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Comparing methods of targeting obesity interventions in populations: An agent-based simulation



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ABSTRACT

Social networks as well as neighborhood environments have been shown to effect obesity-related behaviors including energy intake and physical activity. Accordingly, harnessing social networks to improve targeting of obesity interventions may be promising to the extent this leads to social multiplier effects and wider diffusion of intervention impact on populations. However, the literature evaluating network-based interventions has been inconsistent. Computational methods like agent-based models (ABM) provide researchers with tools to experiment in a simulated environment. We develop an ABM to compare conventional targeting methods (random selection, based on individual obesity risk, and vulnerable areas) with network-based targeting methods. We adapt a previously published and validated model of network diffusion of obesity-related behavior. We then build social networks among agents using a more realistic approach. We calibrate our model first against national-level data. Our results show that network-based targeting may lead to greater population impact. We also present a new targeting method that outperforms other methods in terms of intervention effectiveness at the population level.

1. Introduction

The obesity epidemic has been linked to a web of interdependent causes operating at multiple cascading levels (Galea, Riddle, & Kaplan, 2010; Glass & McAtee, 2006; Huang, Drewnoski, Kumanyika, & Glass, 2009) including environmental influences, genetics, cultural preferences, environmental cues, food pricing and availability, and peer influence (Myers & Rosen, 1999). These complex relationships have been widely studied using conventional study designs and regression-based models. However, it is increasingly understood that obesity is an outgrowth of complex dynamic processes at multiple levels that demonstrate non-linear features such as feedback loops and endogenous peer influences that are not well-captured using conventional approaches (Finegood, 2012; Finegood & Cawley, 2011; Galea et al., 2010; Hammond & Dubé, 2012; Huang & Glass, 2008; Ip, Rahmandad, Shoham, Hammond, & Huang, 2013). The complexity of the obesity epidemic has drawn attention from researchers from a wide range of disciplines seeking new strategies to study the drivers of and solutions to the epidemic. Therefore, increasingly, agent-based computational models (ABMs) have been explored as an alternative approach for addressing scientific and policy questions and as a focal

point for collaborations of multidisciplinary teams.

Agent-based models are computational simulations of real-world dynamic patterns of adaptive behavior (Auchincloss & Diez Roux, 2008; Bonabeau, 2002; Gilbert & Troitzsch, 2005). Their principal strength is the ability to model and capture emergent collective behavior arising from dynamic adaptation of knowledgeable actors who seek strategic solutions in the face of environmental constraints and whose complex interactions create emergent patterns that cannot be predicted or understood using conventional methods that do not permit non-linear dynamics (Epstein, 2006; Epstein & Axtell, 1996; Macy & Willer, 2002; Maglio & Mabry, 2011). In obesity research, ABMs have been used previously to understand the role of the food and physical activity (PA) environments (Auchincloss & Diez Roux, 2008; Widener, Metcalf, & Bar-Yam, 2013; Yang, Diez Roux, Auchincloss, Rodriguez, & Brown, 2011; Yang & Diez-Roux, 2013), social norms (Auchincloss, Riolo, Brown, Cook, & Diez Roux, 2011; Hammond & Ornstein, 2014; Mooney & El-Sayed, 2014; Shoham, Tong, Lamberson, Auchincloss, & Zhang, 2012; Wang, Xue, Chen, & Igusa, 2014), network and peer effects (El-Sayed, Scarborough, Seemann, & Galea, 2012; Hammond & Ornstein, 2014; Shoham et al., 2012; Trogdon & Allaire, 2014), and diffusion of interventions

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(El-Sayed, Seemann, Scarborough, & Galea, 2013; Rahmandad & Sterman, 2008; Widener et al., 2013; Zhang, Giabbanelli, Arah, & Zimmerman, 2014). It is this last application that is our principal focus, to which we now turn.

A central challenge in public health response to the obesity epidemic is the lack of consensus about the optimal strategy for targeting intervention resources. While behavioral interventions to prevent and reduce pathogenic weight gain in various populations have proven difficult, there are strategies that have been tested and found to be, to varying degrees, efficacious. These include interventions to reduce caloric intake and increase physical activity over a sustained period for purposes of weight reduction or obesity prevention. For instance, given a fixed pool of available resources, policy makers, program managers, and other decision makers must decide how to target resources to achieve the maximum desired benefit across a target population. Given a behavioral intervention of fixed efficacy and fixed cost per person (on average), should we target those who are obese, those who live in high-risk areas, or choose at random? This is an ideal problem for agent-based simulation models that can be used to conduct counterfactual experiments to test alternative targeting strategies (El-Sayed et al., 2013). This approach has been effective in tobacco. For example, Levy used a simulation model to show that targeting youth smokers results in limited impact compared to targeting all age groups (Levy, Cummings, & Hyland, 2000).

The main goal of this paper is to develop and use an ABM to evaluate different methods of targeting obesity interventions. Therefore, a model is needed that can, at minimum, incorporate three key factors determining the diffusion of intervention effects throughout a population: personal characteristics of actors, social network ties and social influence, and the role of environmental factors (Andajani-Sutjahjo, Ball, Warren, Inglis, & Crawford, 2004). We assume a fixed funding pool from which a fixed number of persons can be enrolled in a well-validated behavioral intervention.

To evaluate population intervention effectiveness, we begin by selecting the state-of-the-art behavioral intervention shown to be efficacious in randomized experiments of two key behavioral pathways: dietary intake and physical activity. For this analysis, we assume an average intervention effect size based on Cochrane Reviews of obesity prevention interventions (Brown, Avenell, Edmunds, Moore, & Whittaker, 2009; Doak, 2002; Mastellos, Gunn, Felix, Car, & Majeed, 2014; McTigue, Harris, Hemphill, Lux, & Sutton, 2003; Prevention & Glickman, 2012). We identified and reviewed randomized trials of adults who represented all weight classes or overweight and obese. We included only studies that reported behavioral outcomes (change in diet or physical activity) with at least 6 months of follow-up. We prioritized studies that involved intensive non-pharmacological interventions that would be moderate in cost and could be scaled up with sufficient resources. Studies of disease groups (e.g., diabetes) or among only obese adults were excluded. We selected the best studies that also reported pre-post intervention change in diet or PA, where the latter was measured with a pedometer or accelerometer. For each category (diet or PA) we summarized the top and bottom of estimated proportional change. For our final estimate, we chose the midpoint of the range. For dietary change, we used the America on the Move trial for the upper bound estimate (Rodearmel, Wyatt, Stroebale, Smith, & Ogden, 2007; Stroebale, de Castro, Stuht, Catenacci, & Wyatt, 2009) and the Diabetes Prevention Program (DPP) (Group, 2002; Mayer-Davis, Sparks, Hirst, Costacou, & Lovejoy, 2004) for the lower bound. The mid-point estimate is 15% reduction in total kcals of consumption at 6–12 months. For physical activity, we base the upper-bound estimate on the trial by Dinger, Heesch, Cipriani and Qualls (2007) that used pedometers to investigate increased walking after intensive intervention based on the transtheoretical model of behavior change. For a lower bound estimate, we used the Reasonable Eating and Activity to Change Health study (REACH) a randomized trial of 665 overweight men and women ages 40–69 followed for 2 years after an

intensive behavioral intervention tailored to the subjects stage of change (Logue, Sutton, Jarjoura, Smucker, & Baughman, 2005). The mid-point estimate for proportional change in physical activity based on these trials is 17%.

Existing research show that obesity patterns can be contagious; friends and family can affect an individual's behavior (Ali, Amialchuk, Gao, & Heiland, 2012a; Ali, Amialchuk, & Rizzo, 2012b; Baker, Little, & Brownell, 2003; Blanchflower, Landeghem, & Oswald, 2009; Centola, 2011; Christakis & Fowler, 2012, 2007; Crandall, 1988; de la Haye, Robins, Mohr, & Wilson, 2011a, b; Eisenberg, Neumark-Sztainer, Story, & Perry, 2005; El-Sayed et al., 2012; Sentočnik, Atanasjević-Kunc, Drinovec, & Pfeifer, 2014). For instance, an individuals' chance of becoming obese increases as their friends or family became obese. As Trogdon and Allaire (2014) point out, the burgeoning literature on peer effects on obesity has important policy implications: social multiplier effects imply that interventions to reduce obesogenic behaviors may spill over and translate to increase overall population impact. A key goal of this analysis was to evaluate which targeting strategy leads to larger overall impact via social multiplier effects.

We address this problem from a computational modeling point of view, and build an ABM that simulates the outcomes of different targeting methods including selected realistic factors that may interact. There exists a limited but rapidly developing literature for modeling social influence on obesity patterns, and studying network-based obesity interventions. However, the literature seems to provide contradictory conclusions. On one side, Zhang, Tong, Lamberson, Durazo-Arvizu, and Luke (2015) finds no differences between selecting random vs. overweight opinion leaders. El-Sayed et al. (2013) claims that interventions that target the most well-connected individuals in a population will have little or no added value compared with at-random implementation. On the other hand, Bahr, Browning, Wyatt, and Hill (2009) find that random targeting approaches require more individuals to effect the same change as targeting well-connected individuals on cluster edges. Similarly, Trogdon and Allaire (2014) show that the effect of population-level interventions depend on the underlying social network, and selecting the most popular obese agents for weight loss interventions resulted in greater population impact. These models have been estimated using different datasets in both adult and adolescent populations. Moreover, different network structures have been used to build simulated networks. This includes random, lattice, scale-free, small-world and online social networks (Barabasi, 2009).

In all of existing work, the concept of behavioral induction has been used to implement peer influence, which leads to diffusion of behavior change throughout the network. The structure of the network, for instance small-world vs. scale-free, does not affect intervention outcomes significantly (El-Sayed et al., 2013; Trogdon & Allaire, 2014). However, the social diffusion dynamics have differed dramatically, which may explain differences in results. Since the population effectiveness of any simulated intervention is directly determined by the model's assumptions about the diffusion process, it is critical to validate this part of the model before exploring intervention strategies with the model. In this paper, we limit ourselves by holding the diffusion dynamics under consideration constant, focusing exclusively on how different targeting strategies alter population impacts. The question of whether alternate diffusion dynamics may magnify or weaken the impact of interventions across targeting strategies will be the subject of a subsequent analysis.

2. Materials and methods

In this section we introduce the details of our ABM, and describe the diffusion model that was used for simulating the spread of the intervention's effect through social networks. By diffusion model, we refer to the social diffusion dynamics that are assumed for the propagations of behavior change and obesity in a social network. We

then present the details of the network structure used to connect the agents in the model. The five different targeting methods that are compared in this study are introduced after this. At the end, implementation details and parameter values of our ABM are discussed.

2.1. Model development

The model of human metabolism and social diffusion in our ABM builds on the model proposed by Giabbanelli, Alimadad, Dabbaghian, and Finegood (2012), which is based on basic components of energy homeostasis including energy intake, energy expenditure and energy storage. This model has been previously validated using the NLSY dataset (Bureau of Labor Statistics, 2012). It is one of the best models that can be used to simulate the spread of obesity-related behavior change in a networked populations (Li, Zhang, & Pagán, 2016). We begin with an overview of this model and then describe our extensions. In Giabbanelli's model, individuals influence each other with respect to food intake and physical activity. The model also allows for environmental influences. The difference between energy intake (EI) and energy expenditure (EE), defined as energy imbalance (EIB), is used to determine body weight changes over time. A fixed energy density of 32.2 MJ/kg is used for turning energy surplus to body-weight (i.e. gaining 1 kg for each extra 32.2 MJ). Energy expenditure is the sum of physical activity, resting energy expenditure and thermal effect of food metabolism. From this, BMI is calculated assuming height to be fixed. For each individual two parameters are defined: social network influence (SNI) and environment influence (ENV). The value for social network influence is determined by a formula, which is a function of physical activity and energy intake of an agent's friends. A fixed value is used for ENV. These two values are then combined to generate a socio-environmental influence value. If this value is greater than a threshold (fixed for all agents), the agent will change energy intake (EI). Similarly, if this value is greater than another threshold, the agent will update physical activity (PA). No change will happen for values lower than threshold.

We made several additions and modifications to the model as follows. First, our experiments with the model seemed to indicate that single thresholds cause large fluctuations in the amount of energy intake and physical activity and consequently individuals' weight. To resolve this issue, we used a low and a high threshold. If the combined influence is smaller than the low threshold for EI ($T_{EI,low}$), an agent will decrease EI. If the combined influence is larger than the high threshold for EI ($T_{EI,high}$) EI will increase. The same procedure was used for PA. By providing a range for influence not to impact EI and PA, the model stabilizes such that sharp and sudden weight changes are not generated. Fig. 1 shows this process. More technical details including the formulas for agent behaviors are included in supplemental material.

Next, we turned our attention to individuals' variability in thresholds. In the original model, threshold values are fixed across the whole population. In our model, each agent has his/her own threshold values. Similar to the original model, and because these individual-level threshold values are difficult to measure directly, their best values will be determined by "fitting" them to an actual dataset of individual weight change over time (more details are provided in supplemental material). This process is also called model calibration. While the original model assumes a similar environmental influence parameter (ENV) value for the whole population, in our model agents have a distinct ENV that depends on their location. It is set to a lower value in healthier (less obesogenic) environments, and a larger value in a more obesogenic environment. The numerical range for the ENV values was (0.93 to 1.02). $ENV < 1$ represents a healthy environment, $ENV = 1$ a neutral environment (has no effect on individuals), and $ENV > 1$ represents an obesogenic environment. The final change made is on how energy imbalance impacts weight. Instead of using a fixed conversion rate (as in the original model) for mapping energy surplus/deficit to weight change, we used a formula presented by

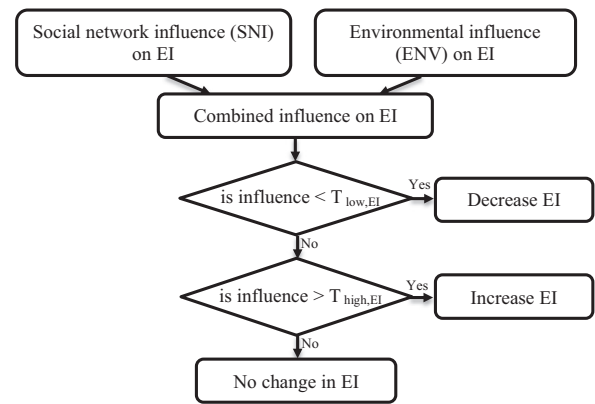


Fig. 1. Depiction of how the model specifies the influence of social networks and environment on agent behavior change. The process of updating energy intake (EI) is shown. A similar process can be imagined for physical activity (PA) by replacing all EIs with PA.

Hall, Heymsfield, Kemnitz, Klein, and Schoeller (2012). According to this formula, for losing 1 kg of weight, an individual with x kg of initial body fat needs to have an energy deficit of $f(x) = 7 \times \ln(x + 1) + 5$ megajoules. This results in more realistic weight change estimates in our model.

2.2. Building social networks among agents

To test the impact of social influence on diffusion of intervention effects, we require a realistic model of social network structure. We use the approach described by Beheshti and Sukthankar (2014) for building network structure. This approach constructs social networks among agents following a power law degree distribution and homophily¹ properties. We borrowed information from existing network data to set the degree of nodes in our network to approximately 12, and the clustering coefficient² (average local) to 0.42. For comparison, the reported clustering coefficient in the social network studied in Framingham Heart Study (Christakis & Fowler, 2007) was 0.66. Reciprocity rate³ was 0.54 for the nodes in our network. This was equal to 0.57 for males and 0.71 for females in data from the Add Health study (Trogon & Allaire, 2014).

2.3. Intervention designs and implementations

We compare five targeting approaches in our experiments, shown in Table 1. The selected targeting approaches consist of random targeting, two conventional targeting approaches and two network-based methods. Similar to previous studies, the number of targeted individuals is the same for all methods (10 percent of the population) (El-Sayed et al., 2013; Sangachin, Samadi, & Cavuoto, 2014; Zhang et al., 2015). These targeting strategies are implemented in the model as follows.

In random targeting, agents are chosen randomly from an appropriate registry or sampling frame. The most common methods involve selecting vulnerable persons (those living in high-risk areas) and high-risk individuals (those who are overweight, sedentary or who have obesogenic diets). In our model, vulnerable individuals live in obesogenic environments such as food deserts or unsafe neighborhoods. The ENV variable, introduced earlier, determines the obesogenicity of the agent's neighborhood. High-risk targeting selects individuals at ran-

¹ Homophily refers to the tendency to be connected to others who are more similar with respect to age, gender and weight status (Hruschka, Brewis, Wutich, & Morin, 2011).

² Clustering coefficient is a measure of the degree to which nodes in a graph tend to cluster together.

³ Reciprocity is a measure of the likelihood of vertices in a directed network to be mutually linked.

Table 1
Comparison of five targeting approaches used in our study.

Strategy:	Random	High-risk	Vulnerable	Network based	Most influential
				Most connected	
Approach to selection:	Choose target individuals at random	Choose target individuals who are at elevated individual risk	Choose target individuals who reside in area designated as high risk	Choose target individuals who have the most social networks connections	Choose targets who will maximize the diffusion of intervention in the network
Example:	Students randomly from a school roster	Overweight adults in a large health plan	Residents of a community identified as a 'food desert'	Adults with the most "facebook friends"	A group of peer leaders plus several others at the edges of clusters
Advantages:	Simple. Does not require risk factor information.	May be optimal if specified intervention works best in high-risk individuals.	May be optimal if specified intervention works best in high-risk environments.	May maximize social multiplier effects through network ties.	
Disadvantages:	Does not leverage social multiplier or diffusion effects; May target individuals with low probability of benefit.	Population impact is counteracted by social isolation of target groups; Can blame the victim.	May be behaviorally inappropriate if environments do not support intervention goals.	Requires direct or indirect knowledge of social network structure in a target group.	

dom based on the agent's BMI assigned at baseline randomly from a realistic distribution.

The first implemented network-based method, the centrality-based approach, chooses agents with the largest number of connections based on network centrality. Individuals with the most connections (edges starting from them to other nodes) in the network (out-degree centrality) are selected. The second network-based strategy is termed "influence maximization", and borrows from a machine learning approach used in other fields like viral marketing and advertisement (Chen, Wang, & Wang, 2010; Morone, & Makse, 2015). This method selects targeted individuals based on a pre-determined optimization goal: given a directed social network and a number k , find k seed nodes (agents) such that activating them (intervention) leads to the maximum expected number of activated nodes, according to a predefined propagation model (Goyal, Lu, & Lakshmanan, 2011; Hajibagheri, Alviri, Hamzeh & Hashemi, 2012; Hajibagheri, Hamzeh, & Sukthankar, 2013). The optimization goal is called the objective function in the literature. In our ABM, the optimization goal is finding a fixed number of nodes in a network that, when selected for intervention, the number of other nodes in the graph that change behavior is maximized. In general, finding the optimum set of initial nodes in a graph is computationally expensive (NP-Hard problem); there is no fixed computational algorithm for finding the optimum nodes in a short time. Different heuristics are employed to find the best near-optimum solution. Kempe et al. (2003) proposed a method using a natural greedy strategy, and proved that it can always find a close-to-optimal solution.

In our model, the influence maximization (IM) targeting approach works in this way: identify one node that if targeted for intervention, maximizes the overall effectiveness of intervention in the population (e.g. result in the lowest number of obese individuals). The second node will be added such that the two nodes will maximize the influence. Additional nodes up to the fixed target number for the intervention is added similarly. In each step, the selected nodes are kept. The IM method does not use the same diffusion mechanism as the main model. A simple linear threshold model (Kempe et al., 2003) is used by the IM method for modeling the diffusions. The threshold value is assigned based on the body-weight of the node. More technical details are provided in supplemental material.

We evaluate the effectiveness of two hypothetical interventions in our experiments: 1) intervention on EI, in which EI of targeted agents is decreased by 15% and 2) intervention on PA, in which a 17% increase is considered for the target agents.

Our ABM was implemented in the NetLogo environment (Wilensky, 1999). Instructions for accessing the source-code are provided in supplemental material. At the beginning of the model run, agents' features were initialized according to the parameters and distributions shown in Table 2. The size of the population was optionally set (equal to the number of samples in NLSY79). Similar results were obtained using larger populations sizes. Values for low and high thresholds for both EI and PA were set to 0.002 and 0.2 respectively. Gender, age, weight and height distributions are assigned based on the data from the year 1986 of National Longitudinal Surveys (NLSY79) dataset (Bureau of Labor Statistics, 2012). This dataset is also used to validate our ABM. These features are used to implement the homophily property of nodes while initializing the network. Threshold values and ENV parameters are calibrated in our model. A set of sensitivity analysis experiments have been performed on the calibrated values. Results of these experiments are available in the supplemental material. In our model, while height remains fixed, weight changes across each time step according to changes in energy intake and physical activity.

The initial population was simulated for two years before applying any intervention. This period was chosen since the NLSY79 dataset is collected in two-year cycles. After this, we used each of five targeting methods to select agents to receive the standard intervention (either for physical activity increase or reduction in dietary intake). The process of

Table 2
Agent-based modeling parameter settings.

Measure	Value
Population size	12686
Gender (female %)	50%
Age (yr)	$21 \leq (24.68 \pm 2.27) \leq 29$
Weight (lb)	$50 \leq (154.55 \pm 56.98) \leq 400$
Height (in)	$40 \leq (67.84 \pm 4.89) \leq 90$
Targeted individuals	10% of total
Simulated length (days)	730
EI intervention effectiveness	15%
PA intervention effectiveness	17%
$T_{EI,low}, T_{PA,low}$	0.002
$T_{EI,high}, T_{PA,high}$	0.2
ENV	[0.93, 1.02]

Footnote:

For age, weight and height, values are shown in the form of $\min \leq (\text{mean} \pm \text{sd}) \leq \text{max}$. The values of four thresholds (T variables) in the model and ENV are calibrated such that realistic patterns of weight change in the population are obtained; sensitivity analysis results are provided as supplemental material.

obtaining EI and PA intervention effectiveness at individual level was described earlier. The objective function that was used for the influence maximization method in our experiments was the minimum number of obese individuals. In other words, the influence maximization method is set to find target individuals that, based on their network ties and a given diffusion model, minimized the obesity prevalence in the population. It should be noted that the objective function could be defined in other ways. For instance, it could be defined such that the number of overweight individuals are minimized, or sum of the number of overweight and obese individuals are minimized. These additional cases are reported in supplemental material. The model is run for an additional two years after intervention roll-out, and the population-wide results are recorded. The results shown in the following section are the average of 100 independent runs of the model for each of 5 targeting experiments. A larger number of runs did not produce different results.

2.4. Model validation

We use the NLSY79 dataset for validating our model (Bureau of Labor Statistics, 2012). The purpose is to evaluate whether the simulated weight changes (due to social and environmental factors) that our model generates are realistic given historical trends observed in the real world. This dataset is a nationally representative sample of 12,686 individuals in the US who have been surveyed starting in 1979. For the purpose of validation, we used biennial changes in weight for the years 1986 to 2012 from this dataset.

3. Results

The results of model validation is shown in Fig. 2. The reported results relate to two years of running our model without any intervention. The mean and standard deviation for the average weight change is equal to 1.8 and 4.0 (pounds) in the NLSY dataset, and 1.5 and 6.6 for our model.

Next, we compared the performance of five targeting methods described earlier, as shown in Figs. 3 and 4 below. Fig. 3 plots change in average agent weight over two years after a behavioral intervention is delivered to reduce EI. As specified by the model, EI is reduced by 15% in agents assigned to the intervention (on average). The figure shows the combined population impact on average body weight, taking account of both diffusion and environmental effects. A dashed line shows the average weight change of the population, had the simulation continued without any intervention. This shows a slight increase in average body mass consistent with population trends, and represents

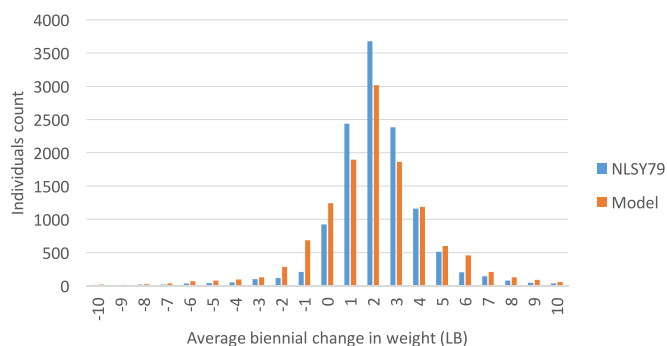


Fig. 2. Comparison between the average biennial change over weight in NLSY79 dataset (blue bars) and our model that was used for the simulation of weight changes (orange bars). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

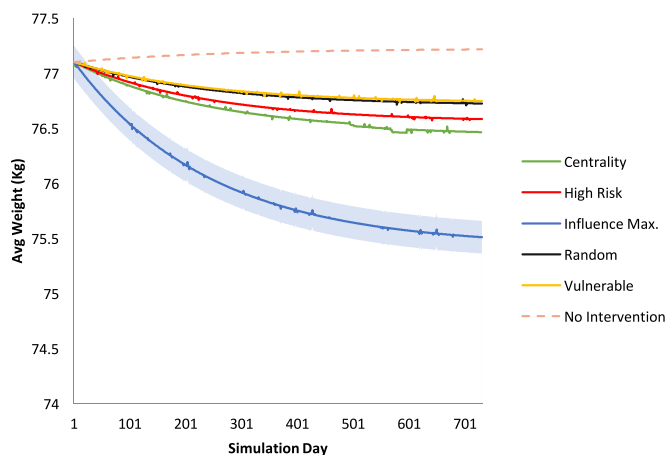


Fig. 3. Simulation results for 5 targeting scenarios after implementation of intervention to reduce dietary intake in 10% of the population. Average weight across the simulated population after applying intervention as obtained by five different targeting, and baseline scenario (no intervention) approaches are shown. Confidence intervals for the influence maximization method are shown using light blue color. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

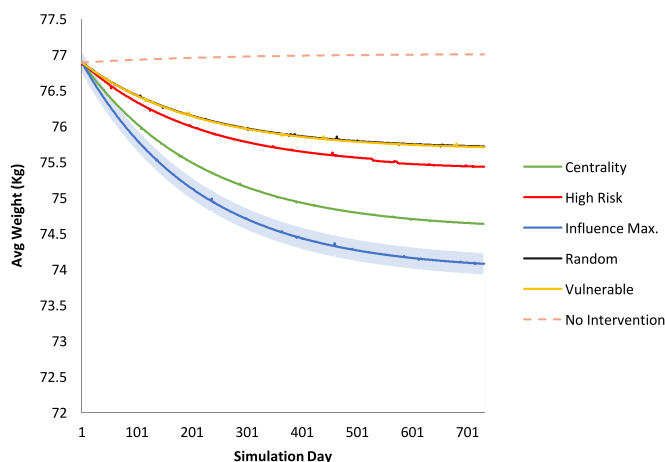


Fig. 4. Simulation results for 5 targeting scenarios after implementation of intervention to increase physical activity in 10% of the population. Average weight across the simulated population after applying intervention as obtained by five different targeting, and baseline scenario (no intervention) approaches are shown. Confidence intervals for the influence maximization method are shown using light blue color. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

the control condition or the causal scenario of no intervention against which the 5 targeting methods can be compared.

Across all five targeting scenarios, the range of change in population average weight was between -0.35 and -1.60 kg (with an average loss of -0.69 kg). Random targeting and vulnerable (agents residing in a more obesogenic area) showed the least overall impact (change in mean weight at 2 years of -0.49 kg and -0.47 kg compared to no intervention (CTNI for short). Targeting high-risk agents (obese) and those with more network connections resulted in more weight loss (-0.63 and -0.75 CTNI respectively). Results showed that selecting subjects on the basis of our IM model resulted in the most average weight loss (-1.7 kg CTNI). Confidence intervals (CIs) of vulnerable and random targeting were overlapping, as well as CIs of high-risk and centrality methods. CIs of these two groups were separate, as well as CI of the IM method (shown in the chart) and others. For energy intake, the rate of aggregate weight loss was similar over time with evidence of convergence to a steady state by day 500. The rate of average weight loss was steeper early in the post-intervention period in the IM targeting scenario.

Fig. 4 shows results over two years after implementation of a physical activity intervention in 10% of the population. The model dictates a 17% increase in physical activity in agents chosen for the intervention. On average, the average decline in body weight was -1.77 kg across the five scenarios ranging from -1.17 (Random) to -2.79 (IM). After physical activity intervention, the random, vulnerable and high risk targeting strategies performed similarly. Targeting based on network centrality yielded a 25% better average decline in weight. Again, the best performing targeting approach was IM, which generated the largest population-wide impact (-2.9 kg CTNI), which was 57 percent more than average. In these results, vulnerable and random methods had overlapping CIs, while other CIs were separate. In addition to five targeting methods discussed here, four other network-based targeting methods are presented in the supplemental material. These four methods, include three different ways of measuring the centrality of nodes (degree centrality was discussed here), and a cluster based targeting method.

We also studied the changes in the prevalence of overweight ($25 \leq \text{BMI} < 29.9$), and obese individuals ($\text{BMI} \geq 30$) in the population. Tables 3 and 4 show the performance of various targeting methods on changing these prevalences. When the targeted agents are receiving an EI intervention, network centrality and high-risk methods yield lower percentages of obese individuals in the population (with 28.9% and 28.3%) than random and vulnerable methods (with 29% for both). Using IM targeting, the number of individuals with obesity drops to 26.6%. Similar patterns were observed for the physical activity intervention. In this case, the IM method yielded the greatest reduction in obesity prevalence (-4.8% for EI and -6% for PA CTNI).

4. Discussion

Harnessing information on the social characteristics of individuals

Table 3

Population prevalence of overweight and obesity after intervention on EI by targeting method.

Method	% Overweight mean \pm SD	% Obese mean \pm SD
Beginning state	33.24 \pm 2.3	31.22 \pm 2.3
Centrality	32.8 \pm 2.1	28.92 \pm 2.08
High risk	32.82 \pm 1.96	28.34 \pm 2.12
Influence max.	33.68 \pm 2.3	26.62 \pm 2.44
Random	33.06 \pm 1.88	29.38 \pm 2.16
Vulnerable	33.04 \pm 2.08	29.4 \pm 2.1
No intervention	33.26 \pm 2.3	31.44 \pm 2.3

Footnote:

Beginning state shows to the initial percentages of the population.

Table 4

Population prevalence of overweight and obesity after intervention on PA by targeting method.

Targeting method:	% Overweight mean \pm SD	% Obese mean \pm SD
Beginning State	33.24 \pm 2.3	31.22 \pm 2.3
Centrality	32.56 \pm 2.58	27.8 \pm 2.32
High Risk	32.62 \pm 1.94	27 \pm 2.3
Influence Max.	33.96 \pm 2.76	25.44 \pm 2.64
Random	32.48 \pm 2.2	28.58 \pm 2.2
Vulnerable	32.64 \pm 2.18	28.56 \pm 2.24
No Intervention	33.26 \pm 2.3	31.44 \pm 2.3

in designing interventions may lead to greater population-level impact (Bahr et al., 2009). However, the impact of harnessing social influence as a basis for intervention targeting is difficult or impossible to estimate using traditional methods. Previous studies about the potential advantages of using social network structure for targeting have been inconclusive. This becomes more important when we consider the relative cost and difficulty of gathering social network information in real human populations. Few interventions have been designed and implemented by analyzing the network structure of the population of interest. The purpose of this paper was to develop and evaluate an agent-based model (ABM) to evaluate performance of 5 possible intervention targeting regimes. Computational methods provide powerful tools to study the effectiveness of alternative targeting strategies by conducting experiments in a so-called *in silico* environment. Our main finding is that subject to the limits and assumptions of our model, we find evidence that using network information to inform targeting outperforms more standard targeting approaches including random selection, or selecting high-risk individuals, or vulnerable contexts.

Our simulations showed that targeting individuals based on their network position leads to greater population effectiveness in obesity interventions, holding the efficacy of the intervention for an individual constant. This is consistent with some existing findings (Bahr et al., 2009; Hammond & Ornstein, 2014; Sangachin et al., 2014; Trogdon & Allaire, 2014). However, these results conflict with other studies. El-Sayed et al. (2013) and Zhang et al. (2015) found that network-based obesity interventions have little or no added value compared with at-random interventions. El-Sayed et al. (2013) assumed that risk of obesity if an individual's contact becomes obese is 1.6 times higher. Sangachin et al. (2014) used a linear threshold model to implement diffusion of obesity. Differences in the choice of diffusion model and key model parameter decisions might explain these inconsistencies. In this study, the usage of a threshold model for simulating obesity-related behavior and its diffusion is based on the model proposed by Giabbanelli et al. (2012). We believe we have improved their approach by replacing a single fixed population threshold with threshold values drawn from a distribution. In addition, we performed sensitivity analysis over the range of threshold values to demonstrate that results are consistently plausible, and not extreme. Our final results are not strongly sensitive to these threshold values. The results of these experiments are reported in supplemental materials.

Most existing work, including studies by Sayed and Zhang, used some variation of degree centrality to evaluate network-based interventions. As Valente (2010) points out, a major limitation of in-degree centrality is that nominations received may be redundant. Borgatti (2006) showed that conventional measures of centrality are inadequate for finding nodes optimally positioned to spread information for diffusion or to be removed from a network to disrupt spread. We address this by using another alternative to degree centrality finding that utilization of social network structures using other approaches can lead to higher population impacts.

This work has several strengths. First, we used a previously published and independently tested model of social network diffusion

of obesity-related behavior and BMI change developed by a team with long experience. Secondly, we calibrated our model by evaluating whether a 2 year run yielded average weight change that is consistent with actual data based on the NLSY study. Third, this is the first study to combine a realistic physiologic model optimized to examine network diffusion, with the ability to examine the role of environmental input, and a realistic and sophisticated method of generating and evaluating social network structure among agents.

One additional strength deserves special comment. We are among the first to incorporate a well-established machine learning method called influence maximization to widen the range of network-based strategies. Influence maximization techniques try to find an optimal number of structural nodes, which, if activated, would cause the spread of intervention to the whole network, or, if immunized, would prevent the diffusion of a large scale epidemic (Morone & Makse, 2015). The idea of influence maximization was initially introduced in the context of viral marketing (Richardson & Domingos, 2002), and was used in other fields like applied physics (Altarelli, Braunstein, Dall'Asta, Wakeling, & Zecchina, 2014). However, it has not received much attention in obesity research. Sangachin et al. (2014) presented a model for the spread of obesity interventions in a networked population, and compared their method against a greedy-based approach that follows an influence maximization strategy. For the influence maximization method, no more data other than the network structure is needed in practice. The need to know the structure of the existing social network is common among all network-based targeting methods, including centrality-based methods.

Network-based approaches select intervention subjects based on their position in a structure of network connections (e.g., friends, classmates, club memberships, teams, organizations, schools, neighborhoods, etc.). This, as we have shown, may target those most likely to transmit the intervention to more people (diffusion of intervention). The disadvantage is that it requires prior knowledge of how potentially enrolled subjects are connected. This type of data can be expensive and time consuming to acquire. Fully dimensional social network data are among the most challenging to collect. In practice, it might be only feasible to ask people to indicate their number of friends, or name some of their close coworkers or relatives. Even by this limited data, a semi-synthetic network structure for a population of interest can be generated and used to determine individuals with highest promise for propagating the intended intervention. Following power law distribution, common values for clustering coefficients and knowledge of patterns of homophily are common features of human networks. These can be used to generate realistic network structures. Moreover, recent technologies like cellular or social media networks have provided us with new tools to capture and study human social networks. These might help policy makers or intervention designers to access social network structure for diverse populations of interest. One way to extend our work is to study the performance of different targeting methods under conditions of incomplete knowledge of the network structure.

We are limited in our ability to generate agent networks with full fidelity to reality given that our agents are “sampled” independently. Therefore, the degree of clustering and homophily is likely to be less than the real world in which human networks operate. We generated networks that match the real world only in the macro-sense of total numbers of ties. The implication of this is that our ability to discern the comparative advantages of network based targeting are likely to be an under-estimate of the true marginal utility of network-based approaches. Moreover, the NLSY79 dataset was chosen to test our experiments on a realistic population. We acknowledge that using real-world distributions of body-weights over time for model validation (as in Fig. 2) is not ideal and does not indicate that our model is able to provide insights into the causal mechanisms generating these changes. It is possible that we are able to recreate overall BMI distribution changes but have the underlying mechanisms wrong. However, given

the absence of longitudinal data on weight trajectories and social network histories, this approach at least allows us to identify a badly performing model. In supplemental materials, we show that consistent results are obtained when we perform our experiments on simulated populations based on the HERITAGE family study dataset (Jackson, Stanforth, Gagnon, Rankinen, & Leon, 2002). Lastly, it is noted that the question of whether there is diffusion in obesity related behaviors is a matter of some controversy. Our results are premised on the idea that there are network diffusion processes at play.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.ssmph.2017.01.006>.

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