The Journal of Prevention of Alzheimer's Disease 13 (2026) 100667

Contents lists available at ScienceDirect

The Journal of Prevention of Alzheimer's Disease

journal homepage: www.elsevier.com/locate/tjpad

Editorial

From advice to agency: making brain health actionable and equitable for middle-aged adults with mild neurocognitive disorder

ARTICLE INFO

Keywords:
Dementia
Prevention
Cognition
Equity
Lifestyle
Midlife

The scoping review by Dookhy and colleagues, published in this issue of the *Journal of Prevention of Alzheimer's Disease*, addresses a central challenge in contemporary dementia prevention: how to translate knowledge of modifiable risk into strategies that people can realistically adopt and sustain [1]. After screening 6349 records, the authors included 17 studies of non-pharmacological brain health strategies used by middle-aged adults with mild neurocognitive disorder (mNCD). These ranged from structured physical and cognitive programs to self-directed dietary, social, and compensatory practices.

Physical activity was prominent, whereas the non-pharmacological prevention and management of cardiometabolic risk factors received less attention. Participants commonly lived in urban areas and were relatively well educated. Most participants were women, yet sex- and gender-related differences in access, engagement, and outcomes were seldom examined. People living in rural areas, people experiencing socioeconomic disadvantage, and people from minoritized racial and ethnic groups were insufficiently represented. None of the included interventions explicitly reported participant co-design.

This question is particularly important given the 2024 *Lancet* Commission's estimate that 14 potentially modifiable factors collectively account for approximately 45% of dementia cases worldwide [2]. This figure has appropriately energized prevention efforts, but it does not imply that almost half of dementia cases would disappear if individuals simply exercised more, ate better, and remained socially active. Population-attributable fractions describe preventive potential at the population level; they neither predict individual outcomes nor capture people's capacity to modify risk. The review therefore exposes three gaps. First, there is an evidence gap because few studies specifically address middle-aged adults with mNCD. Second, there is a translation gap because epidemiologically important risks are not consistently converted into feasible interventions. Third, there is an equity gap because people at greatest risk of dementia may have the least opportunity to access and sustain the programs being evaluated.

Conceptual precision is essential. mNCD is a syndrome rather than an etiologically homogeneous entity. The review brings together clinically and etiologically diverse populations, ranging from individuals with amnestic mild cognitive impairment—sometimes in the context of Alzheimer's disease—to those with cognitive impairment associated with cancer, HIV infection, multiple sclerosis, Huntington's disease, or traumatic brain injury. These conditions may share functional consequences but differ in their mechanisms, prognosis, and responsiveness to intervention. Moreover, several studies extended beyond the authors' 40–65-year definition of midlife without reporting age-stratified results. The review can therefore map the available strategies, but it cannot establish that any particular strategy prevents dementia in middle-aged adults with mNCD. Future trials should define cognitive phenotype and etiology, characterize vascular and biomarker status where relevant, and report age-disaggregated outcomes.

A further limitation concerns what counts as success. Across the included studies, outcomes ranged from cognitive test scores and selected biomarkers to psychological well-being, adherence, and self-reported lifestyle behaviors [1]. Improvements in a cognitive domain over several weeks or months may be clinically valuable, but they should not be equated with prevention of dementia. Likewise, observational associations between dietary or social behaviors and cognition cannot establish a causal effect on dementia risk. Future studies should consider long-term follow-up and adopt a clearer hierarchy of outcomes, distinguishing short-term changes in cognition or behavior from preservation of everyday functioning and quality of life, reduced caregiver burden, and delayed progression to major neurocognitive disorder.

DOI of original article: https://doi.org/10.1016/j.tjpad.2026.100657.

https://doi.org/10.1016/j.tjpad.2026.100667
Received 14 August 2026; Received in revised form 24 August 2026; Accepted 25 August 2026
Available online 6 September 2026

Potential harms and burdens—including fatigue, anxiety induced by risk communication, financial costs, and unequal access—should also be assessed.

The 2025 US POINTER trial provides an instructive counterpoint. Among 2111 adults aged 60–79 years at increased risk of cognitive decline, both structured and self-guided multidomain lifestyle interventions were associated with improved global cognitive scores over two years, with a statistically greater improvement in the structured intervention group [3]. However, the between-group difference was modest, its longer-term clinical significance remains uncertain, and the cohort was older than the population targeted by Dookhy et al. Nevertheless, the trial reinforces an important principle: information alone is not equivalent to intervention. Structure, accountability, social contact, and cardiovascular monitoring may be active ingredients rather than mere features of delivery.

More structured programs may benefit people who can access them while remaining out of reach for those facing employment or caregiving demands, transport or financial barriers, inaccessible program design, digital exclusion, or mental fatigue. Qualitative evidence confirms that barriers operate across patient, clinician, organizational, community, and policy levels [4]. Sustained engagement also depends on motivational processes, including perceived benefit, self-efficacy, enjoyment, and immediate reinforcement. These motivational processes may be affected by cognitive impairment, depression, or apathy [1]. Interventions should therefore minimize the effort required to initiate and maintain behavior, while providing achievable goals, timely feedback, and supportive social contact. This calls for proportionate support: a low-burden core intervention, supplemented according to individual needs and preferences by coaching, supervised physical activity, cognitive rehabilitation, management of hearing and vision loss, and social or occupational support. Evaluation should extend beyond cognition and adherence to include reach, participant burden, functional outcomes, quality of life, and differential effects across equity-related characteristics.

Co-design should also be understood as more than consultation after an intervention has already been conceived. People living with cognitive impairment, their families, and the professionals who support them should be involved in defining meaningful goals, acceptable levels of burden, delivery formats, and relevant outcomes from the outset. This is particularly important in midlife, when employment, caregiving, family responsibilities, and concerns about prognosis may shape both priorities and capacity to engage. Co-design cannot by itself remove structural inequalities, but it can reveal barriers that conventional efficacy trials overlook and help avoid interventions that are theoretically sound yet impractical in everyday life.

Prevention must also move beyond individualized risk messaging and address the collective conditions that shape people's ability to adopt health-promoting behaviors. Population-level interventions can reduce exposure to several dementia risk factors, although their direct effects on dementia incidence remain insufficiently documented [5]. Detection and control of hypertension, identification and management of hearing and vision loss, initiatives that foster social connectedness, smoke-free environments, clean-air policies, education, safe opportunities for physical activity, and access to healthy food cannot be achieved through individual motivation alone.

Clinical counseling remains necessary, but it should foster agency without assigning blame. The risk of cognitive decline arises from complex interactions among biological vulnerabilities (including genetic susceptibility), environmental exposures, and social determinants. Risk reduction is therefore probabilistic, and lifestyle strategies should complement, rather than replace, diagnostic assessment and disease-specific treatment.

The role of relatives also deserves careful consideration. Support from family members or caregivers may facilitate behavioral change, compensate for difficulties in planning or memory, and help sustain engagement over time [1]. However, interventions should not transfer responsibility for prevention to families without assessing their availability, preferences, and burden. Support should strengthen the individual's autonomy and shared decision-making rather than create surveillance or dependency.

For clinical practice, the sequence is pragmatic. Clinicians should clarify the syndrome and identify treatable contributors, then assess modifiable risks alongside the person's capacity and opportunity to act. They should agree on a small number of personally meaningful goals, connect advice to accessible services and follow-up, and reassess both benefit and burden. For research, equity-conscious recruitment, flexible and accessible delivery—including alternatives to exclusively digital provision when required—and co-design with the target populations should be incorporated from the outset. The next generation of prevention studies must ask not only whether an intervention works, but for whom, under what conditions, and with what burden. Brain health will become a credible promise only when the capacity to act is designed into the intervention itself.

Funding

No external funding was received for this work.

Conflict of interest

Hermine Lenoir declares no conflict of interest.

Declaration of competing interest

The author wrote the manuscript in its entirety and used *DeepL* to improve the English language and style. The author critically reviewed and revised the entire manuscript and takes full responsibility for its content.

References

[1] Dookhy J, McCague Y, Hickson J, Stokes D, Kroll T, Frazer K. Brain health strategies in middle-aged individuals with mild neurocognitive disorder: a scoping review. J Prev Alzheimers Dis 2026. In press.
[2] Livingston G, Huntley J, Liu KY, Costafreda SG, Selbæk G, Alladi S, et al. Dementia prevention, intervention, and care: 2024 report of the Lancet standing Commission. Lancet 2024;404:572–628. https://doi.org/10.1016/S0140-6736(24)01296-0.
[3] Baker LD, Espeland MA, Whitmer RA, Snyder HM, Leng X, Lovato L, et al. Structured vs self-guided multidomain lifestyle interventions for global cognitive function: the US POINTER randomized clinical trial. JAMA 2025;334:681. https://doi.org/10.1001/jama.2025.12923.
[4] Mace RA, Cohen JE, Lyons C, Ritchie C, Bartels S, Okereke OI, et al. Socio-ecological barriers to behavior change-oriented dementia prevention: a qualitative study of healthcare professionals' perspectives. Aging Ment Health 2025;29:1055–64. https://doi.org/10.1080/13607863.2024.2430525.
[5] Walsh S, Wallace L, Kuhn I, Mytton O, Lafortune L, Wills W, et al. Population-level interventions for the primary prevention of dementia: a complex evidence review. EClinicalMedicine 2024;70:102538. https://doi.org/10.1016/j.eclinm.2024.102538.

Hermine Lenoir[a,b,*]
[a] *Services de Gériatrie 1 & 2; Centre Mémoire de Ressources et de Recherche Île-de-France-Broca, AP-HP, Hôpital Broca, Paris, France*
[b] *INSERM UMR-S 1144, Optimisation Thérapeutique en Neuropharmacologie, Université Paris Cité, Paris, France*

[*] Corresponding author at: Services de Gériatrie 1 & 2, Hôpital Broca, 54-56 rue Pascal, 75013, Paris, France.
E-mail address: hermine.lenoir@aphp.fr.