Received: 14 October 2024 | Revised: 6 February 2025 | Accepted: 1 March 2025

DOI: 10.1002/trc2.70078

Alzheimer's & Dementia
Translational Research & Clinical Interventions

RESEARCH ARTICLE

Effects of Medicare predictors in health disparities in the risk of Alzheimer's disease

Igor Akushevich[1] | **Arseniy Yashkin**[1] | **Julia Kravchenko**[2]

[1]Biodemography of Aging Research Unit, Social Science Research Institute, Duke University, Durham, North Carolina, USA

[2]Department of Surgery, Duke University School of Medicine, Durham, North Carolina, USA

Correspondence
Igor Akushevich, Biodemography of Aging Research Unit, Social Science Research Institute, Duke University, 2024 W. Main Street, Durham, NC 27708, USA.
Email: igor.akushevich@duke.edu

Funding information
National Institute on Aging, Grant/Award Numbers: R01-AG057801, R01-AG066133; Department of Defense, Grant/Award Numbers: W81XWH-20-1-0253, HT9425-24-1-0255

Abstract

INTRODUCTION: Disparities in Alzheimer's disease (AD) and related dementias (ADRD) persist across race/ethnicity, sex, and US geographic regions, but limited quantitative information exists to explain how specific predictors contribute to these disparities. Many traditional methods lack precision in addressing both exposure (higher prevalence of a predictor) and vulnerability (higher risk associated with a predictor) effects. This study introduces an approach that leverages population attributable fraction (PAF) to analyze and explain AD/ADRD disparities using Medicare data.

METHODS: We applied our method to Medicare claims data from a nationally representative sample of the US adults aged 70, 75, 80, and 85. The analysis focused on six types of disparities: Black–White, Hispanic–White, Native American–White, Asian–White, female–male, and stroke-belt versus non–stroke-belt states. Predictors included Medicare/Medicaid dual eligibility as an indicator of low income and 10 AD/ADRD-related diseases. The method quantified the exposure and vulnerability effects of each predictor on the observed disparities.

RESULTS: Low income and vulnerability to arterial hypertension were the primary contributors to AD/ADRD disparities, with cerebrovascular diseases and depression as notable secondary predictors. The exposure effect dominated for income-related disparities, while hypertension's effect was largely driven by increased vulnerability. Racial disparities (Black–White, Hispanic–White) were most affected by income and hypertension, while female–male and stroke-belt disparities were less influenced by the examined predictors.

DISCUSSION: Our findings indicate that different intervention strategies are needed to address AD/ADRD disparities. Income-related disparities require targeting exposure (e.g., socioeconomic improvements), while hypertension-related disparities suggest a focus on managing vulnerability (e.g., better control of hypertension). The developed approach offers a robust framework for explaining disparities and designing targeted interventions. Further application to other datasets and exploration of

additional predictors could enhance understanding and lead to more effective prevention strategies for AD/ADRD disparities.

KEYWORDS
Alzheimer's disease risk, decomposition methods, exposure and vulnerability effects, health disparities, hypertension, medicare, population attributable fraction, risk-related diseases

Highlights

- Our new approach addresses disparities leveraging the concept of population attributable fraction for Cox models.
- Exposure and vulnerability mechanisms of health disparity generation are evaluated.
- Vulnerability to hypertension is a consistent dominant factor in Alzheimer's disease (AD) risk disparities.
- Predictors explain AD disparities better in Black and Hispanic populations.
- Disparities in AD are driven by exposure to socioeconomic status suggesting targeted interventions.

1 | INTRODUCTION

Advances in public health and medical technologies continue contributing to progressively longer lifespans in the United States;[1] however, persistent health disparities represent major barriers to equitable distributions of these gains.[2–4] The most prominent types of health disparities in Alzheimer's disease (AD) and related dementias (ADRD) are associated with sex, race/ethnicity, and geographic location.[5–10] Effectively targeting these health disparities remains a clear and current challenge,[1,11] in part due to difficulties in identifying actionable causal pathways through which health disparities are generated.

Increasing availability of real-world "big-health" datasets allow for the assessment of health disparities and evaluation of the role of associated risk/protective factors with increasing accuracy with respect to statistical power; data and method-related biases however, remain a significant challenge. One major methodological barrier is that there are no simple and standard empirical or regression-based approaches that allow for the identification and quantification of such pathways without the use of restrictive technical assumptions which introduce notable methodological and evaluation-related biases.

Explaining health disparities in terms of predictors involves decomposing disparities (e.g., rate ratios or rate differences) into contributions associated with specific predictors. Conventional regression models cannot achieve this because they lack the ability to represent differences in relative risks or risk ratios between subgroups in terms of individual-level risk factors. Many traditional approaches are based on a sequence of Cox models. A predictor is said to "explain" a portion of a disparity if the effect associated with the disparity-affected group (e.g., the Black/White indicator) is significantly reduced when the predictor is added to the Cox model. The main limitation of this approach is that because separate Cox models are estimated with different sets of predictors, unobserved differences in baseline hazards lead to hazard ratios that do not reflect true differences in rates. Furthermore, statistical and systematic uncertainties make it ambiguous to isolate the effect of the hypothesized disparity-causing predictor and results are dependent on the order in which predictors are added to the model. Finally, traditional methods capture only differences in model parameter estimates (vulnerability effect) but disparities also can be generated due to differences in risk factor prevalence (exposure effect) which cannot be captured by such methods but should be accounted for in intervention planning.

In this paper, we present a Cox model–based approach capable of explaining health disparities in terms of predictors that will be free of the above limitations. This approach extends the notion of population attributable fraction (PAF) for health disparities and allows the evaluation of both the exposure and vulnerability effects of each predictor. We applied this approach to the US Medicare claims data to evaluate the magnitude of sex, race/ethnicity, and geographic disparities in AD and explain these in terms of predictors available in Medicare.

2 | METHODS

Administrative claim records drawn from a nationally representative 5% sample of the US Medicare population spanning the 1991 to 2020 period were used. These data allow for reconstruction of individual trajectories with identified initial/final date (and age) of follow-up, dates of death and disease onset, and basic demographic characteristics. Four cohorts with baseline ages 70, 75, 80, and 85 were formed using the 2000 to 2020 period with data from 1991 to 2000 used for look-back. Follow-up time was 5 years, and the outcome of interest was AD onset with death treated as independent censoring. Individuals with claims for AD/ADRD at baseline were excluded as well as those who spent >20% of their study time enrolled in Medicare Advantage.

Six types of disparities, with varying underlying mechanisms engendering the disparity in AD risk,[5] were studied: (1) Black–White, (2) White–Native American, (3) Hispanic–White, (4) White–Asian, (5) female–male, and (6) stroke-belt states[12] versus states without common border with the stroke-belt states. Disparities were defined such that the first subpopulation in the disparity pair was the population at a higher risk (therefore, numerically the observed disparity was always a positive value).

The statistical modeling approach is based on the multivariable Cox proportional hazard model and involves the notion of PAF needed to be calculated for population subgroups. The base model is

$$h(t, r, x) = h_0(t) \exp\left(\beta_r r + \sum_i \beta_{0i} x_i + \sum_i \beta_i r x_i\right), \quad (1)$$

where t represents the time-scale variable and denotes the time after the cohort baseline age, r is the indicator of the disparity (specifically, the indicator equals 1 for the subpopulation placing first in the pair as defined in the definition of disparities above), the index i enumerates predictors at baseline, and x is the vector of these predictors.

After the model (1) is estimated, we can construct the hazard functions for both populations by setting $r = 0$ for the advantaged (control) subpopulation and $r = 1$ for the disadvantaged subpopulation; that is,

$$\begin{aligned} h(t, 1, x) &= h_0(t) \exp\left(\beta_r + \sum_i \beta_{1i} x_i\right) = h_0(t) R_r \prod_i R_{1i}^{x_i} \\ h(t, 0, x) &= h_0(t) \exp\left(\sum_i \beta_{0i} x_i\right) = h_0(t) \prod_i R_{0i}^{x_i} \end{aligned}, \quad (2)$$

where $\beta_{1i} = \beta_{0i} + \beta_i$, R's denote relative risks of respective model parameters: $R_r = \exp(\beta_r)$, $R_{0i} = \exp(\beta_{0i})$, and $R_{1i} = \exp(\beta_{1i})$. Importantly, the baseline hazard $h_0(t)$ is the same for both hazard functions. This allows us to construct the model of the disparity with all factors included using the ratio of the two hazards in Equation (2).

The ratio is independent of the baseline hazard without the need for additional specific assumptions. However, before we can calculate the ratio, we need to average each hazard over the subpopulation and present the average hazard functions in terms of PAFs. The PAF for a specific predictor for baseline and disadvantaged subpopulations is expressed in terms of their disease prevalence (P_{0i} and P_{1i}) and relative risks (R_{0i} and R_{1i}):

$$\begin{aligned} \mathrm{PAF}_{0i} &= \frac{P_{0i}(R_{0i}-1)}{P_{0i}(R_{0i}-1)+1} \\ \mathrm{PAF}_{1i} &= \frac{P_{1i}(R_{1i}-1)}{P_{1i}(R_{1i}-1)+1} \end{aligned}, \quad (3)$$

Averaging of Equation (2) results in

$$\begin{aligned} \bar{h}(t, 1, x) &= \frac{R_r h_0(t)}{\prod_i (1-PAF_{1i})} \\ \bar{h}(t, 0, x) &= \frac{h_0(t)}{\prod_i (1-PAF_{0i})} \end{aligned}. \quad (4)$$

For example, for two predictors a and b the first formula is obtained by averaging (2) as follows. There are four groups with respect to presence of the conditions a and b (11, 10, 01, and 00). Prevalence of these four groups are in the approximation of negligible correlations between a and b: P_aP_b, $P_a(1-P_b)$, $(1-P_a)P_b$, and $(1-P_a)(1-P_b)$, respectively. The averaging procedure results in:

$$\begin{aligned} \bar{h}(t, 1, x) &= h_0(t) R_r \left(R_{1a}R_{1b}P_aP_b + R_{1a}P_a(1-P_b)\right. \\ &\quad \left. + R_{1b}(1-P_a)P_b + (1-P_a)(1-P_b)\right) \\ &= h_0(t) R_r \left(R_{1a}P_a + 1 - P_a\right)\left(R_{1b}P_b + 1 - P_b\right) \\ &= \frac{R_r h_0(t)}{(1-PAF_{1a})(1-PAF_{1b})}. \end{aligned} \quad (5)$$

Now we can model the observed risk ratio in terms of predictors as a ratio of subpopulation-specific hazard functions averaged over the individuals of a cohort at baseline:

$$\begin{aligned} RR &= R_r \prod_i f_i, \quad f_i = \frac{1-PAF_{0i}}{1-PAF_{1i}} = \frac{1+P_{1i}(R_{1i}-1)}{1+P_{0i}(R_{0i}-1)} = E_i V_i \\ E_i &= \left(\frac{1+P_{1i}(R_{0i}-1)}{1+P_{0i}(R_{0i}-1)} \cdot \frac{1+P_{1i}(R_{1i}-1)}{1+P_{0i}(R_{1i}-1)}\right)^{1/2} \\ V_i &= \left(\frac{1+P_{1i}(R_{1i}-1)}{1+P_{1i}(R_{0i}-1)} \cdot \frac{1+P_{0i}(R_{1i}-1)}{1+P_{0i}(R_{0i}-1)}\right)^{1/2} \end{aligned} \quad (6)$$

That is, the risk ratio is represented as a product of factors, each associated with a certain predictor. Furthermore, each factor is decomposed into two contributions: exposure effect (E_i) and vulnerability effect (V_i). This decomposition is exact and does not require any

RESEARCH IN CONTEXT

1. **Systematic Review**: Our study addresses significant gaps in the literature concerning health disparities in Alzheimer's disease (AD) using a novel approach based on population attributable fraction. This method quantifies disparities across race/ethnicity, sex, and geography in US Medicare-eligible older adults. Existing methods lack the ability to comprehensively account for both exposure and vulnerability effects of health predictors, particularly in AD and related dementias.
2. **Interpretation**: Our findings demonstrate that low socioeconomic status and vulnerability to arterial hypertension are key contributors to AD disparities. Other diseases such as cerebrovascular conditions and depression also play substantial roles. This research advances the understanding of how specific predictors generate health disparities in AD.
3. **Future Directions**: Future research should focus on refining our understanding of the role of hypertension in AD progression across racial and ethnic groups. Further studies are also needed to assess how treatment interventions for socioeconomic and health disparities can mitigate these observed vulnerabilities.

TABLE 1 Study sample characteristics: the numbers of individuals at baseline and respective numbers of AD cases detected during the 5-year follow-up in the age cohorts.

Number	Age	White	Black	Native	Hispanic	Asian	Male	Female	SB	Non–SB
Individuals	70	1,457,165	140,435	8719	29,937	35,549	810,622	936,752	269,191	1,082,644
AD cases	70	18,027	2700	85	467	313	9026	13,318	4688	12,060
Individuals	75	1,111,863	95,541	6009	25,756	27,278	570,256	743,265	199,299	824,246
AD cases	75	33,812	4044	152	954	565	15,328	25,572	8040	22,971
Individuals	80	871,612	67,407	4117	21,034	20,976	399,620	620,438	148,478	652,854
AD cases	80	51,695	4971	209	1507	876	20,534	40,617	11,242	36,069
Individuals	85	601,069	42,155	2398	13,942	13,444	238,668	455,799	94,724	456,257
AD cases	85	55,603	4399	166	1455	873	18,641	45,578	10,784	39,571

Abbreviations: AD, Alzheimer's disease; SB, stroke belt.

methodological assumptions in addition to those inherent to Cox modeling.

Statistical analysis is identical for all cohorts and types of disparities. First, the hazard ratios of the indicator of compared subpopulations were evaluated in the unidimensional analysis. This estimate provided an observed disparity in the form of a risk ratio. Then we applied the new statistical model for explaining disparities in terms of cofactors. The cofactors were low income, represented by Medicare/Medicaid dual eligibility,[13–16] and 10 risk-related diseases associated with the likelihood of AD/ADRD[17] (Table S1 in supporting information). During the first stage of analysis, four conditions—rheumatic heart disease, systemic hypotension, chronic liver disease, and traumatic brain injury—were found to have no significant contribution to the disparities across all cohorts. These conditions were therefore excluded from subsequent analyses resulting in the final list of disease-related predictors: cerebrovascular disease, arterial hypertension, diabetes mellitus, renal disease, heart failure, and depression. Individual disease presence was measured at baseline for each cohort using previously published Medicare ascertainment algorithms,[18] with all predictors treated as binary and time independent.

The standard errors and confidence intervals for all evaluated measures were calculated using the bootstrapping technique with resampling with replacement to create 100 random samples from our original sample. Sensitivity analysis included three alternative strategies for outcome ascertainment: (1) confirmation period of 180 days for AD onset, (2) no confirmation period for AD onset, and (3) using ADRD instead of AD as the outcome.

3 | RESULTS

The number of individuals at baseline and the number of AD cases during 5-year follow-up are presented in Table 1. The number of individuals at baseline drops for all cohorts according to the population survival curves for these subpopulations (e.g., reported by the Centers for Disease Control and Prevention WONDER online system[19] or Human Mortality Database[20]). The number of cases increases with time and then drops in response to the decrease in the baseline population. The maximum number of cases was detected for age 80 cohort except for White, female, and non–stroke-belt state subgroups.

The prevalence of dual eligibility for Black, Hispanic, and Asian subgroups was higher compared to their White counterparts (Table 2). The prevalence of cerebrovascular diseases (stroke) was lower for the Asian subpopulation. Black Americans had higher prevalence of hypertension, renal disease, and heart failure in all age cohorts. The higher prevalence of diabetes mellitus was detected in Black, Native, or Hispanic subpopulations depending on age. Females had the highest prevalence of depression followed by White, Native, and Hispanic subpopulations, with Asians having the lowest prevalence in all age cohorts.

The hazard ratios of the study predictors obtained in the multivariable Cox model are shown in Table 3. The highest effects were found for depression, low income, and stroke. The observed disparities and the remaining disparities (i.e., unexplained part of the disparities after accounting for the effects of all predictors) represented by the risk ratios obtained using the univariable and multivariable proportional hazard models are shown in Table 4. The contributions of the risk factors used in the analysis explain approximately half of the observed Black–White disparities. The Hispanic–White disparity changed directions if differences in these risk factors in the Hispanic population were mitigated. The White–Native American and White–Asian disparities became more pronounced after accounting the effects of the risk factors used in our study. The explained part of the effects in female–male and stroke-belt disparities is smaller compared to racial disparities with the sign of the explained effect dependent on the baseline age. Figure 1 demonstrates the contribution of low income represented by dual Medicare/Medicaid eligibility to disparities in AD risk. The effect is strongest for Black–White, White–Asian, and stroke-belt disparities. For White–Native American and Hispanic–White disparities the effect is noticeable for the cohort of age 70 only. Dual eligibility also contributes to increasing female–male disparities.

Hypertension was the strongest factor explaining Black–White disparities, and depression had the opposite effect. These findings are in good agreement with the disease-specific factors for explaining Black–White disparities obtained using another methodologic approach.[21] Hypertension is the most important contribution for other disparity

TABLE 2 Subpopulation-specific prevalence of predictors at baseline.

Predictor	Age	White	Black	Native	Hispanic	Asian	Male	Female	SB	Non–SB
Low income/dual eligibility	70	7.9	24.8	29.0	50.9	44.1	9.0	13.1	12.9	11.2
	75	8.4	29.0	32.7	58.2	60.0	9.8	14.8	14.8	12.4
	80	8.8	32.8	35.2	61.2	66.8	9.9	15.6	16.6	12.9
	85	9.5	36.1	37.6	62.0	68.7	9.7	16.1	18.9	13.1
Stroke	70	7.9	10.8	8.4	7.6	5.9	8.4	7.8	9.7	7.5
	75	14.6	17.0	13.8	12.2	11.3	15.5	13.9	16.7	13.9
	80	21.0	21.8	18.6	17.5	16.8	22.6	19.7	23.1	20.1
	85	26.4	25.6	24.1	23.5	21.3	28.5	24.9	28.2	25.5
Hypertension	70	53.0	65.0	55.3	47.4	46.9	51.6	55.2	60.9	51.7
	75	67.7	76.8	67.2	59.7	62.2	65.9	69.5	74.0	66.8
	80	75.6	81.6	72.8	68.3	70.7	73.2	77.0	80.1	75.2
	85	80.4	84.1	76.5	73.7	76.3	77.4	81.8	84.0	80.3
Diabetes mellitus	70	20.3	33.4	34.4	29.6	25.0	22.7	20.9	24.9	20.9
	75	25.2	39.9	38.0	35.6	32.4	28.6	25.6	29.3	26.3
	80	27.3	41.8	39.2	39.8	36.2	31.0	27.5	30.7	28.6
	85	27.3	40.9	36.3	41.9	37.7	31.0	27.6	30.1	28.7
Renal disease	70	6.0	12.4	9.3	6.9	5.6	7.3	5.9	7.6	6.2
	75	9.3	16.3	12.4	8.7	8.7	11.4	8.6	10.9	9.4
	80	12.3	19.5	15.5	10.9	12.7	15.4	11.1	14.2	12.4
	85	15.7	22.3	18.4	15.7	17.4	20.0	14.2	17.4	15.9
Heart failure	70	6.2	10.9	8.9	7.1	3.9	7.1	6.0	7.8	6.1
	75	11.2	16.5	13.5	11.8	7.8	12.5	10.7	13.0	11.1
	80	16.9	21.3	19.4	17.2	12.5	18.6	16.0	18.6	16.7
	85	23.5	26.9	25.2	23.1	17.3	25.2	22.6	24.8	23.3
Depression	70	10.7	8.1	11.8	10.4	4.7	6.8	13.3	10.5	10.2
	75	13.7	9.8	14.5	12.6	6.3	8.9	16.5	13.3	13.1
	80	15.9	11.2	16.4	14.9	8.0	10.5	18.3	15.4	15.2
	85	18.1	12.5	19.7	18.0	9.3	12.4	20.1	17.0	17.5

Abbreviation: SB, stroke belt.

types. Each factor has two contributions also shown in Table 4: exposure E_j and vulnerability V_j. They measure two separate mechanisms of how a specific risk factor can generate health disparities: due to higher prevalence of risk factor (exposure) or to higher risk of AD among individuals with the risk factor (vulnerability). The effect of low income is dominated by the exposure effect: prevalence of dual eligibility is higher for non-White subpopulations.

The effects of diseases are dominated by vulnerability effects. This is especially true for hypertension and heart failure. For cerebrovascular disease, diabetes mellitus, and renal disease the effect of exposure is not minor though still lower than the vulnerability effect. For depression, the exposure effect can exceed the effect of vulnerability such as in White–Asian and female–male disparities.

Bootstrapping shows that the majority of the exposure and vulnerability effects (57.4%) are significant. The fractions of significant exposure effects are 86% for Black–White, 39% for White–Native American, 86% for Hispanic–White, 75% for White–Asian, 96% for female–male, and 96% and stroke-belt disparities. The fractions of significant vulnerability effects are 61% for Black–White, 7% for White–Native American, 25% for Hispanic–White, 32% for White–Asian, 57% for female–male, and 29% for stroke-belt disparities. The top 40 of the highest effects are shown in Table S2 in supporting information.

Sensitivity analysis demonstrated the stability of the results. Table S3 in supporting information presents the estimates obtained using the base approach and three alternative methods: two differing strategies for AD onset ascertainment and the use of ADRD as the outcome under the base ascertainment scheme.[18] The differences between estimates for 90- and 180-day confirmation periods were negligible. The differences in effects for the ascertainment scheme without confirmation are minor with a tendency to be a little lower compared to the base scheme. The differences between the results for AD and ADRD are also minor. The effects in ADRD are higher for the exposure component and lower for the vulnerability component.

TABLE 3 AD hazard ratios for subpopulations obtained using the multivariable Cox model.

	Hazard ratios for comparing groups						
Predictor	**Age**	**BW**[a]	**WN**	**HW**	**WA**	**FM**	**SB**
Low income/dual eligibility	70	1.83,2.08	2.08,2.44	1.81,2.08	2.08,1.99	1.93,2.13	2.11,1.91
	75	1.46,1.71	1.71,1.23	1.14,1.71	1.71,1.49	1.58,1.61	1.68,1.49
	80	1.35,1.47	1.48,1.13	1.04,1.47	1.48,1.05	1.37,1.39	1.50,1.28
	85	1.24,1.40	1.40,1.32	1.02,1.40	1.40,0.79	1.30,1.21	1.40,1.19
Stroke	70	2.00,2.04	2.04,2.08	2.22,2.04	2.04,1.82	1.97,2.16	2.03,2.09
	75	1.77,1.64	1.64,2.19	1.68,1.64	1.64,1.67	1.64,1.72	1.61,1.69
	80	1.47,1.47	1.47,1.79	1.53,1.47	1.47,1.61	1.44,1.56	1.47,1.47
	85	1.36,1.32	1.32,1.53	1.48,1.32	1.32,1.40	1.30,1.44	1.30,1.33
Hypertension	70	1.22,1.10	1.10,1.83	1.41,1.10	1.10,1.07	1.11,1.18	1.10,1.10
	75	1.26,1.05	1.05,1.44	1.48,1.05	1.05,1.43	1.06,1.13	1.01,1.05
	80	1.17,1.01	1.01,1.19	1.37,1.01	1.01,1.35	1.00,1.11	0.97,1.01
	85	1.15,0.99	0.99,1.23	1.36,0.99	0.99,1.40	0.99,1.07	0.90,0.99
Diabetes mellitus	70	1.07,1.18	1.18,0.97	1.22,1.18	1.18,1.47	1.21,1.15	1.19,1.20
	75	1.00,1.12	1.12,0.92	1.05,1.12	1.12,1.17	1.10,1.16	1.11,1.12
	80	1.02,1.07	1.07,1.00	1.15,1.07	1.07,1.23	1.08,1.08	1.02,1.08
	85	0.94,1.02	1.02,1.06	1.10,1.02	1.02,1.08	1.01,1.07	0.99,1.03
Renal disease	70	1.14,1.08	1.08,0.97	1.33,1.08	1.08,2.07	1.15,1.10	1.15,1.14
	75	1.08,1.03	1.03,1.27	1.11,1.03	1.03,1.49	1.09,1.03	1.04,1.06
	80	0.98,0.98	0.98,1.04	1.08,0.98	0.98,0.92	0.99,1.02	0.98,1.00
	85	1.06,0.94	0.94,0.79	0.98,0.94	0.94,1.11	0.97,0.94	0.99,0.95
Heart failure	70	1.02,1.20	1.20,1.43	1.16,1.20	1.20,0.77	1.18,1.19	1.04,1.18
	75	1.13,1.15	1.15,0.69	1.31,1.15	1.15,1.00	1.18,1.16	1.14,1.14
	80	1.03,1.08	1.08,1.28	1.14,1.08	1.08,1.23	1.09,1.10	1.08,1.08
	85	1.02,1.01	1.01,1.06	1.07,1.01	1.01,0.99	1.02,1.02	1.03,1.00
Depression	70	2.23,2.57	2.57,1.80	2.64,2.57	2.57,3.12	2.39,2.72	2.54,2.49
	75	1.89,2.20	2.20,3.02	2.23,2.20	2.20,2.87	2.07,2.34	2.16,2.21
	80	1.85,1.94	1.94,1.69	1.75,1.94	1.94,1.89	1.87,2.02	1.90,1.94
	85	1.46,1.71	1.71,1.32	1.77,1.71	1.71,1.69	1.66,1.77	1.63,1.72

Abbreviation: AD, Alzheimer's disease.

[a]Disparities are denoted: Black–White (BW), White–Native American (WN), Hispanic–White (HW), White–Asian (WA), female–male (FM), and stroke-belt states versus states without common border with the stroke-belt states (SB).

4 | DISCUSSION

In this paper, we present and apply a Cox model–based approach capable of explaining health disparities in terms of the PAF associated with the predictors. This method evaluates two mechanisms of health disparity generation: the effect of exposure, in which a higher prevalence of a predictor exists in one subpopulation, and the effect of vulnerability, in which disparities arise due to a higher risk associated with that predictor in a specific subpopulation. The model represents the observed disparities (measured as a ratio of rates, r_{obs}) as $r_{obs} = r_{unex} \prod_i E_i V_i$, where r_{unex} is the remaining (unexplained) part of the disparity and E_i and V_i are measures representing exposure and vulnerability effects; $r_{obs} > 1$ for all disparities because of our definitions for disparities. If predictors are identified appropriately, then the remaining unexplained effect is lower than measured (i.e., $r_{unex} < r_{unob}$), and if $r_{unob} \approx 1$ then the chosen group of predictors explains the disparity well. This regular situation, $r_{unex} < r_{unob}$, is observed for Black–White and Hispanic–White disparities. This means that chosen predictors explain a larger portion of the AD risk in Black and Hispanic groups than in the White subpopulation. However, the explanatory factors can act to "increase" the size of the disparity. For example, our predictors explain a larger portion of the risk in Asian and Native American subpopulations than in White Americans; however, because the observed risk in Asian and Native Americans was lower than in White Americans, the disparities between these groups increased after applying the set of explanatory predictors. Concerning female–male and stroke-

TABLE 4 Total, exposure, and vulnerability effects for the predictors presented in the form: Total effect of predictor = exposure x vulnerability.

	Age cohort			
	70	75	80	85
Observed Black–White disparity[a]	1.65	1.46	1.30	1.17
Unexplained Black–White disparity[b]	1.43	1.24	1.15	1.05
Low income/dual eligibility[c]	1.11=1.15 * 0.97	1.07=1.11 * 0.96	1.07=1.09 * 0.98	1.05=1.08 * 0.97
Cerebrovascular diseases	1.02=1.03 * 1.00	1.03=1.02 * 1.02	1.00=1.00 * 1.00	1.01=1.00 * 1.01
Arterial hypertension	1.09=1.02 * 1.07	1.16=1.01 * 1.14	1.13=1.00 * 1.13	1.13=1.00 * 1.13
Diabetes	0.99=1.02 * 0.97	0.97=1.01 * 0.96	0.99=1.01 * 0.99	0.97=1.00 * 0.97
Renal disease	1.01=1.01 * 1.01	1.01=1.00 * 1.01	1.00=1.00 * 1.00	1.02=1.00 * 1.02
Heart failure	0.99=1.01 * 0.98	1.00=1.01 * 1.00	0.99=1.00 * 0.99	1.00=1.00 * 1.00
Depression	0.94=0.97 * 0.97	0.93=0.96 * 0.97	0.95=0.96 * 0.99	0.94=0.97 * 0.97
Observed White–Native American disparity	1.26	1.18	1.12	1.25
Unexplained White–Native American disparity	2.10	1.61	1.33	1.56
Low income/dual eligibility	0.77=0.81 * 0.95	0.99=0.90 * 1.09	1.00=0.93 * 1.07	0.93=0.91 * 1.02
Cerebrovascular diseases	0.99=0.99 * 1.00	0.94=1.01 * 0.93	0.96=1.01 * 0.95	0.96=1.01 * 0.95
Arterial hypertension	0.72=0.99 * 0.73	0.80=1.00 * 0.80	0.88=1.00 * 0.88	0.84=1.00 * 0.84
Diabetes	1.05=0.99 * 1.06	1.06=1.00 * 1.07	1.02=1.00 * 1.02	0.99=1.00 * 0.99
Renal disease	1.01=1.00 * 1.01	0.97=1.00 * 0.97	0.99=1.00 * 0.99	1.03=1.00 * 1.03
Heart failure	0.98=0.99 * 0.98	1.06=1.00 * 1.06	0.96=1.00 * 0.97	0.99=1.00 * 0.99
Depression	1.07=0.99 * 1.08	0.90=0.99 * 0.91	1.03=1.00 * 1.04	1.06=0.99 * 1.07
Observed Hispanic–White disparity	1.21	1.21	1.19	1.10
Unexplained Hispanic–White disparity	0.77	0.95	0.96	0.81
Low income/dual eligibility	1.30=1.38 * 0.95	1.02=1.19 * 0.86	0.98=1.12 * 0.87	0.98=1.10 * 0.89
Cerebrovascular diseases	1.01=1.00 * 1.01	0.99=0.99 * 1.00	0.99=0.98 * 1.01	1.03=0.99 * 1.04
Arterial hypertension	1.13=0.99 * 1.15	1.24=0.98 * 1.26	1.24=0.99 * 1.25	1.27=0.99 * 1.28
Diabetes	1.03=1.02 * 1.01	0.99=1.01 * 0.98	1.04=1.01 * 1.03	1.03=1.01 * 1.02
Renal disease	1.02=1.00 * 1.02	1.01=1.00 * 1.01	1.01=1.00 * 1.01	1.01=1.00 * 1.01
Heart failure	1.00=1.00 * 1.00	1.02=1.00 * 1.02	1.01=1.00 * 1.01	1.01=1.00 * 1.01
Depression	1.00=1.00 * 1.01	0.99=0.99 * 1.00	0.97=0.99 * 0.97	1.01=1.00 * 1.01
Observed White–Asian disparity	1.40	1.53	1.46	1.48
Unexplained White–Asian disparity	1.86	2.25	1.79	1.60
Low income/dual eligibility	0.75=0.74 * 1.02	0.82=0.77 * 1.06	1.01=0.88 * 1.15	1.21=0.96 * 1.26
Cerebrovascular diseases	1.03=1.02 * 1.01	1.02=1.02 * 1.00	1.00=1.02 * 0.98	1.00=1.02 * 0.98
Arterial hypertension	1.02=1.00 * 1.02	0.82=1.01 * 0.81	0.81=1.01 * 0.80	0.76=1.01 * 0.76
Diabetes	0.93=0.99 * 0.94	0.98=0.99 * 0.99	0.94=0.99 * 0.95	0.98=0.99 * 0.98
Renal disease	0.95=1.00 * 0.95	0.96=1.00 * 0.96	1.01=1.00 * 1.01	0.97=1.00 * 0.97
Heart failure	1.02=1.00 * 1.02	1.02=1.00 * 1.01	0.99=1.01 * 0.98	1.00=1.00 * 1.00
Depression	1.06=1.10 * 0.96	1.04=1.10 * 0.94	1.07=1.07 * 1.01	1.06=1.06 * 1.00
Observed female–male disparity	1.24	1.21	1.20	1.19
Unexplained female–male disparity	1.20	1.21	1.26	1.27
Low income/dual eligibility	1.02=1.04 * 0.98	1.02=1.03 * 1.00	1.02=1.02 * 1.00	1.03=1.02 * 1.01
Cerebrovascular diseases	0.98=0.99 * 0.99	0.98=0.99 * 0.99	0.97=0.99 * 0.98	0.96=0.99 * 0.97
Arterial hypertension	0.97=1.00 * 0.96	0.96=1.00 * 0.96	0.93=1.00 * 0.93	0.94=1.00 * 0.94
Diabetes	1.01=1.00 * 1.01	0.98=1.00 * 0.99	1.00=1.00 * 1.00	0.98=1.00 * 0.98

(Continues)

TABLE 4 (Continued)

	Age cohort			
	70	75	80	85
Renal disease	1.00=1.00 * 1.00	1.00=1.00 * 1.01	1.00=1.00 * 1.00	1.01=1.00 * 1.00
Heart failure	1.00=1.00 * 1.00	1.00=1.00 * 1.00	1.00=1.00 * 1.00	1.00=1.00 * 1.00
Depression	1.06=1.09 * 0.97	1.05=1.08 * 0.97	1.05=1.07 * 0.98	1.03=1.05 * 0.98
Observed stroke-belt states disparity	1.58	1.48	1.43	1.37
Unexplained stroke-belt states disparity	1.47	1.46	1.42	1.43
Low income/dual eligibility	1.04=1.02 * 1.02	1.04=1.01 * 1.02	1.05=1.01 * 1.03	1.05=1.02 * 1.03
Cerebrovascular diseases	1.02=1.02 * 1.00	1.01=1.02 * 0.99	1.01=1.01 * 1.00	1.00=1.01 * 0.99
Arterial hypertension	1.01=1.01 * 1.00	0.97=1.00 * 0.97	0.97=1.00 * 0.97	0.93=1.00 * 0.93
Diabetes	1.00=1.01 * 1.00	1.00=1.00 * 1.00	0.98=1.00 * 0.98	0.99=1.00 * 0.99
Renal disease	1.00=1.00 * 1.00	1.00=1.00 * 1.00	1.00=1.00 * 1.00	1.01=1.00 * 1.01
Heart failure	0.99=1.00 * 0.99	1.00=1.00 * 1.00	1.00=1.00 * 1.00	1.01=1.00 * 1.01
Depression	1.01=1.00 * 1.00	1.00=1.00 * 0.99	1.00=1.00 * 0.99	0.98=1.00 * 0.99

[a]Observed disparity is represented by the quantity *RR* in Equation (6) and approximated by the hazard ratio estimated in the univariable analysis with the predictor representing the disparity-affected group, for example, the Black/White indicator.
[b]Unexplained part of disparity is represented by R_r in Equation (6); this is defined as $R_r = \exp(\beta_r)$ in Equation (2).
[c]The effects of predictors are determined by factors f_i in Equation (6) that are multiplicatively decomposed by the exposure and vulnerability effects: $f_i = E_j V_j$.

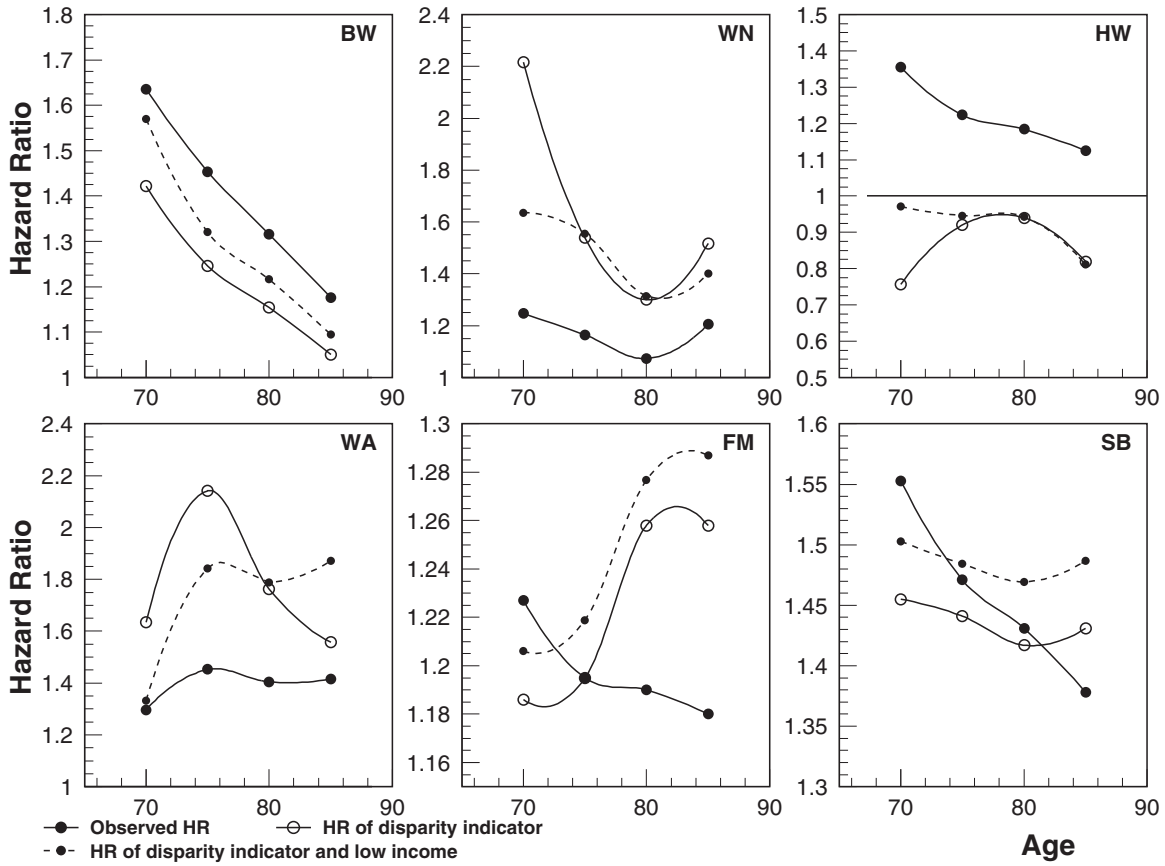

FIGURE 1 Hazard ratio of observed disparities (solid points), the unexplained part of the disparities (open dots), and the effect of low income (dashed line that shows the product of unexplained disparity factors and the factors representing the effect of dual eligibility/low income). BW, Black–White; FM, female–male; HR, hazard ratio; HW, Hispanic–White; SB, stroke belt; WA, White–Asian; WN, White–Native American.

belt disparities, whether the factors reduce or increase the disparities depends on the age at baseline. In both cases the disparities go down after applying the explanatory factors for younger ages and go up for advanced ages.

Our method can be treated as a mediation-based approach fundamentally connected to a general mediation estimation approach[22] and its further developments and implementations such as causal mediation analyses[23–27] and the parametric g-formula adapted for analyses of health disparities.[28] Our method can be assigned to the family of parametric inference algorithms and facilitates causal inferences from time-to-event data by distinguishing the relative importance of differences in mediator prevalence versus mediator effects—the "exposure" and "vulnerability" mechanisms. Another approach capable of providing estimates for the exposure and vulnerability effects is the decomposition technique for hazard-rate models developed by Yun and Powers,[29,30] an adaptation of Oaxaca–Blinder decomposition appropriate for time-to-event data. The method[29,30] is based on two assumptions: evaluation of the mean of the non-linear hazard function as the function of mean risk factors and linearization of the hazard function by using a first-order Taylor expansion; the technique provides estimates of the quality of the approximations induced by these assumptions. The approach used in this study also shares some features of existing methods focused on explaining health disparities in terms of predictors such as rank-and-replace approaches,[31–33] Pollard decomposition models,[34] generalization for registry and administrative data,[35] and the conditional decomposition approach.[36]

Our methodology involves exact mathematical derivations of disparity decomposition using the estimated parameters from a proportional hazards Cox model. Notably, the methodology is straightforward to implement: once the Cox model and predictor prevalence are estimated, exposure and vulnerability effects can be derived using explicit expressions based on the estimated parameters. Thus, a key assumption in our current implementation involves minimizing correlations among predictors, which is a standard consideration in multivariable regression. This assumption is not strictly fundamental and was chosen as an initial simplification; subsequent extensions can introduce corrections in Equations (5) and (6) proportional to correlation coefficients making this assumption less critical. This is sufficient if one merely wishes to characterize statistical associations related to explaining health disparities in terms of predictors. However, causal interpretations of the decomposition necessitate additional assumptions traditional for causal mediation analysis. First, the standard assumptions of no confounding of the mediator(s) and outcome is required. Second, assumptions of sequential ignorability are required for non-parametric identifiability (i.e., consistency in estimates) of the causal mediation effects.[22,37] This means that the treatment (a disparity-generating variable in our case) is first assumed to be ignorable (i.e., statistically independent of potential outcomes and potential mediators) given the pre-treatment covariates, and then the mediator variable is assumed to be ignorable *given* the observed value of the treatment as well as the pretreatment covariates. Third, when examining more than one mediator, assumptions about their causal ordering or interdependence must be made.

Substantively we found that low income was the main predictor for most of the studied disparities. Exceptions were Hispanic–White and White–Native American disparities, for which the effect was notable only for the age 70 cohort (Figure 1 and Table 4). The shape of the effect does not strongly depend on the baseline age: the shapes of the unexplained part of disparities (open dots in Figure 1) and the shape of this with the effect of low income included (dashed line in Figure 1) are similar for all disparities. For low income, the effect of exposure is larger than the effect of vulnerability, indicating that direct interventions aimed at improving the economic well-being of disadvantaged groups would be highly effective.

Among the contributions of AD risk-related diseases arterial hypertension is the strongest predictive factor. The effects of exposure are small for all disparities, but the effect of vulnerability of hypertension dominates to the extent that arterial hypertension becomes the main disease-related predictor in the generation of health disparities related to the AD risk. This is in agreement with analysis of disease-related determinants of Black–White disparities[21] using a Blinder–Oaxaca-based algorithm modified for censored data.[29,30] The identified role of hypertension in increasing the risk of AD in all non-White populations allows us to hypothesize that a more detailed explanation the role of hypertension needs to be obtained, perhaps by involving clinic-related factors such as treatment of hypertension, adherence to treatment, and access to treatment. Two other strong disease-related factors with approximately similar contributions are depression and cerebrovascular diseases. The contributions of exposure and vulnerability for these effects are of similar magnitude, with a slightly higher exposure effect for depression, and a slightly higher vulnerability effect for cerebrovascular diseases. We note that depression is underdiagnosed among the Black subpopulation,[38–40] and this should be kept in mind when interpreting PAFs for depression. Other diseases (diabetes mellitus, renal disease, and heart failure) made situational contributions (Table 4).

In contrast to race/ethnicity disparities, disease-related predictors explained only a small part of female–male and stroke-belt disparities. The patterns of female–male disparities at advanced ages and stroke-belt disparities at age 70 are similar to disparities in the White–Native American and White–Asian subpopulations. For example, the predictors explained a greater portion of the risk of AD in males at advanced ages than in females. Therefore, the unexplained part of female–male disparities becomes larger than the observed female–male disparities in AD risk. Consistent with current knowledge, the exposure effect from cerebrovascular diseases was identified as the dominant exposure factor in explaining the stroke-belt disparities in AD.

Limitations of our analysis are related to the traditional limitation of administrative data use. The outcome is constructed based on an algorithm of disease onset ascertainment.[18] This is addressed using alternative approaches in sensitivity studies. The set of predictors is limited and statistical power for Native Americans should be improved. Only traditional Medicare fee-for-service beneficiaries were analyzed; comparison of empiric estimates with Medicare Advantage beneficiaries are presented in Chen et al.,[41] Haye et al.,[42] and Zissimopoulos et al.[43] Furthermore, the issue of rurality was not addressed, keeping the discussion of geographic disparities at the state-group level.

In sum, in the United States, significant persistent race/ethnicity, sex, and geography-related disparities in the risk and mortality from AD/ADRD persist[6–9,41,42] and in many cases are growing over time.[5,44–47] Recent reviews have emphasized the importance of explaining health disparities in terms of predictors by identifying the causal pathways through which they are generated,[5,10,48] and have suggested the list of potential predictors for different types of disparities.[10,49,50] The approach developed and used in this study provides robust estimates of the contributions of specific predictors to health disparities, as well as their decomposition into exposure and vulnerability effects. Our findings indicate that exposure effects dominate for low income, while vulnerability effects predominate for hypertension and several other diseases. Cerebrovascular diseases and depression were found to have moderate contributions to disparities, with exposure and vulnerability effects of similar magnitude. The higher vulnerability effect observed for certain diseases suggests that disparities may be driven by differences in disease progression, including severity, complications, and treatment, highlighting opportunities to enhance prevention and intervention strategies. Sensitivity analyses confirmed the stability of these estimates.

ACKNOWLEDGMENTS

Publication of this article was supported in part by the National Institute on Aging (grants R01-AG057801 and R01-AG066133) and the Department of Defense (W81XWH-20-1-0253 and HT9425-24-1-0255). The funding organization had no role in the design or conduct of this research.

CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to report. Author disclosures are available in the supporting information.

CONSENT STATEMENT

Data analysis was designed and performed according to the ethical standards of the responsible committee on human studies and the Declaration of Helsinki (1975, revised in 1983) and has been approved by the University Health System Institutional Review Board. Informed consent was not necessary.

REFERENCES

1. Olshansky SJ, Antonucci T, Berkman L, et al. Differences in life expectancy due to race and educational differences are widening, and many may not catch up. *Health Aff*. 2012;31(8):1803-1813.
2. Dickman SL, Himmelstein DU, Woolhandler S. Inequality and the health-care system in the USA. *Lancet*. 2017;389(10077):1431-1441.
3. Gutin I, Hummer RA. Social inequality and the future of US life expectancy. *Annu Rev Sociol*. 2021;47(1):501-520.
4. Dwyer-Lindgren L, Baumann MM, Li Z, et al. Ten Americas: a systematic analysis of life expectancy disparities in the USA. *Lancet*. 2024;404(10469):2299-2313.
5. Akushevich I, Kravchenko J, Yashkin A, et al. Expanding the scope of health disparities research in Alzheimer's disease and related dementias: recommendations from the "Leveraging Existing Data and Analytic Methods for Health Disparities Research Related to Aging and Alzheimer's Disease and Related Dementias" workshop series. *Alzheimer's Dement: Diagn Assess Dis Monit*. 2023;15(1):e12415.
6. Mayeda ER, Glymour MM, Quesenberry CP, Whitmer RA. Inequalities in dementia incidence between six racial and ethnic groups over 14 years. *Alzheimers Dement*. 2016;12(3):216-224.
7. Co M, Couch E, Gao Q, Martinez A, Das-Munshi J, Prina M. Differences in survival and mortality in minority ethnic groups with dementia: a systematic review and meta-analysis. *Int J Geriatr Psychiatry*. 2021;36(11):1640-1663.
8. Co M, Mueller C, Mayston R, Das-Munshi J, Prina M. Ethnicity and survival after a dementia diagnosis: a retrospective cohort study using electronic health record data. *Alzheimers Res Ther*. 2023;15(1):67.
9. Mehta KM, Yeo GW. Systematic review of dementia prevalence and incidence in United States race/ethnic populations. *Alzheimers Dement*. 2017;13(1):72-83. https://doi.org/10.1016/j.jalz.2016.06.2360
10. Maestre G, Hill C, Griffin P, et al. Promoting diverse perspectives: addressing health disparities related to Alzheimer's and all dementias. *Alzheimers Dement*. 2024;20(4):3099-3107.
11. Chetty R, Stepner M, Abraham S, et al. The association between income and life expectancy in the United States, 2001-2014. *JAMA*. 2016;315(16):1750-1766.
12. Howard G, Howard VJ. Twenty years of progress toward understanding the stroke belt. *Stroke*. 2020;51(3):742-750.
13. Joynt KE, Zuckerman R, Epstein AM. Social risk factors and performance under Medicare's value-based purchasing programs. *Circ: Cardiovasc Qual and Outcomes*. 2017;10(5):e003587.
14. Taylor K, Diaz A, Nuliyalu U, Ibrahim A, Nathan H. Association of dual Medicare and Medicaid eligibility with outcomes and spending for cancer surgery in high-quality hospitals. *JAMA Surg*. 2022;157(4):e217586-e217586.
15. Roberts ET, Mellor JM, McInerney M, Sabik LM. State variation in the characteristics of Medicare-Medicaid dual enrollees: implications for risk adjustment. *Health Serv Res*. 2019;54(6):1233-1245.
16. Alberti PM, Baker MC. Dual eligible patients are not the same: how social risk may impact quality measurement's ability to reduce inequities. *Medicine*. 2020;99(38):e22245.
17. Akushevich I, Yashkin A, Ukraintseva S, Yashin AI, Kravchenko J. The construction of a multidomain risk model of Alzheimer's disease and related dementias. *J Alzheimers Dis*. 2023;96(2):535-550. https://doi.org/10.3233/JAD-221292
18. Akushevich I, Kravchenko J, Ukraintseva S, Arbeev K, Yashin AI. Age patterns of incidence of geriatric disease in the US elderly population: Medicare-based analysis. *J Am Geriatr Soc*. 2012;60(2):323-327.
19. Friede A, Reid JA, Ory HW. CDC WONDER: a comprehensive on-line public health information system of the Centers for Disease Control and Prevention. *Am J Public Health*. 1993;83(9):1289-1294.
20. Barbieri M, Wilmoth JR, Shkolnikov VM, et al. Data resource profile: the human mortality database (HMD). *Int J Epidemiol*. 2015;44(5):1549-1556.
21. Akushevich I, Kolpakov S, Yashkin AP, Kravchenko J. Vulnerability to hypertension is a major determinant of racial disparities in Alzheimer's disease risk. *Am J Hypertens*. 2022;35(8):745-751.
22. Imai K, Keele L, Tingley D. A general approach to causal mediation analysis. *Psychol Methods*. 2010;15(4):309.
23. VanderWeele T. *Explanation in Causal Inference: Methods for Mediation and Interaction*. Oxford University Press; 2015.
24. Valeri L, VanderWeele TJ. Mediation analysis allowing for exposure–mediator interactions and causal interpretation: theoretical assumptions and implementation with SAS and SPSS macros. *Psychol Methods*. 2013;18(2):137.
25. VanderWeele TJ, Vansteelandt S. Odds ratios for mediation analysis for a dichotomous outcome. *Am J Epidemiol*. 2010;172(12):1339-1348.
26. VanderWeele TJ. Causal mediation analysis with survival data. *Epidemiology*. 2011;22(4):582.

27. Jackson JW, VanderWeele TJ. Intersectional decomposition analysis with differential exposure, effects, and construct. *Soc Sci Med*. 2019;226:254-259.
28. Sudharsanan N, Bijlsma MJ. Educational note: causal decomposition of population health differences using Monte Carlo integration and the g-formula. *Int J Epidemiol*. 2021;50(6):2098-2107. https://doi.org/10.1093/ije/dyab090
29. Yun M-S. Decomposing differences in the first moment. *Econ Lett*. 2004;82(2):275-280.
30. Powers DA, Yun M-S. 7. Multivariate decomposition for hazard rate models. *Sociol Methodol*. 2009;39(1):233-263.
31. Rao RS, Graubard BI, Breen N, Gastwirth JL. Understanding the factors underlying disparities in cancer screening rates using the Peters-Belson approach: results from the 1998 National Health Interview Survey. *Med Care*. 2004;42(8):789-800.
32. Lê Cook B, McGuire TG, Lock K, Zaslavsky AM. Comparing methods of racial and ethnic disparities measurement across different settings of mental health care. *Health Serv Res*. 2010;45(3):825-847.
33. Lê Cook B, McGuire TG, Zaslavsky AM. Measuring racial/ethnic disparities in health care: methods and practical issues. *Health Serv Res*. 2012;47(3 pt 2):1232-1254.
34. Pollard JH. The expectation of life and its relationship to mortality. *J Inst Actuar*. 1982;109(2):225-240.
35. Akushevich I, Yashkin A, Kovtun M, Stallard E, Yashin A, Kravchenko J. Decomposition of disparities in life expectancy with applications to administrative health claims and registry data. *Theor Popul Biol*. 2023;153:50-68.
36. Gelbach JB. When do covariates matter? And which ones, and how much? *J Labor Econ*. 2016;34(2):509-543.
37. Imai K, Keele L, Yamamoto T. Identification, inference and sensitivity analysis for causal mediation effects. *Statist Sci*. 2010;25(1):51-71.
38. Riley M, Hill-Daniel J. What factors lead to the underdiagnosis of depression among African Americans? *Evidence-Based Practice*. 2020;23(4):41-42.
39. Walton QL, Shepard Payne J. Missing the mark: cultural expressions of depressive symptoms among African-American women and men. *Soc Work Ment Health*. 2016;14(6):637-657.
40. Jones G, Drake C, Lewis CF. Explanations underlying the lack of utility of diagnostic depression scales in Black Americans. *Psychiatr Ann*. 2022;52(12):504-508.
41. Chen Y, Tysinger B, Crimmins E, Zissimopoulos JM. Analysis of dementia in the US population using Medicare claims: insights from linked survey and administrative claims data. *Alzheimer's Dement: Transl Res Clin Interv*. 2019;5:197-207.
42. Haye S, Thunell J, Joyce G, et al. Estimates of diagnosed dementia prevalence and incidence among diverse beneficiaries in traditional Medicare and Medicare Advantage. *Alzheimer's Dement: Diagn Assess Dis Monit*. 2023;15(3):e12472.
43. Zissimopoulos JM, Joyce GF, Jacobson M. Trends in incident dementia diagnosis before and after Medicare risk adjustment. *JAMA Netw Open*. 2023;6(12):e2347708-e2347708.
44. Chen Y, Crimmins E, Ferido P, Zissimopoulos JM. Racial/ethnic disparities in length of life after dementia diagnosis: an 18-year follow-up study of Medicare beneficiaries. *Lancet Reg Health Am*. 2022;8:100179.
45. Mukadam N, Marston L, Lewis G, Mathur R, Rait G, Livingston G. Incidence, age at diagnosis and survival with dementia across ethnic groups in England: a longitudinal study using electronic health records. *Alzheimers Dement*. 2023;19(4):1300-1307.
46. Burke LG, Burke RC, Duggan CE, et al. Trends in observation stays for Medicare beneficiaries with and without Alzheimer's disease and related dementias. *J Am Geriatr Soc*. 2024;72(5):1442-1452.
47. Chen C, Zissimopoulos JM. Racial and ethnic differences in trends in dementia prevalence and risk factors in the United States. *Alzheimer's Dement: Transl Res Clin Interv*. 2018;4(1):510-520. https://doi.org/10.1016/j.trci.2018.08.009
48. Hill CV, Pérez-Stable EJ, Anderson NA, Bernard MA. The National Institute on Aging health disparities research framework. *Ethn Dis*. 2015;25(3):245.
49. O'Neal MA. Women and the risk of Alzheimer's disease. *Front Glob Womens Health*. 2024;4:1324522.
50. Wiese LAK, Gibson A, Guest MA, et al. Global rural health disparities in Alzheimer's disease and related dementias: state of the science. *Alzheimers Dement*. 2023;19(9):4204-4225.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Akushevich I, Yashkin A, Kravchenko J. Effects of Medicare predictors in health disparities in the risk of Alzheimer's disease. *Alzheimer's Dement*. 2025;11:e70078. https://doi.org/10.1002/trc2.70078