

REVIEW ARTICLE

The 2022 symposium on dementia and brain aging in low- and middle-income countries: Highlights on research, diagnosis, care, and impact

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Abstract

Two of every three persons living with dementia reside in low- and middle-income countries (LMICs). The projected increase in global dementia rates is expected to affect LMICs disproportionately. However, the majority of global dementia care costs occur in high-income countries (HICs), with dementia research predominantly focusing on HICs. This imbalance necessitates LMIC-focused research to ensure that characterization of dementia accurately reflects the involvement and specificities of diverse populations. Development of effective preventive, diagnostic, and therapeutic approaches for dementia in LMICs requires targeted, personalized, and harmonized efforts. Our article represents timely discussions at the 2022 Symposium on Dementia and Brain Aging in LMICs that identified the foremost opportunities to advance dementia research, differential diagnosis, use of neuropsychometric tools, awareness, and treatment options. We highlight key topics discussed at the meeting and provide future recommendations to foster a more equitable landscape for dementia prevention, diagnosis, care, policy, and management in LMICs.

KEYWORDS

Alzheimer's disease, dementia, diversity, high-income countries, low- and middle-income countries, risk factors, vascular dementia

Highlights

- Two-thirds of persons with dementia live in LMICs, yet research and costs are skewed toward HICs.
- LMICs expect dementia prevalence to more than double, accompanied by socioeconomic disparities.
- The 2022 Symposium on Dementia in LMICs addressed advances in research, diagnosis, prevention, and policy.
- The Nairobi Declaration urges global action to enhance dementia outcomes in LMICs.

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1 | INTRODUCTION

Approximately 50 million people worldwide have dementia, an estimate projected to reach nearly 150 million by 2050.¹ Current prevalence estimates suggest two of every three people living with dementia (PLWD) reside in low- and middle-income countries (LMIC) (Figure 1). Of concern, the number of PLWD in LMICs is projected to more than double.^{1,2} These increases can be largely attributed to population growth and the shift in population structure toward older adults. While population growth is projected to be imperative in sub-Saharan Africa (SSA), population aging per se is also an important contributory factor in East Asia.¹ Socioeconomic disparities underlying LMICs are a critical factor impacting the understanding of dementia in terms of risk factors and care of patients.^{3,4}

In 2019, the worldwide cost of dementia was estimated at USD1.3 trillion, and this figure could reach more than USD2 trillion by 2030. However, these estimates are mostly driven by HICs.⁶ In fact, predictive models show 74% of the estimated global dementia cost occurs in HICs, despite the fact that most PLWD reside in LMICs.⁷ In LMICs, family care accounts for 65% of the dementia cost, while direct medical care and social care account for only 35%. In contrast, informal care accounts for 44% of dementia costs in HICs.⁷ In regions such as Latin America, caregiver burden is among the largest in the world and more strongly affected by gender disparities.⁸ These figures may reflect under- or misdiagnosis of dementia and poor availability and access to dementia care services in LMICs.⁹

Key common factors across LMICs that could contribute to the lived experience of dementia in these regions include low public awareness of dementia, high stigma and misconceptions, low literacy, insufficient dementia-related knowledge and training among professionals and caregivers, poor interprofessional cooperation, inappropriate predictive and analytical care models, and lack of equitable access to suitable services.^{10,11} Recognizing the importance of such challenges, the World Health Organization (WHO) has developed the "Global Action Plan on the Public Health Response to Dementia 2017 – 2025," which aims to provide policymakers and dementia experts with a roadmap to address dementia globally and regionally.¹² However, the WHO's 2021 report showed that most LMICs do not currently have a national dementia plan, highlighting insufficient progress in addressing the global dementia action plan by 2050.^{7,13}

An important challenge in addressing the impact of dementia in LMICs is the underrepresentation of ethnically and geographically diverse individuals in research studies. Dementia research is mostly conducted in HICs, where only 18% of the world's aging population resides.¹⁴ Only a fraction of dementia clinical trials are conducted in LMICs, limiting the participation of populations from these countries^{15,16} (Figure 2). Consequently, the results of trials conducted in HICs may not be directly applicable to LMICs, due to differences in disease profiles and severity, healthcare access, affordability, infrastructure, skills, and other resources. Determinants of healthy aging are highly heterogeneous according to regional diversity, calling for urgent tailored perspectives in global approaches.⁴ Furthermore, biological

RESEARCH IN CONTEXT

- 1. Systematic review:** Emerging findings suggest that the projected increase in global dementia rates is expected to affect low- and middle-income countries (LMICs) disproportionately, yet most resources are allocated to high-income countries (HICs). The authors of this article highlight advances in dementia research, issues in diagnosis, and care of dementia patients discussed at the 2022 Symposium on Dementia and Brain Aging in LMICs, held in December 2022 in Nairobi, Kenya.
- 2. Interpretation:** Research presented at the symposium highlighted the importance of LMIC-focused research to ensure that characterization of dementia accurately reflects the involvement and specificities of diverse populations.
- 3. Future directions:** A multifaceted approach is necessary to reduce the impact of dementia in LMICs that involves developing and promoting national dementia plans, robust policies, inclusive research, and building capacity. These and other recommendations were set forth by the Nairobi Declaration with the aim of improving dementia prevalence, outcomes, and personal and societal impacts in LMICs.

and genetic variations across populations may influence treatment responses and the frequency of adverse events to investigational products.^{17–19}

Limited dementia research conducted in LMICs may also narrow the scope of studies away from questions particularly relevant to the LMICs. For example, Alzheimer's disease (AD) accounts for around 60% of all dementia in HICs, while vascular dementia (VaD) accounts for only 15%. However, in LMICs, VaD constitutes about 30% of dementia cases.^{20,21} Furthermore, the apolipoprotein E (APOE) genotype may modify dementia risk differently based on ancestral backgrounds.^{22,23} Thus, developing preventive and therapeutic strategies for dementia in LMICs requires consideration of the unique environmental factors and the genetic diversity in these regions. Aging populations in LMICs often differ markedly in culture, living environment, and resources, requiring not only rigorous but disparate approaches to research. It is also not unlikely that cycles of infectious diseases such as the COVID-19 pandemic will modify the overall burden of dementia, not only in LMICs but globally.^{24,25}

In light of these and other challenges, the 2022 Symposium on Dementia and Brain Aging in LMICs, held in Nairobi, Kenya, focused on relevant advances in dementia research, diagnosis, prevention, policy, and care in LMICs. The aim was also to foster national and international collaboration in LMICs. The symposium was convened by the Alzheimer's Association (USA), Newcastle University (UK), and the University of Texas Rio Grande Valley (USA) and funded in part by the

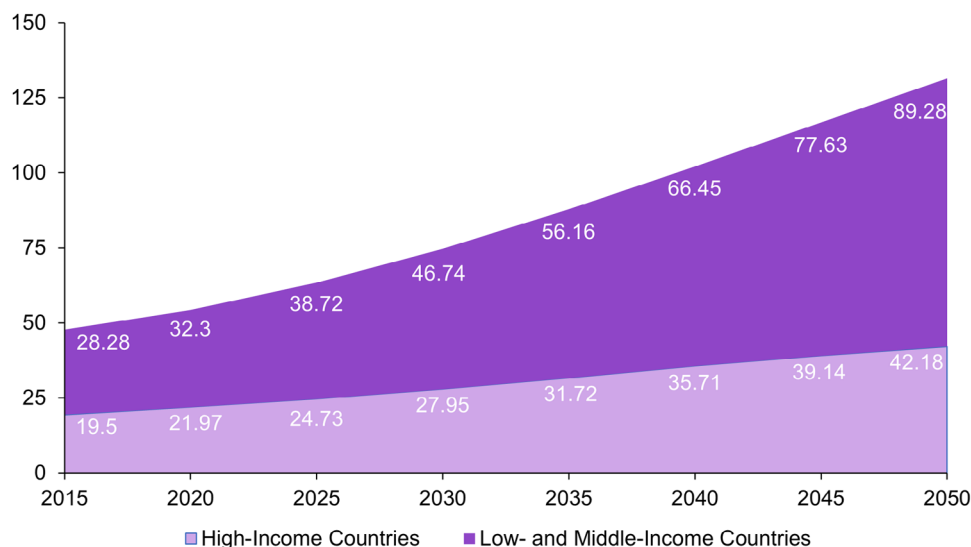


FIGURE 1 Projected prevalence of dementia cases in LMICs compared to HICs. Graph labels: x-axis shows year; y-axis represents numbers in millions. Data modified from Wimo et al. (2018).⁵

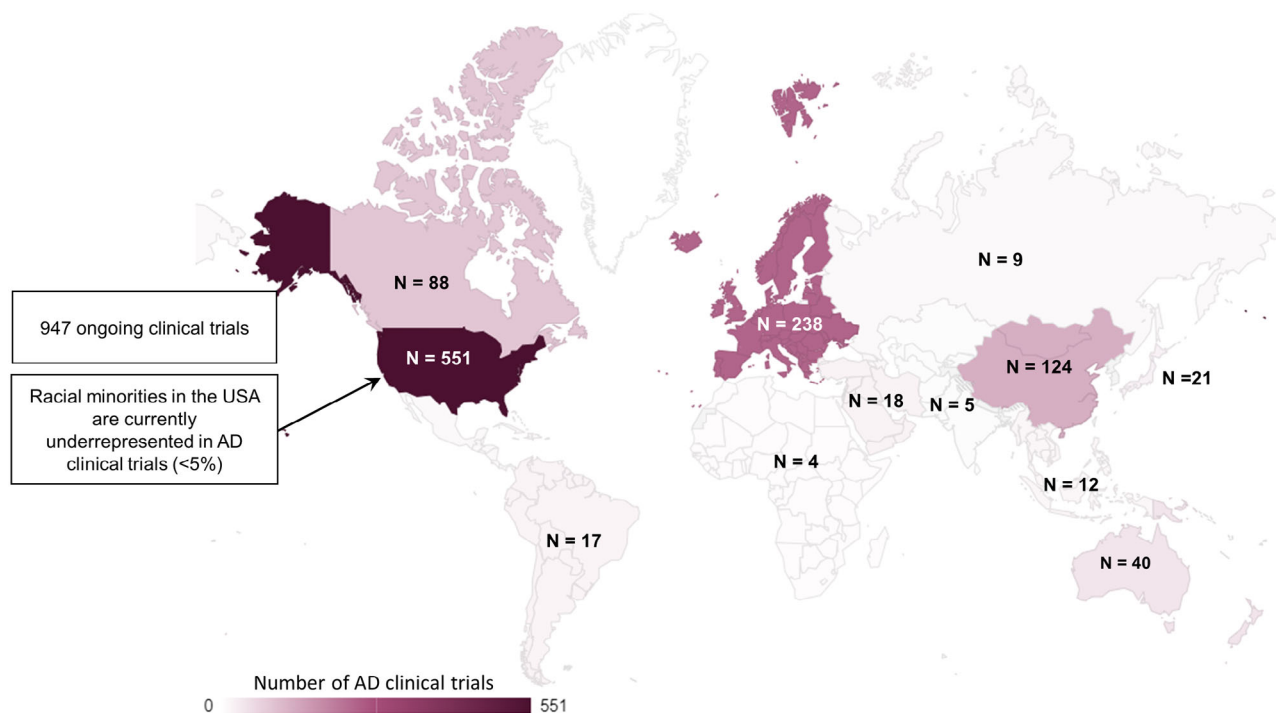


FIGURE 2 Differential numbers of pharmacological clinical trials in AD between LMICs and HICs. Compiled by R Allegri, Argentina, 2022 using data from clinicaltrials.gov.

National Institute on Aging (NIA), USA. Several specific topics, such as cost of dementia and prevalence in LMICs, VaD, and stroke, movement disorders, language and aphasia, genetics, modifiable risk factors, human immunodeficiency virus, dementia care, and policy were discussed by invited experts. In this review, we provide a summary of the topics, highlighting the urgent need for targeted efforts to address the dementia burden in LMICs.

2 | MODIFIABLE RISK FACTORS FOR DEMENTIA

A growing number of studies indicate stable or declining incidence and prevalence of dementia in HICs over the last 25 years.^{26,27} Decline in dementia rates has been attributed mainly to improved cardiovascular health due to lifestyle changes, improved social and welfare systems, prompt diagnosis and treatment of comorbidities, greater access to

medication, and increasing education levels. Such promising findings suggest that identifying and targeting modifiable risk factors may be effective strategies to reduce risk for dementia.²⁷ In contrast, current evidence suggests that dementia rates in low-resource settings, including in LMICs, are not declining but expected to even rise steadily, as indicated by the estimated projections up to 2050.²⁷ While up to 40% of dementia cases worldwide have been attributed to the 12 modifiable risk factors identified by the 2020 Lancet Commission,²⁸ the population attributable fraction (PAF) – the fraction that is theoretically prevented by eliminating risk factors – varies greatly between LMICs and HICs.^{29,30}

Cross-sectional data from the 10/66 Dementia Research surveys have permitted the PAF for dementia associated with nine modifiable risk factors to be calculated in select LMICs, including China, India, and Latin American countries. The modifiable risk factors included lower early-life education, midlife hearing loss, hypertension, obesity, later-life smoking, depression, physical inactivity, social isolation, and diabetes. The results showed that the PAF for dementia was 39.5% in China, 41.2% in India, and 55.8% in Latin America. Furthermore, five risk factors were more prevalent in these LMICs than worldwide estimates, including less childhood education, smoking, hypertension, obesity, and diabetes.²⁹ Importantly, risk models from global settings may not simply be extrapolated to LMICs.³¹ Within Latin America, researchers analyzing longitudinal data from the Brazilian Longitudinal Study of Aging (ELSI-Brazil) study observed varying PAF for different regions of Brazil.³⁰ The 12 modifiable risk factors for dementia accounted for 54% of the cases in poorer regions of Brazil versus 49% in wealthier regions, with less education, hypertension, and hearing loss being the most important risk factors.³⁰ New reports on risk factors in Latin America and Indonesia reveal a larger involvement of factors related to social and health disparities than factors such as age or gender.^{32,33}

Together, these findings indicate that a higher percentage of dementia cases may be attributed to modifiable risk factors in LMICs than in HICs. Therefore, the potential for dementia prevention by addressing modifiable risk factors is particularly significant for LMICs. As many risk factors can cluster,^{34–37} implementing effective policy interventions that enhance access to healthy foods, promote physical activity, and address social and economic disparities in LMICs is crucial. Furthermore, population-based strategies that utilize pragmatic, broadly applied, and low-cost interventions are needed. As an example, the Latin American Initiative for Lifestyle Intervention to Prevent Cognitive Decline (LatAm-FINGERS) trial is a randomized controlled trial for dementia prevention that simultaneously targets multiple modifiable risk factors.³⁸ Similarly, the ongoing World-Wide FINGERS in Africa (AFRICA-FINGERS) project aims to assess culturally informed multimodal intervention strategies for promoting brain health in indigenous African elders at risk of dementia and assess barriers to adherence and sustainability of the intervention via a precision-prevention framework. At the 2022 symposium, the role of several modifiable risk factors for dementia within LMICs was highlighted. In what follows, we provide a summary of some of these important results.

2.1 | Diet

Observational studies indicate that a healthy diet throughout life is essential for maintaining optimal brain health, particularly vascular brain health.³⁹ Individual dietary components such as fruits, vegetables, and fish have been linked with a lower risk of dementia and slower memory decline⁴⁰ and possibly decreased brain amyloid beta (A β) accumulation.⁴¹ Dietary patterns such as the Mediterranean diet (MEDi diet) and Dietary Approaches to Stop Hypertension (DASH diet) have been associated with reduced risk of dementia and AD and lower rates of cognitive decline in some studies.⁴² Such dietary patterns emphasize a high intake of plant-based foods and low saturated lipids and red meat consumption.^{40,43} However, adherence to the MEDi and DASH Diet Intervention (MIND) may be influenced by individual income, evident in some countries like Brazil.⁴⁴ This implies the need for more affordable dietary interventions in LMICs.

While diets rich in whole grains, fruit, and legumes have been linked with dementia risk in some studies, the burden of non-communicable chronic disease due to low dietary fiber in SSA has increased substantially between 1990 and 2019.⁴⁵ A study in the Central African Republic showed that reduced consumption of oleaginous foods was associated with a 3.7- and 2.8-fold higher risk of mild cognitive impairment (MCI) and dementia, respectively.⁴⁶ The rising consumption of ultra-processed foods, which is associated with cognitive decline, is a concerning trend in LMICs.⁴⁷ Furthermore, while higher fish intake and omega-3 fatty acids found in fish are other important dietary components linked with better cognitive health,^{48,49} limited physical and economic access hinder optimal fish intake in SSA.⁵⁰

There is a pressing need for comprehensive programs and policies that address the potential constraints of availability, cost, convenience, and preferences of healthy diets in LMICs. For example, in both urban and rural Indonesia, prevention strategies and public health messages in primary care facilities have been put in place to promote healthy diets containing tempe (a common Indonesian soyabean preparation) or tofu and fruits and provision of physical and psychosocial activities.³² By adopting similar initiatives, LMICs can promote healthy dietary and activity patterns tailored to specific cultural backgrounds and geographical locations with the ultimate aim of reducing dementia risk and improving brain health.

2.2 | Physical activity

An increasing body of evidence underlines the importance of physical activity in dementia risk. Research in the U.S.-based Framingham Heart Study (FHS) cohorts shows that exercise may contribute to reduced risk of dementia and that sedentary individuals are at an increased risk of developing dementia compared to individuals with higher physical activity levels.⁵¹ However, additional studies are needed to investigate the effects of physical activity on dementia risk in LMICs. A 2012 study of aging in Indonesia found that only 1.7% of older men and 0.7% of older women surveyed reported engaging in exercise or sports, with rates ranging from nearly 0% to 3.6% for women and 4.4% for

men across provinces.⁵² Furthermore, significant regional differences show more older people in Jakarta engage in physical activity than in poorer regions such as Borobudur.³² Research in Colombia has also shown that physical activity may substantially reduce the risk of MCI.⁵³ Public health strategies and interventions for enhancing overall health and reducing dementia risk in LMICs that focus on promoting physical activity may be best done via community centers in groups to ensure uptake and adherence.³²

2.3 | Hypertension

Evidence from epidemiological studies suggests hypertension as a significant risk factor for dementia, especially midlife hypertension.⁵⁴ Promising results from clinical trials have also emerged, with the Systolic Blood Pressure Intervention Trial—Memory and Cognition in Decreased Hypertension study (SPRINT-MIND) showing that intensive blood pressure lowering reduces the composite outcome of MCI and probable dementia in those 50 and older.^{43,55,56} From 1975 to 2015, the highest worldwide blood pressure values have been shown to be shifted from HICs to LMICs in South Asia and SSA.⁵⁷ Between 1990 and 2019, the summary exposure value (ie, a metric that captures risk-weighted exposure for a population or risk-weighted prevalence of an exposure) for high systolic blood pressure increased in SSA, the Middle East and North Africa, East Asia, and the Pacific. Despite this shift toward high blood pressure, only one in three individuals in LMICs are aware of their hypertension status, and only approximately 8% have their blood pressure under control.⁵⁸ A cross-sectional, pooled individual-level population-based study examining hypertension care in 44 LMICs found that countries in Latin America and the Caribbean performed better than predicted on hypertension care cascade defined based on having one's blood pressure measured, being diagnosed with hypertension, being treated for hypertension, and achieving control of one's hypertension. However, countries in SSA performed significantly worse on these steps relative to their predicted performance based on per-capita gross domestic product.⁵⁹ Thus, policies and interventions are urgently needed to increase hypertension awareness, diagnosis, control, and treatment rates in LMICs, as those efforts may contribute to not only increased cardiovascular health but also to improved brain health.^{58,60,61}

2.4 | Social networks

Some studies suggest that supportive social networks may reduce the risk of AD and related dementias. Social networks may enhance cognitive resilience or the capacity for cognitive processes to be less impacted by age- or disease-related changes. A cross-sectional study evaluating 2171 U.S. adults revealed that social support in the form of supportive listening was associated with greater cognitive resilience, as indicated by individuals exhibiting higher levels of global cognitive function than predicted, given their cerebral volumes.⁶² This is consistent with observational evidence indicating 30% to 50% lower

subsequent dementia risk is associated with greater social participation in midlife and late life.⁶³ Few studies have investigated the link between social networks and neurocognitive outcomes in LMICs. A recent study conducted in China, Ghana, India, Mexico, Russia, and South Africa among individuals aged ≥ 50 years found that every one-unit increase in the social participation score (a score ranging from 0 to 10, with higher scores corresponding to greater levels of social participation) was associated with a 13% decrease in the odds of developing MCI.⁶⁴ In Latin American countries such as Brazil and Columbia, social isolation or perception of loneliness was determined to be critical in predicting decline or performance in cognitive and functional abilities.³³ In studies from Indonesia, engaging in community activities has been associated with lower dementia risk.³² Thus, psychosocial interventions and public health strategies aimed at increasing social support and participation may help reduce the risk of dementia, particularly in LMICs disproportionately affected by dementia.

2.5 | Sensory impairment: Prevention and intervention

Hearing and visual impairments are frequently underidentified among PLWD, but there is evidence to suggest that they are associated with accelerated cognitive decline, increased neuropsychiatric symptoms, and communication barriers.^{28,65} Furthermore, sensory impairment reduces the quality of life for PLWD and their caregivers.⁶⁶ Recent longitudinal studies in older Americans have linked hearing aid use and cataract surgery with slower episodic memory decline than was observed before interventions.⁶⁷ More research is needed to determine whether early identification and treatment of sensory impairment in cognitively healthy individuals prevent neuropathology and reduce dementia risk (ie, selective primary prevention). Interventions for sensory impairment focused on indicated primary prevention or secondary dementia prevention (ie, preventing conversion from MCI to dementia and preventing the progression of existing dementia, respectively) are also lacking. There is clearly a need for appropriately powered hearing and vision impairment trials because scoping studies suggest consistent evidence is lacking for positive impact of hearing or vision interventions on cognitive function or decline, quality of life, or caregiver burden.⁶⁶ However, recently the Aging and Cognitive Health Evaluation in Elders (ACHIEVE) trial showed that hearing intervention in the Atherosclerosis Risk in Communities cohort might reduce cognitive change over 3 years in older adults (mean 79 years) at increased risk of cognitive decline but not in those at decreased risk of cognitive decline.⁶⁸

To advance such efforts, the European **SENSE-Cog project** has developed multifaceted interventions to improve the lives of elderly individuals with sensory impairment and dementia. Preliminary results showed that the care dyads exhibited improved quality of life and sensory functional ability in a trial conducted in France, England, and Cyprus.⁶⁹ Findings from this project led researchers to develop the **SENSE-Cog project** in South Asia (Pakistan, India, and Bangladesh). All assessment and outcome rating tools were translated into local

languages, and intervention materials were modified to include culturally relevant pictures and consider participants' literacy levels. Preliminary findings from Pakistan demonstrated positive feasibility, acceptability, and tolerability of the intervention by study participants. However, study recruitment, timely hearing aid delivery, procedures for arranging home visits, and communication between referring clinicians and the study team were identified as areas in need of improvement.⁷⁰

3 | VASCULAR COGNITIVE IMPAIRMENT, VASCULAR DEMENTIA, AND STROKE-RELATED COGNITIVE IMPAIRMENT

Vascular cognitive impairment (VCI) captures the entire spectrum of cognitive disorders related to cerebrovascular injury, ranging from mild to severe VCI, which is equated to VaD.⁷¹ In HICs, VaD accounts for 15% of dementia cases, but the burden of VaD is even greater in LMICs, where it accounts for approximately 30% of dementias.^{20,21,72–74} Consistent with this, the prevalence of cerebral small vessel disease causing VCI is also expectedly high in LMICs.^{75,76} Research to elucidate effective preventive care or treatment strategies for VCI is limited in LMICs,^{43,77} and more data are needed particularly from SSA, Eastern Europe, and Latin America.

The 2022 symposium devoted a session to VaD and stroke, covering a range of topics, including mild VCI or preclinical VaD and biomarkers of vascular contributions to dementia, stroke, and post-stroke dementia. In what follows, we provide the summary of key discussions from this session that were relevant to LMICs.

3.1 | VCI and VaD

The prevalence of VCI is particularly high in India and in SSA countries such as Tanzania,⁷⁴ with nearly 40% of dementia cases attributed to VaD.⁷⁷ A hospital-based cohort of 42 patients in India with VaD found that subcortical dementia was the most common subtype of VaD (52%), followed by cortical–subcortical dementia (26%), strategic infarcts (14%), and cortical dementia (7%).⁷⁸ Furthermore, a large study of over 5000 individuals from nine Asian cities found that white matter lesions deemed to be of vascular origin on magnetic resonance imaging (MRI) or computed tomography (CT) are highly prevalent and are associated with worse cognitive performance.⁷⁹ *Post mortem* studies from Brazil have also identified a high prevalence of VaD. For example, a neuropathological examination of 1092 participants from the Biobank for Aging Studies in Brazil showed that cerebrovascular lesions are common, with 35% meeting the criteria for VaD diagnosis.²¹

Key risk factors for VCI are cardiovascular risk factors such as hypertension, dyslipidemia, diabetes, smoking, obesity, atrial fibrillation, and coronary artery disease.^{71,80} While these factors are prevalent in HICs, LMICs face a much greater disparity. For example, over three-quarters of cardiovascular deaths take place in LMICs,⁸¹ yet the availability and affordability of cardiovascular and chronic disease

medications could be low in many LMICs relative to HICs.⁸² In particular, hypertension is on the rise in LMICs, and many are not aware of their hypertension status.^{57,83} Therefore, individuals in LMICs may be more susceptible to developing VCI and potentially more severe VaD.

3.2 | Stroke and post-stroke dementia

Stroke is among the strongest predictors of VCI and VaD.⁸⁰ Furthermore, post-stroke dementia is recognized as a major subtype of VaD according to the Vascular Impairment of Cognition Classification Consensus Study classifications.⁸⁴ Between 1990 and 2019, the global stroke burden increased substantially, with the majority residing in LMICs.⁸⁵ The burden of stroke is especially high in Africa,⁷⁶ with an age-standardized incidence rate of up to 316 per 100,000 population and a prevalence rate of up to 1460 per 100,000 population.^{86,87} In India, distinct patterns of ischemic stroke subtypes have been reported, with intracranial atherosclerosis accounting for 30% of ischemic strokes.⁸⁸ While the prevalence of stroke is higher in LMICs compared to HICs,^{89,90} there are limited data on the prevalence of post-stroke dementia in LMICs. Studies in SSA have reported varying prevalence estimates for post-stroke cognitive impairment, ranging from 6.3% in Nigeria to 25% in Central Africa.⁹¹ The Cognitive Function After STroke (CogFAST) Nigeria Study found that at 3 months after stroke, 40% of participants had cognitive impairment with no dementia, and 8.4% had dementia.⁹² Important determinants of cognitive impairment after stroke in the CogFAST study were increasing age, lower education, pre-stroke cognitive decline, medial temporal lobe atrophy, and pre-stroke diet.⁹²

Collectively, severe VCI or VaD and stroke continue to represent a significant burden in LMICs compared to HICs, and this gap is increasing. Early identification and effective management of cardiovascular risk factors and diseases in LMICs is a crucial step in alleviating the burden of stroke and VaD, which are on the rise in LMICs. To further enhance VaD research and its robust impact in LMICs, sustainable equitable partnerships must be built. In LMICs, several promising future research directions are emerging to address the challenges of VaD in the region. These studies aim to conduct longitudinal studies on larger samples, establish molecular and genetic risk prediction tools for VCI, understand neuroimaging and electroencephalogram determinants of VaD, and elucidate cardiovascular risk factors and cognitive outcomes that lead to VaD.

4 | COGNITIVE DYSFUNCTION IN PARKINSON'S DISEASE

The 2022 symposium included a session on movement disorders with a discussion on mechanisms and genetic factors that may underlie the link between movement disorders and cognitive impairment with a particular focus on Parkinson's disease (PD). Cognitive impairment is the most common non-motor symptom (NMS) of PD, ranging from mild cognitive difficulties in one or more of the cognitive domains to

severe dementia.⁹³ The prevalence of MCI in PD has been estimated to be 40%.⁹⁴ Furthermore, individuals with PD are five to six times more likely to develop dementia, and this risk increases with age and PD symptom severity.⁹⁵ While the WHO has identified LMICs as the region with the most affected PD individuals,⁹⁶ there is a significant lack of data on the burden of PD-associated cognitive impairment in LMICs, including Asia and Latin America.

Research on PD-associated cognitive impairment within Africa was particularly highlighted in the discussions held at the symposium. It was noted that there is a lack of studies on the prevalence and burden of PD-associated cognitive impairment in Africa, with most studies being hospital-based. One of the earliest reports in Africa was from a hospital-based study by Akinyemi et al.⁹⁷ This study determined the frequency, pattern, and predictors of cognitive impairment among 51 Nigerian PD patients that showed the rate of cognitive impairment in PD patients was 21.6% versus 4% in controls, with memory, language, and executive function being most affected.⁹⁷ Furthermore, older age at PD onset was the only independent predictor of cognitive impairment.⁹⁷ Another Nigerian study assessing the frequency of cognitive impairment and depression among 40 PD patients reported that 60% of PD patients had cognitive impairment compared to 5% in controls.⁹⁸ A more recent study assessing the profile and burden of NMS within 825 members of the Nigeria Parkinson Disease Registry reported that frequencies of cognitive complaints ranged between 27% and 46%.⁹⁹ Collectively, these data suggest that cognitive impairment is highly prevalent in the African PD population and similar to that in other populations.⁹⁹

Epidemiological data on PD-associated cognitive impairment are still lacking in LMICs. There is an especially urgent need for robust estimates of prevalence, incidence, and risk factors from population-based studies to better evaluate the burden of PD-associated cognitive impairment in diverse populations. Furthermore, developing cross-culturally validated and efficiently implementing cognitive screens for PD in LMICs is essential. It is especially important since cognitive impairment in PD is associated with a loss of independence, worsening quality of life, increased caregiver burden, and higher mortality risk.¹⁰⁰ Finally, unequal healthcare access, lack of resources, and treatment for people living with PD and associated neurocognitive outcomes need to be addressed in LMICs.

5 | HUMAN IMMUNODEFICIENCY VIRUS AND DEMENTIA

In the era of antiretroviral therapy (ART), human immunodeficiency virus (HIV) has emerged as a lifelong chronic disease that carries several HIV-related complications. One of the major HIV-related complications is cognitive impairment,¹⁰¹ which diminishes the health-related quality of life in people living with HIV (PLWH).¹⁰² HIV-associated neurocognitive disorder (HAND)¹⁰³ has an estimated global prevalence of 43%, with South America and SSA being most affected.¹⁰⁴ A systematic review in 2021 found that prevalence estimates for HAND in SSA varied between 14% and 88% and were impacted by factors like different diagnostic approaches, sampling

methods, and ART status.¹⁰⁵ Another systematic review of 50 studies from 15 SSA countries showed that the prevalence of symptomatic HAND among PLWH aged ≥ 50 years on cART ranged between 19% and 61%.¹⁰⁶ Furthermore, individuals growing old on ART may be susceptible to more cognitive decline as a result of the disease if not treated early as well as the possible effects of long-term ART use on the brain. However, further research needs to be conducted on individuals surviving into old age with HIV.

5.1 | Variations in prevalence of HAND

Variable prevalence estimates for HAND in LMICs may stem from differences in diagnostic approaches and neuropsychological tests. In a 2021 study, researchers applied 20 different methods for determining cognitive impairment in 148 South African PLWH who also underwent structural MRI and diffusion tensor imaging. Researchers observed wide variation (20% to 97%) in estimated rates of cognitive impairment defined by each method, while no method correlated with neuroimaging markers of HIV-associated brain injury.¹⁰⁷ Other factors that may contribute to variable prevalence estimates of HAND are psychosocial and educational factors. A study conducted in South Africa in 2013 found that HIV status and education were the strongest predictors of total scores on the Montreal Cognitive Assessment test (MoCA).¹⁰⁸ Another study in South Africa found that psychosocial factors (ie, less education and greater food insecurity) were stronger predictors of poorer overall cognitive performance than medical factors among PLWH.¹⁰⁹ Therefore, applying solely quantitative methods based purely on cognitive test scores to diagnose cognitive impairment in PLWH may not accurately reflect HIV-associated brain injury. Incorporating clinical judgment and factors that affect performance on neuropsychological tests are important considerations that can enable improved assessment and diagnostic accuracy of cognitive impairment in PLWH.^{107,109}

Currently, criteria for HAND can be met based on cognitive tests alone, and many researchers have argued that this approach is inappropriate.^{110,111} In addition, some studies have cautioned on potentially high false positive classification rates for HAND.¹¹² Thus, clinicians and policymakers should exercise appropriate caution when interpreting research findings on HAND burden. While underestimating the prevalence of cognitive impairment in PLWH will negatively impact the speed at which it can be contained, overestimating the prevalence may lead to misallocating resources that are especially scarce in LMICs.¹⁰⁵ Overestimating cognitive impairment may also increase the risk of stigma and discrimination toward PLWH.^{110,113}

To improve HAND diagnostic criteria, researchers and clinicians from neurology, psychiatry, and infectious disease developed an International HIV-Cognition Working Group, with approximately 50% LMIC membership. The working group has published recommendations for evaluating neuropathology, interpreting cognitive test results, and diagnosing cognitive impairment in PLWH.¹¹¹ The group has proposed the conceptual separation of HIV-associated brain injury from other causes of brain injury in PLWH. It has also recommended moving away from only quantitative methods of diagnosing cognitive impairment in

PLWH while placing emphasis on the clinical context. These recommendations will require validation, field testing, and a broader consensus but may lead to the development of a more precise phenotype for cognitive impairment in PLWH.¹¹¹

6 | LANGUAGE, APHASIA, AND DEMENTIA

Traits of dementia-related speech and language deficits, such as aphasia and subtypes such as dysnomia, characterized by difficulty in finding and recalling words, are valuable early indicators of cognitive impairment.^{114,115} However, research and clinical criteria have largely been limited to Western languages and have not adequately accounted for the impact of linguistic diversity on the manifestation of aphasia. Before 1945, most research on aphasia was conducted in German and French, with subsequent studies primarily conducted in English.¹¹⁶ A study of 1265 articles on aphasia published between 2000 and 2009 reported a pronounced bias toward articles focused on English-speaking patients, accounting for 62% of all papers.¹¹⁶ In comparison, some of the most widely spoken languages in the world (eg, Arabic, Hindi, Bengali, Russian, and Portuguese) accounted for only <0.5% of the aphasia literature. More than 90% of aphasia treatment studies were based on English, German, and Dutch-speaking patients.¹¹⁶ Such findings underscore the extreme bias toward Western languages in aphasia or dysnomia¹¹⁷ assessment and treatment, limiting clinical findings' applicability and generalizability to diverse populations.

6.1 | Heterogeneity in aphasia manifestation

It is important to note that aphasias manifest heterogeneously across different languages. In a study of linguistic impairment patterns in Bengali speakers, Bengali-speaking AD participants tended to significantly underuse pronouns, whereas English-speaking AD patients tended to overuse them.¹¹⁸ Other studies have shown that German¹¹⁹ and Hebrew-speaking AD patients¹²⁰ exhibited no impairment in verb inflection, whereas English-speaking AD patients tended to have more inflectional errors than the respective control patients.¹²¹ It has been suggested that reference to the past is challenging for patients with agrammatic aphasia, but a study of time reference in Thai speakers with agrammatic aphasia showed inconsistent results.¹²² Thai agrammatic speakers had more vulnerability to future reference than the present and the past.¹²² Thus, the morphology and characteristics of a patient's language are important considerations for the accurate assessment, diagnosis, and treatment of aphasia. Future studies are essential to enhance our understanding of cross-linguistic differences in aphasia, particularly across morphologically distinct and underexplored languages.

Language-specific considerations should be made for bilingual and multilingual patients. Most bilingual aphasias manifest in parallel in both languages, but there is also evidence of differential or selective aphasia.¹²³ For example, some studies show that in bilingual patients with aphasia, the first language is best preserved,¹²⁴ while others show

that the last language is best preserved.¹²³ Older studies also suggest that the emotionally relevant language may be better preserved, or bilingual patients may exhibit different types of aphasias in different languages.^{123,125} In bilingual patients with semantic dementia, the less proficient language was lost at presentation, with some patients even failing to recognize the language. This was seen across English and several Indian languages like Kannada, Hindi, Tamil, and Telugu.¹²⁶ In bilingual agrammatic speakers of Swahili and English in Africa, agrammatic patients make no errors in the past or future tense in Swahili. At the same time, they have selective deficits in producing past tense in English.¹²⁷ Such observations have relevant implications for assessing aphasia in multilingual patients in LMICs. This also implies aphasia treatments may not be generalizable to various linguistic and cultural backgrounds.

6.2 | Effect of dual language use on cognition

It is also important to note that dual language use may affect cognitive function. In this regard, there are two main competing hypotheses: (1) the subtractive effect of bilingualism where bilinguals show deficiencies in neuropsychological test performance, particularly naming and verbal fluency tests, compared to monolinguals and (2) the additive effect of bilingualism where bilinguals may outperform monolinguals in tasks involving executive controls, particularly tasks requiring inhibitory control in non-verbal tasks.¹²⁸⁻¹³⁰ The benefit of bilingualism has also been shown in verbal memory tests in which inhibitory control is required to reduce proactive and retroactive interference.¹³¹ The advantage of bilinguals in tasks requiring inhibitory control may be the consequence of bilinguals inhibiting the language not in use at any given moment¹³² or their constant need to keep track of both languages in order to select and activate the appropriate language (switching).^{128,130,133} It has also been shown that keeping two languages active increases cognitive reserve and may delay the emergence of dementia.^{128,134} Furthermore, bilingualism has been associated with structural and functional alterations in the brain.¹³⁵⁻¹³⁷ Further research is needed to better understand how bilingualism is related to neurocognitive processes though, as some studies have failed to replicate empirical evidence of the protective effect of bilingualism on dementia progression.¹³⁸⁻¹⁴⁰

In summary, to enhance our understanding of aphasia across languages and cultures, it is important to develop culturally sensitive assessment tools and appropriately tailored interventions. Neglecting cultural and linguistic diversity, as well as multilingualism in aphasia, may lead to incorrect diagnosis and ineffective treatment strategies. Such oversight could lead to inappropriate or inaccurate theories that cannot be generalized to diverse populations.

7 | GENETICS OF DEMENTIA

Profound insights into the genetics of AD have been gained from focusing on genes that cause autosomal dominant familial AD (FAD), as well

as genes that have a substantial influence on late-onset AD (LOAD), such as APOE.¹⁴¹ Building on this foundation, more recent research has identified additional genetic variants that modify AD risk.^{142,143} Historically, most research on the genetic risk of AD focused on populations with Caucasian European backgrounds.¹⁴⁴ However, more recent studies in LMICs and other global population groups highlight the importance of diversity and inclusion of all populations in genetic research because many genetic variants that influence AD risk are not present in European local ancestry but instead reflect genetic admixtures derived from European, African, Amerindian, and East Asian ancestries.¹⁴⁵ This more recent inclusion of diverse global populations contributes to tremendous advances in unraveling the variation and complexity of genetic risk and resilience to AD and related dementias.¹⁴⁶

7.1 | Autosomal dominant familial AD in Latin America

Studies of geographically and genetically isolated populations in Antioquia Colombia have led to the exciting understanding of a form of FAD caused by an E280A point mutation in the presenilin 1 gene (*PSEN1*).¹⁴⁷ Following the identification of the first cluster family in the 1980s, subsequent pedigree identification, genealogy, and follow-up work throughout rural Antioquia identified 25 affected families sharing a common ancestry that could be traced back to a founder in the 18th century.¹⁴⁸ This cohort now consists of 25 multigenerational families and includes around 5000 individuals and about 1200 living *PSEN1* E280A mutation carriers.¹⁴⁹

Collaborative studies on this Colombian kindred have established key characteristics of AD neurocognitive and neuropathological progression in *PSEN1* E280A carriers. Neuropsychological testing identified several stages of progressive clinical deterioration that can take as long as 25 years, including asymptomatic pre-MCI, symptomatic pre-MCI, MCI, and dementia with overall clinical deterioration.^{150,151} Positron emission tomography (PET) imaging of *PSEN1* E280A carrier brains found fibrillar A β accumulation beginning during asymptomatic stages at a mean age of approximately 28 years.¹⁵² On the other hand, elevated levels of tau deposition were observed 6 years before AD clinical onset, at the mean age of 38 years, suggesting a decade gap between the development of amyloid and tau-PET pathology.¹⁵³ In addition, *PSEN1* E280A carriers have low levels of cerebrospinal fluid (CSF) A β 1-42¹⁵⁴ and high levels of plasma tau phosphorylated at threonine 217 (p-tau217) and neurofilament light (NfL) up to 20 years before the first symptoms of MCI.¹⁵⁵ *Post mortem* studies also showed a high degree of cerebral small vessel disease pathology in *PSEN1* E280A carriers in Colombia, suggesting covert vascular pathology in *PSEN1* that is not directly associated with brain deposition of amyloid.¹⁵⁶ This identification of AD biomarkers many years before clinical symptoms and the association of biomarkers with clinical phases of AD progression has important implications for early AD diagnosis and the evaluation of disease-modifying AD therapies in clinical trials.

Delayed dementia onset beyond one standard deviation from the average is associated with decreased cortical tau pathology and increased activity of the proteasome system, without any modification of A β -associated pathology.¹⁵⁷ Interestingly, resistance to early-onset FAD conferred by the rare APOE ϵ 3/ ϵ 3 Christchurch R136S mutation was discovered in a *PSEN1* E280A carrier who did not develop MCI until her 70s.¹⁵⁸ Brain imaging and autopsy revealed low levels of tau pathology and neurodegeneration in key cortical areas, despite a high A β plaque burden.¹⁵⁹ This suggests a role of APOE3 Christchurch variant in the clinical presentation, age of onset, and biomarker progression in autosomal-dominant AD. More recently, another case was identified who, despite carrying the E280A mutation, remained cognitively intact until 67 years of age since this individual also carried a heterozygous rare variant of the Reelin gene. This remarkable protection was associated not with the profile or severity of AD pathology but with increased neuronal density in the entorhinal cortex.¹⁶⁰ Finally, Colombian individuals with AD or related dementias carrying additional *PSEN1* pathogenic variants that cause early-onset FAD, as well as multiple rare variants, develop other forms of autosomal-dominant early-onset dementia.¹⁴⁵

Together, these studies underscore the genetic insights that can be gained from studies that include an admixture of genetic alleles from multiple ancestries and reinforce the need to include diverse populations for gene-trait association studies. The discovery of additional genes in diverse populations is critical to filling knowledge gaps in the functional genomic landscape of AD pathogenesis, such as explaining clinical observations and providing clues to potential novel disease-modifying therapies. It is expected that the Recruitment and Retention of Alzheimer's Disease Diversity Genetic Cohorts in the AD sequencing project (READD-ADSP) will hold great potential for finding new variants in LMICs, as well as nine SSA countries involved in the African Dementia Consortium¹⁶¹ and the Multipartner Consortium to expand the genetics of dementia research in Latin America (ReDLat).¹⁴⁶

7.2 | Effects of APOE on AD risk across diverse populations

APOE is the strongest genetic risk factor for LOAD, with three isoforms – ϵ 2, ϵ 3, and ϵ 4 – that differentially influence AD risk.¹⁶² Although the initial studies of cohorts with European ancestry suggested that APOE ϵ 4 homozygotes had a substantially higher AD risk than APOE ϵ 3 homozygotes,¹⁶³ subsequent research has found that the risk of developing AD in the presence of the APOE ϵ 4 allele varies across populations with diverse ancestral backgrounds.^{23,164-166} It has been shown that the strongest risk of AD from the APOE ϵ 4 allele is in East Asians, followed by non-Hispanic Whites, and considerably lower risk in African-ancestry populations such as Nigerian and East Africans and African Americans.^{20,22,164,165,167,168} Interestingly, ancestry analysis showed that the differential risk of AD from APOE ϵ 4 between populations and individuals is due to differences in the local ancestry surrounding the APOE gene rather than differences in global ancestry. Specifically, when the APOE ϵ 4 allele is present on a haplotype

originating from African ancestry, the risk of AD is substantially lower than on a haplotype originating from European ancestry.^{22,169}

Genetic interaction studies to identify and characterize the protective variant in the African local ancestry around *APOE* showed that a protective locus lay approximately two megabases (mB) upstream of the *APOE* locus and reduced the risk effect of *APOE* ϵ 4 homozygotes in African local ancestry by 75%. However, this protective allele is only in 11% of the African ancestry population and very rare in others. Thus, while it contributes to lowering the risk for AD from *APOE*, other factors are important.¹⁶⁵ Single-nucleus RNA sequencing of the frontal cortex of homozygous *APOE* ϵ 4 AD patients revealed that the *APOE* gene was differentially expressed between individuals with African or European local ancestry, with higher levels in European local ancestry frontal cortex, particularly in astrocytes and microglia.¹⁷⁰

These studies and related research highlight the differences in the genetic contributions to AD across various ancestries. Future studies focused on diverse populations are essential as they can provide valuable insights into AD pathology that may not be available when studying single populations. By identifying protective factors for AD, such as *APOE* ϵ 4 in African-Ancestry populations, we can move forward with AD therapeutics efforts that cater to the needs of all populations.

8 | COGNITIVE TESTING AND DIAGNOSIS

8.1 | Delays and underdiagnosis of dementia in LMICs

There is a critical need for early and accurate diagnosis of AD to ensure that eligible individuals receive treatments at the earliest time possible.^{171,172} Furthermore, early dementia diagnosis can provide patients and families the opportunity to begin long-term legal, financial, and care planning. However, 75% of dementia cases worldwide and 90% in LMICs go undiagnosed.¹⁷³ Low availability of biomarkers in LMICs contributes to this discrepancy, as discussed in more detail in section 9.

Barriers to timely diagnosis of dementia, including cultural trends, exist at multiple levels. Patients and family members may have limited awareness of dementia, attribute their symptoms to normal aging, or experience feelings of shame and fear. In addition, clinicians may lack the necessary training, enough time, and cognitive tools to assess dementia patients.¹⁷⁴ Only recently have there been efforts to assess which cognitive tests are best to use in LMICs, and particularly in people with low education, to identify cognitive deficits.¹⁷⁵

At a higher level, health systems with poor infrastructure and too few resources may impede early dementia diagnosis.¹⁷⁶ In HICs, racial and ethnic disparities in the timeliness and breadth of dementia diagnosis have also been reported. For example, one study found that, compared with White Medicare beneficiaries in California, those who identified as Asian, Black, or Hispanic were less likely to receive a timely dementia diagnosis.¹⁷⁷ In LMICs across Latin America, the combination of different barriers and socioeconomic disparities strongly impacts underdiagnosis.^{178,179} Intriguingly, Asian beneficiaries also received

fewer diagnostic evaluation elements.¹⁷⁷ A study in Bangalore showed that among 855 total patients with dementia, the median time from symptom onset to diagnosis (TTD) was 2 years. Among patients with young onset dementia, the median TTD was 3 years. In addition, patients with VaD were diagnosed significantly earlier following symptom onset than patients with AD or frontotemporal dementia (FTD). These findings indicate substantial delays in the diagnosis of dementia in urban India that are likely exacerbated in rural and lower educated regions.¹⁸⁰

8.2 | Cross-cultural adaptation and validation of cognitive testing

Effective neuropsychological tests applicable across varied cultures are important in the assessment of cognitive function. While several effective neuropsychological tools are widely available for use in HICs, this is not the case for most LMICs.¹⁸¹ In India, it has been necessary to develop various neuropsychological batteries for use in older persons, especially within the rural, low-educated elderly population.^{182,183} In Indonesia use of different screening test may be responsible for very high estimates of dementia (up to 33%) and standardization and validation of screening tests is important for accurate estimates.³² To reduce disparities in dementia diagnosis, neuropsychological tools, and resources must be adapted and validated for use in LMICs.

Researchers have begun validating and culturally adapting the Eight-Item Informant Interview to Differentiate Aging and Dementia (AD-8) for use in Ethiopia¹⁸⁴; Alzheimer Disease Centers' Neuropsychological Test Battery in the Uniform Data Set (UDS-III), Tablet-based brain health assessment (BHA), and Pfeffer Functional Activities Questionnaire (PFAQ) for use in Botswana (unpublished data) and a brief version of the Addenbrooke Cognitive Examination (ACE) for use in Brazil.^{185,186} Several cognitive tests have also been cross-culturally adapted and standardized for use in Latin America and Africa.¹⁸⁷⁻¹⁹³ These efforts involve forward- and back-translating tools into target languages, conducting cognitive interviews to assess respondents' interpretations of test questions, collecting feedback from expert panels on test language and cultural appropriateness, and performing statistical analyses to isolate the questions with the highest predictive power for identifying cognitive impairment.^{181,184,186,194} In India, it has been shown that there is also utility in modifying verbal-language/orientation-memory (VLOM) ratios from the ACE battery for early identification and clinical differentiation of AD from other forms of dementia.^{195,196}

To improve dementia detection in under-resourced areas lacking comprehensive neuropsychological testing, validation and cultural adaptation efforts, including the need to cater to multiple languages for cognitive screening tools, are needed in more LMICs.¹⁹⁷ Future efforts should consider how an individual's performance on a cognitive test may be impacted by their culture, acculturation, degree of bilingualism, and both quality and level of education and socioeconomic inequality, which can include unfamiliarity with – and fear of – test-based assessments.¹⁹⁸

9 | BIOMARKERS AND BIOBANKING

9.1 | Biomarkers

Various biomarkers for dementia have been developed that not only provide valuable insight into disease pathology but also pave the way for more efficient risk stratification, diagnosis, and monitoring in the context of therapeutic and preventive interventions. This includes neuroimaging biomarkers of protein deposition, as well as structural and functional alterations (eg, structural and functional MRI, amyloid and tau PET) and fluid biomarkers (ie, CSF and blood-based biomarkers).¹⁷¹ However, MRI and PET are not widely available in most LMICs, and current knowledge on dementia biomarkers is derived mostly from studies of White individuals.

Recently, blood-based biomarkers of AD have been shown to significantly improve the diagnostic accuracy of AD by providing less invasive, more feasible, and more cost-effective approaches compared to PET neuroimaging and CSF analysis.¹⁹⁹ In the United States, evidence from diverse ethnic and racial groups has been lacking, though recent studies have included African American and Hispanic participants. However, compared to studies on non-Hispanic White individuals, the numbers remain small. Systematic review and meta-analysis found that only five studies compared AD fluid biomarkers between African American or Black Africans and White individuals.^{23,200} Meta-analyses of these studies showed that CSF total tau (t-tau) and p-tau181 levels were consistently lower in African Americans than in White individuals with normal cognition and MCI. Such findings underscore the importance of considering ethnic and racial factors that may influence AD biomarker levels.²⁰¹ Regionally oriented frameworks can help to develop more effective and feasible biomarkers in LMICs and other low-resource-setting regions. Improved sampling protocols, for example, through finger prick testing with no need for centrifugation and storage at room temperature, are now emerging, which should facilitate biomarker studies in remote or low-resource settings.²⁰²

9.2 | Biobanks

Brain banks are essential for the study of brain aging and neurodegenerative disorders, particularly AD.²⁰³ While the majority of PLWD reside in LMICs, most brain banks are in HICs. Establishing biobanks in LMICs is difficult due to a lack of institutional funds and external stakeholder support, including poor willingness toward brain donation. However, biobanking in LMICs can provide valuable insights into disease pathology within the context of diverse ancestry/geographical backgrounds.^{204–207}

In Africa, the Ibadan Brain Ageing, Dementia And Neurodegeneration (IBADAN) brain bank and the African Neurobiobank ELSI Project^{205,208} have demonstrated that 19% to 27% of older Africans are willing to donate brains for research and have also drawn attention to the necessity of paying attention to the legal issues related to biobanking in a unique African context.^{206,209,210} The Biobank for Aging Studies (BAS) in Sao Paulo, Brazil²⁰⁴ has shown that 22% of cases

with a Clinical Dementia Rating (CDR) score of zero met the criteria for a neuropathological disease diagnosis, with AD being the most common.²¹ It has also been shown that VaD and cerebral small vessel disease are higher in Brazil than in other clinicopathological studies.²¹¹ Data from the BAS further demonstrated that African ancestry may be highly protective against AD by showing a negative correlation between African ancestry and neuritic plaques.²⁰⁰ Additionally, it has been shown that the APOE ϵ 4 genotype has different effects on AD pathology and clinical features, depending on the patient's genetic lineage. It was shown that APOE ϵ 4 carriers with high African ancestry proportion and worse functional cognitive scores were not affected as they would be in Europeans.¹⁶⁵

These results demonstrate the need for localized study of AD and dementia biomarkers in LMICs, as geography-dependent factors such as ancestry can have significant impacts on the disease. The scarcity of brain banks in LMICs is further illustrated in Latin America and the Caribbean region. Besides the Brazilian BAS, currently only four other functioning biobanks are preserving and collecting brain tissue: Argentina, Colombia, Mexico, and Dominican Republic. More often than not, brain banks in this region are associated with specific research programs and populations, such as the brain bank in Medellin, Colombia. On the other hand, Sri Lanka's NeuroBioBank may provide a model for establishing biobanks in low-resource settings. With funding from the National Institutes of Health (NIH) and National Institute of Neurological Disorders and Stroke (NINDS), this biobank has collected biological samples from 500 healthy controls and roughly 2000 patients with brain diseases. It has also established mobile clinics across Sri Lanka and conducted genetic tests to assess genetic traits and risk factors for rare neurodegenerative diseases.²¹² Notably, researchers found that 285 patients tested negative for known mutations associated with neurodegeneration, indicating novel mutations may remain available for discovery. This presents opportunities for companies in the biomedical industry to collaborate with biobanks, to enable resource sharing and aid in new mutation discovery and gene therapy development.²¹³

10 | DEMENTIA CARE AND POLICY

The 2022 symposium also devoted a session on dementia care and policy to a discussion of challenges and best practices for dementia care in LMICs. The session highlighted that the most effective strategies for dementia policy in LMICs were those that are culturally appropriate and meaningful to the communities that are impacted by neurocognitive illnesses. In particular, lack of institutional funding, stigma toward dementia, and low education on the causes of dementia and AD were highlighted as leading causes of poorer dementia diagnostic and treatment outcomes in SSA and other LMICs.^{174,214,215}

To broaden the scope of dementia research in LMICs and build evidence on dementia care and services, the Strengthening Responses to Dementia in Developing Countries project (STRIDE, www.stride-dementia.org/) was launched in seven LMICs comprising Brazil, Indonesia, India, Jamaica, Kenya, Mexico, and South Africa.²¹⁶

TABLE 1 Recommendations of Nairobi Declaration 2022 on dementia impact.²²⁰

Rethink global approach to dementia by focusing on underserved and underrepresented populations.
Increase investments in LMICs to address challenges and seize opportunities related to various dementia subtypes.
Implement and evaluate population-level risk reduction strategies by engaging policymakers and advocacy organizations.
Consider nutritional and psychosocial factors for promoting brain health, in addition to improving education and cardiovascular health.
Equip health and social care services with necessary resources in LMICs as well as in low-resourced HICs to meet the needs of the aging population.
Support research into more pragmatic, affordable, and effective solutions.
Train highly motivated early career researchers (ECRs) in higher education institutions in both HICs and LMICs.
Promote research in LMICs by establishing a research framework with international collaboration and prevent the brain drain from LMICs.

STRiDE Kenya launched a dementia anti-stigma intervention in rural communities that have a poorer understanding of the condition. The intervention educated communities about dementia symptoms, addressed myths and misconceptions about dementia, showed the impact of discrimination and human rights violations against PLWD, and encouraged social inclusion of people living with dementia. One month after the intervention, STRiDE Kenya reported that participants were less likely to believe that doctors should force treatment on unwilling patients and more likely to believe that greater spending on dementia care is a useful investment. Furthermore, an ongoing study entitled "Integration and evaluation of a community-level dementia screening program in Kenya (DEM-SKY)"²¹⁷ has begun working to normalize dementia in Africa by targeting 2400 older persons aged 60 and older. This project aims to increase timely and accurate dementia detection using evidence-based tools, improve access to quality dementia care, prioritize dementia in policy-level discussions, and increase funding for dementia care and research.

Jamaica and other Caribbean countries face significant impacts associated with dementia care, where the current costs of informal care outweigh costs in the public sector. This means considerable intervention is needed to prepare health and social care systems to care for people with dementia. Underpinned by a theory of change adapted by the STRiDE Jamaica team and over 50 representatives from multiple sectors, researchers prioritized a knowledge exchange approach to creating services to increase formal care and reduce the impact on informal caregivers. With the funding and resource support from the research grant, the local team focused on building greater coordination among private- and public-sector stakeholders to make further progress on state support for medication subsidization, formal care, and support for caregivers. STRiDE Jamaica also provided formal caregivers with trainings via webinars and conferences on how to appropriately manage dementia. Informal caregivers, that is, family and community-based caregivers, were likewise provided with training to develop skills in care activities, such as stimulating activities, as well as daily living tasks, including shopping, preparing food, and transportation.²¹⁸

11 | CONCLUSIONS

Dementia has become an escalating challenge for public health, society, and the economy in LMICs, with multiple factors at play. Such factors

include a high prevalence of risk factors, poor dementia awareness, stigma and misconception surrounding dementia, inequitable access to healthcare resources, and socioeconomic disparities. Therefore, a multifaceted approach is necessary to reduce the impact of dementia in LMICs. Developing national dementia plans and implementing robust policies focused on enhancing dementia care infrastructure and services are crucial. Furthermore, national and international collaborations that lead to sharing knowledge and resources are key factors. By ensuring the inclusion of individuals from LMICs in research studies and pharmacological (and non-pharmacological) clinical trials (Figure 2) and building research capacity in these regions,²¹⁹ we can benefit from more effective preventive and treatment strategies for dementia.

In conclusion, we would like to echo the recommendations of the Nairobi Declaration made at the 2022 Symposium on Dementia and Brain Aging in LMICs to call upon the global community to take the previously mentioned actions to improve dementia prevalence, outcomes, and personal and societal impacts in LMICs²²⁰ (Table 1).

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REFERENCES

- Nichols E, Steinmetz JD, Vollset SE, et al. Estimation of the global prevalence of dementia in 2019 and forecasted prevalence in 2050: an analysis for the Global Burden of Disease Study 2019. *Lancet Public Health*. 2022;7:e105-e125. doi:10.1016/S2468-2667(21)00249-8
- Prince MJ, Acosta D, Castro-Costa E, Jackson J, Shaji KS. Packages of care for dementia in low- and middle-income countries. *PLoS Med*. 2009;6:e1000176. doi:10.1371/journal.pmed.1000176
- Risk factors related to population diversity and disparity determine healthy aging. *Nat Med*. 2023;29:2183-2184. doi:10.1038/s41591-023-02531-0
- Ibáñez A, Legaz A, Ruiz-Adame M. Addressing the gaps between socioeconomic disparities and biological models of dementia. *Brain*. 2023;146:3561-3564. doi:10.1093/brain/awad236
- Wimo A, Gauthier S, Prince M. *World Alzheimer Report 2018: The state of the art of dementia research: New frontiers*. Alzheimer's Disease International; 2018.
- Mattap SM, Mohan D, McGrattan AM, et al. The economic burden of dementia in low- and middle-income countries (LMICs): a

- systematic review. *BMJ Glob Health*. 2022;7:e007409. doi:10.1136/bmjgh-2021-007409
7. World Health Organization. *Global status report on the public health response to dementia*. World Health Organization; 2021.
 8. Ibáñez A, Pina-Escudero SD, Possin KL, et al. Dementia caregiving across Latin America and the Caribbean and brain health diplomacy. *Lancet Healthy Longev*. 2021;2:e222-e231. doi:10.1016/s2666-7568(21)00031-3
 9. Ferri CP, Jacob KS. Dementia in low-income and middle-income countries: different realities mandate tailored solutions. *PLOS Med*. 2017;14:e1002271. doi:10.1371/journal.pmed.1002271
 10. Prince M, Comas-Herrera A, Knapp M, Guerchet M, Karagiannidou M, World Alzheimer report 2016: improving healthcare for people living with dementia: coverage, quality and costs now and in the future 2016. <http://www.alz.co.uk/> (accessed July 24, 2023)
 11. Johnston K, Preston R, Strivens E, Qaloewai S, Larkins S. Understandings of dementia in low and middle income countries and amongst indigenous peoples: a systematic review and qualitative meta-synthesis. *Aging Ment Health*. 2020;24:1183-1195. doi:10.1080/13607863.2019.1606891
 12. World Health Organization. *Global action plan on the public health response to dementia 2017-2025*. World Health Organization; 2017.
 13. James T, Mukadam N, Sommerlad A, et al. Protection against discrimination in national dementia guideline recommendations: a systematic review. *PLOS Med*. 2022;19:e1003860. doi:10.1371/journal.pmed.1003860
 14. United Nations, Department of Economic and Social Affairs, Population Division (2020). *World Population Ageing 2019 (ST/ESA/SER.A/444)*. n.d.
 15. Franzen S, Smith JE, van den Berg E, et al. Diversity in Alzheimer's disease drug trials: the importance of eligibility criteria. *Alzheimers Dement J Alzheimers Assoc*. 2022;18:810-823. doi:10.1002/alz.12433
 16. Llibre-Guerra JJ, Heavener A, Brucki SMD, et al. A call for clinical trial globalization in Alzheimer's disease and related dementia. *Alzheimers Dement*. 2023;19:3210-3221. doi:10.1002/alz.12995
 17. Cummings J, Lee G, Ritter A, Zhong K. Alzheimer's disease drug development pipeline: 2018. *Alzheimers Dement N Y N*. 2018;4:195-214. doi:10.1016/j.trci.2018.03.009
 18. Cummings J, Zhou Y, Lee G, Zhong K, Fonseca J, Cheng F. Alzheimer's disease drug development pipeline: 2023. *Alzheimers Dement N Y N*. 2023;9:e12385. doi:10.1002/trc2.12385
 19. Babulal GM, Quiroz YT, Albeni BC, et al. Perspectives on ethnic and racial disparities in Alzheimer's disease and related dementias: update and areas of immediate need. *Alzheimers Dement J Alzheimers Assoc*. 2019;15:292-312. doi:10.1016/j.jalz.2018.09.009
 20. Kalaria RN, Maestre GE, Arizaga R, et al. Alzheimer's disease and vascular dementia in developing countries: prevalence, management, and risk factors. *Lancet Neurol*. 2008;7:812-826. doi:10.1016/S1474-4422(08)70169-8
 21. Suemoto CK, Ferretti-Rebustini REL, Rodriguez RD, et al. Neuropathological diagnoses and clinical correlates in older adults in Brazil: a cross-sectional study. *PLOS Med*. 2017;14:e1002267. doi:10.1371/journal.pmed.1002267
 22. Rajabli F, Feliciano BE, Celis K, et al. Ancestral origin of ApoE ε4 Alzheimer disease risk in Puerto Rican and African American populations. *PLOS Genet*. 2018;14:e1007791. doi:10.1371/journal.pgen.1007791
 23. Naslavsky MS, Suemoto CK, Brito LA, et al. Global and local ancestry modulate APOE association with Alzheimer's neuropathology and cognitive outcomes in an admixed sample. *Mol Psychiatry*. 2022;27:4800-4808. doi:10.1038/s41380-022-01729-x
 24. Mok VCT, Pendlebury S, Wong A, et al. Tackling challenges in care of Alzheimer's disease and other dementias amid the COVID-19 pandemic, now and in the future. *Alzheimers Dement J Alzheimers Assoc*. 2020;16:1571-1581. doi:10.1002/alz.12143
 25. de Erausquin GA, Snyder H, Brugha TS, et al. Chronic neuropsychiatric sequelae of SARS-CoV-2: protocol and methods from the Alzheimer's Association Global Consortium. *Alzheimers Dement N Y N*. 2022;8:e12348. doi:10.1002/trc2.12348
 26. Wu Y-T, Beiser AS, Breteler MMB, et al. The changing prevalence and incidence of dementia over time—current evidence. *Nat Rev Neurol*. 2017;13:327-339. doi:10.1038/nrneurol.2017.63
 27. 2023 Alzheimer's disease facts and figures. *Alzheimers Dement*. 2023;19:1598-1695. doi:10.1002/alz.13016
 28. Livingston G, Huntley J, Sommerlad A, et al. Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. *The Lancet*. 2020;396:413-446. doi:10.1016/S0140-6736(20)30367-6
 29. Mukadam N, Sommerlad A, Huntley J, Livingston G. Population attributable fractions for risk factors for dementia in low-income and middle-income countries: an analysis using cross-sectional survey data. *Lancet Glob Health*. 2019;7:e596-e603. doi:10.1016/S2214-109X(19)30074-9
 30. Suemoto CK, Mukadam N, Brucki SMD, et al. Risk factors for dementia in Brazil: differences by region and race. *Alzheimers Dement*. 2023;19:1849-1857. doi:10.1002/alz.12820
 31. Stephan BCM, Pakpahan E, Siervo M, et al. Prediction of dementia risk in low-income and middle-income countries (the 10/66 Study): an independent external validation of existing models. *Lancet Glob Health*. 2020;8:e524-e535. doi:10.1016/S2214-109X(20)30062-0
 32. Hogervorst E, Schröder-Butterfill E, Handajani YS, Kreager P, Rahardjo TBW. Dementia and dependency vs. proxy indicators of the active ageing index in Indonesia. *Int J Environ Res Public Health*. 2021;18:8235. doi:10.3390/ijerph18188235
 33. Santamaria-Garcia H, Sainz-Ballesteros A, Hernandez H, et al. Factors associated with healthy aging in Latin American populations. *Nat Med*. 2023;29:2248-2258. doi:10.1038/s41591-023-02495-1
 34. Blanken AE, Nation DA. Does gender influence the relationship between high blood pressure and dementia? Highlighting areas for further investigation. *J Alzheimers Dis*. 2020;78:23-48. doi:10.3233/JAD-200245
 35. Peters R, Booth A, Rockwood K, Peters J, D'Este C, Anstey KJ. Combining modifiable risk factors and risk of dementia: a systematic review and meta-analysis. *BMJ Open*. 2019;9:e022846. doi:10.1136/bmjopen-2018-022846
 36. Siervo M, Harrison SL, Jagger C, Robinson L, Stephan BCM. Metabolic syndrome and longitudinal changes in cognitive function: a systematic review and meta-analysis. *J Alzheimers Dis JAD*. 2014;41:151-161. doi:10.3233/JAD-132279
 37. Wimo A, Guerchet M, Ali G-C, et al. The worldwide costs of dementia 2015 and comparisons with 2010. *Alzheimers Dement J Alzheimers Assoc*. 2017;13:1-7. doi:10.1016/j.jalz.2016.07.150
 38. Crivelli L. LatAm-FINGERS (Latin America): world-Wide FINGERS network: the first global network of multidomain dementia prevention trials. *Alzheimers Dement*. 2020;16:e046953. doi:10.1002/alz.046953
 39. World Health Organization. *Risk reduction of cognitive decline and dementia: WHO guidelines*. World Health Organization; 2019.
 40. Pistollato F, Iglesias RC, Ruiz R, et al. Nutritional patterns associated with the maintenance of neurocognitive functions and the risk of dementia and Alzheimer's disease: a focus on human studies. *Pharmacol Res*. 2018;131:32-43. doi:10.1016/j.phrs.2018.03.012
 41. Wijesinghe P, Wijeweera G, De Silva KR. Healthy Brain Ageing and Longevity; the Harmony of Natural Products, APOE Polymorphism, and Melatonin. In: Jagota A, ed. *Sleep Clocks Aging Longev*. Springer International Publishing; 2023:143-164. doi:10.1007/978-3-031-22468-3_7
 42. Mahinrad S, Sorond F, Gorelick PB. The role of vascular risk factors in cognitive impairment and dementia and prospects for prevention. *Clin Geriatr Med*. 2023;39:123-134. doi:10.1016/j.cger.2022.07.007

43. Kalaria R, Akinyemi R, Paddick S, Ihara M. Current perspectives on prevention of vascular cognitive impairment and promotion of vascular brain health. *Expert Rev Neurother*. 2023. (in press).
44. Ferreira NV, Lotufo PA, Marchioni DML, et al. Association between adherence to the MIND diet and cognitive performance is affected by income: the ELISA-Brasil Study. *Alzheimer Dis Assoc Disord*. 2022;36:133-139. doi:10.1097/WAD.0000000000000491
45. Zhuo M, Chen Z, Zhong M-L, et al. The global disease burden attributable to a diet low in fibre in 204 countries and territories from 1990 to 2019. *Public Health Nutr*. 2022;26:1-12. doi:10.1017/S1368980022001987
46. Pilleron S, Desport J-C, Jésus P, et al. Diet, alcohol consumption and cognitive disorders in Central Africa: a Study from the EPIDEMCA program. *J Nutr Health Aging*. 2015;19:657-667. doi:10.1007/s12603-015-0487-y
47. Gomes Gonçalves N, Vidal Ferreira N, Khandpur N, et al. Association between consumption of ultraprocessed foods and cognitive decline. *JAMA Neurol*. 2023;80:142. doi:10.1001/jamaneurol.2022.4397
48. Sala-Vila A, Satizabal CL, Tintle N, et al. Red blood cell DHA is inversely associated with risk of incident Alzheimer's disease and all-cause dementia: framingham offspring study. *Nutrients*. 2022;14:2408. doi:10.3390/nu14122408
49. Satizabal CL, Himali JJ, Beiser AS, et al. Association of red blood cell omega-3 fatty acids with MRI markers and cognitive function in midlife: the Framingham heart study. *Neurology*. 2022;99:e2572-e2582. doi:10.1212/WNL.00000000000021296
50. de Bruyn J, Wesana J, Bunting SW, Thilsted SH, Cohen PJ. Fish Acquisition and consumption in the African great lakes region through a food environment lens: a scoping review. *Nutrients*. 2021;13:2408. doi:10.3390/nu13072408
51. Tan ZS, Spartano NL, Beiser AS, et al. Physical activity, brain volume, and dementia risk: the Framingham study. *J Gerontol A Biol Sci Med Sci*. 2017;72:789-795. doi:10.1093/gerona/glw130
52. Arifin EN, Braun KL, Hogervorst E. Three pillars of active ageing in Indonesia. *Asian Popul Stud*. 2012;8:207-230. doi:10.1080/17441730.2012.680334
53. O'Donovan G, Lee I-M, Hamer M, et al. The burden of mild cognitive impairment attributable to physical inactivity in Colombia. *Eur Rev Aging Phys Act*. 2022;19:28. doi:10.1186/s11556-022-00307-y
54. Walker KA, Power MC, Gottesman RF. Defining the relationship between hypertension, cognitive decline, and dementia: a review. *Curr Hypertens Rep*. 2017;19:24. doi:10.1007/s11906-017-0724-3
55. SPRINT MIND Investigators for the SPRINT Research Group, Williamson JD, Pajewski NM, Auchus AP, et al, SPRINT MIND Investigators for the SPRINT Research Group. Effect of intensive vs standard blood pressure control on probable dementia: a randomized clinical trial. *JAMA*. 2019;321:553-561. doi:10.1001/jama.2018.21442
56. Peters R, Xu Y, Fitzgerald O, et al. Blood pressure lowering and prevention of dementia: an individual patient data meta-analysis. *Eur Heart J*. 2022;43:4980-4990. doi:10.1093/eurheartj/ehac584
57. NCD Risk Factor Collaboration. Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19.2 million participants. *The Lancet*. 2016;387:1377-1396. doi:10.1016/S0140-6736(16)30054-X
58. Schutte AE, Srinivasapura Venkateshmurthy N, Mohan S, Prabhakaran D. Hypertension in low- and middle-income countries. *Circ Res*. 2021;128:808-826. doi:10.1161/CIRCRESAHA.120.318729
59. Geldsetzer P, Manne-Goehler J, Marcus M-E, et al. The state of hypertension care in 44 low-income and middle-income countries: a cross-sectional study of nationally representative individual-level data from 1.1 million adults. *The Lancet*. 2019;394:652-662. doi:10.1016/S0140-6736(19)30955-9
60. Owolabi M, Olowoyo P, Mocumbi A, et al. African control of hypertension through innovative epidemiology and a vibrant ecosystem (ACHIEVE): novel strategies for accelerating hypertension control in Africa. *J Hum Hypertens*. 2023. doi:10.1038/s41371-023-00828-8
61. Mahinrad S, Sorond FA, Gorelick PB. Hypertension and cognitive dysfunction: a review of mechanisms, life-course observational studies and clinical trial results. *Rev Cardiovasc Med*. 2021;22:1429-1449. doi:10.31083/j.rcm2204148
62. Salinas J, O'Donnell A, Kojis DJ, et al. Association of social support with brain volume and cognition. *JAMA Netw Open*. 2021;4:e2121122. doi:10.1001/jamanetworkopen.2021.21122
63. Sommerlad A, Kivimäki M, Larson EB, et al. Social participation and risk of developing dementia. *Nat Aging*. 2023;3:532-545. doi:10.1038/s43587-023-00387-0
64. Smith L, Shin JI, López Sánchez GF, et al. Social participation and mild cognitive impairment in low- and middle-income countries. *Prev Med*. 2022;164:107230. doi:10.1016/j.ypmed.2022.107230
65. Bowen M, Edgar DF, Hancock B, et al. The prevalence of visual impairment in people with dementia (the PROVIDe study): a cross-sectional study of people aged 60-89 years with dementia and qualitative exploration of individual, carer and professional perspectives. *NIHR Journals Library*. 2016.
66. Dawes P, Wolski L, Himmelsbach I, Regan J, Leroi I. Interventions for hearing and vision impairment to improve outcomes for people with dementia: a scoping review. *Int Psychogeriatr*. 2019;31:203-221. doi:10.1017/S1041610218000728
67. Maharani A, Dawes P, Nazroo J, Tampubolon G, Pendleton N. SENSE-Cog WP1 group. Longitudinal relationship between hearing aid use and cognitive function in older Americans. *J Am Geriatr Soc*. 2018;66:1130-1136. doi:10.1111/jgs.15363
68. Lin FR, Pike JR, Albert MS, et al. Hearing intervention versus health education control to reduce cognitive decline in older adults with hearing loss in the USA (ACHIEVE): a multicentre, randomised controlled trial. *The Lancet*. 2023;402:786-797. doi:10.1016/S0140-6736(23)01406-X
69. Leroi I, Simkin Z, Hooper E, et al. Impact of an intervention to support hearing and vision in dementia: the SENSE-Cog Field Trial. *Int J Geriatr Psychiatry*. 2020;35:348-357. doi:10.1002/gps.5231
70. Sheikh S, Tofique S, Zehra N, et al. SENSE-Cog Asia: a feasibility study of a hearing intervention to improve outcomes in people with dementia. *Front Neurol*. 2021;12:654143.
71. Van Der Flier WM, Skoog I, Schneider JA, et al. Vascular cognitive impairment. *Nat Rev Dis Primer*. 2018;4:18003. doi:10.1038/nrdp.2018.3
72. Wolters FJ, Ikram MA. Epidemiology of vascular dementia. *Arterioscler Thromb Vasc Biol*. 2019;39:1542-1549. doi:10.1161/ATVBAHA.119.311908
73. Paddick S-M, Longdon AR, Kisoli A, et al. Dementia prevalence estimates in sub-Saharan Africa: comparison of two diagnostic criteria. *Glob Health Action*. 2013;6:19646. doi:10.3402/gha.v6i0.19646
74. Paddick S-M, Longdon A, Kisoli A, et al. The prevalence of dementia subtypes in rural Tanzania. *Am J Geriatr Psychiatry Off J Am Assoc Geriatr Psychiatry*. 2014;22:1613-1622. doi:10.1016/j.jagp.2014.02.004
75. Lam BYK, Cai Y, Akinyemi R, et al. The global burden of cerebral small vessel disease in low- and middle-income countries: a systematic review and meta-analysis. *Int J Stroke Off J Int Stroke Soc*. 2023;18:15-27. doi:10.1177/17474930221137019
76. Akinyemi RO, Owolabi MO, Ihara M, et al. Stroke, cerebrovascular diseases and vascular cognitive impairment in Africa. *Brain Res Bull*. 2019;145:97-108. doi:10.1016/j.brainresbull.2018.05.018
77. Arshad F, Mm S, Paplikar A, Rajendran S, Kalkonde Y, Alladi S. Vascular cognitive impairment in India: challenges and opportunities for prevention and treatment. *Cereb Circ—Cogn Behav*. 2022;3:100034. doi:10.1016/j.cccb.2021.100034

78. Alladi S, Kaul S, Meena AK, Somayajula S, Umadevi M, Reddy JM. Pattern of vascular dementia in India: study of clinical features, imaging, and vascular mechanisms from a hospital dementia registry. *J Stroke Cerebrovasc Dis Off J Natl Stroke Assoc*. 2006;15:49-56. doi:10.1016/j.jstrokecerebrovasdis.2004.09.004
79. Lam BYK, Yiu B, Ampil E, et al. High burden of cerebral white matter lesion in 9 Asian cities. *Sci Rep*. 2021;11:11587. doi:10.1038/s41598-021-90746-x
80. Iadecola C, Duering M, Hachinski V, et al. Vascular cognitive impairment and dementia: JACC scientific expert panel. *J Am Coll Cardiol*. 2019;73:3326-3344. doi:10.1016/j.jacc.2019.04.034
81. World Health Organization. *Global action plan for the prevention and control of noncommunicable diseases 2013-2020*. World Health Organization; 2013.
82. Khatib R, McKee M, Shannon H, et al. Availability and affordability of cardiovascular disease medicines and their effect on use in high-income, middle-income, and low-income countries: an analysis of the PURE study data. *The Lancet*. 2016;387:61-69. doi:10.1016/S0140-6736(15)00469-9
83. Matuja SS, Ngimbwa J, Andrew L, et al. Stroke characteristics and outcomes in urban Tanzania: data from the Prospective Lake Zone Stroke Registry. *Int J Stroke Off J Int Stroke Soc*. 2023;17:17474930231219584. doi:10.1177/17474930231219584
84. Skrobot OA, Black SE, Chen C, et al. Progress toward standardized diagnosis of vascular cognitive impairment: guidelines from the vascular impairment of cognition classification consensus study. *Alzheimers Dement J Alzheimers Assoc*. 2018;14:280-292. doi:10.1016/j.jalz.2017.09.007
85. Feigin VL, Brainin M, Norrving B, et al. World Stroke Organization (WSO): global Stroke Fact Sheet 2022. *Int J Stroke Off J Int Stroke Soc*. 2022;17:18-29. doi:10.1177/17474930211065917
86. Owolabi M, Akarolo-Anthony S, Akinyemi R, et al. The burden of stroke in Africa: a glance at the present and a glimpse into the future: review article. *Cardiovasc J Afr*. 2015;26:S27-S38. doi:10.5830/CVJA-2015-038
87. Akinyemi RO, Ovbiagele B, Adeniji OA, et al. Stroke in Africa: profile, progress, prospects and priorities. *Nat Rev Neurol*. 2021;17:634-656. doi:10.1038/s41582-021-00542-4
88. Kaul S, Alladi S, Jabeen S, et al. Intracranial atherosclerosis is the most common stroke subtype: ten-year data from hyderabad stroke registry (India). *Ann Indian Acad Neurol*. 2018;21:209. doi:10.4103/aian.AIAN_86_18
89. Pandian JD, Kalkonde Y, Sebastian IA, Felix C, Urimubenshi G, Bosch J. Stroke systems of care in low-income and middle-income countries: challenges and opportunities. *Lancet Lond Engl*. 2020;396:1443-1451. doi:10.1016/S0140-6736(20)31374-X
90. Jacob MA, Ekker MS, Allach Y, et al. Global differences in risk factors, etiology, and outcome of ischemic stroke in young adults—a worldwide meta-analysis: the GOAL Initiative. *Neurology*. 2022;98:e573-e588. doi:10.1212/WNL.00000000000013195
91. Mavrodaris A, Powell J, Thorogood M. Prevalences of dementia and cognitive impairment among older people in sub-Saharan Africa: a systematic review. *Bull World Health Organ*. 2013;91:773-783. doi:10.2471/BLT.13.118422
92. Akinyemi RO, Allan L, Owolabi MO, et al. Profile and determinants of vascular cognitive impairment in African stroke survivors: the CogFAST Nigeria Study. *J Neurol Sci*. 2014;346:241-249. doi:10.1016/j.jns.2014.08.042
93. Fang C, Lv L, Mao S, Dong H, Liu B. Cognition deficits in Parkinson's disease: mechanisms and treatment. *Park Dis*. 2020;2020:2076942. doi:10.1155/2020/2076942
94. Baiano C, Barone P, Trojano L, Santangelo G. Prevalence and clinical aspects of mild cognitive impairment in Parkinson's disease: a meta-analysis. *Mov Disord Off J Mov Disord Soc*. 2020;35:45-54. doi:10.1002/mds.27902
95. Aarsland D, Kurz MW. The epidemiology of dementia associated with Parkinson's disease. *Brain Pathol*. 2010;20:633-639. doi:10.1111/j.1750-3639.2009.00369.x
96. Schiess N, Cataldi R, Okun MS, et al. Six action steps to address global disparities in Parkinson disease: a world health organization priority. *JAMA Neurol*. 2022;79:929-936. doi:10.1001/jamaneurol.2022.1783
97. Akinyemi RO, Okubadejo NN, Akinyemi JO, Owolabi MO, Owolabi LF, Ogunniyi A. Cognitive dysfunction in Nigerians with Parkinson's disease. *Mov Disord*. 2008;23:1378-1383. doi:10.1002/mds.22087
98. Ojo OO, Okubadejo NU, Ojini FI, Danesi MA. Frequency of cognitive impairment and depression in Parkinson's disease: a preliminary case-control study. *Niger Med J J Niger Med Assoc*. 2012;53:65-70. doi:10.4103/0300-1652.103544
99. Ojo OO, Wahab KW, Bello AH, et al. A cross-sectional comprehensive assessment of the profile and burden of non-motor symptoms in relation to motor phenotype in the Nigeria Parkinson disease registry cohort. *Mov Disord Clin Pract*. 2021;8:1206-1215. doi:10.1002/mdc3.13346
100. Litvan I, Aarsland D, Adler CH, et al. MDS Task Force on mild cognitive impairment in Parkinson's disease: critical review of PD-MCI. *Mov Disord Off J Mov Disord Soc*. 2011;26:1814-1824. doi:10.1002/mds.23823
101. Deeks SG, Lewin SR, Havlir DV. The end of AIDS: HIV infection as a chronic disease. *Lancet Lond Engl*. 2013;382:1525-1533. doi:10.1016/S0140-6736(13)61809-7
102. Alford K, Daley S, Banerjee S, Vera JH. Quality of life in people living with HIV-associated neurocognitive disorder: a scoping review study. *PLOS ONE*. 2021;16:e0251944. doi:10.1371/journal.pone.0251944
103. Antinori A, Arendt G, Becker JT, et al. Updated research nosology for HIV-associated neurocognitive disorders. *Neurology*. 2007;69:1789-1799. doi:10.1212/01.WNL.0000287431.88658.8b
104. Wang Y, Liu M, Lu Q, et al. Global prevalence and burden of HIV-associated neurocognitive disorder: a meta-analysis. *Neurology*. 2020;95:e2610-e2621. doi:10.1212/WNL.00000000000010752
105. Nweke MC, Okemuo AJ, Uduonu EM, Ugwu PI, Nwachukwu C, Mshunqane N. Meta-analysis of factors affecting prevalence estimates of HIV-associated neurocognitive disorder in sub-Saharan Africa. *South Afr J Sci*. 2021;117:1-10. doi:10.17159/sajs.2021/8575
106. Mwangala PN, Mabrouk A, Wagner R, Newton CRJC, Abubakar AA. Mental health and well-being of older adults living with HIV in sub-Saharan Africa: a systematic review. *BMJ Open*. 2021;11:e052810. doi:10.1136/bmjopen-2021-052810
107. Dreyer AJ, Nightingale S, Heaps-Woodruff JM, et al. Rates of cognitive impairment in a South African cohort of people with HIV: variation by definitional criteria and lack of association with neuroimaging biomarkers. *J Neurovirol*. 2021;27:579-594. doi:10.1007/s13365-021-00993-x
108. Robbins RN, Joska JA, Thomas KGF, et al. Exploring the utility of the Montreal Cognitive Assessment to detect HIV-associated neurocognitive disorder: the challenge and need for culturally valid screening tests in South Africa. *Clin Neuropsychol*. 2013;27:437-454. doi:10.1080/13854046.2012.759627
109. Dreyer AJ, Nightingale S, Andersen LS, et al. Cognitive performance in a South African cohort of people with HIV and comorbid major depressive disorder. *J Neurovirol*. 2022;28:537-551. doi:10.1007/s13365-022-01093-0
110. Nightingale S, Cinque P, Joska JA, Price RW, Underwood J, International HIV-Cognition Working Group. A new approach to cognitive impairment in people with HIV. *Lancet HIV*. 2022;9:e815-e817. doi:10.1016/S2352-3018(22)00267-3
111. Nightingale S, Ances B, Cinque P, et al. Cognitive impairment in people living with HIV: consensus recommendations for a new approach. *Nat Rev Neurol*. 2023;19:424-433. doi:10.1038/s41582-023-00813-2

112. Gisslén M, Price RW, Nilsson S. The definition of HIV-associated neurocognitive disorders: are we overestimating the real prevalence? *BMC Infect Dis*. 2011;11:356. doi:10.1186/1471-2334-11-356
113. Nightingale S, Winston A, Letendre S, et al. Controversies in HIV-associated neurocognitive disorders. *Lancet Neurol*. 2014;13:1139-1151. doi:10.1016/S1474-4422(14)70137-1
114. Klimova B, Kuca K. Speech and language impairments in dementia. *J Appl Biomed*. 2016;14:97-103. doi:10.1016/j.jab.2016.02.002
115. Salehi M, Reisi M, Ghasisin L. Lexical retrieval or semantic knowledge which one causes naming errors in patients with mild and moderate Alzheimer's disease. *Dement Geriatr Cogn Disord Extra*. 2017;7:419-429. doi:10.1159/000484137
116. Beveridge MEL, Bak TH. The languages of aphasia research: bias and diversity. *Aphasiology*. 2011;25:1451-1468. doi:10.1080/02687038.2011.624165
117. Georgiou EE, Prapiadou S, Thomopoulos V, et al. Naming ability assessment in neurocognitive disorders: a clinician's perspective. *BMC Psychiatry*. 2022;22:837. doi:10.1186/s12888-022-04486-x
118. Bose A, Dash NS, Ahmed S, et al. Connected speech characteristics of Bengali speakers with Alzheimer's disease: evidence for language-specific diagnostic markers. *Front Aging Neurosci*. 2021;13:707628. doi:10.3389/fnagi.2021.707628
119. Blanken G, Dittmann J, Haas J-C, Wallesch C-W. Spontaneous speech in senile dementia and aphasia: implications for a neurolinguistic model of language production. *Cognition*. 1987;27:247-274. doi:10.1016/S0010-0277(87)80011-2
120. Kavé G, Levy Y. Morphology in picture descriptions provided by persons with Alzheimer's disease. *J Speech Lang Hear Res JSLHR*. 2003;46:341-352.
121. Ahmed S, Haigh A-MF, De Jager CA, Garrard P. Connected speech as a marker of disease progression in autopsy-proven Alzheimer's disease. *Brain*. 2013;136:3727-3737. doi:10.1093/brain/awt269
122. Siriboonpipattana W, Nickels L, Bastiaanse R. An investigation of time reference in production and comprehension in Thai speakers with agrammatic aphasia. *Aphasiology*. 2021;35:1168-1189. doi:10.1080/02687038.2020.1781777
123. Pearce JMS. A note on aphasia in bilingual patients: pitres' and Ribot's laws. *Eur Neurol*. 2005;54:127-131. doi:10.1159/000089083
124. Ribot T. *Diseases of memory: an essay in the positive psychology*. D. Appleton and Company; 1882.
125. Paradis M. Bilingualism and neuropsychiatric disorders. *J Neurolinguistics*. 2008;21:199-230. doi:10.1016/j.jneuroling.2007.09.002
126. Ellajosyula R, Narayanan J, Patterson K. Striking loss of second language in bilingual patients with semantic dementia. *J Neurol*. 2020;267:551-560. doi:10.1007/s00415-019-09616-2
127. Abuom TO, Oblor LK, Bastiaanse R. Using Swahili and English to test explanations of agrammatism. *Aphasiology*. 2011;25:559-575. doi:10.1080/02687038.2010.538417
128. Bialystok E, Abutalebi J, Bak TH, Burke DM, Kroll JF. Aging in two languages: implications for public health. *Ageing Res Rev*. 2016;27:56-60. doi:10.1016/j.arr.2016.03.003
129. Ardila A, Rosselli M. Cognitive world: neuropsychology of individual differences. *Appl Neuropsychol Adult*. 2018;25:29-37. doi:10.1080/23279095.2016.1232264
130. Bak TH, Nissan JJ, Allerhand MM, Deary IJ. Does bilingualism influence cognitive aging? *Ann Neurol*. 2014;75:959-963. doi:10.1002/ana.24158
131. Rosselli M, Loewenstein DA, Curiel RE, et al. Effects of bilingualism on verbal and nonverbal memory measures in mild cognitive impairment. *J Int Neuropsychol Soc JINS*. 2019;25:15-28. doi:10.1017/S135561771800070X
132. Costa A, Hernández M, Costa-Faidella J, Sebastián-Gallés N. On the bilingual advantage in conflict processing: now you see it, now you don't. *Cognition*. 2009;113:135-149. doi:10.1016/j.cognition.2009.08.001
133. Kroll JF, Bobb SC, Hoshino N. Two languages in mind: bilingualism as a tool to investigate language, cognition, and the brain. *Curr Dir Psychol Sci*. 2014;23:159-163. doi:10.1177/0963721414528511
134. Perani D, Abutalebi J. Bilingualism, dementia, cognitive and neural reserve. *Curr Opin Neurol*. 2015;28:618-625. doi:10.1097/WCO.0000000000000267
135. Abutalebi J, Della Rosa PA, Green DW, et al. Bilingualism tunes the anterior cingulate cortex for conflict monitoring. *CEREB Cortex N Y N* 1991. 2012;22:2076-2086. doi:10.1093/cercor/bhr287
136. Abutalebi J, Canini M, Della Rosa PA, Sheung LP, Green DW, Weekes BS. Bilingualism protects anterior temporal lobe integrity in aging. *Neurobiol Aging*. 2014;35:2126-2133. doi:10.1016/j.neurobiolaging.2014.03.010
137. Klein D, Mok K, Chen J-K, Watkins KE. Age of language learning shapes brain structure: a cortical thickness study of bilingual and monolingual individuals. *Brain Lang*. 2014;131:20-24. doi:10.1016/j.bandl.2013.05.014
138. Paap KR, Greenberg ZI. There is no coherent evidence for a bilingual advantage in executive processing. *Cognit Psychol*. 2013;66:232-258. doi:10.1016/j.cogpsych.2012.12.002
139. Hilchey MD, Klein RM. Are there bilingual advantages on nonlinguistic interference tasks? Implications for the plasticity of executive control processes. *Psychon Bull Rev*. 2011;18:625-658. doi:10.3758/s13423-011-0116-7
140. Duñabeitia JA, Hernández JA, Antón E, Macizo P, Estévez A, Fuentes LJ, et al. The inhibitory advantage in bilingual children revisited: myth or reality? *Exp Psychol*. 2014;61:234-251. doi:10.1027/1618-3169/a000243
141. Scarce-Levie K, Sanchez PE, Lewcock JW. Leveraging preclinical models for the development of Alzheimer disease therapeutics. *Nat Rev Drug Discov*. 2020;19:447-462. doi:10.1038/s41573-020-0065-9
142. Thornton P, Sevalle J, Deery MJ, et al. TREM2 shedding by cleavage at the H157-S158 bond is accelerated for the Alzheimer's disease-associated H157Y variant. *EMBO Mol Med*. 2017;9:1366-1378. doi:10.15252/emmm.201707673
143. Vilalta A, Zhou Y, Sevalle J, et al. Wild-type sTREM2 blocks A β aggregation and neurotoxicity, but the Alzheimer's R47H mutant increases A β aggregation. *J Biol Chem*. 2021;296:100631. doi:10.1016/j.jbc.2021.100631
144. Kunkle BW, Grenier-Boley B, Sims R, et al. Genetic meta-analysis of diagnosed Alzheimer's disease identifies new risk loci and implicates A β , tau, immunity and lipid processing. *Nat Genet*. 2019;51:414-430. doi:10.1038/s41588-019-0358-2
145. Acosta-Urbe J, Aguilón D, Cochran JN, et al. A neurodegenerative disease landscape of rare mutations in Colombia due to founder effects. *Genome Med*. 2022;14:27. doi:10.1186/s13073-022-01035-9
146. Ibanez A, Yokoyama JS, Possin KL, et al. The multi-partner consortium to expand dementia research in Latin America (ReDLat): driving multicentric research and implementation science. *Front Neurol*. 2021;12:631722. doi:10.3389/fneur.2021.631722
147. Lopera F. Clinical features of early-onset Alzheimer disease in a large kindred with an E280A presenilin-1 mutation. *JAMA J Am Med Assoc*. 1997;277:793-799. doi:10.1001/jama.277.10.793
148. Lalli MA, Cox HC, Arcila ML, et al. Origin of the PSEN1 E280A mutation causing early-onset Alzheimer's disease. *Alzheimers Dement J Alzheimers Assoc*. 2014;10:S277-S283. doi:10.1016/j.jalz.2013.09.005. e10.
149. Sepulveda-Falla D, Glatzel M, Lopera F. Phenotypic profile of early-onset familial Alzheimer's disease caused by presenilin-1 E280A mutation. *J Alzheimers Dis JAD*. 2012;32:1-12. doi:10.3233/JAD-2012-120907
150. Aguirre-Acevedo DC, Lopera F, Henao E, et al. Cognitive decline in a colombian kindred with autosomal dominant Alzheimer disease: a

- retrospective cohort study. *JAMA Neurol.* 2016;73:431. doi:10.1001/jamaneurol.2015.4851
151. Acosta-Baena N, Sepulveda-Falla D, Lopera-Gómez CM, et al. Pre-dementia clinical stages in presenilin 1 E280A familial early-onset Alzheimer's disease: a retrospective cohort study. *Lancet Neurol.* 2011;10:213-220. doi:10.1016/S1474-4422(10)70323-9
 152. Fleisher AS, Chen K, Quiroz YT, et al. Florbetapir PET analysis of amyloid- β deposition in the presenilin 1 E280A autosomal dominant Alzheimer's disease kindred: a cross-sectional study. *Lancet Neurol.* 2012;11:1057-1065. doi:10.1016/S1474-4422(12)70227-2
 153. Quiroz YT, Sperling RA, Norton DJ, et al. Association between amyloid and tau accumulation in young adults with autosomal dominant Alzheimer disease. *JAMA Neurol.* 2018;75:548-556. doi:10.1001/jamaneurol.2017.4907
 154. Fleisher AS, Chen K, Quiroz YT, et al. Associations between biomarkers and age in the presenilin 1 E280A autosomal dominant Alzheimer Disease kindred: a cross-sectional study. *JAMA Neurol.* 2015;72:316. doi:10.1001/jamaneurol.2014.3314
 155. Quiroz YT, Zetterberg H, Reiman EM, et al. Plasma neurofilament light chain in the presenilin 1 E280A autosomal dominant Alzheimer's disease kindred: a cross-sectional and longitudinal cohort study. *Lancet Neurol.* 2020;19:513-521. doi:10.1016/S1474-4422(20)30137-X
 156. Littau JL, Velilla L, Hase Y, et al. Evidence of beta amyloid independent small vessel disease in familial Alzheimer's disease. *Brain Pathol.* 2022;32:e13097. doi:10.1111/bpa.13097
 157. Sepulveda-Falla D, Chavez-Gutierrez L, Portelius E, et al. A multifactorial model of pathology for age of onset heterogeneity in familial Alzheimer's disease. *Acta Neuropathol (Berl).* 2021;141:217-233. doi:10.1007/s00401-020-02249-0
 158. Arboleda-Velasquez JF, Lopera F, O'Hare M, et al. Resistance to autosomal dominant Alzheimer's disease in an APOE3 Christchurch homozygote: a case report. *Nat Med.* 2019;25:1680-1683. doi:10.1038/s41591-019-0611-3
 159. Sepulveda-Falla D, Sanchez JS, Almeida MC, et al. Distinct tau neuropathology and cellular profiles of an APOE3 Christchurch homozygote protected against autosomal dominant Alzheimer's dementia. *Acta Neuropathol (Berl).* 2022;144:589-601. doi:10.1007/s00401-022-02467-8
 160. Lopera F, Marino C, Chandrahas AS, et al. Resilience to autosomal dominant Alzheimer's disease in a Reelin-COLBOS heterozygous man. *Nat Med.* 2023;29:1243-1252. doi:10.1038/s41591-023-02318-3
 161. Akinyemi RO, Owolabi MO, Okubadejo N, Ogunniyi A, Kalaria RN. African dementia consortium. the African dementia consortium. *Lancet Neurol.* 2023;22:28-29. doi:10.1016/S1474-4422(22)00475-6
 162. Corder EH, Saunders AM, Strittmatter WJ, et al. Gene dose of apolipoprotein E type 4 allele and the risk of Alzheimer's disease in late onset families. *Science.* 1993;261:921-923. doi:10.1126/science.8346443
 163. Corder EH, Saunders AM, Risch NJ, et al. Protective effect of apolipoprotein E type 2 allele for late onset Alzheimer disease. *Nat Genet.* 1994;7:180-184. doi:10.1038/ng0694-180
 164. Farrer LA, Cupples LA, Haines JL, et al. Effects of age, sex, and ethnicity on the association between apolipoprotein E genotype and Alzheimer disease. A meta-analysis. APOE and Alzheimer disease meta analysis consortium. *JAMA.* 1997;278:1349-1356.
 165. Rajabli F, Beecham GW, Hendrie HC, et al. A locus at 19q13.31 significantly reduces the ApoE ϵ 4 risk for Alzheimer's disease in African Ancestry. *PLOS Genet.* 2022;18:e1009977. doi:10.1371/journal.pgen.1009977
 166. Naslavsky MS, Scliar MO, Yamamoto GL, et al. Whole-genome sequencing of 1,171 elderly admixed individuals from Brazil. *Nat Commun.* 2022;13:1004. doi:10.1038/s41467-022-28648-3
 167. Chen C-H, Mizuno T, Elston R, et al. A comparative study to screen dementia and APOE genotypes in an ageing East African population. *Neurobiol Aging.* 2010;31:732-740. doi:10.1016/j.neurobiolaging.2008.06.014
 168. Wijesinghe P, Shankar SK, Yasha TC, et al. Vascular contributions in Alzheimer's disease-related neuropathological changes: first autopsy evidence from a South Asian aging population. *J Alzheimers Dis.* 2016;54:1607-1618. doi:10.3233/JAD-160425
 169. Blue EE, Horimoto ARVR, Mukherjee S, Wijsman EM, Thornton TA. Local ancestry at APOE modifies Alzheimer's disease risk in Caribbean Hispanics. *Alzheimers Dement J Alzheimers Assoc.* 2019;15:1524-1532. doi:10.1016/j.jalz.2019.07.016
 170. Griswold AJ, Celis K, Bussies PL, et al. Increased APOE ϵ 4 expression is associated with the difference in Alzheimer's disease risk from diverse ancestral backgrounds. *Alzheimers Dement.* 2021;17:1179-1188. doi:10.1002/alz.12287
 171. Jack CR, Bennett DA, Blennow K, et al. NIA-AA Research Framework: toward a biological definition of Alzheimer's disease. *Alzheimers Dement J Alzheimers Assoc.* 2018;14:535-562. doi:10.1016/j.jalz.2018.02.018
 172. Kallmyer B, Daven M, Thornhill L, Clifford K, Conant R, Carrillo M. Editorial: impact of Aduhelm approval on care and policy. *J Prev Alzheimers Dis.* 2021;8:396-397. doi:10.14283/jpad.2021.42
 173. Gautier S, Webster C, Servaes S, Morais JA, Rosa-Neto P. *World Alzheimer Report 2022: Life after diagnosis: Navigating treatment, care and support.* Alzheimer's Disease International; 2022.
 174. Alzheimer's Disease International. *World Alzheimer Report 2019: Attitudes to dementia.* Alzheimer's Disease International; 2019.
 175. Bellaj T, Ben Jemaa S, Khelifa M, Ben Djebara M, Gouider R, Le Gall D. The development of the dementia screening battery-100: instrument presentation, reliability, and construct validity. *Dement Geriatr Cogn Disord Extra.* 2017;7:215-229. doi:10.1159/000477437
 176. Parker M, Barlow S, Hoe J, Aitken L. Persistent barriers and facilitators to seeking help for a dementia diagnosis: a systematic review of 30 years of the perspectives of carers and people with dementia. *Int Psychogeriatr.* 2020;1-24. doi:10.1017/S1041610219002229
 177. Tsoy E, Kiehofer RE, Guterman EL, et al. Assessment of Racial/ethnic disparities in timeliness and comprehensiveness of dementia diagnosis in California. *JAMA Neurol.* 2021;78:657-665. doi:10.1001/jamaneurol.2021.0399
 178. Parra MA, Baez S, Sedeño L, et al. Dementia in Latin America: paving the way toward a regional action plan. *Alzheimers Dement.* 2021;17:295-313. doi:10.1002/alz.12202
 179. for The Latin America and the Caribbean Consortium on Dementia (LAC-CD), Ibanez A, Parra MA, Butler C, for The Latin America and the Caribbean Consortium on Dementia (LAC-CD). The Latin America and the Caribbean Consortium on Dementia (LAC-CD): from networking to research to implementation science. *J Alzheimers Dis.* 2021;82:S379-S394. doi:10.3233/JAD-201384
 180. Ellajosyula R, Narayanan J, Hegde S, et al. Delay in the diagnosis of dementia in urban India: role of dementia subtype and age at onset. *Int J Geriatr Psychiatry.* 2022;37. doi:10.1002/gps.5843. gps.5843.
 181. Plattner IE, Mbakile-Mahlanza L, Marobela S, et al. Developing a computerized brief cognitive screening battery for Botswana: a feasibility study. *Arch Clin Neuropsychol.* 2019;34:682-689. doi:10.1093/arclin/acy071
 182. Porrselvi A, Shankar V. Status of cognitive testing of adults in India. *Ann Indian Acad Neurol.* 2017;20:334. doi:10.4103/aian.AIAN_107_17
 183. Ahmed T, Kumar K, Zhang P. A systematic review of the status of neuropsychological research and dementia in South Asia. *Discov Psychol.* 2023;3:16. doi:10.1007/s44202-023-00078-2
 184. Gugssa SA, Zenebe Y, Seeger S, Comeau D, Derbew M. Cross-cultural adaptation of AD-8 into Amharic for operationalizing the detection of

- cognitive impairment in a primary care setting of Ethiopia. In Review; 2022. doi:10.21203/rs.3.rs-1820446/v1
185. Amaral-Carvalho V, Lima-Silva TB, Mariano LI, et al. Brazilian Version of Addenbrooke's cognitive examination—revised in the differential diagnosis of Alzheimer's disease and behavioral variant frontotemporal dementia. *Arch Clin Neuropsychol*. 2022;37:437-448. doi:10.1093/arclin/acab071
 186. Okada De Oliveira M, Cesar KG, Allen IE, Mioshi E, Nitrini R, Dozzi Brucki SM. Development of the Brazilian Mini-Addenbrooke's cognitive examination (BR M-ACE). *Alzheimers Dement*. 2019;15:P796-P797. doi:10.1016/j.jalz.2019.06.2876
 187. Allegri RF, Villavicencio AF, Taragano FE, Rymberg S, Mangone CA, Baumann D. Spanish boston naming test norms. *Clin Neuropsychol*. 1997;11:416-420. doi:10.1080/13854049708400471
 188. Blanco R, Román F, Iturry M, et al. Cuestionario de detección de deterioro cognitivo AD8-arg para su uso Atención Primaria de la salud en Argentina. *Neurol Argent*. 2016;8:231-236. doi:10.1016/j.neuarg.2016.10.001
 189. Roman F, Iturry M, Rojas G, Barceló E, Buschke H, Allegri RF. Validation of the Argentine version of the Memory Binding Test (MBT) for Early Detection of Mild Cognitive Impairment. *Dement Neuropsychol*. 2016;10:217-226. doi:10.1590/S1980-5764-2016DN1003008
 190. Maito MA, Santamaría-García H, Moguilner S, et al. Classification of Alzheimer's disease and frontotemporal dementia using routine clinical and cognitive measures across multicentric underrepresented samples: a cross sectional observational study. *Lancet Reg Health—Am*. 2023;17:100387. doi:10.1016/j.lana.2022.100387
 191. Moguilner S, Birba A, Fittipaldi S, et al. Multi-feature computational framework for combined signatures of dementia in underrepresented settings. *J Neural Eng*. 2022;19:046048. doi:10.1088/1741-2552/ac87d0
 192. Ben Jemaa S, Marzouki Y, Fredj M, Le Gall D, Bellaj T. The adaptation and validation of an Arabic version of the Cornell scale for depression in dementia (A-CSDD). *J Alzheimers Dis*. 2019;67:839-848. doi:10.3233/JAD-180448
 193. Ben Jemaa S, Attia Romdhane N, Bahri-Mrabet A, Jendli A, Le Gall D, Bellaj T. An Arabic version of the cognitive subscale of the Alzheimer's disease assessment scale (ADAS-Cog): reliability, Validity, and Normative Data. *J Alzheimers Dis*. 2017;60:11-21. doi:10.3233/JAD-170222
 194. Mbakile-Mahlanza L. Validation of a cognitive assessment battery in the Botswana population 2020. <https://iadrp.nia.nih.gov/project/validation-cognitive-assessment-battery-botswana-population> (accessed July 8, 2023)
 195. Mathuranath PS, Nestor PJ, Berrios GE, Rakowicz W, Hodges JR. A brief cognitive test battery to differentiate Alzheimer's disease and frontotemporal dementia. *Neurology*. 2000;55:1613-1620. doi:10.1212/01.wnl.0000434309.85312.19
 196. Meghana R, Jain S, Malo PK, Stezin A, Issac TG. Potential modifications on verbal-language/orientation-memory ratio from Addenbrooke's cognitive examination III to predict mild cognitive impairment from healthy controls. *J Neurosci Rural Pract*. 2023;14:531-532. doi:10.25259/JNRP_223_2023
 197. Paddick S-M, Gray WK, McGuire J, Richardson J, Dotchin C, Walker RW. Cognitive screening tools for identification of dementia in illiterate and low-educated older adults, a systematic review and meta-analysis. *Int Psychogeriatr*. 2017;29:897-929. doi:10.1017/S1041610216001976
 198. Rosselli M, Uribe IV, Ahne E, Shihadeh L. Culture, ethnicity, and level of education in Alzheimer's disease. *Neurotherapeutics*. 2022;19:26-54. doi:10.1007/s13311-022-01193-z
 199. Hansson O, Edelmayer RM, Boxer AL, et al. The Alzheimer's Association appropriate use recommendations for blood biomarkers in Alzheimer's disease. *Alzheimers Dement J Alzheimers Assoc*. 2022;18:2669-2686. doi:10.1002/alz.12756
 200. Schlesinger D, Grinberg LT, Alba JG, et al. African ancestry protects against Alzheimer's disease-related neuropathology. *Mol Psychiatry*. 2013;18:79-85. doi:10.1038/mp.2011.136
 201. Chaudhry A, Rizig M. Comparing fluid biomarkers of Alzheimer's disease between African American or Black African and white groups: a systematic review and meta-analysis. *J Neurol Sci*. 2021;421:117270. doi:10.1016/j.jns.2020.117270
 202. Simrén J, Ashton NJ, Blennow K, Zetterberg H. Blood neurofilament light in remote settings: alternative protocols to support sample collection in challenging pre-analytical conditions. *Alzheimers Dement Amst Neth*. 2021;13:e12145. doi:10.1002/dad2.12145
 203. Gauthier S, Rosa-Neto P, Morais J, Webster C. *World Alzheimer Report 2021: Journey through the diagnosis of dementia*. Alzheimer's Disease International; 2021.
 204. Grinberg LT, de Ferretti RE L, Farfel JM, et al. Brain bank of the Brazilian aging brain study group—a milestone reached and more than 1,600 collected brains. *Cell Tissue Bank*. 2007;8:151-162. doi:10.1007/s10561-006-9022-z
 205. Akinyemi RO, Salami A, Akinyemi J, et al. Brain banking in low and middle-income countries: raison D'être for the Ibadan brain ageing, dementia and neurodegeneration (IBADAN) Brain Bank Project. *Brain Res Bull*. 2019;145:136-141. doi:10.1016/j.brainresbull.2018.08.014
 206. Akinyemi R, Ojagbemi A, Akinyemi J, et al. Gender differential in inclination to donate brain for research among Nigerians: the IBADAN Brain Bank Project. *Cell Tissue Bank*. 2019;20:297-306. doi:10.1007/s10561-019-09769-4
 207. Ogeng'o JA, Cohen DL, Sayi JG, et al. Cerebral amyloid beta protein deposits and other Alzheimer lesions in non-demented elderly east Africans. *Brain Pathol Zurich Switz*. 1996;6:101-107. doi:10.1111/j.1750-3639.1996.tb00790.x
 208. Akinyemi RO, Jenkins C, Nichols M, et al. Unraveling the ethical, legal, and social implications of neurobiobanking and stroke genomic research in Africa: a Study Protocol of the African Neurobiobank for precision stroke medicine ELSI project. *Int J Qual Methods*. 2020;19:160940692092319. doi:10.1177/1609406920923194
 209. Singh A, Arulogun O, Akinyemi J, et al. Biological sample donation and informed consent for neurobiobanking: evidence from a community survey in Ghana and Nigeria. *PLOS ONE*. 2022;17:e0267705. doi:10.1371/journal.pone.0267705
 210. Adigun M, Ojebuyi BR, Akinyemi J, et al. Legal implications of stroke biobanking and genomics research in Sub-Saharan Africa. *J Law Med*. 2022;29:579-598.
 211. Grinberg LT, Nitrini R, Suemoto CK, et al. Prevalence of dementia subtypes in a developing country: a clinicopathological study. *Clin Sao Paulo Braz*. 2013;68:1140-1145. doi:10.6061/clinics/2013(08)13
 212. Wijekoon N, Gonawala L, Wijesinghe P, Steinbusch HW, Mohan C, De Silva KR. A biobank in Sri Lanka that links East and West. *Lancet Neurol*. 2020;19:972. doi:10.1016/S1474-4422(20)30405-1
 213. Wijekoon N, Gonawala L, Ratnayake P, et al. Gene therapy for selected neuromuscular and trinucleotide repeat disorders – An insight to subsume South Asia for multicenter clinical trials. *IBRO Neurosci Rep*. 2023;14:146-153. doi:10.1016/j.ibneur.2023.01.009
 214. Agyeman N, Guerchet M, Nyame S, et al. "When someone becomes old then every part of the body too becomes old": experiences of living with dementia in Kintampo, rural Ghana. *Transcult Psychiatry*. 2019;56:895-917. doi:10.1177/1363461519847054
 215. Hurzuk S, Farina N, Pattabiraman M, et al. Understanding, experiences and attitudes of dementia in India: a qualitative study. *Dement Lond Engl*. 2022;21:2288-2306. doi:10.1177/1471301222118774
 216. Breuer E, Comas-Herrera A, Freeman E, et al. Beyond the project: building a strategic theory of change to address dementia care, treatment and support gaps across seven middle-income countries. *Dement Lond Engl*. 2022;21:114-135. doi:10.1177/14713012211029105

217. Musyimi C, Ndetei D, Muyela LA, Masila J, Mutunga E, Farina N. Integration and evaluation of a community-level dementia screening program in Kenya (DEM-SKY): a Protocol. *J Alzheimers Dis*. 2023;95:1771-1776. doi:[10.3233/JAD-230107](https://doi.org/10.3233/JAD-230107)
218. Govia I, Robinson JN, Amour R, et al. Mapping long-term care in Jamaica: addressing an ageing population. *Sustainability*. 2021;13:8101. doi:[10.3390/su13148101](https://doi.org/10.3390/su13148101)
219. Leroi I, Chaudhry N, Daniel A, et al. A roadmap to develop dementia research capacity and capability in Pakistan: a model for low- and middle-income countries. *Alzheimers Dement N Y N*. 2019;5:939-952. doi:[10.1016/j.trci.2019.11.005](https://doi.org/10.1016/j.trci.2019.11.005)
220. Maestre G, Carrillo M, Kalaria R, et al. The Nairobi Declaration—Reducing the burden of dementia in low- and middle-income countries (LMICs): declaration of the 2022 Symposium on Dementia and Brain Aging in LMICs. *Alzheimers Dement*. 2023;19:1105-1108. doi:[10.1002/alz.13025](https://doi.org/10.1002/alz.13025)

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