Alzheimer’s Disease: How to Prevent It in Advance?

A Multidisciplinary Perspective from Pathophysiology to Design-Driven Early Prevention

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Abstract: Background: Alzheimer’s disease (AD) is a progressive neurodegenerative disorder imposing a growing burden on aging societies. While treatment receives much attention, early prevention remains underexplored by the public.

Aim: This paper examines how AD can be prevented through daily lifestyle interventions, drawing on genetic insights, biomarker research, pharmacological progress, and design-oriented approaches.

Methods: The paper reviews AD etiology, pathophysiology, global aging trends, and existing prevention programs (e.g., FINGERS). It also analyzes two design concepts (Ambient Assisted Living and a smart vase for reminiscence therapy) and reports original survey data on public awareness.

Findings: Global aging, declining birth rates, and rising young-onset AD (including a case at age 19) make early prevention urgent. Over 93% of survey respondents were unaware AD can be prevented in advance. Multidomain lifestyle interventions show cognitive benefits (e.g., 25% improvement in FINGERS), and design-driven solutions can support brain stimulation, safety monitoring, and social engagement.

Conclusion: Early prevention of AD is scientifically feasible and socially necessary. Designers have a critical role in translating medical evidence into everyday tools that help people—especially those over 45 and the elderly—prolong cognitive health.

Key words: Alzheimer’s disease; early prevention; lifestyle intervention; design for dementia; ambient assisted living.

1. Introduction

Alzheimer's disease (AD) is a progressive, irreversible neurological disorder that begins with mild memory loss and may lead to the complete loss of conversational ability and responsiveness to the environment. According to the literature, 70% of AD patients experience at least one episode of getting lost [1,2]. Public awareness remains alarmingly low: a 2022 CNN survey found that more than 80% of Americans are unfamiliar with the disease [3]. Motivated by this gap, the author conducted a quick street survey during their school years and found that 93.6% of respondents did not know that AD can be prevented in advance.

The 21st century is defined by global aging. By 2050, the world's population aged 60 and over will reach 2.1 billion [4]. Neurodegenerative diseases, especially AD, have thus become a major healthcare challenge. At the same time, early prevention strategies are gaining scientific support. For example, the Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability (FINGERS) demonstrated that a multidomain lifestyle intervention (diet, exercise, cognitive training, vascular management) can significantly improve cognitive outcomes [5,6].

This paper has three objectives:
(1) To synthesize current knowledge on the pathophysiology, etiology, and global context of AD, with special attention to young-onset cases;
(2) To evaluate existing prevention programs and design-oriented technologies;
(3) To provide a foundation for designers to create accessible, daily-life interventions that delay or prevent AD.

2. Alzheimer's Disease: Fundamentals

2.1 Definition and Epidemiology

AD is characterized by β-amyloid plaques and tau neurofibrillary tangles in the brain, which disrupt neuronal communication and lead to cell death [7,8]. As of April 2023, approximately 55 million people worldwide suffer from AD [9]. Between 2000 and 2016, AD deaths increased by 139%, whereas deaths from heart disease (the leading cause of death) fell by 6% [10]. AD is now the seventh leading cause of death among people aged 65 and older [7] and one of the most socially costly diseases in developed nations [11,12].

2.2 History

AD is named after German psychiatrist Alois Alzheimer (1864–1915), who in 1906 first described the combination of progressive cognitive decline with amyloid plaques and neurofibrillary tangles. Research stagnated for over half a century but revived in the 1970s–1980s. In the 1990s, genetic advances identified genes linked to disease onset [13].

2.3 Clinical Stages

- **Mild stage:** Noticeable memory loss, time/space disorientation, difficulty with complex tasks. The author's own 82-year-old grandfather was found to have cerebellar atrophy and hippocampal abnormalities, diagnosed as early AD.
- **Moderate stage:** Language problems, increased dependency, risk of getting lost. A 68-year-old family friend experienced a lost episode.
- **Severe stage:** Complete loss of independent daily living.

2.4 The Growing Reality of Young-Onset Alzheimer's

AD is not exclusive to the elderly.

- **Case 1 (China, 2023):**A 19-year-old male was diagnosed with probable AD without any recognized genetic mutations – the youngest confirmed case to date [14,15].
- **Case 2 (France):** The youngest French case is around age 20 [16]. France has an estimated 33,000 AD patients under 65. The "Le Chemin" residence in Cesson admits patients under 60, the youngest being 38. Neuropsychologist Anne-Laure Lesage notes: "A person who is not stimulated loses much faster than those who are often stimulated" [17].

These cases underscore that AD can affect people in their 20s, 30s, and 40s, making early prevention relevant to a much broader population than previously assumed.

3. Pathophysiology and Etiology

3.1 Core Pathological Mechanisms

AD involves two hallmark proteinopathies: extracellular β- amyloid (Aβ) plaques and intracellular tau neurofibrillary tangles(NFT). Aβ plaques first appear in neocortical areas and spread inward, while NFT originate in the medial temporal lobe and spread outward. Aβ and tau act synergistically: Aβ alone causes neuronal hyperactivity, tau alone induces inhibition, and when both are present, tau dominates, leading to neural circuit dysfunction [18].

3.2 Basic Mechanisms

- **βamyloid plaques:** Disrupt synaptic transmission, activate microglial-mediated neuroinflammation, induce oxidative stress, and trigger apoptosis.
- **Tau tangles:** Hyperphosphorylation of tau causes detachment from microtubules, impairing axonal transport and causing nutrient deficiency.
- **Neuronal degeneration and cognitive decline:** Loss of connectivity, energy shortage, and eventual cell death in memory-related regions (e.g., hippocampus) [19,20].

3.3 Etiological Factors

Genetic factors (APOE ε4, rare APP/PSEN1/PSEN2 mutations, Down's syndrome) account for only 1–2% of cases. The majority of risk is modifiable and includes: cardiovascular conditions (hypertension, diabetes, high cholesterol), lifestyle factors (poor diet, physical inactivity, smoking), environmental exposures, head trauma, chronic inflammation, and psychosocial factors such as loneliness and social isolation [21].

4. Global Demographic Context and Challenges

4.1 Global Aging

By 2030, one in six people worldwide will be aged 60 or older. By 2050, the global population aged 60+ will double to 2.1 billion. Figure 2 and 3 show that in France, over-60 population is projected to reach 35% by 2050; in China, it will exceed 40% [22].

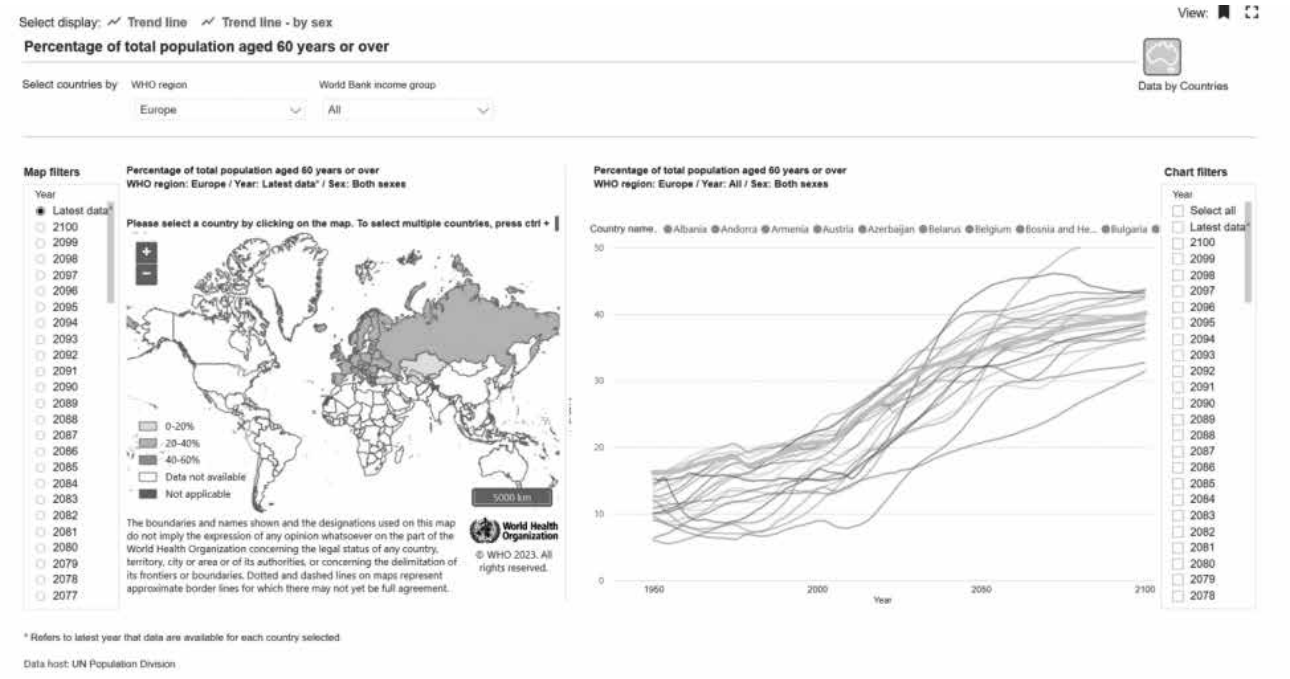

Figure 1 Aging trends in European countries 1950-2100

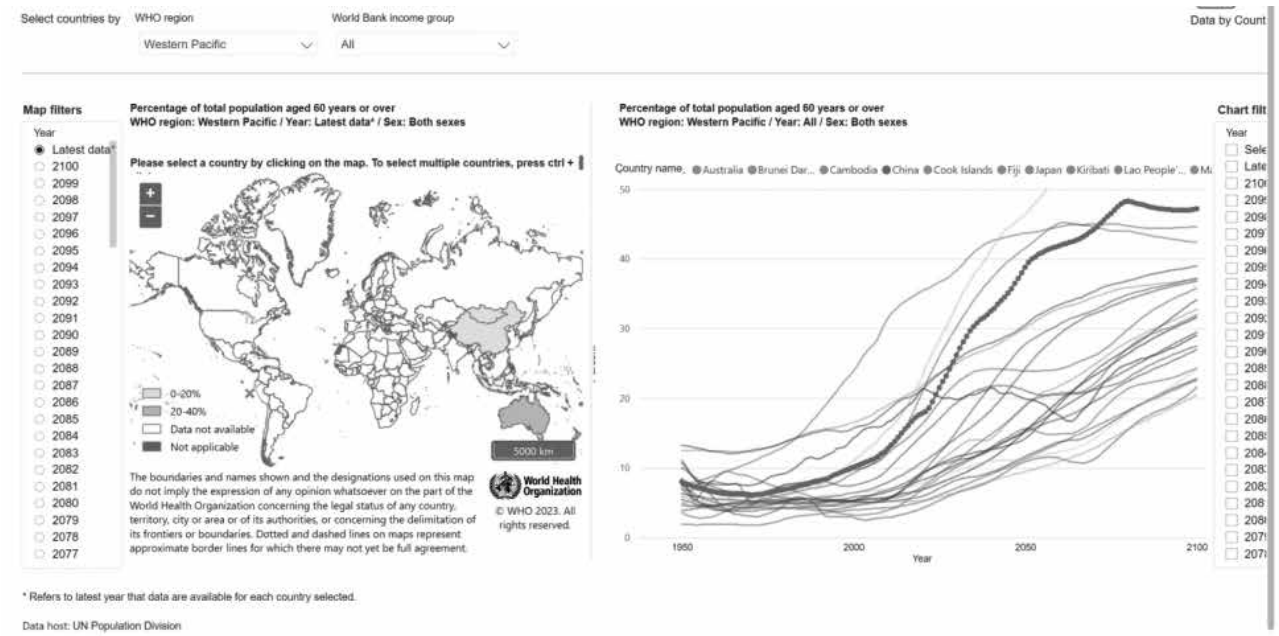

Figure 2 The aging trend in countries of the Western Pacific 1950-2100

4.2 Declining Birth Rates and OnlyChild Families

Global birth rates are declining rapidly (Figure 4) [23]. In Europe, 47% of families with children are only-child families [22]. The combination of aging parents, aging grandparents, and a single adult child creates a heavy caregiving burden. The expected economic impact is substantial: AD is projected to represent 9.6% of the French population and 6.2% of the French working population by 2050 [24].

These demographic trends make early prevention not only a medical imperative but also a socio-economic necessity.

5. Current Diagnosis, Treatment, and Their Limitations

5.1 Diagnostic Methods

Diagnosis involves clinical evaluation, cognitive screening (MMSE, MoCA [25]), neuroimaging (structural MRI [26], amyloid/tau PET [27]), and CSF/blood biomarkers (e.g., p-tau217) [28–30]. Early detection is improving but remains inaccessible in many regions.

5.2 Pharmacological Treatments

- **Cholinesterase inhibitors (donepezil, rivastigmine, galantamine):** Symptomatic, limited efficacy, side effects.
- NMDA antagonist (memantine): Symptomatic only.
- **Antiamyloid therapy (aducanumab):** Controversial efficacy, risk of brain swelling. Once started, it is a lifelong treatment.

All current medications are curativenone; they do not stop or reverse disease progression.

5.3 NonPharmacological and Emerging Interventions

Cognitive training, physical exercise, and dietary adjustments provide supportive benefits but are not diseasemodifying. Emerging therapies (tautargeting, stem cells, gene therapy) remain experimental.

6. Existing Prevention Programs and DesignDriven Solutions

6.1 The FINGERS (Finland)

The FINGERS (Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability) is a landmark randomized controlled trial. It uses a multidomain approach: nutritional guidance (Mediterranean diet), physical exercise, cognitive training, and management of cardiovascular risk factors. After two years, the intervention group showed a 25% higher composite cognitive score, an 83% improvement in executive function, and a 150% improvement in processing speed compared to controls [5,6].

Limitations: High cost, unclear longterm sustainability, lack of biomarker endpoints, and limited generalizability to non-Finnish populations.

6.2 Ambient Assisted Living (AAL) and Smart Home Technologies

AAL systems integrate sensors (presence, temperature, motion, WiFi bracelets) and actuators (light control, emergency alarms) to support aging in place. Figures 3–6 illustrate sensor deployment in homes and nursing homes. Benefits include fall detection, medication reminders, emergency response, and cognitive support. However, only 10% of over75s in the EU use the internet, creating a digital divide [31]. High costs and privacy concerns also limit adoption.

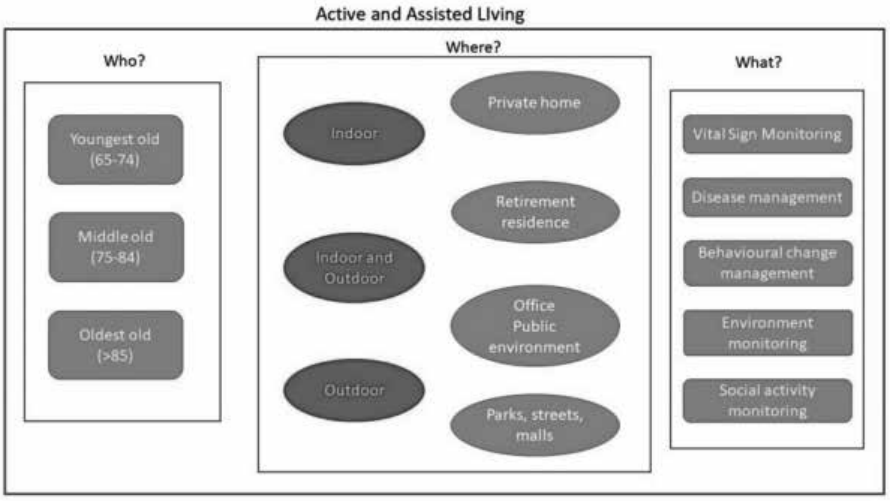

Figure 3 Basic aspects to consider in AAL system design and development

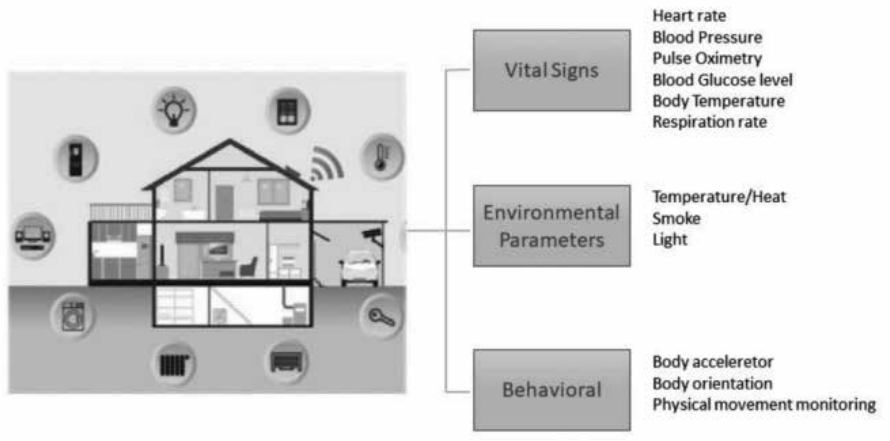

Figure 4 Sensors in homes collect data on residents' locations, object interactions, and health status.

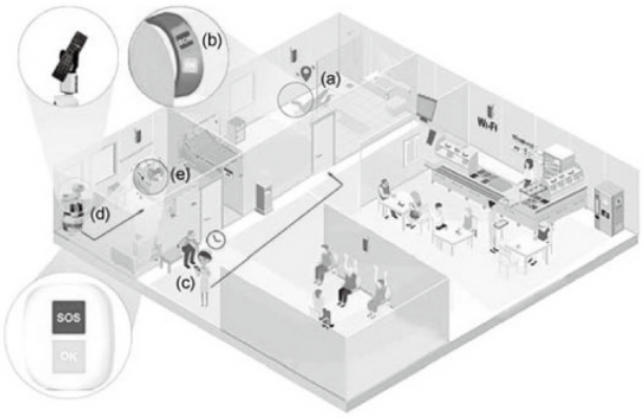

Figure 5 Nursing homes use sensors for caregiver support and indoor positioning. Wi-Fi bracelet detects falls and alerts caregivers. Robot assists bedridden residents.

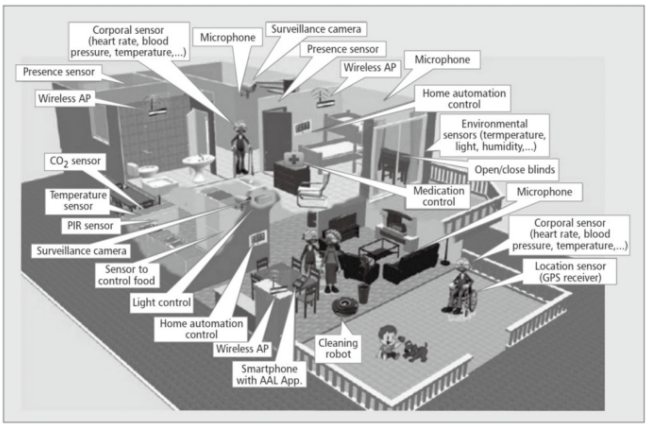

Figure 6 Examples of AAL homes equipped with sensors (e.g., presence, temperature) and actuators (e.g., light, home automation, drug control).

6.3 Conceptual Design: Wearable Headset (Brandon Lewandowski)

A conceptual headset with two spectrometers placed against the temples to scan brain tissue for early AD anomalies. Not yet marketed (Figure 7).

Figure 7 The picture shows Brandon Lewandowski's head-mounted detection design

6.4 RECALL: Intelligent Vase for Reminiscence Therapy [34]

RECALL combines plant growing with photo-based reminiscence therapy. Using gesture interaction, the device plays back family photos to stimulate memory. Although still a concept, photo-based interventions have recognized therapeutic value in triggering autobiographical memories and reducing behavioral symptoms.

6.5 The Role of Design in Early Prevention

From a design perspective, the author identifies two core questions:

1. How can people prolong AD onset through daily life?
2. How can people of a certain age maintain social engagement and cognitive activity, especially when living alone?

Design can contribute by:

- Translating complex multidomain interventions (like FINGERS) into simple, engaging daily routines.
- Creating affordable, nonstigmatizing products that encourage mental stimulation (e.g., puzzles, memory games, social platforms).
- Developing supportive environments (smart home features) that are intuitive for the elderly.

7. Original Survey Findings

Method: Two questionnaires were administered.

- General public (n≈150, convenience sample): 93.6% did not know AD can be prevented in advance; over 80% were unaware of youngonset AD.
- AD community (caregivers and family members, n=30): 63.3% of patients had gotten lost (once or more); 23.45% had fallen; 72.5% had medication errors; 21% of families reported a nearaccident due to forgetting boiling water.

These findings confirm a severe awareness gap and the urgent need for accessible prevention tools.

8. Conclusion

Global aging, declining birth rates, and the increasing incidence of youngonset Alzheimer's disease demand a paradigm shift from reactive treatment to proactive early prevention. Genetic predisposition explains only 1–2% of cases; the majority of risk factors are modifiable through lifestyle and environmental interventions.

Existing programs such as FINGERS provide strong evidence that multidomain lifestyle changes can improve cognitive outcomes. Meanwhile, designdriven solutions—from smart home systems to reminiscence therapy devices—show promise in making prevention practical and engaging.

Only through such multidisciplinary efforts can we hope to delay AD onset and reduce its individual, familial, and societal burden.

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