

Does a Cancer Diagnosis in Mid-to-Later Life Modify Racial Disparities in Memory Aging?

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Background: It is unknown whether an incident cancer diagnosis differentially impacts acute and long-term memory aging between older White and Black Americans.

Methods: Incident cancer diagnoses and memory (immediate and delayed recall, combined with proxy-reported memory) were assessed at biennial study interviews in the US Health and Retirement Study (N=14,235, 1998-2016). We used multivariable segmented linear mixed-effects models to evaluate the rate of change in standardized memory score (SD/decade) in the years before, acutely at the time of, and in the years following an incident cancer diagnosis, compared to cancer-free adults, by race.

Results: Black participants experienced faster memory decline than White participants (cancer-free group: -1.211 vs. -1.077 ; $P < 0.0001$). An incident cancer diagnosis was associated with an acute memory drop in White, but not Black participants (-0.065 vs. 0.024 ; $P < 0.0001$). However, White cancer survivors experienced slower memory decline than cancer-free White adults before and after diagnosis, but this memory advantage was not observed among Black cancer survivors.

Conclusions: Racial disparities in memory aging are not modified by an incident cancer diagnosis. The acute cancer-related memory decline and long-term memory advantage experienced by White, but not Black, cancer survivors relative to cancer-free older adults, requires further investigation.

Key Words: cancer, cognitive aging, health disparities, memory
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Racial disparities in memory aging and dementia risk in the United States are well-documented and are largely attributable to forms of historic and contemporary racism.^{1–5} These forms of racism operate at multiple levels and across the life course, and include but are not limited to interpersonal discrimination, residential segregation, inequitable access to educational and employment opportunities, and inequitable access to health care.⁴ The impact of an incident cancer diagnosis in mid-to-later life on racial disparities in memory aging older cancer survivors is unknown. This is a major evidence gap, as the population of older cancer survivors in the United States is growing and projected to include >19 million adults aged 65 and above by 2040.⁶ Hence, a rapidly growing population of older cancer survivors is set to experience survivorship health effects alongside cognitive changes and dementia risk as they age.

Paradoxically, older cancer survivors experience lower lifetime dementia risk and slower memory decline during aging than cancer-free individuals.^{7,8} At the same time, older cancer survivors experience acute memory deterioration following diagnosis,^{8–10} which may be due to neurotoxic effects of chemotherapies and other treatments, surgical delirium, or stress associated with a cancer diagnosis.^{11–13} An incident cancer diagnosis in mid-to-later life may impact the magnitude of ongoing racial disparities in memory aging that exist before diagnosis, due to racial disparities in access to quality cancer care, timely diagnosis, and treatments.^{14–18} However, racial disparities in the acute and long-term effects of a cancer diagnosis on memory aging have not been investigated.

We aimed to investigate whether an incident cancer diagnosis in mid-to-later life modifies Black-White racial disparities in memory aging in a population-based cohort of US adults aged above 50 years (Fig. 1). Specifically, we aimed to compare rates of memory aging in the years before, acutely at the time of, and in the years after a first incident cancer diagnosis in mid-to-later life between Black and White Americans. We hypothesized that: (1) incident cancer diagnosis would exacerbate the overall magnitude of racial disparities in memory aging; (2) Black cancer survivors would experience a smaller long-term prediagnosis and postdiagnosis memory advantage compared with cancer-free Black individuals than White cancer survivors compared to cancer-free White individuals; and (3) Black cancer survivors would experience a stronger acute memory decline at the time of diagnosis than White cancer survivors.

METHODS

Study Design and Sample

Data were from adults aged above 50 at baseline in the US Health and Retirement Study (HRS) from 1998 to 2016.¹⁹ The

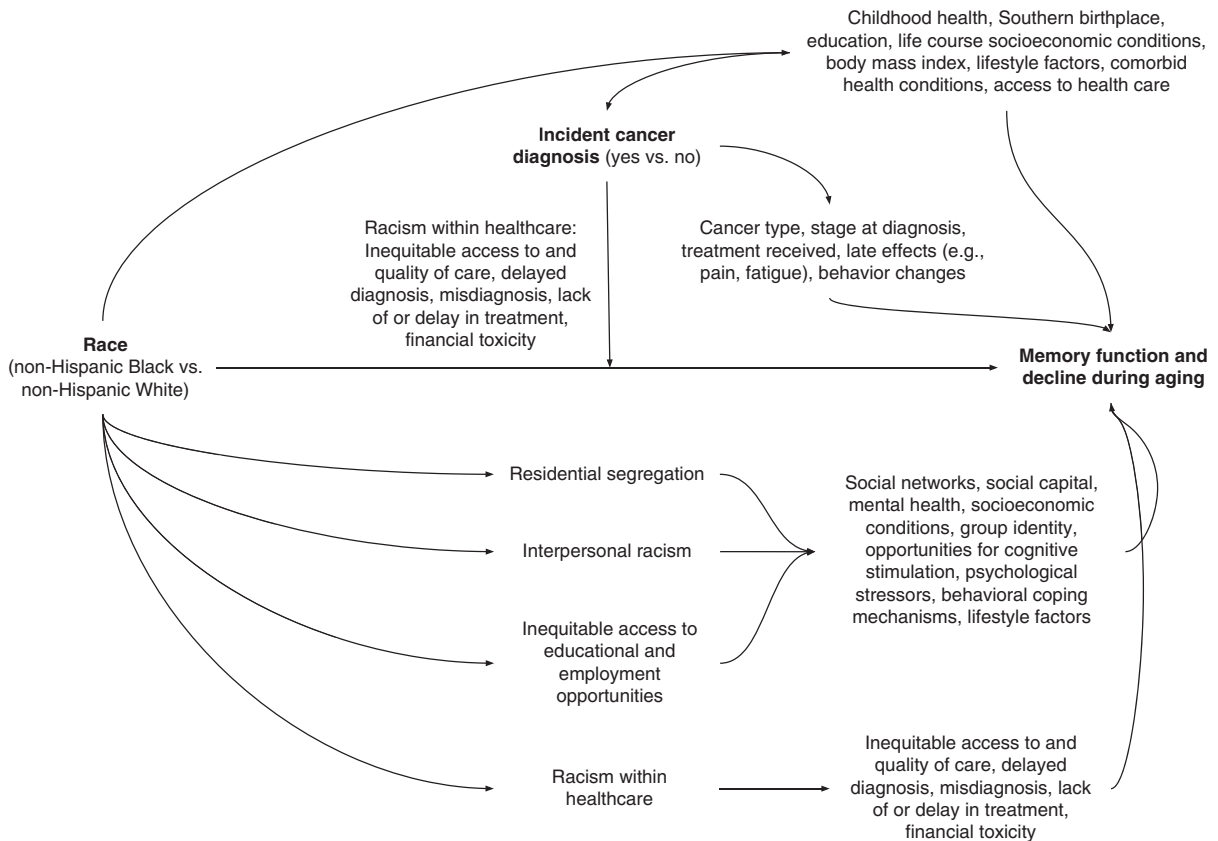


FIGURE 1. Conceptual model.

HRS data are collected through biennial in-person and telephone interviews. Proxy interviews with a spouse, other family member, or friend are conducted for participants who are too impaired to directly participate.¹⁹ The HRS is approved by the Health Sciences and Behavioral Sciences Institutional Review Board at the University of Michigan (HUM00061128) and all respondents provided informed consent upon recruitment into the study. The present analysis was approved by the Health Sciences and Behavioral Sciences Institutional Review Board at the University of Michigan (HUM00170138).

Participants eligible for the present analysis were those born prior to 1949 who self-identified as Black or African American (Black) or White (White) and who participated in the 1998 HRS interview (the baseline for this analysis). Full details on our design are available elsewhere.¹⁰ Eligible participants had no self-reported cancer history before 1998, complete memory outcome and covariate data in 1998, and at least 1 follow-up interview between 1998 and 2016. After excluding 3608 noneligible participants, our final analytic sample included 14,235 individuals who contributed 99,603 observations over the follow-up (see Fig. 2 for the study flow diagram).

Measures

Memory

Memory function is assessed at each biennial study interview as immediate and delayed recall of a 10-word list read out loud by the interviewer.²⁰ For proxy participants, memory is assessed by the proxy informant using the 16-item version of the Jorm Informant Questionnaire for Cognitive Decline (IQCODE) and a 5-point Likert scale of proxy-reported

memory. To reduce bias that could be introduced by excluding the proxy participants, who are the most likely to be cognitively impaired, we combined the direct and proxy assessments of memory using a validated algorithm developed by Wu et al.²¹ At each time point, memory function scores were standardized to the baseline (1998) distribution, such that the units for change in memory over time correspond to 1 SD of the baseline distribution. For cancer survivors, memory scores before diagnosis were assigned negative time in years, and memory scores following diagnosis were assigned positive time in years, to allow evaluation of change in precancer and postcancer memory slopes relative to cancer-free adults.⁴

Race

Self-identified race was collected in the 1998 HRS interview by asking participants: “Do you consider yourself primarily White or Caucasian, Black or African American, American Indian, or Asian, or something else?” We coded our race variable as White (White) or Black or African American (Black). We restrict to these categories of racialization given robust and consistent evidence on disparities in cognitive aging and dementia risk between these groups, given historic systemic racism including, but not limited to, racial residential segregation, disparities in educational access and quality, and employment opportunities.^{22–24}

Incident Cancer

Incident cancer status was assessed at each biennial study interview as a new self-reported physician diagnosis of cancer, excluding non-melanoma skin cancer. The month

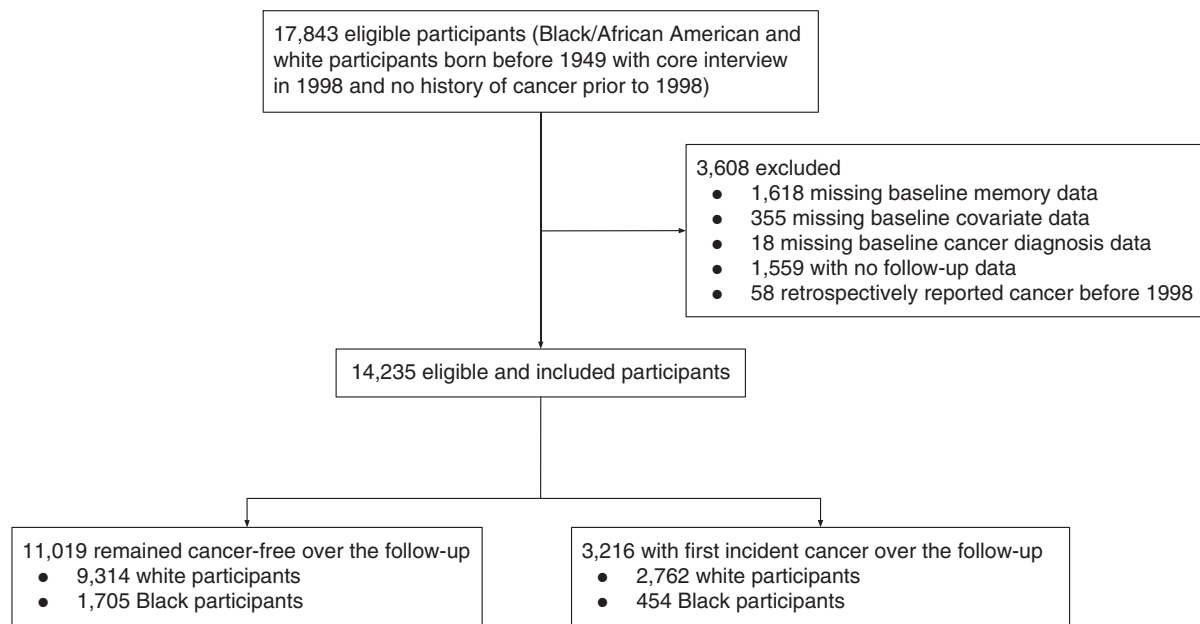


FIGURE 2. Study flow diagram.

and year of cancer diagnosis was self-reported, allowing us to determine the timing of memory assessments relative to cancer diagnosis (ie, number of years precancer and post-cancer diagnosis for each memory assessment). Missing dates of diagnosis were assigned the mid-point between the last cancer-free interview and the interview when the cancer was reported ($n=54$; $<1\%$ of all diagnoses). For participants who died between interviews ($n=6611$), we ascertained cancer diagnoses from postdeath exit interviews with a spouse, family member, or friend.

Covariates

Potential confounders that could be common causes of cancer and rate of memory decline were assessed at the baseline (1998) interview: age (years), sex (male; female), self-rated childhood health (excellent/very good; good; fair/poor), Southern region of birthplace to account for early-life exposure to Jim Crow policies (yes; no), years of education, body mass index (BMI; centered at 25), alcohol consumption according to the National Institute of Alcohol Abuse and Alcoholism definitions²⁵ (none; low risk; binge), ever smoked (yes; no), and self-reported history of physician-diagnosed hypertension, diabetes, and stroke (yes; no) for each. We did not include variables from subsequent interview time points that may be affected by a cancer diagnosis or differential by race and thus lie on a putative causal pathway between cancer and rate of memory decline or race and rate of memory decline. For example, we did not adjust for clinical variables such as cancer treatment or stage at diagnosis in analyses restricted to cancer survivors. Inclusion of these variables would induce overadjustment bias into the estimated relationships, for which we intended to estimate total effects, rather than direct effects not attributable to mediators on the putative causal pathway.⁷

Statistical Analysis

We described baseline characteristics of the sample using univariate statistics. We used multivariable linear mixed-effects

models with restricted maximum likelihood estimation to estimate rates of change in memory function (slopes) for cancer-free participants and cancer survivors, by race. Age was the timescale, describing the average rate of memory decline per decade of age for participants who remained cancer-free over the follow-up. To estimate differences in the rate of memory change associated with a cancer diagnosis, we included slope terms for precancer memory assessments (assigned negative time in years) and postcancer memory assessments (assigned positive time in years) for participants who were diagnosed with cancer, as well as a time-varying indicator set to 0 for all time points before diagnosis, and 1 for all time points following diagnosis. This time-varying indicator allowed us to estimate the magnitude of acute change in memory following diagnosis, for those with an incident cancer. Age was centered at 75 years for cancer-free participants, and age at cancer diagnosis was centered at 75 for participants with cancer, allowing us to align the memory aging slopes for these groups.

We formally assessed racial disparities in (1) memory aging slopes and (2) the impact of a cancer diagnosis on memory aging slopes. First, we ran a model in the full sample that included statistical interaction terms between race and age, and race and each cancer indicator variable. We used the Wald test to evaluate statistical significance of the interaction terms. The full model specification is shown in the Supplemental Material (Supplemental Digital Content 1, <http://links.lww.com/WAD/A378>). We then specified models stratified by race and visualized the predicted memory aging slopes for cancer-free participants (age centered at 75) and participants with cancer (age at diagnosis centered at 75) by race, with corresponding 95% confidence intervals (CIs). All models allowed the memory intercepts and slopes to vary as random effects with an unstructured covariance matrix to allow for estimation of the correlations between the random effects.

We conducted a sensitivity analysis to examine the potential influence of selective attrition following a cancer

TABLE 1. Baseline Characteristics of the Sample, by Race and Cancer Status, in the 1998 Wave of the US Health and Retirement Study

Characteristic	White (n = 12,076)			Black (n = 2,159)	
	Total (n = 14,235)	Incident Cancer (n = 2762)	Cancer-free (n = 9314)	Incident Cancer (n = 454)	Cancer-free (n = 1705)
Age, mean (SD), y	65.9 (10.0)	65.8 (8.8)	66.1 (10.3)	64.9 (9.0)	65.1 (10.1)
Female, n (%)	8258 (58.0)	1344 (48.7)	5508 (59.1)	227 (50.0)	1179 (69.2)
Education, mean (SD), y	12.3 (3.0)	12.7 (2.8)	12.6 (2.8)	10.6 (3.5)	10.8 (3.5)
Childhood self-rated health, n (%)					
Excellent/very good	10,774 (75.7)	2142 (77.6)	7152 (76.8)	324 (71.4)	1156 (67.8)
Good	2602 (18.3)	462 (16.7)	1625 (17.5)	96 (21.2)	419 (24.6)
Fair/poor	859 (6.0)	158 (5.7)	537 (5.8)	34 (7.5)	130 (7.6)
Southern birthplace, n (%)	5233 (36.8)	776 (28.1)	2710 (29.1)	366 (80.6)	1381 (81.0)
BMI, mean (SD)	27.0 (5.1)	26.9 (4.9)	26.6 (5.0)	28.3 (5.4)	28.8 (5.9)
Alcohol use, n (%)					
None	9819 (69.0)	1723 (62.4)	6317 (67.8)	351 (77.3)	1428 (83.8)
Low risk	4148 (29.1)	966 (35.0)	2844 (30.5)	90 (19.8)	248 (14.6)
Binge	268 (1.9)	73 (2.6)	153 (1.6)	13 (2.9)	29 (1.7)
Hypertension, n (%)	5984 (42.0)	1061 (38.4)	3625 (38.9)	277 (61.0)	1021 (59.9)
Diabetes, n (%)	1652 (11.6)	266 (9.6)	929 (10.0)	87 (19.2)	370 (21.7)
Stroke, n (%)	876 (6.2)	163 (5.9)	555 (6.0)	30 (6.6)	128 (7.5)

BMI indicates body mass index (calculated as weight in kilograms divided by height in meters squared).

diagnosis on our results, as cancer survival rates are lower on average for Black versus White Americans.²⁶ We used logistic regression adjusted for baseline age, sex, and race to evaluate whether the likelihood of postdiagnosis study attrition was differential by race. We then added prediagnosis memory to the model to determine whether postdiagnosis attrition was differential by prediagnosis memory and if this relationship varied by race, which would indicate selective survival bias. We ran 2 models to evaluate these relationships, examining 2 forms of memory scores: (1) baseline memory in 1998, and (2) within-person smoothed memory trajectories before cancer diagnosis, as best unbiased linear predictions from fitted models incorporating all prediagnosis memory observations.²⁷ Analyses were conducted using SAS 9.4 (Cary, NC) and StataSE 17.0 (College Station, TX), with data visualizations created in R (Vienna Austria).

Data Availability

The data used for analyses are available from the HRS website (hrs.isr.umich.edu/data-products).

RESULTS

The study sample comprised 14,235 individuals [mean (SD) age, 65.9 (10.0) y; 58.0% female] who contributed 99,603 observations from 1998 through 2016 (Table 1). There were 2159 (15.2%) participants who identified as Black, and 12,076 (84.8%) who identified as White (Table 1). A total of 3216 first incident cancer cases occurred over the 18-year follow-up period, giving a cumulative

incidence of 22.6%. Black participants had a slightly lower mean age at diagnosis than Whites (73.0 vs. 73.7 y, respectively; Table 2). Similar percentages of observations from cancer-free individuals and those with an incident cancer were contributed by each racial group (Table 2).

Results from multivariable segmented linear mixed effects models demonstrated substantial racial disparities in memory aging, regardless of incident cancer status (Table 3; Fig. 3). Among participants who remained cancer-free, mean memory function at age 75 was -0.541 SD (95% CI: -0.573 , -0.509) among Whites, and -1.611 SD (95% CI: -1.686 , -1.536) among Blacks (Wald P -value <0.0001 ; Table 3; Fig. 3). Participants diagnosed with cancer had slightly higher memory function immediately before their diagnosis than those without cancer (difference for Whites: 0.052 SD; 95% CI: 0.022 , 0.082 ; difference for Blacks: 0.040 SD; 95% CI: -0.031 , 0.112 ; Table 3).

Among participants who remained cancer-free over the follow-up, mean memory decline was -1.077 SD/decade of age (95% CI: -1.088 , -1.065) among White participants, and -1.211 SD/decade of age (95% CI: -1.241 , -1.181) among Black participants (Wald P -value <0.0001 ; Table 3; Fig. 3). White cancer survivors experienced an acute memory decline immediately at the time of diagnosis (-0.065 SD; 95% CI: -0.090 , -0.040), while Black cancer survivors experienced a small, nonstatistically significant increase in their memory scores, on average (0.024 SD; 95% CI: -0.041 , 0.088 ; Table 3, Fig. 3). With respect to long-term rates of memory aging, White cancer survivors had a memory advantage compared to cancer-free White participants both in the years before diagnosis (difference in memory slope

TABLE 2. Cancer Statistics Among Non-Hispanic White and Black Participants Diagnosed With Cancer, the US Health and Retirement Study, 1998-2016

Characteristic	Total (n = 3216)	White (n = 2762)	Black (n = 454)
Age at time of cancer diagnosis, mean (SD), y	73.6 (8.8)	73.7 (8.8)	73.0 (9.0)
Total cancer observations, n (%)	22,391 (100.0)	19,325 (86.3)	3066 (13.7)
Precancer observations, n (%)	13,603 (60.8)	11,703 (52.3)	1900 (8.5)
Postcancer observations, n (%)	8783 (39.2)	7618 (34.0)	1165 (5.2)

TABLE 3. Racial Disparities in Memory Aging, and the Impact of a First Incident Cancer Diagnosis in Mid-to-Later Life on Racial Disparities in Memory Aging, US Health and Retirement Study, 1998–2016, n = 14,235

Characteristic	White Participants (n = 12,076)		Black Participants (n = 2159)		P for Racial Disparity in Estimates†
	β	95% Confidence Interval	β	95% Confidence Interval	
Memory function (SD units) and memory change (SD units/decade)					
Participants with no cancer during follow-up					
Memory function at age 75	-0.541	-0.573, -0.509	-1.611	-1.686, -1.536	<0.0001
Memory slope with linear age (centered at 75)	-1.077	-1.088, -1.065	-1.211	-1.241, -1.182	<0.0001
Memory slope with quadratic age (SD units/decade ²)	-0.309	-0.314, -0.304	-0.265	(-0.278, -0.254)	—
Participants with an incident cancer diagnosis					
Difference in memory score right before cancer diagnosis*	0.052	0.022, 0.082	0.040	-0.031, 0.112	0.778
Acute change in memory at the time of diagnosis*	-0.065	-0.090, -0.040	0.024	-0.041, 0.088	0.014
Difference in memory slope before diagnosis*	0.048	0.020, 0.076	0.022	-0.045, 0.090	0.786
Difference in memory slope after diagnosis*	0.087	0.046, 0.128	-0.011	-0.116, 0.094	0.496

Estimates are from models stratified by race, and adjusted for age, age², sex, southern birthplace, self-rated childhood health, years of education, body mass index, alcohol use, and history of hypertension, diabetes, and stroke.

Model specification (person *i* at time *j*): $Memory_{ij} = \beta_0 + \beta_1 \text{cancer_diagnosis} + \beta_2 \text{cancer_now}_j + \beta_3 \text{time_to_diagnosis}$ (zero for time after cancer) $+ \beta_4 \text{time_since_diagnosis}$ (zero for time before cancer) $+ \beta_5 \text{age}_j + \beta_6 \text{age}_j^2 + \beta_7 \text{age_at_cancer_diagnosis} + \sum \beta_k \text{covariates}$. Cancer_now_j is a time-varying binary indicator that equals 0 at time points before diagnosis, and 1 at the time of and after diagnosis.

*Compared with participants with no cancer over the follow-up, as the reference group.

†From Wald test of statistical interaction between race and each characteristic of interest, from model including the full sample.

compared with cancer-free Whites: 0.048 SD/decade of aging; 95% CI: 0.020, 0.076) and in the years following diagnosis (difference in memory compared to cancer-free Whites: 0.087 SD/decade of aging; 95% CI: 0.046, 0.128; Table 3). However, among Black participants, long-term memory aging slopes were similar between those with and without an incident cancer diagnosis (Table 3, Fig. 3).

A total of 586/2762 White participants with cancer (21.2%) and 116/454 Black participants with cancer (25.6%) stopped contributing data following their reported cancer diagnosis [odds ratio (OR)=1.35; 95% CI: 1.06, 1.70, adjusted for baseline age and sex]. Among both Black and White participants with cancer, higher precancer memory scores were associated with lower likelihood of postdiagnosis attrition (baseline memory: OR = 0.85 per SD unit; 95% CI: 0.74, 0.97; prediagnosis memory trajectory: OR = 0.37 per SD unit/decade of age; 95% CI: 0.26, 0.53). The relationships between prediagnosis memory and postdiagnosis attrition did not vary by race (*P* for interaction = 0.123 for baseline memory, and 0.369 for prediagnosis memory trajectory), indicating that selective survival bias conditioned on race and prediagnosis memory does not impact our findings. Mean and median baseline memory scores and prediagnosis memory trajectories by race and attrition status are shown in Supplemental Table 1 (Supplemental Digital Content 1, <http://links.lww.com/WAD/A378>).

DISCUSSION

In this nationally representative cohort of middle-aged and older US adults, we observed substantial Black-White disparities in memory aging that were not meaningfully affected by a first incident cancer diagnosis in mid-to-late life.

Black adults experienced a lower memory intercept and slope than White adults, regardless of cancer status, consistent with evidence that racism across the life course affects memory aging in mid-to-later life.^{1–5,24,28–30} Consistent with prior clinical and population-based evidence, we observed an acute memory decline immediately following cancer diagnosis, but a long-term memory advantage among middle-aged and older White cancer survivors compared with White cancer-free adults. These cancer-associated memory differences were not observed among middle-aged and older Black cancer survivors and cancer-free adults. These racial differences were small and require further investigation and replication in other studies. Potential proximal contributors to these racial differences may be different distributions of cancer types, treatments, and stages at diagnosis between older Black and White cancer survivors, and these should be investigated in future studies. Overall, results of this study indicate that postcancer memory disparities between Black and White cancer survivors largely reflect precancer disparities.

Comparison With Existing Literature

Consistent with previous research, we observed that middle-aged and older Black adults had lower memory function at baseline and faster rates of memory aging compared with Whites.^{5,24,28–31} Racial disparities in memory aging and dementia risk in the United States have been attributed to historical and contemporary forms of racism, including residential segregation, educational access and quality, employment opportunities, and access to health care.^{1–5,22–24,30–32} Black participants in our study completed an average of 10.8 years of education, compared with 12.6 years among White participants, and were

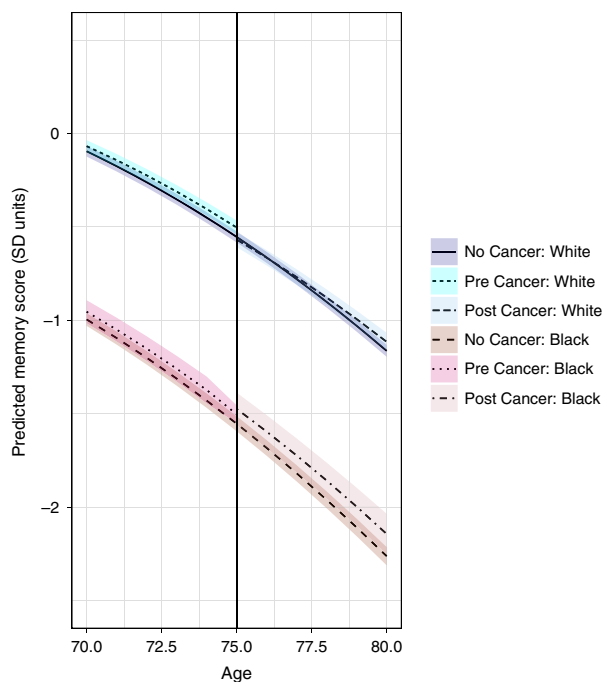


FIGURE 3. Predicted precancer and postcancer memory slopes (95% confidence intervals shaded) for incident cancer cases and predicted memory slopes with aging for those who remained cancer-free over the follow-up, by race. Age at cancer diagnosis is centered at 75 years for incident cancer cases, and age is centered at 75 years for those who remained cancer-free over the follow-up. The predicted values are from fully adjusted models, with all covariates held at their mean for continuous variables or at their reference categories for categorical variables. Memory is standardized to the 1998 baseline distribution (mean of 0 and SD of 1).

disproportionately likely to have been born in the Southern US at a time when Jim Crow policies legislated many forms of structural racism.³³ Southern US place of birth has been associated with increased risk of stroke, dementia, and dementia mortality among both Black and White older adults, although with a disproportionately greater magnitude of risk among Black older adults.^{34,35} To the best of our knowledge, this is the first study to examine acute and long-term racial disparities in memory aging in a population-based cohort utilizing repeated measures of memory pre- and post-cancer diagnosis with a cancer-free comparison group.

Potential Mechanisms

Our finding of an acute memory decline immediately following cancer diagnosis among White, but not Black adults, was unexpected. Acute memory decline following a cancer diagnosis, often referred to as “chemobrain,”^{9,12,36,37} is thought to be primarily due to neurotoxic effects of chemotherapy treatment, as well as some hormonal therapies, the stress of a cancer diagnosis, or potentially the carcinogenesis process itself.^{8–13} Our findings could reflect cancer treatment disparities that negatively affect Black cancer patients, who are more likely to experience diagnostic delays,^{16–18} less likely to receive timely chemotherapy,^{14,15,38} and less likely to adhere to chemotherapy regimens^{16,17} than White cancer patients, all of which could cause differential acute memory function by race shortly following diagnosis. These persistent Black-White racial disparities in receipt and

adherence to cancer care are due to myriad reasons including maltreatment by the medical system,³⁸ poor physician access,¹⁸ delayed referral to specialist care,¹⁷ prohibitive costs/lack of insurance,¹⁷ and competing socioeconomic responsibilities.¹⁶ Future research on racial disparities in aging outcomes among cancer survivors should incorporate direct measures of racism at multiple levels (eg, interpersonal racism, racism in health care, structural racism) to better disentangle these mechanisms. Future studies should also investigate the roles of racial disparities in cancer clinical factors and quality of cancer care as proximal contributors to memory aging disparities among older cancer survivors. Finally, investigation of the role of pre-cancer comorbidities, such as cardiovascular disease, in racial disparities in memory aging among older cancer survivors is warranted.

The reasons for the paradoxical inverse relationship between cancer and long-term memory aging that we observed are a topic of ongoing investigation. Methodological study biases such as selective survival of cognitively healthier individuals with cancer, diagnostic bias, competing risks bias, and confounding bias are possible noncausal explanations.⁷ We found that incident cancer cases who were cognitively healthier in the years before their diagnosis were less likely to die or drop out of the HRS following diagnosis, but this selective survival pattern was not differential by race. The memory advantage experienced by cancer survivors in the years before diagnosis suggests a potential common cause of cancer and dementia, such as inverse biological regulation of carcinogenesis and neurodegeneration.⁷ However, the lack of an inverse cancer-memory relationship among middle-aged and older Black adults in this study is unexplained and deserves further investigation.

Strengths and Limitations

Cancer diagnoses were self-reported and subject to measurement error. Covariates were self-reported in the HRS interviews, and errors in covariate measurement due to these self-reports or interview question limitations could result in residual confounding of the model estimates. We did not adjust for baseline physical activity as a confounder, as this variable was crudely measured in the HRS, but estimates were negligibly changed when we did adjust for the HRS physical activity variable (results not shown). We were underpowered to evaluate the effect of an incident cancer diagnosis on memory aging by sex/gender in addition to race, which is an important area for future exploration as the magnitudes of racial and ethnic disparities in cognitive aging vary between men and women.³⁹ Our results may not generalize to older adults from racial or ethnic groups not represented in this study, and future research should investigate disparities in memory aging after cancer in more diverse study samples. As one of the first studies to demonstrate racial disparities in long-term precancer and postcancer diagnosis memory aging and acute memory changes at cancer diagnosis, our study provides important evidence for future hypothesis generation in studies with more in-depth clinical data from multiracial cancer survivors. A future next step is to link the HRS cohort data with Medicare claims to investigate the roles of cancer type and treatment modality as proximal contributors to racial disparities in memory aging among older cancer survivors.

A strength of this study is its large, nationally representative sample of middle-aged and older US adults over a

long follow-up period with high cohort retention. We utilized rich data on memory function combined with proxy-reported memory using a validated algorithm.²¹ Our inclusion of proxy interviews minimizes bias that may be introduced by the exclusion of the most cognitively impaired individuals from analysis. We had longitudinal data on memory prior to cancer diagnosis, which is often unavailable in clinic-based studies of cancer survivorship that recruit after diagnosis, allowing us to identify the within-person change in memory function associated with a cancer diagnosis. We utilized data from exit interviews with informants and proxy interviews to capture cancer diagnoses among participants who died between interviews or who were too impaired to directly take part in the study interview, which helped minimize misclassification of cancer diagnostic status. Our sensitivity analyses indicated that post-diagnosis selective survival or study attrition do not explain the racial disparities in memory aging observed in this study.

CONCLUSION

Memory aging and dementia risk in the growing population of older US cancer survivors will be important in shaping overall older population health in the decades to come. We found, for the first time, that Black-White racial disparities in memory aging are not modified by a first incident cancer diagnosis in mid-to-late life. However, we observed a long-term inverse association between cancer and rate of memory aging, as well as an acute short-term memory decline associated with a cancer diagnosis among White, but not Black middle-aged and older adults. Future studies should investigate the roles of racial disparities in cancer type, stage at diagnosis, treatment received, and quality of care in influencing racial disparities in memory aging among older cancer survivors. Linkages between longitudinal cohort studies of aging and sources of cancer clinical data are needed to accelerate this type of research on cancer and aging. The present findings may inform future hypotheses for research on the intersection of racial disparities in cognitive aging with racial disparities in cancer care and outcomes.

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