A New Approach for Quantifying Structural Racism and Discrimination with an Application to Alzheimer's Disease Risk

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Abstract

In this report we develop and apply a new methodological framework that allows researchers to measure the proportion of the total disparity in the risk of a disease associated with structural racism and discrimination (SRD). Oaxaca-Blinder-based decomposition generalized for censored data, rank-and-replace methods, and trend partitioning are the methodologic core of this framework which is then applied to the analysis of sources of racial disparities in AD risk. Arterial hypertension was found to be responsible for exceptionally large proportions of the total Black/White disparity in AD incidence and a major part of this effect was associated with dual eligibility––the SRD-related variable we used in the analysis. The post-onset treatment and management of arterial hypertension could be a main source generating the disparities. SRD, acting through multiple pathways before and after the onset of a risk-factor disease, can be a major contributing factor to these racial disparities.

Introduction

Racism and other forms of discrimination, both on the individual and structural level ensures that minority groups are more likely to have accumulated a significant stock of adverse health exposures directly leading to sizeable disparities in the risk of Alzheimer's disease and dementia across the racial spectrum of the U.S. Many of the adverse health outcomes experienced in later life are directly or indirectly impacted by structural racism through several distinct, but interrelated, pathways such as socioeconomic and occupational inequality, environmental injustice, social deprivation, and general healthcare-related inequities[1,2] driven by inadequate access to health insurance, healthcare facilities and substandard treatment of individual health conditions[1]. An important aspect of this problem are the persistent racial disparities in the incidence of Alzheimer's Disease (AD), related dementia and other neurocognitive disorders[3] which are diagnosed 1.33-1.50 times more often in Black individuals than in their White counterparts[4-6].

In this report we develop and apply a new methodological framework that allows researchers to measure the proportion of the total disparity in the risk of a disease (in our case AD) associated with structural racism and discrimination (SRD). This approach includes the sequential application of three innovative approaches. First, associations between AD risk and associated risk factors are calculated using the generalized Oaxaca-Blinder-based models[7-10] resulting in two effects for each predictor: exposure (higher prevalence of a risk factor in a population group) and vulnerability (higher effect of a risk factor in a population group)[11]. Then, the relative impact of SRD on the total vulnerability effect is assessed using rank-and-replace approaches[12,13] in which the distribution of non-SRD-related variables is equalized between the advantaged and disadvantaged populations creating pseudopoulations that differ only due to SRD-related variables. The contribution of SRD is obtained as the difference between the predicted effects of SRD-related variables in these pseudopopulations. The exposure effect, if large, is analyzed using trend partitioning to express this effect in terms of the relative contributions of causal risk factors over time and the contribution of SRD to the total exposure effect again extracted by application of rank-and-replace methods.

Methods: The methodology to measure SRD

Disparities in AD risk using Oaxaca-Blinder model generalized for censored data: effects of exposure and vulnerability. We used a Blinder-Oaxaca algorithm[7,8] adapted for use with censored longitudinal data[9,10] to identify the racial disparities in AD incidence between White and Black Medicare beneficiaries aged 65+ and explain them in terms of differences in exposure (a higher prevalence of the disease) and vulnerability (a higher effect of the disease on AD risk) to AD-risk-related diseases and all other risk factors[11]. Methodologically, the approach is based on Poisson regression fitted to person-period data and using piecewise constant age-specific intercepts. This results in a decomposition of the difference between the incidence rates for both groups,

$r_B - r_W = E + C = \sum_{k=1}^{K} E_k + \sum_{k=1}^{K} C_k$, where r_B and r_W are the incidence rates in the Black and White subgroups; k is the summation index that enumerates K risk factors including diseases/disease patterns and age groups (i.e., the age-specific intercepts reflecting the effects of all other variables not included in the model); E_k and C_k are the effects of exposure and vulnerability which are expressed in terms of race-specific prevalence of risk factors explicitly included in the model and their estimated effect sizes on AD risk based on a race-specific Poisson generalized linear model (PGLM).

Identifying the SRD effect in differences in vulnerability using rank-and-replace methods. The rank-and-replace[12,13] method is then used to identify the proportion of the total effect accounted for by SRD. Such method allows us to quantify the effect of any given subset of predictors (e.g., those judged to be non-SRD-related) by equalizing the distribution of non-SRD predictors between two population groups. Specifically, the variables of interests are ranked (by the value of a linear predictor representing the effects of all non-SRD-variables) within two race/ethnicity-specific subpopulations, and then the values of the linear predictors in the Black (or other disadvantaged) subpopulation are replaced with those of the White group in order of the created rank. The fraction of the total disparity due to SRD is obtained by comparing the estimates in the original model with those obtained after the rank-and-replace procedures are performed. In PGLM the race specific AD rates are expressed as $R_r = \exp\left(b_r + \sum_i \beta_i^{SRD} x_i^{SRD} + \sum_i \beta_i^{non-SRD} x_i^{non-SRD}\right)$. Averaging of $\log R_r$ over race-specific group gives three terms: $b_r + b_r^{SRD} + b_r^{non-SRD}$. The ratio of their exponents gives the estimate of HR of AD for Black vs. White individuals which has three respective components: $HR = HR_b \cdot HR_{SRD} \cdot HR_{non-SRD}$. These components can be estimated for the original dataset and for the dataset after the rank-and-replace procedure. HR_b and HR_{SRD} are equal for both datasets, and $HR_{non-SRD} = 1$ for the dataset after the rank-and-replace procedure.

Identifying the SRD effect in differences in exposure using partitioning methods. Trend partitioning approach for two diseases is used for analyses of the exposure effect detected using the generalized Oaxaca-Blinder approach. Traditionally, such partitioning treats disease prevalence and mortality as its primary outcomes and differences in incidence and survival as its primary causal factors (as applied to diabetes mellitus[14,15], lung cancer[16], bladder cancer[17], and Alzheimer's disease[18]). In this study, two modifications are implemented for the analyses of SRD effects: i) the onset of an outcome (e.g., AD) will replace mortality (i.e., survival-time from a risk factor disease represents the time to AD onset), ii) death will be considered as an alternative censoring event. This generalized partitioning approach is then applied to risk-factor diseases with high exposure effects previously identified using Oaxaca-Blinder-based models to quantify the causes of this exposure effect in terms of racial difference in a risk-factor disease's incidence, post-onset survival (time to AD), and its prevalence at data entry (age 65). Age-time combinations characterized by abnormally strong/weak absolute values/rates of the change in exposure-related disparity sizes or magnitude of the contributing components will be identified for further analysis using rank-and-replace methods.

Results: application of the methodology to the racial disparities in AD risk

We included the following AD-risk-related diseases: arterial hypertension[19,20], cerebrovascular disease[21,22], several other disease of the circulatory system[23-26] (including ischemic heart disease, atherosclerotic cardiovascular disease, and heart failure), diabetes mellitus[27,28], renal disease[29,30], traumatic brain injury[31], and depression[32,33]. First, we used a classic disease indicator model and found that (Table 1): arterial hypertension was the leading contributor to the disparities with a large gap between it and depression,

Table 1. Select Results of Oaxaca-Blinder Decomposition

	Exposure	Vulnerability	Total	Prevalence		PGLM Coefficient	
				Black	White	Black	White
Classic Disease Indicators Model							
Arterial hypertension	46.0	204.5	250.5	70.8	63.6	0.57	0.05
All Other Factors	-165.0	15.0	-150.5				
Disease Pattern Model							
Hypertension & Diabetes	77.4	68.0	145.4	17.2	10.6	0.88	0.29
All Other Factors	-208	162.3	-45.4				

the next most influential contribution. Then, diseases with minor contributions (total effect <5%) were excluded from further analysis and 32 morbidity-profile indicators of all possible mutually exclusive combinations of the remaining diseases (arterial hypertension, cerebrovascular disease, diabetes mellitus, renal disease and depression) were used as predictors. This analysis confirmed the role of arterial hypertension as the most powerful modifiable predictor of racial disparities in AD incidence and identified the combination of hypertension and diabetes as highly influential within that relationship (with stroke, and renal disease playing notable, but significantly lesser roles). Notably, even though prevalence of hypertension was, as expected higher in Blacks, only 20% of the associated total effect was due to exposure while 80% was due to vulnerability.[34]

We then selected the race-specific populations of individuals with arterial hypertension and performed rank-and-replace analysis to isolate the effect of SRD from the total vulnerability effect. We used the AD-risk-related diseases (excluding hypertension) from the previous step of analysis to represent the effects of non-SRD-related health factors and dual eligibility (a proxy for poverty and poor living conditions available in Medicare data) to represent the effects of lifetime exposure to SRD. The PLGM was estimated for the combined all-race population with race-specific intercepts. Then an individual linear predictor representing the sum of the effects of all non-SRD-related health factors (e.g., $\beta_1 X_1 + \beta_2 X_2 ...$) was calculated and ranked for each race-specific group separately. Individuals in Black and White groups were matched in percentile groups by the values of the linear predictor and then the individual values for non-SRD-related variables for Black were replaced by their respective values from the White population. Finally, the originally estimated PLGM with the replaced non-SRD-variables is used for predictions. The estimated age-adjusted rates of AD were 2,850 (per 100,000) for White and 3,482 for Black individuals with hypertension. Pure intercept-based rates are a little higher for Whites: 1,582 vs. 1,497. The difference between total and pure intercept-based rates are determined by the contributions of SRD- and non-SRD-related variables which are respectively distributed as 39% and 61% for White and 59% and 41% for Black. The hazard ratios associated with SRD were $\exp(b_{Black}^{SRD}) = 1.57$ and $\exp(b_{White}^{SRD}) = 1.22$ and, therefore, responsible for a 35% higher risk of AD incidence in Blacks with only minor differences in race-specific intercepts and the contribution of non-SRD variables remaining after the effect SRD variables to racial disparities was accounted for. The observed risk ratio for total rates is 1.22 that is lower comparing to the ratio that follows from Table 1 (1.68 obtained as exp(0.57)/exp(0.05)); the difference is due to matching in the rank-and-replace procedure and multivariable modeling for Table 1. The two contributions to the observed risk ratio come from pure intercept contribution $HR_b = 0.95$ and the contribution from SRD variables $HR_{SRD} = 1.29$. The contributions of non-SRD variables to the total rates were similar for both races, therefore $HR_{non-SRD}$ is close to one and only minor effect of the rank-and-replace procedure that must result in exact equality $HR_{non-SRD} = 1$, was detected.

A more diverse selection of SRD and non-SRD-related predictors representing different pathways of both the impact of SRD and AD onset for individuals with arterial hypertension and possibly other risk-factor diseases will be presented at the PAA 2022 meeting.

Discussion and Conclusion

The objective of the study was to assess the impact of SRD on racial disparities in the risk of AD. To address this problem, we developed a new methodologic approach for quantifying the effect of SRD in AD risk through the sequential application of i) generalized Oaxaca-Blinder-based models resulting in exposure and vulnerability effect foreach predictor, ii) the rank-and-replace methods to evaluate the relative impact of SRD on the total vulnerability effect, iii) the generalized partitioning approach to analyze the effect of exposure. Application of this methodology to the analysis of Black/White disparities in AD incidence in Medicare-eligible individuals age 65+ found that: i) chronic age-related diseases especially arterial hypertension were responsible for exceptionally large proportions of the total Black/White disparity in AD incidence with about 20% of the total contribution due to differences in prevalence (exposure effect) and 80% to differences in the effects of the morbidity profile on AD risk (vulnerability effect); and ii) a significant part of the vulnerability effect was associated with SRD. The results of our study allowed us to speculate that the primary determinants of racial disparities in AD incidence are differences in exposure and vulnerability to risk-factor diseases (first of all, arterial hypertension). The post-onset treatment and management of these conditions could be a main source generating the disparities. SRD, acting through multiple pathways before and after the onset of a risk-factor disease, can be a major contributing factor to these racial disparities.

The methodology used in this study requires splitting the available explanatory variables into two categories: SRD-related and non-SRD related. The non-SRD-related variables can include disease-patterns, physical

function status, genetics, health-related behavior, and basic demographic indicators. SRD-related variables can include[1]: indices of segregation, local access-to-care, education, life-long employment characteristics and other factors associated with financial well-being, and concentrated disadvantage (e.g. environmental injustice etc.) as well as treatment characteristics. Since individuals at 65 are facing the result of a lifetime of experience and exposure to SRD, factors associated with SES and other potential mediators between SRD and the outcome, can be treated as SRD-related. Our study used a very limited set of SRD- and non-SRD-predictors, and its quality can be improved through inclusion of a wider set of variables reflecting systemic SRD in individuals with arterial hypertension and possibly other risk-factor diseases.

Only two steps of our methodology were applied in the illustrative example in this report. The last step containing the analyses of exposure effect using the trend partitioning analysis for disparities was not used because of the fairly minor effects of exposure to hypertension on AD risk (5 times less than the effect of vulnerability). Here we briefly illustrate how this approach would work. Figure 1 shows the results of the extended version of trend partitioning analysis[14-18] to analyze disparities in arterial hypertension prevalence between Black and White subpopulations in terms of four causal components: incidence, survival, and pre-existing prevalence at data entry (65 years) and pre-existing prevalence at data entry (1992 year). The results show that, over time the racial disparities in arterial hypertension prevalence are overwhelmingly the result of racial differences in initial morbidity, i.e., prevalence at 65, following by the contributions from survival and incidence that are much less important. In 2015, the four components had the relative contributions: prevalence at 65: 158.5%, prevalence at 1992: 7.4%; incidence: -53.4%, and survival: -12.4%. negative percentage means that the component gives the contribution opposite to the total prevalence disparities, i.e., these components act to make hypertension prevalence higher for White individuals. An approach to explain why race-specific prevalence of arterial hypertension at 65 are so different requires attraction of additional datasets that can provide health-related information for populations before age 65.

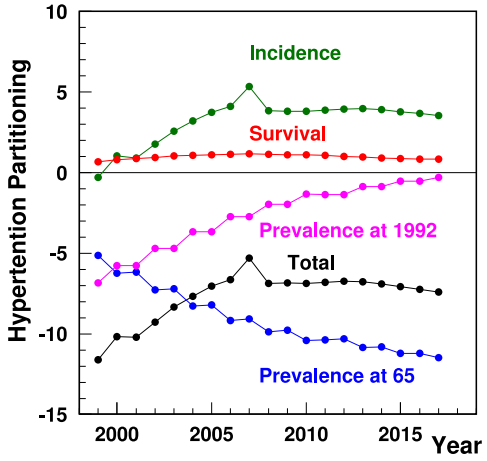

Figure 1. Partitioning of disparities in arterial hypertension age-adjusted prevalence (in %). Black curve shows total effect and other curves represents the effects of components.

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