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Biopsychosocial pathways in dementia inequalities: introduction to the Michigan Cognitive Aging Project

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Abstract

Racial/ethnic inequalities in dementia risk are a major public health and health justice concern. Group differences that persist despite adjustment for socioeconomic and vascular indicators suggest that known dementia risk factors exhibit differential impact across race/ethnicity and/or there are unrecognized dementia risk factors that are racially patterned. This paper provides targeted examples of both possibilities. First, depressive symptoms and white matter hyperintensities represent two known dementia risk factors that more strongly relate to negative cognitive outcomes among Black older adults than Whites, pointing to the need to consider contextual factors. Second, racial discrimination and external perceived control predict worse brain and cognitive aging above and beyond known risk factors. These psychosocial factors warrant explicit consideration in dementia cohort studies. Several challenges appear to be particularly relevant to the study of dementia inequalities, including selective survival and recruitment. These challenges complicate not only cross-study comparisons, but also within-study causal inferences. This paper provides recommendations for addressing these challenges in order to accelerate high-quality research on dementia inequalities. Stemming from these recommendations, the paper introduces the design and methods of the Michigan Cognitive Aging Project, a new, racially-balanced cohort study of Black and White adults transitioning to late life. In sum, careful research with community partners is needed to more fully explore the factors and contexts that create and sustain racial/ethnic disparities, as well as those that buffer against them. The ultimate goal of this research is to facilitate the dismantling of structural barriers to health justice for diverse older people.

Keywords

Health disparities; African American/Black; Hispanic/Latinx; Cognition; Aging

Dementia is a growing public health concern. The U.S. is aging rapidly, due to changes in both birth trends and life expectancies (Roberts et al., 2018). The number of U.S. older adults (i.e., aged 65 and older) increased from 3.1 million (4%) in 1900 to 49.2 million (15%) in 2016. Rapid population aging is accompanied by an explosion in the number of dementia cases, as older age is the strongest risk factor for dementia (Guerreiro & Bras, 2015). Dementia is an umbrella term for a clinical syndrome characterized by progressive

cognitive declines severe enough to interfere with daily life. The most common cause of dementia is Alzheimer's disease. Of note, the prevalence (i.e., proportion of the population living with a disease) of dementia declined between 2000 and 2012, due in part to higher educational attainment in the population (Langa et al., 2017). However, the aging of the population means that the raw number of individuals living with dementia will continue to rise. Indeed, the number of older Americans living with dementia due to Alzheimer's disease is projected to increase from 6.2 million in 2021 to 12.7 million in 2050, a doubling in less than 30 years (Alzheimer's Association, 2021). Like many diseases, dementia is not randomly distributed in the population. It disproportionately affects social groups that have been historically marginalized as the result of structural and interpersonal racism, including Hispanic/Latinx (hereafter "Latinx") and non-Latinx Black (hereafter "Black") older adults (Tang et al., 2001; Mayeda et al., 2016; Weuve, et al., 2018). While dementia prevalence has declined at the population level, racial disparities in dementia have remained stable (Power et al., 2021).

An overwhelming body of research indicates that racial/ethnic differences in dementia reflect a health inequality, a particular type of health difference linked to social disadvantage that is avoidable, unnecessary, and unjust (Whitehead, 1992; Braveman, 2006). Grounded in fundamental cause theory (Link & Phelan, 1996), Glymour and Manly (2008) outline how racially patterned social conditions (e.g., social determinants of health; World Health Organization, 2008) can initiate a cascade of individual and proximal mediators of racial/ethnic disparities in cognitive aging. Indeed, a large proportion of racial/ethnic differences in late-life cognitive health are attributable to modifiable social determinants of health, such as educational attainment, educational quality, and income (Manly & Mungas, 2015; Sisco et al., 2015; Carvalho et al., 2015; Haq & Penning, 2019), as well as related physical health factors (Gottesman et al., 2016). However, cognitive inequalities are not fully explained by educational attainment, educational quality, income, or cardiovascular indicators (Sisco, et al., 2015; Tang et al., 2001; Demirovic et al., 2003).

The observation that racial/ethnic inequalities persist despite adjusting for these important known mediators suggests at least two possibilities. First, known dementia risk factors may demonstrate differential impact across racial/ethnic groups. Failure to account for group by risk interactions or to characterize contextual factors that lead to differential impact limits our ability to model and ultimately eliminate disparities. Second, dementia researchers are not routinely measuring racially-patterned risk and protective factors that influence dementia risk. For example, there may be risk factors for dementia that are unique to certain racial/ethnic groups and have not been considered in major cohort studies that comprise mostly non-Latinx White older adults. The first section of this paper provides empirical examples advancing both of these possibilities, demonstrating how an explicit focus on psychosocial factors can improve our understanding of dementia inequalities. The second section highlights two major challenges to this work: selective survival and recruitment. While such challenges are relevant to many types of psychological research, they may be particularly impactful in the study of racial/ethnic differences in cognitive aging. Finally, this paper introduces the Michigan Cognitive Aging Project, a new data collection effort designed to minimize the impacts of these challenges.

Biopsychosocial Explanations for Persistent Inequalities

Differential Impact

Differential impact refers to group differences in the strength of association between a risk factor and an outcome, not group differences in the level of a risk factor. Evidence for differential impact is available across psychological and biological categories of dementia risk.

Depressive Symptoms.—The most prominent psychological risk factor for dementia is depressive symptoms (Livingston et al., 2017). Meta-analyses confirm that depressive symptoms are prospectively linked to greater dementia incidence (Diniz et al., 2013; Mourao et al., 2016). The direction of the association between depressive symptoms and cognitive aging is not clear-cut. However, longitudinal modeling indicates that depressive symptoms prospectively predict faster rates of memory decline over 12 years, and reporting more depressive symptoms than expected at one occasion predicts worse memory performance than expected at the subsequent occasion, but not vice versa (Zahodne et al., 2014).

Racial/ethnic differences in depressive symptoms are inconsistent. While Black and Latinx adults tend to report more depressive symptoms than non-Latinx Whites (hereafter “White”), clinical depression is often lowest in Black samples (Taylor & Chatters, 2020). Regardless of these level differences, depression tends to be more chronic and impairing among Black adults compared with Whites (Williams et al., 2007). Recent work has extended these observations to cognitive aging. Even though Black older adults who participated in the national norming study for the NIH Toolbox reported fewer depressive symptoms than Whites on average, negative associations between depressive symptoms and cognition were stronger among Blacks (Zahodne et al., 2014).

These findings are in line with the related concepts of compound disadvantage (Wheaton & Clarke, 2003), cumulative disadvantage (Dannefer, 2003), and double jeopardy (Jackson, 1985), which predict that the greatest health disadvantages occur for disadvantaged individuals living in a disadvantaged context. For example, Black Americans are more likely to live in neighborhoods characterized by poor quality schools and limited access to healthcare, jobs, and beneficial social networks than Whites (Firebaugh & Acciai, 2016), though there is great within-group heterogeneity in socioeconomic status and neighborhood contexts among Black Americans. Black and/or Latinx adults with psychiatric disorders are also less likely to receive mental health treatments (Alegría et al., 2008), including psychotherapy (González et al., 2010), antidepressants (González et al., 2008), and alternative therapies (Woodward et al., 2009). Among older adults, these racial differences in neighborhood contexts and mental health services may all contribute to the differential impact of depressive symptoms.

White Matter Hyperintensities.—White matter hypertensities (WMH) refer to diffuse areas of high signal intensity on magnetic resonance imaging (Prins & Scheltens, 2015) and reflect cerebrovascular pathology (i.e., leukoaraiosis). In general, WMH increase with age and are associated with a variety of cardiovascular risk factors, including smoking and hypertension (Liao et al., 1997). Importantly, WMH prospectively predict dementia

incidence, above and beyond age and systemic cardiovascular diseases (Brickman et al., 2012; Brickman et al., 2015). With regard to racial/ethnic differences, a community-based study in New York City reported greater WMH volumes among non-demented Black and Latinx older adults compared with Whites (Brickman et al., 2009), while another community-based study in Chicago found no differences in WMH volumes across Black and White older adults (Aggarwal et al., 2010). A large, population-based study in the U.S. South found that Blacks showed lower overall prevalence of WMH but a higher prevalence of more severe WMHs than Whites (Liao et al., 1997). Similarly, a community-based study in London showed that African Caribbeans have a higher prevalence of severe WMHs than Whites (Shibata et al., 2013). Of note, the finding that WMH volume is greater among Blacks than Whites appears to be strongest for individuals with lower socioeconomic status (Waldstein et al., 2017).

Regardless of these level differences, cross-sectional studies indicate that WMH are more strongly related to worse language and speed/executive functioning (Zahodne et al., 2015) and worse visuospatial abilities (Mungas et al., 2009) among Black older adults than Whites. However, longitudinal work suggests that baseline WMH are more strongly predictive of subsequent rates of cognitive decline in Latinx and White older adults than Blacks (Gavett et al., 2018). Together, these studies support differential impact of WMH but indicate that group differences may not be the same for cognitive level versus change. Of note, racial/ethnic inequalities in dementia are more likely to be driven by racial/ethnic differences in cognitive level than differences in rates of late-life cognitive change (Manly & Mungas, 2015). On the whole, these neuroimaging results support differential impact in that WMHs, a key biological predictor of dementia risk, may be most strongly associated with cognitive outcomes among older Black and/or Latinx adults. These studies provide additional evidence for compound disadvantage (Wheaton & Clarke, 2003) and may point to synergy between risk factors that are more prevalent in certain racial/ethnic minority groups. Observations that the brain predictors of cognition and function differ across race/ethnicity also underscore the need for more diverse samples in research on dementia biomarkers (Glymour et al., 2018), as well as more within-group studies of Black and Latinx adults.

Summary.—While this section highlighted depressive symptoms and WMH as illustrative examples of psychological and biological risk factors that exhibit differential impact across race/ethnicity, differential impact has also been described for other health factors relevant to dementia risk, such as type 2 diabetes (Mayeda et al., 2014). Thus, testing for differential impact and considering contributing contexts may be broadly relevant when considering dementia risk and protective factors across racial/ethnic groups.

Novel Dementia Risk Factors

Racial/ethnic inequalities in cognitive aging that are not fully explained by commonly-measured dementia risk factors may also point to the existence of additional, under-recognized risk factors that are racially patterned. Many psychosocial factors are not routinely measured in cohort studies (c.f., depressive symptoms). Even less attention has been paid to psychosocial factors that reflect structural and interpersonal racism, which are increasingly recognized as causes of health disparities. In particular, emerging

literature points to discrimination and external perceived control are two racially-patterned psychosocial constructs with implications for dementia risk.

Discrimination.—Discrimination was first linked to lower cognitive performance in a cross-sectional study of 407 Black participants in the Minority Aging Research Study and Memory and Aging Project (Barnes et al., 2012). In that study, more frequent subjective experiences of being treated unfairly in common, everyday situations (i.e., everyday discrimination) was associated with worse concurrent performance on tests of episodic memory and perceptual speed. More recent longitudinal work in the nationally-representative Health and Retirement Study (HRS) has revealed that more frequent everyday discrimination predicted worse performance on measures of executive functioning, processing speed, and visuconstruction two to four years later, controlling for global cognition at the time of the baseline discrimination assessment (Zahodne et al., 2020). In addition, Black-White differences in everyday discrimination partially mediated group differences in initial episodic memory performance, and more frequent everyday discrimination was associated with faster rates of episodic memory decline over six years (Zahodne, Sol & Kraal, 2019). Together, these disparate studies indicate that racially-patterned discrimination may contribute to racial inequalities in cognitive aging and dementia risk.

Clark and colleagues' (1999) biopsychosocial model of racism states that experiences of discrimination can trigger psychological and physiological stress responses that lead to a variety of negative health outcomes. As a stressor, discrimination may be particularly deleterious to health because it threatens highly salient aspects of self-identity (Thoits, 2013) and is both unpredictable and uncontrollable (Williams & Mohammed, 2009). With regard to psychological responses, qualitative and quantitative work indicates that experiencing discrimination can lead to depressed mood and lower perceived control (Taylor & Chatters, 2020; Broman et al., 2000; Bullock & Houston, 1987). With regard to physiological responses, cross-sectional and longitudinal studies have linked everyday discrimination to multiple negative outcomes, including cardiometabolic (Beatty Moody et al., 2019; Sims et al., 2012; Everson-Rose et al., 2015; Whitaker et al., 2017) and inflammatory (Beatty Moody, Brown, Matthews, & Bromberger, 2014; Zahodne, Kraal, et al., 2019; Friedman et al., 2009; Lewis et al., 2010) factors.

Empirical support for Clark and colleagues' (1999) biopsychosocial model in the area of cognitive aging is growing. In the HRS, negative associations between everyday discrimination and episodic memory performance are mediated by greater depressive symptoms and external control (Zahodne, Sol & Kraal, 2019), as well as by elevated C-reactive protein, a blood biomarker of systemic inflammation (Zahodne, Kraal, et al., 2019). In addition to evidence that discrimination gets “under the skin,” there is also evidence that discrimination gets “into the skull.” Among Black older adults in the Washington Heights-Inwood Columbia Aging Project (WHICAP), greater lifetime discrimination (i.e., sum of self-reported major life events involving unfair treatment) was associated with lower hippocampal volumes, while everyday discrimination prospectively predicted faster accumulation of WMH over four years (Zahodne et al., in prep). In all of these studies, Black participants reported more everyday discrimination than Whites. Together, they provide

evidence that discrimination may have a negative impact on late-life cognitive functioning via its psychological and physiological effects.

External Perceived Control.—Originating in social learning theory (Rotter, 1966), perceived control (i.e., locus of control) refers to the extent to which individuals feel they have control over important life outcomes (Lefcourt, 2014). Early work described internal and external control as two ends of a single continuum; however, more recent work conceptualizes internal and external control as separate dimensions that can covary (Lefcourt, 2014). Internal control is akin to mastery or self-efficacy, while external control is characterized by beliefs about environmental or interpersonal constraints that limit instrumentality (Lachman et al., 2011). Importantly, racial differences in perceived control are more evident in external control than internal control. Black and Latinx adults report more external control but not less internal control than Whites (Ross & Mirowsky, 2013). One explanation for these divergent patterns is that views of the self (i.e., internal control) are shaped more by an individual's micro-environment (e.g., family, peers), while views of environmental constraints (i.e., external control) are shaped more by an individual's macro-environment (e.g., larger social structures) (Hughes & Demo, 1989). Indeed, both socioeconomic disadvantage (Ross & Mirowsky, 2013) and interpersonal discrimination (Broman et al., 2000; Bullock & Houston, 1987) have been linked to more external control.

Less internal control and/or more external control are prospectively linked to worse cognitive aging (Caplan & Schooler, 2003; Seeman et al., 1996) and incident dementia (Zahodne et al., in prep). Grounded in cognitive behavioral theory (Bandura, 1997), theories for a causal influence of perceived control on cognition include behavioral, motivational, and affective mechanisms (Lachman, 2006). Given the prominent social gradient observed for both perceived control (Ross & Mirowsky, 2013) and cognitive performance (Lee et al., 2003), associations between these constructs are likely to reflect structural mechanisms. Specifically, perceived control may serve as a proxy for a host of socioeconomic factors and other aspects of social class that are consequential for cognitive aging but are not routinely measured in dementia cohort studies. Thus, perceived control has high potential as a relatively easy-to-measure construct to quantify the extent to which an individual is affected by structural/environmental disadvantages, which can advance knowledge of racial/ethnic inequalities in dementia.

Empirical evidence for the role of perceived control in racial/ethnic inequalities in cognitive aging is growing. In both the Survey of Midlife in the United States and WHICAP, external control partially mediated cross-sectional Black-White and/or Latinx-White disparities in cognition above and beyond everyday discrimination, education, income, and health (Zahodne et al., 2017; Zahodne et al., in press). In a longitudinal study in the HRS, external control partially mediated both Black-White and Latinx-White disparities in initial episodic memory, but not subsequent rates of memory change (Zahodne, Sol & Kraal, 2019). Together, these disparate studies provide converging evidence that racial/ethnic differences in external control contribute to cognitive inequalities above and beyond other known risk factors.

In studies considering both discrimination and external control as separate, *simultaneous* mediators, external control has consistently emerged as a stronger contributor to racial inequalities in cognitive aging outcomes (Zahodne et al., 2017; Zahodne et al., in press). In addition, external control shows much more reliable Latinx-White differences than discrimination (Zahodne, Sol & Kraal, 2019; Zahodne et al., in press), as Latinx older adults often report *less* everyday discrimination than Whites (Zahodne et al., in press; Lewis et al., 2012). This pattern of results might suggest that measures of external control capture the cognitively-relevant psychosocial experiences of marginalized social groups better than commonly-used measures of everyday discrimination. Interestingly, studies considering discrimination and external control as *sequential* mediators supports discrimination as an upstream determinant of external control in psychosocial pathways leading to racial/ethnic inequalities in cognitive aging (Zahodne, Sol & Kraal, 2019).

Beyond observational studies, intervention research has revealed that racial disparities in external control also contribute to racial differences in the efficacy of cognitive interventions for older adults. In the Advanced Cognitive Training for Independent and Vital Elderly clinical trial of over 2,800 older adults from multiple sites in the U.S., both reasoning and memory interventions yielded smaller cognitive benefits for Black participants, compared with Whites, and these disparities were partially mediated by more external control among Blacks at baseline (Zahodne et al., 2015). These results may indicate that explicitly combating structural barriers to behavior change may improve the efficacy of cognitive interventions with marginalized older adults.

Summary.—Psychosocial factors are relatively under-studied dementia risk factors that help to explain racial/ethnic inequalities that persist despite statistical control for socioeconomic and physical health. In particular, measures of discrimination and external control provide useful proxies for structural and interpersonal racism that reliably predict a variety of psychological, physiological, neurological, and cognitive outcomes. The cognitive aging relevance of these context-dependent psychosocial constructs contributes to the strong evidence base for social determinants of health. Targeting an individual's environment (rather the individual) in order to modify perceived discrimination, external locus of control, and related cognitive risk may be a primary avenue to reduce racial/ethnic inequalities in dementia. Dementia cohort studies should also consider measuring racially-patterned psychosocial factors, particularly external control, as well as its contextual determinants.

Challenges in the Study of Dementia Inequalities

Incorporating biopsychosocial factors into the study of dementia inequalities is inherently challenged by issues related to sampling and survival, as well as the manifold differences in health and social circumstances across race/ethnicity that we can and cannot measure. This section highlights two particular challenges: selective survival and study recruitment. It is important to note that these are not the only challenges facing research on dementia inequalities. For example, substantial scholarship has been devoted to the important issue of measurement when quantifying and interpreting racial/ethnic differences in both neuropsychological test performance (Manly, 2005; Aiken Morgan et al., 2010) and dementia ascertainment (Gianattasio et al., 2019). The illustrative examples of selective

survival and recruitment were chosen, in part, because they have received relatively less attention in the neuropsychological literature to date.

Selective Survival

A classic demonstration of survival-related selection bias can be seen in prospective cohort studies estimating the association between smoking and incident dementia (Hernán et al., 2008). In this systematic review, studies that initially enrolled younger participants (i.e., minimum age between 55 and 75) showed an *elevated* risk of incident dementia among smokers, whereas studies that initially enrolled older participants (i.e., minimum age 75+) showed a *reduced* risk of incident dementia among smokers. A causal interpretation of these results might suggest that the oldest adults should take up smoking to ward off dementia, but there is no physiological evidence to support this recommendation. A more plausible explanation relates to selection bias. Specifically, many smokers who develop dementia or die as a result of their smoking have already done so by age 75. Those smokers who manage to survive to age 75+ dementia-free are likely to have some degree of (unmeasured) resilience that enabled them to remain healthy *despite* their smoking, and the seemingly protective effects of smoking likely reflect these other, unmeasured factors.

In the case of dementia inequalities, stark racial inequalities in survival are likely to limit our ability to measure cognitive disparities in later life due to comparing a more heavily-selected Black group to a less-heavily selected White group. Indeed, parallel analyses within disparate cohort studies of mid- and late-life adults have revealed that Black-White differences in cognitive functioning are consistently smaller in older participants than younger participants (Zahodne et al., 2016). This narrowing of racial health disparities at older ages due to selective survival helps to explain “age-as-leveler” effects observed for many health outcomes (Ferraro & Farmer, 1996), including the mortality cross-over effect in which Black Americans show a mortality disadvantage at younger ages and a mortality advantage at older ages (Wing et al., 1985). Indeed, simulation studies have demonstrated that known racial differences in survival can fully explain observed patterns of decreasing Black-White disparities in incident stroke at later ages (Mayeda et al., 2018). Importantly, selective survival not only affects the magnitude of group differences observed in older samples, it also undermines causal inferences about biopsychosocial contributors to dementia inequalities.

While survival bias has the potential to complicate any study of dementia, stark racial differences in survival make it particularly relevant to the study of dementia inequalities. Many dementia researchers are turning to younger cohorts in order to minimize the impact of selective survival and seek the origins of racial disparities, which are first evident much earlier than age 65. Researchers interested in dementia inequalities should employ study designs and statistical approaches that minimize the impact of bias due to selective survival and fully acknowledge the potential role of resultant biases in explaining results.

Recruitment

The magnitude and pattern of racial/ethnic differences in longitudinal cognitive trajectories vary across cohorts, as estimates of racial/ethnic disparities are highly sensitive to study

design characteristics, as well as regional context. Some studies report that Black and/or Latinx adults exhibit *faster* memory decline than Whites (Gross et al., 2015; Masel & Peek, 2009), while others report *slower* declines (Karlamañgla et al., 2009; Wilson et al., 2015) or *no differences* (Wilson et al., 2010; Karlamañgla et al., 2009; Wilson et al., 2016). These studies differed widely not only in geographic catchment areas (e.g., national vs. regional), but also recruitment strategies (e.g., population-based vs. convenience vs. clinical samples). Non-random sampling is a major threat to both internal and external validity and appears to be a particular problem for studies interested in dementia inequalities.

Black and Latinx older adults are often underrepresented in dementia cohort studies (Birkenbihl et al., 2020). For example, the Alzheimer's Disease Neuroimaging Initiative represents a major source of extant knowledge about the brain correlates of dementia but overwhelming includes White participants (93%). Reasons for the underrepresentation of historically marginalized racial/ethnic groups are multifactorial, and some depend on the specific study design. Underrepresentation in clinical studies reflects, in part, structural barriers to healthcare access, utilization, and satisfaction (Bierman et al., 1998). Qualitative research has highlighted experiences of unequal treatment and racism, cultural trauma due to historical and contemporary events, racial identity and cultural norms, and limited cultural competency and/or racial congruence as barriers to older Blacks' participation in clinical dementia research (Lincoln et al., 2018). In population-based samples of older adults, underrepresentation appears to be much less pronounced but persists for studies with more invasive procedures (Ofstedal & Weir, 2011).

A recent systematic review reported improvements in the representations of historically marginalized populations in dementia research (Gilmore-Bykovskyi et al., 2019). For example, a recent study of Black-White differences in incident cognitive impairment in the National Alzheimer's Coordinating Center's (NACC) Uniform Data Set reported that Black participants made up nearly 16% of the study sample, which is in line with national demographics (Gleason et al., 2019). However, diversifying study samples may involve divergent recruitment strategies by race/ethnicity that can mask population-level disparities and undermine causal inferences. For example, many of the Alzheimer's Disease Research Centers represented in NACC use a combined clinical-community recruitment strategy that is racially patterned. Specifically, less than 14% of non-demented Black participants were recruited via physician referral, as compared with over 24% of non-demented White participants. Blacks in that study demonstrated a *lower* rate of progression than Whites, which directly contradicts findings from population-based studies (Tang et al., 2001; Mayeda et al., 2016). Importantly, recruitment via physician referral positively predicted progression, raising the possibility that the unexpected Black-White differences merely reflect divergent recruitment by race.

In summary, racial/ethnic differences in recruitment are relevant not only to reconciling disparate findings *across* studies, but also to interpreting racial/ethnic differences *within* a single study. Researchers interested in dementia inequalities should strive for increased representation of Black and Latinx participants in cohort studies, but mirroring population demographics does not eliminate the potential for selection bias. Random sampling can minimize the likelihood of non-representative subpopulations. Further, over-sampling

historically underrepresented groups will improve power for between-group studies and enhance the potential to examine within-group variability, which is often overlooked in health disparities research. Finally, researchers should follow best practices for the recruitment, retention, and inclusion of underrepresented groups to minimize selection bias, as well as fully acknowledge the implications of divergent recruitment strategies for their results.

Advancing Research in Dementia Inequalities

Recommendations and Future Directions

The work reviewed above leads to several recommendations for research on biopsychosocial pathways in dementia inequalities. For example, studies of brain and cognitive aging should systematically measure not only socioeconomic factors, but also other markers of or proxies for psychosocial disadvantage, including racial discrimination and external control. Increased inclusion of historically underrepresented racial/ethnic groups is also needed, particularly in biomarker studies, and the differential impact of risk and protective factors should be evaluated. Studies interested in between-group differences should strive for racially/ethnically balanced samples rather than mirroring population demographics. However, efforts to increase the recruitment of racial/ethnic minority groups must minimize and model the impact of selection bias resulting from non-random sampling and divergent recruitment strategies. Life course studies are particularly important for understanding the origins of dementia inequalities due to selective survival and cumulative advantage/disadvantage.

Future directions should also include protective pathways among historically marginalized groups rather than taking an exclusive deficit perspective. Because observed racial/ethnic differences reflect the aggregate effects of both risk and resilience pathways, documented disparities may underestimate the negative impacts of racially-patterned social disadvantage. Considering both risk and resilience pathways will not only improve the accuracy of risk estimates but also point to additional, culturally-relevant intervention targets with high potential for effectiveness due to their natural occurrence within a community. For example, a recent study in the HRS found that racial/ethnic disparities in episodic memory were offset by higher levels of religious involvement among Black and Latinx older adults (Kraal et al., 2019).

Michigan Cognitive Aging Project

A new data collection effort to address dementia inequalities is the Michigan Cognitive Aging Project (MCAP), which follows a racially balanced sample of Black and White adults transitioning to late-life in Southeast Michigan with biological, psychological, and social measures. MCAP was designed to address many of the above-identified challenges through sampling, recruitment, retention, and measurement. MCAP is approved by the University of Michigan Institutional Review Board.

Procedures.—An address-based sample of adults aged 55–70 was constructed using 2016 voter registration lists from Washtenaw and Wayne counties, combined with

sociodemographic data available from the U.S. Census. Census tracts with high racial and socioeconomic diversity were oversampled, and individuals aged 60–65 were oversampled within each census tract. Starting in 2017, direct mailings were sent to 5,503 individuals from the 2016 voter registration list. The average response rate was 10.32%, which exceeds the 3–5% response rate typically reported for direct mail recruitment (Messer, 2006).

Participants were initially screened using the following inclusion criteria: (1) age 55 or older; (2) English speaking; and (3) no self-reported diagnosis of dementia or severe memory impairment. Of individuals who responded to the initial mailing, 71.13% enrolled in the study, reflecting an overall enrollment rate of 7.34%. Of note, a comparison of participation rates and cost effectiveness across recruitment methods concluded that direct mail using voter registration lists was a more effective strategy for recruiting older adults from the community than commercial databases, community advertisements, or clinic referrals (Katula, 2007). To demonstrate responsiveness to participant preferences and foster mutual collaboration, recruitment was expanded to include chain referral in response to overwhelming requests from enrolled participants from the address-based sample (ABS) to give personal acquaintances an opportunity to participate. Participants from the initial ABS are identified in the data set, enabling investigations of recruitment differences.

To minimize barriers to participation, participants are given the option of being interviewed in their home, at research space in midtown Detroit, or on the University of Michigan campus in Ann Arbor. Biomarker substudies (blood draw, magnetic resonance imaging) are optional. To date, approximately 83% of participants have provided blood samples when asked, and 67% of participants have been both willing and eligible for neuroimaging. (Supplementary Table 1 displays the racial/ethnic composition of MCAP, which is largely a racially balanced sample of Black and White adults. Based on characteristics shown in Table 1, participants are representative of the study catchment areas, supporting the effectiveness of the recruitment and enrollment approach.

To maximize retention, each participant is asked to provide contact information for at least one individual who would be expected to always know the participant's whereabouts (i.e., proxy contacts). All recruitment data are managed with Ripple Science (www.ripplescience.com/). Annual newsletters are used to maintain contact with participants and encourage updating of contact information. To date, newsletter content has included introductions to study staff, aggregate sociodemographic and health data on the MCAP sample, brief summaries of our research group's publications written at an eighth grade reading level, local COVID-19 resources, and tips for healthy aging. Due to the COVID-19 pandemic, telephone-based follow-ups began in 2020. To date, two-year retention of survivors is approximately 90%.

Racial Differences.—More Black participants were recruited through chain referral compared with Whites (28.23% versus 7.83%; $\chi^2(1)=31.64$; $\phi=0.26$; $p<0.001$), but there was no difference in the preference for in-home visits for Blacks versus Whites (20.16% versus 16.13%; $\chi^2(1)=1.26$; $\phi=0.005$; $p=0.262$). Racial differences shown in Table 1 are consistent with the literature and show moderate to large racial differences in SES, physical health, and depressive symptoms.

Recruitment Differences.—Respondents recruited through chain referral differed from participants in the ABS. While groups did not differ on age, participants recruited via chain referral reported lower education (12.95 versus 14.52; Cohen's $d = 0.63$; $p < 0.001$), income (\$28,248.98 versus \$58,073.03; Cohen's $d = 0.49$; $p < 0.001$) and wealth (\$108,275.57 versus \$231,666.52; Cohen's $d = 0.22$; $p = 0.036$), were less likely to own their home (27.17% versus 58.13%; $\phi = -0.24$; $p < 0.001$), and were more likely to be living under the 2017 poverty threshold (51.11% versus 20.65%; $\phi = 0.27$; $p < 0.001$) compared with ABS participants. Participants recruited via chain referral also reported more health conditions (3.27 versus 2.87; Cohen's $d = 0.23$; $p = 0.040$) and depressive symptoms (10.02 versus 7.62; Cohen's $d = 0.39$; $p = 0.001$). Evidence that participants recruited via chain referral had lower SES and worse health than participants from the ABS is consistent with the original conceptualization of chain referral as a method to identify participants from populations that have been historically underrepresented in research (Heckathorn, 1997).

Neuropsychological Data.—Summarizing data from comprehensive neuropsychological batteries into cognitive domains is commonly done in cohort studies to improve psychometric reliability of analyzed variables, to reduce the risk of Type I error, and to provide scores on a common metric (e.g., z-score metric). Therefore, theory-based confirmatory factor analysis (CFA) was used to summarize the neuropsychological data, consistent with national cohort studies (e.g., Jones et al., 2021). Residual variances were allowed between indicators drawn from the same test (e.g., Color Trails I and Color Trails II). A five-factor model fit well: CFI = 0.953; RMSEA = 0.047 (90% CI: 0.039 – 0.055); SRMR = 0.047. As shown in Supplementary Table 2, all indicators showed standardized loadings above 0.4. To facilitate analyses across researchers and software, cognitive factor scores were exported and z-scored. A global cognition score was computed by averaging the five z-scores. As shown in Supplementary Table 3, there were large to very large racial differences in the unadjusted neuropsychological scores, and a primary goal of MCAP is to characterize life course factors that contribute to these disparities.

Summary.—The Michigan Cognitive Aging Project (MCAP) attempts to overcome several challenges to high-quality research on dementia inequality. Specifically, MCAP recruits a younger and less age-heterogeneous sample of adults transitioning to late life to minimize bias related to racial differences in survival. Recruitment combines ABS and chain referral approaches, and participants can choose the location of data collection, in order to minimize selection bias, adapt to community preferences, and access underrepresented subpopulations. The sample is racially balanced (roughly 50% Black and 50% White) to maximize power for between-group and within-group analyses. Studies of biopsychosocial pathways are facilitated with in-depth measurements of life course socioeconomic and psychosocial factors that may contribute to later-life cognitive disparities via biological pathways. Comprehensive neuropsychological testing is conducted to obtain high-quality cognitive aging phenotypes. Finally, participant retention is maximized through regular, meaningful communication, as well as proxy contact.

Conclusions

Dementia disproportionately affects social groups that have been historically marginalized as the result of structural and interpersonal racism. The observation that racial/ethnic inequalities in dementia persist even after adjusting for common measures of socioeconomic status and vascular disease suggests that known dementia risk factors exhibit differential impact across racially-patterned contexts and/or that there are additional racially-patterned risk factors that are less commonly measured in dementia cohort studies. Biopsychosocial evidence is mounting that racial discrimination and external control help to explain disparities in brain and cognitive aging.

Several challenges are particularly relevant to the study of dementia inequalities. Selective survival and recruitment complicate not only cross-study comparisons, but also within-study causal inferences. These challenges can be at least partially addressed by taking a life course approach, explicitly modeling and acknowledging the influence of selection bias, partnering with communities to recruit more representative study samples, oversampling participants from historically underrepresented groups while acknowledging the implications of divergent recruitment strategies, and measuring both psychosocial risk and protective factors. In Michigan, MCAP follows a racially-balanced, representative sample of Black and White adults transitioning to late life with in-depth biological, psychosocial, and neuropsychological assessments to advance knowledge on modifiable factors that create and sustain Black-White disparities in dementia. The ultimate goal of this program of research is to optimize cognitive aging at the individual and population levels.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Public significance statement:

Racial/ethnic inequalities in dementia are a major public health and health justice concern. This paper discusses mechanisms of dementia inequalities, describes several challenges to the study of dementia inequalities, and provides recommendations for future research.

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Table 1.

Sample characteristics and racial differences

	Whole sample (N=500 ^a)	White (N=217)	Black (N=248)	Effect Size (Cohen's <i>d</i> or ϕ)
Age	63.49 (3.12)	63.69 (3.20)	63.50 (3.12)	0.06
Sex/gender (% women)	59.40	55.76	62.90	0.07
Education (years)	14.23 (2.63)	15.21 (2.45)	13.28 (2.39)	0.80*
Annual household income (in \$1,000)	52.58 (73.40)	73.12 (89.58)	31.77 (43.33)	0.59*
Poverty ^b (%)	26.28	7.91	43.28	0.40*
Wealth (in \$1,000) ^c	208.90 (626.04)	381.35 (881.17)	41.82 (155.34)	0.54*
Own home (%)	52.41	75.46	31.58	0.44*
Health conditions ^d	2.94 (1.71)	2.60 (1.65)	3.29 (1.73)	0.41*
Depressive symptoms ^e	8.06 (6.09)	5.83 (0.40)	6.25 (0.40)	1.05*

^aIncludes participants with any racial/ethnic identity

^bBased on the 2017 U.S. Department of Health and Human Services poverty guidelines

^cBased on participant's estimate of assets minus debts using wording from the Survey of Midlife in the United States (MIDUS 2)

^dSum of the presence/absence of hypertension, diabetes, hypercholesterolemia, heart disease, lung disease, cancer (excluding minor skin cancer), arthritis, hip fracture, eye disease based on questions used in the Health and Retirement Study (HRS)

^eTen-item version of the Centers for Epidemiological Studies – Depression scale; scores range from 0 to 30

* $p < .001$