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Illustrating a ‘Consequential’ Shift in the Study of Health Inequalities: A Decomposition of Racial Differences in the Distribution of Body Mass

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Abstract

Purpose—We present a conceptual introduction to ‘distributional inequalities’ - differences in distributions of risk factors or other outcomes between social groups - as a consequential shift for research on health inequalities. We also review a companion analytic methodology, ‘distributional decomposition’, which can assess the population characteristics that explain distributional inequalities.

Methods—Using the 1999–2012 U.S. National Health and Examination Survey, we used statistical decomposition to document gender-specific, Black-White inequalities in the distribution of body mass index (BMI), and assess the extent to which demographic (age), socioeconomic (family income, education), and behavioural predictors (caloric intake, physical activity, smoking, alcohol consumption) are associated with broader distributional inequalities in BMI.

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Results—Black people demonstrated favourable or no different caloric intake, smoking, or alcohol consumption than Whites, but worse levels of physical activity. Racial inequalities extended beyond the obesity threshold to the broader BMI distribution. Demographic, socioeconomic, and behavioral characteristics jointly explained more of the distributional inequality among men than among women.

Conclusions—Black-White distributional inequalities are present both among men and women, though the mechanisms may differ by gender. The notion of ‘distributional inequalities’ offers an additional purchase for studying social inequalities in health.

Keywords

obesity; BMI; distributions; health inequalities; race; Rose

Introduction

Overview

The field of epidemiology is currently engaged in self-reflection about whether its research is ‘of consequence’; whether it sufficiently concerns itself with the broad context for the production of population health, rather than the quest for evermore precise estimates of single causal factors^{1,2}. This paper contemplates a potentially consequential shift in approach to the epidemiology of health inequalities. Specifically, we formalize the notion of ‘distributional health inequalities³’, which we define as the difference in distributions of risk factors, or other health outcomes, between social groups within or between societies. Using an exemplar distributional issue, U.S. racial inequalities in body mass index (BMI), we provide an empirical workup to demonstrate how to investigate the extent and sources of distributional health inequalities

Distributional Inequalities as a Conceptual Shift in the Field of Health Inequalities Research

Based on Rose’s theoretical insights regarding the health of populations and their causes^{5–7}, we posit that this sizeable literature, and its underlying paradigmatic approach, still have two consequential shortcomings.

First, as Rose suggested, for many public health problems, risk is not well-characterized in dichotomous terms, but rather as some form of a continuous distribution^{3,5}. By contrast, the vast majority of health inequalities research examines social-group differences in the risk or likelihood of some binary outcome. For example, the health effects of body mass are known to extend throughout most of the distribution of body mass⁸, but a recent comprehensive review article demonstrates that studies of racial inequalities in body mass are largely relegated to investigating risk of obesity⁹.

Second, Rose argues that the health differences observed between two populations are not solely the consequence of differences in extreme risk factors, but also differences in subtler exposures that incrementally nudge or push a whole population, *en masse*, towards more adverse outcomes^{5,6}. For example, studies of racial inequalities in obesity often pursue

questions about exposure to severely unfavorable conditions – for example, the probability of living in severely unfavorable food environments, such as ‘food deserts’ and ‘food swamps’¹⁰. But, what if it is not only that more black people are exposed to such severe obesogenic conditions? What if greater obesity is also the result of ‘weaker’ exposures – those that don’t necessarily produce massive changes in body mass, but rather cause incrementally higher body mass - which nudges black people, *en masse*, towards a worsened BMI distribution (and therefore a larger proportion of obese people)⁵? For example, given prior work on higher levels of allostatic load in black people compared to white people¹¹ (thought to be the consequence of more stressful everyday living conditions^{12–14}) might racial differences in allostatic load nudge, or push black people *en masse* towards higher BMIs?

It is these shortcomings that are addressed by the notion of distributional inequalities, because the concept offers a whole-of-population orientation to health inequalities and to their causes.

Routine Epidemiological Methods Don’t Adequately Assessing Distributional Inequalities

The notion of distributional inequalities also demands a methodological departure for epidemiology. There are two primary reorientations to make explicit. The first is that, to understand distributional differences, we require methods which estimate continuous outcome distributions without imposing undue parametric assumptions^{15–17}. The second is that the methods must provide population-level parameter estimates, because the inequality is defined as a difference between populations, or groups, rather than a difference between individuals⁵.

At the moment, the most commonly applied distributional technique in epidemiology is quantile regression, which has a different goal: to disaggregate the overall, average relative risk of an outcome associated with some exposure (over the entire distribution of the outcome) in order to obtain separate relative risks at each specified quantile of the outcome distribution¹⁸. Quantile regression therefore: (a) does not model the whole distribution of an outcome (although one could conduct infinite quantile regressions to arrive at estimates for each quantile of a distribution, this is neither practical nor interpretable), (b) does not assess differences between two outcome distributions (nor the sources of difference)¹⁸ and, (c) does not provide population-level parameter estimates, instead providing quantile-specific, individual-level relative-risk estimates.

A recent resurgent interest in BMI distributions has sparked a ‘back-to-the-basics’ approach, whereby a set of more descriptive metrics (mean, standard deviation, 5th and 95th percentiles) have been used to assess distributional inequalities in BMI (by gender, race, and education)^{19,20}. While these metrics do provide population parameters for a continuous outcome, they do not assess the parametricity of distributions, nor analyze the predictors of observed distributional inequalities.

An oft-forgotten tool is the Kolmogorov-Smirnov test statistic (KST), which provides a global assessment of whether two distributions are significantly different from each other

(and thus meets the two main criteria) but does not detail differences at each point in the distributions, or provide a means for assessing predictors of these differences¹⁰.

Decomposition of Distributional Health Inequalities: A Tutorial

Distributional decomposition methods originate in the econometrics literature, and because it fulfills the criteria of (a) analyzing continuous, non/semi-parametric outcomes and, (b) producing population-level parameters of both distributional inequalities and their sources, we suggest that they offer the most robust means currently available for analyzing distributional inequalities^{15–17,22–24}. In what follows, we provide a brief tutorial and accompanying empirical workup to show how distributional decompositions can be applied to analyze distributional inequalities. Our purpose is not to provide all the technicalities of these methods, which are available elsewhere^{15,16}, but instead to convey the basics of the method as an introduction for an epidemiological audience.

Data Source and Variables

Our analyses draw on 1999–2012 data from the National Health and Nutrition Examination Survey (NHANES). Race was measured by self-report of being only non-Hispanic White or only non-Hispanic Black. BMI was calculated using interviewer-measured data (weight in kilograms divided by height in meters squared). Pregnant women were excluded from analyses, as were individuals with extreme BMIs (below 10 and above 50). The potential predictors of distributional inequalities we considered were those that are commonly known predictors of obesity (measured by self report):^{12–15} age (25–65 years), family income (<100% of the federal poverty line, 100%–199% of the federal poverty line, or >400% of the federal poverty line), highest level of education, health insurance status, total caloric intake (kcal) reported as the average of two 24-hour dietary recalls, physical activity (none, moderate, or vigorous) based on self-reported minutes per day, smoking status (non-smoker or smoker), and alcohol consumption (none, moderate, or heavy). Fewer than eight percent of subjects had missing data and were omitted from analyses. The final sample consisted of 3123 White men, 1287 Black men, 3024 White women, and 1364 Black women. Sample characteristics were described through weighted proportions of each variable category in each gender-race group (Table 1).

Basic Description of Distributional Inequalities

First, we examined the proportion of people in each commonly-used body mass category (underweight, normal weight, overweight, obese) in each gender-race group (Table 1). Next, we applied typical epidemiological methods to describe gender-specific racial inequalities in the distribution of BMI: (a) we replicated Krishna et. al's metrics (mean, standard deviation, 5th percentile of BMI, 95th percentile of BMI) (Table 2)¹⁹ and, (b) we calculated the KST to see if gender-specific racial distributions of BMI were globally different from each other (Table 2)²¹.

We then produced kernel density distributions of the outcomes for each of two populations or groups (e.g., the BMI distributions of black men and white men or, of black and white women, as presented in Figure 1). Next, we computed the difference (inequality) between the two kernel density distributions at each point of the two distributions. At a given point in

the distribution, this can be interpreted as the difference in the proportion of the population of one group (e.g., black men) and the proportion of the population of the other group (e.g., white men)^{15–17}. This was repeated for every point in the distribution and depicted graphically (Figure 1a and 1b)¹⁵. It is this distributional inequality that we sought to explain in the next step using distributional decomposition.

Distributional Decomposition

Distributional decomposition is often referred to as DiNardo-Fortin-Lemieux (DFL) decomposition, after its originators¹⁵. Using DFL, we assessed the extent to which each predictor variable entered in the model (and combined predictors) accounted for the distributional inequality at a given distributional point (Figure 2a and Figure 2b, supplemental Figure 1). Conceptually, the estimate for each predictor can be interpreted as the absolute proportion of the inequality at a specific point in the distribution that is explained by that predictor (or by combined predictors)³⁴. The estimation procedure involves mathematical reweighting, which constructs conditional distributions that identify the BMI distribution of one group, conditional on one or more predictor-profiles of the other group^{15–17,35}.

Following DiNardo et al. (1996), we denote $f(BMI, z | g)$ the joint distribution of BMI and individual-level predictors z conditional on group membership g , where z is a vector of individual-level predictors, and g an indicator variable = 0 for all non-Hispanic black and = 1 for non-hispanic white. Using these notations, the non-hispanic black BMI density can be written as:

$$f^B \equiv \int_{z \in \Omega_z} f(BMI, z | g=0) dz = \int_{z \in \Omega_z} f(BMI | z, g=0) \times f(z | g=0) dz$$

and counterfactual density of non-Hispanic black were they given the non-Hispanic white predictor-profile is:

$$f^C \equiv \int_{z \in \Omega_z} f(BMI | z, g=0) \times f(z | g=1) dz = \int_{z \in \Omega_z} \psi_z f(BMI, z | g=0) dz$$

where Ω_z is the domain of definition of the predictors in z capturing population characteristics and ψ_z is the reweighting function¹⁶

The ‘counterfactual’ scenario (in this case, counterfactual BMI distributions) is thus measuring how much the observed distribution of one group (the BMI distribution of black people) is expected to change if that group (black people) were to take on the predictor profile of the comparison group (white people)^{15,16,34}.

We use this counterfactual scenario to consider the following distributional decomposition of the observed (actual) differences between the BMI densities of black (f^B) and white (f^W):

$$f^B - f^W = [f^B - f^C] + [f^C - f^W]$$

The first term -- the difference between the actual BMI density of non-Hispanic black (f^B) and the counterfactual scenario (f^C) -- identifies the part of the gap explained by differences in predictor-characteristics, z . The second term -- the difference between the actual BMI density of non-Hispanic white (f^W) and the counterfactual scenario (f^C) -- measures distributional differences not accounted for by observable predictor-characteristics (i.e. the unexplained part). We could further factorize f^B into a product of conditional densities, and then consider more detailed decompositions of the racial BMI gap, which identify the distinct contribution of each predictor (or set of predictors) in separately.^{16,17} Stata code for replicating analyses provided in the Appendix³⁴. All analyses incorporated survey weights provided by NHANES.

Additional information on DFL decomposition, including major considerations (dependent relationship between predictors and the issue of ‘common support’), as well as comparison of DFL composition to more currently popular matching methods is available in the supplemental file.

Results

Descriptive Statistics (Tables 1 and 2)

Description of BMI distributions

Men: Differences in the proportions in each BMI category indicated that black and white men had differences in their BMI distributions (Table 1). Black men were more likely to be obese than white men (37.35% versus 32.60%), and were more likely to be underweight (1.27% versus 0.68%), but less likely to be normal weight (25.32% versus 26.01%) or overweight (36.07% versus 40.71%) ($p < 0.01$).

Black men had only a slightly higher mean BMI than white men (28.99 kg/m² versus 28.46 kg/m²) but, despite similar average BMI, the standard deviations of the two distributions were dissimilar, with black men more spread out compared to white men (6.26 kg/m² versus 5.34 kg/m²). The tails of the distribution were also longer for black men compared to white men. The left tails of the BMI distributions for black men versus white men differed less - at the 5th percentile of their population, black men had a BMI of 20.11 kg/m² while, for white men, this figure was 20.90 kg/m². On the other hand, the right tail was quite a bit larger for black men. At the 95th percentile, black men's BMI was 41.43 kg/m², while for white men it was 38.61 kg/m². Finally, the KST also indicated that, overall, the BMI distributions of White and Black men differed significantly ($p = 0.002$).

Comparison of kernel density distributions revealed that the key racial distinction in the BMI distributions of men was that white men had a larger proportion of individuals concentrated around the mean, while black men were more dispersed, with a greater proportion than whites at both tails of the distribution (Figure 1).

Women: Black and white women also had differences in their BMI distributions (Table 1). Black women were much more likely to be obese than white women (56.67% versus 33.83%), but were less likely to be in every other category ($p < 0.001$).

Distributional metrics also suggested a distributional inequality. The difference in mean BMI for women was slightly larger (31.75 kg/m² for black women compared to 28.12 kg/m² for white women). Black women also had a larger standard deviation in BMI compared to white women (7.06 kg/m² versus 6.67 kg/m²). Unlike for men, however, black women had a shorter left tail (at the 95th percentile, black BMI was 21.12 kg/m², while for white women it was 19.53 kg/m²). The right tail for black women was significantly longer than for white women (45.10 kg/m² versus 40.81 kg/m²). The KST suggested overall, the BMI distributions of white and black women differed significantly ($p < 0.0001$).

Description of Predictor-Characteristics—The predictor-profiles of black men and white men also differed (Table 1). Black men tended towards a socioeconomic profile that was riskier but, with the exception of physical activity, had a more favourable substance-use and health-behavioral profile. Women were similar to men in their profiles: black women had riskier socioeconomics, and a superior substance use profile. Black women also had a better behavioural profile than white women, with the exceptions of physical activity (which was worse among black women) and caloric intake (which was not statistically different across racial groups) (Table 1).

Distributional Decomposition (Figures 1 and 2 and Supplemental Figures 1 and 2)

Men: When black men were re-weighted to have the predictor-profile of white men, the BMI distribution of black men became narrower and more closely resembled the BMI distribution of white men. However, there still remained fewer Black men at the mean, and more on either tail (Figure 2, Supplemental Figure 2). The interpretation of these findings is that among men, differences in key, routine individual-level predictors associated with obesity play some role in explaining Black-White inequalities in the distribution of BMI, but do not explain the whole inequality.

Women: In contrast to racial inequalities among men, when the predictor-profile of white women was applied to black women, black women's counterfactual BMI distribution shifted very little towards the actual BMI distribution of white women. In absolute terms, although the right tail of black women's distribution shifted considerably towards the left (indicating fewer black women at higher BMIs), it was still larger than that the right tail for white women, and did not shift more Black women into the normal weight range (Figure 2, Supplemental Figure 3). In sum, routine predictors accounted for some of the differences between the BMI distributions of black and white women, but a substantial residual remained.

Discussion

We describe the notion of 'distributional inequalities' as a consequential way of understanding health inequalities. The main finding of our study is that, beyond racial inequalities in obesity, there are broader racial inequalities in the distribution of BMI, and that routine predictors of obesity offer some, but not full, explanation of these distributional

inequalities. Moreover, DFL decomposition makes the residual more apparent than in most health inequalities analyses, because it graphically depicts what has been left unexplained.

Our study is not without limitations. Predictors measured by self-report may not be accurate. Data limitations also prevent more nuanced specifications of several variables (e.g., income was not reported as a dollar amount and type of health insurance not captured). Perhaps the most significant limitation of our analyses is the lack of control for unreported or unknown sources of confounding. Unlike the methodological advances made in epidemiology and other social sciences to address this issue, distributional techniques lag in this respect^{36,37}.

Our study provides motivation and empirical guidance for examining distributional health inequalities, and suggests many directions for future investigation. Substantive issues include the societal conditions that are the cause of these inequalities. Methodological issues include improvements in accounting for unknown and unmeasured confounding.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1. Kernel Density Distributions of BMI and Decomposed Density Functions for Blacks and Whites, by Gender: National Health and Nutrition Examination Survey, 1999–2012 (N=8798)

Among men, racial differences in the distribution of BMI can be characterized by a similar mean, but greater proportions of Blacks on either tail of the BMI distribution; among women, the Black distribution of BMI is substantially right-shifted compared to the White distribution, resulting in heavier mean BMI and greater proportion of Black women in the right (heavier) tail of the BMI distribution.

Figure 2. Superimposed Counterfactual Kernel Density Distribution of BMI when Blacks are Reweighted with the Predictor Profile of Whites, by Gender: National Health and Nutrition Examination Survey, 1999–2012 (N=8798)

Among men, the counterfactual scenario (when the predictor profile of Whites is mathematically transferred to Blacks), creates a narrower black BMI distribution, but with distributional inequalities still evidence; among women, the counterfactual scenario explains even less of the distributional inequality.

Weighted Proportions Among Respondents Aged 18 Years and Older, by Gender and Race: National Health and Nutrition Examination Survey, 1999–2012 (N=8798)

Table 1

	Men		Women	
	White n=3123	Black n=1287	White n=3024	Black n=1364
BMI				
Underweight	0.68	1.27	2.31	1.10
Normal	26.01	25.32	36.97	15.69
Overweight	40.71	36.07	26.88	26.54
Obese	32.60	37.34	33.83	56.67
Age				
25–34	25.03	24.84	23.21	22.73
35–44	25.78	25.16	24.80	24.63
45–54	25.88	24.76	26.85	26.98
55+	23.31	25.24	25.13	25.66
Family Income				
<100% poverty line	10.91	19.98	11.97	23.39
100%–199% poverty line	16.20	22.05	16.90	25.95
200%–399% poverty line	27.14	31.37	28.08	27.42
>400% poverty line	45.75	26.59	43.06	23.24
Education				
Less than secondary	11.82	26.67	10.62	22.87
Secondary	25.36	29.86	23.91	23.90
Some post-secondary	30.42	28.98	32.51	35.48
Post-secondary	32.40	14.49	32.97	17.74
Health Insurance				
Uninsured	19.06	29.06	15.28	19.94
Insured	80.94	70.94	84.72	80.06
Total Caloric Intake (kcal)	2704.59	2546.89	1837.31	1838.64
Alcohol Consumption				

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	Men		Women		
	White n=3123	Black n=1287	White n=3024	Black n=1364	
None	13.25	22.93	26.69	48.83	p<0.001
Moderate	72.08	65.05	62.57	44.50	
Heavy	14.68	12.02	10.75	6.67	
Physical Activity					
None	33.38	42.12	33.86	51.69	p<0.001
Moderate	28.70	20.06	35.45	26.39	
Vigorous	37.92	37.82	30.69	21.92	
Smoking Status					
Non-smoker	43.93	44.98	51.42	63.05	p<0.001
Smoker	56.07	55.02	48.58	36.95	

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Distributional Inequalities in Body Mass Index, by Gender and Race: National Health and Nutrition Examination Survey, 1999–2012 (N=8798)

Table 2

	Men		Women	
	White	Black	White	Black
BMI				
Mean	28.46	28.99	28.12	31.75
SD ^b	5.34	6.27	6.67	7.06
5th Percentile	20.90	20.11	19.53	21.12
95th Percentile	38.61	41.43	40.81	45.10

^aKolmogorov-Smirnov Test Statistic

^bStandard Deviation