Annals of Epidemiology 24 (2014) 563–569

Contents lists available at ScienceDirect

Annals of Epidemiology

journal homepage: www.annalsofepidemiology.org

Original article

Reducing racial disparities in obesity: simulating the effects of improved education and social network influence on diet behavior

Mark G. Orr PhD [a,*], Sandro Galea MD, DrPH [b], Matt Riddle PhD [c], George A. Kaplan PhD [d]

[a] Social and Decision Analytics Laboratory-Virginia Bioinformatics Institute, Virginia Polytechnic Institute and State University, Arlington, VA
[b] Department of Epidemiology, Columbia University, New York, NY
[c] Decision and Information Science, Argonne National Laboratory, Lemont, IL
[d] Department of Epidemiology, University of Michigan, Ann Arbor, MI

ARTICLE INFO

Article history:
Received 23 August 2013
Accepted 23 May 2014
Available online 29 May 2014

Keywords:
Obesity
Health disparities
Complex systems
Simulation
Agent-based modeling

ABSTRACT

Purpose: Understanding how to mitigate the present black–white obesity disparity in the United States is a complex issue, stemming from a multitude of intertwined causes. An appropriate but underused approach to guiding policy approaches to this problem is to account for this complexity using simulation modeling.
Methods: We explored the efficacy of a policy that improved the quality of neighborhood schools in reducing racial disparities in obesity-related behavior and the dependence of this effect on social network influence and norms. We used an empirically grounded agent-based model to generate simulation experiments. We used a 2 × 2 × 2 factorial design that represented the presence or absence of improved neighborhood school quality, the presence or absence of social influence, and the type of social norm (healthy or unhealthy). Analyses focused on time trends in sociodemographic variables and diet quality.
Results: First, the quality of schools and social network influence had independent and interactive effects on diet behavior. Second, the black–white disparity in diet behavior was considerably reduced under some conditions, but never completely eliminated. Third, the degree to which the disparity in diet behavior was reduced was a function of the type of social norm that was in place; the reduction was the smallest when the type of social norm was healthy.
Conclusions: Improving school quality can reduce, but not eliminate racial disparities in obesity-related behavior, and the degree to which this is true depends partly on social network effects.

Introduction

There is no doubt that most behavioral, social, environmental, and biological factors that affect health and health disparities are the result of complex, multifactorial, and multilevel processes, taking place over time. However, for the most part, our analytic approaches struggle with this complexity [1]. The search for independent causes in epidemiology and other health and social sciences is attractive because it can suggest targeted narrowly defined interventions. However, when appreciating the complex determination of population health outcomes, approaches that seek to isolate independent causes as a guide to intervention may mislead by oversimplification. On the other hand, approaches that embrace a dynamic, complex, multifactorial, and multilevel perspective can be daunting from both a conceptual and analytical perspective.

Obesity is an important, and growing, public health problem [2]. However, it is an extraordinarily complex problem, perhaps best exemplified by the Foresight group's systems map of the drivers of obesity that include many dozens of pathways over many levels with considerable interaction and feedback [3]. The number of obesogenic factors and their interconnections does not easily lend itself to traditional analytical approaches such as multivariate regression.

Understanding the causes of obesity and how to prevent it becomes even more challenging when we consider racial and ethnic disparities in levels of obesity and differences in trends in obesity [4]. Because levels and trends in obesity vary considerably between groups, and race and ethnicity stand for the complex intersection of many levels of determinants [5], it is not clear what types of interventions would be most effective in reducing disparities in obesity.

Agent-based modeling is an alternative approach with considerable potential to capture the key characteristics of the obesity system: multiple-levels of analysis, interdependence between levels, heterogeneity in agents or actors, and interdependence within levels (e.g., between individual people in a social network). Furthermore, agent-based models can capture key causal mechanisms and

* Corresponding author. Social and Decision Analytics Laboratory-Virginia Bioinformatics Institute, Virginia Polytechnic Institute and State University, 900 N. Glebe Road, Arlington, VA 22203. Tel.: +1 571 858 3116; fax: +1 571 858 3015.
E-mail address: morr9@vbi.vt.edu (M.G. Orr).

1047-2797/$ – see front matter © 2014 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.annepidem.2014.05.012

transparently highlight policy levers. This approach implements the key features of a phenomenon as a computer simulation, in which individuals are represented as agents, and a set of explicit rules are defined that dictate how individuals interact with the environment and with each other [1,6,7].

With this in mind, we introduce an agent-based model that allows us to capture a large set of dynamic and interactive factors that contribute to obesity and to generate simulations that examine the short- and long-term effects of various approaches to intervening on the obesity epidemic. Although not encompassing anywhere near all the factors identified by the Foresight group [3], the model does encompass behavioral, social, and environmental determinants of dietary intake and physical activity, the chief proximal determinants of obesity.

In this initial presentation of our model, we focus on diet alone. This affords the most straightforward view of how the model works and the potentials of such a model for policy decisions. It should be noted, however, that individual levels of physical activity and neighborhood opportunities for physical activity were included in the model.

Our approach in this article was to model the potential effects of a social policy, an improvement in school quality for neighborhoods in which it is poor, on the reduction in black–white disparities in diet. School quality may affect health through multiple pathways, both directly, affecting individual choices and economic viability, and indirectly affecting neighborhood-level contextual factors [8]. Furthermore, in our model, we included the effects of endogenous social networks and norms on diet behaviors because of the increasing and strong evidence that diet behavior is influenced by social norms [9–11].

Our hypotheses are as follows. Hypothesis I: policy effects and social network effects will have independent effects on the diet of both blacks and whites, but will also interact; when social norms are aligned with a healthy diet, the effects of policy will be boosted; when not aligned with a healthy diet, the effects of policy will be reduced.

Hypothesis II: targeting neighborhood school quality will reduce the black–white disparities in diet because neighborhoods in our model with the poorest quality of schools also tended to have the greatest concentration of black residents. However, we do not expect to completely eliminate the black–white disparity because both subpopulations will be affected by changes in neighborhood school quality.

Hypothesis III: the effects of targeting neighborhood school quality will be self-sustaining, even after the policy is no longer in place, because of the propagation of diet behaviors via social network effects.

Methods and procedures

Description of the model

The population of agents in our model represented the economic and racial distributions of black and non-Hispanic whites in the 100 largest metropolitan statistical areas in the United States. This was accomplished by constructing 64 neighborhoods and matching the race or ethnic and economic distributions of these neighborhoods to empirical data sources [12]. Each neighborhood had 25 households, resulting in 1600 housing units. Please see the online supplemental material for details on these procedures.

Agents in the model were born, changed neighborhoods (residential mobility), went to school at the age of 6 years, got jobs after they left school, retired at the age of 65 years, had one child at age the age of 25 years, and died at age-specific rates reflective of the US population. In the process, these agents exhibited health behaviors (smoking, diet, and exercise) that produced health outcomes (body mass index, cardiovascular disease, and death). The health behaviors were dependent on the agent's education level, neighborhood school quality, walkability, access to healthy foods, and the behaviors of others. The agents were connected via social ties (to represent friendship networks) and social norms among friends affected the extent of social influence. The parameters in the model were, wherever possible, empirically grounded to extant data sources as described in the online supplemental material.

Key variables in the model

Figure 1 illustrates the variables and related processes in the model related to diet, a subset of the full set of variables in the model. The full set of variables and processes are described in detail in the online supplemental material. It is important to emphasize that

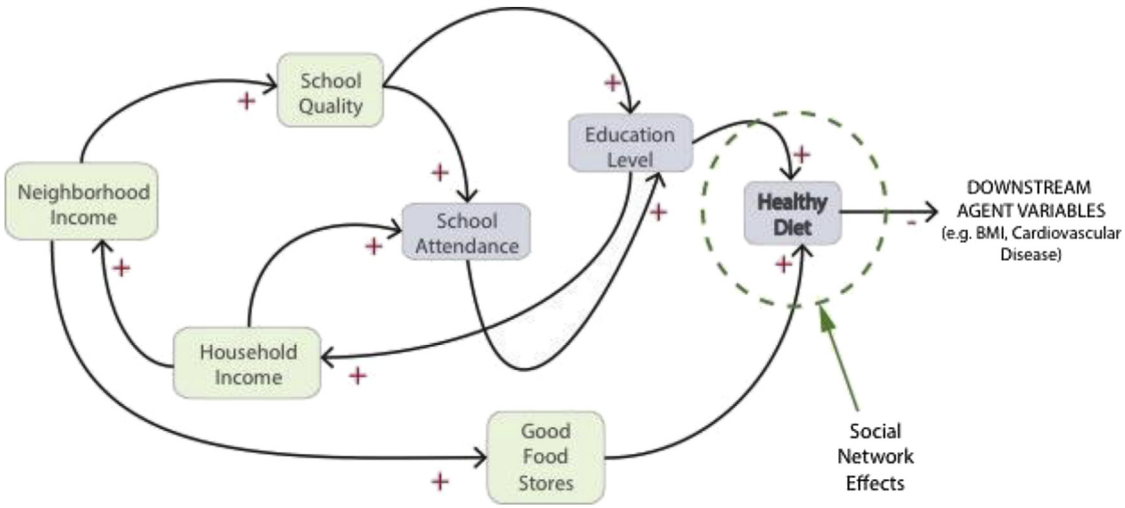

Fig. 1. Diagram of agent-rule structure related to agent healthy diet. The *light-green boxes* depict neighborhood-level variables; *light-blue* depicts agent-level variables. The *dotted-green circle* highlights that an agent's diet behavior is also a function of direct social ties to other agents in the agent's social network. The *red plus* and *minus signs* express the nature of the causal rule; positive is direct, negative is inverse (there are no negatives). Smoking and physical activity behaviors (at the agent level) were left out of the diagram for simplicity.

although the simulations presented in this article focus on the variables in Figure 1, they are conducted in the context of the full model.

Healthy diet, our measure of diet behavior, was modeled to reflect the distribution of the Healthy Eating Index-2005 [13]. This reflected the quality of nutrient intake and its associations with other key individual and neighborhood variables. As shown in Figure 1, an agent's diet was directly affected by the amount of good food stores in the agent's neighborhood and the agent's education level—as these variables increased in value, so did the quality of the agent's healthy diet [14,15].

School quality was expressed in terms of student-to-teacher ratios. Although not capturing all the factors that determine school quality, student-to-teacher ratio is importantly associated with the economic returns from increasing education, and some evidence suggests other benefits as well [16–18].

Social network effects are depicted in the model by the green-dashed circle in Figure 1. Each agent was assigned a social norm threshold for what it considered to be a desirable level of healthy diet. This threshold was compared with the average healthy diet of others in the agent's social network (the agent's set of friends). If the agent's threshold was higher than the average of the others', then the agent decreased its own healthy diet; if lower, it increased its healthy diet. The specific formulation of this process is described in detail in the online supplemental material.

The agent's education level was a function of both the school quality in its neighborhood and the number of years that school was attended. An agent's school attendance increased as both the income of the agent's household and neighborhood-level school quality increased. Household income was a function of the aggregate education level of all agents. Neighborhood income was simply the aggregate of the households in a neighborhood—it directly affected the school quality and good food stores in the neighborhood. On the far right side of Figure 1 are downstream agent variables that were partly a function of diet. These are not directly analyzed in this article.

The specific equations that underlie Figure 1 and the associated empirical work supporting the model parameters are presented in the online supplemental material.

Model analysis and experimental design

The primary experimental design manipulated whether the school quality policy (described in the following) was in place (policy or no policy respectively, two-level), social network effects (present or not, two-level), and the social norm type (low or high social norm threshold, two-level). Thus, we used a 2 (policy) × 2 (social network effect) × 2 (social norm type) design for a total of eight conditions per each of two experiments. Functionally, two pairs of conditions were identical—when there were no social network effects, social norm type did not have an impact on system behavior. Thus, we present only six conditions per experiment. For each experiment, each condition was simulated 20 times with a different random seed. All simulations ran for 110 time steps. This was long enough to reflect, on average, about three generations of agents. We report averages for each time step over the 20 simulations for each condition for all key state variables (described in the following) at three time points in the simulation.

Experiments 1 and 2 differed only with respect to the length the policy manipulation was in place. It was either continuous throughout the simulation, that is, across all time steps (experiment 1) or only early-on (up until time step 50) in the simulation (experiment 2).

School policy and social network manipulations

The policy manipulation of school quality targeted the neighborhoods in the lowest 20% of school quality. The strength of the manipulation in our simulation experiments was large, reducing the student-to-teacher ratio by 61% (equivalent to four standard deviations from the average value of the student-to-teacher ratio). In short, we improved the student-to-teacher ratio in the lowest 20% of the neighborhoods to be four standard deviations above the average value of school quality. In effect, this changed the average student-to-teacher ratio for the targeted neighborhoods from 15.03 to 7.60.

The manipulation of social networks worked in two ways. First, we simply blocked social influence between agents when the social network effect was not present. Second, when social influence was present, the social norm type induced agents, typically, to either increase healthy diet or decrease healthy diet by placing the agents' social norm threshold either below (at 50.4) or above (at 58.5) the average healthy diet (at 55.2) of all agents in the model, respectively.

Table 1
The key observed state variables for the simulations

Variable name	Description of variable representation
Healthy diet	Healthy Eating Index-2005
Gross income	Household-level gross income
Good food stores	Neighborhood-level good food stores in terms of the natural log of the ratio of the number of supermarkets per 10,000 to the average number of supermarkets per 10,000 (the second term is 0.71 in our model)
Education level	Aggregate of years in school and school quality that links returns on education to an agent's earnings
Years in school	Years an agent is in school
School quality	Neighborhood-level student-to-teacher ratio
Percent targeted	Percent of blacks and whites targeted by the policy manipulation

Statistical analysis

All analysis is derived from what we call the standard model output—a tracking of the average values of model state variables (described next) separately for black and white agents over simulation time steps. We analyzed the standard model output in several ways.

Analysis of key state variables. Table 1 presents a description of the key state variables that we analyzed in the experiments. In Table S1a–c (online supplement), we provide the average of the standard errors for three time points (beginning, middle, and end of simulation) for each key state variable.

Analysis of healthy diet. To analyze the changes in healthy diet, we used a two-way mixed analysis of variance (ANOVA). Policy, a between-subjects factor, was defined as described previously (policy or no policy). Period, a within-subjects factor, was defined as the first and the last time step in a simulation run. Note that the term "subjects" refers to simulation runs, not agents. We tested the statistical significance for both main effects and interactions for both factors. In Table 2, we show the average values of healthy diet

Table 2
The average of healthy diet by policy and period separately by race for each simulation in experiment 1

Race	Policy	Exp 1a			Exp 1b			Exp 1c		
		Begin	End	Main eff.	Begin	End	Main eff.	Begin	End	Main eff.
Black	No	55.06	52.74	53.90	56.31	59.36	57.84	53.75	48.02	50.89
	Yes	55.06	58.34	56.70	56.31	66.10	61.21	53.75	52.84	53.30
	Main eff.	55.06	55.54	—	56.31	62.73	—	53.75	50.43	—
White	No	61.15	59.69	60.42	63.04	69.11	66.08	60.68	55.60	58.14
	Yes	61.15	61.78	61.46	63.04	71.99	67.52	60.68	57.69	59.19
	Main eff.	61.15	60.73	—	63.04	70.55	—	60.68	56.65	—

eff. = effects; Exp = experiment.

Table 3
The average difference between blacks and whites in healthy diet by policy and period in experiment 1

Policy	Exp 1a			Exp 1b			Exp 1c		
	Begin	End	Main eff	Begin	End	Main eff.	Begin	End	Main eff.
No	−6.10	−6.95	−6.52	−6.73	−9.76	−8.24	−6.93	−7.58	−7.25
Yes	−6.10	−3.44	−4.77	−6.73	−5.89	−6.31	−6.93	−4.85	−5.89
Main eff.	−6.10	−5.19	—	−6.73	−7.82	—	−6.93	−6.21	—

eff. = effects; Exp = experiment.

for the 10th and the last time steps for both the main effects and cell means when crossing the policy and period factors. This is provided separately for blacks and whites for each experiment. In the case of a statistically significant interaction, we used a dependent *t* test within each level of policy to test for statistically significant differences in healthy diet over time. For all ANOVA and related *t* tests, we used a criterion value of alpha less than 0.001 to account for the 18 ANOVA effects and 24 planned comparisons conducted throughout experiment 1 (a–c).

Furthermore, in Table 3, we show the average difference between blacks and whites in healthy diet for the 10th and the last time steps, using the same format as Table 2. We used a two-way mixed ANOVA to test for changes in the difference in healthy diet between blacks and whites because of the school policy manipulation. Given statistically significant interactions, we used an independent *t* test at the end point in the simulation to test whether school policy affected the magnitude of difference in healthy diet between blacks and whites.

Results

Experiment 1

Experiment 1a manipulated student-to-teacher ratio when no social network effects were in place. For this experiment, the policy was continuously applied from time step 10 to 110. Figure 2A illustrates the full time series of healthy diet for experiment 1a. Table 2 shows the beginning and ending values of healthy diet for experiment 1a along with the main effects for both policy and period. For blacks, a two-way mixed ANOVA yielded a main effect for policy, $F(1,38) = 49.72$, $P < .001$, no main effect for period, $F(1,38) = 7.71$,

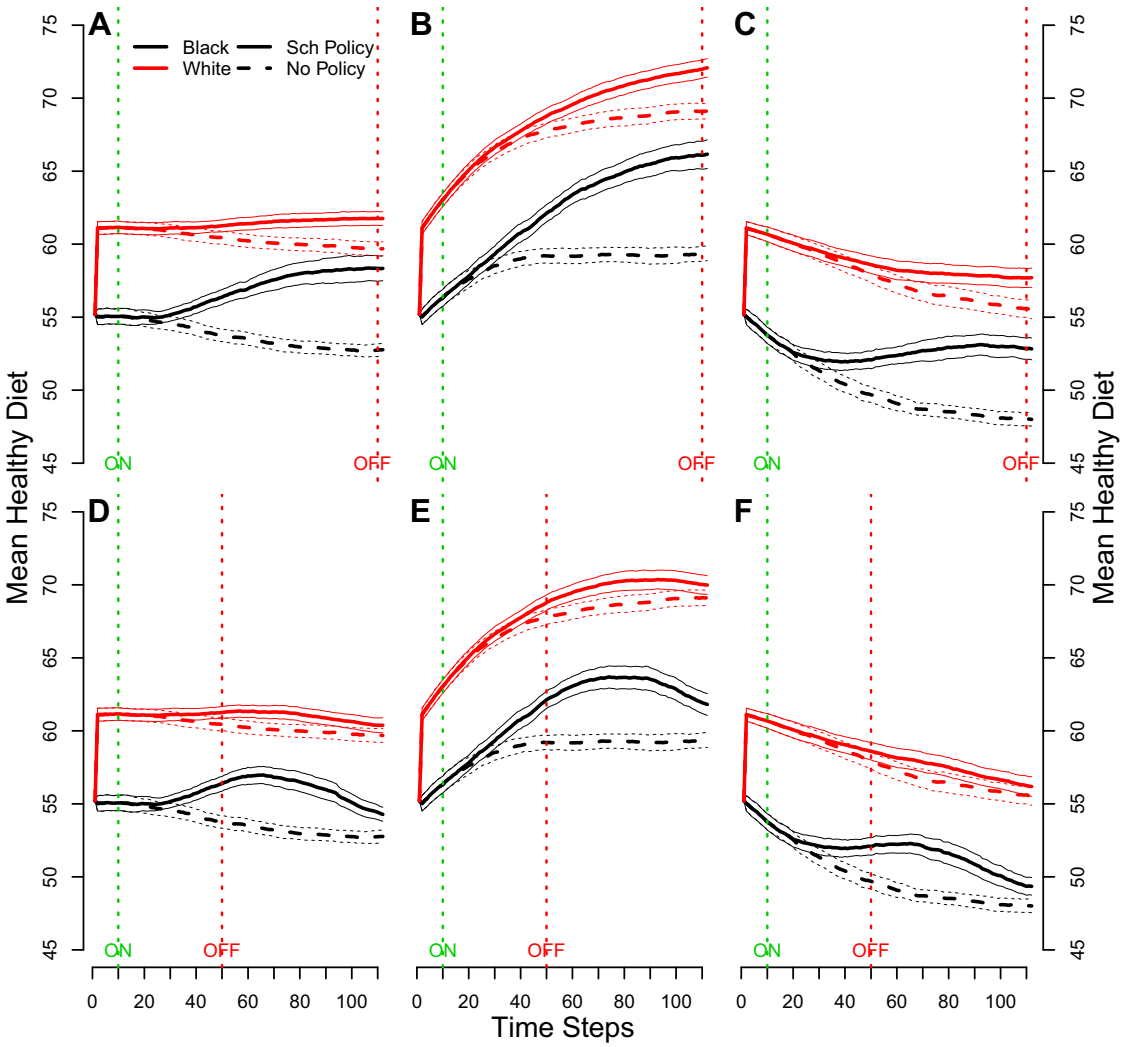

Fig. 2. Average healthy diet for blacks and whites over model time steps for experiments 1 and 2. *Panels A–C* show experiment 1 (continuous policy). *Panels D–F* show experiment 2 (policy early-on). *Panel A* shows the effects of policy without social network effects in place; *panel B* shows social network effects with low social norm threshold; *panel C* shows social network effects with high social norm threshold. *Panels D–F* have the same structure as *A–C* but for experiment 2. The *vertical red-* and *green-dashed lines* depict when policy was turned on and off, respectively. For all panels, the thick lines depict the mean value of healthy diet; the thin lines depict the 95% confidence intervals.

ns (non-significant) and a significant policy by period interaction, $F(1,38) = 257.11$, $P < .001$. Both the no policy and policy conditions yielded significant period differences, $t(19) = 13.27$, $P < .001$, and $t(19) = 10.86$, $P < .001$, respectively, although the directions of change were different. For whites, ANOVA yielded no main effect for policy, $F(1,38) = 11.74$, *ns*, a main effect for period, $F(1,38) = 19.32$, $P < .001$, and a significant policy by period interaction, $F(1,38) = 119.16$, $P < .001$. The no policy condition yielded a significant period difference, $t(19) = 13.15$, $P < .001$, but the policy condition did not, $t(19) = 4.01$, *ns*.

We also addressed whether the school policy was able to reduce the health disparity in healthy diet between blacks and whites. Table 3 shows the average difference between blacks and whites in healthy diet for the beginning and ending of experiment 1a along with the main effects for both policy and period. A two-way mixed ANOVA yielded a main effect for policy, $F(1,38) = 25.76$, $P < .001$, a main effect for period, $F(1,38) = 18.53$, $P < .001$ and a significant policy by period interaction, $F(1,38) = 69.69$, $P < .001$. The planned contrast of interest was to compare policy with no policy at the end point—the difference between blacks and whites was smaller by 51% when the school policy was in place $t(38) = 8.20$, $P < .001$.

Experiment 1b manipulated policy when social network effects were in place and the social norm type was set to increase healthy diet. Figure 2B illustrates the full time series of healthy diet for experiment 1b. Table 2 shows the beginning and ending values of healthy diet for experiment 1b along with the main effects for both policy and period (i.e., the beginning and ending values). For blacks, a two-way mixed (policy, between; period, within) ANOVA yielded a main effect for both policy, $F(1,38) = 64.84$, $P < .001$ and period, $F(1,38) = 740.00$, $P < .001$ and a significant policy by time period interaction, $F(1,38) = 204.60$, $P < .001$. Given the significant interaction, we applied dependent t tests within each level of policy. Both the no policy and policy conditions yielded significant time period differences, $t(19) = 19.53$, $P < .001$, and $t(19) = 21.99$, $P < .001$, respectively. For whites, ANOVA yielded a main effect for both policy, $F(1,38) = 16.31$, $P < .001$ and period, $F(1,38) = 5599.00$, $P < .001$, and a significant policy by period interaction, $F(1,38) = 205.00$, $P < .001$. Both the no policy and policy conditions yielded a significant period difference, $t(19) = 48.67$, $P < .001$ and $t(19) = 56.91$, $P < .001$, respectively.

As for experiment 1a, we also addressed whether school policy was able to reduce the health disparity in healthy diet between blacks and whites in Table 3 (see Exp 1b). A two-way mixed ANOVA yielded a main effect for policy, $F(1,38) = 22.38$, $P < .001$, a main effect for period, $F(1,38) = 19.16$, $P < .001$ and a significant policy by period interaction, $F(1,38) = 59.77$, $P < .001$. The planned contrast of interest was to compare policy with no policy at the end point—the difference between blacks and whites was smaller by 40% when the school policy was in place $t(38) = 7.30$, $P < .001$.

Experiment 1c was identical to 1b except the social norm type was set to decrease healthy diet. Figure 2C illustrates the full time series of healthy diet for experiment 1c. Table 2 shows the beginning and ending values of healthy diet for experiment 1c along with the main effects for both policy and period (i.e., the beginning and ending values). For blacks, a two-way mixed (policy, between; period, within) ANOVA yielded a main effect for both policy, $F(1,38) = 43.14$, $P < .001$ and period, $F(1,38) = 322.1$, $P < .001$ and a significant policy by period interaction, $F(1,38) = 169.4$, $P < .001$. Given the significant interaction, we applied dependent t tests within each level of policy. The no policy condition yielded significant period differences, $t(19) = 32.16$, $P < .001$, but the policy condition did not, $t(19) = 2.81$, *ns*. For whites, ANOVA yielded no main effect for policy, $F(1,38) = 7.21$, $P < .001$ but a significant main effect for period, $F(1,38) = 1533.9$, $P < .001$, and a significant policy by period interaction, $F(1,38) = 102.5$, $P < .001$. Both the no policy and policy conditions yielded a significant period difference, $t(19) = 38.98$, $P < .001$ and $t(19) = 18.74$, $P < .001$, respectively.

We also addressed whether school policy was able to reduce the health disparity in healthy diet between blacks and whites in Table 3 (see Exp 1c). A two-way mixed ANOVA yielded a main effect for policy, $F(1,38) = 14.28$, $P < .001$, a main effect for period, $F(1,38) = 12.65$, $P < .01$ and a significant policy by period interaction, $F(1,38) = 46.28$, $P < .001$. The planned contrast of interest was to compare policy with no policy at the end point—the difference between blacks and whites was smaller by 36% when the school policy was in place $t(38) = 6.41$, $P < .001$.

To summarize the results thus far, we point out three patterns that emerged from experiment 1. First, the effects of policy alone are illustrated clearly in experiment 1a for which there was a significant change in healthy diet for blacks in the absence of social network effects. The effect of social networks without the presence of the policy was evident in both experiment 1b and c. When the social norm type was characterized by a low threshold, healthy diet increased; a high threshold yielded a decrease in healthy diet. In terms of interactivity, the effects on healthy diet were strongest when policy was aligned with the social norm type. That is, the largest changes in healthy diet were found in experiment 1b where both the policy and the social norm type were designed to improve healthy diet. In contrast, when the policy and social norm type were opposed, as in experiment 1c, there was a clear competition between the two.

Second, the policy conditions, although strong, did not completely eliminate the racial disparities in diet behavior. This may be explained by the fact that the policy manipulation targeted race only indirectly via neighborhood-level school quality. In effect, the average student-to-teacher ratio was reduced by about 35% for blacks compared with about 60% for the neighborhoods that were directly targeted (data not shown). Furthermore, in all conditions of experiment 1 it appears that the time curves of healthy diet were leveling off for both blacks and whites. So, even if we extended the simulation in time, we probably would not have completely reduced the black–white disparity in healthy diet.

Third, the difference between blacks and whites, at the end of the simulation, was largest when the social network effects were in place and the social norm type was set to increase the healthy diet. This was probably due to the simple fact that social network effects were present in both blacks and whites.

The online supplementary material provides a more global view of the systems behavior over experiments 1a–c. Although not essential for understanding the dynamics of diet behavior in our model, these data provide a broader view of how the system as a whole operates across the experiments.

Experiment 2

Experiment 2 was identical to experiment 1 except that the school quality policy was only applied early-on in the simulation, from time step 10 to 50, and then turned off. The primary interest in experiment 2 was to understand how healthy diet would change in response to the removal of the policy implementation at time step 51. Thus, we focus the results only on the time course of healthy diet and do not provide detailed statistical testing of the differences in healthy diet over time or between blacks and whites. Experiment 2a is presented in Figure 2D. It is clear that the effects of policy are not maintained when the social networks effects were not present. This finding was repeated in both experiments 2b and 2c (see Fig. 2E and F, respectively) in which social network effects were in place. With social norm type set to improve the healthy diet, there was a continued increase in healthy diet for blacks which then reversed and began to decline. For whites, there was a much smaller decline.

With social norm type set to decrease healthy diet, the results were similar for blacks and whites—after the policy was turned off, levels of healthy diet declined to the levels shown with no school quality policy intervention.

To summarize, the policy effects on healthy diet in experiment 2 were transitory. We hypothesized that the social network effects, when present, might drive the system toward an increased healthy diet once the policy implementation was removed in experiment 2b. Our simulations did not support this hypothesis. Once the policy manipulation was removed, healthy diet moved toward the level of the respective no policy condition.

Discussion

These results reinforce and elaborate on several themes in the literature on determinants of population health and racial disparities in health. First, there exists a potential to leverage upstream macrosocial policies to affect the change in population-level racial disparities in health behaviors [19]. Although our model focused on only one aspect of school quality (student-to-teacher ratio), the results fall in-line with contemporary thought regarding the potential effects of education on health. Education may operate via several mechanisms, some working through income and occupation (e.g., increased access to health care; more comprehensive health insurance and safer working environments) and some directly linked to education itself (e.g., lower discounting rates for returns to future health; increased cognitive skills; increased ability to delay gratification; and societal rank) [20,21]. Furthermore, implementing changes in education is thought to be feasible through various policy channels [22,23], and thus has the potential to fundamentally change the patterning of disparities in obesity and related behaviors in the United States.

Second, the impact of policy efforts toward reducing obesity needs to take into account social context [24]. Specifically, the effects of policy may be systematically dependent on the initial conditions of the social networks (i.e., the type of social norm) at the time of policy implementation. Also, there may exist the potential to align policy efforts with the initial conditions of the social network or for the policy to circumvent these initial conditions.

Third, because of the complex and dynamic determination of health behaviors [25] and their links to health outcomes, it may be difficult to estimate the length of time and degree of efficacy that arise from macrosocial policy changes. Tobacco control provides an excellent example—despite a slow and steady decline in the prevalence of current smokers (aged 18–24 years) from 1965 to 1995, the trends since 1995 are indicative of a much slower decline. Our model implies a similar scenario when considering macrosocial, upstream effects on obesity—slow incremental changes and the potential for less than 100% efficacy.

Finally, in the current model, contrary to our expectations and the work of others [26], social network effects did not generate a sustained change in behavior, so a relatively short perturbation of upstream policy may not drive the system to a healthy state after the policy is no longer in place. Although the assumptions of our model accord with recent work demonstrating the social diffusion of obesity-related behaviors [27–29], once the policy is no longer in place, factors other than social influence dominated individual-level behaviors, such as education level and neighborhood-level good food stores.

As with any computational modeling enterprise, implications about the real world are relative to the fidelity of the model said world. Our model is based on causal statements (in equation form) that represent hundreds of variables. Where possible, we grounded the model assumptions with extant data sources. Not infrequently the data are not completely up to the task and various adjustments need to be made to estimate parameters from studies, some of which are cross-sectional versus longitudinal with varying lengths of follow-up, some adjusted for covariates versus some not, with various studies covering different populations and different periods of time. In some cases, we needed to estimate parameters without any background data. For example, the social network influence equations, although plausible, are not informed by any single study. The sign is probably correct, but the magnitude may be in question. We present three separate sensitivity analyses in the online supplementary material and urge the reader to review them.

Regarding the direct implications for policy, the results of this study should only be taken as an indication of the potential for reducing racial disparities in obesity-related behaviors via changes in educational opportunity. Neither the time course nor the magnitude of the effect should be taken at face value. This limitation, if it should be considered as such, is important given the extent to which the assumptions of the model are empirically grounded (see previous paragraph). However, the real value of this study lies in the recognition that macrosocial upstream policies that do not target racial disparities or target-specific racial groups specifically may serve as an effective long-term strategy to help ameliorate disparities specific to obesity-related behaviors.

In summary, we believe that the results indicate the value of a carefully specified agent-based model that brings together a variety of factors thought to influence dietary choice. Such a model has allowed us to simulate the effects of changing education policy and the nature of friendship networks on diet quality, allowing for considerable feedback and interaction across levels. Although the results should be seen within the lens of "first attempts," the results illustrate the value of such efforts, and the need for further elaboration and testing of *in-silico* models.

Acknowledgment

This work was supported in part by an Epidemiology Merit Fellowship to M.G.O. from the Mailman School of Public Health, Columbia Univ., grant No. 60466 from the Robert Wood Johnson Foundation to S.G. and G.A.K., a TIIH Scholar Award to G.A.K. from the Institute for Integrative Health, and by a small grant from the Network on Inequalities, Complex Systems, and Health (HHSN276200800013C). The authors would like to thank Nathan Osgood, Ronald Mintz, and Dylan Knowles regarding technical development of the agent-based model and Andrew Kosenko for aid in manuscript preparation.

Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.annepidem.2014.05.012.

References

[1] Galea S, Riddle M, Kaplan GA. Causal thinking and complex system approaches in epidemiology. Int J Epidemiol 2010;39(1):97–106.
[2] Glickman D, Parker L, Sim LJ, Cook H, Miller EA. Accelerating progress in obesity prevention: Solving the weight of the nation. [Book]: National Academies Press; 2012 [updated 6/6/12]; Available from: http://www.nap.edu/catalog.php?record_id=13275.
[3] Vanderbroeck P, Goossens J, Clemens M. Tackling obesities: Future choices—building the obesity system map In: (UK) GOfS, editor.2007.
[4] Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence among US adults, 1999-2008. J Am Med Assoc 2010;303(3):235–41.
[5] Kaplan GA. What's wrong with social epidemiology, and how can we make it better? Epidemiological Rev 2004;26:124–35.
[6] El-Sayed AM, Seemann L, Scarborough P, Galea S. Are network-based interventions a useful antiobesity strategy? An illustration of simulation models of causal inference in epidemiology. Am J Epidemiol 2013;178(2):287–95.

[7] Galea S, Hall C, Kaplan GA. Social epidemiology and complex system dynamic modelling as applied to health behaviour and drug use research. Int J Drug Policy 2009;20(3):209–16.
[8] Cohen AK, Rai M, Rehkopf DH, Abrams B. Educational attainment and obesity: a systematic review. Obes Rev 2013;14:989–1005.
[9] Barclay KJ, Edling C, Rydgren J. Peer clustering of exercise and eating behaviours among young adults in Sweden: a cross-sectional study of egocentric network data. BMC Public Health 2013;13:784.
[10] Pelletier JE, Graham DJ, Laska MN. Social norms and dietary behaviors among young adults. Am J Health Behav 2014;38(1):144–52.
[11] Salvy S-J, de la Haye K, Bowker JC, Hermans RCJ. Influence of peers and friends on children's and adolescents' eating and activity behaviors. Physiol Behav 2012;106:369–78.
[12] Massey DS, Gross AB, Shibuya K. Migration, segregation, and the geographic concentration of poverty. Am Sociological Rev 1994;59(3):425–45.
[13] Guenther PM, Reedy J, Krebs-Smith SM, Reeve B. Evaluation of the Healthy Eating Index-2005. J Am Diet Assoc 2008;108:1854–64.
[14] Franco MA, Diez Roux A, Nettleton J, Lazo M, Brancati F, Caballero B, et al. Availability of healthy foods and dietary patterns: the Multi-Ethnic Study of Atherosclerosis. Am J Clin Nutr 2009;89:897–904.
[15] Krebs-Smith SM, Cook A, Subar AF, Cleveland L, Friday J. US adults' fruit and vegetable intakes, 1989 to 1991: a revised baseline for the Healthy People 2000 objective. Am J Public Health 1995;85(12):1623–9.
[16] Card D, Krueger AB. Does school quality matter? Returns to education and the characteristics of public schools in the United States. J Polit Economy 1992;100(1):1–40.
[17] Card D, Krueger AB. School quality and the return to education. In: Burtless G, editor. Does money matter?: The effect of school resources on student achievement and adult success. Washington, D.C.: Brookings Institute; 1996. pp. 118–9.
[18] Mishel L, Rothstein R, editors. The class size debate. Washington, D.C.: Economic Policy Institute; 2002.
[19] Galea S, editor. Macrosocial determinants of population health. New York: Springer; 2007.
[20] Chandola T, Clarke P, Morris JN, Blane D. Pathways between education and health: a causal modelling approach. J R Stat Soc A 2006;169(Part 2):337–59.
[21] Cutler DM, Lleras-Muney A. Education and health: evaluating theories and evidence. Cambridge, MA: National Bureau of Economic Research; 2006. Contract No.: 12352.
[22] Low M, Low B, Baumler E, Huynh P. Can education policy be health policy? Implications of research on social determinants of health. J Health Polit Policy L 2005;30:1131–62.
[23] Woolf SH, Johnson RE, Phillips RL, Philipsen M. Giving everyone the health of the educated: an examination of whether social change would save more lives than medical advances. Am J Public Health 2007;97:679–83.
[24] Bahr DB, Browning RC, Wyatt HR, Hill JO. Exploiting social networks to mitigate the obesity epidemic. Obesity 2009;17(4):723–8.
[25] Sterman JD. Learning from evidence in a complex world. Am J Public Health 2006;96(3):505–14.
[26] Orr MG, Evans CR. Understanding long-term diffusion dynamics in the prevalence of adolescent sexual initiation: a first investigation using agent-based modeling. Res Hum Development 2011;8(1):48–66.
[27] Kayla de la Haye GR, Mohr Philip, Wilson Carlene. Obesity-related behaviors in adolescent friendship networks. Social Networks 2010;(32):161–7.
[28] Kayla de la Haye GR, Mohr Philip, Wilson Carlene. Homophily and Contagion as explanations for weight similarities among adolescent friends. J Adolesc Health 2011;(49):421–7.
[29] Shoham DA, Tong L, Lamberson PJ, Auchincloss AH, Zhang J, Dugas L, et al. An actor-based model of social network influence on adolescent body size, screen time and playing sports. PLOS One 2012;7(6):e39795.