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Ethnoracial Disparities in Alzheimer's Disease: Target on Cardiovascular Risks via Lifestyle Changes?

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Dementia in Minorities

Dementia and its most common form, Alzheimer 's disease (AD), are crippling diseases affecting more than 5 million Americans and 30 million people worldwide [1]. Without cure, the cost of AD is astronomical. In the U.S., the combined cost of unpaid hours of caretakers and total cost of health care, long-term care, and hospice services is estimated to be $419 billion for individuals 65 years and older with dementia [1]. America's aging population surviving past 65 years old is the largest growing segment of the population. By the year 2050, 14-16 million people 65 years will be expected to have AD with an associated cost of $1.2 trillion, not including unpaid caretaker hours [2].

Mounting evidence that these diseases disproportionately affect minority ethnoracial groups, particularly African-Americans and Hispanics, make disease mitigation in these populations particularly crucial. Although ethnicity has never been established as an independent predictor of AD, the Alzheimer's Association estimates that African-Americans and Hispanics are about 1.5-2 times more likely to have AD and other dementias compared with non-Hispanic Caucasians of the same age [1]. Moreover, these minority populations demonstrate earlier and more severe AD symptoms [3], yet paradoxically live longer with the disease compared with non-Hispanic Caucasians [4].

The etiologies of the disparity of AD prevalence, symptomology, and time to mortality are not well understood. Ostensibly, they are most likely multifactorial, rooted in a complex interaction of social (e.g., literacy, socioeconomic status), biological (e.g., genotype, vascular function), and other factors [5]. Social disparities in such factors as education and literacy unfortunately exist in ethnoracial minorities. Lower levels of these sociocultural factors are associated with decreased cognitive performance [6,7]. The inaccurate perception that memory loss is part of normal healthy aging may also stymy pursuit of treatment in ethnoracial minorities [8]. Inheriting the genotype of the ε4 apolipoprotein polymorphism has been associated with the increased risk for developing AD. In spite of the disparate prevalence and symptomology, the presence of the ε4 is similar in African-Americans and non-Hispanic Caucasians and increases the relative risk of AD similarly in both populations [9]. Additionally, the effect of the ε4 is attenuated in Hispanics [9]. These issues make the detection and the prevention more complicated in ethnic and racial minorities.

Developments are being made in the use of biomarkers to detect early warning signs of AD pathology, such as cerebrospinofluid levels of amyloid β and tau protein. However, biomarker testing can be costly, invasive, and not easily accessible to minorities. Traditional modifiable cardiovascular risk factors remain an easy target in midlife as a surrogate for possible increased risk of AD, and could be used in conjunction with developing biomarkers to track early alterations in cognitive function [10,11]. A vascular hypothesis has been proposed as a feasible route to AD [12]. According to this hypothesis, cardiovascular risk factors accumulate gradually leading to chronic brain hypoperfusion and a neuroglial energy crisis. This energy crisis in turn culminates neurodegeneration and AD [12]. A variety of risk factors for cardiovascular disease have been associated with outcomes leading to AD. For example, hypertension and diabetes are related to chronic brain hypoperfusion [13,14] and hyperlipidemia with amyloid β accumulation [15]. The prevalence of modifiable cardiovascular risk factors is greater in African-Americans and Hispanics than in non-Hispanic Caucasians. African-Americans are known to have increased risk of hypertension [16], while Hispanics have increased risk of insulin resistance [17]. Reducing the number of aforementioned cardiovascular risk factors throughout the lifespan may lead, in theory, to a delay and/or prevention of AD pathology [12]. This shift towards primary prevention can be attained through lifestyle interventions involving diet and exercise [18]. One dietary intervention may be increasing dairy consumption. Simply adding dairy products to the routine diet can have hypotensive effects in middle-aged and older adults with elevated blood pressure [19]. Importantly, increased dairy consumption has also been shown to be associated with reduced risk of AD [20]. Regular physical activity is beneficial for the prevention and treatment of cardiovascular risk factors. Saliently, large epidemiological studies have shown that physical activity is associated with reduced risk of AD [21,22], and a randomized controlled exercise intervention trial resulted in improved global cognitive functioning compared with usual care controls [23]. More importantly, these benefits occurred even in patients already experiencing mild cognitive impairment and lasted 6 months following study completion.

Although lifestyle modification to reduce the risk of cardiovascular diseases and ultimately AD is a feasible strategy to slow and prevent AD pathology, clearly more research is needed. Because of the existing disparity of AD prevalence and symptomology in ethnoracial minorities, it would be extremely valuable from the public health perspective to establish lifestyle modification strategies for AD risk reduction in these populations. The impact of reducing only 10-25% of existing cardiovascular risk factors could prevent up to 3 million cases of AD worldwide, and this would greatly benefit ethnoracial minorities in the U.S [24].

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