible to assume that social synergy is the mechanism at play in peer effects. Indeed, it means that an increase in the peers' average BMI of a given gender positively influences the marginal utility of his own BMI ($\psi_{fm} > 0; \psi_{mf} > 0$). Heterogeneity in social interactions is reflected by the fact that ψ_{fm} and ψ_{mf} can be different. For instance, as mentioned earlier, since girls are in general more mature and influencial than boys at the same age, it is natural to assume that the former have more influence on the latter's marginal utility than the reverse.

The maximization program of type-f individuals can be written using a similar utility function, where social interaction parameters can differ from those of type-m ones. Hence, a type-f individual solves the following program:

$$\max_{y_{i,f},e_{i,f}} \quad U_f(e_{i,f},\mathbf{y}) = -y_{i,f} - \frac{e_{i,f}^2}{2} + \psi_{ff} y_{i,f} \mathbf{g}'_{3i} \mathbf{y}_f + \psi_{fm} y_{i,f} \mathbf{g}'_{4i} \mathbf{y}_m \tag{15}$$

$$s.t. \quad y_{i,f} = \alpha_0 - \alpha_1 e_{i,f} + \alpha_2 x_{i,f} + \eta_{i,f} \tag{16}$$

The first order conditions of the type-m maximization program lead to

$$\mathbf{y}_m = \alpha \boldsymbol{\iota}_m + \beta_{mm} \mathbb{G}_1 \mathbf{y}_m + \beta_{mf} \mathbb{G}_2 \mathbf{y}_f + \alpha_2 \mathbf{x}_m + \boldsymbol{\epsilon}_m \tag{17}$$

where $\alpha = \alpha_0 - \mu$, $\beta_{mm} = \mu\psi_{mm}$, $\beta_{mf} = \mu\psi_{mf}$, and $\boldsymbol{\epsilon}_m = \boldsymbol{\eta}_m$, where $\mu = \alpha_1^2$ represents the squared marginal productivity of effort on weight level. Similarly, the first order conditions for type-f individuals lead to

$$\mathbf{y}_f = \alpha \boldsymbol{\iota}_f + \beta_{ff} \mathbb{G}_3 \mathbf{y}_f + \beta_{fm} \mathbb{G}_4 \mathbf{y}_m + \alpha_2 \mathbf{x}_f + \boldsymbol{\epsilon}_f \tag{18}$$

where $\beta_{ff} = \mu\psi_{ff}$, $\beta_{fm} = \mu\psi_{fm}$, and $\boldsymbol{\epsilon}_f = \boldsymbol{\eta}_f$. Assuming that the absolute value of the β's is less than one, if we concatenate Equations (17) and (18), we obtain the following best-response functions for the whole population of students, given the others' weight level (Nash equilibrium):

$$\mathbf{y} = \alpha \boldsymbol{\iota} + \beta_{mm} \mathbb{G}_1 \mathbf{y} + \beta_{mf} \mathbb{G}_2 \mathbf{y} + \beta_{ff} \mathbb{G}_3 \mathbf{y} + \beta_{fm} \mathbb{G}_4 \mathbf{y} + \alpha_2 \mathbf{x} + \boldsymbol{\epsilon}, \tag{19}$$

which coincides with equation (2). Notice that homogeneity implies that all ψ's are equal ($= \psi$), and thus $\beta_{mm} = \beta_{mf} = \beta_{ff} = \beta_{fm} = \beta$ (equation (3)).

This theoretical result has a practical relevance for the evaluation of exogenous shocks and interventions (Section 5). In fact, the conditions above imply that we can separately identify all the parameters of the utility function provided that we have a proxy for effort. While effort is generally not observed, it is possible to find a good proxy for effort in observational databases at the individual level (*e.g.*, a measure of eating habits, physical exercise, *etc.*). When no such data are available, one can always recover the parameters of the preferences and the production function, *for a given level of* μ. Indeed, each of the four social sub-utility parameters (the ψ's) are proportional to its corresponding β, the proportionality coefficient being μ^{-1}.

Finally, throughout the simulation exercise of section 5 we have assumed that the intervention shifts the intercept α_0 of the BMI production function in equation (14) . This is a benchmark assumption which allows us to be relatively agnostic with respect to the underlying mechanism. However, we could have alternative hypotheses about the channels through which the intervention affects the BMI in the population of interest: for instance, policy makers may have good reasons to believe that the intervention affects either the marginal productivity of effort (α_1) of directly the way peers' BMI influences the marginal utility of own BMI (ψ's). These different scenarios yield different conclusions in terms of policy evaluation, and the amplitude of these discrepancies could be evaluated by an appropriate calibration of the model above, following the footsteps of section (5).

Appendix B

Proof of proposition 1: identification

To prove our proposition, we assume that $\mathbf{S}(\boldsymbol{\beta})$ is invertible (see footnote 8 for sufficient conditions) and we use the formula of the inverse of matrix established using the Newton Binomial formula. The following steps are necessary to prove our proposition:

1. Let $k = 1, 2, 3, 4, ...$ and derive the expression of $\mathbf{S}_k(\boldsymbol{\beta})^{-1}$ using:[39]
$$\mathbf{S}_k(\boldsymbol{\beta}) = \sum_{i=0}^{k\geq 1} \binom{k}{i} \left[(\beta_{mm}\bar{\mathbb{G}}_1)^{k-i} + (k-i)\beta_{mf}(\beta_{mm}\bar{\mathbb{G}}_1)^{k-i-1}\bar{\mathbb{G}}_2\right].\left[(\beta_{ff}\bar{\mathbb{G}}_3)^{i} + i\beta_{fm}(\beta_{ff}\bar{\mathbb{G}}_3)^{i-1}\bar{\mathbb{G}}_4\right]$$
2. Sum over all k's and re-write $\mathbf{S}(\boldsymbol{\beta})^{-1}$ such that $\mathbf{S}(\boldsymbol{\beta})^{-1} = \mathbf{I} + \sum_{k=1}^{\infty} \mathbf{S}_k(\boldsymbol{\beta})$.
3. Using the latter expression, derive an expression of $\mathbf{W}_i(\boldsymbol{\beta}) = \bar{\mathbb{G}}_i\mathbf{S}(\boldsymbol{\beta})^{-1}$ and $\mathbf{W}_i(\boldsymbol{\beta})\bar{\mathbb{G}}(\boldsymbol{\delta})$ $\forall i \in \{1, 2, 3, 4\}$.
4. Write $\{\mathbf{W_i}(\boldsymbol{\beta})\left[\gamma\mathbf{x} + \bar{\mathbb{G}}(\boldsymbol{\delta})\mathbf{x} + \boldsymbol{\iota\alpha}\right]\}_{\{i=1,2,3,4\}}$ as a function of instruments and extract intruments and the associated restrictions on the parameters of the model, pre-multiplied by matrix $\mathbf{J}$.

For sake of simplicity, let susbscripts mm, mf, ff and fm in $\boldsymbol{\beta}$ be replaced by $1, 2, 3, 4$ respectively. Using the steps enumerated above and developing for $k \in 1, 2, 3, 4$, one can write $\mathbf{S}_k(\boldsymbol{\beta})$ using the expression below:

$$\mathbf{S}_1(\boldsymbol{\beta}) = \left[\beta_1\bar{\mathbb{G}}_1 + \beta_2\bar{\mathbb{G}}_2\right] \times \left[\beta_3\bar{\mathbb{G}}_3 + \beta_4\bar{\mathbb{G}}_4\right]$$

$$\mathbf{S}_2(\boldsymbol{\beta}) = \left[\beta_1^2\bar{\mathbb{G}}_1^2 + 2\beta_1\beta_2\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\right] + 2\left[\beta_1\bar{\mathbb{G}}_1 + \beta_2\bar{\mathbb{G}}_2\right] \times \left[\beta_3\bar{\mathbb{G}}_3 + \beta_4\bar{\mathbb{G}}_4\right] + \left[\beta_3^2\bar{\mathbb{G}}_3^2 + 2\beta_3\beta_4\bar{\mathbb{G}}_3\bar{\mathbb{G}}_4\right]$$

$$\begin{aligned}\mathbf{S}_3(\boldsymbol{\beta}) = \quad & \left[\beta_1^3\bar{\mathbb{G}}_1^3 + 3\beta_1^2\beta_2\bar{\mathbb{G}}_1^2\bar{\mathbb{G}}_2\right] + 3\left[\beta_1^2\bar{\mathbb{G}}_1^2 + 2\beta_1\beta_2\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\right] \times \left[\beta_3\bar{\mathbb{G}}_3 + \beta_4\bar{\mathbb{G}}_4\right] \\ + \quad & 3\left[\beta_1\bar{\mathbb{G}}_1 + \beta_2\bar{\mathbb{G}}_2\right] \times \left[\beta_3^2\bar{\mathbb{G}}_3^2 + 2\beta_3\beta_4\bar{\mathbb{G}}_3\bar{\mathbb{G}}_4\right] + \left[\beta_3^3\bar{\mathbb{G}}_3^3 + 3\beta_3^2\beta_4\bar{\mathbb{G}}_3^2\bar{\mathbb{G}}_4\right]\end{aligned}$$

[39] Recall that we order all matrices so that the first n_r^f rows correspond to type-f individuals of network r, and the remaining n_r^m rows are for type-m individuals in network r. This leads by construction to the following identities: $\mathbb{G}_{1,r}.\mathbb{G}_{4,r} = 0_{n_r}$, $\mathbb{G}_{3,r}.\mathbb{G}_{2,r} = 0_{n_r}$, $\mathbb{G}_{1,r}.\mathbb{G}_{3,r} = 0_{n_r}$, $\mathbb{G}_{3,r}.\mathbb{G}_{1,r} = 0_{n_r}$, $\mathbb{G}_{2,r}^{k\geq 2} = 0_{n_r}$, $\mathbb{G}_{4,r}^{k\geq 2} = 0_{n_r}$, $\mathbb{G}_{4,r}.\mathbb{G}_{3,r} = 0_{n_r}$ and $\mathbb{G}_{2,r}.\mathbb{G}_{1,r} = 0_{n_r}$.

$$
\begin{aligned}
\mathbf{S}_4(\boldsymbol{\beta}) = \quad & \left[\beta_1^4\bar{\mathbb{G}}_1^4 + 4\beta_1^3\beta_2\bar{\mathbb{G}}_1^3\bar{\mathbb{G}}_2\right] + 4\left[\beta_1^3\bar{\mathbb{G}}_1^3 + 3\beta_1^2\beta_2\bar{\mathbb{G}}_1^2\bar{\mathbb{G}}_2\right] \times \left[\beta_3\bar{\mathbb{G}}_3 + \beta_4\bar{\mathbb{G}}_4\right] \\
+ \quad & 6\left[\beta_1^2\bar{\mathbb{G}}_1^2 + 2\beta_1\beta_2\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\right] \times \left[\beta_3^2\bar{\mathbb{G}}_3^2 + 2\beta_3\beta_4\bar{\mathbb{G}}_3\bar{\mathbb{G}}_4\right] + 4\left[\beta_1\bar{\mathbb{G}}_1 + \beta_2\bar{\mathbb{G}}_2\right] \\
\times \quad & \left[\beta_3^3\bar{\mathbb{G}}_3^3 + 3\beta_3^2\beta_4\bar{\mathbb{G}}_3^2\bar{\mathbb{G}}_4\right] + \left[\beta_3^4\bar{\mathbb{G}}_3^4 + 4\beta_3^3\beta_4\bar{\mathbb{G}}_3^3\bar{\mathbb{G}}_4\right]
\end{aligned}
$$

We then write $\mathbf{S}^{-1}(\boldsymbol{\beta}) = \mathbf{I}+\mathbf{S}_1(\boldsymbol{\beta})+\mathbf{S}_2(\boldsymbol{\beta})+\mathbf{S}_3(\boldsymbol{\beta})+\mathbf{S}_4(\boldsymbol{\beta})+\sum_{k=5}^{\infty}\mathbf{S}_k(\boldsymbol{\beta})$ using the expressions of $\mathbf{S}_k(\boldsymbol{\beta})$ given above. We are then able to write, $\forall i \in \{1,2,3,4\}$, $\mathbf{W_i}(\boldsymbol{\beta})\left[\gamma\mathbf{x} + \bar{\mathbb{G}}(\boldsymbol{\delta})\mathbf{x} + \boldsymbol{\iota\alpha}\right]$ as:

$$
\begin{aligned}
\mathbf{W_1}(\boldsymbol{\beta})\left[\gamma\mathbf{x} + \bar{\mathbb{G}}(\boldsymbol{\delta})\mathbf{x} + \boldsymbol{\iota\alpha}\right] = \gamma\bar{\mathbb{G}}_1\mathbf{x} \quad & + \quad (\gamma\beta_1+\delta_1)\left[\bar{\mathbb{G}}_1^2 + \beta_1\bar{\mathbb{G}}_1^3 + \beta_1^2\bar{\mathbb{G}}_1^4 + \beta_1^5\bar{\mathbb{G}}_1^2\right]\mathbf{x} \\
& + \quad (\gamma\beta_2+\delta_2)\left[\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\right]\mathbf{x} + \beta_1(2\gamma\beta_2+\delta_2)\left[\bar{\mathbb{G}}_1^2\bar{\mathbb{G}}_2\right]\mathbf{x} \\
& + \quad \beta_2(2\gamma\beta_3+\delta_3)\left[\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3\right]\mathbf{x} + \beta_2(2\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\bar{\mathbb{G}}_4\right]\mathbf{x} \\
& + \quad \left[\bar{\mathbb{G}}_1 + \beta_1\bar{\mathbb{G}}_1^2 + \beta_2\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2 + \beta_1^2\bar{\mathbb{G}}_1^3 + 2\beta_1\beta_2\bar{\mathbb{G}}_1^2\bar{\mathbb{G}}_2 + ...\right]\boldsymbol{\iota\alpha} \\
& + \quad \bar{\mathbb{G}}_1\sum_{k=5}^{\infty}\mathbf{S}_k(\boldsymbol{\beta})\left[(\gamma + \bar{\mathbb{G}}(\boldsymbol{\delta}))\mathbf{x} + \boldsymbol{\iota\alpha}\right]
\end{aligned}
$$

$$
\begin{aligned}
\mathbf{W_2}(\boldsymbol{\beta})\left[\gamma\mathbf{x} + \bar{\mathbb{G}}(\boldsymbol{\delta})\mathbf{x} + \boldsymbol{\iota\alpha}\right] = \gamma\bar{\mathbb{G}}_2\mathbf{x} \quad & + \quad (\gamma\beta_3+\delta_3)\left[\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3 + \beta_3\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3^2 + \beta_3^2\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3^3 + \beta_3^3\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3^3\right]\mathbf{x} \\
& + \quad (\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_2\bar{\mathbb{G}}_4\right]\mathbf{x} + \beta_3(2\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3\bar{\mathbb{G}}_4\right]\mathbf{x} \\
& + \quad \beta_3^2(3\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3^2\bar{\mathbb{G}}_4\right]\mathbf{x} + \beta_3^3(4\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3^3\bar{\mathbb{G}}_4\right]\mathbf{x} \\
& + \quad \left[\bar{\mathbb{G}}_2 + \beta_3\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3 + \beta_3^2\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3^2 + 2\beta_3\beta_4\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3\bar{\mathbb{G}}_4 + ...\right]\boldsymbol{\iota\alpha} \\
& + \quad \bar{\mathbb{G}}_2\sum_{k=5}^{\infty}\mathbf{S}_k(\boldsymbol{\beta})\left[(\gamma + \bar{\mathbb{G}}(\boldsymbol{\delta}))\mathbf{x} + \boldsymbol{\iota\alpha}\right]
\end{aligned}
$$

$$
\begin{aligned}
\mathbf{W_3}(\boldsymbol{\beta})\left[\gamma\mathbf{x} + \bar{\mathbb{G}}(\boldsymbol{\delta})\mathbf{x} + \boldsymbol{\iota\alpha}\right] = \gamma\bar{\mathbb{G}}_3\mathbf{x} \quad & + \quad (\gamma\beta_3+\delta_3)\left[\bar{\mathbb{G}}_3^2 + \beta_3\bar{\mathbb{G}}_3^3 + \beta_3^2\bar{\mathbb{G}}_3^4 + \beta_3^3\bar{\mathbb{G}}_3^5\right]\mathbf{x} \\
& + \quad (\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_3\bar{\mathbb{G}}_4\right]\mathbf{x} + \beta_3(2\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_3^2\bar{\mathbb{G}}_4\right]\mathbf{x} \\
& + \quad \beta_3^2(3\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_3^3\bar{\mathbb{G}}_4\right]\mathbf{x} + \beta_3^3(4\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_3^4\bar{\mathbb{G}}_4\right]\mathbf{x} \\
& + \quad \left[\bar{\mathbb{G}}_3 + \beta_3\bar{\mathbb{G}}_3^2 + \beta_4\bar{\mathbb{G}}_3\bar{\mathbb{G}}_4 + 2\beta_3\beta_4\bar{\mathbb{G}}_3^2\bar{\mathbb{G}}_4 + ...\right]\boldsymbol{\iota\alpha} \\
& + \quad \bar{\mathbb{G}}_3\sum_{k=5}^{\infty}\mathbf{S}_k(\boldsymbol{\beta})\left[(\gamma + \bar{\mathbb{G}}(\boldsymbol{\delta}))\mathbf{x} + \boldsymbol{\iota\alpha}\right]
\end{aligned}
$$

$$
\begin{aligned}
\mathbf{W_4}(\boldsymbol{\beta})\left[\gamma\mathbf{x} + \bar{\mathbb{G}}(\boldsymbol{\delta})\mathbf{x} + \boldsymbol{\iota\alpha}\right] = \gamma\bar{\mathbb{G}}_4\mathbf{x} \quad & + \quad (\gamma\beta_1+\delta_1)\left[\bar{\mathbb{G}}_4\bar{\mathbb{G}}_1 + \beta_1\bar{\mathbb{G}}_4\bar{\mathbb{G}}_1^2 + \beta_1^2\bar{\mathbb{G}}_4\bar{\mathbb{G}}_1^3 + \beta_1^3\bar{\mathbb{G}}_4\bar{\mathbb{G}}_1^4\right]\mathbf{x} \\
& + \quad (\gamma\beta_2+\delta_2)\left[\bar{\mathbb{G}}_4\bar{\mathbb{G}}_2\right]\mathbf{x} + \beta_1(2\gamma\beta_2+\delta_2)\left[\bar{\mathbb{G}}_4\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\right]\mathbf{x} \\
& + \quad \beta_2(2\gamma\beta_3+\delta_3)\left[\bar{\mathbb{G}}_4\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3\right]\mathbf{x} + \beta_2(2\gamma\beta_4+\delta_4)\left[\bar{\mathbb{G}}_4\bar{\mathbb{G}}_2\bar{\mathbb{G}}_4\right]\mathbf{x} \\
& + \quad \left[\bar{\mathbb{G}}_4 + \beta_1\bar{\mathbb{G}}_4\bar{\mathbb{G}}_1 + \beta_2\bar{\mathbb{G}}_4\bar{\mathbb{G}}_2 + \beta_1^2\bar{\mathbb{G}}_4\bar{\mathbb{G}}_1^2 + ...\right]\boldsymbol{\iota\alpha} \\
& + \quad \bar{\mathbb{G}}_4\sum_{k=5}^{\infty}\mathbf{S}_k(\boldsymbol{\beta})\left[(\gamma + \bar{\mathbb{G}}(\boldsymbol{\delta}))\mathbf{x} + \boldsymbol{\iota\alpha}\right]
\end{aligned}
$$

The above expressions provide sufficient conditions of identification of our parameters using the IV method. These conditions extend the ones obtained in Bramoullé, Djebbari, and Fortin (2009) regarding the independence of the interaction matrices of our model and restrictions on our parameters.

Specifically, considering the expressions given above, we can see that naturally occuring intruments of our endogenous variables include different order of our interaction matrices and interactions of different orders of these matrices. For example, instruments of our first endogenous variable include $\mathbf{J}\mathbf{G_1}\mathbf{x}$, $\mathbf{J}\mathbf{G_1}^2\mathbf{x}$, $\mathbf{J}\mathbf{G_1}^3\mathbf{x}$ and higher degrees of the matrix $\mathbf{J}\mathbf{G}_1$ multiplied by vector $\mathbf{x}$ of characteristics if both $(\gamma\beta_1 + \delta_1) \neq 0$ and matrices $\mathbf{G_1}, \mathbf{G_1}^2, \mathbf{G_1}^3, \mathbf{G_1}^4, etc.$ are linearly independent. Following the same method and using the other expressions above, we can see that minimal conditions for IV variables to work for each of the four endogenous variables are $(\gamma\beta_2+\delta_2) \neq 0$, $(\gamma\beta_3+\delta_3) \neq 0$ and $(\gamma\beta_4+\delta_4) \neq 0$. In addition, γ needs to be different from zero and matrices $\bar{\mathbb{G}}_1$, $\bar{\mathbb{G}}_2$, $\bar{\mathbb{G}}_3$, $\bar{\mathbb{G}}_4$, $\bar{\mathbb{G}}_1^2$, $\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2$, $\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3$, $\bar{\mathbb{G}}_3^2$, $\bar{\mathbb{G}}_1^3$, ..., $\mathbf{I}$ need to be independent, which corresponds to the condition that vector columns of matrix $\mathbf{Q}_K$ of instruments should be linearly independent. Additional conditions appear whenever one is concerned about adding instruments of higher order of interaction matrices multiplication. In this case, the additional conditions on parameters of the model take the form of $\beta_i \neq 0$ $\forall i \in \{2,3,4\}$ and $((j-1)\gamma\beta_l + \delta_l) \neq 0$ and linear independence of j^{th} order interaction of social interaction matrices such that $\mathbf{C}\mathbf{G}_i\bar{\mathbb{G}}_l$ adds up to the independence conditions stated above, where $\mathbf{C}$ is either a single interaction matrix or a non-zero product of interaction matrices. For example, $\mathbf{J}\mathbf{G}_1\bar{\mathbb{G}}_2\bar{\mathbb{G}}_4\mathbf{x}$ may be used as an instrument if $\beta_2 \neq 0$, $(2\gamma\beta_4 + \delta_4) \neq 0$ and matrices $\bar{\mathbb{G}}_1$, $\bar{\mathbb{G}}_2$, $\bar{\mathbb{G}}_3$, $\bar{\mathbb{G}}_4$, $\bar{\mathbb{G}}_1^2$, $\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2$, $\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3$, $\bar{\mathbb{G}}_3^2$, $\bar{\mathbb{G}}_1^3$, ..., $\mathbf{I}$ and $\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2\bar{\mathbb{G}}_4$ are linearly independent. Also, $\mathbf{J}\mathbf{G}_4\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3^2\mathbf{x}$ may be used as an additional instrument if $\beta_2 \neq 0$, $\beta_3 \neq 0$, $(3\gamma\beta_3 + \delta_3) \neq 0$ and matrices $\bar{\mathbb{G}}_1$, $\bar{\mathbb{G}}_2$, $\bar{\mathbb{G}}_3$, $\bar{\mathbb{G}}_4$, $\bar{\mathbb{G}}_1^2$, $\bar{\mathbb{G}}_1\bar{\mathbb{G}}_2$, $\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3$, $\bar{\mathbb{G}}_3^2$, $\bar{\mathbb{G}}_1^3$, ..., $\mathbf{I}$ and $\bar{\mathbb{G}}_4\bar{\mathbb{G}}_2\bar{\mathbb{G}}_3^2$ are linearly independent.