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Addressing Population Health and Health Inequalities: The Role of Fundamental Causes

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Abstract

Objectives—As a case study of the impact of universal versus targeted interventions on population health and health inequalities, we used simulations to examine (1) whether universal or targeted manipulations of collective efficacy better reduced population-level rates and racial/ethnic inequalities in violent victimization; and (2) whether experiments reduced disparities without addressing fundamental causes.

Methods—We applied agent-based simulation techniques to the specific example of an intervention on neighborhood collective efficacy to reduce population-level rates and racial/ethnic inequalities in violent victimization. The agent population consisted of 4000 individuals aged 18 years and older with sociodemographic characteristics assigned to match distributions of the adult population in New York City according to the 2000 US Census.

Results—Universal experiments reduced rates of victimization more than targeted experiments. However, neither experiment reduced inequalities. To reduce inequalities, it was necessary to eliminate racial/ethnic residential segregation.

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Contributors

M. Cerdá designed the study, contributed to the creation of the agent-based model, conducted the literature review, and wrote the article. M. Tracy created the agent-based model, conducted the simulations, and wrote sections of the article. J. Ahern contributed to the study design and substantially edited all sections of the article. S. Galea contributed to the study design and the creation of the agent-based model, and substantially edited all sections of the article.

Human Participant Protection

This study was approved by the institutional review board of Columbia University.

Conclusions—These simulations support the use of universal intervention but suggest that it is not possible to address inequalities in health without first addressing fundamental causes.

The work of Geoffrey Rose transformed our conception of public health prevention efforts. Rose introduced the notion of a universal strategy of prevention, which targets a whole population regardless of variation in individuals' risk status.[1,2] This strategy is grounded on 2 important assumptions: (1) the distribution of risk in a population is shaped by contextual conditions that differ between populations, and (2) most cases arise from the large population with only an average level of risk, rather than from the small population at high risk.[1,2] Although each individual at average risk has a low probability of disease incidence, so many are exposed that the number of cases arising from this group is large. Thus, intervening on the entire population improves the risk distribution for all, resulting in the most effective improvement in population health. Rose differentiated such a universal strategy from the targeted strategy, which dominates much of biomedicine to this day. The targeted strategy identifies and intervenes on individuals with high disease risk. This strategy is appropriate to the individuals treated, as it is tailored to their specific risk factors. However, because it does not deal with the root of the problem by shifting the population risk distribution, a targeted strategy must continue indefinitely treating those at highest risk.[3]

Rose's strategy of universal intervention has been criticized for not addressing the structural factors that lead to different distributions of risk between social groups, such that those with the lowest initial level of risk are the first to derive benefits from universal interventions, potentially exacerbating health inequalities.[4–6] This has been seen in interventions in areas such as smoking prevention, smoking cessation, cervical cancer screening, and neonatal intensive care whereby a universal intervention was associated with attendant widening of intergroup differences in health.[7–9] Such a view is consistent with fundamental cause theory, which argues that higher social status, as indexed by knowledge, money, power, social connectedness, and prestige is always associated with better access to resources that optimize health, even though health and its predictors may change with time.[10–12] Hence, an intervention may shift the mean distribution of disease, but if the intervention fails to address the underlying economic and political forces that lead to a different risk exposure across social groups, those with more resources (and thus lower initial risk) will benefit more from the intervention so that inequalities may increase with the intervention.

Questions about the effect of universal versus targeted prevention strategies on population health and health inequalities, and the role that fundamental causes play in population health, are critical to the articulation of effective public health planning strategies. Although an energetic debate exists about the potential merits and shortcomings of targeted versus universal interventions,[4,13–15] we are not aware of any empirical tests that examine the impact of universal versus targeted public health interventions on both population-level rates of disease and inequalities in disease. We aimed to fill this gap by quantifying the impact of universal and targeted interventions on both population health and health inequalities and testing whether it was possible for interventions to effectively address population health and health inequalities without addressing fundamental causes of health. Empirical testing of these questions would require large-scale population-based experiments that manipulate social exposures. Such experiments are prohibitively expensive or logistically impossible to

implement. We instead addressed these questions through the use of agent-based simulation modeling that allowed us to simulate large populations in silico.

We used a case study to test the impact of universal versus targeted interventions on population health and health inequalities: manipulating collective efficacy to reduce both population-level rates and racial/ethnic inequalities in violent victimization. The concept of collective efficacy arises from social disorganization theory and involves the ability of community residents to collectively harness resources and effectively respond to negative situations for the benefit of the community (informal social control), combined with the degree to which community residents mutually trust and respect each other (social cohesion).[16] Collective efficacy has been consistently associated with reduced neighborhood victimization across observational studies in the United States and other countries.[16–21] Interventions are currently under way in cities across the United States and other countries to mobilize collective efficacy as a way to improve public health.[22–26]

We used collective efficacy and victimization for our case study because the focus of intervention (i.e., collective efficacy) and the health indicator (i.e., violent victimization) are socially distributed, and the role of fundamental causes of health is particularly relevant in this case. Collective efficacy arises in more stable, less economically disadvantaged neighborhoods.[16,17,27,28] Victimization, in turn, is racially and economically patterned: in 1980–2008, Blacks were disproportionately represented as homicide victims and offenders. They were 6 times more likely to die from homicide than were Whites, and the offending rate was 8 times higher among Blacks than among Whites.[29] An important determinant of the elevated rates of homicide among Blacks is the disproportionate segregation of Blacks into economically disadvantaged neighborhoods,[30–36] where there are lower levels of protective social processes such as collective efficacy as well as exposure to multiple other risk factors for violent victimization.[37,38] Hence, racial residential segregation is a fundamental cause of violent victimization as well as multiple other correlated health-related problems.[37]

We used in silico experiments that capitalize on innovative complex systems approaches to answer 2 major questions: (1) what is the comparative impact of universal versus targeted experimental manipulations of collective efficacy on population-level rates of violent victimization and on Black–White inequalities in victimization? and (2) when the level of racial residential segregation is altered, does the impact of collective efficacy on population-level rates of violent victimization and of Black–White inequalities in victimization change?

We used agent-based modeling (ABM) to simulate a series of in silico neighborhood experiments. Because ABMs consist of simulations that follow prescribed rules about the characteristics of agents, their networks, contexts, and behaviors, investigators can simulate scenarios in which only 1 aspect of the initial conditions is changed, thus allowing us to conduct counterfactual neighborhood policy “experiments” without issues of resource costs or ethical concerns. These in silico experiments can serve as a first step to build the evidence base on tractable interventions that can then be tested in community-randomized trials.

METHODS

We created an ABM simulating the dynamic processes that govern exposure to violence, including contact between individuals and the influence of the neighborhood environment (for a diagram summarizing the processes, see Appendix 1, available as a supplement to this article at http://www.ajph.org). We implemented and compared 2 neighborhood experimental manipulations of collective efficacy, 1 universal and 1 targeted, under the contexts of complete and no residential segregation. Our intention was not to emulate a realistic context of residential segregation but to use extremes to illustrate the impact that residential segregation can have on interventions. We developed the ABM using Recursive Porous Agent Simulation Toolkit (Repast) software version 3.1 (Argonne National Laboratory, Argonne, IL), which uses Java programming language version 7 (Oracle, Redwood Shores, CA), and implemented it in Eclipse version 4.2 (Eclipse Foundation, Ottawa, Canada). The model followed the overview, design concepts details protocol[39,40]; for more details about model parameters, including a flowchart and pseudo-code demonstrating the processes in the model, see Appendices 4 and 5 (available as a supplement to this article at http://www.ajph.org).

The purpose of the ABM was to compare the effects that universal and targeted experimental manipulations of collective efficacy have on population rates of violent victimization as well as Black–White inequalities in victimization, under alternate scenarios of racial and economic residential segregation. The broader objective of the model, then, was to determine whether a universal or targeted intervention approach could reduce health inequalities without addressing fundamental causes of those inequalities (e.g., residential segregation).

Entities, State Variables, and Scales

The model consisted of adult “agents” residing in a physical environment divided into neighborhoods. The static and time-varying variables characterized individual agents, in addition to their location on the grid representing the physical environment and the identity number of the neighborhood where they live. Individual behaviors included violent perpetration, violent victimization, other traumatic event exposure, and development of posttraumatic stress disorder (PTSD). We developed equations predicting the probability of each agent behavior using data from 2 longitudinal studies: the National Epidemiologic Survey of Alcohol and Related Conditions[41] and the World Trade Center study.[42]

The model physical environment consisted of a square 200 × 200 grid of cells divided into 16 neighborhoods. Each neighborhood was characterized by its location on the grid and list of resident agents. In addition, we assigned initial values of neighborhood collective efficacy at baseline in response to the neighborhood's income and violence levels, using an equation calculated from the New York Social Environment Study.[43,44] (For information on the 3 studies we used to calibrate the model and how we measured each agent and neighborhood characteristic and which data source we used to calibrate each characteristic, see Appendices 2 and 3, available as a supplement to this article at http://www.ajph.org.)

Each time step of the model represented 1 year. We ran simulations for 40 years, with the first 10 years discarded as a "burn-in period," during which the agent population accumulated a history of violence and other traumatic experiences but other agent characteristics (e.g., age, income) remained unchanged.

Process Overview and Scheduling

The model proceeded in discrete annual time steps. Within each time step, 7 modules were processed in the following order (a flow-chart demonstrating processes in the model and pseudocode for the model are available in Appendices 4 and 5):

1. aging,
2. resolution of PTSD and income decline from the previous time step,
3. potential victimization and perpetration,
4. actual violent incidents,
5. other traumatic events and development of PTSD,
6. changes in income in response to violence and PTSD, and
7. updates to neighborhood characteristics.

Within each module, we processed agents and neighborhoods in sequential order, except for the occurrence of actual violent incidents, for which we randomly ordered potential perpetrators when seeking potential victims. This random shuffling of potential perpetrators ensured diversity in the pairs of perpetrators and victims who interacted in a completed violent event during the course of the model run.

Design Concepts

The model implemented several hallmark features of agent-based models, including emergence, adaptation, sensing, interaction, stochasticity, and collectives. Specifically, emergence was present, as population levels of violence and PTSD emerged from the behaviors and experiences of the individual agents, which in turn were influenced by the characteristics of their neighborhoods and their interactions with other agents.

Adaptation was modeled, as traumatic event exposure (including violent perpetration, victimization, and other traumatic events) and PTSD, once experienced, increased an agent's probability of future traumatic events and PTSD during subsequent time steps, reflecting vulnerability to revictimization and the strong influence of prior psychological problems on future psychological distress.[45–48]

As for sensing, we assumed that individual agents knew their own characteristics (e.g., age, gender), which influenced their behaviors. They were also assumed to know the characteristics of the neighborhood in which they resided, and agents with the potential to perpetrate violence were able to detect the nearby presence of potential victims.

Interaction was critical to the model dynamics, in that violence occurred in the model through the direct interaction of a potential victim and potential perpetrator in the physical

space. Specifically, each potential perpetrator searched the physical space within a 20-cell radius; any potential victims in that area who had not already been victimized at that time step were then “victimized” by the perpetrating agent with a certain probability, depending on the level of collective efficacy in the neighborhood. Thus, a perpetrator may have had many victims, but each victim only had 1 perpetrator, and some potential victims remained unharmed if not in proximity to a potential assailant or if in a neighborhood with high collective efficacy, which we theorized to protect potential victims from violence through the intervention of potential witnesses.[17] The level of victimization committed in this model thus best represents violent acts committed by strangers, in which few repeat perpetrators commit the majority of violent acts.[49]

We used stochasticity in assigning agent characteristics and behaviors. Specifically, we interpreted all agent demographic and behavioral parameters as probabilities and assigned characteristics and behaviors by drawing a random number between 0 and 1 and comparing the selected number to the agent's calculated probability. As a result, the population composition varied slightly across model runs but population patterns of violence demonstrated expected frequencies and correlates.

Collectives were present in the model in the form of agents grouped together in neighborhoods. We averaged the characteristics of all the agents located within the boundaries of each neighborhood to derive the neighborhood's average level of income and violent victimization.

Finally, to allow observation for model testing, we recorded the values of agent and neighborhood parameters for each unit at each time step. For model analysis, we recorded only population-level variables for each time step (e.g., percentage of agents who were victimized). To account for the stochastic nature of the model, we ran each model scenario 200 times, with the median, 5th percentile, and 95th percentiles reported from across the 200 runs.

Initialization

At initialization, the agent population consisted of 4000 individuals aged 18 years and older with sociodemographic characteristics assigned to match distributions of the adult population in New York City according to the 2000 US Census (for a table specifying the default values of the initialization parameters of the model, see Appendix 6, available as a supplement to this article at http://www.ajph.org).[50] We divided the grid representing the physical space into 16 neighborhoods, and each cell in the grid could be occupied by only 1 agent. Assignment of agent locations and determination of neighborhood boundaries depended on the objectives of the model run with respect to racial and economic residential segregation. We implemented 2 residential segregation scenarios in different model runs: complete segregation of agents by race and income and no racial or economic segregation. To achieve complete segregation by race and income, each of the 16 neighborhoods in the model corresponded to 1 of the 16 possible combinations of race/ethnicity and household income, with only agents assigned that particular combination of race and income residing in that neighborhood. For example, all White agents with an income of $75 000 or more lived in 1 neighborhood, whereas Black agents with an income of $75 000 or more lived in

another neighborhood. The size of the neighborhood was proportionate to the size of the race/income combination in the total population, with the width of the neighborhood on the grid reflecting the racial distribution and the height of the neighborhood on the grid reflecting the income distribution (for a snapshot of the grid, see Appendix 7, available as a supplement to this article at http://www.ajph.org). By contrast, for populations with no racial or economic segregation, we randomly assigned agents to a location on the grid, which was divided into 16 neighborhoods of equal size, producing neighborhoods that each had residents with a mix of race and income characteristics.

Other parameters set at baseline included the magnitude of the neighborhood influence on agent behaviors. Because of previous evidence for the influence of neighborhood characteristics on exposure to violence,[51–54] we allowed 5% of individual agents' probabilities of violent victimization and violent perpetration to be determined by their neighborhood characteristics. We set the radius within which potential perpetrators searched for victims at initialization to 20 cells. To assign baseline levels of collective efficacy to each neighborhood, we aggregated individual collective efficacy ratings from New York Social Environment Study data to the New York City neighborhood (i.e., community district) level.[44,55] Appendix 8, available as a supplement to this article at http://www.ajph.org, describes the equation used to predict neighborhood collective efficacy.)

Finally, we set the probability of a violent act being completed when potential victims were in sufficient proximity to potential perpetrators at 0.70 for high collective efficacy neighborhoods, reflecting estimates of a 30% reduction in violence associated with higher community collective efficacy.[16] By contrast, all interactions between potential perpetrators and potential victims resulted in completed violent acts in low collective efficacy neighborhoods.

The environment did not change during the course of the model run, so the model did not use input data to represent time-varying processes.

An overview of the 7 modules implemented at each time step follows (for the specific data sources and equations we used to calculate behavioral probabilities, see Appendix 9, available as a supplement to this article at http://www.ajph.org):

1. Aging: Following the burn-in period, each agent aged by 1 year at each time step.
2. Resolution of PTSD and income decline from the previous time step: Resolution of PTSD followed an exponential decay function on the basis of patterns of PTSD symptom duration among untreated individuals,[47] with sharp declines in the first year after the development of PTSD and more gradual declines thereafter. For agents who had experienced only violent victimization at the previous time step (and not PTSD), we returned income to its previous category. For agents who had experienced PTSD at the previous time step, we returned income to its previous category only if PTSD had resolved at the current time step.
3. Potential victimization and perpetration: At each time step, each agent had a certain probability of committing a violent act and of being a victim of a violent act. Probabilities of violent perpetration and violent victimization depended on the

individual's age, sex, marital status, education level, household income, prior history of violent perpetration, history of violent victimization, and history of PTSD.[56–59] Although racial inequalities have been noted for both violent victimization and perpetration,[46,60–62] we did not include race/ethnicity as a specific determinant of violence because race/ ethnicity itself does not cause violence.[62] Racial inequalities in outcomes could thus emerge from the model through racial patterning of other risk factors for violence, including income and residential location.

4. Actual violent incidents: After calculating an agent's probability of violent perpetration and victimization, we selected 2 random numbers between 0 and 1. If the selected number was less than the agent's calculated probability of victimization or perpetration, respectively, the agent had the potential to commit or experience violence; whether a violent act actually occurred, however, also depended on a potential victim's exposure to a potential perpetrator, and vice versa. This circumstance captures an often overlooked but fundamental determinant of violence[44] and uses one of the main advantages of agent-based models for studying violence (i.e., the ability to incorporate interactions between individuals).

5. Other traumatic events and development of PTSD: Because PTSD is a strong predictor and outcome of victimization, we also incorporated it as a potential agent outcome in the model.[56–58] Agents who had experienced violent victimization or another traumatic event or who had perpetrated violence at each time step had the potential to develop PTSD at that time step.[46,47,63]

6. Changes in income in response to violence and PTSD: If an agent was a victim of violence, that agent experienced a reduction in income, represented by a drop to the next lowest income category. This 1-year income decline was meant to re-flect the short-term declines in income that may be associated with victimization (e.g., costs associated with physical injury or property damage resulting from violence).[64,65] Furthermore, agents who developed PTSD also experienced a drop in income to the next lowest category, with income returning to its previous level only when PTSD resolved. This reflects the potentially more long-term costs associated with the mental health consequences of violence, including lost wages and reduced productivity and the costs of mental health services.[65]

7. Updates to neighborhood characteristics: At each time step, we recalculated the average levels of income and violent victimization for each neighborhood to account for changes in income and experiences of violence among neighborhood residents. We also recalculated neighborhood collective efficacy to account for changes in neighborhood levels of income and violence.

To calibrate the model, we used an iterative process comparing ABM estimates to empirical data on the prevalence of violent victimization, perpetration, and PTSD; we adjusted parameters (e.g., probabilities of violence) and initial conditions (e.g., radius within which potential perpetrators search for victims) until ABM estimates more closely matched expected estimates on the basis of empirical data.[66]

Simulation Experiments

We ran universal and targeted experimental manipulations of neighborhood collective efficacy with a range of doses (ranging from one half of an SD to a value of 5.0) and 2 alternative durations (1 year and 30 years), producing a range of experimental effects. To assess the role of fundamental causes in the experiments, we repeated each experiment in a context of no racial residential segregation and a context of complete racial residential segregation. We also undertook sensitivity analyses to test the robustness of the results to the initial conditions of the model and to evaluate the effects of alternate interventions and of interventions conducted in the context of alternate segregation scenarios, thereby ensuring that our primary results reflected overall patterns in the simulation results.

The first series of experiments designed to reduce violent victimization were aimed at all neighborhoods in the model and are thus termed “universal” experiments. We first assigned baseline levels of neighborhood collective ef-ficacy on the basis of neighborhood income and violence; under the universal experiment, we increased neighborhood collective efficacy by a set amount for all neighborhoods, ranging from one half of an SE (0.14) to the maximum possible value of 5.0. We ran models with a 1-year duration of experiment, with neighborhood collective efficacy remaining at the experiment levels for 1 time step and then changing according to changes in neighborhood income and violence. We also repeated models with a 30-year experiment, in which experiment levels of neighborhood collective efficacy remained in effect throughout the entire model run.

The second series of experiments were targeted to high-violence neighborhoods only—these were the neighborhoods with above average levels of violent victimization at each time step. As in the universal experiment, we assigned baseline levels of neighborhood collective efficacy; then high-violence neighborhoods experienced an increase in collective efficacy by a set amount for either a 1-year or 30-year duration.

We conducted a series of sensitivity analyses to check the robustness of the model results to alternate specifications of segregation, intervention conditions, and initial conditions (see Appendix 11, available as a supplement to this article at http://www.ajph.org).

RESULTS

We successfully calibrated the ABM so that the estimates of violent victimization, violent perpetration, and PTSD the model produced were consistent with previously published estimates and estimates from a New York City population (for a table contrasting the published and model estimates, see Appendix 10, available as a supplement to this article at http://www.ajph.org). On average, 3.8% of the agent population experienced violent victimization each year, whereas 28.2% of agents were victims of violence at least once in the course of the model run. A smaller proportion of agents perpetrated violence each year (0.85%), with 14.0% committing a violent act against another agent at least once during the model run. Although race/ethnicity was not explicitly used in determining probabilities of violence and PTSD, racial inequalities emerged from the model run, as in reality, with Black agents exhibiting higher levels of annual and lifetime violent victimization, perpetration, and PTSD.

Population-Level Rates of Violent Victimization

Figure 1 presents estimates of annual violent victimization for different levels and durations of universal and targeted experiments increasing neighborhood collective efficacy, in an agent population completely segregated by race and income as well as an agent population with no racial or economic segregation. Specifically, we compared violence in populations in which we did not implement any experiment (i.e., we assigned neighborhood collective efficacy at baseline and changed it in response to changes in neighborhood levels of violence and income) with violence in populations in which neighborhood collective efficacy was artificially increased by 0.5 SDs to the maximum level, either in all neighborhoods in the model (i.e., universal experiment) or only in the highest violence neighborhoods (i.e., targeted experiment).

We repeated model runs with the experiment lasting for 1 year and for 30 years (i.e., the duration of the model run). Both universal and targeted experiments successfully reduced annual violent victimization in the population in all scenarios. In 1-year experiments (Figure 1a and c), there was a successive reduction in violent victimization for every 0.5 SD increase in neighborhood collective efficacy. Thirty-year experiments (Figure 1b and d) produced a substantial decrease in victimization, compared with the no experiment scenario, of a similar magnitude across levels of the collective efficacy experiment.

At all levels of collective efficacy, a universal increase of collective efficacy resulted in a lower prevalence of victimization than did targeted increases in collective efficacy. It was necessary to increase collective efficacy to the maximum value in high-violence neighborhoods to exert a larger effect than that exerted by a small universal increase of collective efficacy. We found comparable effects in a context of no segregation.

Racial/Ethnic Inequalities in Violent Victimization

Figure 2 presents race-specific estimates of annual violent victimization for different levels and durations of universal and targeted experiments in agent populations completely segregated by race and income and with no racial or economic segregation. Although both universal and targeted collective efficacy experiments reduced average levels of violent victimization among both Blacks and Whites, in populations in which race and income segregated agents, a consistently higher proportion of Black agents experienced victimization in all models (Figure 2a and b). Racial inequalities in violent victimization in the segregated context remained largely unchanged by the experiments. On average, we found a 1.4% difference between Blacks and Whites in victimization (95% confidence interval [CI] = 0.6, 2.4) under no intervention. When we implemented 1-year universal neighborhood collective efficacy experiments, the difference ranged between 1.5% (95% CI = 0.7%, 2.3%) and 1.6% (95% CI = 0.6%, 2.5%), whereas 1-year targeted experiments resulted in a difference ranging from 1.3% (95% CI = 0.5%, 2.1%) to 1.5% (95% CI = 0.6%, 2.4%; Figure 3a).

In populations with no segregation, levels of victimization were closer for Blacks and Whites, and experiments had a greater impact on Blacks than in segregated populations (Figure 2c and d). Under no intervention, Blacks and Whites differed by 0.6% in

victimization (95% CI = 0.1%, 1.1%; Figure 3c and d). When we implemented 1-year universal neighborhood collective efficacy experiments, the difference ranged from of 0.5% (95% CI = 0.1%, 1.0%) to 0.6% (95% CI 0.1%, 1.1%), whereas 1-year targeted experiments also resulted in a difference of 0.6% (95% CI = 0.0%, 1.2%).

Figure 4 shows the percentage reduction in average annual violent victimization overall and among Blacks and Whites, by level of neighborhood collective efficacy increase for both universal and targeted experiments, compared with models in which we did not implement any experiment. All experiments produced a reduction in violent victimization, with increasing reductions associated with successive increases in neighborhood collective efficacy and larger reductions produced by universal versus targeted experiments. However, in populations segregated by race and income (Figure 4a and b), the benefits of experiments accrued disproportionately to White agents, who experienced substantially larger reductions in violent victimization than did Black agents. For example, annual violent victimization was reduced by 24.4% among White agents when neighborhood collective efficacy was increased to the maximum for all neighborhoods versus a reduction of only 14.0% for Black agents. However, in populations with no racial or economic segregation (Figure 4c and d), violent victimization was reduced similarly for both Black and White agents.

(Results of sensitivity analyses are available in Appendices 11–15, as a supplement to this article at http://www.ajph.org.) The pattern of findings remained the same under different segregation and intervention scenarios as well as under alternative assumptions about the influence of neighborhood conditions.

DISCUSSION

Using a simulation, we found that universal interventions that increased collective efficacy by a small amount for the entire population had the same or larger effect on victimization than did experiments that selectively increased collective efficacy by a large amount in high-risk neighborhoods. However, neither universal nor targeted experiments reduced racial inequalities in victimization under situations of complete segregation. In such contexts, experiments benefited Whites more than Blacks, preserving racial inequalities in victimization. Addressing the structural drivers of risk achieved the largest impact on inequalities—that is, by eliminating racial residential segregation.

Our findings provide an empirical test of Rose's ideas about a population strategy of prevention. Consistent with his predictions, a small shift in collective efficacy across all neighborhoods resulted in the same or greater reduction in victimization than did a targeted shift in high-violence neighborhoods.[1,2] This suggests that although the risk of violence involvement is highest among neighborhoods with high rates of violence, it is the large number of neighborhoods with modestly elevated rates of violence that contribute the largest proportion of victimization cases. Prevention strategies directed at all neighborhoods (i.e., universal, population-based strategies) may thus be more effective in reducing the overall amount of violent events in a population than are strategies aimed at the small fraction of historically violent neighborhoods (i.e., targeted strategies).[67] Previous evaluations of the impact of universal versus targeted strategies on health have focused on individual-level

interventions[3,15]; we have advanced the literature on prevention policy by focusing on contextual interventions that are carried out at the neighborhood level.

However, although universal interventions may effectively address population-level rates of health, our study suggests that it may not be possible for interventions to address racial/ethnic inequalities in health without first addressing their fundamental causes. Consistent with the fundamental causes of health perspective,[5,10–12] the association between race/ethnicity and victimization persisted despite experimental manipulations of neighborhood collective efficacy. Because of residential segregation, race/ethnicity embodies an array of economic resources that define health no matter what intervening social intervention is enacted.[37] In this case, Blacks were concentrated in more economically disadvantaged neighborhoods, where temporary increases in collective efficacy (and thus temporary decreases in victimization) decayed over time because of the persistent unaddressed levels of neighborhood disadvantage. It was necessary to first address such unequal distribution of racial/ethnic groups across neighborhoods to ensure that Blacks and Whites experienced a comparable benefit from a collective efficacy experiment.

These results illustrate the tight link between social processes such as collective efficacy and neighborhood residential segregation and suggest that current collective efficacy interventions[22–26] that attempt to increase collective efficacy while leaving patterns of residential segregation in place will have a limited impact on racial/ethnic inequalities in population health. Instead, for public health policy to both improve population health and reduce health inequalities, a combined approach is advisable. This involves joint investment in policies that encourage public health advances (e.g., universal neighborhood-level violence prevention interventions) and policies that weaken the link between public health innovations and socioeconomic resources (e.g., policies that reduce resource inequalities, including tax policies, regulation of lending practices, fair housing policies, or college admissions policies).[12]

We have illustrated the contributions that simulation approaches such as ABM can make to conducting virtual experiments. ABM allowed us to answer questions about community-level experiments that would have been difficult to answer using real-life social experiments. That is, through simulations, we were able to enact a series of counterfactual experiments, reflecting different doses of collective efficacy, at different durations, administered to different targets (i.e., universal vs targeted), and assuming different patterns of racial and economic residential segregation. By simulating counter-factuals, we were also able to decouple race/ ethnicity from socioeconomic status and assess the impact that neighborhood dynamics and neighborhood experiments have on racial/ethnic inequalities in victimization. Because of systematic individual selection into neighborhoods by race/ethnicity and socioeconomic status, that would not have been possible in observational studies.[68,69]

Limitations

Our conclusions should be considered with the following limitations. First, we did not consider the role of adverse experimental effects or costs on our outcomes of interest. Prior studies suggest that assumptions about intervention costs and potential adverse effects can

influence the relative effectiveness of targeted versus universal interventions.[3,15] Future studies of neighborhood-level experiments need to incorporate data on cost and adverse effects into the calculation of experimental outcomes. Second, our agents reflected the composition of New York City neighborhoods, so generalizability beyond comparably multi-ethnic urban areas is limited.

Third, because of our lack of New York City–specific measures of violent perpetration and our consequent inability to link New York City neighborhood data with measures of perpetration, we used information on the relationship between neighborhood characteristics and the risk of victimization to estimate the relationship between neighborhood characteristics and the risk of perpetration. To the extent that these 2 relationships differ, this could have affected our findings on the neighborhood experiments. However, the close match between empirical data on perpetration and the perpetration prevalence estimates that emerged from our ABM allay this concern. Fourth, the validity of an ABM is contingent on the quality of data used to inform the parameterization. Extensive calibration of the model helped us ensure that it reflected known distributions before experiments were simulated.

Fifth, to develop an interpretable model, and because of data limitations, the model required a set of simplifying assumptions, including specification of restricted mechanisms through which neighborhood interventions could in-fluence agent behaviors, and the use of a simplified set of situational determinants of violence that did not include factors such as nature of the violent act or type of weapon. Our intention was not to present a full representation of the processes that create racial/ ethnic differentials in victimization but to explore specific interactions between key neighborhood and individual-level processes hypothesized in the literature and to evaluate results using different scenarios. Finally, we limited our experimental manipulations to a single intervention increasing collective efficacy. Combinations of interventions, including hybrid strategies that incorporate universal and targeted interventions, may be more effective at reducing population levels and inequalities in violent victimization.

Conclusions

We presented a quantitative simulation method to compare universal and targeted contextual interventions and to test the implications of fundamental cause theory for prevention policy. Our methods build on Rose's work on prevention policies and on Link and Phelan's work on fundamental causes of health.[1,2,4] Although universal interventions may produce the largest effects on population health, our findings suggest that it may not be possible to address racial/ethnic inequalities in health without first addressing the fundamental causes of such inequalities. Simulations such as ours hold promise for helping public health policymakers evaluate potential intervention strategies from the perspective of population health and health inequalities.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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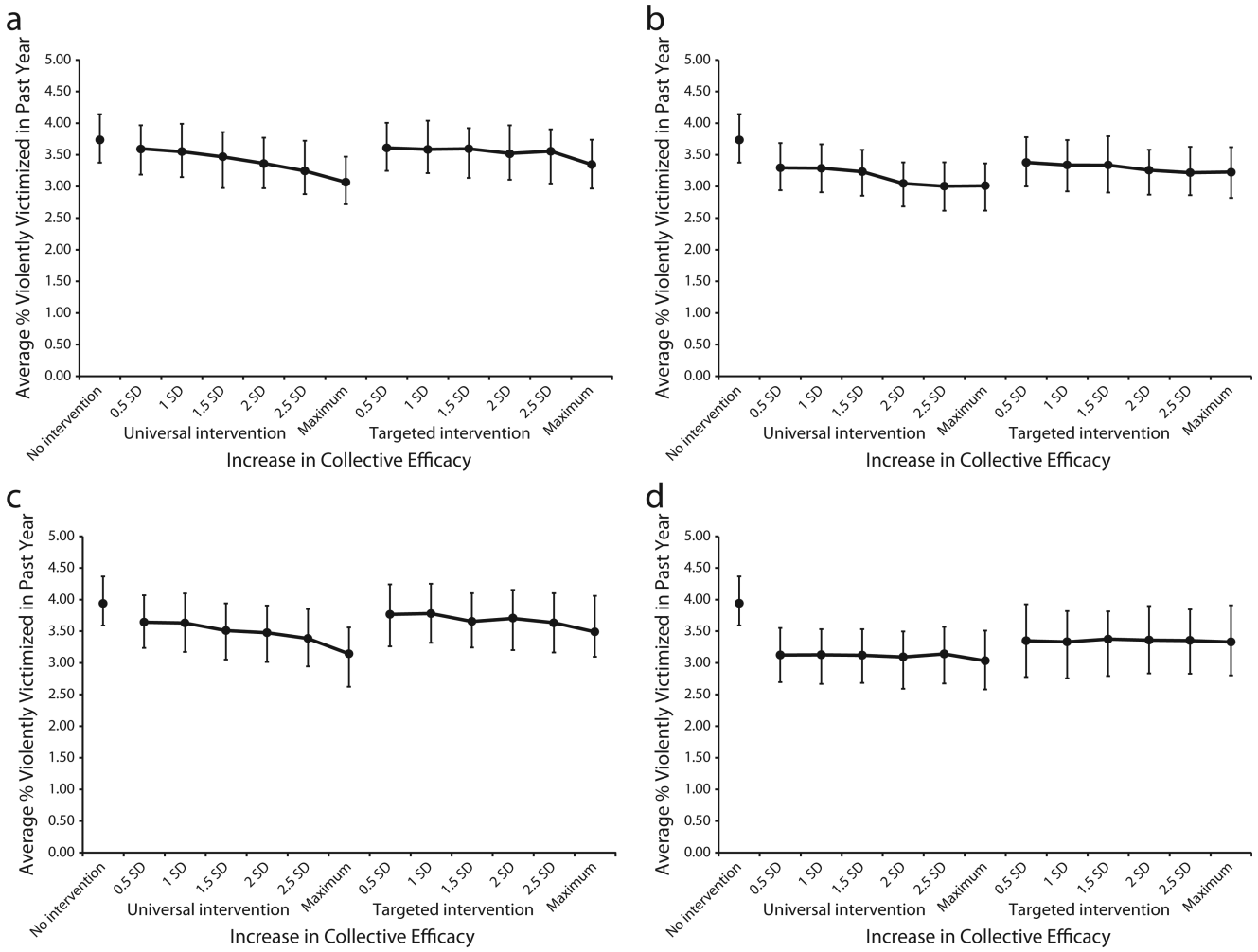
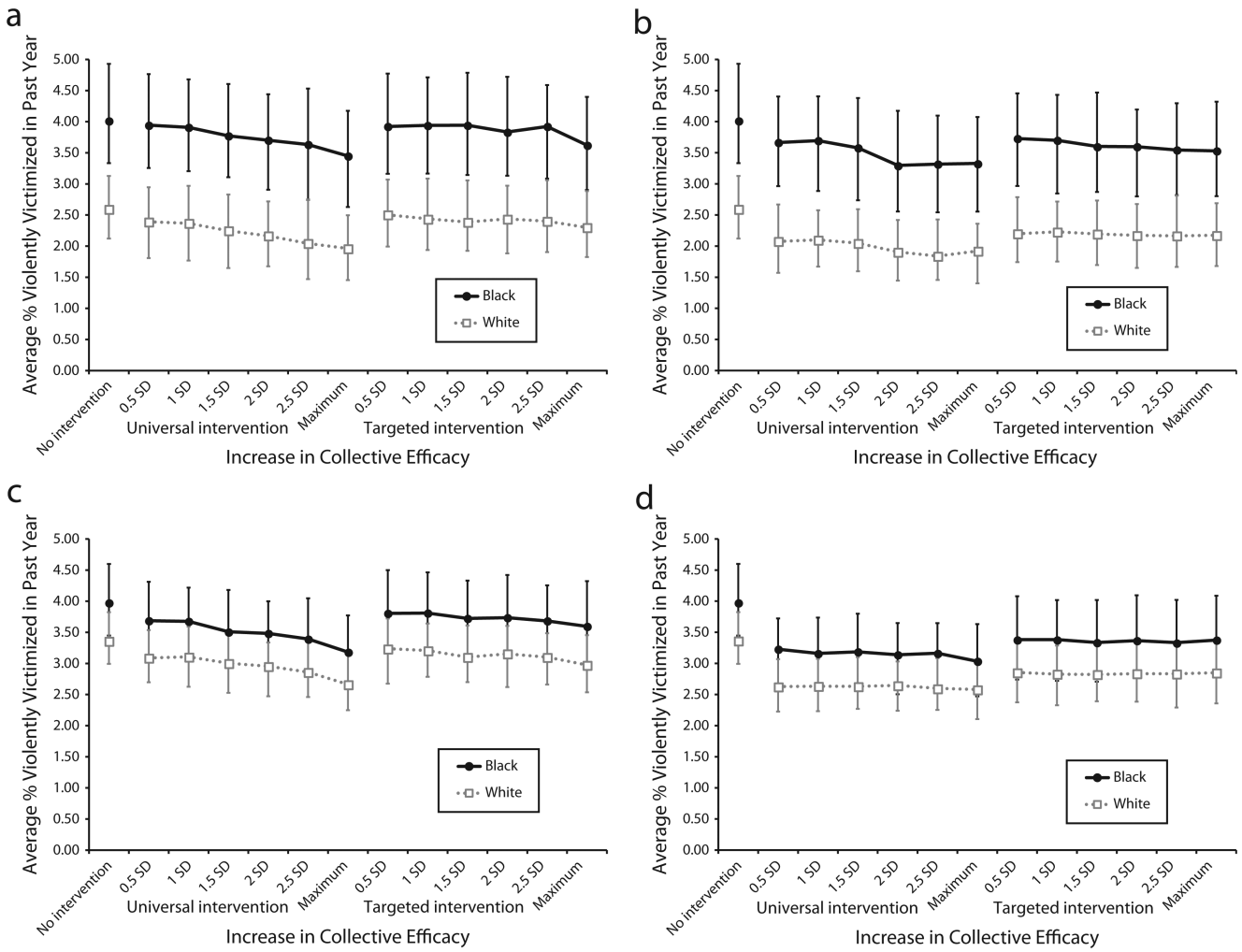


Note. Whiskers indicate 95% confidence intervals.

FIGURE 1.

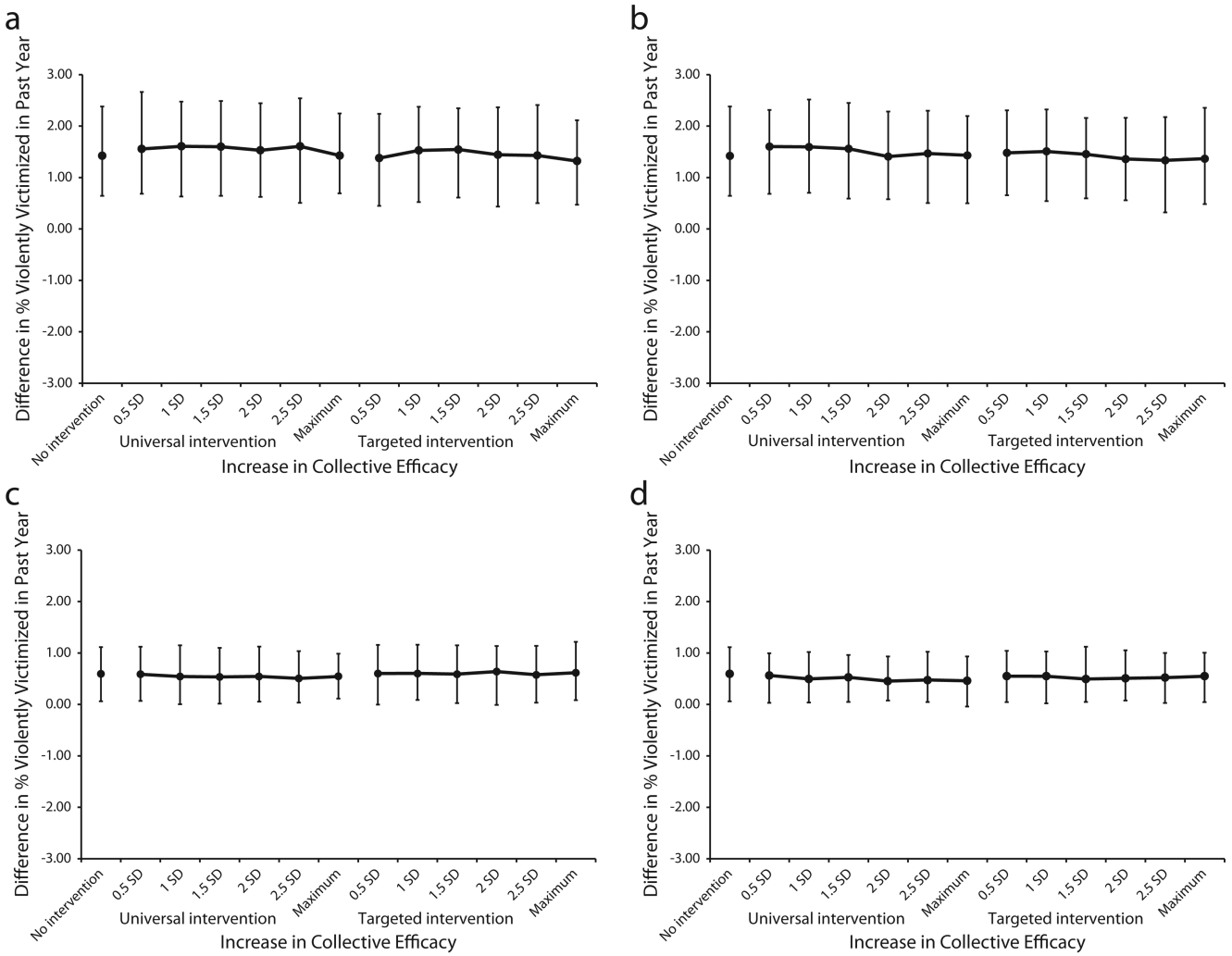
Estimates of annual violent victimization comparing universal and targeted neighborhood collective efficacy interventions with (a) 1-year duration segregated by race and income, (b) 30-year duration segregated by race and income, (c) 1-year duration assigned to random locations, and (d) 30-year duration assigned to random locations.



Note. Whiskers indicate 95% confidence intervals.

FIGURE 2.

Estimates of absolute difference in annual violent victimization between Blacks and Whites comparing universal and targeted neighborhood collective efficacy interventions with (a) 1-year duration segregated by race and income, (b) 30-year duration segregated by race and income, (c) 1-year duration assigned to random locations, and (d) 30-year duration assigned to random locations.



Note. Whiskers indicate 95% confidence intervals.

FIGURE 3.

Estimates of annual violent victimization among Blacks and Whites, comparing universal and targeted neighborhood collective efficacy interventions with (a) 1-year duration segregated by race and income, (b) 30-year duration segregated by race and income, (c) 1-year duration assigned to random locations, and (d) 30-year duration assigned to random locations.

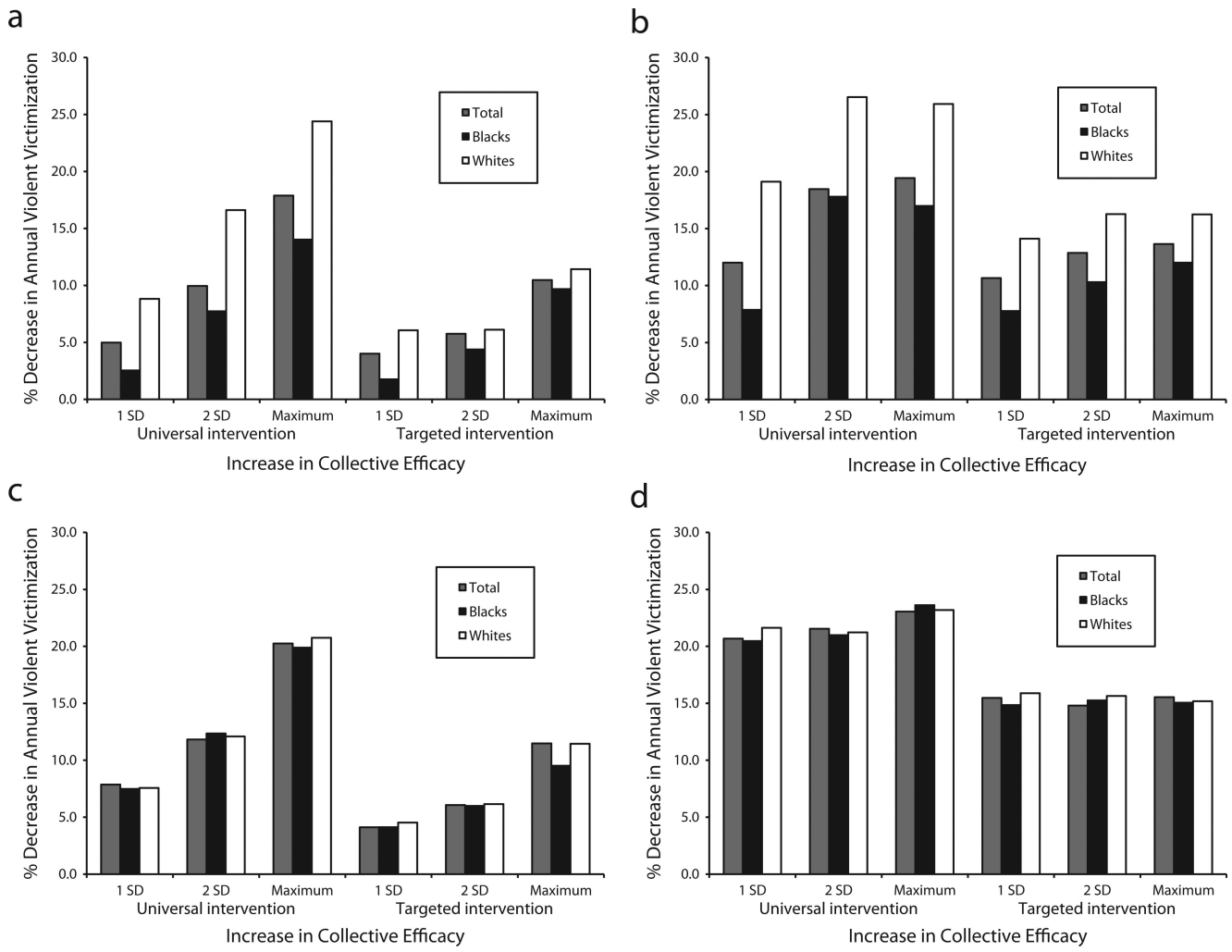


FIGURE 4.

Estimates of percentage reduction in annual violent victimization, overall and among Blacks and Whites, by increase in collective efficacy, comparing universal and targeted neighborhood collective efficacy interventions with (a) 1-year duration segregated by race and income, (b) 30-year duration segregated by race and income, (c) 1-year duration assigned to random locations, and (d) 30-year duration assigned to random locations.