

# Vulnerability to Hypertension Is a Major Determinant of Racial Disparities in Alzheimer's Disease Risk

Igor Akushevich,<sup>1,\*</sup> Stanislav Kolpakov,<sup>1</sup> Arseniy P. Yashkin,<sup>1,\*</sup> and Julia Kravchenko<sup>2</sup>

## BACKGROUND

Higher incidence levels of Alzheimer's disease (AD) in Black Americans are well documented. However, quantitative explanations of this disparity in terms of risk-factor diseases acting through well-defined pathways are lacking.

## METHODS

We applied a Blinder-Oaxaca-based algorithm modified for censored data to a 5% random sample of Medicare beneficiaries age 65+ to explain Black/White disparities in AD risk in terms of differences in exposure and vulnerability to morbidity profiles based on 10 major AD-risk-related diseases.

## RESULTS

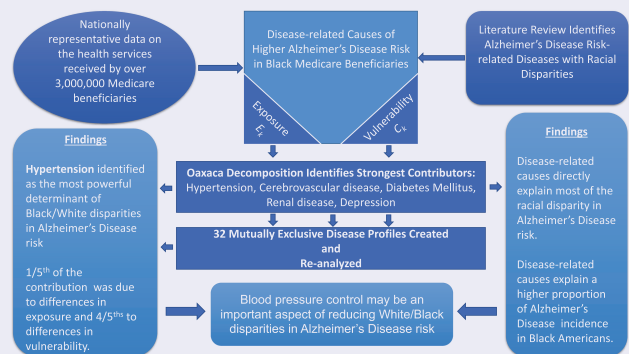
The primary contribution to racial disparities in AD risk comes from morbidity profiles that included hypertension with about 1/5th of their contribution due to differences in prevalence (exposure effect) and 4/5ths to differences in the effects of the morbidity profile on AD risk (vulnerability effect). In total, disease-related effects explained a higher proportion of AD incidence in Black Americans than in their White counterparts.

## CONCLUSIONS

Disease-related causes may represent some of the most straightforward targets for targeted interventions aimed at the reduction of

racial disparities in health among US older adults. Hypertension is a manageable and potentially preventable condition responsible for the majority of the Black/White differences in AD risk, making mitigation of the role of this disease in engendering higher AD incidence in Black Americans a prominent concern.

## GRAPHICAL ABSTRACT



**Keywords:** Alzheimer's disease; blood pressure; hypertension; Medicare; older adults; racial disparity.

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Alzheimer's disease (AD) is a progressive neurodegenerative disorder that is associated with a significant financial and health-related burden on affected individuals, their families, and the US healthcare system.<sup>1</sup> This burden is not evenly distributed across racial groups: Black Americans consistently demonstrate higher AD incidence and prevalence than their non-Black counterparts.<sup>1-3</sup> The effects of such differences go beyond their direct impact on AD, affecting the onset of other diseases, including those associated with higher AD

risk. Indeed, many diseases known to be associated with increased risk of AD onset such as diabetes mellitus,<sup>4</sup> hypertension,<sup>5,6</sup> renal disease,<sup>7</sup> depression,<sup>8</sup> cerebrovascular disease,<sup>9</sup> cardiovascular diseases,<sup>1,10</sup> and traumatic brain injury<sup>11</sup> have notable race-related differences<sup>12-15</sup> and can contribute to disparities in AD risk.

In this study, we use a modified Blinder-Oaxaca algorithm adapted for use with censored longitudinal data<sup>16,17</sup> to identify the race-related differences in AD incidence between

Correspondence: Arseniy P. Yashkin ([apy4@duke.edu](mailto:apy4@duke.edu)).

\* Co-first authors: Akushevich and Yashkin have contributed equally to the manuscript.

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<sup>1</sup>Biodemography of Aging Research Unit, Social Science Research Institute, Duke University, Durham, North Carolina, USA; <sup>2</sup>Department of Surgery, Duke University School of Medicine, Duke University, Durham, North Carolina, USA.

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White and Black Medicare beneficiaries age 65+ and decompose this difference into two components: (i) the proportion of the total effect due to the differences in exposure (e.g., differences in prevalence levels) to diseases known to be risk factors for AD; and (ii) the proportion of the total effect due to differences in the vulnerability (e.g., differences in the effect of a disease on AD risk) to diseases known to be risk factors for AD. This approach allows for (i) identifying and ranking the main disease-related causes of the disparities in AD incidence between White and Black Americans; (ii) quantifying the size of the effect as well as the primary pathway (e.g., exposure, vulnerability, or both) by which it is generated; and (iii) assessing the combined magnitude of the effects of other factors, not available in the data and/or included in the modeling.

## METHODS

We used administrative claims data drawn from a nationally representative 5% sample of the total US Medicare population over the 2000–2017 period (5%-Medicare). This data provides information on the diagnoses made and procedures performed during episodes of care paid for by either Medicare Part A (facility-based services) or Medicare Part B (professional services), along with basic demographic and enrollment information about the beneficiaries.

The baseline date was defined as the earliest date when an individual had full traditional fee-for-service Medicare Parts A and B coverage after reaching the age of 65. Individuals were then observed until reaching the end of their follow-up period defined as the earliest among the date of AD onset, date of death, or censoring with the latest possible cutoff date being December 31, 2017. Individuals enrolled in traditional Medicare for less than 80% of their total follow-up time were excluded from the analysis. The final sample size consisted of 3,121,553 White and 320,720 Black Medicare beneficiaries.

The onset of AD was the primary outcome of interest. Ten diseases were selected based on the results of a literature review of probable AD disease-related risk factors and the presence of race-related differences in the epidemiology of these conditions: hypertension,<sup>5,6,18</sup> cerebrovascular disease,<sup>9</sup> several other diseases of the circulatory system<sup>1,10</sup> (including ischemic heart disease, atherosclerotic cardiovascular disease, and heart failure), diabetes mellitus,<sup>4</sup> renal disease,<sup>7</sup> traumatic brain injury,<sup>11</sup> and depression.<sup>8</sup>

Disease onset was assigned to the date of the first claim where an appropriate International Classification of Disease-9/10 code (Supplementary Table S1 online) was recorded and confirmed by the presence of a second distinct claim with the same code within 90 days. Death occurring within this period was treated as a confirmatory record.<sup>19</sup> The age-specific incidence rates were calculated for 29 age groups: one-year groups for ages 65–90, and multi-year groups for ages 90–91, 92–94, 95–99, 100–110; ages > 110 were considered invalid and censored. These rates were then age-adjusted using the US standard population for the year 2000.

In the first step of the analysis, indicators of the presence of the 10 study diseases were used as predictors of racial disparities in AD risk. Diseases with minor contributions (total effect of <5%) were excluded from further analysis. The second step

of the analysis, the primary focus of this manuscript, used 32 indicators of specific morbidity profiles comprising all possible combinations of the remaining 5 diseases (hypertension, cerebrovascular disease, diabetes mellitus, renal disease, and depression) as predictors of racial disparities in AD risk instead of the disease indicators used in the first step.

The extent to which each condition contributes to the total racial disparity in AD risk was calculated using the modified Blinder-Oaxaca decomposition generalized by Powers and Yun<sup>16,17</sup> for the use of censored data. This modification was based on Poisson regression with piecewise constant intercepts fitted to person-period (split-age-group) data. This allows for estimation of the contributions of exposure and vulnerability to the difference between the race-specific risks of AD. The hazard rate decomposition results in:

$$r_B - r_W = E + C = \sum_{k=1}^K E_k + \sum_{k=1}^K C_k$$

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/morbidity profiles and age groups (i.e., the age-specific intercepts reflecting the effects of all other variables not included in the model);  $E_k$  is the effect of exposure or the difference in the prevalence of each risk factor between the Black and White subgroups;  $C_k$  is the effect of vulnerability or the difference in the magnitude of the effect of a risk factor between the Black and White subgroups. Specifically,

$$E_k = \frac{\beta_{Bk}(f_{Bk} - f_{Wk})}{\sum_k \beta_{Bk}(f_{Bk} - f_{Wk})} (F_{BB} - F_{WB}), \quad C_k = \frac{f_{Bk}(\beta_{Bk} - \beta_{Wk})}{\sum_k f_{Bk}(\beta_{Bk} - \beta_{Wk})} (F_{WB} - F_{WW}).$$

Thus, evaluation of  $E_k$  and  $C_k$  includes calculation of the race-specific prevalence of morbid conditions ( $f_{Bk}$  and  $f_{Wk}$ ) within each age group and the estimation of effect sizes ( $\beta_{Bk}$  and  $\beta_{Wk}$ ) of risk factors on AD risk in a race-specific Poisson generalized linear model (PGLM) with no intercept. The equations for  $E_k$  and  $C_k$  contain four rates  $F_{WW}$ ,  $F_{WB}$ ,  $F_{BW}$ , and  $F_{BB}$  predicted by the PGLM, where the first and second sub-indices indicate that the risk factor prevalence (first index) and effect size (second index) were taken from the respective race-specific subpopulation. We note that the total exposure and vulnerability effects are  $E = \sum_k E_k = F_{BB} - F_{WB}$  and  $C = \sum_k C_k = F_{WB} - F_{WW}$ , and the rates  $F_{WW}$  and  $F_{BB}$  coincide with race-specific crude rates estimated empirically. In our tables, we report transformations of  $E_k$  and  $C_k$ :  $\tilde{E}_k = E_k / (r_B - r_W)$  and  $\tilde{C}_k = C_k / (r_B - r_W)$ . This improves the practical interpretability of the results by ensuring that  $\sum_k (\tilde{E}_k + \tilde{C}_k) = 1$  or 100%.<sup>16</sup>

## RESULTS

Descriptive statistics, aggregated across the entire study period are presented in Table 1. The study-wide proportion of AD onset was similar for both races: 6.97% in Whites ( $N = 3,121,553$ ) and 7.03% in Blacks ( $N = 320,720$ ). The baseline ages between the two groups (67.1 for Blacks, 67.5

**Table 1.** Summary statistics<sup>a</sup>

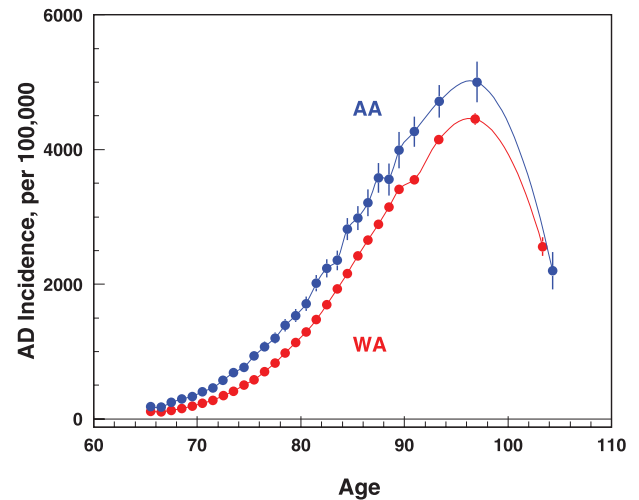
	White	Black
N	3,121,553	320,720
Female	57.4	58.9
Age at baseline	67.5(5.3)	67.1(5.0)
Follow-up (years)	10.1 (7.1)	8.6(6.9)
Alzheimer's disease	6.97	7.03
Age at Alzheimer's disease onset	83.8 (7.3)	82.3 (8.0)
Death	36.6	33.9
Age at death	83.0 (8.8)	80.5 (9.2)
Hypertension	66.1	71.3
Cerebrovascular disease	22.9	23.5
Diabetes mellitus	27.0	39.1
Renal disease	19.7	26.6
Depression	17.1	11.6
Ischemic heart disease	34.3	30.9
Heart failure	24.2	25.2
Atherosclerotic cardiovascular disease	4.3	3.8
Other diseases of circulatory system	57.1	53.0
Traumatic brain injury	5.5	3.0

<sup>a</sup>Numbers presented are sample proportions or means (SD) as appropriate.

for Whites) were comparable, Blacks showed lower ages of onset for both AD (82.3 vs. 83.8) and death (80.5 vs. 83.0) and therefore lower overall follow-up times (8.6 years vs. 10.1 years). Hypertension was the most frequent disease in both race groups, followed by other circulatory diseases, ischemic heart disease, and diabetes. Blacks had higher levels of hypertension (71.3% vs. 66.1%), diabetes (39.1% vs. 27.0%), and renal disease (26.6% vs. 19.7%), but lower levels of depression (11.6% vs. 17.1%).

The age-adjusted incidence rates of AD, per 100,000 individuals, were 904 (95% confidence interval [CI]: 900–908) for Whites and 1,210 (95% CI: 1,195–1,225) for Blacks. The corresponding disparity in AD incidence was 306 per 100,000 person-years. Similarly, the age-specific incidence of AD was significantly higher for the Blacks in all age groups (Figure 1) and reaches the inflection point for exponential growth 5 years later (age 80) than in White counterparts (age 75).

The results of the first step of the analysis (i.e., Oaxaca-Blinder approach applied to 10 individual diseases) showed that the strongest contribution to racial disparities in AD risk between Black and White populations was due to hypertension (Table 2; Panel A), with differences in exposure (race-specific prevalence levels) and vulnerability (race-specific effect sizes on AD risk) accounting respectively for 46.0% and 204.5% of the total racial difference in AD risk (offset by disparity-reducing effects of other factors). The impact of hypertension was stronger than the effect of all other diseases combined. Five diseases (Supplementary Table 2 online; Panel A) including atherosclerotic cardiovascular disease, heart failure, ischemic heart disease, other diseases of the circulatory system, and traumatic brain



**Figure 1.** Age-specific incidence of Alzheimer's disease. Age-specific incidence per 100,000 (dots) and associated 95% confidence intervals (bars) for African Americans (blue dots/bars) and White Americans (red dots/bars).

injury demonstrated minor contributions (total effect less than 5%) and were excluded from further analyses of morbidity profiles.

The results of the second step of the analysis (i.e., Oaxaca-Blinder approach applied to 32 morbidity profiles) confirmed the leading role of hypertension in explaining the racial disparities in AD risk with relatively small contributions of profiles not including hypertension. Therefore, we focused on profiles involving hypertension (Table 2; Panel B) with the full results presented in Supplementary Table S2 online for reference. In addition to the effects of exposure ( $\tilde{E}_k$ ) and vulnerability ( $\tilde{C}_k$ ), we present the race-specific estimates of disease/morbidity profile prevalence for Black ( $f_B$ ) and White ( $f_W$ ) Americans obtained using empiric analysis and the PGLM response coefficients for Blacks ( $\beta_{Bk}$ ) and Whites ( $\beta_{Wk}$ ) populations. The full results of PGLM models are presented in Supplementary Tables S3 and S4 online.

The combined contribution of morbidity profiles that included hypertension, the most substantial contributors to higher AD risk in Black Americans, was equal to 426.3% of the total size of the racial disparity ( $\approx 306$  per 100,000 person-years). Of this contribution, 83.4% and 342.0% (or 1/5th and 4/5th of the total hypertension effect of 426.3%) were associated with the exposure and vulnerability pathway respectively. The most pronounced contribution was from the hypertension + diabetes morbidity profile (145.4%), with 77.4% of this contribution due to the exposure and 68.0% due to the vulnerability pathway. Morbidity profiles of hypertension combined with cerebrovascular or renal disease also demonstrated high contributions to racial disparities in AD risk, but their impact was less pronounced compared to the hypertension + diabetes morbidity profile. In the White population, there was a substantial contribution (acting to lower the size of the disparity in AD incidence) from depression-related morbidity profiles with the most pronounced contribution associated with the hypertension + depression ( $-36.3\%$  total:  $-44.4$  exposure;  $8.0$  vulnerability) morbidity profile.

**Table 2.** Results of Oaxaca-Blinder decomposition<sup>a</sup>

	Exposure	Vulnerability	Total	Prevalence		PGLM coefficient <sup>t</sup>	
				Black	White	Black	White
Panel A: disease indicators							
Hypertension	46.0	204.5	250.5	70.8	63.6	0.57	0.05
Other diseases	-36.7	1.7	-35.0				
Age-specific effect of all other factors <sup>b</sup>	-128.3	12.8	-115.5				
Panel B: morbidity profiles <sup>c</sup>							
Hypertension (combined)	83.4	342.9	426.3				
HT + DM	77.4	68.0	145.4	17.2	10.6	0.88	0.29
HT	-41.6	122.6	81.0	24.9	27.9	1.04	0.31
HT+DM + RD	43.2	25.3	68.5	5.1	2.2	1.15	0.40
HT + CB + DM + RD	38.5	17.7	56.3	2.9	1.3	1.76	0.85
HT + CB + DM	31.5	23.7	55.2	4.4	3.0	1.70	0.89
HT + RD	14.5	18.2	32.6	3.3	2.4	1.27	0.44
HT + CB + DM + RD + DP	13.4	4.7	18.1	1.0	0.6	2.28	1.60
HT + CB + RD	3.6	8.5	12.1	1.3	1.1	1.79	0.78
HT + DM + RD + DP	4.9	3.4	8.3	0.8	0.6	1.99	1.33
HT + CB + DM + DP	3.4	4.4	7.8	1.0	0.9	2.30	1.64
HT + DM + DP	-0.8	5.9	5.1	1.5	1.5	1.94	1.35
HT + RD + DP	-4.1	2.3	-1.9	0.4	0.6	2.06	1.23
HT + CB + RD + DP	-3.7	1.6	-2.1	0.3	0.4	2.26	1.50
HT + CB	-31.3	24.9	-6.3	4.3	5.7	1.71	0.85
HT + CB + DP	-21.2	3.7	-17.4	0.7	1.4	2.36	1.60
HT + DP	-44.4	8.0	-36.3	1.7	3.4	2.01	1.32
All other morbidity profiles	-61.6	13.7	-47.9				
Age-specific effect of all other factors <sup>b</sup>	-152.1	-126.3	-278.4				

Abbreviations: AD, Alzheimer's disease; PGLM, Poisson generalized linear model.

<sup>a</sup>Model approximation errors were not more than 4% relative to the age-adjusted AD risks

<sup>b</sup>Presented in detail in [Figure 2](#).

<sup>c</sup>HT, hypertension; CB, cerebrovascular disease; DM, diabetes mellitus; RD, renal disease; DP, depression: since these are based on split-episode person-years their prevalence are necessarily  $\leq$  the general sample summary in [Table 1](#).

The strong effect of hypertension driving the disparities in AD risk between White and Black populations is partially offset by the disparity-reducing contributions from the age-specific unexplained effects and, to a lesser extent, non-hypertension-related morbidity profiles. [Figure 2](#) illustrates comparative age-specific intercepts reflecting the effects associated with all other variables not included in the model (or the remaining effect unexplained by the differences in the diseases/morbidity profiles) from both the disease indicator and morbidity profile-based models as well as the logarithms of age-specific estimates of AD incidence that correspond to the intercept-only model. The estimates for the model with morbidity profiles are much lower than those for the model with disease on indicators. This means that the disease-indicator model has a higher proportion of unexplained effects and therefore the model with morbidity profiles is the better model.

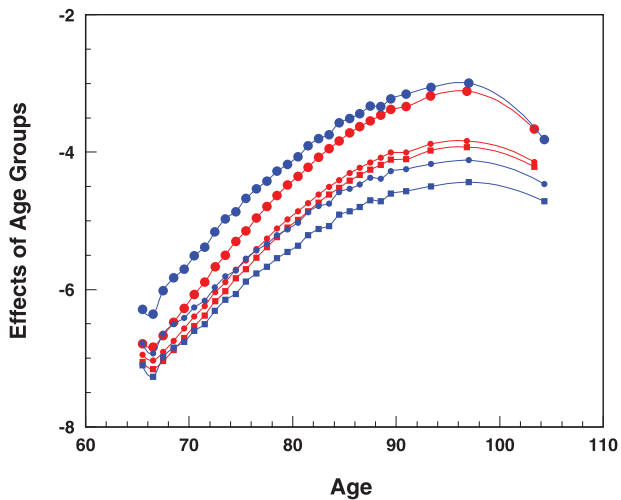
Finally, to check the stability of the results, a series of sensitivity analyses focusing on the effect of hypertension

was performed. The results (summarized in [Supplementary Table S5](#) online) show that our findings were consistent across multiple alternative specifications and are discussed as needed below.

## DISCUSSION

Using a Blinder-Oaxaca decomposition modified for use with censored data,<sup>16,17</sup> we evaluated the relative impacts of 2 pathways (exposure and vulnerability) through which a spectrum of AD-risk-related diseases contributed to the disparities in AD incidence between White and Black Medicare beneficiaries. Although less commonly used than the base method, this modification has been successfully applied to analyses of health-related patterns in sociology,<sup>20</sup> economics,<sup>21</sup> and health outcomes research.<sup>22</sup>

The Black-to-White ratio of age-adjusted incidence of AD obtained in our study was 1.34, comparable to results



**Figure 2.** Age-specific effects of all other factors in the Oaxaca-Blinder models. The age-specific intercept terms for Black (blue) and White (red) Americans represent the effects of all other factors not included in the model at each age-group. Estimates based on models with disease indicators (small dots) and morbidity profiles (small squares) are shown. Large dots show the logarithm of observed age-specific incidence shown in [Figure 1](#).

previously reported.<sup>2,3</sup> We found that hypertension was the strongest contributor to the disparity in AD risk between White and Black Medicare beneficiaries. This is consistent with the higher levels of prevalence of both AD<sup>23</sup> and hypertension<sup>13,14</sup> in Black Americans, including those estimated in this study.

The primary pathway by which morbidity profiles with hypertension influenced disparities in AD risk between Black and White Americans was vulnerability (i.e., differences in the effect on AD risk); although, exposure (i.e., differences in prevalence) played an important role in specific individual morbidity profiles. This suggests that blood pressure control is an important aspect of reducing White/Black disparities in AD risk. Indeed, recent studies have shown that intensive blood pressure control, in addition to significant cerebrovascular and mortality benefits,<sup>24</sup> was associated with lower risk of mild cognitive impairment<sup>25</sup> and dementia onset.<sup>26</sup> Consequently, differences in antihypertensive pharmacological therapy may be an important, easily modifiable, factor influencing the vulnerability of the Black population to AD risk. Previous studies showed antihypertensive treatment reducing the risk of AD and dementia, but these results remain inconsistent.<sup>27–29</sup> It has been suggested that renin-angiotensin system (RAS) acting medications—namely, angiotensin-converting enzyme (ACE) inhibitors and angiotensin-II receptor blockers (ARB)—are more effective in reducing AD risk than other antihypertensive medications by reducing the risk of development of AD-related neurofibrillary tangle pathology.<sup>29,30</sup> It has been hypothesized that ARBs are potentially more effective in reducing AD risk in White and Black females and White males. The impacts of ACE inhibitors on AD risk are more complex due to ACE-mediated conversion of amyloid- $\beta_{1-42}$  to amyloid- $\beta_{1-40}$ ; the role of different ACE-inhibitors (e.g., enalapril, captopril, perindopril, and lisinopril) in inhibiting the potentially protective mechanisms

of conversion of amyloids- $\beta$  varies depending on the specific medication.<sup>31,32</sup> There are also well-documented race-specific differences in the effectiveness of hypertension medication between White and Black patients: arterial hypertension resistant to antihypertensive medications is more prevalent in Black Americans<sup>33</sup> and ACE inhibitors and ARB monotherapy were less effective at controlling blood pressure in Black patients,<sup>34,35</sup> with higher endogenous sodium and lower renin levels.<sup>32</sup> Together with the fact that Black older adults with hypertension and dementia have been shown less likely to receive ARBs and/or ACE inhibitors and are more likely to have lower levels of adherence to antihypertensive medications than White patients,<sup>36</sup> it is possible that lower use of RAS medications by Black patients could be associated with their higher vulnerability to AD risk found in our study. It should be noted that estrogen has a role in RAS-mediated effects on AD risk and that this effect also varies across race-specific population groups.<sup>37</sup> Therefore, the differences in the race-specific relationships between antihypertensive therapy and AD risk could be indicative of effects that are independent of blood pressure.<sup>38,39</sup>

Occurrence of injury of target organs caused by increased blood pressure and high prevalence of comorbid diabetes and chronic kidney disease contribute to higher morbidity and mortality among Black Americans at any given blood pressure level.<sup>33,40,41</sup> When viewed from this perspective, our results show that the strongest contributing morbidity profile was hypertension+diabetes, which accounted for 145.4% of the total disparity. Although the prevalence of this morbidity profile in Blacks was only 6.6% higher than in Whites, this difference was associated with a strong exposure effect (77.4%) at a comparable level of vulnerability (68.0%). In general, morbidity profiles including hypertension were characterized by strong vulnerability effects, in several cases capable of overturning exposure effects that otherwise would have reduced the size of the disparity. The increased vulnerability of Blacks to the effects of hypertension was especially notable for two morbidity profiles: hypertension alone and hypertension + cerebrovascular disease for which a lower prevalence and associated beneficial exposure effect was overpowered by Black vulnerability. In contrast, morbidity profiles acting to reduce the racial disparities in AD risk were primarily associated with the presence of depression. Unlike morbidity profiles increasing the size of the disparity, depression acted primarily through associated exposure effects (with no strong vulnerability effects observed) consistent with higher prevalence of depression in the White subgroup. Such effects are consistent with race-specific prevalence proportions of depression estimated in other studies.<sup>42</sup>

The modified Blinder-Oaxaca algorithm used in this study also provides age and race-specific intercepts which show the fraction of the difference in AD risk that was not explained by the predictors included in the model. The difference between race-specific curves in [Figure 2](#) shows that the effects of diseases are more pronounced for the Black population (i.e., disease-related effects explain a higher proportion of the final incidence rate in Black Americans). So much so that if the disparity generated by differences in disease-related effects (recall that hypertension accounts for 70% of all disease-related differences; see [Supplementary Table S5](#)

online; Panel A; Row 1) is mitigated then the Black AD incidence shown in Figure 1 would fall below that of White Americans. The age-dependent dynamics of race-specific disparity in the estimated intercept, which reflects the risk of AD for individuals without the considered diseases, changes at ages 75+. At ages below 75 Black and White Americans without hypertension and other diseases considered in this study have similar risks of AD, however, then (at ages 80+) Black Americans become less likely to be diagnosed with AD. This latter effect may be related to the lower rates of progression/survival in Black vs. White Americans.<sup>43</sup>

This study has the following limitations. Identifying the AD population from administrative claims is challenging: AD is difficult to diagnose prior to autopsy, often co-exists with dementias of other pathologies,<sup>44</sup> and can be the result of a misdiagnosis of other pathologies including those cerebrovascular in nature.<sup>44,45</sup> To address this limitation, we conducted a sensitivity study, extending the outcome to include diagnoses of other common dementia types in addition to AD alone (see Supplementary Table S1 online), the results were highly consistent with the findings of the primary analysis (Supplementary Table S5 online; Panel B; and Supplementary Table S6 online). Our data had no information on claims submitted through Medicare Advantage (MA) plans, private managed care alternatives to traditional Medicare. The magnitude of the potential bias associated with this exclusion varies with the size of the MA subgroup at any given point in time. This decreased from  $\approx 18\%$  of the total in 1999, to a low of  $\approx 13\%$  in 2005, increasing thereafter to  $\approx 33\%$  in 2017.<sup>46</sup> We conducted sensitivity studies to assess the impact of inclusion based on the proportion of time spent in a MA plan (and therefore unobserved) and found that the findings reported in this study were stable (Supplementary Table S5 online; Panel A; Rows 4 and 5).

In conclusion, the leading contribution to the racial disparities in AD risk comes from hypertension with about 1/5th of its contribution due to differences in hypertension prevalence (exposure effect) and 4/5ths due to differences in the effects of hypertension on AD risk (vulnerability effect). The contributions of other diseases considered in this study were much weaker, even when combined. Given that hypertension is a manageable and potentially preventable<sup>5,6</sup> condition, mitigating the effects of this disease in engendering higher AD incidence in Black Americans should be a prominent public health concern. Furthermore, interventions focused on Black American communities, especially with high numbers of 75+ individuals, are urgently needed. Improving hypertension management after disease onset would lead to drastic reductions in the Black/White disparity in AD risk since a sizeable proportion of the total disparity is caused by post-onset vulnerability. Emphasis should be placed on the effects of antihypertensive pharmacological therapy as this pathway is most amenable to immediate modification and targets one of the most influential single sources of disparity.

## SUPPLEMENTARY MATERIAL

Supplementary data are available at *American Journal of Hypertension* online.

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## DISCLOSURE

The authors have no conflicts of interest to report. The funding organization had no role in the design or conduct of this research. All individuals who have contributed to this paper in a significant manner are listed as authors. No other significant contributions were made. All authors meet the criteria for authorship stated in the Uniform Requirements for Manuscripts Submitted to Biomedical Journals. Drs Akushevich and Yashkin have contributed equally to the manuscript. The focus of individual specific contributions was: Dr Akushevich (study concept and design, data acquisition, data analysis, data interpretation, initial draft, final approval); Dr Yashkin (study design, data acquisition, data interpretation, subsequent drafts and critical revision, final approval); Dr Kolpakov Nikitin (study design, mathematical input, critical revision, final approval); Dr Kravchenko (study design, data interpretation, critical revision, final approval).

## DATA AVAILABILITY

The data used in this study cannot be shared publicly due to confidentiality reasons. Please contact the Centers for Medicare and Medicaid Services and/or ResDac for information on how to obtain the data.

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