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Are Network-Based Interventions a Useful Antiobesity Strategy? An Application of Simulation Models for Causal Inference in Epidemiology

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Recent research suggests that social networks may present an avenue for intervention against obesity. By using a simulation model in which artificial individuals were nested in a social network, we assessed whether interventions targeting highly networked individuals could help reduce population obesity. We compared the effects of targeting antiobesity interventions at the most connected individuals in a network with those targeting individuals at random. We tested 2 interventions, the first "preventing" obesity among 10% of the population at simulation outset and the second "treating" obesity among 10% of the obese population yearly, each in 2 separate simulations. One simulation featured a literature-based parameter for the network spread of obesity, and the other featured an artificially high parameter. Interventions that targeted highly networked individuals did not outperform at-random interventions in simulations featuring the literature-based parameter. However, in simulations featuring the artificially high parameter, the targeted prevention intervention outperformed the at-random intervention, whereas the treatment intervention implemented at random outperformed the targeted treatment intervention. Results were qualitatively similar across network topologies and intervention scales. Although descriptive studies suggest that social networks influence the spread of obesity, policies targeting well-connected individuals in social networks may not improve obesity reduction. We highlight and discuss the potential applications of counterfactual simulations in epidemiology.

computer simulation; intervention; obesity; public health; social networks

Abbreviation: ABC, agent-based counterfactual.

The rates of overweight and obesity have increased dramatically in high-income countries during the past 30 years (1–4). Obesity is an important determinant of chronic disease morbidity and mortality (5–7) and is associated with the risk of hypertension, hypercholesterolemia, coronary heart disease (5), stroke (8), and diabetes mellitus (9).

Compounding the concern about the broader implications of rising obesity rates has been uncertainty about interventions that might mitigate the epidemic. Despite substantial investment in behavioral and pharmacotherapeutic interventions to reduce obesity (10–12), the efficacy of many of these interventions has been questionable (13, 14). For this reason, a recent study suggesting that social interactions may influence the spread of obesity within a densely interconnected social network (15) has been greeted with enthusiasm (16). Identifying the influences of social networks on obesity may well suggest a potential novel approach to intervention, targeting key network influences to reduce overall population obesity risk.

Beyond concerns about the appropriate methodology for measuring network effects longitudinally (17, 18), there remains a substantial inferential leap from observational data suggesting that there are social network influences on the risk of obesity over time to confidence that manipulating social networks can substantially *reduce* obesity (19–21). There has been a growing call for randomized social interventions in population health research to address this challenge (22, 23). However, randomization is not without challenges, including resource constraints, ethical issues, the physical impossibility of randomization to certain exposures,

and long latency periods between social exposure and the onset of symptoms (21).

Computerized simulation approaches, such as agent-based models, are of growing interest in population health research (24, 25). Agent-based models are computerized simulation models that can be used to simulate individuals nested in simulated environments over simulated time. The simulated individuals behave according to programmed rules that define baseline characteristics, locations in space, and interactions with their environments and with one another. These simulated individuals are dynamic and adaptive over time, autonomous from one another, and heterogeneous with regard to baseline characteristics. Moreover, they can be nested within networks that can simulate diverse motifs of human interaction, such as households, families, social networks, neighborhoods, and communities. We propose the use of agent-based counterfactual (ABC) simulations, simulations of counterfactual universes that use artificial computerized models, to allow for discrete in silico "policy experiments" from which researchers can infer the influences of perturbations within particular exposure parameters on outcomes of interest within the simulation.

Although this approach is only in its early stages, there are already fruitful examples of the use of ABC simulations in epidemiologic inquiry. For example, 2 recent studies used similar computerized simulations to characterize the mechanisms underlying social disparities in walking behavior and food choices, respectively. Yang et al. (26) used an agent-based model in which walking choices were influenced by demographic and spatial characteristics as well as distances to different activities, walking ability, and attitudes toward walking to simulate walking behaviors within a city. By comparing walking behavior across 4 counterfactual simulations, each with different levels of safety and walkable land use, the authors demonstrated that differences in these factors in more deprived compared with less deprived neighborhoods might explain socioeconomic disparities in walking behavior. Another study (27) used an agent-based model to understand socioeconomic disparities in healthy food consumption. In this model, aggregate household food preferences in a neighborhood, which are products of household socioeconomic status, predicted the availability of stores offering healthy food in that neighborhood. The authors demonstrated that with socioeconomic segregation, stores offering healthy food became less prevalent in low-income neighborhoods. Furthermore, through a series of simulations, they demonstrated that both increasing healthy food preferences in low-income households and decreasing the price of healthy foods were necessary to improve availability of healthy food in low-income settings.

In the present analysis, we had 2 aims. First, we used an ABC simulation model to explore the implications of interventions targeting social networks to reduce obesity. To do this, we simulated the relative efficacy of interventions that target the most highly connected individuals in a social network relative to those targeting individuals at random. Second, we illustrated the potential of ABC simulations as a powerful tool in etiological inference building on observational analyses in settings where randomized social interventions are impractical.

MATERIALS AND METHODS

Model

We used data from the Health Surveys for England in 1999 and 2004 as well as data about the relative risk of obesity among those with obese contacts compared with those without obese contacts from Christakis and Fowler (15) to design an agent-based social network model of obesity in England among a simulated birth cohort born in 1981.

Initial conditions for the baseline model were as follows: Each agent was stochastically assigned gender, ethnicity, social class, and educational level, adherent to distributions of each characteristic in England, such that the proportion of Asian, black, and white agents, for example, mimicked that of the English population overall. Each agent was nested within 1 of 6 spatial contexts, representing different ethnic and social class compositions, and was placed in a context preferentially by demographic characteristics (ethnicity and social class). A proportion of the population was assigned obese status at the model outset by demographic and neighborhood allocation similar to the population 18 years of age who are obese in England. Each agent represented an individual 18 years of age at the model outset, aging by 1 year for each time step, each agent's risk for developing obesity in that time step was calculated as a function of gender, ethnicity, social class, education, and social contacts and was implemented (Web Appendix available at http://aje.oxfordjournals.org/).

To model the effects of social networks on the spread of obesity, agents were also nested within a segregated social network that was generated by using a biased preferential attachment growth model to create a scale-free (Barabási-Albert model) (28) social network with assortative mixing. The network was initialized from a seed network composed of a small number of agents. Each new agent added to the network was connected to up to 4 existing agents with a probability of connecting to an existing agent that was proportional to the number of connections that that existing agent already had. Moreover, an additional bias was included to preferentially connect agents with like characteristics. Whereas 25% of new agents to the network were connected without regard to their characteristics (i.e., ethnicity, social class, education), 50% of new agents to the network were restricted to connecting with existing agents of similar ethnicity, again with a probability of connecting to existing agents with the same ethnicity that was proportional to the number of connections that that agent already had. Twenty-five percent of new agents added to the network were restricted to connecting with existing agents of similar ethnicity and social class, again with a probability of connecting to an existing agent with the same ethnicity and social class that was proportional to the number of connections that agent already had.

For contacts of obese nodes, we assigned a higher probability of becoming obese in each time step on the basis of findings from Christakis and Fowler (15), such that if an agent's contact became obese in a previous time step, that agent had a 1.16 times higher risk of developing obesity in

the current time step. For more detailed information about the construction of the model and its limitations, see the Web Appendix.

Simulations

We tested 2 interventions. The first was a prevention intervention that rendered a proportion of the population incapable of becoming obese throughout the simulation. It was administered first among 10% of the population at random and then among the most well-connected 10% of the population. The second intervention featured a treatment for obesity, which returned a proportion of the entire obese population back to normal body mass index each year. It was implemented among 10% of the obese population each year, first at random, and then among the most well-connected 10% of the population. To understand the influence of the strength of the network effect of obesity on our findings, we tested each of these interventions both on the baseline model and by using an altered model in which the parameter indicating the relative risk of developing obesity if a network contact became obese in the previous time step was increased from 1.16 (the Christakis and Fowler parameter) to 10.

We ran further simulations to ensure the robustness of our findings to the population scale of each intervention as well as to differences in network topology as discussed above. To account for the potential for different outcomes by intervention scale, we simulated each of the interventions applied to 30% of the population as well. To account for potential sensitivity to network topology, we replicated each of our simulations by using a segregated Erdős-Rényi model and a clustered network (construction described in detail in the Web Appendix).

To further characterize the mechanism underlying our findings regarding the effects of targeting our treatment intervention, we ran 2 further simulations. By using the scale-free network model with an artificially high network communicability parameter of 10 (the relative risk of developing obesity if a network contact became obese in the previous time step), we implemented a permanent treatment intervention whereby in each time step, 10% of the obese population reverted back to normal weight and was made incapable of developing obesity in future time steps. This intervention was implemented both at random and by targeting the most well-connected individuals in the population.

All intervention simulations were compared with a control simulation (with the same network effect parameter and network topology) without any intervention. All results were subject to Monte Carlo simulation with 100 iterations.

RESULTS

Among the most well-connected 10% of individuals in the scale-free social network, the mean number of contacts was 25.4 per agent, and the median number of contacts was 17 (not shown). Among the remaining agents in the model, the mean number of contacts was 6.1 per agent, and the median number of contacts was 5 (not shown).

Figure 1 shows obesity prevalence by age among 10,000 agents representing a cohort born in 1981 in England in a simulation without intervention (baseline), simulating an intervention that prevented 10% of the population from becoming obese at random, and simulating an intervention that prevented the most well-connected 10% of the population from becoming obese. There was almost no difference in the prevalence of obesity throughout the life course between simulations that included interventions and the baseline simulation. Similarly, there was no difference in the prevalence of obesity between the simulation featuring the intervention implemented among the most well-connected individuals and that featuring the intervention implemented at random. Similarly, Figure 2 shows differences in obesity prevalence in a simulation without intervention (baseline), in a simulation of

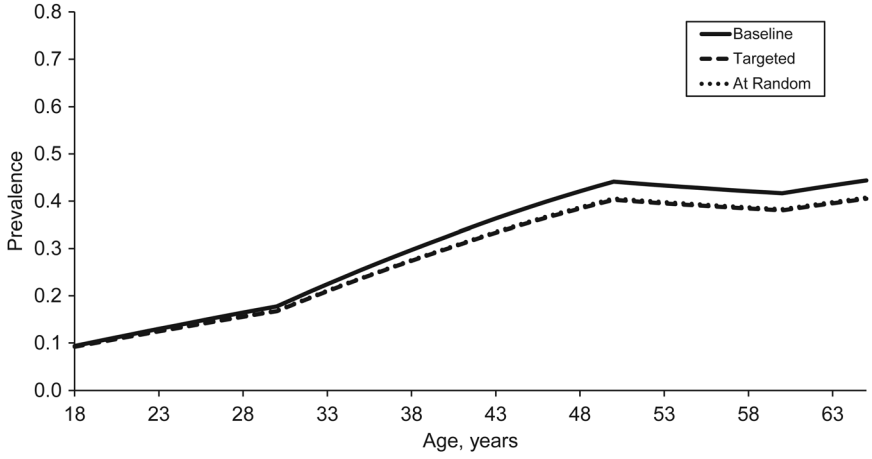

Figure 1. Obesity prevalence by age among 10,000 agents representing a cohort born in 1981 in England with no intervention (solid line), simulating an intervention that prevented 10% of the population from becoming obese at random (dashed line), and simulating an intervention that prevented the most well-connected 10% of the population from becoming obese (dotted line). Note that the dashed and dotted lines are overlaid on one another. Obesity was defined as a body mass index (weight (kg)/height $(m)^2$) >30.

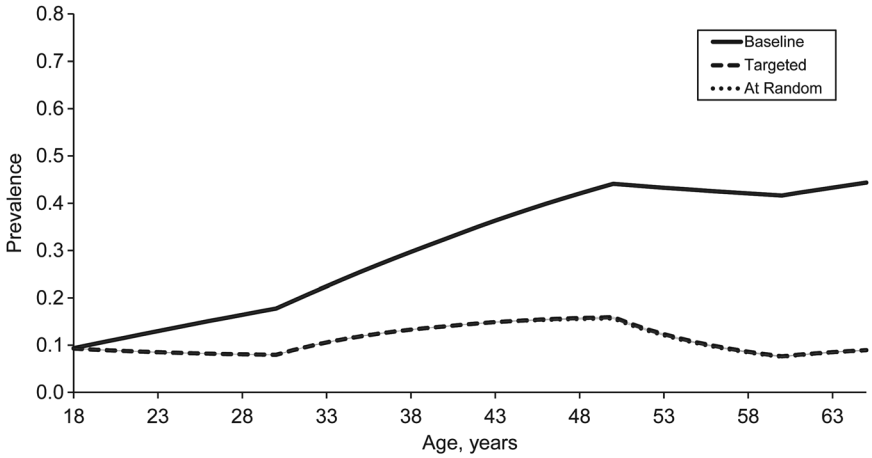

Figure 2. Obesity prevalence by age among 10,000 agents representing a cohort born in 1981 in England with no intervention (solid line), simulating an intervention that treated obesity among the most well-connected 10% of the obese population each year (dashed line), and simulating an intervention that treated obesity among 10% of the obese population at random each year (dotted line). Note that the dashed and dotted lines are overlaid on one another. Obesity was defined as a body mass index (weight (kg)/height $(m)^2$) >30.

an intervention that treated obesity among 10% of the obese population each year at random, and in a simulation of an intervention that treated obesity among the most well-connected 10% of the obese population each year, with similar findings. Although both intervention simulations showed lower prevalence of obesity throughout the life course than the baseline simulation, the targeted intervention did not outperform the intervention implemented at random in reducing obesity prevalence.

Figures 3 and 4 are analogous to the previous 2 figures, although demonstrating the results of simulations run in the model with the artificially high network effect on obesity risk. Figure 3 shows the results of preventive interventions implemented both among the most well-connected individuals and at random relative to baseline. The lowest prevalence of obesity occurred in the simulation with the intervention targeting the most well-connected obese individuals, followed by the simulation featuring the at-random intervention, and then the baseline simulation with no intervention. By contrast, Figure 4, which shows the results of the treatment intervention implemented both among the most well-connected individuals and at random relative to baseline, demonstrates the lowest prevalence of obesity in the simulation featuring the intervention implemented at random,

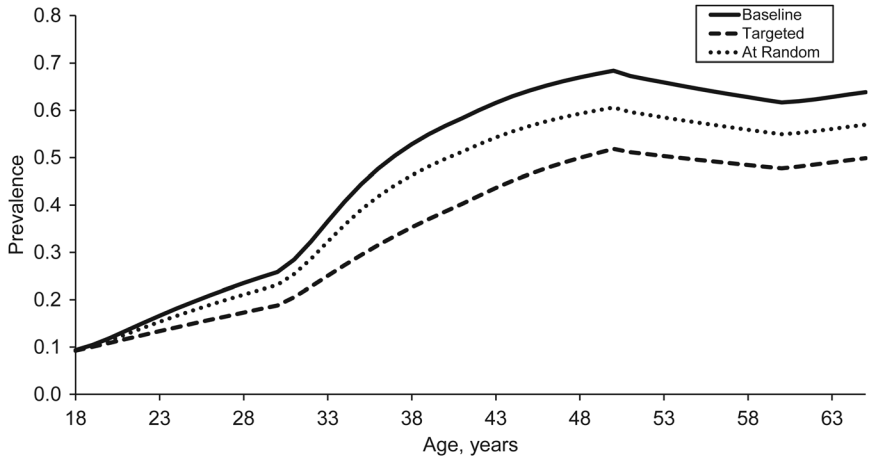

Figure 3. Obesity prevalence by age among 10,000 agents representing a cohort born in 1981 in England in simulations with artificially high network effects on obesity risk with no intervention (solid line), simulating an intervention that prevented the most well-connected 10% of the population from becoming obese (dashed line), and simulating an intervention that prevented 10% of the population from becoming obese at random (dotted line). Throughout these simulations, if a network contact became obese, an agent's relative risk of becoming obese increased to 10 compared with 1.16 in the baseline model. Obesity was defined as a body mass index (weight (kg)/height $(m)^2$) >30.

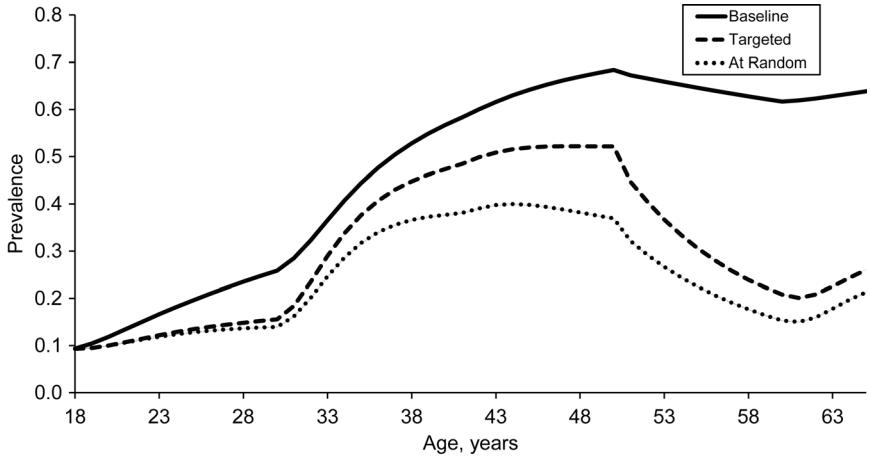

Figure 4. Obesity prevalence by age among 10,000 agents representing a cohort born in 1981 in England in simulations with artificially high network effects on obesity risk with no intervention (solid line), simulating an intervention that treated obesity among the most well-connected 10% of the obese population each year (dashed line), and simulating an intervention that treated obesity among 10% of the obese population at random each year (dotted line). Throughout these simulations, if a network contact became obese, an agent's relative risk of becoming obese increased to 10 compared with 1.16 in the baseline model. Obesity was defined as a body mass index (weight (kg)/height $(m)^2$) >30.

followed by the simulation featuring the targeted intervention, and then the baseline simulation. Results in Figures 1–4 were replicated across network topologies and intervention scales with no qualitative differences in findings.

Figure 5, analogous to Figure 4, shows the results of a permanent treatment intervention wherein treated individuals were made incapable of becoming obese again in future time steps. Relative to Figure 4, which shows the results of treatment interventions that imposed no restrictions on individuals' future risks of developing obesity, Figure 5 demonstrates that eliminating future risk of obesity eliminated nearly the entire gap in efficacy between the intervention implemented at random and that targeting the most well-connected individuals in the population.

DISCUSSION

In this ABC simulation of the progression of obesity through a densely interconnected social network among a simulated population of 10,000 individuals representing an English cohort born in 1981 between ages 18 and 65 years, we found no difference in the progression of obesity when either preventive or treatment interventions were applied differentially to well-connected individuals in a network or randomly across the population.

Our study adds to a small but growing literature that has simulated obesity interventions predicated on epidemiologic observations about the spread of obesity through social networks. Bahr et al. (29) used social networks to test hypotheses with regard to effective interventions against the obesity epidemic. By using the body mass index (calculated as weight (kg)/height $(m)^2$) distribution from US data in 2000 to initialize their network, as well as the basic rule that the likelihood of progression between classes of body mass index (underweight, appropriate weight, overweight, or obese) was a function of the body mass index class of the majority of an agent's contacts, they found stable results across network topologies (e.g., lattice, random, small-world, or scale free). They concluded that weight loss among friends of friends was more important than weight loss among friends alone in affecting the weight loss of an index individual, that pinning the body mass index of random individuals in the network could promote a more healthy body mass index distribution, and that interventions among well-connected individuals would be more effective than interventions among individuals at random.

Our findings contrast with those of Bahr et al. (29). We found that keeping constant the body mass index of 10% of the population chosen at random did not considerably decrease obesity prevalence. Differences in these outcomes may be explained by differences in our work from that of Bahr et al. more broadly. First, Bahr et al. modeled a majority rule, whereby the probability of an individual becoming obese in each time step was a function of the obesity state of the majority of the individual's contacts. Rather, we modeled each node's probability of becoming obese in the next time step as a function of obesity among 1 or more of its contacts, more akin to the mechanism of spread demonstrated by Christakis and Fowler (15). Second, our analysis was nested within a broader agent-based model that accounted for heterogeneity in agent characteristics and the social segregation of social networks. Third, our model was parameterized directly from data about a real population. The model implemented by Bahr et al., by contrast, was not parameterized from data from a real population and did not account for heterogeneity in either agent characteristics or spatial context.

Our observations suggest that even if observational findings (15) demonstrating increased risk of obesity among individuals with obese contacts are valid, targeting influential

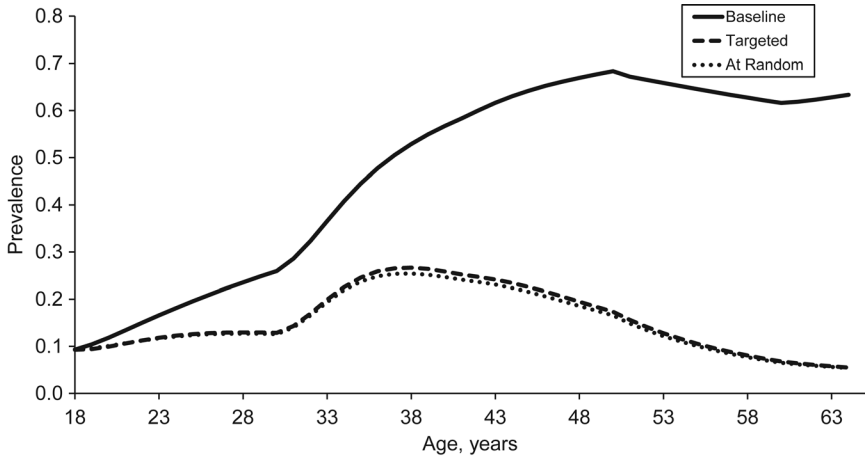

Figure 5. Obesity prevalence by age among 10,000 agents representing a cohort born in 1981 in England in simulations with artificially high network effects on obesity risk with no intervention (solid line), simulating an intervention that permanently treated obesity among the most well-connected 10% of the obese population each year (dashed line), and simulating an intervention that permanently treated obesity among 10% of the obese population at random each year (dotted line). Throughout this simulation, following treatment, agents were no longer capable of developing obesity at any further point in time. Moreover, if a network contact became obese, an agent's relative risk of becoming obese increased to 10 compared with 1.16 in the baseline model. Obesity was defined as a body mass index (weight (kg)/height $(m)^2$) >30.

individuals is not likely to translate to an effective population-based intervention strategy. Comparison of outcomes from simulations carried out with a literature-based network influence parameter with those with artificially high network influences on obesity risk suggested that, with respect to other influences operating in the complex etiology of obesity, network influences on obesity risk are simply not of sufficient strength to warrant network targeting of antiobesity interventions.

We found, perhaps counterintuitively, that in simulations with artificially high network effect parameters, the treatment intervention implemented at random was more effective than the same intervention targeting the most well-connected individuals. This observation, however, is quite plausible. The most well-connected obese individuals are most likely, by definition, to have social contacts with individuals in the network. They are therefore most likely to both "expose" others to obesity and to be "exposed" to obesity via the network effect. Interventions aimed at preventing obesity among the most well-connected individuals are, therefore, likely to be more effective than those among individuals at random because targeting the intervention is the most effective way to limit overall exposure within the network. However, in a treatment intervention, even if well-connected individuals are "cured" at a given time-step, they are more likely than others to be further exposed because of the number of contacts they share and, therefore, to contract the condition again following the treatment. This rebound effect may explain the finding here. Supporting this explanation are our findings (Figure 5) demonstrating little difference in the outcomes of treatment interventions implemented at random compared with targeting the most well-connected individuals when these interventions were made permanent, blocking any potential for rebound because treated individuals were no longer vulnerable.

The costs and logistical difficulties associated with randomized social interventions testing the effects of targeting antiobesity interventions among the most well-connected individuals in a social network would likely preclude the design and implementation of such studies that could test the hypothesis that our ABC simulations tested here. Moreover, aside from the costs and logistical difficulties, the influence of social exposure on obesity risk may occur over several years (30–32), suggesting that adequate follow-up in such a randomized intervention would require several decades. Therefore, the use of ABC simulations allowed us to draw important inferences about the causal relationship between targeting the most well-connected individuals and mitigating obesity prevalence over time, above and beyond observational data, in the setting of limited opportunities to assess this relationship experimentally with a randomized social intervention. Kaufman et al. (21) suggest that a key conceptual contribution of randomized social interventions to social epidemiology is that the experimental conceptual paradigm of randomized interventions forces investigators to explicitly define interventions of interest in study design—formulating a cogent articulation of the counterfactual to be considered and, therefore, the causal relationship to be tested. Similarly, the construction of the ABC simulations featured here also forces investigators to explicitly define their interventions of interest prior to simulation.

A central limitation to randomized social interventions is that contexts may predict outcomes in randomized trials by influencing compliance and participation rates (33). Participation and compliance rates particular to a context where a randomized social intervention study may be situated may limit the generalizability of findings in other contexts where participation and compliance rates may differ. ABC simulations are not as vulnerable to the same limitation because they allow investigators to program differential participation and

compliance rates into repeated simulations and, therefore, to assess the impact of participation and compliance on outcomes of interest. In this way, ABC simulations allow for testing of the potential effects of specific hypothetical policies across contexts. Similarly, the transmissibility of social exposures may impair the assumption of unit independence among study participants in randomized social interventions via the well-documented "spill over" effect (21); however, ABC simulations allow investigators to model repeated simulations, controlling for the effects of exposure transmissibility and thereby facilitating an improved understanding of the effects of these externalities on outcomes of interest among individuals and within the population.

That said, the use of ABC simulations is not without limitations. Among them is balancing mechanistic rigor with model parsimony during model construction. In this regard, although building more detail into models may yield a more faithful representation of our mechanistic understanding of the phenomena under study, this may overcomplicate model interpretation and sacrifice predictive power. Along those lines, however, there is a need for caution, more generally, when interpreting model output quantitatively. Given the high number of variables, often parameterized from disparate sources, that may be included in agent-based models, qualitative inference may be more appropriate (24, 34). Also, as discussed elsewhere (35), validating agent-based models is fraught with conceptual challenges because investigators have essentially only 2 avenues by which they can situate their models in reality: They can parameterize their models with real data, or they can build their models from hypothesized conceptual relationships and then compare their output with real-world observations (35). In the former circumstance, only the relationships between factors in the model on the front end can be validated, whereas in the latter, only the outcomes of the model can be validated—but rarely can both be evaluated simultaneously (35). Finally, agent-based models are computationally intensive, which can pose practical limitations to the scope and complexity of models investigators might build (35). Although randomized interventions remain the "gold standard" in causal inference regarding the health effects of social exposures, ABC simulations do present a viable alternative in settings where such intervention studies are implausible or impractical.

When considering the findings of the present research, the reader should consider several limitations. First, it is plausible that there are other interventions that could manipulate network dynamics to mitigate obesity spread that were not tested here and that would yield different results. However, our analysis was not intended to assess the hypothetical outcomes of all plausible policies involving social networks on obesity outcomes, but only the specific policy of targeting well-connected individuals we outlined here. Second, our agent-based model was parameterized by using data from England. Therefore, our findings may not generalize to other contexts globally. However, given several parallels between the obesity epidemic in England and that in other high-income countries, we feel our findings should be taken into account when considering the hypothetical outcomes of network-based approaches in other high-income contexts. Third, there are several limitations to our model about which the reader should be aware. The model allowed for neither social mobility nor residential mobility among agents. Moreover, we used serial cross-sectional data to parameterize the model, which imposed several limitations on the findings. Unfortunately, these limitations were imposed on our model because of lack of available data for parameterization, as we discuss in the Web Appendix. Fourth, validation of the model was limited to comparing life course trajectories in obesity prevalence projected by our model with those from real data. However, given that the model was parameterized from population data, this is less concerning compared with other similar models in the literature (29). Fifth, although we simulated the effects of 2 potential antiobesity interventions, prevention and treatment, our findings should not be used to compare the outcomes of preventive and treatment approaches head-to-head. These interventions were not constructed for this purpose; rather they were designed to draw out the role of network targeting in antiobesity interventions. Cost-benefit analyses account for the costs of proposed interventions when comparing these interventions head-to-head, and it is likely that in reality, our treatment intervention would be substantially more expensive than our prevention approach, and therefore comparisons across these interventions, without accounting for cost, are inappropriate. Sixth, because serial cross-sectional data were used for the parameterization of our model, producing estimates of variance faithful to the parameter estimates upon which the model was constructed was beyond its scope. However, all mean estimates of obesity prevalence were averaged over 100 Monte Carlo runs to correct for artifacts resulting from the stochastic implementation of the model.

Despite these limitations, our findings have important implications for policy and future research. With regard to the policy implications of the present analysis, our findings suggest that interventions that focus on targeting the most well-connected individuals in a population will have little or no added value compared with at-random implementation toward curbing the risk of obesity among individuals or in the population overall. However, investigators interested in understanding the implications of social networks in mitigating the obesity epidemic might consider other interventions that attempt to exploit social networks to mitigate obesity risk overall and by subgroup. Moreover, several studies have demonstrated the progression of other noninfectious health outcomes, such as smoking (36, 37), back pain (38), alcohol consumption (39), and adolescent risk behavior (37), through a densely interconnected social network. Investigators interested in these fields may also consider modeling the impacts of interventions targeting the most well-connected individuals in a social network. Finally, methodological development of agent-based modeling for epidemiology is needed. Future work, for example, may consider improving the capacity of these models to faithfully translate variance estimates on parameters used in their construction into the outcomes they are designed to assess. This information would allow investigators to identify the range of potential outcomes from interventions and could also be used to identify mechanistic factors that influence that range.

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