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Original article

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

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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

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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

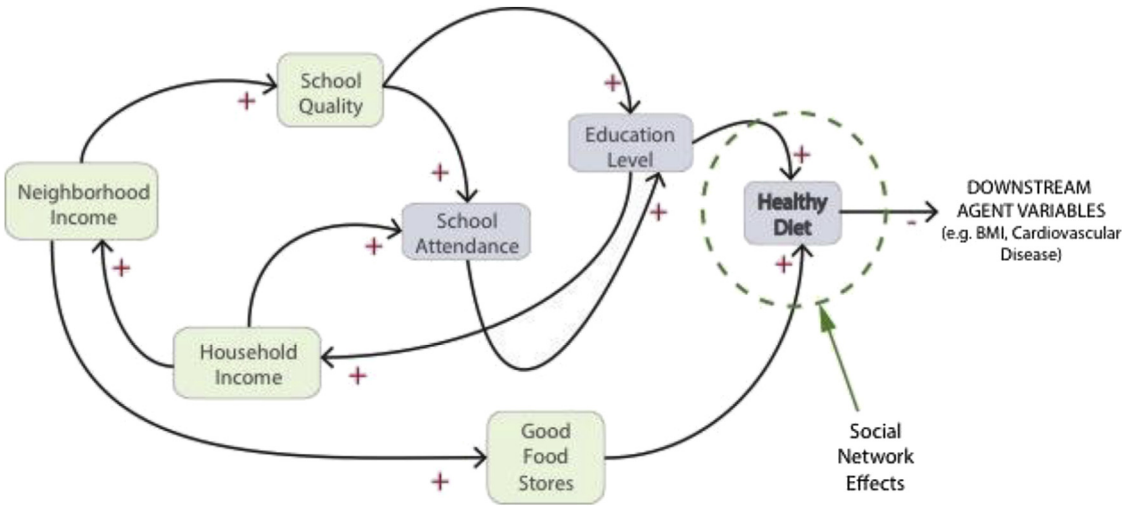

Fig. 1. Diagram of agent-rule structure related to agent healthy diet. The *light-green boxes* depict neighborhood-level variables; *light-blue* depicts agent-level variables. The *dotted-green circle* highlights that an agent's diet behavior is also a function of direct social ties to other agents in 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.