Practice of Epidemiology

Using Decomposition Analysis to Identify Modifiable Racial Disparities in the Distribution of Blood Pressure in the United States

Sanjay Basu*, Anthony Hong, and Arjumand Siddiqi

* Correspondence to Dr. Sanjay Basu, Stanford University School of Medicine, Medical School Office Building, X322, 1265 Welch Road, Mail Code 5411, Stanford, CA 94305-5411 (e-mail: basus@stanford.edu).

To lower the prevalence of hypertension and racial disparities in hypertension, public health agencies have attempted to reduce modifiable risk factors for high blood pressure, such as excess sodium intake or high body mass index. In the present study, we used decomposition methods to identify how population-level reductions in key risk factors for hypertension could reshape entire population distributions of blood pressure and associated disparities among racial/ethnic groups. We compared blood pressure distributions among non-Hispanic white, non-Hispanic black, and Mexican-American persons using data from the US National Health and Nutrition Examination Survey (2003–2010). When using standard adjusted logistic regression analysis, we found that differences in body mass index were the only significant explanatory correlate to racial disparities in blood pressure. By contrast, our decomposition approach provided more nuanced revelations; we found that disparities in hypertension related to tobacco use might be masked by differences in body mass index that significantly increase the disparities between black and white participants. Analysis of disparities between white and Mexican-American participants also reveal hidden relationships between tobacco use, body mass index, and blood pressure. Decomposition offers an approach to understand how modifying risk factors might alter population-level health disparities in overall outcome distributions that can be obscured by standard regression analyses.

health disparities; hypertension; population studies; statistical methods

Abbreviations: BMI, body mass index; NHANES, National Health and Nutrition Examination Survey.

Editor's note: An invited commentary on this article appears on page 354, and the authors' response appears on page 358.

High blood pressure remains the second leading preventable risk factor for death in the United States, behind tobacco smoking (1). Hypertension (a systolic blood pressure of at least 140 mm Hg or a diastolic blood pressure of at least 90 mm Hg (2)) is highest among black adults and lowest among Mexican-American adults and some other Latino groups (3, 4).

Emerging literature suggests that the full blood pressure distribution carries important health implications. The relationship between blood pressure and mortality appears J-shaped, with some increased mortality risk at low blood pressures and greater mortality risk beginning at "normal"

values and rising incrementally with increased blood pressure (5, 6). Atherosclerotic risk also appears to increase with incremental increases in systolic and diastolic blood pressure starting with pressures in the normal range (7). Collectively, these findings indicate that disparities in high blood pressure should be analyzed not only as disparities in hypertension prevalence, but also as disparities across the entire distribution of systolic and diastolic blood pressures.

Although the theoretical importance of a distributional perspective has long between established in epidemiology, a critical gap in the field has been finding methodological approaches to empirically engage with the distributional perspective. The Kolmogorov-Smirnov test statistic tests whether overall differences between 2 distributions can be considered significant, but it cannot identify the degree to which a given risk factor might explain these differences (8). Quantile

regression provides estimates of how risk factors may alter an outcome within each quantile of the population distribution of a disease risk factor, but it requires parametric assumptions that are difficult to uphold for many empirical distributions (9). The extant literature also reveals that key hypertension risk factors may not explain blood pressure disparities in a linear way across blood pressure distributions; there are complex relations between factors such as body mass index (BMI) (3), tobacco smoking (10, 11), alcohol consumption (11–13), sodium intake (14, 15), and ultimate hypertension risk.

Here, we introduce a nonparametric decomposition technique derived from the field of econometrics (16) that identifies how changes in modifiable risk factors for the outcome variable could reduce disparities at different points along the distribution of an outcome variable. We can thus ask: How much do modifiable risk factors such as BMI, smoking, alcohol consumption, and sodium intake account for disparities at each point in the blood pressure distribution? If we were to eliminate racial differences in observable modifiable risk factors, how much would we expect racial disparities in blood pressure to be mitigated at each point in the blood pressure distribution? To demonstrate the method, we focused on racial disparities in blood pressure in the US National Health and Nutrition Examination Survey (NHANES) (17) and compare the insights from this approach to the insights gleaned from a traditional "cutpoint" analysis of hypertension.

METHODS

Data sources

NHANES was chosen for our analysis because it is the basis for US government estimates of hypertension disparities among racial/ethnic groups (4). NHANES data from 2003 through 2010 were chosen for the analysis because hypertension disparities remained stable between racial/ethnic groups over these years (4) and because survey questions about risk factors for high blood pressure were consistently asked among these survey waves. We chose the following common risk factors for high blood pressure to analyze:
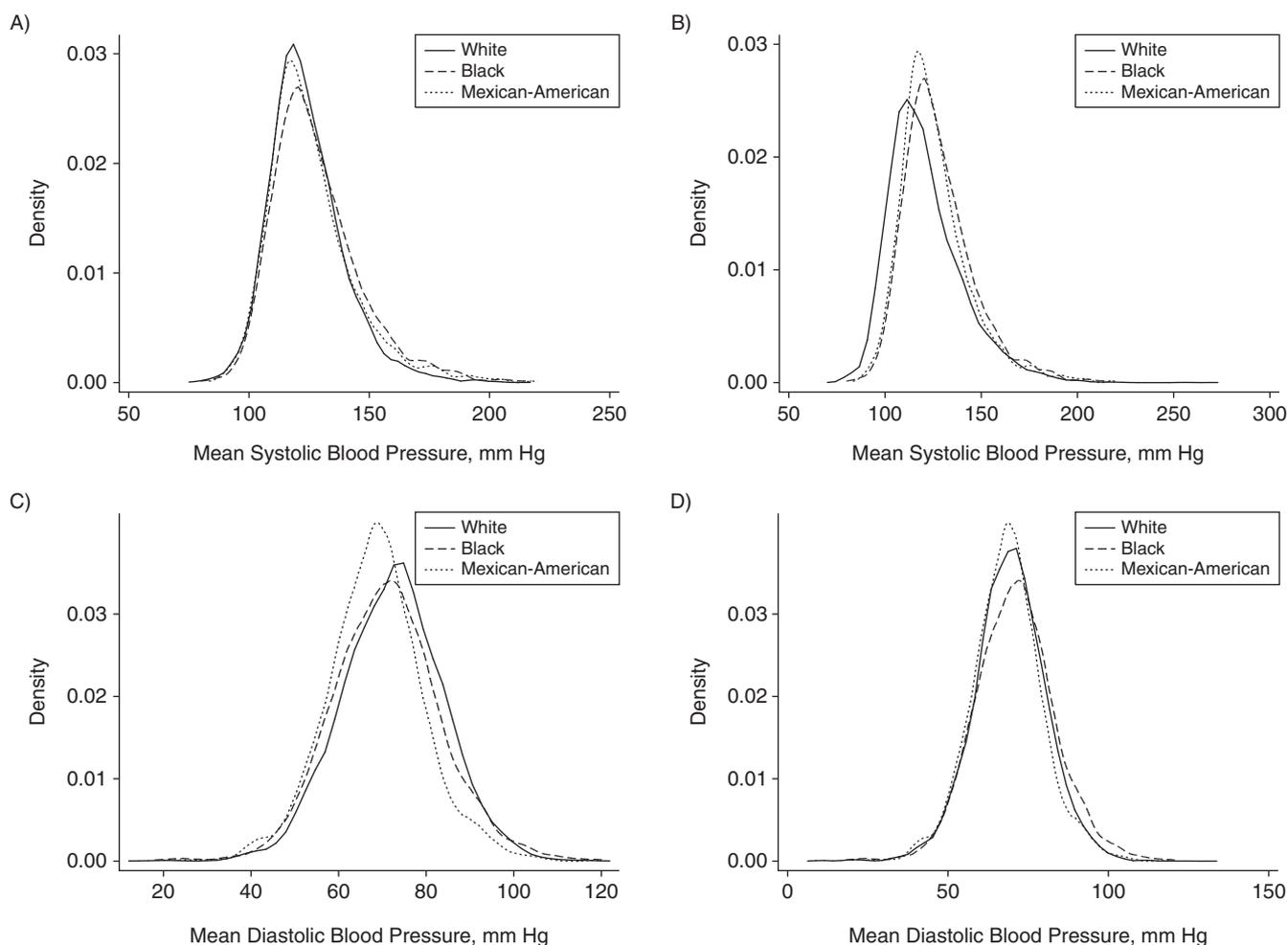
Figure 1. Age-adjusted distributions of blood pressure in the US National Health and Nutrition Examination Survey, 2003–2010 (17). A) Systolic blood pressure among men; B) systolic blood pressure among women; C) diastolic blood pressure among men; and D) diastolic blood pressure among women. Densities reveal the probability of each blood pressure at each level of mm Hg, using an Epanechnikov kernel smoothing function applied to 50 evaluation points across the range of observed blood pressures.

tobacco smoking (whether the participant smoked at least 100 cigarettes in their lifetime), alcohol consumption (average number of alcohol drinks per day over the last 12 months), sodium intake (in mg/person/day from 24-hour dietary recalls ([18], [19])), and BMI (weight in kilograms divided by height in meters squared) determined from height and weight measurements during the survey. We related these to the mean of 3 recorded systolic and diastolic blood pressures. Frequencies and distributions of each high blood pressure risk factor by racial/ethnic group and sex are listed in Web Table 1 (available at http://aje.oxfordjournals.org/). Because more than 97% of subjects had complete data, no imputation was performed for missing values. Data from 25,510 US-born nonpregnant adults were used for the analysis.

Statistical analysis

The derivation of the method is provided in the Web Appendix, along with statistical code to replicate the analysis. Here, we describe the intuition behind the method. The decomposition approach accomplished the 2 primary objectives of our analysis: to quantify the difference in the blood pressure distributions among racial/ethnic groups and to determine the extent to which modifiable risk factors account for differences at each point along the blood pressure distribution. We compared black and Mexican-American participants with the reference group of white participants, both to contrast our results with national disparity statistics that use white persons as a reference group ([20]–[22]) and to test our hypothesis that white participants are actually not always the most "advantaged" in terms of risk factor levels at all parts of the distribution of blood pressure.

First, we constructed age-adjusted, sex-specific estimates of blood pressure distributions among each racial/ethnic group (Figure 1) ([23]). Next, we measured and plotted the difference in blood pressure along each point of the distribution. To assess the contribution of each risk factor independently and of the risk factors jointly, we constructed counterfactual distributions that reflect how one group's distribution of blood pressure would be expected to change if its risk factor profile looked more like that of a comparator group (e.g., how much the distribution of systolic blood pressure among black participants would be expected to shift if that had the same risk factor profiles as white participants). These counterfactual distributions were defined by first writing the marginal distribution of blood pressure as the joint distribution of blood pressure integrated over risk factors for each group. Then, applying the law of iterated expectations ([24]), we expressed the group-wise marginal distribution of blood pressure as the product of the conditional blood pressure density and conditional risk factor densities. This allowed us to consider a number of counterfactual situations such as the blood pressure outcome for black participants if they had the risk factor profiles of white participants (Figure 2).

The reweighting procedure (Web Table 2) allows us to decompose the difference in blood pressure between groups into the sum of 2 components. The first, the explained component, is the portion of between-race differences in blood pressure due to differences in risk factors for high blood pressure. The second, the unexplained or residual compo-
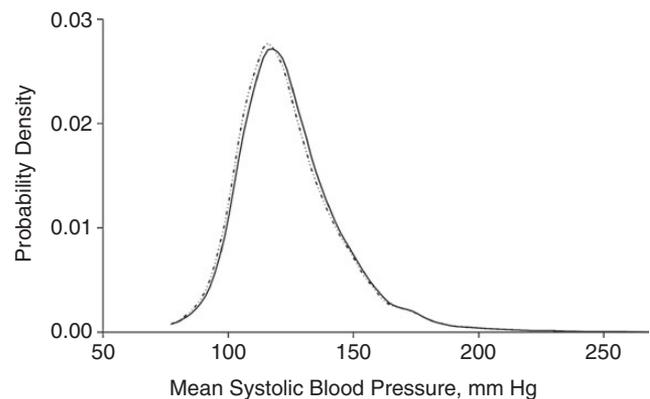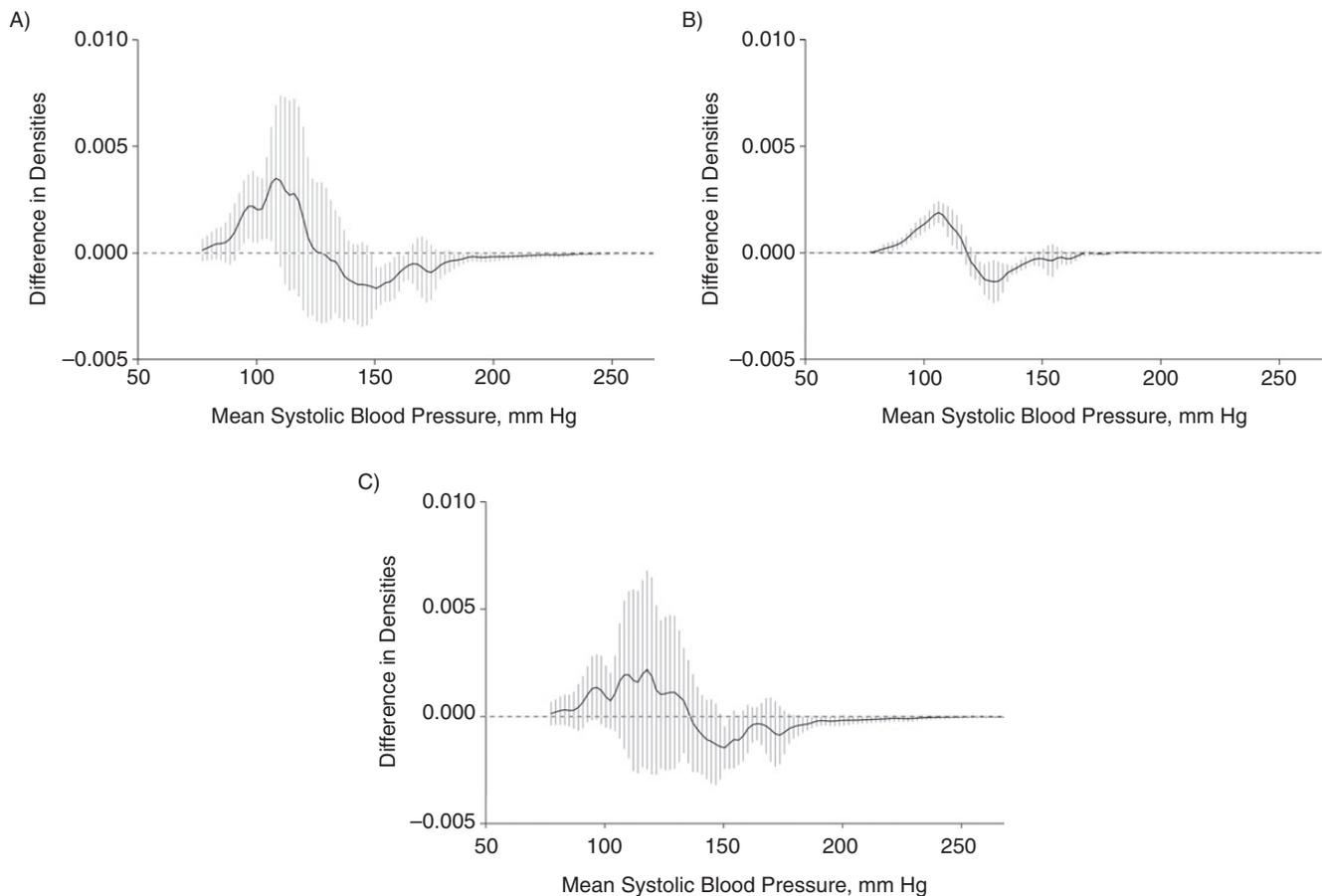

Figure 2. Counterfactual analysis of age-adjusted distributions of blood pressure in the US National Health and Nutrition Examination Survey, 2003–2010 ([17]). Counterfactual analysis involves comparing the density of systolic blood pressure for black participants (solid line) with the counterfactual case in which the systolic blood pressure distribution in black participants is reweighted to reflect what it would appear if they had the same body mass index distribution as did white participants (dotted-dashed line). The *y*-axis refers to the probability density at each point along the distribution. As shown, the blood pressure distribution is shifted slightly to the left (lower systolic blood pressures) if the distribution in black participants is reweighted to reflect the body mass index distribution of white participants with all else being held equal.

nent, captures the distributional disparities that are not explainable through differences in the observed risk factor characteristics—that is, the distributional differences that do not change when reweighting the distribution of one group to match the risk factors of the other group (Figure 3).

Sensitivity analyses

The decomposition method was applied to systolic blood pressure and then reapplied to diastolic blood pressure distributions as a robustness check. Comparisons were made between the decomposition results and standard logistic regressions of the attributes against prevalence rates of prehypertension (systolic blood pressure of 120–139 mm Hg or diastolic blood pressure of 80–89 mm Hg), stage 1 hypertension (systolic blood pressure of 140–159 mm Hg or diastolic blood pressure of 90–99 mm Hg), stage 2 hypertension (systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥100 mm Hg), and overall hypertension (either stage 1 or 2). Age-adjusted values using the direct method for age-standardization were applied to all analyses; survey sample weights were also applied to correct for nonresponse and differential sampling among groups ([23], [25]). Subgroup analyses were performed by repeating the decompositions on only those individuals not currently taking antihypertensive medications (*n* = 18,757) to filter out potential consequences of differential health care access and quality. A further sensitivity analysis was performed by replacing BMI with waist circumference (in centimeters, measured during the NHANES medical examination) and then including both BMI and waist circumference separately in the decompositions to identify

Figure 3. Decomposition of blood pressure in the US National Health and Nutrition Examination Survey, 2003–2010 (17). The analysis identifies how much disparity would remain between blood pressure distributions of whites and blacks after the black systolic blood pressure distribution is reweighted to reflect the white distribution of each modifiable risk factor. A) First, we plotted the systolic blood pressure distribution in white participants minus the distribution in black participants. The net difference in distributions is positive at lower values of systolic blood pressure because there were more white participants than black participants with lower blood pressure; conversely, the net difference in distributions is negative at higher values of systolic blood pressure because there are fewer white participants than there were black participants with high blood pressure. B) Next, we plotted what the difference in distributions would be after the distribution of systolic blood pressure in black participants was reweighted to reflect the distribution of body mass index among white participants. As shown, after the body mass index reweighting, the net differences would be slightly reduced, but black participants would still have more prevalent high blood pressure than would white participants. C) Finally, we plotted the "residual" or unexplained difference, which is the portion of the net disparity between the distributions that does not change despite reweighting the distribution in black participants by all observed risk factors (in this case, a majority of the difference in distributions between white and black participants remains unexplained by differences in body mass index). Gray vertical bars indicate 95% confidence intervals at each point along the distribution.

potential variations in associations of body composition captured by a metric of central adiposity rather than body mass. All analyses were conducted using Stata, version MP-12.1 (StataCorp LP, College Station, Texas).

RESULTS

Observed disparities

A standard cutpoint analysis revealed that black men had a significantly higher prevalence of hypertension than did either white or Mexican-American men, with Mexican-American men having significantly lower rates of hypertension than all other men ($P < 0.05$; Table 1). Among women, black women had a significantly higher prevalence of

hypertension than did either white or Mexican-American women, but the latter 2 groups did not significantly differ at the level of $P < 0.05$. In this cutpoint analysis of hypertension disparities, disparities between black and white participants were essentially equal at stages 1 and 2 of hypertension ($\approx 6\%$ difference among men and $\approx 12\%$ among women in the proportion of black vs. white participants with stage 1 or stage 2 hypertension). As also shown in Table 1, disparities between Mexican-American and white participants were greatest for prehypertension ($\approx 13\%$ difference in prevalence between Mexican-American and white men and $\approx 9\%$ between Mexican-American and white women) but lower for both stages of hypertension ($\approx 1\%$ for men and women).

By examining the overall distributions of blood pressure among groups (Figure 1), it was possible to observe a more

Table 1.   Hypertension Disparities by Sex and Race/Ethnicity Among Nonpregnant US-Born Adults in the US National Health and Nutrition Examination Survey, 2003–2010[a]

Sex and Race/ Ethnicity	No. of Participants	Hypertension Category							
		Prehypertension[b]		Overall Hypertension[c]		Stage 1 Hypertension Only[d]		Stage 2 Hypertension Only[e]	
		%	95% CI	%	95% CI	%	95% CI	%	95% CI
Male	12,643								
Non-Hispanic white	7,838	15.6	12.4, 18.8	27.7	26.1, 29.3	26.3	24.7, 27.9	18.6	17.3, 19.9
Non-Hispanic black	3,150	19.3	12.9, 25.6	35.7	32.8, 38.7	33.2	30.3, 36.0	24.9	22.1, 27.7
Mexican-American	1,655	8.0	0.9, 15.1	29.0	24.4, 33.7	27.3	22.7, 32.0	17.7	14.2, 21.2
Female	12,867								
Non-Hispanic white	7,723	15.9	11.9, 19.9	24.6	23.2, 25.9	22.7	21.3, 24.0	18.8	17.5, 20.1
Non-Hispanic black	3,263	24.7	17.7, 31.6	36.8	34.4, 39.2	34.1	31.6, 36.6	30.3	27.9, 32.7
Mexican-American	1,881	6.9	1.8, 12.0	24.9	21.6, 28.3	22.6	19.1, 26.1	17.4	14.2, 20.6

Abbreviation: CI, confidence interval.

[a] Prevalence rates are age-standardized using the direct method and incorporate survey sample weights to generate nationally representative results.

[b] Systolic blood pressure of 120–139 mm Hg or diastolic blood pressure of 80–89 mm Hg.

[c] Either stage 1 or 2 hypertension.

[d] Systolic blood pressure of 140–159 mm Hg or diastolic blood pressure of 90–99 mm Hg.

[e] Systolic blood pressure $\geq$160 mm Hg or diastolic blood pressure $\geq$100 mm Hg.

complex picture of disparities than was possible through the cutpoint analysis. As shown in Figure 1, the major disparities among black and white men appeared at systolic blood pressures below 110 mm Hg or above 130 mm Hg and diastolic blood pressures above 70 mm Hg rather than at the extreme right tail of the blood pressure distribution. A similar pattern was observed among women (Figure 1). In the distribution analysis, it was also clearer that Mexican-American participants did not always experience the lowest blood pressures despite their overall lower prevalence rates of hypertension. As shown in Figure 1, a portion of Mexican-American participants had systolic pressures above 170 mm Hg, producing an extended right-tail in the distribution of systolic blood pressure that was hidden in the cutpoint analysis. In other words, there

Table 2.   Association Between Modifiable Risk Factors and Hypertension in the US National Health and Nutrition Examination Survey, 2003–2010[a]

Risk Factor	Race/Ethnicity					
	Non-Hispanic White		Non-Hispanic Black		Mexican-American	
	aOR	95% CI	aOR	95% CI	aOR	95% CI
Tobacco smoking	1.0	0.8, 1.2	1.3	0.9, 2.0	1.2	0.7, 2.1
Alcohol, log drinks/day	1.1	0.9, 1.3	1.2	0.9, 1.6	1.3	0.8, 2.0
Sodium, log mg/person/day	1.0	0.7, 1.3	1.1	0.8, 1.4	1.7	0.9, 3.2
Body mass index[b]	1.1	1.0, 1.1[c]	1.0	0.9, 1.1	1.2	1.1, 1.3[d]
Waist circumference, log cm	4.3	0.9, 18.6	8.8	0.9, 79.4	0.1	0.0, 3.0

Abbreviations: aOR, adjusted odds ratio; CI, confidence interval.

[a] All logistic regressions were adjusted for age and sex. Hypertension is defined as systolic blood pressure of at least 140 mm Hg or diastolic pressure of at least 90 mm Hg on average of 3 readings from the National Health and Nutrition Examination Survey (2003–2010). Risk factors included tobacco smoking (dichotomous variable; whether the participant smoked at least 100 cigarettes in their lifetime), alcohol consumption (continuous variable; average number of alcohol drinks per day over the past 12 months), sodium intake (continuous variable in mg/person/day, estimated as usual daily intake from two 24-hour dietary recalls), body mass index (continuous variable calculated from medical examination), and waist circumference (continuous variable measured in cm during medical examination). Skewed variables were log-transformed as shown.

[b] Weight (kg)/height (m)^2.

[c] $P < 0.01$.

[d] $P < 0.001$.

is a high-risk subgroup population among Mexican-American participants who are missed by conventional analyses.

Decomposition of risk factors on disparities between black and white participants

We analyzed how the observed disparities relate to 4 commonly discussed modifiable risk factors: tobacco smoking, alcohol drinking, sodium intake, and BMI. Analyzing the differences in hypertension prevalence through a standard logistic regression (Table 2) revealed that only 1 of the 4 modifiable risk factors—BMI—was significantly related to the odds of hypertension after adjustment for age and sex. These results were consistent with the logistic regressions repeated for the outcome of prehypertension, as well as for the outcomes of stage 1 and stage 2 hypertension (Web Table 3).

Through the decomposition analysis, a much more nuanced and detailed picture emerged of how risk factors alter overall disparities in systolic blood pressure distributions among groups. As shown in Web Figure 1A and 1B, the baseline blood pressure disparity between black and what participants was approximately 1–6 mm Hg higher among black men and women than among white men and women after inclusion of sample weights to estimate population-representative distributions. In decompositions in which we isolated the role of individual risk factors (Web Figure 1C and 1D), we observed that tobacco smoking patterns actually shifted the systolic blood pressure distribution for white participants upwards, narrowing the disparity between the 2 groups, particularly at high levels of systolic blood pressure (given the higher rates of smoking among white participants; see Web Table 1). This means that the disparity between white and black participants masked the fact that white men and women would be better off if lowering their tobacco smoking rates to be closer to those of black men and women (Web Table 1 and Web Figure 1C and 1D).

This fact was hidden by the larger association of other risk factors in the opposite direction. As shown in Web Figure 1C, sodium intake differences between white and black men pushed black male systolic blood pressure up significantly relative to the white male distribution at high levels of blood pressure ($P < 0.05$). Although the average sodium intake among black men is lower than the average among white men, the decomposition result alerted us to the fact that the difference reverses at very high levels of blood pressure; sodium intake becomes higher among black men than white men at the high end of the blood pressure distribution, worsening the disparity between black and white participants. Additionally, as shown in Web Figure 1D, BMI differences between white and black women are significant in increasing the disparity between them ($P < 0.05$). The largest contribution of BMI, however, was at lower levels of systolic blood pressure, where the systolic blood pressure distribution for black women was increased by as much as 2.5 mm Hg (95% confidence interval: 2.0, 3.0) because of the differences in BMI between black and white women. The greatest association was near the 45th percentile of the systolic blood pressure distribution (near 125 mm Hg).

Overall, although higher rates of tobacco smoking among white participants leads to a relative increase in systolic blood pressure, thereby narrowing the disparities between black and white participants, the association is negated by differences in sodium intakes and BMIs. Black-white disparities at principally lower systolic blood pressure levels were explained by BMI differences, and those at higher systolic blood pressure levels were significantly but only partially explained by differences in sodium intakes ($P < 0.05$).

At high levels of blood pressure around the 80th percentile (near 150 mm Hg), the association is such that being black and having levels of risk factors similar to those seen in white participants increases blood pressure—in other words, black participants with this blood pressure level have generally better risk factor characteristics than do their white counterparts, but the risk factors that we studied could not explain the higher blood pressure levels observed among black participants compared with white participants. This suggests, as shown in Web Figure 1C and 1D, that there is something else contributing to the black-white disparity—for example, differences in medical treatment or social conditions.

Decomposed impact of risk factors on disparities between Mexican-American and white participants

The decomposition analysis of disparities between Mexican-American and white participants similarly revealed a more nuanced set of relations between risk factors and disparities across the blood pressure distribution than did the standard cutpoint analysis. As illustrated in Web Figure 2, systolic blood pressure was approximately 1.7 mm Hg higher among white men and women than among Mexican-American men and women on average, but it was generally not statistically significant across the entire distribution at the level of $P < 0.05$ (Web Figure 2A and 2B).

Yet, despite this nonsignificant difference, the decompositions (Web Figure 2C and 2D) revealed important findings about how risk factor variations affect the gap between groups. We observed that tobacco smoking is increasing the gap systolic blood pressures between white and Mexican-American men by elevating systolic blood pressures in men (because tobacco smoking is higher among white men; see Web Table 1). If whites were to adopt the lower tobacco smoking rates seen in Mexican-American men (Web Table 1), the systolic blood pressure gap would be narrowed mostly at lower blood pressure ranges (<120 mm Hg). Conversely, BMI differences were again more important at lower ends of the systolic blood pressure distribution, where the inequality in BMI (which is higher in Mexican-American participants) inflated blood pressure levels in Mexican-American participants as compared with those in white participants among both men and women (Web Figure 2C and 2D).

Sensitivity analyses

Results from decomposition analysis of diastolic blood pressure distributions paralleled those from systolic blood pressure decomposition analysis (Web Figures 3 and 4). Results also remained consistent when repeating the decompositions on only those individuals who reported not currently taking antihypertensive medications (Web Figures 5 and 6) because there are similar distributional shapes for risk factors among persons who are not currently taking antihypertensive

medications and those who are. This result suggests that the antihypertensive medications produce a shift in the distributions overall, but each risk factor would influence disparities in the same direction regardless of medication use. Furthermore, our results also remained consistent with the primary results when we replaced BMI with waist circumference (Web Figures 7 and 8) or included both variables (Web Figures 9 and 10), except among men, in whom waist circumference is lower among black men than white men. Hence, the disparity between black and white men is actually reduced by the differences in waist circumferences per the decomposition (by increasing white men's blood pressure).

DISCUSSION

A critical epidemiologic challenge in health disparities research has been to find a strategy to compare entire distributions of a disease or risk factor of interest, clarifying how changes to differences in exposures or experiences of different groups can manifest in differences in outcomes of interest. To date, approaches that allow for comparisons across entire distributions have been limited by parametric assumptions that rarely apply to actual observed distributions of data and that limit the analysis of how risk factor changes may alter overall distributions of risk (9). In the present study, we applied a strategy of distributional decomposition (16) to analyze blood pressure disparities and their determinants.

This approach offers both substantive and methodological insights to the epidemiologic literature on high blood pressure. Substantively, we found that although a standard cut-point analysis using logistic regression would highlight only BMI as a key risk factor to explain disparities in prehypertension and various stages of hypertension, our decomposition approach provided several further insights, such as that differences in tobacco smoking that were influential in worsening systolic blood pressures in white participants compared with black participants were hidden in the standard cutpoint analysis and that white-black disparities were being narrowed at high blood pressure levels (but not in a healthy manner) by high rates of tobacco smoking among white participants. This is particularly interesting in the context of extant literature in which tobacco smoking is often correlated with lower blood pressure levels but confounded by low body mass among many smokers (11); in the present analysis, we can separate out the 2 risk factors to identify how blood pressure is related to smoking when taking into account the distribution of BMI. We compared participants who were members of racial/ethnic minorities to white participants both to parallel national disparities statistics that use white persons as a reference group (20–22) and to understand differences between the distributions of risk factors in the group often considered most "advantaged" and those groups considered less advantaged; we found that white participants were not always more advantaged in terms of their risk factor distributions, and indeed factors that elevated blood pressure in white participants were often hidden behind other factors that increased disparities between white and minority participants. Furthermore, BMI differences helped to explain disparities between black and white participants and between Mexican-American and white participants at principally lower systolic blood pressure

levels even among persons who were hypertensive, which also adds to the extant literature in which BMI differences are typically only correlated to overall hypertension disparities rather than understood to affect some but not all portions of the hypertensive blood pressure range (26). This implies that disparities among the cohorts with higher blood pressures may not be sufficiently narrowed by obesity reduction alone. Because we provided our statistical code in an "open source" manner, the decomposition approach to be applied to any number of other risk factors, groups, and outcome variables.

As with any statistical analysis of epidemiologic data, however, our analysis is subject to limitations inherent to the data itself. We did not consider persons in racial/ethnic groups other than non-Hispanic white, non-Hispanic black, and Mexican-American because of small sample sizes and inconsistent composition of the "other" racial/ethnic categories in NHANES data across survey waves. One such inconsistency includes the notable change in the composition of non–Mexican-American Latino populations due to sampling changes in 2007; this change prevented us from extending this analysis to non–Mexican-American Latinos. A further limitation is that NHANES is restricted to civilian noninstitutionalized populations. NHANES also uses a cross-sectional design, which provides only a 1-time estimate of blood pressure. The 1-time assessment can overestimate or underestimate blood pressure distributions in unpredictable directions, although this problem would not be expected to systematically affect 1 racial/ethnic group over another in a manner that would bias our analyses.

There are also key limitations inherent to our method that are important to note. One limitation of traditional decomposition is the "common support problem," which indicates that decomposition results are unreliable if there are limited cross-group differences in the distribution of covariates or regions of the covariate distributions that are only occupied by the comparison group and not the reference group. Conveniently, and purposefully, we chose to study hypertension risk factors for which white participants have a narrower distribution of values so that we did not have a common support issue. However, for cases in which such a dilemma arises, one strategy is to discard extreme tails of the distribution to yield an estimator that is consistent for the common support only. A second strategy is to assess whether the estimated value for a propensity score is such that there are no matches in the sample. A third strategy involves conducting multiple (at least 2) separate but analogous "subdecompositions," one in which the conditioning variables are those in group 1 (e.g., white participants) that have a narrower distribution and one in which the conditioning variables are those in group 2 (e.g., black participants) that have a narrower distribution. For each subdecomposition, the reference group becomes the group with the narrower distribution, so that in the counterfactual scenario, there is always common support. In both scenarios, it is the gap between 2 groups that is being estimated, and such an analysis avoids a problem of common support.

These issues raise opportunities for future research to optimize the algorithm presented here. Our initial analysis here creates a backbone example of the application of

decomposition as an analytical strategy for epidemiologists to compare whole population distributions of disease risk factors. The approach allows us to identify how modifications that may be achieved through public health interventions could affect different aspects of disparities across the full spectrum of risk in a population and reveal hidden relationships that might be obscured by other analytical approaches.

REFERENCES

1. Danaei G, Ding EL, Mozaffarian D, et al. The preventable causes of death in the United States: comparative risk assessment of dietary, lifestyle, and metabolic risk factors. *PLoS Med*. 2009;6(4):e1000058.
2. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA*. 2003;289(19):2560–2572.
3. Burt VL, Cutler JA, Higgins M, et al. Trends in the prevalence, awareness, treatment, and control of hypertension in the adult US population. Data from the health examination surveys, 1960 to 1991. *Hypertension*. 1995;26(1):60–69.
4. Keenan NL, Rosendorf KA; Centers for Disease Control and Prevention (CDC). Prevalence of hypertension and controlled hypertension—United States, 2005–2008. *MMWR Surveill Summ*. 2011;60(Suppl):94–97.
5. Boutitie F, Gueyffier F, Pocock S, et al. J-shaped relationship between blood pressure and mortality in hypertensive patients: new insights from a meta-analysis of individual-patient data. *Ann Intern Med*. 2002;136(6):438–448.
6. O'Donnell MJ, Yusuf S, Mente A, et al. Urinary sodium and potassium excretion and risk of cardiovascular events. *JAMA*. 2011;306(20):2229–2238.
7. Allen NB, Siddique J, Wilkins JT, et al. Blood pressure trajectories in early adulthood and subclinical atherosclerosis in middle age. *JAMA*. 2014;311(5):490–497.
8. Berger VW, Zhou Y. Kolmogorov–Smirnov tests. In: Everitt B, Howell D. *Encyclopedia of Statistics in Behavioral Science*. Hoboken, NJ: John Wiley & Sons, Inc.; 1999. http://onlinelibrary.wiley.com/doi/10.1002/0470013192.bsa332/full. Published October 15, 2005. Accessed May 19, 2014.
9. Hao L, Naiman DQ. *Quantile Regression*. New York, NY: Sage; 2007.
10. Cooper R, Cutler J, Desvigne-Nickens P, et al. Trends and disparities in coronary heart disease, stroke, and other cardiovascular diseases in the United States: findings of the National Conference on Cardiovascular Disease Prevention. *Circulation*. 2000;102(25):3137–3147.
11. Friedman GD, Klatsky AL, Siegelaub AB. Alcohol, tobacco, and hypertension. *Hypertension*. 1982;4(5 pt 2): III143–III150.
12. Klatsky AL. Alcohol, coronary disease, and hypertension. *Annu Rev Med*. 1996;47:149–160.
13. Klatsky AL, Gunderson EP, Kipp H, et al. Higher prevalence of systemic hypertension among moderate alcohol drinkers: an exploration of the role of underreporting. *J Stud Alcohol*. 2006; 67(3):421–428.
14. Mente A, O'Donnell MJ, Rangarajan S, et al. Association of urinary sodium and potassium excretion with blood pressure. *N Engl J Med*. 2014;371(7):601–611.
15. O'Donnell M, Mente A, Rangarajan S, et al. Urinary sodium and potassium excretion, mortality, and cardiovascular events. *N Engl J Med*. 2014;371(7):612–623.
16. DiNardo J, Fortin NM, Lemieux T. Labor market institutions and the distribution of wages, 1973–1992: a semiparametric approach. Cambridge, MA: National Bureau of Economic Research; 1995. http://www.nber.org/papers/w5093. Published April 1, 1995. Updated September 31, 1996. Accessed April 3, 2014.
17. Centers for Disease Control and Prevention. *National Health and Nutrition Examination Survey, 1999–2010*. Atlanta, MA: Centers for Disease Control and Prevention; 2011. http://wwwn.cdc.gov/Nchs/Nhanes/Search/nhanes_continuous.aspx. Accessed July 6, 2015.
18. Dodd KW, Guenther PM, Freedman LS, et al. Statistical methods for estimating usual intake of nutrients and foods: a review of the theory. *J Am Diet Assoc*. 2006;106(10):1640–1650.
19. Tooze JA, Midthune D, Dodd KW, et al. A new statistical method for estimating the usual intake of episodically consumed foods with application to their distribution. *J Am Diet Assoc*. 2006;106(10):1575–1587.
20. Centers for Disease Control and Prevention (CDC). Racial/ Ethnic disparities in the awareness, treatment, and control of hypertension—United States, 2003–2010. *MMWR Morb Mortal Wkly Rep*. 2013;62(18):351–355.
21. Ortiz E. *Hypertension Awareness and Control Programs at the National Heart, Lung, and Blood and Institute (NHLBI)*. Bethesda, MD: National Institutes of Health; 2009. http://iom.nationalacademies.org/~/media/Files/Activity%20Files/Disease/ReducingHypertension/Ortiz.pdf. Accessed July 6, 2015.
22. National Heart, Lung and Blood Institute. Activities to promote diversity and address health disparities. In: *NHLBI Fact Book, Fiscal Year 2012*. Bethesda, MD: National Institutes of Health; 2012. http://www.nhlbi.nih.gov/about/documents/factbook/2012/chapter12. Accessed July 6, 2015.

23. Centers for Disease Control and Prevention. *Continuous NHANES Web Tutorial*. Atlanta, GA: Centers for Disease Control and Prevention; 2013. http://www.cdc.gov/nchs/tutorials/Nhanes/index_continuous.htm. Accessed July 6, 2015.

24. Hill RC, Griffiths WE, Lim GC. *Principles of Econometrics*. 3rd ed. Hoboken, NJ: John Wiley & Sons, Inc.; 2008.

http://principlesofeconometrics.com/poe3/poe3toc.pdf. Accessed June 6, 2014.

25. Age standardization by the direct method. *J R Coll Gen Pract Occas Pap*. 1984;(26):7.

26. Egan BM, Zhao Y, Axon RN. US trends in prevalence, awareness, treatment, and control of hypertension, 1988–2008. *JAMA*. 2010;303(20):2043–2050.