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DEMENTIA RISK REDUCTION

REDUCING DEMENTIA RISK:

A SUMMARY OF THE SCIENCE AND PUBLIC HEALTH IMPACT

The Public Health Center of Excellence on Dementia Risk Reduction coordinates risk reduction efforts and helps public health agencies share best practices. The Center translates the latest science on dementia risk reduction into actionable tools, materials and messaging that public health agencies can use to reduce dementia risk for all people — including those in diverse, underserved and higher-risk communities.

Find the summaries of science and additional tools, resources and data at: alz.org/riskreduction

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Wake Forest® School of Medicine

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Reducing the risk of age-related cognitive decline and dementia is a major and urgent public health priority affecting individuals, families, communities and health care systems nationwide. In response, the National Institutes of Health and the World Health Organization, among others, have focused attention on lifestyle and behavioral actions that could preserve cognitive function and reduce risk of cognitive decline and possibly dementia.

In 2018, the U.S. Congress passed – and in 2024 reauthorized – the Building Our Largest Dementia (BOLD) Infrastructure for Alzheimer's Act, which is aimed at building a nationwide public health infrastructure to address Alzheimer's disease. This includes expanding efforts on dementia risk reduction. As part of this initiative, the Centers for Disease Control and Prevention (CDC) designated the Alzheimer's Association as the Public Health Center of Excellence on Dementia Risk Reduction (Center). In collaboration with Wake Forest University School of Medicine, the Center convened a panel of nationally and internationally recognized experts in public health, epidemiology, clinical practice, aging and cognitive science to review and synthesize the current state of the science on risk factors for cognitive decline and dementia. As a result of this work, the Center released a series of summaries reviewing the state of the evidence on a variety of potential risk factors. And, this work helped inform the Association's development of 10 Healthy Habits for Your Brain. In 2022, the Center worked with Wake Forest to expand this scientific review to include social determinants of health related to dementia risk, further strengthening the foundation for public health strategies to support brain health across the lifespan.

Now, the Center, again in collaboration with Wake Forest and experts in relevant disciplines, has updated the science summaries to reflect the latest research. The updated and expanded summaries cover multiple potential risk factors: alcohol, depression, diabetes and obesity, diet and nutrition, education, exercise, hypertension, sensory impairment, sleep, social engagement, smoking, and traumatic brain injury.

Each summary assesses the current state of evidence, explores implications for public health and discusses the role of social determinants of health. The summaries are intended to support public health agencies in implementing evidence-informed interventions tailored to the diverse communities they serve – and to position individuals and communities for successful cognitive aging.

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EDUCATION AND COGNITIVE ENGAGEMENT

WHAT IS ALREADY KNOWN

Enriched educational and cognitive experiences are linked to lower dementia risk. Research is exploring key mechanisms, such as the stronger impact of education quality and literacy compared with the number of years of schooling. Early education shapes cognitive ability, while lifelong enrichment through work and leisure more strongly affects cognitive decline. Animal studies show that cognitive enrichment fosters neural growth, even in old age. Brain plasticity persists lifelong and is influenced by behavior and lifestyle. These findings suggest cognitive enrichment both in early critical windows of life and throughout the lifespan impart unique opportunities to support brain health.

BACKGROUND AND EVIDENCE BASE

Evidence from preclinical studies

Animal studies show that brain structure and function remain responsive to external experiences throughout life. In adults, up to 10% of synapses can turn over within a single week. Aged animals in cognitively enriched environments develop more complex neural connections, larger cell bodies, and increased neuron counts. Novelty appears to be a key factor: repeated exposure to the same tasks yields limited benefits, while new stimuli and challenges promote the formation of new brain cells and connections. Most human studies on cognitive enrichment are observational, limiting causal conclusions. However, preclinical findings support a causal role for sustained, novel mental stimulation in promoting brain health and inform our understanding of cognitive enrichment in humans.

Evidence from clinical studies

In human studies, cognitive enrichment is often divided in three domains of life: 1) educational quality and attainment, 2) occupational demands, and 3) leisure activities.

Educational quality and attainment: Educational attainment continues to increase globally, a trend that has been directly linked to decreased rates of incidence of dementia including in the United States. Inclusivity in educational opportunities continues to be a challenge, although the gender gap, which may have historically contributed to higher dementia risk in women, is closing. Years of educational attainment are robustly associated with level of cognitive ability and lower odds of dementia across multiple studies. However, whether education relates to the rate of cognitive decline with age is debated. Several meta-analyses and large-scale epidemiologic studies suggest that education may instead promote a higher cognitive starting point and greater capacity to maintain cognitive function in the face of neuropathologic changes (e.g., Alzheimer's plaques and tangles) versus slowing overall rate of cognitive aging. These patterns suggest that education may promote one's "cognitive reserve".

Additionally, the mechanisms of education on dementia are likely multifaceted. Several studies suggest that educational quality (as measured via literacy and reading levels) but not quantity may be a key ingredient. Among adults with similarly low education in the United States (<5 years), those who were not literate demonstrated a three-fold increased risk of having dementia at baseline and a two-fold risk of developing dementia over the study period. Quality of education is a critical consideration when examining systematic disparities in educational opportunities across historically underserved populations in the United States, such as racial and ethnic minorities and women. Other key mediators of education include intelligence, occupational attainment, and health literacy. One recent study using genetic predictive models showed that intelligence and education were bidirectionally related to one another and to dementia risk, but that the effects of education on dementia risk were explained via intelligence. Further, combined analyses examining seven population-based studies showed that education and occupational complexity independently contribute to dementia risk; however, there was a stepwise pattern such that the greatest protection was conferred in adults with both higher education and occupational complexity. Notably, over a quarter of the effect of education on dementia risk was explained through occupational attainment, highlighting the important overlap among these interrelated factors (education, intelligence, and occupational attainment).

Occupational demands: Occupational activities are another central source of sustained cognitive stimulation that appear to offer protection against future dementia risk. High stimulation occupations are conceptualized as positions that have demanding tasks and high job decision latitude (e.g., "job control"). In a seminal study showcasing how occupation may shape the brain, taxi drivers in London, a city known for its complex street system, had significantly larger volumes in the areas of the brain responsible for spatial navigation and memory (posterior hippocampus) compared with non-drivers or even

compared with bus drivers who navigate a prescriptive route each day. Ongoing cohort and epidemiologic studies continue to replicate these effects. A recent population-based study estimated that adults engaged in occupations with higher cognitive stimulation demonstrate 21% decreased risk of dementia 10 years later. Beyond cognitive stimulation, there are likely multiple neuroprotective factors that contribute to occupation-related brain health, including access to health care, financial resources, social connection, and sense of purpose, all of which have been previously linked to dementia risk and continue to be an area of study.

Leisure activities and cognitive training: Cognitively stimulating leisure activities are defined as unstructured, enjoyable activities that engage and challenge the mind, often involving learning, novelty, or problem-solving, and are independent of work or routine daily tasks. The activities should be something that is challenging or involves learning something new. A systematic review of five meta-analytic studies showed that greater engagement in mentally stimulating activities was associated with a 31% reduced risk of cognitive impairment and a 42% reduced risk of dementia, as well as better overall cognitive performances and slower cognitive decline. A study of monozygotic twins who share the same genetic make-up demonstrated a 26% reduced risk of dementia in the twin who engaged in greater cognitive and social activities during midlife — an effect that was particularly prominent in twin dyads who had high genetic risk for Alzheimer's disease (APOEe4 carriers). These findings highlight the importance of novelty and mental challenge in maintaining cognitive benefits. Activities that once stimulated the brain may lose their effect if they become habitual or passive, highlighting the need for ongoing learning and adaptation.

Other opportunities to directly engage in cognitive stimulation are through increasingly available cognitive training platforms. A recent Cochrane review evaluating the evidence on randomized controlled trials (RCTs) that tested computerized cognitive training paradigms in normal older adults suggested there may be small benefits in global cognitive skills following brief (12-26 week) training programs. However, there was notable heterogeneity in the dose, duration, intensity, and methodological approaches (e.g., lack of blinding, small samples) of studies that contributed to overall low quality of evidence and unclear persistence or domain specificity (e.g., memory vs. processing speed) of these benefits. Ultimately, computerized training platforms may be beneficial but are a financial and time investment, given the evidence supporting beneficial effects of many other types of (often free) leisure cognitive activities.

IMPLICATIONS FOR PUBLIC HEALTH

Educational quality, lifelong learning opportunities, and health literacy represent key public health opportunities for reducing dementia risk. In fact, trends showing generational increases in educational access and quality track with decreased dementia incidence rates in the United States. Education promotes cognitive ability level ("cognitive reserve"), which enhances the brain's ability to cope with age-related changes and neurodegenerative diseases. In addition to formal education, engaging in cognitively stimulating occupational and leisure activities — such as reading and learning new skills — can further support cognitive health into the oldest ages. Promoting equitable access to quality education from an early age and encouraging lifelong learning and leisure-time cognitive engagement can help reduce dementia prevalence, particularly in highest risk, underserved communities. Public health initiatives that support both equal educational opportunities and cognitively enriching activities across the lifespan are essential components of a sustainable dementia prevention strategy in the United States.

The role of social determinants of health

Educational attainment and quality are closely tied to income, employment opportunities, housing stability, access to health care, and exposure to chronic stress, all of which influence long-term brain health. These social and economic conditions shape health behaviors and exposures across the life course, ultimately affecting cognitive aging and dementia risk. Individuals with lower levels of education are more likely to experience socioeconomic disadvantages, reduced access to cognitively enriching environments, and limited health literacy, which together contribute to increased risk for cognitive decline and dementia. Importantly, disparities in educational opportunities often reflect broader structural inequities, including systemic racism and underinvestment in schools serving low-income and minority communities. Addressing these disparities through public health and policy interventions, such as improving access to high-quality early education, supporting school completion, and expanding affordable post-secondary opportunities, can help reduce inequities in cognitive aging and promote brain health across the lifespan.

EDUCATION AND COGNITIVE ENGAGEMENT

DISCUSSION

Preclinical and clinical studies highlight the importance of cognitive enrichment in dementia risk reduction. Animal research shows that novel experiences enhance neural growth and resilience, supporting lifelong mental stimulation. In humans, higher levels of education correlate with lower dementia risk, though education quality may matter more than years of schooling. Disparities in educational access contribute to risk differences, particularly in historically underserved populations. Cognitively demanding jobs and leisure activities could reduce dementia risk by 20-40%. Cognitive training may offer benefits, but evidence from multi-domain interventions, such as the FINGER trial, which combined cognitive training with exercise, dietary guidance, and vascular risk management, suggests that a comprehensive approach may be more effective than any single component alone. Future research should refine person- and activity-specific neuroprotective factors. Public health efforts must ensure equitable education, lifelong cognitive engagement, and supportive occupational and leisure opportunities.

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TRAUMATIC BRAIN INJURY AND COGNITIVE DECLINE

WHAT IS ALREADY KNOWN

Each year, there are an estimated 2.5 to 4 million cases of traumatic brain injury (TBI) in the United States. TBI is a disruption in normal brain function caused by a bump, blow, or jolt to the head, or a penetrating injury. These injuries are most common in the youngest and oldest age groups. TBI can raise the risk of dementia in two ways: directly, by making the brain more vulnerable after injury, and indirectly, by increasing the chances of developing other health problems linked to cognitive decline, such as heart disease, epilepsy, and mental health disorders. To lower the risk of TBI and its long-term effects, public health strategies that focus on both preventing injuries and managing chronic health conditions are needed.

The type of injury varies by age. Falls are the leading cause of emergency department visits for children aged 0 to 4 years and for adults aged 65 years and older. For older children and adolescents, being struck by or against an object is the leading cause of TBI, while for older adolescents to middle-aged adults, injuries are related to sports, motor vehicle accidents, or military service.

BACKGROUND AND EVIDENCE BASE

Evidence of an association between TBI and cognitive decline

Acute cognitive effects of TBI typically involve reductions in executive function, processing speed, and learning and memory. The degree to which cognitive recovery in these domains occurs varies, depending on injury severity and other factors (e.g., age, injury characteristics such as diffuse axonal injury or focal intracranial bleed), though recovery is expected in mild TBI (mTBI). Older adults who experience injuries are less likely to fully recover. While cognition can return to preinjury levels after a single TBI, depending on injury severity, risk for dementia may be increased later in life. The associations between TBI and specific dementia subtypes or neuropathology vary, but TBIs occurring in older adults can increase the overall risk of dementia.

The mechanisms underlying the relationship between TBI and later-life cognitive decline are not well understood. TBI is thought to increase risk for cognitive decline later in life through both direct and indirect pathways. These include chronic neuroimmune activation, structural injury that results in cumulative neuronal and cellular dysfunction, and the disruption of the blood-brain barrier as a result of the injury. Polypathology is common at autopsy, particularly in those with moderate to severe TBI, which supports the notion that dementia risk occurs

through multiple processes. Initial studies of biomarkers to identify those at risk for dementia following TBI indicate UCH-L1, ptau181, GFAP, and NfL may have utility in measuring chronic structural brain changes and correlate with cognitive function several years following TBI. However, more research is needed to determine optimal combination and timing of biomarker measurement to assess ongoing chronic brain injury processes.

Genetic vulnerability may also be a key factor. Polymorphism of the APOE gene has received attention in this field, though the evidence is mixed regarding the ε4 allele and long-term outcomes after TBI beyond its recognized contribution to dementia risk. Genome-wide association studies of TBI are limited, though one investigation identified 15 loci, the strongest being NCAM1, APOE, FTO, and FOXP2.

In addition to increased dementia risk due to direct changes associated with TBI, growing evidence suggests that the link between TBI and dementia later in life occurs through indirect pathways as well. Specifically, multiple cross-sectional and longitudinal cohort studies have reported increased rates of chronic conditions that are identified risk factors for dementia, such as cardiovascular/cardiometabolic disease, stroke, seizures, endocrine dysfunction, and psychiatric disorders. Independent of TBI, these comorbid conditions may be risk factors for dementia in the general population. Among those with moderate to severe TBI, greater rates of these comorbid conditions are associated with decline in functional motor and cognitive function over time.

Potential causes of traumatic brain injuries

TBI most commonly results from direct impacts to the head, such as those sustained in motor vehicle accidents, sports, falls, or assaults. These injuries can occur in everyday situations, such as not wearing a helmet while biking or not using a seatbelt in a car. These direct causes are well-recognized and have evidence-based prevention strategies. It is also important to understand social and health-related factors that increase the risk of sustaining a TBI. The following sections explore how adverse childhood experiences (ACEs), violence, and falls contribute to TBI risk, particularly in vulnerable populations.

ACEs: ACEs are potentially traumatic events that a child witnesses or experiences and can include abuse (emotional, physical, or sexual) and household challenges (such as intimate partner violence, substance abuse, mental illness, separation/divorce, and incarceration of a family member). Up to 61% of adults have reported at least 1 type of ACE, and 1 in 6 have reported experiencing four or more types. Women and individuals of racial/ethnic minority groups have a greater risk of experiencing four or more types of ACEs. ACEs can disrupt neurodevelopment and affect health and well-being throughout the lifespan. Health outcomes associated with ACEs, such as homelessness, substance abuse, and violence, can increase a person's risk of experiencing TBI and thereby increase their risk of cognitive decline.

Violence: TBI may result from violence. Abusive head trauma is a leading cause of child abuse deaths in children under the age of five. Domestic, elderly, and intimate partner violence involves unique effects of repetitive or cumulative injury, as well as brain injury effects due to non-fatal strangulation. Survivors of violence are more likely to report ongoing cognitive, physical, and emotional symptoms long-term, and TBI may be underdiagnosed among these survivors because these symptoms can mimic those associated with other mental health or comorbid conditions.

Falls: At least one in four individuals aged 65+ are experiencing falls. Longer recovery among older adults can complicate persistent cognitive sequelae following TBI from cognitive decline associated with pathological aging. Additionally, about 10% to 25% of falls in this population cause fractures, with hip fractures being the most common. Those who experience falls are two to three times more likely to sustain a second fall. Individuals in this age group are likelier to fall indoors than outdoors, and the incidence of falls is even higher among certain populations, such as older adults living in institutions, those recovering from a stroke, and those with diabetes or Parkinson's disease.

IMPLICATIONS FOR PUBLIC HEALTH

Preventing TBI and its long-term effects can be achieved by targeting the circumstances that can precipitate a TBI event, as noted above. Such prevention should be encouraged to preserve brain health in addition to overall general health. Targets must include all ages, relationships and families, and at-risk communities. Social-ecological models and approaches are needed for prevention to be effective in the public health space. Addressing risk factors associated with TBI can have a large and compounding effect on preventing cognitive impairment in older adulthood. These risk factors include falls, violence/abuse, risk taking behaviors, and multifaceted environmental and relational contributions to ACEs. Additionally, continued monitoring and ongoing care directed toward reducing higher rates of chronic conditions associated with dementia risk beyond the post-TBI recovery period is critical.

The role of social determinants of health

The influence of social determinants of health (SDOH) lies at the intersection of poor outcomes following TBI and overall increased risk for dementia. For example, factors such as lower education, lack of health insurance, and lower income are associated with worse cognitive outcomes at one-year post-injury following mTBI. Additionally, living in a disadvantaged neighborhood is linked to more severe and persistent symptoms both shortly after the injury and up to six months later. These social factors have also been identified as independent risk factors for dementia, even among individuals without a history of TBI, after accounting for demographic and background variables. Ultimately, SDOH represents key underlying contributors to the association between TBI, and dementia risk given their position at the intersection of poor TBI outcome following injury and increased dementia risk.

TRAUMATIC BRAIN INJURY AND COGNITIVE DECLINE

DISCUSSION

Interventions to reduce the risk of cognitive decline associated with TBI should focus on all age groups, with interventions targeted at the leading causes of TBI by age group. To prevent injuries from accidents in children, playground surfaces should be safe, soft, and composed of appropriate materials. Children should ride in appropriate car seats and booster seats. Adults and children of all ages should wear seatbelts and use helmets and other safety gear for recreational activities. The safety gear should be well maintained, age-appropriate, and worn consistently and correctly. In older adults, multidimensional strategies, such as assistive devices and environmental modifications, should be implemented to prevent and reduce falls. By implementing multi-level public health interventions tailored to different age groups, we can significantly reduce the incidence of TBI and promote better brain health across the lifespan.

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HYPERTENSION AND COGNITION

WHAT IS ALREADY KNOWN

High blood pressure is strongly linked to poorer brain health, including increased risk for cognitive decline and dementia. Up to 80% of individuals with Alzheimer's disease show evidence of vascular damage in the brain, such as small vessel disease, microinfarcts, or blood-brain barrier disruption, for which hypertension is a leading risk factor. Over 50% of adults have hypertension by age 50. The evidence for hypertension's role in cognitive decline is consistent across cultures and races and spans the translational spectrum from basic biology to epidemiologic and clinical trial science.

BACKGROUND AND EVIDENCE BASE

Evidence from population studies

There is substantial evidence that chronic hypertension over the lifespan is the most prevalent risk factor for cognitive impairment in aging, with midlife hypertension strongly associated with later cognitive deficits.

- Midlife vascular factors, particularly hypertension, were associated with 25-year incident dementia in the diverse Atherosclerosis Risk in Communities (ARIC) observational cohort.
- The AGES-Rejyavik Study documented the joint importance of mid- and late-life blood pressure on subsequent cognitive decline.
- The Harvard Aging Brain Study reported interactive associations among vascular risk, beta-amyloid burden, and cognitive decline in clinically normal older adults.
- In a British birth cohort study, Insight 46, vascular risk across adulthood was associated with late-life brain pathology.
- In the Coronary Artery Risk Development in Young Adults (CARDIA) study, cumulative blood pressure exposure over time, beginning in young adulthood, was associated with mobility and cognitive function in midlife.
- Most recently, a study used UK Biobank data from more than 200,000 participants to determine the relative causal contributions to dementia of individual biological and lifestyle factors that tend to cluster together in midlife. The results showed that, of the biological factors (i.e., systolic blood pressure, LDL cholesterol level, and hemoglobin A1c level), only systolic blood pressure in midlife was an independent predictor of incident dementia later in life.

Biological evidence

Cerebrovascular disease, a condition affecting the blood vessels in the brain and a complication of hypertension, is a major contributor to cognitive decline and dementia. Vascular brain changes frequently co-occur with Alzheimer's disease–related pathology, including ß-amyloid and tau accumulation. Hypertension is the primary risk factor for small-vessel ischemic disease, a type of cerebrovascular disease, and cortical white matter abnormalities in the brain, both of which are highly predictive of cognitive decline and dementia. Observational studies and clinical trials suggest that better control of hypertension reduces risk for Alzheimer's and other dementias, with the strongest association for blood pressure (BP) lowering in middle age. These data support intensive BP treatment (target systolic blood pressure <130 mm Hg) as an important strategy for the prevention of cognitive impairment and suggest that benefits of intensive BP control on prevention of cognitive impairment continue even with only a few years of intensive treatment.

There is evidence for a synergistic interaction between the accumulation of Aß and vascular damage in the brain. However, it is unclear whether vascular decline precedes amyloid accumulation or vice versa.

Another mechanism that could explain the link between hypertension, vascular health, and cognitive health is arterial stiffness. The brain, heart, and kidneys are especially vulnerable to arterial stiffness, and all three organs show age-related changes in physiology associated with organ system failure. Arterial stiffness increases

pulse wave velocity, and the increased transmission of a larger forward wave may expose fragile peripheral small arteries and micro-vessels to damaging levels of pulsatility, particularly in the brain. Such damage may contribute to the microvascular disorders that are common in aging in these organs.

Evidence supporting hypertension treatment to reduce the risk of dementia

The most recent evidence that reduction of a cardiovascular (CVD) risk factor could minimize the risk for clinically significant cognitive decline comes from the Systolic Blood Pressure Intervention Trial–Memory and Cognition in Decreased Hypertension (SPRINT-MIND). Participants were randomized to either a standard blood pressure management goal (systolic blood pressure <140 mm Hg) or to an intensive blood pressure management goal (systolic blood pressure <120 mm Hg). After only 3.2 years of treatment, participants assigned to the lower systolic blood pressure goal had a 19% lower risk for developing mild cognitive impairment (MCI), a precursor stage of dementia, compared with those assigned to the systolic blood pressure goal of <140 mm Hg. Participants also had a 17% lower risk for developing dementia, but this reduction was not statistically significant as fewer cases of dementia than of MCI developed during the follow-up period. Participants assigned to the lower blood pressure goal also had reduced development of abnormal white matter lesions in their brains, indicating a possible mechanism for the observed preservation of cognitive function. This statistically positive impact on reducing the risk for cognitive impairment or dementia persisted for at least 7 years and benefited people in their 80s. Finally, a large study in China, the China Rural Hypertension Control Project, showed a 15% reduction in dementia for those treated to a systolic blood pressure goal of 120 mm Hg for 48 months.

IMPLICATIONS FOR PUBLIC HEALTH

There is a vascular component in most cases of dementia, with increasing evidence that hypertension in mid- and later life is strongly associated with dementia and should thus be a focus of prevention strategies. Public health efforts should reach populations with the highest rates of uncontrolled hypertension: older adults, especially those living in poverty, Black Americans, and American Indian and Alaska Native people. There is substantial epidemiologic evidence for a link between long term hypertension and dementia incidence, and therefore an additional population of focus for prevention should be middle-aged adults.

Nearly six million people in the United States aged 75 years or older met the criteria for inclusion in the SPRINT trial. This age group is rapidly growing and at the greatest risk for cognitive decline and dementia and would therefore be a prominent subgroup to target for meeting blood pressure goals as a way to reduce the risk of cognitive dysfunction quickly and safely.

The role of social determinants of health

Barriers to accessing healthy foods and safe environments for exercise can further exacerbate hypertension and other vascular risk factors for dementia. Diet and poor access to healthy dietary components have been linked to both dementia and hypertension. Access to healthy foods including vegetables, leafy greens, berries, whole-grain, and fish is important to reducing risk for hypertension and its complications.

Finally, a person's living environment substantially influences their risk for development of hypertension and their ability to manage it. Air pollution and limited access to safe outdoor spaces for physical activity disproportionately affect communities with lower socioeconomic status, many of which have high prevalence of Black, Hispanic, and other people of color.

HYPERTENSION AND COGNITION

DISCUSSION

Evidence shows that vascular health and cognitive health are closely related. Chronic hypertension is the most prevalent risk factor for cognitive impairment in aging, and the incidence of high blood pressure increases with age; by age 75, up to 80% of individuals will have been diagnosed with and/or treated for hypertension. Prevention is always the most effective way to change the course of a disease, and the scientific evidence is strong for treating hypertension as an effective means to reduce the risk of cognitive dysfunction in older age. Importantly, there are many existing treatments for doing so. However, despite the wide availability of hypertension treatments, access to those treatments is a major concern for underrepresented populations, many of which have higher rates of hypertension and dementia. Public health programs should reach these populations with hypertension prevention and treatment strategies to reduce the risk of, or delay the development of, cognitive decline in later life.

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The Public Health Center of Excellence on Dementia Risk Reduction is made possible by the Centers for Disease Control and Prevention of the U.S. Department of Health and Human Services as part of a financial assistance award totaling $3,707,435, with 100 percent funded by CDC/HHS. The contents of this document are those of the Alzheimer's Association and do not necessarily represent the official views of, nor an endorsement by, CDC/HHS or the U.S. Government.

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DIABETES, OBESITY AND COGNITION

WHAT IS ALREADY KNOWN

For the past 60 years, rates of obesity and diabetes have been rising rapidly in the United States. The Centers for Disease Control and Prevention estimates that more than 40% of the U.S. population is obese, and more than 10% of the total population has diabetes. These conditions often co-occur, and both increase risks for cognitive decline and dementia and other health problems such as heart disease and disability.

BACKGROUND AND EVIDENCE BASE

Evidence of an association between obesity, diabetes and cognitive impairment

The adverse consequences of obesity begin early, placing children at increased risk for obesity and Type 2 Diabetes Mellitus (T2DM) as adults. Mid-life obesity and T2DM result in increased risk for later-life dementia. Obesity is strongly linked to insulin resistance and elevated blood glucose, which in turn affect cognition and brain structure even before the onset of T2DM. Obesity and increased insulin resistance are also linked to poorer control of T2DM in later life, which may accelerate risks of cognitive decline and dementia.

There are many pathways through which obesity and T2DM adversely affect brain health, and many of these are driven by hormones associated with inflammation and metabolism that are secreted by adipose tissue (fat cells) and the pancreas.

Both obesity and T2DM can disrupt energy metabolism in the brain, which in turn may lead to accelerated cognitive decline. They may also increase risks for anxiety and depression, which may affect cognition and increase risk for dementia.

Evidence of prevention and treatment

The National Academies concluded that prevention of obesity and T2DM is a promising pathway to reduce the risk of cognitive decline and dementia. The most effective approach may involve adopting healthier lifestyles, including increased physical activity and balanced diets that are lower in calories and processed foods.

Treatment in mid-life for persons already experiencing obesity is possible through interventions promoting weight loss and increased physical activity and holds promise for reducing the increased risks for dementia that obesity conveys; however, the evidence that obesity treatment reduces risks is less strong than the evidence for prevention. In later life, there is even less evidence that treatment of obesity is effective in reducing risks for dementia. Rapid decline in weight during late life may be a signal of early dementia, particularly among individuals who do not currently have obesity.

Recently, there has been considerable interest in the potential for treating obesity with newer classes of weight loss medications to reduce dementia risk. While these medications may eventually prove effective, they do not diminish the importance of lifestyle-based approaches. Even if pharmacological treatments are shown to reduce risk, they are likely to be most effective when combined with sustained changes in diet and physical activity.

Together, current evidence suggests that preventing obesity, particularly at younger ages, may be more promising for reducing the risk of dementia later in life than treating it after onset. While some large-scale interventions, such as the Look AHEAD study, have shown benefits for metabolic health, their impact on cognitive outcomes remains unclear.

Better control of T2DM is often associated with better cognitive functioning. There are some reports from observational studies that treatment of T2DM through medical management reduces the risks it conveys for dementia; however, there is less evidence for this from clinical trials. There is also less evidence that treatment of T2DM with lifestyle changes reduces risks for dementia.

IMPLICATIONS FOR PUBLIC HEALTH

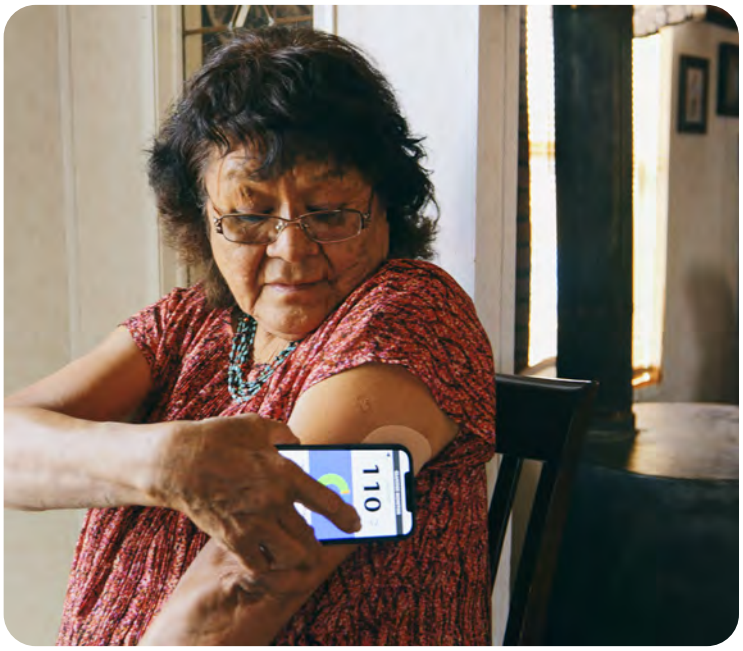
Providing communities with greater awareness of the need to intervene and prevent obesity through education and community-based programs promoting better diet and greater physical activity is likely to result in long-term preservation of memory and other cognitive functions. Greater efforts to target and carry out tailored interventions among those at higher risk for future cognitive problems – namely, populations with a higher burden of unrecognized or untreated obesity and/or T2DM – could yield benefits in terms of reduced burden of cognitive impairment and dementia across communities as a whole.

The role of social determinants of health

Characteristics of one's neighborhood may influence risks for obesity and T2DM. For example, greater exposure to air pollution increases these risks, as does residing in economically disadvantaged neighborhoods. Lack of access to healthy foods also increases risks, in part through the potential that this leads to inadequate nutrition. Living in neighborhoods with fewer green spaces and access to recreational facilities may also increase risks as it limits opportunities to engage in physical activity. Inadequate sleep also increases risks, which may be a product of environmental noise and living spaces that compromise sleep quality.

Obesity and T2DM are consistently associated with increased risk for cognitive decline and dementia across all populations. In the United States, some groups, particularly Black and Hispanic individuals, have higher rates of these conditions, which may contribute to disparities in dementia prevalence.

In the United States, obesity tends to be more prevalent among women while T2DM tends to strike men earlier in life. These differences are partly driven by biology, but there are also strong components related to social and cultural aspects such as lifestyles, stressors, exposures, and attitudes about health and health care. There is some evidence that men may be more susceptible to relatively greater increased risks for dementia that obesity and T2DM convey than women; however, it is clear that they adversely affect both sexes.



DISCUSSION

Opportunities to prevent obesity and T2DM, and thereby reduce the risk of later cognitive problems, have primarily targeted children and young to middle-aged adults. These include programs focused on behavior change in children, especially through parental counseling and school-based initiatives, as well as individual-, group-, and community-based strategies for adults that promote physical activity and healthier diets.

While obesity and T2DM often co-occur and may interact to increase dementia risk, each condition also has distinct effects on brain health. Obesity may impair vascular function and brain structure, while T2DM can disrupt insulin signaling and glucose metabolism in the brain. As such, prevention and treatment strategies for T2DM, such as improved glycemic control, may offer cognitive benefits independent of weight loss.

For these approaches to be successful, it is critical to use broad based population level public health interventions while ensuring these approaches are tailored to the needs of individuals and communities.

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EXERCISE AND ITS EFFECT ON COGNITION

WHAT IS ALREADY KNOWN

The United States Department of Health and Human Services recommends that all adults get at least 150 minutes of moderate-intensity exercise or 75 minutes of vigorous-intensity exercise, or an equivalent combination, per week to maintain overall health and function, and reduce the risk of a number of chronic diseases. It has become clear that high-intensity aerobic exercise improves not just the function of the body but also function of the brain. The World Health Organization included physical activity as a top priority in its review of 12 nonpharmacological interventions with the potential to reduce the risk of cognitive decline and dementia. Regular exercise as a means to enhance or protect cognitive health is gaining momentum not just among health policy experts but also in messaging to the public.

BACKGROUND AND EVIDENCE BASE

More than three decades of research in both animal and human studies provides strong support that aerobic exercise benefits the brain. Exercise is associated with increased neurorepair, increased clearance of hyperphosphorylated tau, improved glucose metabolism, reduced inflammation, reduced ß-amyloid plaques in the brain, and reduced oxidative stress. In rodents, aerobic exercise leads to hippocampal neurogenesis (a key brain structure for memory), increased levels of brain-derived neurotrophic factor, improved endothelial function and blood flow, slowing of atrophy, physiological stress reduction, and improved cognition. Observational studies in humans report that aerobic exercise results in improved cognitive function and a reduced risk of cognitive decline and Alzheimer's disease, and it has positive effects on brain volume and Alzheimer's disease biomarkers.

A growing number of clinical trials provide evidence that aerobic exercise increases brain volumes and blood flow to the brain and improves cognitive functions. A recent meta-analysis of 19 studies that examined the effects of physical activity on mild cognitive impairment in older adults at risk of developing dementia (17 of which were randomized controlled trials) concluded that exercise improves cognition. The effect size was 1.3-fold higher when the analysis examined only the effects of aerobic exercise (excluding light physical activity). Thus it is clear that interventions involving high-intensity aerobic exercise substantially benefit cognitive health; however, both moderate-level and lower-intensity exercise may slow cognitive decline among older adults with mild cognitive impairment.

Some studies, such as the recently reported Exercise in Adults with Mild Memory Problems (EXERT) trial, show that even among once-sedentary adults with mild cognitive impairment exercise interventions can be feasibly and sustainably delivered in the community and by community partners even among once-sedentary adults with mild cognitive impairment. One important lesson learned is that older adults must have ongoing support and guidance as they carry out a prescribed exercise program. The EXERT trial's study success can likely be attributed to the trainer support that participants received on a weekly basis throughout their participation in the trial. This support is especially important for older adults who are not regular exercisers before starting an exercise program, and for older adults with mild cognitive impairment who consequently face daily challenges. Without support, such interventions that are designed to protect cognitive function in older adults will not likely succeed.

Combining exercise with other lifestyle behaviors such as balanced nutrition, cognitive stimulation, and monitoring and managing clinical markers such as blood pressure and blood glucose may enhance cognitive benefits and promote overall brain health. Increased physical activity was included in the Alzheimer's Association U.S. POINTER clinical trial as a component of two multidomain lifestyle interventions that also aimed to enhance nutrition, cognitive and social challenge, and health monitoring. The two interventions differed in structure, intensity, and accountability. Both interventions

resulted in clinically significant improvements in cognitive test scores, with greater improvements among participants receiving the more intensive intervention.

IMPLICATIONS FOR PUBLIC HEALTH

Growing evidence for a relationship between physical activity and long-term preservation of cognitive function underscores the importance of efforts to promote exercise, including creation of and participation in structured community exercise programs. Assessment and implementation of new avenues for exercise participation in diverse communities and across all age groups will strengthen the message that exercise is a helpful pathway for reducing the burden of cognitive decline and dementia. However, the success of intervention strategies will depend on access and sustainability of delivery within the community.

The role of social determinants of health

There are numerous social and cultural factors that may impact an individual's ability to engage in physical activity. Individuals with higher social economic status are more likely to be physically active than others, possibly as a result of having greater access to exercise facilities and safe spaces to exercise. Women are less likely to engage in regular physical activity than men, which may be attributable to cultural norms, gender roles, and lack of social support. Overall, a broad range of societal factors may contribute to differences in physical activity, including one's residence, race and ethnicity, occupation, gender, religion, education, social capital, and local governmental policies. Collectively, these offer many opportunities to decrease barriers to engaging in physical activity and to increase exercise levels to reduce risks of cognitive decline.

DISCUSSION

Despite the generally positive research findings that exercise improves cognitive function in both healthy adults and adults with varying stages of cognitive decline, results are not always consistent. This inconsistency is likely related to differences in study design, such as the use of supervised versus unsupervised home-based exercise interventions, and differences in the intensity, weekly frequency, and overall duration of interventions. Health-restoring effects of exercise take time; trials with interventions lasting fewer than six months rarely show cognitive benefits. Longer trials more commonly show benefits on executive function, but those with interventions lasting fewer than 12 months rarely show benefits on memory. Thus, it can be concluded that interventions need to be of an appropriate intensity and an appropriate duration for individuals to experience benefits. Research continues on the exact amount, frequency, and duration to provide the best benefit for brain health.

A long-term commitment to an intense exercise regimen may cause some individuals to wonder whether the potential benefits are worth the challenges and discomfort that can be associated with beginning and adhering to such an exercise program. Thus, public health interventions need to provide ongoing support (coaching) to enable participants to continue exercising at appropriate levels of intensity for an appropriate period of time so that they can experience the full therapeutic benefit. There is a need for sustainable community-based programs that can be delivered by the community, using community-based infrastructure and resources. Health care providers should also be incentivized to promote "exercise as medicine" interventions to their patients, and to provide appropriate referrals to evidence-based community programs that can properly and effectively assist and support the patient. Public health messaging could also support a step-wise approach to exercise – starting small and gradually increasing intensity and frequency over time. Additional challenges with respect to recommendations arise because many trials have historically enrolled populations that are not demographically representative of the overall population, and studies are time and resource limited resulting in programs ending when the funding ends.

EXERCISE AND ITS EFFECT ON COGNITION

In conclusion, there is increasing evidence that physical exercise, and aerobic training in particular, has favorable effects on multiple health outcomes, including protection against cognitive decline and the development of Alzheimer's disease. Although the success of intervention strategies will depend on the ultimate sustainability of their delivery within the community, it is clear that adults should be encouraged to participate in vigorous aerobic activity in line with the United States Department of Health and Human Services guidelines.

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SMOKING AND COGNITIVE DECLINE

WHAT IS ALREADY KNOWN

Smoking and exposure to cigarette smoke increase the risk of cognitive decline, cognitive impairment, and dementia including Alzheimer's disease. This occurs at all stages of life. Studies have suggested heavy smoking in middle age may increase one's risk of dementia later in life by up to 50%. Lifetime exposure to cigarette smoke, even among non-smokers, also may adversely affect cognitive function. Use of oral tobacco products has also been associated with increased risks for cognitive impairment. Maternal smoking exposes the fetus to many toxins that may harm development and lead to lower cognitive functioning that may extend late into adulthood. Children and adolescents who begin smoking at or before age 15 may be at particularly greater risk for cognitive decline and loss of brain tissue later in life compared with those who begin smoking after age 15. Studies on vaping – a relatively new phenomenon – continue, but there are some preliminary indications that vaping may adversely affect brain health and increase risk for cognitive deficits.

BACKGROUND AND EVIDENCE BASE

Evidence of negative health effects of cigarette smoking on the brain

There are more than 7,000 chemicals in cigarette smoke, mostly produced by the combustion of tobacco. Many of these chemicals are toxic and/or carcinogenic. Of all the ingredients in cigarettes, nicotine is the primary cause of addiction. Nicotine is present in non-combustible products such as electronic cigarettes, heat-not-burn products, and oral pouches, which are all increasingly popular. While non-combustible nicotine products may be safer than traditional cigarettes, the long-term use of these products on health is uncertain because they are relatively new. In addition to its addictive properties, research suggests nicotine partially contributes to the negative health effects attributed to cigarette smoke.

Long-term exposure to cigarette smoke has negative effects throughout the body, but the brain and cerebrovascular system are particularly at risk. Smoking is associated with accelerated rates of brain tissue loss later in life. This is evident in a reduced volume of brain gray matter, where neuron cell bodies are located, and in degraded white matter tracts, which form communication pathways between brain regions. Cigarette smoke also increases stiffness in the small blood vessels of the brain, which contributes to a reduction of cerebral blood flow.

While these effects on the brain and cerebrovasculature can be directly measured, they are also indirectly evident in worsened cognitive function among people who smoke. In particular, long-term smoking reduces cognitive flexibility, attention, and short- and long-term memory. Nicotine can improve mood and cognitive function, but with long-term use, people who smoke become tolerant to these effects and experience withdrawal symptoms that worsen mood and cognitive function when they abstain from nicotine-containing products.

The effects of cigarette smoking on other parts of the body can indirectly worsen brain health. For instance, cigarette smoke worsens cardiovascular health by increasing blood pressure and decreasing heart rate variability, which in turn negatively affects brain tissue, cerebral blood flow, and cognitive function. The negative health effects of smoking tend to be associated with the heaviness and duration of use. However, an abundance of research has shown that smoking cessation improves brain and cerebrovascular health. Better health is predicted by the number of years an ex-smoker has quit. Smoking cessation may be most effective among younger individuals but appears to produce meaningful benefits at any age and regardless of the heaviness and duration of smoking.

Evidence of an association between cigarette use and dementia

Cigarette smoke accelerates brain aging and promotes the development of Alzheimer's disease and cerebrovascular disease. Exposure to both first-hand and second-hand cigarette smoke is associated with risk

for Alzheimer's. The general negative effects of cigarette smoke on brain structure and function may accelerate the underlying mechanisms of Alzheimer's disease.

Alzheimer's pathology is associated with the accumulation of amyloid plaques and neurofibrillary tangles in brain regions modulating the cholinergic system. These processes ultimately reduce the release of the neurotransmitter acetylcholine and reduce the number of nicotinic acetylcholine receptors. This results in the loss of cognitive function as well as balance and movement. Nicotine mimics the effects of acetylcholine. Thus, some studies have suggested that short-term use of nicotine may be helpful in restoring cognitive function in people with Alzheimer's, and some researchers have suggested that nicotine could have protective effects against Alzheimer's. However, the relationship between long-term nicotine use and Alzheimer's is confounded by the fact that nicotine is overwhelmingly delivered in cigarette smoke.

Other chemicals in cigarette smoke contribute to Alzheimer's and other types of dementia, such as neurotoxic heavy metals that increase plaque formation in the brain, and oxidizing stress agents that increase inflammation and lead to brain cell death. Oxidative stress also damages the blood brain barrier and impairs cerebral vasodilation, leading to cerebrovascular dysfunction, less cerebral blood flow, and neurodegeneration. By these and other mechanisms, cigarette smoking is associated with increased risk of microbleeds and strokes. Electronic cigarettes contain many of these same chemicals, albeit at lower levels than cigarette smoke. However, the association between electronic cigarette use and dementia is unknown, as the majority of electronic cigarette users are under the age of 45 years.

Harms associated with lifetime exposure to cigarette smoke

Environmental exposure to cigarette smoke adversely affects cognitive abilities and increases risks for dementia. Children may be especially sensitive as there is an inverse association between environmental cigarette smoke exposure and cognitive deficits among children even at low levels of exposure, although the cognitive development of infants is negatively related to the heaviness of their mothers' smoking. Among children and adolescents, smoking may inhibit the brain's development, compromise important cognitive functions, and result in smaller brain volumes.

IMPLICATIONS FOR PUBLIC HEALTH

The heaviness and duration of cigarette smoking is associated with a higher risk of Alzheimer's, cognitive impairment, and dementia. There is compelling evidence that long-term smoking cessation can reverse a person's risk of developing cognitive decline. When compared with continual smokers, long-term quitters and never smokers have a decreased risk of both overall dementia and vascular dementia. Indeed, stopping smoking may be one of the best ways to reduce the risk of dementia and cognitive impairment in later life. However, smoking cessation is difficult, and less than 1 in 10 smokers successfully quit each year. Programs to prevent children and adolescents from starting to smoke may be most beneficial. The earlier in life a person begins smoking or using other nicotine products, the more likely they are to develop daily use and symptoms of dependence.

There is growing evidence that vaping and the use of oral tobacco products, especially when initiated at younger ages, are associated with impairments in memory, attention, and executive function. While long-term studies are still ongoing, early findings suggest these behaviors may contribute to cognitive challenges that could persist or worsen over time. Preventing initiation and promoting cessation remain important public health goals.

The role of social determinants of health

There are many social and cultural factors associated with the use of tobacco products. It is well-known that smoking is generally more prevalent among men than women in the United States. It is also more prevalent among individuals with less formal education and fewer economic resources. Smoking is more common among those living in rural communities and in the southern United States. Peer pressure may be a motivating factor for the initiation of smoking. It is also often linked to behaviors such as alcohol use and sedentarism and may be higher among those suffering from depression. Exposure to environmental smoke is increased by parental smoking and influenced by peer groups. It is prevalent among occupations in which coworkers or clients are more often smokers.

SMOKING AND COGNITIVE DECLINE

DISCUSSION

The evidence for a negative impact of smoking on cognitive health, both among those who smoke cigarettes and among those with environmental exposure to cigarette smoke, is strong. People at any age who smoke, and especially those with young children in their household, should be encouraged to stop and be provided with smoking cessation interventions. Smoking cessation provides many health benefits in addition to those for cognitive health.

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SLEEP AND COGNITION

WHAT IS ALREADY KNOWN

As people age, sleep disturbances increase and sleep quality decreases. Disruptions in either sleep quality or circadian rhythm can have significant health implications. Just over half (54%) of older adults report that they sometimes or most of the time wake too early without being able to fall back to sleep, and just under half (44%) report that they rarely or never sleep through the night without waking for more than a few minutes. People with advanced dementia experience increased sundowning, fragmented nighttime sleep, changes in the sleep cycle and REM sleep, increased napping, and a high prevalence of sleep apnea. The relationship between such sleep disturbances and cognitive decline is now understood to be bidirectional, with disturbed sleep having a causal relationship with neurodegenerative disease and neurodegeneration leading to an increase in sleep disturbances.

BACKGROUND AND EVIDENCE BASE

A bidirectional association between disturbed sleep and cognitive decline is supported by evidence from several studies. In a study of women with normal cognitive function, cognitive decline over a 15-year period was associated with subsequent poor sleep quality (measured as poor sleep efficiency, taking longer to fall asleep, and waking up after falling asleep). Another study reported an association between current poor sleep quality and subsequent clinically significant cognitive decline, after adjustment for potential confounders (e.g., demographics, lifestyle factors, comorbidities, and medication use). Less efficient sleep and taking longer to fall asleep were associated with an increased risk of mild cognitive impairment and dementia. A study of adults without current dementia but at genetic risk for developing Alzheimer's disease showed that sleep duration was significantly reduced (by 1.9 hours) in people with a genetic predisposition to Alzheimer's disease, suggesting that sleep duration might be useful as a marker for future cognitive decline in this group. Another study found that sleep duration of six hours or less at age 50 or 60 was more strongly associated with the subsequent development of dementia than was sleep of six hours or less at age 70. Short sleep at age 60 was the most strongly linked to subsequent dementia risk, suggesting it may be particularly harmful to have short sleep duration around age 60.

Findings from animal studies suggest a potential mechanism for explaining the relationship between sleep and cognitive function. Two studies in mice suggested that sleep helps prevent the accumulation of harmful substances (such as amyloid-β, a peptide found in the brains of people with Alzheimer's disease). Sleep deprivation resulted in significant accumulation of amyloid-β in one study, and another study showed that sleeping mice cleared twice as much amyloid-β from their brains as conscious mice did through the *glymphatic system*, a brain waste-removal system that is believed to be most active during slow-wave (deep) sleep. Together, findings suggest that when sleep is poor, there is either greater production or reduced clearance of the toxins associated with Alzheimer's disease.

Studies in humans have linked shorter sleep duration and poorer sleep quality to greater amyloid-β deposition. Alterations in sleep architecture, such as fragmented slow-wave (deep) sleep and reductions in sleep spindles also have been linked to Alzheimer's disease pathology and poorer cognitive performance. In addition, idiopathic rapid eye movement sleep behavior disorder (iREM-SBD), a condition in which people commonly act out their dreams while asleep, is a prodromal marker of Lewy body dementia and Parkinson's disease, highlighting the involvement of sleep in the context of neurodegenerative conditions beyond Alzheimer's disease.

In addition to impaired sleep at night, napping during the day is also common in older adults. The relationship between napping and cognitive function is complex. Overall, older people who reported more frequent napping and taking naps longer than two hours in duration demonstrated a significantly greater risk of cognitive decline compared with those who napped less frequently. However, when napping was considered in the context of recent sleep quality at night (measured by sleep duration and sleep efficiency), it was found that napping during the day after a person experienced a poor night's sleep was not associated with this increased risk of cognitive decline; after adjusting for possible confounding variables, this risk appeared to be greater only for those who napped during the day after experiencing good sleep quality at night.

Changes in circadian rhythm (mental and physical behaviors over a 24-hour period associated with the light-dark cycle) have also been found to be related to cognitive decline and dementia risk. Sleep-disordered breathing (e.g., sleep apnea) increases with age and is present in about 25% of older adults by 75 years of age. It is associated with an increased risk of dementia, possibly by reducing the brain's oxygen supply. Interestingly, it has also been associated with a reduced clearance of amyloid-β in cerebrospinal fluid over a two-year period.

Some studies have explored whether treating sleep disruptions can reduce the risk of cognitive impairment. A study that evaluated the effect of continuous positive airway pressure (CPAP) therapy on cognitive function found no cognitive benefit after six months of CPAP therapy compared with a sham treatment, while another reported cognitive improvements after just three weeks of CPAP use. A small study also showed that CPAP therapy reduced amyloid-β accumulation. Meanwhile, certain insomnia medications, such as benzodiazepines and "Z drugs," have been linked to increased risk of cognitive decline. Zolpidem, a commonly used sleep aid, may interfere with glymphatic clearance during sleep. In contrast, a small study in healthy mostly middle-aged adults found that suvorexant, a dual orexin receptor antagonist, acutely reduced Alzheimer's-related proteins in cerebrospinal fluid. More research studies are needed to better understand how different types of sleep medications affect the brain, both in the short- and long-term.

Sleep disturbances are also associated with an increased risk of developing multiple chronic diseases that affect brain health. For example, longitudinal data from the Swedish National study of Aging and Care in more than 4,000 Swedish individuals aged 60+, showed that moderate to severe sleep disturbances were associated with more rapid development of chronic morbidities, particularly neuropsychiatric and musculoskeletal conditions. Cardiovascular health protects against cerebrovascular disease and stroke, with implications for dementia prevention. Obtaining sleep duration of seven to nine hours is now recognized as a key contributor to cardiovascular health, and increasing attention is being paid to other aspects of sleep (e.g., sleep quality, regularity, timing) in cardiovascular health and disease.

Cognitive-behavioral therapy for insomnia (CBT-i) is a non-pharmacological treatment for insomnia. It is effective across adulthood, and in 2016 the American College of Physicians recommended CBT-i as the first-line treatment for chronic insomnia. To date, however, there is not enough research evidence to evaluate the extent to which CBT-i improves cognitive or brain health outcomes. In general, it is not yet known whether targeting sleep is an effective method for reducing a person's risk of developing cognitive impairment and/or dementia.

IMPLICATIONS FOR PUBLIC HEALTH

It is known that improving sleep has beneficial effects on other health outcomes (including mortality, cardiovascular disease, inflammation, obesity, and others), which can improve overall quality of life. Standard sleep hygiene practices can be recommended for any person wanting to improve his or her sleep. Such practices include day-time exercise; avoiding afternoon caffeine intake and fluid, food, nicotine, and alcohol intake before bed; keeping the bedroom dark and cool; and avoiding using electronics in the bedroom. Additionally, low-cost mobile health technology that is focused on behavioral interventions that can assist people wanting to improve sleep quality is increasingly available. Such technology can monitor sleep remotely and may be included in public health initiatives in the coming years.

The role of social determinants of health

Public health interventions that address root causes of sleep disturbances – such as late-night neighborhood noise levels and economic conditions that require individuals to work multiple jobs – may improve an individual's ability to achieve adequate sleep. Sleep health disparities, driven by social, structural, and environmental inequities, play a critical and often underappreciated role in shaping global dementia risk. These disparities are intersectional, emerging across sex/gender, race/ethnicity, and socioeconomic position. Women experience greater sleep fragmentation and insomnia compared with men, particularly during midlife and the menopausal transition — a critical period also associated with rising risk for Alzheimer's disease and related dementias. In the United States, Black and Hispanic/Latino adults tend to report shorter, poorer quality sleep, and have a higher prevalence of undiagnosed and untreated sleep disorders than non-Hispanic Whites. These differences persist beyond individual behavioral or health profiles, suggesting the impact of broader structural determinants, including limited access to health care, cumulative exposure to psychosocial stressors, and residence in disadvantaged neighborhoods. Ecological factors such as housing quality, urban noise, excessive heat, light pollution, and neighborhood crime may further degrade sleep and disproportionately affect low-income populations.

SLEEP AND COGNITION

DISCUSSION

While the evidence is clear that sleep and cognitive function are interrelated, it is less clear whether treating disordered sleep can reduce a person's risk of cognitive decline. Accurate measurement of sleep quality and quantity requires objective measurement of sleep and cannot rely solely on self-reports. Today, widely available new technology-based tools permit more precise measurement of sleep quality outside of the laboratory setting and may become helpful for identifying individuals who may benefit from specific treatments to improve sleep. Additionally, research addressing underlying problems that contribute to poor sleep (such as sleep apnea, diabetes, and cardiovascular disease) is in its early stages. Furthermore, this research is examining whether improved treatments for the sleep-related disorders associated with these conditions may secondarily reduce the risk for dementia.

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Glossary

Sleep refers to a natural, reversible state of reduced responsiveness and activity.

Sleep quality encompasses factors such as sleep duration, continuity, depth, and how restorative the sleep is.

Circadian rhythm is the body's internal biological clock that regulates the timing of sleep and wakefulness over a 24-hour cycle.

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DIET, NUTRITION AND COGNITION

WHAT IS ALREADY KNOWN

Diet directly and indirectly influences cognitive health. This influence occurs particularly through nutrition, which is the process by which the body takes in and uses food for growth, repair, and maintenance. Key nutrients that support cognitive function include antioxidant vitamins (such as vitamins E, A, and C, as well as carotenoids), bioactive compounds (such as flavonoids and polyphenols), B vitamins, fiber, healthy fats (including omega-3 and monounsaturated fatty acids), and essential minerals.

For overall optimal health, United States Dietary Guidelines and the American Heart Association recommend a dietary pattern centered on plant-based foods such as vegetables, whole fruits, whole grains, nuts and seeds, plant-based sources of protein such as beans and legumes, and unsaturated vegetable oils. The recommendation is also to minimize or avoid consumption of red and processed meats, excess sodium, saturated fats, added sugars, refined grains, highly processed foods, and excess alcohol. This dietary approach is also associated with cognitive health and aligns with popular dietary patterns, such as the Mediterranean -style and Nordic (Scandinavian-style) diets, both of which have demonstrated positive effects on cognitive health. The World Health Organization (WHO) recommends the Mediterranean diet for adults with normal cognition or mild cognitive impairment as part of a strategy to help slow cognitive decline and dementia.

BACKGROUND AND EVIDENCE BASE

Evidence of an association between diet, nutrition and cognitive health

The exact mechanism between diet and cognition is not fully understood; however, these nutrients and bioactive compounds may help maintain neuronal health, support endothelial and mitochondrial function, preserve blood–brain barrier integrity, and modulate anti-inflammatory and oxidative stress pathways, all of which are important for brain health outcomes. Additionally, diet plays a crucial role in managing cardiovascular risk factors — such as diabetes, dyslipidemia, hypertension, and obesity — that are linked to cognitive decline.

Research on diet and cognition is complex due to challenges such as isolating the effects of individual nutrients and noting a person's dietary pattern using detailed validated dietary assessment tools and considering various social, economic, cultural, and geographical influences. Randomized controlled trials (RCTs) on dietary interventions are also difficult to conduct. Most studies in this field are observational, assessing the long-term relationships between nutrition and cognition or change in cognition over time with age.

Recent systematic reviews show that adherence to healthy dietary patterns is associated with better cognitive performance, slower cognitive decline, and reduced dementia risk. In contrast, unhealthy dietary patterns are linked to poorer cognitive outcomes. Further research is needed to better understand the specific effects of diet on dementia development and its different forms. This includes exploring how diet may interact with other lifestyle factors, such as physical activity and sleep. It is also important to incorporate culturally relevant dietary patterns and to study populations across the full spectrum of cognitive function. Future studies should include younger individuals and more ethnically and geographically diverse groups to ensure findings are broadly applicable.

Evidence for an association between single nutrients, food groups and cognition

Recent reviews have evaluated the impact of individual nutrients and food groups on cognitive health. While studies on isolated nutrients often yield mixed results, some evidence supports the beneficial role of B vitamins, vitamin E, omega-3 fatty acids, and other antioxidants including carotenoids, and flavonoids, in protecting cognitive function. However, experts agree that the best approach is to obtain nutrients from whole foods, not supplements.

Research highlights the importance of a nutrient-rich diet, emphasizing whole foods and beverages that support cognitive health. The WHO strongly advises against using supplements as a preventive strategy for cognitive decline, citing insufficient evidence for benefit. While multivitamins may show some promise, they are not as effective as obtaining nutrients directly from food. The Alzheimer's Association similarly does not recommend any vitamin, mineral, herbal supplement, or medical food for the prevention or treatment of Alzheimer's or other dementias, due to a lack of consistent clinical evidence.

Evidence for an association between dietary patterns and cognition

A growing body of evidence shows that balanced eating patterns, rather than focusing on individual nutrients or

foods, are associated with better cognitive health. This highlights the cumulative benefits of adopting a varied, nutrient-dense diet. The USDA's Nutrition Evidence Systematic Review concluded that there is a moderate level evidence for a reduced risk of age-related cognitive decline, mild cognitive impairment, dementia, and/or Alzheimer's disease as a result of dietary patterns with higher intakes of vegetables, fruits, legumes, beans, nuts, fish, seafood, and unsaturated vegetable oils/fats, and lower intakes of red/processed meats, sugar-sweetened beverages, and ultra-processed foods.

Specific diets such as the Mediterranean, DASH (Dietary Approaches to Stop Hypertension), and MIND (Mediterranean-DASH Diet Intervention for Neurodegenerative Delay) diets have been investigated widely in relation to cognition, cognitive decline, dementia risk, brain imaging biomarkers, brain volume and Alzheimer's disease pathology. Each of these diets share a broader dietary pattern that emphasizes the promotion of foods and nutrients discussed above. Based on longitudinal studies among older adults, the MIND and Mediterranean diets have the most compelling evidence. These plant-based dietary patterns may lower systemic inflammatory biomarkers, and neuroinflammation, and help with vascular health. The MIND diet was recently tested in the U.S. POINTER clinical trial as a component of two multidomain lifestyle interventions that also aimed to enhance physical activity, cognitive and social challenge, and health monitoring. The two interventions differed in structure, intensity, and accountability. Both interventions resulted in clinically significant improvements in cognitive test scores, with greater improvements among participants receiving the more intensive intervention.

IMPLICATIONS FOR PUBLIC HEALTH

The findings outlined above suggest that transitioning to a diet rich in fruits and vegetables with lean proteins and healthy fats may benefit cognitive health for all adults. Public health recommendations should encourage individuals, especially those at risk for cognitive decline, to adopt dietary patterns that prioritize plant-based foods, lean proteins, and healthy fats, while limiting intake of unhealthy fats and sugars. Such dietary changes should be tailored to individual needs and cultural contexts, ensuring they are realistic, affordable, accessible, manageable, and sustainable.

The role of social determinants of health

Dietary habits are influenced by cultural, social, and economic factors, including food access and affordability. Availability of healthy foods is positively associated with better memory, whereas limited access to healthy foods is associated with cognitive decline, food insecurity, and higher likelihood of depressive symptoms. Public health initiatives, such as community-based programs and policies improving food availability, are crucial in reducing these disparities. Programs like the Supplemental Nutrition Assistance Program (SNAP) have been linked to slower memory decline, reinforcing the need to ensure equitable access to nutritious foods, particularly for underserved communities. Public health initiatives should address disparities in access to healthy food and promote healthier food environments.

DISCUSSION

Current research underscores the importance of nutrition in cognitive health, particularly the protective effects of primarily plant-based diets. These diets, with their emphasis on whole, nutrient-dense foods, have been shown to slow cognitive decline, improve brain function, support brain volume, and reduce the risk of dementia. Nutritional research faces ongoing challenges in measuring dietary intake, standardizing diet scoring methods, determining the optimal timing for dietary interventions, understanding the effects of supplements compared with whole foods, and accounting for an individual's culture and dietary preferences. Studies are also needed to assess the effects of dietary interventions on cognition based on when in life (adolescence, mid-life, older age) dietary patterns are changed.

DIET, NUTRITION AND COGNITION

In conclusion, dietary interventions, especially when integrated with other lifestyle modifications such as physical activity, cognitive training, social interaction, and cardiovascular risk management, can be an effective strategy for improving cognitive health. As with all interventions, they must be adaptable, culturally relevant, and sustainable in the long term. Addressing dietary access and equity is crucial to ensure that these benefits reach all populations.

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Glossary

Diet refers to the types and amounts of food and beverages consumed by an individual.

Dietary patterns refers to the combination and quantities of foods and beverages consumed over time.

Nutrition refers to the process of using food for growth, metabolism and tissue repair.

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SOCIAL ENGAGEMENT AND COGNITION

WHAT IS ALREADY KNOWN

Social engagement is defined as meaningful and sustained contact with at least one other person that is intrinsically and mutually beneficial and pertaining to a shared interest, activity, or goal. A 2023 report from the U.S. Surgeon General noted that research suggests loneliness – a related construct to social engagement – can shorten a person's life by an estimated 15 years, or the equivalent of smoking 15 cigarettes per day.

Social engagement is considered a key component of successful aging and an integral part of overall health. Its relationship specifically to cognitive decline and dementia remains a subject of study. Of note, social engagement and loneliness are two different concepts. The opposite of social engagement is social isolation. But that is not the same thing as loneliness. Socially engaged individuals can suffer from loneliness and socially isolated people may not feel lonely at all. Both loneliness and social isolation have been associated with increased rates of cognitive decline, but they are not synonymous.

The interconnectedness of social, physical, and cognitive activity – and the tendency of trials to combine one or more elements in a single intervention – makes it difficult to assess social engagement's effect on reducing the risk for cognitive decline.

BACKGROUND AND EVIDENCE BASE

Social engagement in mid-life may help protect against dementia in later life, by protecting cognitive function and slowing cognitive decline throughout the life course. It also may provide benefits to many older individuals, including persons living with dementia. Among older individuals with a genetic risk for dementia, rates of dementia were lower among those who frequently engaged in social activities. Studies have also found that the variety and frequency of social engagement are important for preventing further cognitive decline among those with early-stage memory loss/mild cognitive impairment.

One possible mechanism by which social engagement can facilitate cognitive function and protect against cognitive decline is through cognitive reserve, which is thought to result from increased neuronal connections and more efficient processing in the brain. Greater cognitive reserve may allow the brain to maintain function and cope with conditions associated with cognitive impairment, such as cerebrovascular disease and atrophy. Cognitive reserve evolves throughout the lifespan and, theoretically, can be enhanced by interventions at any stage of life. One study concluded that social engagement may lower risk of cognitive decline by as much as 70% in those who frequently socially engage.

Multi-site collaborations are needed to examine and scale-up existing community-based strategies to facilitate and foster social engagement, especially among priority populations. Once priority populations are identified, strategies and interventions may be further developed, tailored, and tested accordingly.

IMPLICATIONS FOR PUBLIC HEALTH

To have the greatest effect, social engagement must be culturally appropriate and tailored to individual interests. Several groups may be at increased risk for social isolation/lower levels of social engagement:

- Older women may outlive their spouses and lose the social connections they had as a couple.
- Members of racial and ethnic minority communities may have weaker social ties due to the impact of migration and immigration – and the effects of discrimination across the lifespan can result in exclusion and isolation from the broader community.
- Lesbian, Gay, Bisexual, Transgender, Queer and additional identity (LGBTQ+) individuals may be subjected to discrimination and lifetime stigma – and may be ostracized from family members and other community supports.
- Rural residents tend to live farther apart from other individuals, have less access to transportation infrastructure, and often experience poor and sparse digital connectivity.

For public health strategies designed to impact social engagement in the post-COVID era, hybrid formats (i.e., social engagement activities with both an in-person and remote option) are likely needed for the foreseeable future. Recent novel interventions include community choirs, dance, virtual conversation groups, and storytelling. Investigators may adapt and test international programs in the United States for evidence of efficacy.

The role of social determinants of health

Public health research increasingly recognizes that successful aging is not solely a biological process but one deeply embedded in social contexts that either foster or hinder cognitive resilience. Social and environmental barriers can create chronic stressors and reduce cognitive stimulation, thereby accelerating cognitive aging. Public health efforts must therefore include community- and policy-level strategies that address these upstream determinants. By investing in age-friendly infrastructure, inclusive community programs, and digital access, particularly for historically excluded populations, communities can begin to reshape the conditions that enable or inhibit cognitive vitality in later life.

DISCUSSION

To date, most data on social engagement to enhance brain health comes from observational studies. Few randomized clinical trials or interventional studies exist, especially those that focus solely on social engagement and cognitive function. Such studies are needed to address causality and the directionality of impact (does social isolation affect cognitive function or does cognitive impairment impede social engagement), the distinct role of social engagement separate from physical and cognitive activity, and the importance of social engagement in midlife and from mid- to late life. In addition, overlapping concepts are often found in the literature (e.g., social isolation vs. loneliness, social participation vs. social contact), confounding a clear understanding of what interventions might be needed and signaling a need for refined studies of social engagement in aging.

Future work should explore the most effective types of social engagement, how much social engagement is needed for cognitive benefit, and the best ways to define and measure social engagement.

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DEPRESSION AND COGNITIVE DECLINE

WHAT IS ALREADY KNOWN

Depression and dementia are prevalent and often co-occurring conditions among older adults, yet both are frequently under-recognized in clinical and community settings. While major depression becomes less common with age, milder and chronic depressive symptoms tend to increase in prevalence. Understanding the relationship between depression and dementia is important for informing early detection and care strategies. Depression may increase the risk of developing dementia, but it may also be an early indicator of neurodegenerative changes in the brain. In some cases, depressive symptoms may reflect the earliest manifestations of dementia-related pathology, rather than being a separate or preceding condition. Recognizing this complexity can help improve diagnostic accuracy, guide intervention timing, and shape more effective public health responses to aging-related cognitive decline.

BACKGROUND AND EVIDENCE BASE

Depression is a mood disorder that exists on a spectrum, from transient feelings of sadness to clinically diagnosed major depressive disorder (MDD), which is characterized by persistent low mood, loss of interest, and functional impairment. In research and clinical settings, depression is often measured using standardized diagnostic criteria or symptom scales, but definitions and severity thresholds can vary across studies. This variability is important to consider when interpreting the evidence linking depression and dementia.

Although much attention has focused on depressive "pseudodementia," a classical presentation of impaired cognition due to underlying undiagnosed depression instead of neuropathology, decades of research have revealed that it is rare. Instead, depression may occur as an antecedent to dementia, including Alzheimer's disease. Both depression and anxiety have been shown to be associated with dementia and may be independent risk factors for Alzheimer's disease. However, there are discrepancies in the literature. In a study of over 500,000 military conscripts in Denmark, depression during adulthood was associated with increased risk of dementia later in life. In a 2021 longitudinal observational study, 16,608 people without prevalent dementia were followed for 6 years; depression in both adulthood and late life was associated with increased dementia risk, while depression occurring only in late life was associated with lower risk of dementia. Resolution of adult-onset depression symptoms was not linked to a higher risk of developing dementia. In contrast, the longitudinal Swedish Twins Study (with over 40,000 participants) found that late-life depression was associated with a greater risk of Alzheimer's disease while mid- to late-life depression increased the risk of non-Alzheimer's disease subtypes of dementia that typically occur at younger ages.

Depression often presents as a coincident symptom in mild cognitive impairment (MCI), and its presence is associated with an increased likelihood of progression to Alzheimer's disease. There is also an association between depression and cerebrovascular disease in older adults that has led to the term "vascular depression." In 443 Black participants enrolled in the National Alzheimer's Coordinating Center (NACC) dataset, depression and white matter hyperintensity volume were associated with a higher prevalence of dementia. In summary, there appears to be a bidirectional relationship between depression and Alzheimer's disease, including genetic predisposition, immune dysregulation, accumulation of Alzheimer's disease-related biomarkers (e.g., amyloid-β and tau), and alterations in brain structure including cerebrovascular disease.

There is less information and prospectively-collected data to address questions about depression and how its treatment earlier in life may affect cognition. This is largely because of the long time lag between diagnosis and treatment for depression, and cognitive decline or dementia later in life. The literature on whether depression in earlier life predicts the likelihood of developing dementia is mixed, with nearly all studies relying on history obtained from participants about decades-old depressive episodes because prospective evaluation over an entire lifetime is impractical. Similarly, data are limited on whether treatment of depression at younger ages may prevent cognitive decline and dementia in the distant future.

IMPLICATIONS FOR PUBLIC HEALTH

The association between depression and dementia highlights the need for proactive mental health strategies within public health systems. Early identification of depression may offer a critical window to reduce or delay cognitive decline. Public health initiatives should prioritize integrating depression screening into routine care for adults, particularly in midlife and older age, and ensure that treatment pathways are accessible and evidence-

based. While more research is needed to determine whether treating depression directly reduces dementia risk, the potential for long-term cognitive benefits reinforces the value of early intervention.

The role of social determinants of health

Social and environmental conditions play a significant role in shaping both depression and dementia risk. Individuals experiencing economic hardship, social isolation, or limited access to mental health care are more likely to have undiagnosed or untreated depression, which may contribute to accelerated cognitive decline. Structural barriers such as health care access, housing instability, and stigma around mental illness further complicate timely diagnosis and treatment. Addressing these upstream factors through community-based outreach, culturally responsive care, and cross-sector collaboration is essential to promoting mental and cognitive health across diverse aging populations.

DISCUSSION

Depression is closely linked to subsequent development of dementia, particularly Alzheimer's disease, vascular, and mixed dementia. Screening efforts for depression remain paramount in order to promote awareness and treatment. Early observational data suggests recovering from depression may be associated with bringing risk of subsequent dementia back down, but further research is needed to determine whether management of depression reduces dementia risk.

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SENSORY IMPAIRMENTS AND COGNITION

WHAT IS ALREADY KNOWN

Age-related dysfunctions in vision, hearing, and smell have each been linked separately to cognitive impairment and cognitive decline. Historically, it has been challenging to disentangle assessments of cognitive impairment from assessments of sensory impairment. However, more recent data from cognitive tests that did not rely on both unimpaired hearing and vision have established moderate evidence for a link between sensory impairment and cognitive decline/dementia. Recent meta-analyses support an association between cognitive decline and vision and hearing impairments in particular, which are the most easily modifiable sensory impairments. Multisensory decline is common as people age, but few large population-based studies collect the data needed to examine the relationship between multisensory loss and cognitive decline and dementia.

BACKGROUND AND EVIDENCE BASE

Evidence for a relationship between sensory impairment and aging

Sensory impairment, or reduced function in one or more senses, is common with age. A recent study found that many older Americans experience deficits in multiple senses, suggesting a shared aging process across all five senses. Hearing loss often results from nerve degeneration in the inner ear, though other parts of the auditory system can also be affected. Vision impairment in older adults is typically caused by conditions such as macular degeneration, glaucoma, cataracts, diabetic retinopathy, and refractive errors. Loss of smell is also widespread, affecting up to half of older adults, with prevalence increasing with age.

The link between sensory impairment and cognitive decline is complex. Shared underlying conditions such as vascular disease may damage both the brain and sensory systems. Aging may also create a bi-directional relationship: declining sensory function can impair cognition, while brain changes can alter sensory processing. Additionally, sensory loss may increase cognitive load and contribute to social isolation, depression, and reduced activity, all of which are associated with cognitive decline.

Evidence for a relationship between sensory impairment and cognitive decline

A 2017 study assessed participants with both the Global Sensory Impairment (GSI) score, an integrated measure of dysfunction in the five classical senses (vision, hearing, smell, taste, and touch), and a modified Montreal Cognitive Assessment. Worse GSI scores at baseline predicted worse cognitive function five years later.

Hearing: Hearing loss has been associated with increased rates of cognitive decline and dementia risk. Older adults with severe hearing impairment often have widespread cognitive impairments. A recent meta-analysis found that age-related hearing loss may be a modifiable risk factor for cognitive decline, cognitive impairment, and dementia.

Vision: Two recent meta-analyses provide evidence of an association between vision impairment and dementia risk. Both analyses reported that the relative risk of dementia or cognitive impairment was greater among people with vision impairment than among those without vision impairment.

Smell: A 2016 longitudinal cohort study of more than 1,400 cognitively normal adults linked olfactory impairment to incident amnestic mild cognitive impairment and to the progression from mild cognitive impairment to Alzheimer's dementia. Importantly, among people with normal olfaction, there were no dementia events over the study period. A 2014 review noted that many age-related dementias, such as Alzheimer's, vascular dementia, Parkinson's disease, and frontotemporal dementia, are associated with olfactory impairment. Loss of smell may lead to diminished quality of life, depression, and potential issues with food safety and hygiene.

Does intervening on hearing or vision improve outcomes for people with dementia?

Because of the interconnections between sensory and cognitive function, it is reasonable to wonder if interventions to address sensory impairment could also have an effect on cognitive function. Interventions could consist of preventing disease or injury as well as treating deficits.

One review examined the results of studies that evaluated the effect of vision and hearing interventions on outcomes in people with dementia. The authors reported that most

published studies on this topic were small and of moderate quality, but many showed evidence of benefit. An analysis of data from the Health and Retirement Study reported that the use of hearing aids slowed the rate of memory decline in older adults after they began using hearing aids relative to their rate of memory decline before using hearing aids. The multi-center ACHIEVE trial randomized participants with untreated hearing loss but without significant cognitive impairment to a hearing aid intervention or an educational control. While the trial did not observe a significant treatment effect in the total population, the hearing aid intervention was more effective in participants who were older and had more risk factors for cognitive decline. With respect to treatment of vision deficits, an analysis of participants in the English Longitudinal Study of Ageing showed cataract surgery was associated with a lower rate of memory decline compared with a control group of participants who did not have cataracts. Thus, there is some evidence to suggest treating sensory deficits positively affects cognitive decline, but further studies are needed.

IMPLICATIONS FOR PUBLIC HEALTH

Interventions for hearing and vision impairment have been shown to improve noncognitive outcomes in the general population. While some studies suggest that treating vision and hearing problems in older adults may reduce their risk of cognitive decline, the overall evidence is mixed, and more research is needed to determine whether these interventions can prevent or delay dementia. In people who already have dementia, sensory interventions are not expected to reverse or prevent the disease, but they may improve quality of life and functional outcomes. The current evidence suggests that people with dementia may benefit from, and are unlikely to be harmed by, sensory interventions, especially those that are minimally invasive, such as glasses and hearing aids. However, the effects of these interventions on cognitive outcomes in people with dementia remain unclear. In recognition of the relationship between sensory and cognitive impairment and the overall benefit of addressing sensory health, the U.K.'s National Institute for Health and Care Excellence guidelines recommend sensory examinations for people with cognitive deficits, including hearing assessments for people with suspected or diagnosed dementia and eye tests every two years for those living with dementia.

The role of social determinants of health

Many Americans do not receive appropriate sensory care. More than six million have chronic vision loss of some type, but fewer than half receive appropriate care for these conditions; only 1 in 6 people who could benefit from hearing aids use them. Furthermore, assessment and treatment of sensory impairments are unequal among populations, leading to significant inequities in sensory health outcomes by socioeconomic status and racial/ethnic groups.

DISCUSSION

There are many areas in which additional research is needed to understand the connections between sensory impairment and cognitive function. Also, it is difficult to compare findings across studies to date because of inconsistencies in the cognitive assessments used in hearing and vision studies. Nonetheless, there is evidence of an association between sensory impairment (particularly in vision, hearing, and olfaction) and cognitive decline and dementia. The evidence is mixed as to whether addressing hearing or vision impairments can reduce the risk of cognitive decline, but people with dementia may benefit from, and are unlikely to be harmed by, sensory interventions, especially those that are minimally invasive. Additional randomized clinical trials are needed to determine whether specific interventions (such as hearing aids and cataract surgery) can improve cognitive outcomes in adults with high risk of or probable dementia.

Although screening programs for vision/hearing problems have been tested, they may not be cost-effective for the general population of middle-aged and older adults. Therefore, screening should be focused on populations at greatest risk of sensory and cognitive impairment.

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DRINKING ALCOHOL AND COGNITIVE DECLINE

WHAT IS ALREADY KNOWN

Excessive alcohol consumption is a major contributor to lost lives and lost health in the United States. The United States federal guidelines define the limits of low-risk drinking as up to 2 standard drinks per day for a male and 1 standard drink per day for non-pregnant females. A standard drink is typically defined as containing about 14 grams (0.6 ounces) of pure alcohol, which is roughly equivalent to 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of distilled spirits. Drinking above these limits can increase one's long-term risk for cancer, injuries and violence, certain forms of heart disease, liver damage, and death. Drinking heavily can also lead to alcohol use disorder, a form of addiction in which the individual cannot control their drinking even when it has adverse social or medical consequences.

Excessive alcohol consumption can damage the brain in several ways. Alcohol itself can damage nerve cells at high doses, and prolonged heavy drinking leads to a specific form of cognitive decline and dementia (termed alcoholic dementia). Even occasional binge drinking, defined as 5 or more drinks in a day for a man or 4 or more for a woman, can raise blood pressure and risk for atrial fibrillation, the most common heart rhythm disorder, both of which can lead to stroke. Individuals who consume excessive alcohol may also neglect other parts of a healthy diet, leading to nutritional deficiencies that can damage the brain.

Whether there is damage to the brain when a person engages in alcohol consumption below or at the recommended limits is uncertain. Some studies suggest that it is safe for the brain, while others do not. In particular, individuals at higher risk for dementia, such as those with mild cognitive impairment or with a genetic tendency toward dementia, may face increased risk even from limited drinking. These findings are based on correlational studies, which identify associations but cannot establish a direct causal relationship between alcohol use and cognitive decline due to potential confounding factors.

BACKGROUND AND EVIDENCE BASE

Heavy alcohol consumption has long been known to produce distinctive chronic brain syndromes. In severe cases, this can lead to alcoholic dementia, characterized by widespread and lasting brain damage. However, even before reaching that level of severity, excessive alcohol use may impair cognitive functions such as memory, attention, and decision-making. These subtler effects can accumulate over time and may not be immediately recognized as part of a broader pattern of cognitive decline.

In addition to direct neurotoxicity, excessive drinking can lead to injury to the brain through several other pathways. For example, individuals with alcohol use disorder, who may consume alcohol at the expense of healthy food, can develop vitamin B1 (thiamine) deficiency, which in turn can injure brain cells and cause a distinctive form of dementia called Korsakoff syndrome. Falls that occur during episodes of heavy drinking can produce concussions and, with repeated injury, chronic traumatic encephalopathy, a progressive degenerative brain disease.

In addition, heavy alcohol consumption can lead to strokes, which cumulatively can also cause dementia through multiple pathways. First, heavy drinking causes high blood pressure, which is the major risk factor for all types of stroke. Second, even occasional episodes of heavy drinking can trigger atrial fibrillation, which is the most common abnormal heart rhythm. This combination of drinking and atrial fibrillation is so common that it has been termed the "holiday heart syndrome." Atrial fibrillation can then lead to blood clots in the heart that may travel to the brain, leading to strokes.

In contrast with excessive drinking, consumption within recommended limits remains a controversial area for research, and the effect of limited alcohol consumption on risk for dementia is not yet settled. For example, an expert panel convened by the National Academies of Sciences, Engineering, and Medicine concluded in January 2025 that too little evidence existed to confidently understand how limited drinking affects risk of dementia, Alzheimer's disease, or cognitive decline.

Important reasons exist to be cautious about even limited alcohol consumption and its effect on the brain. For example, nerve cells are more sensitive to damage from alcohol consumption than are cells in other organs. Even 1-2 drinks can raise blood alcohol concentrations enough to affect individuals' ability to complete complex tasks like driving. In large studies, people who consume higher amounts of alcohol are more likely to have more overall brain shrinkage and more shrinkage of the hippocampus, a particular hallmark of Alzheimer's disease, with no evidence for a threshold. Finally, alcohol acts as a mild blood thinner, reducing the clotting ability of the blood.

This could increase the risk of hemorrhagic strokes, where bleeding occurs into the brain.

Other evidence suggests that limited alcohol consumption is unlikely to worsen cognition markedly for most adults in the same way that heavy drinking does. For example, while heavy drinking increases blood pressure, drinking within recommended limits appears not to. Consistent with that finding, the risk of ischemic stroke also does not increase with limited drinking. When consumed in limited amounts with meals, alcohol also tends to reduce average blood sugar, which may slow the development of diabetes, a major risk factor for cognitive decline. A combined analysis of 15 individual studies in 2023 concluded that dementia risk did not differ meaningfully among older adults based on their alcohol intake if they remained within recommended limits.

IMPLICATIONS FOR PUBLIC HEALTH

Excessive alcohol consumption is a major public health concern, in part because of its damaging effects to the brain. Excessive alcohol consumption is most common among individuals in their 20s and 30s where its major health consequences are increases in injury and violence, but older adults are particularly susceptible to harm from alcohol because their metabolism of alcohol and baseline health can worsen with age. Similarly, women tend to consume less alcohol than men, but harms from alcohol occur at lower levels of consumption among women. Thus, strong efforts to eliminate excessive drinking are needed for individuals of all ages and regardless of sex, beginning with better education about the definition of excessive drinking. Unfortunately, many adults in the United States and globally remain unaware of the specific risks alcohol poses to brain health. These risks include impaired memory, reduced attention and executive function, increased likelihood of mood and anxiety disorders, and at higher levels of consumption, structural brain changes and increased risk for cognitive decline and dementia.

At present, more limited amounts of alcohol consumption have not consistently been found to increase risk for cognitive decline or dementia, although it appears to increase risk for other diseases, such as female breast cancer and atrial fibrillation. Moreover, some individuals, such as those with early cognitive impairment, may be at particular risk with even limited drinking. Therefore, middle-aged and older adults who consume alcohol within recommended limits should review their alcohol consumption with a health professional regularly and be prepared to modify it as their health changes or as new evidence emerges about this highly complex substance.

The role of social determinants of health

Alcohol consumption varies markedly across different cultures. Many religious groups abstain completely from alcohol, for example, while others may use wine in specific religious ceremonies. Similarly, some cultures (e.g., Mediterranean countries such as Italy and Spain) tend to encourage limited amounts of alcohol consumption in social settings with meals, which blunts the spikes in blood alcohol concentration that can damage brain cells. In general, alcohol consumption is most harmful in cultural settings where binge drinking occurs.

In addition to the ways that social factors can affect alcohol consumption and its consequences, alcohol can also affect social factors in turn. Alcohol use disorder and problem drinking are powerful social factors, leading to alienation from friends and family, poor self-care, and loss of employment and insurance. Social isolation and lack of self-care can contribute to cognitive decline, exacerbating the harm caused by excessive alcohol consumption itself.

DISCUSSION

Alcohol consumption remains one of the most complicated medical topics to study, in part because it may not have the same types of effects if consumed within recommended limits that it does at higher doses. This unusual relationship, sometimes called a "J-shaped curve," does not occur for substances that are either always harmful (e.g., cigarette smoking) or always beneficial (e.g., eating a balanced diet). In addition, investigators have not yet conducted long-term randomized trials of alcohol consumption (similar to what normally occurs for new medications), leaving doctors and patients with evidence that is not definitive, can be difficult to interpret, and seems to change frequently. Nonetheless, the key federal guidelines about alcohol consumption – do not drink more than 1 drink in a day for women or 2 drinks in a day for men – have not changed in decades and remain the basis for maintaining a healthy lifestyle that may reduce risk for dementia.

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