Title

Examining Neighborhood Socioeconomic Status as a Mediator of Racial/Ethnic Disparities in Hypertension Control Across Two San Francisco Health Systems.

Journal

Circulation. Cardiovascular quality and outcomes, 15(2)

ISSN

1941-7713

Authors

Liu, Emily F
Rubinsky, Anna D
Pacca, Lucia
et al.

Publication Date

2022-02-01

DOI

10.1161/circoutcomes.121.008256

Peer reviewed

HHS Public Access

Author manuscript

Circ Cardiovasc Qual Outcomes. Author manuscript; available in PMC 2023 February 01.

Published in final edited form as:

Circ Cardiovasc Qual Outcomes. 2022 February ; 15(2): e008256. doi:10.1161/CIRCOUTCOMES.121.008256.

Examining Neighborhood Socioeconomic Status as a Mediator of Racial/Ethnic Disparities in Hypertension Control Across Two San Francisco Health Systems

Emily F. Liu, MPH[1], **Anna D. Rubinsky, PhD**[2], **Lucia Pacca, PhD**[3,4], **Mahasin Mujahid, PhD, MS**[1], **Valy Fontil, MD, MPH, MAS**[3,4], **Mindy C. DeRouen, PhD, MPH**[2,5], **Jessica Fields, BA**[2,3,4], **Kirsten Bibbins-Domingo, PhD, MD, MAS**[2,3,4], **Courtney R. Lyles, PhD**[2,3,4]

[1]School of Public Health, University of California, Berkeley, Berkeley, CA, United States

[2]Department of Epidemiology and Biostatistics, University of California, San Francisco, San Francisco, CA United States

[3]UCSF Center for Vulnerable Populations, Zuckerberg San Francisco General Hospital, San Francisco, CA, United States

[4]Division of General Internal Medicine at Zuckerberg San Francisco General Hospital, University of California, San Francisco, San Francisco, CA, United States

[5]Helen Diller Family Comprehensive Cancer Center, University of California, San Francisco, San Francisco, CA, United States

Abstract

Background.—A contextual understanding of hypertension control can inform population health management strategies to mitigate cardiovascular disease events. This retrospective cohort study links neighborhood-level data with patients' health records to describe racial/ethnic differences in uncontrolled hypertension and determine if and to what extent these differences are mediated by nSES.

Methods.—We conducted a mediation analysis using a sample of patients with hypertension from two healthcare delivery systems in San Francisco over two years (n = 47,031). We used generalized structural equation modeling, adjusted for age, sex, and healthcare system, to estimate the contribution of nSES to *disparities* in uncontrolled hypertension between White patients and Black, Hispanic/Latino, and Asian patients, respectively. Sensitivity analysis removed adjustment for healthcare system.

Results.—Over half the cohort (62%) experienced uncontrolled hypertension during the study period. Racial/ethnic groups showed substantial differences in prevalence of uncontrolled hypertension and distribution of nSES quintiles. Compared to White patients, Black and Hispanic/Latino patients had higher adjusted odds of uncontrolled hypertension: OR = 1.79, 95% CI: 1.67,

Corresponding Author: Courtney R. Lyles, PhD, Address: 1001 Potrero Avenue, Building 10, Room 1320B, UCSF Box 1364, San Francisco, CA 94143, Phone Number: (628) 206-6483, Courtney.Lyles@ucsf.edu.

Disclosures

None.

1.91 and OR = 1.38, 95% CI: 1.29, 1.47, respectively, and nSES accounted for 7% of the disparity in both comparisons. Asian patients had slightly lower adjusted odds of uncontrolled hypertension when compared to White patients: OR = 0.95, 95% CI: 0.89, 0.99, and the mediating effect of nSES did not change the direction of the relationship. Sensitivity analysis increased the proportion mediated by nSES to 11% between Black and White patients and 13% between Hispanic/Latino and White patients, but did not influence differences between Asian and White patients.

Conclusions.—Among patients with hypertension in this study, nSES mediated a small proportion of racial/ethnic disparities in uncontrolled hypertension. Population health management strategies may be most effective by focusing on additional structural and interpersonal pathways such as racism and discrimination in health care settings.

Keywords

Hypertension; Health Status Disparities; Population Health Management; Ethnic Groups; Social Environment

Introduction

Cardiovascular disease (CVD) is the leading cause of death[1] and a primary source of differences in life expectancy between Black and White individuals in the United States (US).[2] These differences have persisted despite the overall decline in the US death rate from CVD in the last decade, with Black individuals bearing a disproportionate burden of CVD-related risk factors and mortality.[3] Given the long-term health and economic impact of CVD on individuals and communities,[4–6] it is a public health priority to further our understanding of the sources of these racial/ethnic disparities to improve population health. Novel approaches that integrate neighborhood-level determinants with patient-level data[7, 8] and apply analytic methods from social epidemiology can help to develop targeted interventions that address social determinants of health.[9, 10]

Hypertension control is a primary strategy in preventing CVD events,[11] underscored by its addition to Healthy People 2030 as a leading health indicator.[12] Healthy People 2030, the health prevention agenda for the US federal government, set a target to increase the proportion of adults with controlled hypertension to 60.8%.[12] In 2015–2016, only 48.3% of hypertensive adults had their blood pressure under control, and White individuals had a higher prevalence of hypertension control (50.8%) compared to Black (44.6%) and Asian (37.4%) individuals.[13] The AHA and ACC clinical practice guidelines focus on the individual-level mechanisms of hypertension,[11] but the few studies that examine racial/ethnic disparities in hypertension control have found that differences between Black and White individuals and between Hispanic/Latino and White individuals are not fully attenuated after adjusting for individual demographics and health behaviors.[14,15] Given that differences persist, broader contextual factors may serve as a root cause of these disparities.

In the United States, health-promoting resources and environments are differentially distributed among racial/ethnic groups as a consequence of racial residential segregation and past and present disinvestment in neighborhoods where racial/ethnic minorities reside.[16] These physical and social environments have been found to influence cardiovascular risk

and outcomes such as ideal cardiovascular health,[17] body mass index,[18] and hypertension prevalence.[19] Neighborhood socioeconomic status (nSES), an index comprising measures of multiple domains of the social environment (income, education, and occupation), could be an important intermediate step along the pathway between race/ethnicity and hypertension control that helps to explain these persistent disparities in cardiovascular health. Therefore, there is a need to utilize multiple sources of validated data (e.g. electronic health record data on hypertension control from diverse health care settings combined with nSES index) to better understand this pathway and more effectively support health promotion. A contextual understanding of hypertension control has the potential to scaffold the use of clinical practice guidelines and inform population health management strategies to mitigate CVD events. This study aimed to accomplish this goal by examining racial/ethnic differences in uncontrolled hypertension across two different health systems in San Francisco and determining if and to what extent these differences are mediated by nSES.

Methods

Data Availability

The patient data for this study is protected health information and unavailable to be shared. The data used to develop the neighborhood socioeconomic index is publicly available from the United States Census Bureau and can be accessed at data.census.gov. The code for the analysis is available from the corresponding author upon reasonable request.

Study Sample

Our study population consisted of patients aged 18 years and older who received outpatient care from two large healthcare systems in San Francisco [University of California San Francisco (UCSF) Health and the San Francisco Health Network (SFHN)] over a two year period between January 1, 2015 and December 31, 2017, and who had a hypertension diagnosis made at any time before the start of the observation period and a home address in the San Francisco Bay Area documented in the electronic health record (EHR). Patients were included if they had at least two outpatient blood pressure measurements over the two-year study period. Those with documented self-identified race/ethnicity other than the four largest categories of White, Black, Asian, or Hispanic/Latino were excluded from this analysis in which race/ethnicity was the primary variable of interest.

Data Sources

Using electronic health record (EHR) data, we extracted patient cohorts based on the same definitions across both healthcare systems. UCSF Health is an academic medical center and integrated healthcare network in the San Francisco Bay Area that sees 45,000 hospital admissions and 1.7 million outpatient visits annually. SFHN is the public healthcare system for the City and County of San Francisco that provides safety-net services for low-income residents primarily on Medicaid. We identified patients with hypertension based on ICD-9-CM or ICD-10-CM diagnoses documented prior to or during the study period and ascertained patients' most recent home addresses captured within the EHR. Patients with addresses outside the 9-county Bay Area were excluded. We then geocoded patient addresses to latitude and longitude using ArcGIS Business Analyst software version

2016 (Esri) and assigned patients to 2010 US Census tracts based on these geographic coordinates.

This multi-campus study was approved by the University of California (UC) Institutional Review Board (IRB) Reliance Registry, with UC San Francisco serving as the reviewing IRB and UC Berkeley serving as the relying IRB. The UC San Francisco IRB determined that no informed consent was required from participants in the sample.

Study Variables

Primary independent variable: Patient race/ethnicity (White non-Hispanic, Black non-Hispanic, Hispanic/Latino, Asian non-Hispanic) was based on self-identified race/ethnicity documented in the EHR. While the EHR collected information for other racial/ethnic groups, including American Indian or Alaska Native (n = 63), Multi-race (n = 147), Native Hawaiian or Other Pacific Islander (n = 532), and Other (n = 3290), these groups were excluded from the analysis to mitigate unequal group sizes and align with racial/ethnic groups in prior literature.

Primary Outcome: Uncontrolled hypertension was defined as a mean systolic blood pressure greater than or equal to 140 mm Hg or a mean diastolic blood pressure greater than or equal to 90 mm Hg based on a moving average across any two-consecutive outpatient visits[11, 20] during the two-year study period. We assessed uncontrolled hypertension as a dichotomous variable using the pre-2017 ACC/AHA blood pressure guidelines for hypertension (≥140/90 mm Hg) to coincide with care targets during the study period.[5]

Mediator: We examined nSES as the primary potential mediator, using a validated index score constructed from seven neighborhood-level indicators representing census tract-level education, income/wealth, employment, and housing from the 2013–2017 American Community Survey (ACS) 5-year estimates.[21] The ACS data were concurrent to the study period and aggregated information by the most recent geographic boundaries in its last year of the estimate period; in 2017, census tracts had last been updated in the 2010 census.[22] We used census tract-level scores of nSES, as census tracts approximate neighborhoods of roughly 4,000 residents and are known to be more stable place-based indicators than zip codes and better able to capture place-based social inequalities.[23, 24] Scores of nSES (with higher scores indicating higher nSES) were categorized into quintiles based on their distribution across census tracts in the San Francisco Bay Area (defined as including 9 counties in this California region: Alameda, Contra Costa, Marin, Napa, San Francisco, San Mateo, Santa Clara, Solano, and Sonoma).

Covariates: We examined race/ethnicity as a socially-defined construct that encapsulates institutionalized, personally mediated, and internalized racial bias or discrimination.[25] As such, we interpreted racial disparities in comorbidities and antihypertensive treatment to indicate that these factors were potential mediators – rather than confounders – of the relationship of interest.[26–30] We decided a priori that adjustment for these mediators was not appropriate in our analysis because we conceptualized racism and associated racial discrimination to lead to differences in these factors, which could, in turn, lead to differences

in hypertension control. We used a directed acyclic graph to identify the minimum set of confounders to include age and self-reported sex assigned at birth, both from the EHR of each healthcare system (Figure 1).

Statistical Analysis

We examined the distribution of all study variables in the overall cohort – comprised of patients from both healthcare systems – and the distribution of the outcome and potential mediator by racial/ethnic group. Differences across groups were assessed using chi-squared tests.

This mediation analysis estimated the contribution of nSES to *disparities* in uncontrolled hypertension between each racial/ethnic minority patient group and White patients. We used generalized structural equation modeling (GSEM) with a 100-replication bootstrapping. Our GSEM model simultaneously fit (1) ordinal logistic regression estimating the effect of race/ethnicity on nSES and (2) logistic regression estimating the effect of nSES on uncontrolled hypertension. Both fitted regression equations in the GSEM model were adjusted for age, self-reported sex assigned at birth, and healthcare system, and clustered on census tract to account for geographic correlation. Models were adjusted for confounders and variables associated with the outcome, but not on the causal pathway (i.e. precision variables). The results of the GSEM model were used to calculate the odds ratio (OR) estimates of the direct effect of race/ethnicity on uncontrolled hypertension independent of nSES (i.e. OR of uncontrolled hypertension for each racial/ethnic minority group versus White patients, when nSES and other covariates are held constant), the natural indirect effect due to patients' nSES (i.e. the mediational path in which race/ethnicity leads to differences in uncontrolled hypertension through nSES), and the total effect (i.e. racial disparity in uncontrolled hypertension after adjusting for covariates but not adjusting for nSES). Finally, the proportion mediated by nSES was found by dividing the indirect effect by the total effect.

We hypothesized that adjusting for healthcare system in our GSEM model would attenuate the mediating effect of nSES. While it was included to address practice variation between the two healthcare systems, UCSF Health and SFHN fundamentally serve two different populations and adjusting for healthcare system artificially constrains the true underlying racial/ethnic disparity in uncontrolled hypertension as well as the distribution of nSES. Therefore, we conducted a sensitivity analysis and removed the fixed effect for healthcare system.

Results were considered statistically significant at a p-value ≤ 0.05. All statistical analyses were performed in Stata 16 software.[31]

Results

Among the 97,205 patients with hypertension and a home address documented in the EHR, we identified 60,030 patients with at least two outpatient BP measurements during the study period. We excluded 8,333 patients who did not reside in the San Francisco Bay Area and 4,666 patients who did not identify as White, Black, Asian, Hispanic/Latino, or missing

race/ethnicity information. We had a final analytic sample of 47,031 patients within 1,485 census tracts with an average of 32 patients per tract (Figure 2).

Over the two-year study period, 62% of the cohort experienced uncontrolled hypertension (Table 1). Patients were primarily between 35 and 64 years old (47%) and White (40%). Thirty percent of the sample consisted of Asian patients, followed by Black (16%) and Hispanic/Latino (15%) patients. Sex assigned at birth was evenly distributed between males and females.

Black patients had the highest prevalence of uncontrolled hypertension (72%) compared to all other racial/ethnic groups (Table 1). Hispanic/Latino patients had the second highest prevalence of uncontrolled hypertension (67%), whereas Asian patients and White patients had similar lower prevalence (57%). White patients were much less likely to live in lower nSES neighborhoods (26% in the two lowest quintiles) than other racial/ethnic groups, particularly compared to Black (69%) and Hispanic/Latino (59%) patients (Table 1). More than 50% of White patients resided in the highest nSES neighborhoods, and White patients were two to three times more prevalent in the highest nSES quintile compared to other racial/ethnic groups.

Overall, the direct effects (independent of nSES) accounted for the majority of the total effect between race/ethnicity and uncontrolled hypertension. Compared to White patients, Black and Hispanic/Latino patients had higher odds of uncontrolled hypertension when adjusting for age, sex, and healthcare system: OR = 1.79, 95% CI: 1.67, 1.91 and OR = 1.38, 95% CI: 1.29, 1.47, respectively (total effect, Table 2). After additional adjustment for nSES, racial/ethnic differences in uncontrolled hypertension were only mildly attenuated for Black and Hispanic/Latino patients (direct effect, Table 2). Nonetheless, we found that nSES accounted for 7% of the disparity in uncontrolled hypertension between Black and White patients and between Hispanic/Latino and White patients.

In contrast, Asian patients had slightly lower odds of uncontrolled hypertension when adjusting for age, sex, and healthcare system when compared to White patients: OR = 0.95, 95% CI: 0.89, 0.99 (total effect, Table 2), and after additional adjustment for nSES, racial/ethnic differences in uncontrolled hypertension increased in magnitude in the same direction (direct effect, Table 2). While we found a statistically significant mediating effect of nSES, it was not strong enough to shift the directionality of the differences in uncontrolled hypertension between Asian and White patients.

As we hypothesized, the mediating effect of nSES on differences in uncontrolled hypertension increased for all three racial/ethnic comparisons when the fixed effect for healthcare system was removed in our sensitivity analysis. The proportion mediated by nSES increased to 11% between Black and White patients and 13% between Hispanic/Latino and White patients. In the comparison between Asian and White patients, the mediating effect of nSES became strong enough to offset the direct effect and render the total effect of differences in uncontrolled hypertension null (Table 2).

Discussion

The aim of this study was to describe racial/ethnic differences in uncontrolled hypertension and to understand if and to what extent neighborhood socioeconomic status mediates these differences. We found statistically significant differences in uncontrolled hypertension across all three racial/ethnic comparisons, and nSES mediated 7% of differences between Black and White patients and between Hispanic/Latino and White patients. No meaningful mediation effect was found between Asian and White patients. In our sensitivity analysis after removing adjustment for healthcare system, the mediating effect of nSES on uncontrolled hypertension increased to 11% for Black-White differences and 13% for Hispanic/Latino-White differences. Adjusting for healthcare system to address practice variation between the two patient populations may have also artificially constrained variation in the distribution of racial/ethnic groups and nSES and attenuated the true mediating effect of nSES on uncontrolled hypertension.

There is only a small set of studies that have examined the effects of neighborhood environment on racial/ethnic health disparities in cardiometabolic risk factors and outcomes, and few investigating its role as a mediator. Among these studies, neighborhood factors accounted for reductions ranging from 9% to 29% in racial/ethnic differences depending on the outcome under consideration.[17–19, 32] In the single study focused on uncontrolled hypertension as the outcome, Morenoff et al. found that, among 569 individuals in Chicago with hypertension who reported taking antihypertensive medication, adjustment for neighborhood sociodemographic structure (including socioeconomic status, racial/ethnic composition and age composition) did not change Black-White differences in hypertension control.[15] In contrast to Morenoff et al., we have much larger sample size and did not adjust or stratify for medication status. In contrast, we found that nSES alone played some mediating role, albeit relatively small, in observed Black-White and Hispanic/Latino-White differences in uncontrolled hypertension. We also observed that the mediating effect of nSES was not consistent across racial/ethnic comparisons, suggesting that the mediator role of neighborhood environment may differ across racial/ethnic groups.[33]

The interpretation of our results requires several considerations. Neighborhoods have the potential to influence health and well-being through concurrent access to health care resources and health-promoting physical and social environments. For example, previous literature has shown that lower-income neighborhoods are associated with poorer medication adherence, a factor that contributes to poor hypertension control and may partially explain racial differences in blood pressure control.[34, 35] This study used patients' most recent home address to determine neighborhood membership, but this methodology does not allow insight into the longitudinal effects of neighborhoods. Further studies should explore the patients' residential history when available and corresponding changes in nSES over time. The San Francisco Bay Area is characterized by high rent, gentrification, and displacement, so while we saw only modest mediation by nSES in our results, we do not discount the possibility that the effect is attenuated by changes in the duration of exposure to neighborhood environment.[36]

Moreover, racial/ethnic differences in cardiovascular health, including hypertension control, may be attributed to both the acute and cumulative interaction of multi-level and multi-factorial causes.[37] Neighborhood environment is only one potential pathway through which the social determinants of health may manifest. Thus, it is unlikely that neighborhood environment would fully mediate racial/ethnic differences. It is possible that the mechanisms reflected in nSES were not as meaningful in shaping hypertension control in our sample population. It is also possible that race/ethnicity, a social rather than biological construct and a proxy for structural racism and discrimination, may impact individual hypertension outcomes more strongly than those captured in nSES alone. Collectively, we need more research in this space to carefully model race/ethnicity and neighborhood predictors of health outcomes using rigorous methods (and specifying where these serve as imperfect proxies for other larger societal structures and policies) to ensure that our work to eliminate healthcare disparities can be targeted at the right levels given the intersecting and multi-level determinants of health.

Healthcare systems are increasingly prioritizing population health and considering the role of social determinants of health, including structural and place-based factors. From a population health management perspective, results of this study suggest that neighborhood-specific interventions may not be sufficient in eliminating racial/ethnic disparities in uncontrolled hypertension, and we are encouraged to investigate potential pathways that directly address psychosocial stressors including racism and discrimination in health care settings and other environments to better inform alternative intervention strategies. Towards this end, healthcare systems can develop reimbursement, regulatory, and data infrastructures, as well as shifts in the care paradigm, to regularly collect social determinants of health measures in EHRs. This information could then be incorporated into organizational policy and clinical decision-making, with the goal of proactively identifying, monitoring, and ultimately intervening on environmental and social factors that are relevant to the health of communities and patient cohorts.

Our study is novel in its use of EHR data to study the mediation effect of neighborhood environment on racial/ethnic differences. EHRs provide access to longitudinal clinical data for a large patient population, and because of this, we were able to limit our sample to patients who had at least two clinically-measured blood pressure readings in an outpatient setting rather than determining hypertension control status from a single blood pressure reading or self-report. This could address potential misclassification of hypertension control due to blood pressure variability[38] and erroneous self-reported blood pressure measurements.[39] The EHR data provided both comprehensive clinical measures and patient address, and by combining diverse patient populations from two healthcare systems, we were able to construct a large, racially diverse sample more likely to capture a range of socioeconomic factors and health behaviors representative of our geographic region and less likely to underestimate racial/ethnic disparities in our target population.

Limitations

We were unable to identify longitudinal residential address history using available EHR data, and therefore, unable to establish temporality between the mediator and the outcome

and to examine the longitudinal effect of neighborhood environment. Additionally, our use of EHR data and exclusion criteria may bias our sample population to capture patients who were, to some extent, engaged in the healthcare system. One-third of patients with hypertension were excluded because they did not have at least two outpatient visits over the study period, and it is possible that these patients were systematically different from those in our sample. Due to data availability in the EHR, patients were categorized into broad racial/ethnic groups that are most commonly collected during routine care. We acknowledge that these racial categorizations conceal more nuanced experiences of racial bias and discrimination and future research is needed to meaningfully disaggregate these data.

Based on established work in this space, we likewise chose to use census tracts to represent neighborhood, but recognize that these boundaries are arbitrary and vary by individuals, so the attribution of nSES based on census tracts may not be accurate for all patients. Moreover, we used a composite index of nSES and were unable to identify specific socioeconomic factors that play a role in racial disparities in uncontrolled hypertension. Although this index included several neighborhood-level socioeconomic indicators, it did not include other structural factors and built environment features such as access to transportation or distance from healthcare clinics and pharmacies. Despite previous validation of this measure, a more comprehensive set of neighborhood features, including those amenable to change, deserve further investigation.

Conclusion

Among patients with hypertension, nSES mediates a small proportion of racial/ethnic disparities in uncontrolled hypertension. It is only one of many potential pathways that may play a role in the inequitable distribution of cardiovascular disease risk factors among racial/ethnic groups in the United States. Still, racial/ethnic disparities in uncontrolled hypertension are more than a reflection of individual risk factors, and given these findings, population health management strategies may be most effective by focusing on additional structural and interpersonal pathways.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

We would like to acknowledge Alice Fishman for her administrative support with this project.

Sources of Funding

This work was funded by the Population Health Data Initiative within the University of California, San Francisco School of Medicine Dean’s Office and the University of California San Francisco Executive Vice Chancellor’s Office. C.R.L. also received support from a UCSF/Genentech Mid-Career Development Award.

Non-standard Abbreviations and Acronyms:

ACC	American College of Cardiology

ACS	American Community Survey
AHA	American Heart Association
HER	electronic health record
GSEM	generalized structural equation model
nSES	neighborhood socioeconomic status
SFHN	San Francisco Health Network
UC	University of California
UCSF	University of California, San Francisco

References

1. Health, United States 2018. National Center for Health Statistics. 2019. Accessed October 25, 2021. https://www.cdc.gov/nchs/data/hus/hus18.pdf
2. Kochanek KD, Arias E, Anderson RN. How did cause of death contribute to racial differences in life expectancy in the United States in 2010? NCHS Data Brief, no 125. National Center for Health Statistics. 2013. Accessed October 25, 2021. https://www.cdc.gov/nchs/data/databriefs/db125.pdf
3. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. Circulation. 2005; 111:1233–1241. doi: 10.1161/01.CIR.0000158136.76824.04 [PubMed: 15769763]
4. Khavjou O, Phelps D, Leib A. Projections of Cardiovascular Disease Prevalence and Costs: 2015–2035. American Heart Association. 2016. Accessed August 19, 2021. https://www.heart.org/-/media/files/get-involved/advocacy/burden-report-technical-report.pdf?la=en
5. Moran AE, Odden MC, Thanataveerat A, Tzong KY, Rasmussen PW, Guzman D, Williams L, Bibbins-Domingo K, Coxson PG, Goldman L. Cost-effectiveness of hypertension therapy according to 2014 guidelines. N Engl J Med. 2015; 372:447–55. doi: 10.1056/NEJMsa1406751. [PubMed: 25629742] Erratum in: N Engl J Med. 2015; 372:1677.
6. Davis KE. Expenditures for Hypertension among Adults Age 18 and Older, 2010: Estimates for the U.S. Civilian Noninstitutionalized Population. Statistical Brief, no 404. Agency for Healthcare Research and Quality. 2013. Accessed October 25, 2021. https://meps.ahrq.gov/data_files/publications/st404/stat404.shtml
7. Lê-Scherban F, Ballester L, Castro JC, Cohen S, Melly S, Moore K, Buehler JW. Identifying neighborhood characteristics associated with diabetes and hypertension control in an urban African-American population using geo-linked electronic health records. Prev Med Rep. 2019; 15:100953. doi: 10.1016/j.pmedr.2019.100953 [PubMed: 31367515]
8. Schinasi LH, Auchincloss AH, Forrest CB, Diez Roux AV. Using electronic health record data for environmental and place based population health research: a systematic review. Ann Epidemiol. 2018; 28:493–502. doi: 10.1016/j.annepidem.2018.03.008 [PubMed: 29628285]
9. Galea S, Abdalla SM, Sturchio JL. Social determinants of health, data science, and decision-making: Forging a transdisciplinary synthesis. PLoS Med.; 17:e1003174. doi: 10.1371/journal.pmed.1003174
10. Gamache R, Kharrazi H, Weiner JP. Public and Population Health Informatics: The Bridging of Big Data to Benefit Communities. Yearb Med Inform. 2018; 27:199–206. doi:10.1055/s-0038-1667081 [PubMed: 30157524]
11. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, DePalma SM, Gidding S, Jamerson KA, Jones DW, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice

Guidelines. Circulation. 2018; 138:e426–e483. doi:10.1161/CIR.0000000000000597 [PubMed: 30354655]

12. Healthy People 2030: Leading Health Indicators. Healthy People 2030. U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion. Accessed March 22, 2021. https://www.healthypeople.gov/2030/Leading-Health-Indicators
13. Fryar CD, Ostchega Y, Hales CM, Zhang G, Kruszon-Moran D. Hypertension Prevalence and Control Among Adults: United States, 2015–2016. NCHS Data Brief, no. 289. 2017. Accessed October 25, 2021. https://www.cdc.gov/nchs/data/databriefs/db289.pdf
14. Hertz RP, Unger AN, Cornell JA, Saunders E. Racial Disparities in Hypertension Prevalence, Awareness, and Management. Arch Intern Med. 2005; 165:2098–2104. doi:10.1001/archinte.165.18.2098 [PubMed: 16216999]
15. Morenoff JD, House JS, Hansen BB, Williams DR, Kaplan GA, Hunte HE. Understanding Social Disparities in Hypertension Prevalence, Awareness, Treatment, and Control: The Role of Neighborhood Context. Soc Sci Med 1982. 2007; 65:1853–1866. doi:10.1016/j.socscimed.2007.05.038
16. Rothstein Richard. The Color of Law: A Forgotten History of How Our Government Segregated America. NY; London: Liveright Publishing Corporation; 2017.
17. Mujahid MS, Moore LV, Petito LC, Kershaw KN, Watson K, Diez Roux AV. Neighborhoods and racial/ethnic differences in ideal cardiovascular health (the Multi-Ethnic Study of Atherosclerosis). Health Place. 2017; 44:61–69. doi:10.1016/j.healthplace.2017.01.005 [PubMed: 28167269]
18. Do DP, Dubowitz T, Bird CE, Lurie N, Escarce JJ, Finch BK. Neighborhood context and ethnicity differences in body mass index: a multilevel analysis using the NHANES III survey (1988–1994). Econ Hum Biol. 2007; 5:179–203. doi:10.1016/j.ehb.2007.03.006 [PubMed: 17507298]
19. Mujahid MS, Roux AVD, Cooper RC, Shea S, Williams DR. Neighborhood stressors and race/ethnic differences in hypertension prevalence (the Multi-Ethnic Study of Atherosclerosis). Am J Hypertens. 2011; 24:187–193. doi:10.1038/ajh.2010.200 [PubMed: 20847728]
20. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003; 42:1206–1252. doi: 10.1161/01.HYP.0000107251.49515.c2 [PubMed: 14656957]
21. Yang J, Schupp C, Harrati A, Clarke C, Keegan T, Gomez S. Developing an area-based socioeconomic measure from American Community Survey data. Cancer Prevention Institute of California. Accessed September 1, 2019. https://cancerregistry.ucsf.edu/resources/research-tools
22. Geography and the American Community Survey: What Data Users Need to Know. U.S. Census Bureau. 2020. Accessed October 25, 2021. https://www.census.gov/content/dam/Census/library/publications/2020/acs/acs_geography_handbook_2020.pdf
23. Krieger N, Chen JT, Waterman PD, Soobader MJ, Subramanian SV, Carson R. Choosing area based socioeconomic measures to monitor social inequalities in low birth weight and childhood lead poisoning: The Public Health Disparities Geocoding Project (US). J Epidemiol Community Health. 2003; 57:186–99. doi: 10.1136/jech.57.3.186 [PubMed: 12594195]
24. Krieger N, Waterman P, Chen JT, Soobader MJ, Subramanian SV, Carson R. Zip code caveat: bias due to spatiotemporal mismatches between zip codes and US census-defined geographic areas--the Public Health Disparities Geocoding Project. Am J Public Health. 2002; 92:1100–2. doi: 10.2105/ajph.92.7.1100 [PubMed: 12084688]
25. Jones CP. Levels of racism: a theoretic framework and a gardener's tale. Am J Public Health. 2000; 90:1212–1215. doi:10.2105/ajph.90.8.1212 [PubMed: 10936998]
26. Kressin NR, Orner MB, Manze M, Glickman ME, Berlowitz D. Understanding contributors to racial disparities in blood pressure control. Circ Cardiovasc Qual Outcomes. 2010; 3:173–180. doi:10.1161/CIRCOUTCOMES.109.860841 [PubMed: 20233981]
27. Al Kibria GM. Racial/ethnic disparities in prevalence, treatment, and control of hypertension among US adults following application of the 2017 American College of Cardiology/American Heart Association guideline. Prev Med Rep. 2019; 14:100850. doi:10.1016/j.pmedr.2019.100850 [PubMed: 31061780]

28. Gu A, Yue Y, Desai RP, Argulian E. Racial and Ethnic Differences in Antihypertensive Medication Use and Blood Pressure Control Among US Adults With Hypertension: The National Health and Nutrition Examination Survey, 2003 to 2012. Circ Cardiovasc Qual Outcomes. 2017; 10:e003166. doi:10.1161/CIRCOUTCOMES.116.003166 [PubMed: 28096206]
29. Manze M, Rose AJ, Orner MB, Berlowitz DR, Kressin NR. Understanding racial disparities in treatment intensification for hypertension management. J Gen Intern Med. 2010; 25:819–825. doi:10.1007/s11606-010-1342-9 [PubMed: 20386998]
30. Bailey ZD, Krieger N, Agénor M, Graves J, Linos N, Bassett MT. Structural racism and health inequities the USA: evidence and interventions. Lancet. 2017; 389:1453–1463. doi: 10.1016/S0140-6736(17)30569-X [PubMed: 28402827]
31. StataCorp. 2019. Stata Statistical Software: Release 16. College Station, TX: StataCorp LLC.
32. Diez Roux AV, Mujahid MS, Hirsch JA, Moore K, Moore LV. The impact of neighborhoods on cardiovascular risk: the MESA Neighborhood Study. Glob Heart. 2016; 11:353–363. doi:10.1016/j.gheart.2016.08.002 [PubMed: 27741982]
33. Bauer GR. Incorporating intersectionality theory into population health research methodology: challenges and the potential to advance health equity. Soc Sci Med. 2014; 110:10–17. doi:10.1016/j.socscimed.2014.03.022 [PubMed: 24704889]
34. Lewey J, Shrank WH, Avorn J, Liu J, Choudhry NK. Medication adherence and healthcare disparities: impact of statin co-payment reduction. Am J Manag Care. 2015; 21:696–704. [PubMed: 26633094]
35. Schoenthaler AM. Reexamining medication adherence in black patients with hypertension through the lens of the social determinants of health. J Clin Hypertens. 2017; 19:1025–1027. doi:10.1111/jch.13071
36. Mujahid MS, Sohn EK, Izenberg J, Gao X, Tulier ME, Lee MM, Yen IH. Gentrification and Displacement in the San Francisco Bay Area: A Comparison of Measurement Approaches. Int J Environ Res Public Health. 2019; 16:2246. doi: 10.3390/ijerph16122246
37. Shankardass K, Dunn JR. How Goes the Neighbourhood? Rethinking Neighbourhoods and Health Research in Social Epidemiology. In O'Campo P, Dunn JR (Eds.): Rethinking Social Epidemiology. 2012; 137–156. doi: 10.1007/978-94-007-2138-8_7
38. Parati G, Ochoa JE, Lombardi C, Bilo G. Assessment and management of blood-pressure variability. Nat Rev Cardiol. 2013; 10:143–155. doi:10.1038/nrcardio.2013.1 [PubMed: 23399972]
39. Johnson KA, Partsch DJ, Rippole LL, McVey DM. Reliability of self-reported blood pressure measurements. Arch Intern Med. 1999; 159:2689–2693. doi:10.1001/archinte.159.22.2689 [PubMed: 10597759]

What is Known

- Individual-level demographics and health behaviors do not fully explain racial/ethnic disparities in hypertension control.
- Health-promoting resources and environments are differentially distributed among racial/ethnic groups and these physical, social, and economic environments have been linked to cardiovascular risk and outcomes.

What the Study Adds

- Patient-level electronic health record data from multiple health systems can be integrated with publicly-available census data to study potential environmental mediators of racial/ethnic disparities in health outcomes.
- Findings show that neighborhood socioeconomic status mediates 6.6% of the difference in uncontrolled hypertension between Black and White patients and 7.2% of the difference between Hispanic/Latino and White patients.

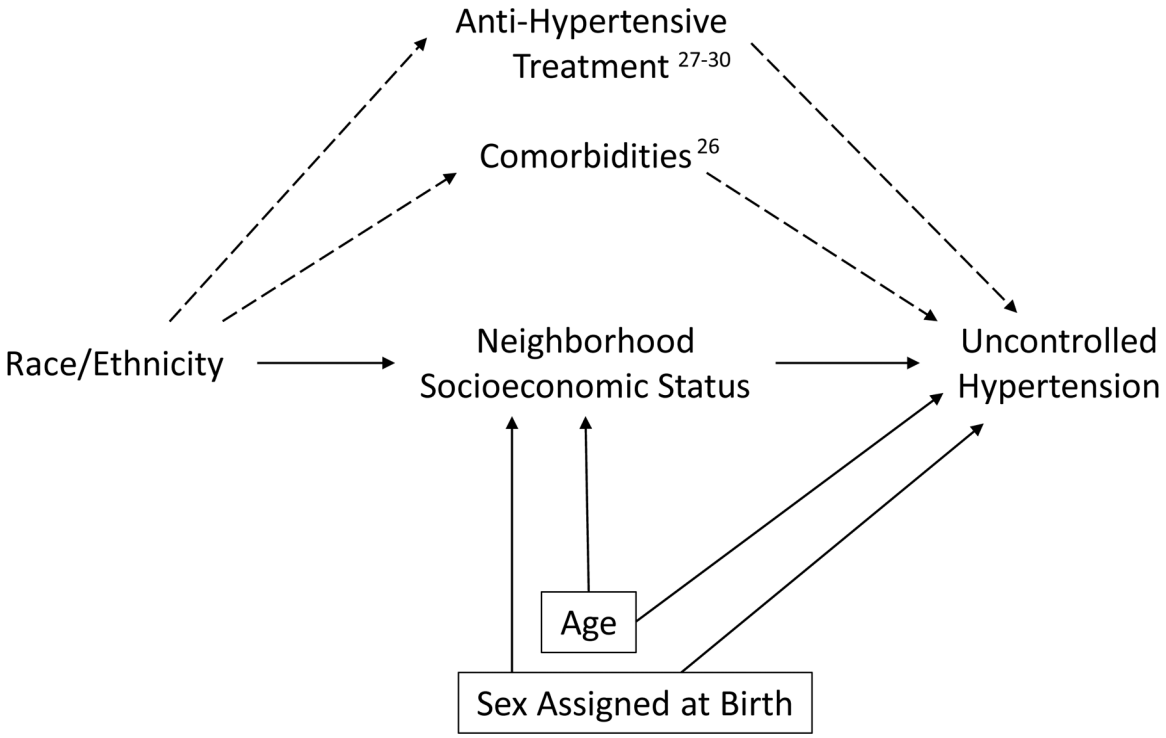

Figure 1. Directed acyclic graph identifying the hypothesized mediating role of nSES to disparities in uncontrolled hypertension between each racial/ethnic minority patient group and White patients.

Solid lines show the relationships examined in the current analysis. Dashed lines show potential mediators from race/ethnicity to uncontrolled hypertension that were considered but not included in the current analysis. Constructs in boxes indicate confounders. Precision variables are not shown.

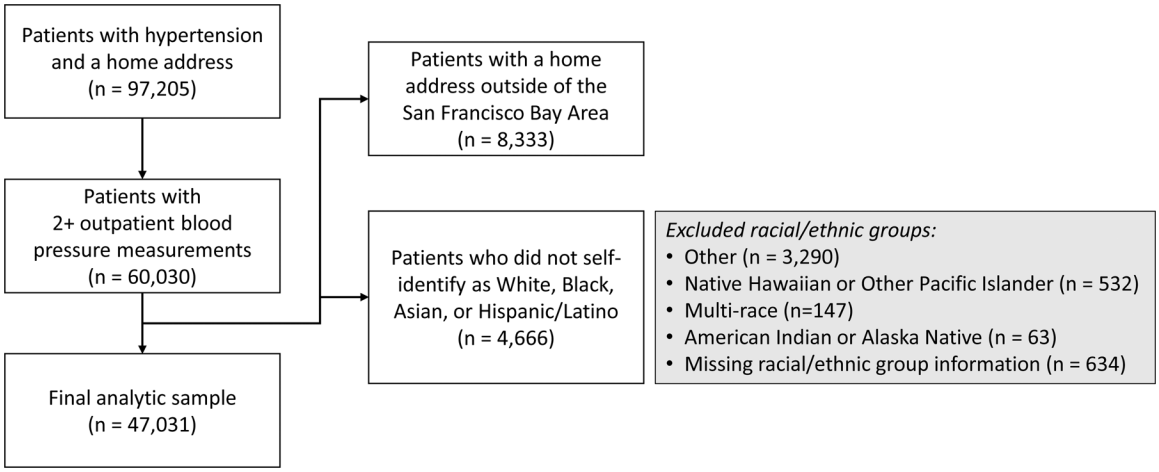

Figure 2. Cohort flowchart

Table 1.

Characteristics of patients with a prior hypertension diagnosis and at least two outpatient blood pressure measurements between June 1, 2015 and May 30, 2017 from two health systems in the San Francisco Bay Area, CA [*]

	Total (n = 47,031) n (%)	White (n = 18,566) n (%)	Asian (n = 13,948) n (%)	Black (n = 7,483) n (%)	Hispanic/Latino (n = 7,034) n (%)	P-value[†]
Uncontrolled Hypertension	29,325 (62)	11,006 (59)	8,272(59)	5,350 (72)	4,697 (67)	< 0.001
Health System						< 0.001
UCSF Health	29,213 (60)	15,269 (82)	7,545 (54)	3,475 (46)	2,924 (42)	
SFHN	17,818 (40)	3,297 (18)	6,403 (46)	4,008 (54)	6403 (46)	
nSES Quintiles						< 0.001
1 (lowest)	11,081 (24)	1,958 (11)	3,551 (25)	3,488 (47)	2,084 (30)	
2	9,454 (20)	2,798 (15)	3,015 (21)	1,622 (22)	2,019 (29)	
3	8,208 (17)	3,420 (18)	2,721 (20)	832 (11)	1,235 (17)	
4	9,346 (20)	4,486 (24)	2,884 (21)	936 (12)	1040 (15)	
5 (highest)	8,942 (19)	5,904 (32)	1,777 (13)	605 (8)	656 (9)	
Age group						< 0.001
18–34	1,458 (3)	465 (3)	288 (2)	315 (4)	390 (6)	
35–64	21,944 (47)	7,413 (61)	6,031 (43)	4,585 (61)	3,915 (56)	
65–79	17,117 (36)	7,609 (28)	5,354 (38)	2,131 (28)	2,023 (29)	
80+	6,512 (14)	3,079 (17)	2,275 (16)	452 (6)	706 (10)	
Sex assigned at birth						< 0.001
Female	23,909 (51)	8,051 (43)	8,046 (58)	3,929 (53)	3,883 (55)	
Male	23,122 (49)	10,515 (57)	5,902 (42)	3,554 (47)	3,151 (45)	

Abbreviations: UCSF, University of California San Francisco; SFHN, San Francisco Health Network; nSES, Neighborhood socioeconomic status

[*] San Francisco Bay Area defined as including 9 counties in this California region: Alameda, Contra Costa, Marin, Napa, San Francisco, San Mateo, Santa Clara, Solano, and Sonoma

[†] Hypothesis tests using chi-squared tests of independence to compare the distribution of each characteristic across the four racial/ethnic categories (i.e. White, Black, Asian, Hispanic/Latino)

Table 2.

Estimates of direct, indirect, and total effects of the association* between race/ethnicity and uncontrolled hypertension and the percent mediated by neighborhood socioeconomic status among patients in two healthcare systems in the San Francisco Bay Area, CA (n = 47,031).

	Direct effect† Race/Ethnicity → Uncontrolled HTN OR (95% CI)	Natural indirect effect‡ Race/Ethnicity → nSES → Uncontrolled HTN OR (95% CI)	Total effect§ OR (95% CI)	% mediated‖
Primary Analysis				
Black	**1.73 (1.61, 1.86)**	**1.04 (1.01, 1.07)**	**1.79 (1.67, 1.91)**	6.57%
Hispanic/Latino	**1.35 (1.26 1.44)**	**1.02 (1.00, 1.14)**	**1.38 (1.29, 1.48)**	7.22%
Asian	**0.93 (0.88, 0.98)**	**1.02 (1.00, 1.03)**	**0.95 (0.89, 0.99)**	-
White	Ref	Ref	Ref	-
Sensitivity Analysis				
Black	**1.80 (1.67, 1.94)**	**1.08 (1.04, 1.11)**	**1.94 (1.80, 2.08)**	11.04%
Hispanic/Latino	**1.44 (1.36, 1.52)**	**1.05 (1.03, 1.08)**	**1.52 (1.44, 1.60)**	12.53%
Asian	0.97 (0.92, 1.02)	1.04 (1.02, 1.06)	1.01 (0.96, 1.07)	-
White	Ref	Ref	Ref	-

Bolded values denote a p-value ≤ 0.05

Abbreviations: HTN, hypertension; nSES, neighborhood socioeconomic status; OR, odds ratio; CI, confidence interval

*Primary analysis is adjusted for age, sex assigned at birth, and healthcare system; sensitivity analysis is adjusted for age and sex assigned at birth.

†Natural direct effect is the odds of uncontrolled hypertension independent of neighborhood socioeconomic status for one racial/ethnic group compared to the reference group (White patients).

‡Natural indirect effect is the odds of uncontrolled hypertension that is transmitted through neighborhood socioeconomic status for one racial/ethnic group compared to the reference group (White patients).

§Total effect is the sum of the direct and indirect effects.

‖The percent mediated is the proportion of the total effect that can be attributed to the natural indirect effects