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Leveraging social influence to address overweight and obesity using agent-based models: the role of adolescent social networks

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Abstract

The prevalence of adolescent overweight and obesity (hereafter, simply “overweight”) in the US has increased over the past several decades. Individually-targeted prevention and treatment strategies targeting individuals have been disappointing, leading some to propose leveraging social networks to improve interventions. We hypothesized that social network dynamics (social marginalization; homophily on body mass index, BMI) and the strength of peer influence would increase or decrease the proportion of network member (agents) becoming overweight over a simulated year, and that peer influence would operate differently in social networks with greater overweight. We built an agent-based model (ABM) using results from R-SIENA. ABMs allow for the exploration of potential interventions using simulated agents. Initial model specifications were drawn from Wave 1 of the National Longitudinal Study of Adolescent Health (Add Health). We focused on a single saturation school with complete network and BMI data over two waves (n=624). The model was validated against empirical observations at Wave 2. We focused on overall overweight prevalence after a simulated year. Five experiments were conducted: (1) changing attractiveness of high-BMI agents; (2) changing homophily on BMI; (3) changing the strength of peer influence; (4) shifting the overall BMI distribution; and (5) targeting dietary interventions to highly connected individuals. Increasing peer influence showed a dramatic decrease in the prevalence of overweight; making peer influence negative (ie, doing the opposite of friends) increased overweight. However, the effect of peer influence varied based on the underlying distribution of BMI; when BMI was increased overall, stronger peer influence increased proportion of overweight. Other interventions, including targeted dieting, had little impact. Peer influence may be a viable target in overweight interventions, but the distribution of

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body size in the population needs to be taken into account. In low-obesity populations, strengthening peer influence may be a useful strategy.

Keywords

USA; National Longitudinal Study of Adolescent Health; Add Health; social networks; agent-based models; peer influence; obesity; stigma

Introduction

The prevalence of childhood obesity has risen markedly over the past several decades (Ogden, Carroll et al. 2010; Ogden, Lamb et al. 2010), although it appears to have leveled off in recent surveys (Ogden, Carroll et al. 2012). The ineffectiveness of many available intervention strategies has led to a search for novel approaches to preventing and treating childhood obesity (Waters, de Silva-Sanigorski et al. 2011). For this reason, social influences including neighborhood environments (Gordon-Larsen, Nelson et al. 2006) and peer influences (Smith and Christakis 2008) have been examined.

One promising line of research involves network interventions (Valente and Davis 1999; Valente 2012). Longitudinal analyses of children's friendship networks (Shoham, Tong et al. 2012; Simpkins, Schaefer et al. 2013) and play groups (Gesell, Tesdahl et al. 2012) shows peer influence to operate independently of peer selection, although research has not been consistent (de la Haye, Robins et al. 2011).

Social learning theory proposes that individuals' behaviors may arise and be reinforced through observing and imitating others, including peers (Bandura 1977). The mechanism underlying peer influence likely involves conformity (Brechwald and Prinstein 2011), while neuroscientific studies suggest that social rejection is experienced as a form of pain (Kross, Berman et al.). Peer influence is thought to play a role in the development and maintenance of chronic disease and associated risk factors, including overweight and obesity. Peer influence is often modeled as a social network phenomenon. Friendship networks provide information allowing researchers to extend social influence beyond the direct influence of friends. Christakis and Fowler (Christakis and Fowler 2007) found that obesity appears to spread not only between immediate contacts (alters) of study subjects (egos), but also their 2^{nd} and 3^{rd} degree contacts (eg, friends' friends' friends). This suggests that overweight may follow a "diffusion of innovations" model (Rogers 1983), making networks particularly relevant (Valente 1995).

Alters provide the linkage between egos and the larger social network, and therefore they mediate any flow of information, norms, etc. through the network. Simulations conducted by Bahr and colleagues (Bahr, Browning et al. 2009) demonstrated that under various peer influence scenarios, the larger network must be taken into account. However, their study focused on a highly stylized network (grid lattice, although other structures were tried) and a cellular automata framework (ie, agent represented by cells in the grid follow rules based on only their own neighboring cells).

Real world networks are known to differ from simulated networks in important ways including clustering, mean path length, and degree distribution (Hamill and Gilbert 2009). Individual characteristics of networks can be effectively simulated one at a time: path length is reduced in small world networks (Watts and Strogatz 1998); lattice and small world networks show high clustering; and degree distribution can be effectively "grown" using a preferential attachment model (Barabasi and Albert 1999). These sorts of networks have been successfully used to model, eg, the implications for network structure on an epidemic of infectious disease (Rahmandad and Sterman 2008). Attempts have been made to combine several aspects of real-world networks, such as social circles (Hamill and Gilbert 2009) and multiscale network simulation (Gutfraind, Meyers et al. 2012). A further limitation of stylized approaches to simulating social networks is the lack of dynamics seen in the real world. While the Barabasi preferential attachment model (Barabasi and Albert 1999) does allow for dynamic growth of the network, it allows for no loss of ties once they are formed. For these reasons, basing simulations on a real-world network would be desirable.

However, real world networks present a challenge for understanding peer influences including the mechanisms underlying such influence (Cunningham, Vaquera et al. 2012). While we can observe clustering of health-related behaviors in networks (e.g., obesity), we cannot directly observe the mechanisms that gave rise to such clustering. Endogenous (peer influence) effects may give rise to the same clustering phenomenon as exogenous (shared environmental) effects and shared background characteristics (selection or homophily) effects. Manski calls this the "reflection problem," since effects mirror one another (Manski 1993). There have been several critiques of the social network "contagion" hypothesis in academic (Cohen-Cole and Fletcher 2008; Cohen-Cole and Fletcher 2008; Halliday and Kwak 2009; Shalizi and Thomas 2010; Shalizi and Thomas 2011) and popular (Johns 2010) literatures. In essence, three mechanisms might account for observed contagion: true social influence; confounding by shared environments; and homophily ("love of sameness") on shared predisposition to BMI and related behaviors. The actor-based stochastic model (Snijders, van de Bunt et al. 2010; Steglich, Snijders et al. 2010) offers perhaps the only way to tackle this problem by iteratively modeling the evolution of social network structure and the behavior of individuals in the network. This method has been implemented in the R package SIENA (Simulation Investigation for Empirical Network Analysis). Parameter estimates from R-SIENA could then be used to explore various interventions, using an observed dynamic network as a basis.

It is unclear how social networks could be exploited to promote behavior change. One suggested approach is to target the behavior of key nodes, or "opinion leaders," which can be provisionally defined as the individuals that have the highest in-degree (Valente and Davis 1999). However, as Valente has noted, there is limited empirical work to support network-based interventions (Valente 2009), and given the great expense and effort involved in collecting network data, simulations may offer insights as to which interventions are more or less likely to succeed. Agent-Based Models (ABMs) allow us to create simulated agents in a computational platform, assign them traits (such as behaviors) and rules for interacting with other agents and the environment. We can then run simulation experiments, and observe the network change and behavior change of individuals and the system as a whole. The primary goal of our simulations is to gain insights into what network mechanisms are

salient (or irrelevant) for obesity and which obesity-related approaches might leverage social networks.

In this study, we built an agent-based model (ABM) of adolescent body mass index (BMI) and tested the impact of social influence on combined overweight and obesity prevalence in the population (hereafter referred to simply as "overweight", defined as a BMI of at least $25 kg/m^2$). We validated this model by comparing network characteristics (distribution of in-degree, out-degree, and triad census) and behavior (mean BMI, distribution of BMI) between simulated networks and the observed network after one simulated year. We examined several aspects of social influence, including the robustness of the system to changes in particular parameters, which can be interpreted as behavior rules. We focused on peer selection, strength of peer influence, and whether targeted weight loss in the overweight population can better reduce prevalence of overweight in the network as a whole.

Methods

Population for deriving parameter estimates

Data were drawn from the first two waves of the National Longitudinal Study of Adolescent Health (Add Health), a representative sample of US high schools. The two waves of data were collected a year apart (2004 and 2005). Details of the study design can be found elsewhere (Harris, Halpern et al. 2009). We focused on one of the largest saturation high schools, referred to elsewhere as Jefferson High School (Moody 2004). This high school is unique because of its rural location and racial homogeneity. A total of 624 respondents were present in both waves and had complete self-reported information on weight and height. We calculated BMI by dividing weight (in kilograms) by height squared (in meters squared). The model included network parameters (outdegree, reciprocity, transitive triplets, homophily on sex, grade, age, and income, attractiveness of higher BMI, sociability of those with higher BMI, and homophily on BMI). Summary values for the population at baseline are reported in Table 1.

Overview of the empirical analysis

The SIENA package in R was used to obtain initial parameter estimates, which were the bases for specifying rules in our agent-based model. The R-SIENA model is a type of discrete choice model (Train 2009); actors in the model are assumed to make particular choices, and the utility associated with various choice is inferred from the choices actually made. Results of this model were previously published (THE AUTHORS). Table 2 shows the parameters from our empirical analysis. We cannot elaborate on details of the R-SIENA model here, but in essence, there are four types of parameters estimated by the model. Two rate parameters estimate how much change there is in the network ("Basic rate parameter friendship") and how much change there is in BMI ("Rate parameter for BMI behavior"). The other two types of parameters are network and behavior effects. Confidence intervals are reported for parameter measures, allowing us to judge the range of values and whether the null value lies within the confidence intervals. We are primarily interested in BMI-related effects in our model: "attractiveness of alter's BMI", which is negative if overweight or obese respondents are less likely to be named as friends (see (Strauss and Pollack 2003));

"Similarity of ego's and alter's BMI", which is positive if there is homophily on BMI (i.e., two respondents are more likely to form a friendship tie if they are similar in BMI); and "BMI average similarity", which is a measure of peers' influencing one another's BMI (i.e., the tendency to assimilate to the average friends' BMI). Other homophily parameters include sex, grade, age, and income similarity. Other structural effects in the model include outdegree (negative, because there is a tendency to limit friendships), reciprocity (the tendency to return friendship nominations), transitive triplets (egos are more likely to form friendships with their friends' friends), and sociability related to ego's BMI (all things being equal, those with higher BMI have a tendency to name more friends). Actors in the model may either make a network change or a behavior change, and only one change may be made at a time and in one-unit increments (for example, add or drop a single tie; increase by one unit of BMI). The BMI linear and quadratic shape parameters relate current BMI to future gain or loss, as those with BMIs above the average have a tendency to gain more weight; the quadratic shape parameter is positive because this tendency is compounded at higher BMI values, which is consistent with underlying addictive behavior (Snijders, van de Bunt et al. 2010).

Translating parameter estimates into probabilities for behavior in the agent-based model

Variables are standardized by dividing by the range of possible values then centering at the mean. Parameters may be interpreted as the log-odds of taking a particular action for each one-unit increase in the standardized variable. For example, the mean similarity in BMI in the population (i.e., the average difference between any two agents) was 0.865, while the range of potential BMI similarity was 33 (the largest value of BMI minus the smallest value). For a respondent to move in the direction of friends' BMI, the similarity score would increase by 1/33, or 0.0303; moving away from the average of alters would similarly decrease this score by that amount. The difference in similarity score can then be multiplied by the parameter estimate and exponentiated, much as one would do in a logistic regression model. The odds ratio of moving one unit in the direction of average friend's BMI vs. staying at the same BMI is thus exp(0.0303*14.1) = 1.53. Taking into account the tendency for overweight individuals to continue on a trajectory of weight gain ("BMI linear shape" and "BMI quadratic shape" parameters), we can calculate the contribution of all three parameters to an objective function for BMI. We can make similar calculations for the network objective function.

The objective function is analogous to a utility function for each respondent; it captures how satisfied he or she is with current network configuration or BMI (or perhaps more realistically, the factors contributing to BMI such as diet, which we could not observe longitudinally with the Add Health data). The objective function may then be translated into an expected probability for choosing one behavior or another. More thorough discussions of discrete choice models and their interpretation may be found elsewhere (Train 2009).

Building an agent-based model

Based on these parameters, an agent-based social network model was built using the NetLogo platform (Wilensky 1999). NetLogo is a freely available agent-based modeling software package that is easy to implement yet flexible in terms of functionality. Each agent

represents an Add Health respondent (n=624). Code was written to translate parameters into objective functions, and the initial agent attributes were observed Add Health attributes at Wave 1. The empirical network at Wave 1 was also used as the initial condition for friendship ties between agents. Because of the friendship ties, agents serve as the local environment for one another.

In order to keep close to the empirical data, models were run for a simulated "year", defined by the rate functions (12.87 + 4.17 = 17.04) and the number of agents (624). In other words, each agent was chosen on average 17.04 times, creating 10,632 total opportunities for change. Each change opportunity was a "tick" or step in the model. At each tick, an agent was randomly chosen, and then asked to make either a network change or a behavior change, with probability proportional to the rate function for each type of change (ie, 75.5% probability of a network change; 24.5% of a BMI change). If it is a network change, the agent calculates the value of the objective function for its current network configuration. Next, the agent calculates what the objective function would be if it were to add a new tie or drop an existing tie. Because there are (n-1) potential ties with other actors, plus one configuration with no ties, the agent calculates 624 objective function values. These values are then translated into probabilities for any particular network "move" (including retaining the same network), arrayed into a list of cumulative probabilities, and compared against a random draw from the uniform distribution with range of (0,1). If the randomly selected draw falls within the range of the cumulative probability for a particular move, the agent chooses that move. An overview of the modeling procedure is shown in Figure 1. In order to simplify the model, we assume that all parameters represent causal effects that are independent of one another.

The rule for BMI change according to the objective function values is similar, but computationally much simpler: the only choices are to move down one unit, stay the same, or move up one unit. Agents compare their own BMI to the BMI of others. Agents with higher BMIs have an inherent tendency to increase weight, which is also part of the calculation. If an agent has friends with lower BMI, this serves as a check against gaining weight, while having friends with higher BMI promotes further weight gain. The objective function for maintaining the same BMI is thus:

$$u_{same}=\exp[(0.16^{*}\mathrm{BMI})+(0.015^{*}\mathrm{BMI}^{2})+(14.1^{*}\mathrm{SIM})]$$

where BMI is the agent's current BMI, centered on the population average, and SIM is the average similarity between the agent's current BMI and its friends' average similarity, scaled by the BMI range (33) and centered by the population average (0.865). The agent also evaluates the objective function for increasing and decreasing BMI by 1 unit at each time step, then calculates a probability for increasing or decreasing BMI or staying the same by dividing the exponentiated value for a particular choice by the sum of the exponentiated values for all 3 possible choices. In effect, the threshold for gaining weight is lowered if the average friend has greater body size than the agent, and increased if the average friend has a smaller body size. The threshold for gaining weight is also lowered if the agent has a large BMI relative to the average.

All simulations were run 100 times with different random seeds, allowing us to generate box plots and 95% confidence limits of the distribution of model runs. Model runs took approximately 5 minutes each; on a 4-core, 2.66GHz, 64-bit Windows workstation using all four processors, the 100 runs were completed in under 3 hours.

Model validation

In order to verify that the model was capturing network dynamics and patters of behavior, we compared simulated results at the end of the model run to empirical observations of the network and BMI at Wave 2 of Add Health. Statistical analyses and plots were created using the R statistical analysis program. We focused on three key measures of the network: the distribution of in-degree (number of friends "named" by each agent); distribution of out-degree (number of alters that "named" the agent as a friend); and the triad census, according to the standard taxonomy for naming the 16 possible configurations of 3 agents (Holland and Leinhardt 1970). For the triad census, we were especially concerned about the possibility of over-simulating 3-cycles, which are rarely observed in real friendship networks, and under-simulating cliques of size 3, which should be relatively common (Davis 1970; Snijders, van de Bunt et al. 2010). For BMI, we compared observed BMI at wave 2 and simulated BMIs after one simulated year. If simulations are accurately capturing the distributions, we should see the empirical estimates falling within the 95% confidence limits.

Experiments

We conducted a suite of experiments, based on the empirical network. In these experiments, we emphasize general qualitative features of the results, rather than precise measures of prevalence. Our goal with these experiments is to guide intuition for model refinement and future study (including intervention studies) rather than to obtain a precise measure of the effect of certain "what-if" scenarios.

Experiment 1. Does marginalization reinforce BMI and overweight?—First, we asked if peer selection (assuming peer influence is operative) drives overweight rates up or down. Several hallmarks of complex systems make the answer to this question non-intuitive, especially feedbacks between peers and non-linearity of the system. If overweight students tend to form friendships amongst themselves, this could both reinforce obesity-related behaviors (which would tend to increase obesity prevalence), and isolate non-overweight students from peers' obesity-related behaviors (which may mitigate obesity prevalence). On the other hand, overweight students' ties with non-overweight friends may mitigate overweight in the overweight group, but increase it in the non-overweight group. We began with the original value of marginalization (−0.007), then tripled this value (− 0.021) to make it strongly negative. Translating into odds ratios, an agent with a BMI of 30 kg/m^2 would have an odds ratio of 0.81 for receiving a friendship tie compared to an agent with a BMI of 20 kg/m^2 (10 units lower). We also examined what effect removing marginalization would have on the BMI of the system, as well as making high BMI more attractive than low BMI by reversing the sign and tripling the absolute value (+0.021).

Experiment 2. Does increasing homophily on BMI contribute to higher rates of overweight?—To test the effect of peer selection on the BMI of agents, the experiment was conducted on changing the parameter (network similarity) to be 0, negative, or quadrupled. The purpose of this experiment is to test if making friends based on different levels of similarity will have different effects on their BMI. This process is called "homophily", and may be understood by the expression "birds of a feather flock together" (McPherson, Smith-Lovin et al. 2001). To translate this into odds ratios, the estimated parameter value for similarity (−0.54) tells us that agents have 1.14 times the odds of choosing friends with the same BMI, compared to an alter of average similarity. Quadrupling this parameter translates into an odds ratio of 1.34.

Experiment 3. Does increasing the strength of peer influence increase the prevalence of overweight?—We then asked how the strength of peer influence relates to the supposed "spread" of overweight in the system. Christakis and Fowler's work argues that obesity (like influenza) can be caught from friends and relatives (Christakis and Fowler 2007). This may operate via local social influence like peer or role modeling (Bandura 1986), or perhaps as a result of shifting group norms (Hruschka, Brewis et al. 2011). Extrapolating linearly, this suggests that stronger peer influence should lead to higher overweight rates, while weaker influence would lead to lower rates. To do this experiment, we changed the parameter, behavior similarity to be 0, negative number (2 times the influence strength, but in the opposite direction, ie, tell the agent to "do the opposite of my friends") and larger positive number twice the original value (assimilate more strongly to friends' BMI). The purpose of this is to test if peer influence has a positive effect on the spread of overweight. The OR for increasing a unit closer to the average alter vs. staying the same would be 1.54 under the original parameter value (14.1); when we double the parameter to 28.2, this OR increases to exp (28.2*0.0303) = 2.33.

Experiment 4. How does peer influence interact with the distribution of BMI? —Since Wave 1 of the Add Health study was begun in 1994, there have been dramatic increases in overweight among adolescents. The prevalence of overweight has increased to approximately 1 out of 3 adolescents (Go, Mozaffarian et al. 2013); obesity (for children, above the 95^{th} percentile) has increased over 50% since 1988, to 17.5% in boys and 14.7% in girls (Ogden, Carroll et al. 2012). Peer influence may be very different in a population with such high combined rates of overweight and obesity. Therefore, we explored how the strength of influence interacts with the distribution of BMI in the population. We conducted this experiment by shifting the entire distribution of BMI from its initial value (22.4 kg/m^2) by 1, 2, 3, and 4 units up. We then compared negative and strongly positive influences with the original peer influence parameter (see experiment 3 above).

Experiment 5. Are interventions targeting highly connected overweight agents more successful than randomly selecting overweight agents?—Next, we explored a targeted intervention strategy suggested by Valente and Davis (Valente and Davis 1999). We explored two scenarios. First, we randomly selected 10% of agents (n=62), restricted to those who were in the combined overweight or obese category (BMI>=25kg/m^2), who were then placed on a hypothetical successful diet (drop 4 units

BMI). Next, we again selected 10% of the agents (again, n=62) who were overweight or obese, but only choosing those agents with the highest in-degree, i.e., those who might be considered “opinion leaders”. Our thinking was that if these high in-degree individuals drop weight, they may influence the behavior of those they are connected with.

All experiments were conducted at baseline, and the model was run for one simulated year.

Results

Model validation

We ran 100 simulations by using different random seeds. The ABM fit the observed behavior data (body mass index, or BMI), with a correlation coefficient of 0.906 between the simulated mean BMI for 100 runs and real BMI. Simulations were compared to the empirical network and behavior statistics, with a focus on degree, reciprocity, and transitive triads. Figure 2 shows the comparison of the BMI distributions between the simulated data (pooling the data from 100 runs) and the real data. We see that the simulated data fit the real data very well, with the solid line (observed) falling inside the box plots across the distribution.

Next, we compared out-degree (number of friends named) between the simulated data and the observed network (Table 3). Simulated network structural characteristics (out-degree, reciprocated dyads, transitive triads and changes of ties) fit the observed data very well. For example, the empirical network had 1472 transitive triads, while our simulated networks averaged for 1480.76 triads with standard deviation 198.59. Network structure in the observed and simulated networks agrees well, with the average simulation statistics within 2% of the observed value.

We further examined the triad census of the empirical and simulated networks. In general, triad counts for the most important types of triads were similar between observed and simulated networks. There were 262,010 dyads within triads (Holland and Leinhardt (1976) type 102) in the observed network; for the simulations, average count was 250,010. The number of complete cliques of size 3 (Holland and Leinhardt type 300) was 56 in the observed and an average of 135 in the simulated runs. On the other hand, 3-cycles (type 030C) were rare in both observed (11) network and simulated (1) runs.

The final validation step was to compare the distribution of out-degree (number of friends named) and in-degree (number of friends naming the ego). The following plots (Figure 3 and Figure 4) show that the simulated data (100 runs) fit the real data well, with the exception of degrees zero and one. The simulated models consistently under-simulated the number of isolates. Note that the maximum out-degree is 10 in the empirical data, as imposed by the friendship questionnaire employed in Add Health; the theoretical limit on in-degree is much higher (623, for an ego named by all others in a network of 624).

Result for experiment 1 (impact of marginalizing overweight students)

When social marginalization was increased, the prevalence of overweight slightly decreased, although this did not to appear to be substantial [Figure 5; Table 4]. In conjunction with

social influence, isolating obese adolescents might tend to limit their influence on the system, while promoting friendships with leaner adolescents will have the opposite effect. Marginalization does not appear to be a promising strategy given the weak effect on overweight prevalence and the potential for negative psychosocial consequences (Crow, Eisenberg et al. 2008; Tang-Peronard and Heitmann 2008),. Conversely, our findings support efforts to improve social integration of obese adolescents, as such integration is unlikely to have any negative effect on healthy weight peers.

Results for experiment 2 (impact of homophily)

We varied the preference for agents to form or maintain friendships with others who are similar to themselves in body size (homophily on BMI), which would indicate greater clustering based on body size, and potentially more reinforcement of healthy or overweight. We can see that there is no appreciable difference in overall overweight prevalence between different levels of homophily in this model. This means that although students make friends based on the similarity, the effect of similarity do not substantively impact on overweight prevalence. When we artificially set the parameters for homophily on BMI to zero, negative the original, and strongly positive (4 times the original) values, this had no impact on overweight in the system. This suggests that homophily body size does not drive the results.

Results for experiment 3 (impact of social influence)

The percentage of overweight and obese agents differs dramatically among different levels of impact of social influence. As influence increased in this model, the prevalence of overweight decreased. In contrast to a finding of simple “contagion”, influence appears to serve as a check on weight gain. We see that the percentage of overweight and obese is lowest when the social influence is doubled while the percentage is highest with negative influence (Figure 7). When the friends of the student have more influence on the student’s behavior (BMI), the percentage will be lower since there are more normal weight agents in this model. Thus, peer influence may serve as a buffer to overweight, at least in the case where overweight prevalence is relatively low. This suggests that peer influence may be a tool for preventing or reducing overweight in adolescents.

Results for experiment 4 (impact of varying the BMI distribution)

Peer influence relates to the average peers’ value in the network. That is, in a network where most agents are healthy weight (BMI<25kg/m^2), increasing influence will tend to pull back overweight and obese agents. The situation may be different in populations where overweight is the norm. Figure 8 shows the effect of increasing the mean BMI in 1-unit increments. As the mean BMI approaches the cut point for overweigh (25kg/m^2), the absolute value of the slope relating strength of peer influence to the prevalence of overweight decreases to zero; above this value, the slope increases, meaning that greater peer influence increases the proportion of overweight adolescents in a population where most students are overweight. These results thus call for caution in making blanket recommendations for interventions, as peer influence may be beneficial only when healthy weight and healthy behaviors are the norm.

Results for experiment 5 (random vs. targeted weight loss interventions)

Finally, we discuss the results of an overweight intervention that targets particular network members, and compare these interventions with those targeting randomly selecting overweight agents. Figure 9 shows the percentages of overweight and obese students under different selecting strategies. We can see that there is no large difference between selecting overweight and opinion leaders in overweight students (two box plots on the right). The percentages of overweight and obese for the interventions which selected opinion leaders from among the overweight students is lower than randomly selecting obese students by about 1 percentage point (approximately 6 study subjects). On the other hand, there was little difference in the proportion of underweight (defined as BMI $\leq 17 kg/m^2$) across interventions: the mean (95% CI) proportion underweight was 4.9 (3.5, 6.3) under no intervention; 4.9 (3.4, 6.5) under randomly selecting overweight agents; and 4.8 (3.4, 6.3) targeting overweight opinion leaders.

Discussion

The models presented here attempt to move beyond pure simulations by incorporating real-world network structure and dynamics. We have demonstrated a method of translating analytical models into an agent-based model. Conclusions of the models include that (1) peer influence may serve as a buffer to overweight and obesity rather than a vector of its spread, and (2) interventions based on real-world networks may not be as successful as highly clustered (grid lattice-based) simulations would suggest. Furthermore, while the model of Bahr (2009) suggested dieting with friends of friends would be successful, identifying the edges of overweight or obesity clusters in real-world networks is difficult.

Models that explored the role of stigmatization deserve special attention. Stigma's role in perpetuating the AIDS epidemic is well know, yet at the same time, there was a robust push from advocacy groups to actively de-normalize smoking behavior and, by extension, marginalize smokers themselves (Bayer 2008). Bayer argues that stigma is defensible if there are also benefits to the groups being stigmatized, for example, if low-income (and therefore less empowered) smokers are more likely to quit as a result of taxes that burden them, a policy debate that has direct relevance to soda taxes and other efforts to combat childhood obesity (Gortmaker, Swinburn et al. 2011). Using Add Health network data, Strauss (2003) has found that overweight adolescents tend to be socially marginalized, which is one of the aspects of stigmatization. In particular, they nominate about the same number of friends as their non-overweight peers, but these nominations tend to be reciprocated at much lower rates than non-overweight adolescents. This marginalization or stigmatization comes at high social and psychological cost, especially for girls (Crow, Eisenberg et al. 2008; Tang-Peronard and Heitmann 2008), and may increase other unhealthy behaviors (especially tobacco use), exacerbating the problem of childhood obesity (Ratcliff, Jenkins et al. 2011). In a commentary on Bayer's article, Burris (Burris 2008)argues that measures such as taxation are not stigmatizing in and of themselves, although they may certainly support stigma. We therefore strongly oppose policies of stigmatization. We did not find any appreciable effect of increasing marginalization on obesity prevalence, but this does not mean that marginalization is unimportant for obesity.

Following the model of smoking (eg, the Truth campaign), de-normalization of particular unhealthy eating behaviors may be a promising approach, and need not involve stigmatizing. At the same time, such de-normalization of behaviors must be careful to avoid de-humanizing and shaming individuals, which are the hallmarks of stigmatization (Nussbaum 2004; Burris 2008).

We wish to highlight one of the unexpected findings of our simulations: the inverse relationship between the strength of the peer influence parameter and the prevalence of overweight at the end of the simulations. The metaphor of obesity spreading like a virus (or "catching" obesity from friends) is misleading, as contagion may work both ways (ie, one can "catch" health and healthy behaviors as well). Peer influence will act to pull agents at the edges of the distribution toward the mean; as the mean increases, so will what is considered "normal". This phenomenon has been observed in the increase in what is considered a normal body size overall (Burke, Heiland et al. 2009) and heterogeneity by racial-ethnic-gender group (Lynch, Liu et al. 2009). On the other hand, there is a basic biological tendency to accumulate and store fat, likely arising out of natural selection, as first proposed by Neel (Neel 1962). Our study population was relatively lean, with few overweight or obese adolescents; in a population with higher mean BMI, peer influence may serve to reinforce overweight and obesity, and our future work will explore this phenomenon using BMI distributions that are similar to those observed today.

This brings attention to a limitation of the use of real-world data. Because our model is closely tied to the Add Health data analyzed by THE AUTHORS, the results may not be generalizable outside of the population on which it is based. Nevertheless, one of the strengths of the ABM is their generality and flexibility (Epstein 2006; North and Macal 2007); gaining future insights into the mechanisms (and potential policy targets) may require us to move beyond specific data as an input and to explore ranges of mechanisms that cannot be captured through analytical methods. Related to this, the model does not extend beyond one simulated year, when in fact we know that individuals' weight continues to increase. Furthermore, we made a decision to include independent parameters in our model and explore changes one at a time. However, in the real world, peer influence and social marginalization may be interdependent and contingent. For example, if the social marginalization parameter were stronger, this may also entail a stronger peer influence parameter. We plan to explore such interdependence by relaxing this independence assumption in future work.

In the base case scenario, our model does a good job of predicting dynamics at the system level. However, predicting individual agents' behavior is not possible in this model. In part, the divergence between individual and population-level fit may be due to the underlying SIENA model that serves as the primary input to our ABM; for large networks, SIENA fits a simulation model to population-level targets, although a maximum likelihood option is available which is more statistically efficient for small networks (Snijders, Koskinen et al. 2010). A second reason for the discrepancy may be due to the general inability of any model to predict individual outcomes, a phenomenon that has been observed in epidemiology (Rockhill 2001; Rose 2001), while Macintyre offers a philosophical explanation of the impossibility of predicting human social behavior (MacIntyre 1984).

We admit that the intervention proposed (a 4 unit drop among key agents) is an exogenous shock, and the means by which such weight loss is accomplished is unspecified by our model. We would argue, however, that while the specified weight loss is extreme and thus not realistic, the purpose of the model is not to explore whether one weight loss method is superior to another. Rather, our goal was to demonstrate the impact of network structure and dynamics on such a hypothesized intervention; to demonstrate that network members may be resilient to even highly successful interventions; and to show that proposed intervention strategies relying on peer leaders is unlikely to yield great benefit in the case of adolescent overweight and obesity. Similar findings were recently reported by El-Sayed and colleagues, using an ABM based on the work of Christakis and Fowler (El-Sayed, Seemann et al. 2013) but lacking the empirical network or network dynamics examined in our work. We note that experiments targeted only highly-connected overweight agents did not lead to any spillover effect on underweight, which would have been an unfortunate side effect.

Finally, we have demonstrated the novel finding that given an individual predisposition to gain weight, social norms can serve as a check (negative feedback). Identifying what specific normative mechanisms allow for social homeostasis of weight would be a fruitful endeavor. We have only assumed a limited range of mechanisms of peer influence, specifically, modeling behavior of average friends, and the potential stigmatization that can arise from overweight/obese. Furthermore, interventions that strengthen peer influence, such as promoting positive behaviors, may help reduce obesity incidence. At the same time, there is a potential to undermine health promotion campaigns, for example, by forcing students to interact with one another within official school settings mandated by authorities. This may have the unintended effect of watering down and reducing social influence, a phenomenon deemed “policy resistance” (Sterman 2006). Many other mechanisms and outcomes are possible, and we will explore these in future work.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Highlights

- Social networks may help us understand adolescent overweight and obesity
- Agent-based models allow for exploration of interventions on dynamic social networks
- Dieting interventions targeted to highly connected individuals are not promising
- Interventions that increase peer influence may be worthwhile
- Interventions are sensitive to underlying distributions in the network

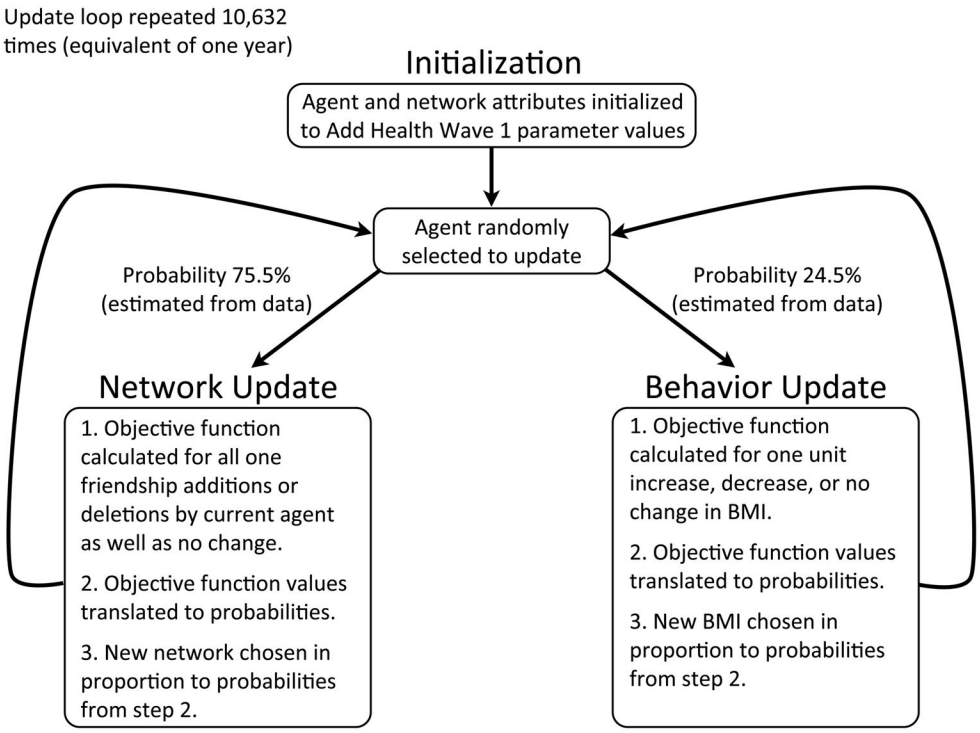
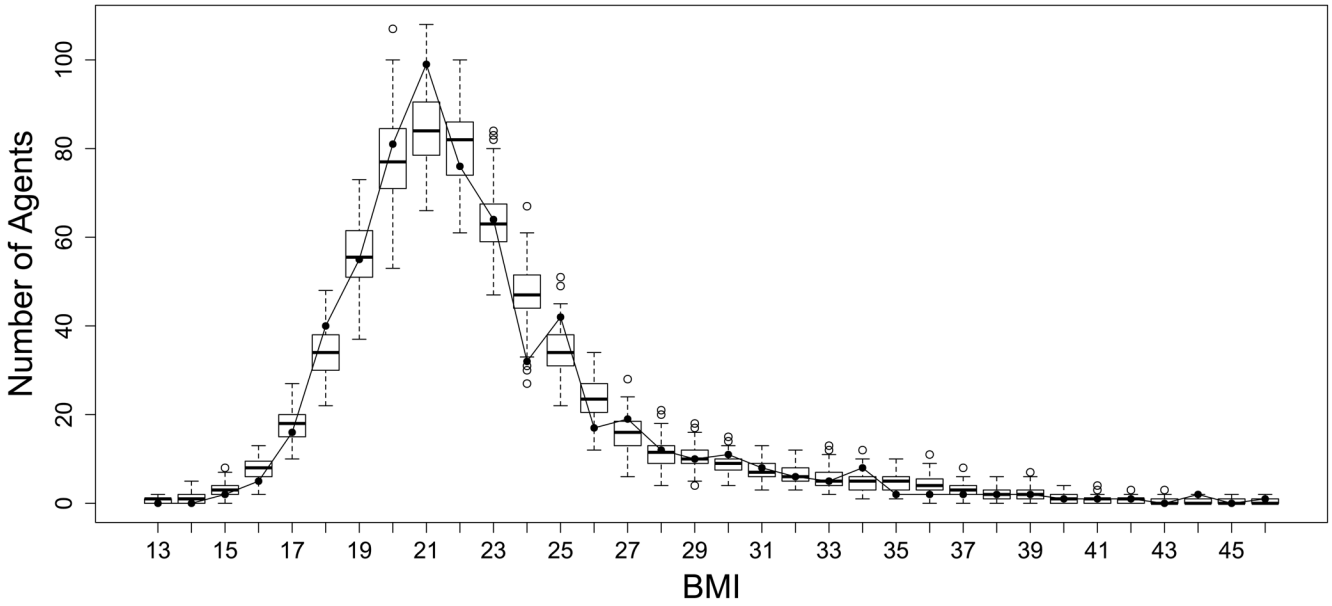


Figure 1.



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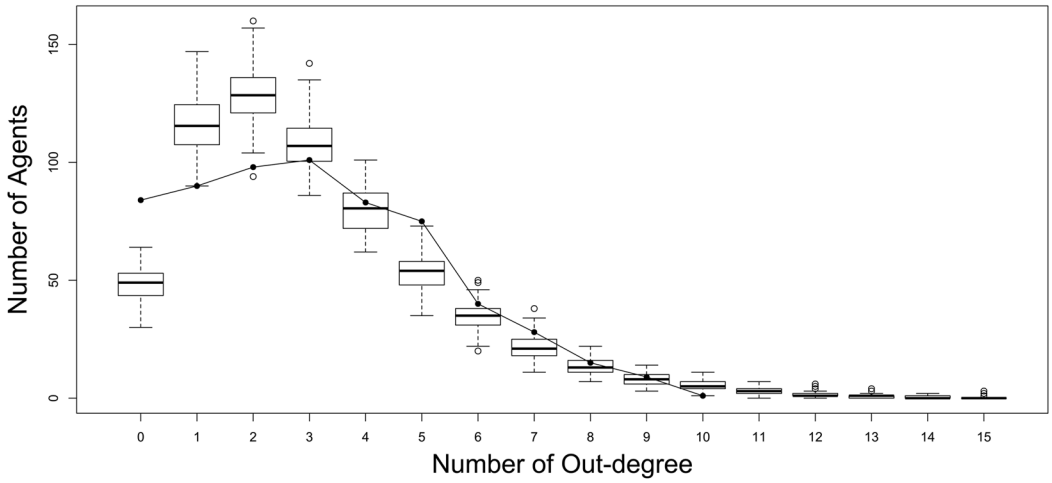

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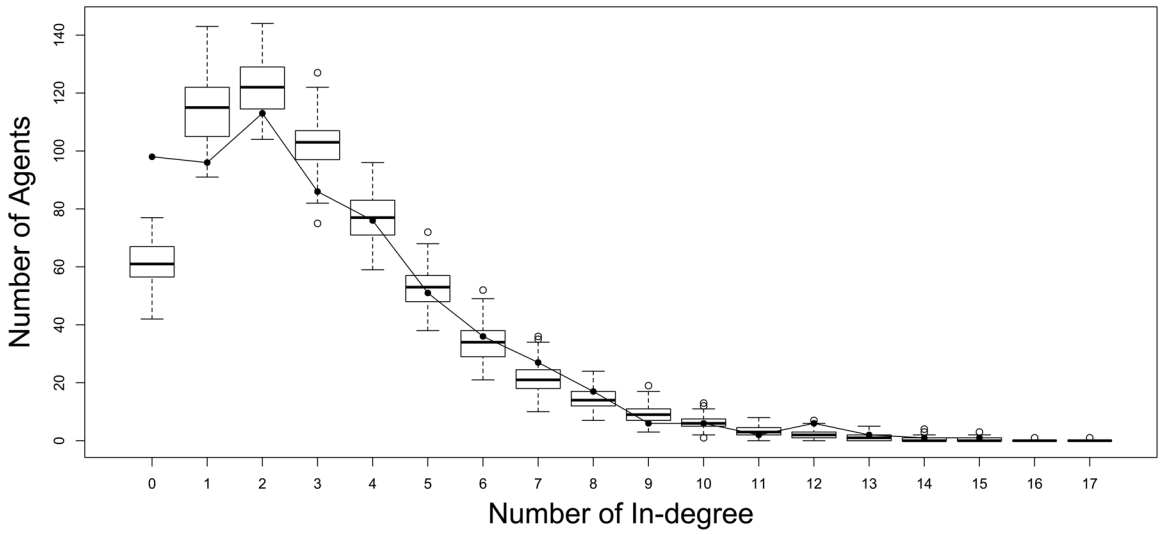

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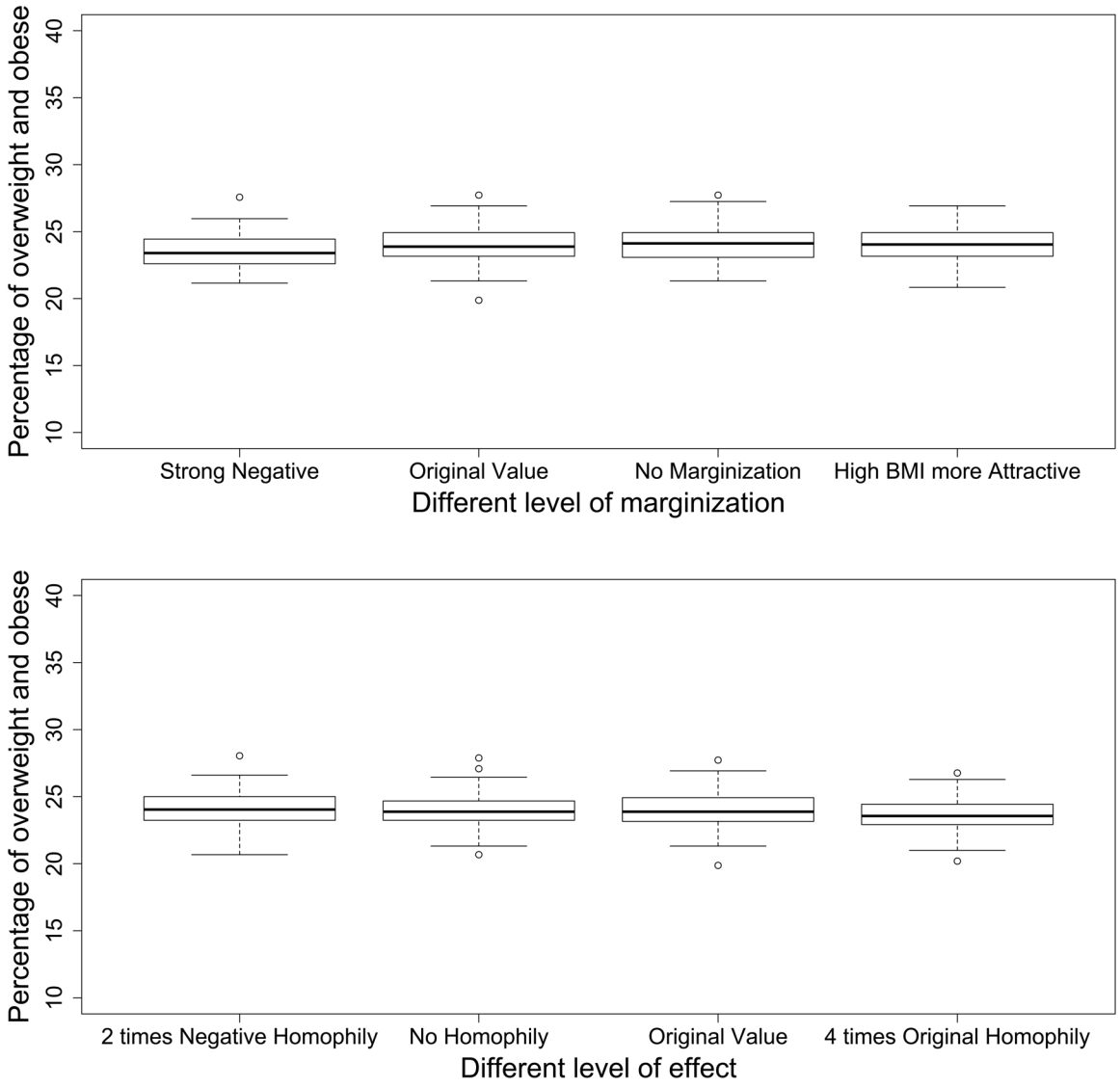

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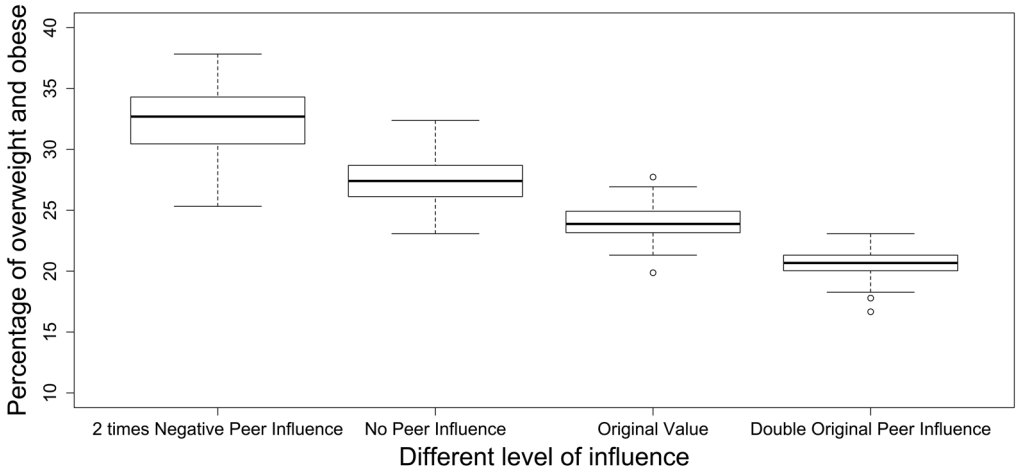

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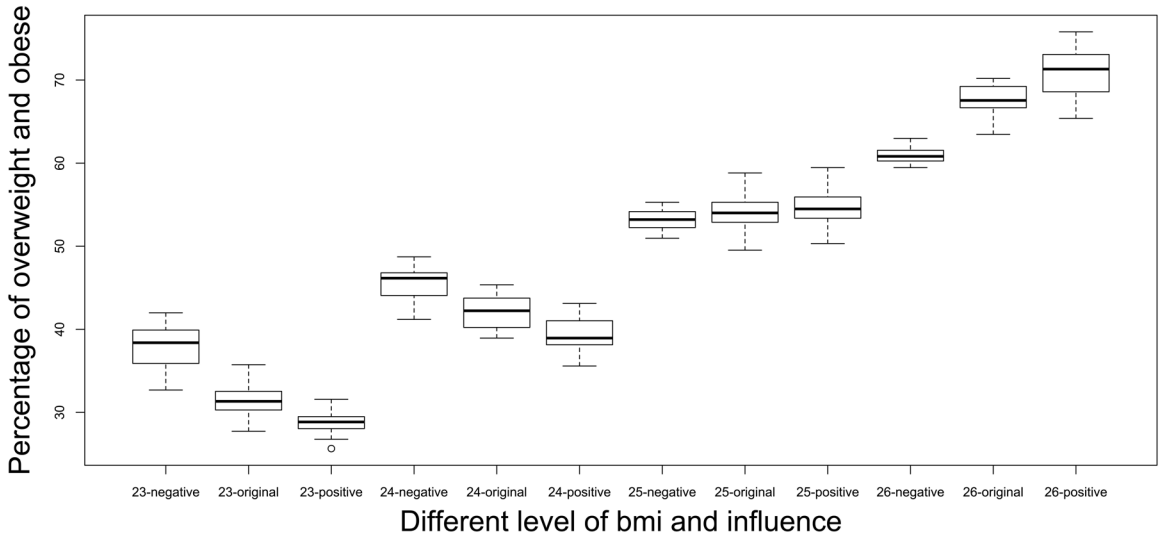

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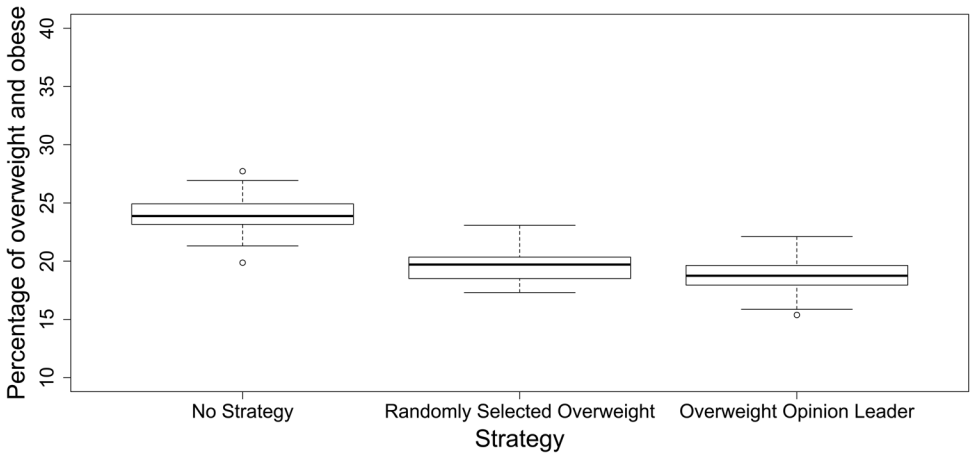

Figure 8.

Table 1

Baseline characteristics

Measure	Value
Number of respondents	624
Mean age at baseline (SD)	16.1 (1.1)
Range of grades at baseline	9 to 11
Male	47.4%
Household income, in $1000 (SD)	45.2 (26.7)
Mean BMI at baseline (SD)	22.4 (4.4)
Range of BMI at baseline (min – max)	13 to 43
Total number of ties at baseline	2201
Mean out-degree (SD) at baseline	3.5 (2.3)

SD= standard deviation

BMI= Body mass index, in kg/m^2

Table 2

Parameter Estimation from SIENA model, with 95% confidence limits

Basic rate parameter friendship	12.87 (12.62, 13.85)
Outdegree (density)	−3.56 (−3.64, −3.48)
Reciprocity	2.26 (2.13, 2.39)
Transitive triplets	0.48 (0.43, 0.53)
Same sex	0.18 (0.10, 0.26)
Same grade	0.49 (0.41, 0.57)
Age similarity	0.91 (0.62, 1.20)
Income similarity	0.060 (−0.23, 0.35)
Marginalization based on alter's BMI	−0.007 (−0.017, 0.003)
Sociability related to ego's BMI	0.014 (0.003, 0.030)
Similarity of ego's and alter's BMI	0.54 (0.14, 0.95)
Rate parameter for BMI behavior	4.17 (3.57, 4.76)
BMI linear shape	0.16 (0.11, 0.22)
BMI quadratic shape	0.015 (0.004, 0.012)
BMI average similarity between ego and alter	14.10 (7.76, 20.44)

Table 3

Overall comparisons of network

	Observed	Simulated (standard deviation)
Number of ties	1943	1952.72(67.64)
Number of transitive triads	1472	1480.76 (198.59)
Number of reciprocated dyads	856	861 (37.32)
Number of tie changes	2484	2448.32 (204.47)

Table 4

Summary of experimental results

Experiment 1: Change Marginalization		**Mean Percent Overweight (95% CI)**
Strong Negative Marginalization		23.5 (21.0, 26.1)
Original Value		24.0 (21.4, 26.6)
No Marginalization		24.0 (21.4, 26.6)
High BMI more Attractive		24.1 (21.4, 26.7)
Experiment 2: Change Homophily		
2 times Negative Homophily		24.1 (21.4, 26.8)
No Homophily		23.9 (21.4, 26.5)
Original Value		24.0 (21.4, 26.6)
4 times Original Homophily		23.7 (21.3, 26.1)
Experiment 3: Change Influence		
2 times Negative Peer Influence		32.3 (27.4, 37.3)
No Peer Influence		27.3 (23.7, 30.9)
Original Value		24.0 (21.4, 26.6)
Double Original Peer Influence		20.6 (18.6, 22.7)
Experiment 4: Change influence under shifted BMI distribution		
Mean BMI 23	Negative	38.0 (33.1, 42.8)
	Original	31.6 (28.0, 35.1)
	Positive	28.8 (26.5, 31.1)
Mean BMI 24	Negative	45.5 (41.8, 49.3)
	Original	42.0 (38.2, 45.7)
	Positive	39.4 (35.6, 43.2)
Mean BMI 25	Negative	53.2 (50.8, 55.6)
	Original	54.0 (49.7, 58.3)
	Positive	54.7 (50.3, 59.1)
Mean BMI 26	Negative	61.0 (59.1, 62.8)
	Original	67.7 (64.4, 71.1)
	Positive	70.9 (65.7, 76.1)
Experiment 5: Targeted vs. random interventions		**Mean Percent Overweight (95% CI)**
No Strategy		24.0 (21.4, 26.6)
Randomly Selected Overweight		19.5 (17.0, 22.1)
Overweight Opinion Leader		18.7 (15.9, 21.6)
		Mean Percent Underweight (95% CI)
No Strategy		4.9 (3.5, 6.3)

Randomly Selected Overweight	4.9 (3.4, 6.5)
Overweight Opinion Leader	4.8 (3.4, 6.3)