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

J. Zhang [a], L. Tong [a], P.J. Lamberson [b], R.A. Durazo-Arvizu [a], A. Luke [a], D.A. Shoham [a, *]

[a] Department of Public Health Sciences, Stritch School of Medicine, Loyola University Chicago, Maywood, IL 60153, USA
[b] Kellogg School of Management, Northwestern University, 2169 Campus Dr, Evanston, IL 60208, USA

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

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1. Introduction

The prevalence of childhood obesity has risen markedly over the past several decades (Ogden et al., 2010a; Ogden et al., 2010b), although it appears to have leveled off in recent surveys (Ogden 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 et al., 2011). For this reason, social influences including neighborhood environments (Gordon-Larsen 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 et al., 2012; Simpkins et al., 2013) and play groups (Gesell et al., 2012) shows peer influence to operate independently of peer selection, although research has not been consistent (de la Haye 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 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 2nd and 3rd degree contacts (e.g., 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 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 (i.e., 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, e.g., 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 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 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, 2008a, 2008b; Halliday and Kwak, 2009; 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 et al., 2010a; Steglich 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.

2. Methods

2.1. 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 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.

2.2. 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-

Table 1
Baseline characteristics.

Measure	Value
Number of respondents	624
Mean age at baseline (SD)	16.1 (1.1)
Range of grades at baseline	9–11
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}.