Reducing Dementia Risk:

A Summary of the Science and Public Health Impact

The Alzheimer's Association is grateful for the work of Wake Forest School of Medicine in the preparation of this publication, which is supported by the Centers for Disease Control and Prevention (CDC) of the U.S. Department of Health and Human Services (HHS) as part of a financial assistance award totaling $673,487, with 100 percent funded by CDC/HHS. The contents 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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Table of Contents

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The prevention of aging-related cognitive impairment and dementia is a major and urgent public health priority as well as a priority for individuals, families, and communities. In response, the popular press, the National Institutes of Health, and the World Health Organization, among others, have recently focused attention on lifestyle and behavioral actions that could preserve cognitive function and prevent memory impairment and dementia. Because evidence for the effectiveness of specific health-related behaviors and practices has begun to emerge, the U.S. Congress provided funding for the Centers for Disease Control and Prevention (CDC) to strengthen the public health infrastructure on Alzheimer's disease, including by expanding efforts at dementia risk reduction and prevention.

As part of this effort, the CDC selected the Alzheimer's Association to serve as the Public Health Center of Excellence on Dementia Risk Reduction. The Association then partnered with Wake Forest School of Medicine to convene a panel of nationally and internationally renowned scientists with expertise in specific areas of dementia and cognitive impairment prevention research. The panel's charge was to review, evaluate, and synthesize the currently available knowledge on preventing or delaying the onset of cognitive decline and dementia. The panel identified 12 areas of promise. Each area was reviewed by a content expert and presented to the panel for further discussion.

From these 12 areas of promise, eight were selected, based on the level of research support and strength of evidence, to inform emerging efforts by public health agencies throughout the United States to address the risk for cognitive decline and dementia. Those eight areas are: **diabetes and obesity, physical activity, social engagement, diet and nutrition, vascular health, sleep, smoking and alcohol, and sensory impairments**.

Only one of the final eight areas — interventions for hypertension — satisfied the most stringent form of evidence with results from a randomized controlled trial (RCT), the gold standard in evidence generation. The remaining seven areas have varying levels of epidemiologic evidence, but they all have sufficiently strong evidence to include in a toolbox for consideration by public health agencies when addressing dementia risk in the diverse communities they serve. In addition, the link between cognitive decline/dementia and **traumatic brain injury** has sufficiently strong evidence across the lifespan — from earliest childhood to the oldest old — that it, too, should be a target for public health action. The expert panel recommends the Public Health Center of Excellence on Dementia Risk Reduction identify interventions to address these risk factors and work with public health agencies to implement interventions within their communities to position their citizens for successful cognitive aging, including reducing risk for cognitive impairment and dementia.

Cognitive decline and dementia involve complex processes. Many of the risk factors are interrelated, and the interventions are often complementary. As a result, an approach that combines strategies for prevention may be the most effective.

For each of the eight areas plus traumatic brain injury, Wake Forest School of Medicine, the expert panel members, and the Alzheimer's Association developed a summary that examines the current state of evidence for preventing cognitive decline and dementia and that discusses the implications for public health. Those summaries follow.

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Cardiovascular Risk Factors, Cognitive Decline, and Dementia:

What's Good for the Heart Is Good for the Brain

What Is Already Known

There is substantial evidence for a relationship between vascular risk factors and brain disorders, including cognitive decline and dementia. Up to 80% of people with Alzheimer's disease (AD) have significant vascular pathology in the brain. This evidence is remarkably consistent and spans the translational spectrum from basic biology to epidemiologic and clinical trial science.

Background and Evidence Base

Mechanistic evidence

Beta-amyloid (Aβ), a protein that accumulates in the brains of those with Alzheimer's disease, produces oxidative stress that in turn causes neurovascular injury by constricting capillaries in the brain. This constriction can lead to chronic hypoperfusion, exacerbating neurodegeneration and cognitive dysfunction. There is evidence for a synergistic interaction between the accumulation of Aβ 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 vascular 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 peripheral small arteries and microvessels 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 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.
- A 2014 review and meta-analysis reported associations between arterial stiffness, cerebral small vessel disease, and cognitive impairment.
- The Harvard Aging Brain Study reported interactive associations among vascular risk, Aβ 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.

Evidence supporting vascular risk treatment to reduce the risk of later dementia

The most recent evidence that reduction of a cardiovascular (CVD) risk factor could minimize the risk for clinically significant cognitive decline came from the Systolic Blood Pressure Intervention Trial (SPRINT). 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). 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. (It should be noted that this trial terminated early because of the cardiovascular benefits of treatment identified in the overall SPRINT trial.) 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.

Evidence for the effects of statins on reducing the risk of cognitive decline and dementia is equivocal. While it is known that clinical and subclinical cardiovascular disease increases the risk of cognitive impairment and dementia and that the antihypertensive drugs statins reduce rates of ischemic stroke (a significant contributor to vascular dementia), the role of statins in preserving cognition is unclear. This gap in knowledge led to the development of the PREVENTABLE study, a new trial that is testing the hypothesis that statins could reduce the occurrence of the composite endpoint of death, dementia, and persistent disability among 20,000 community-living adults aged 75 years and older without cardiovascular disease or dementia at baseline. The secondary hypothesis is that statins could reduce the occurrence of the composite endpoint of MCI and dementia and a composite cardiovascular outcome. Results of this trial, due in 2027, will provide evidence for whether treatment with statins can protect against cognitive decline in aging.

Implications for Public Health

There is a vascular component in most cases of ADRD, with increasing evidence that hypertension in midlife is strongly associated with dementia in older adulthood. Hypertension is thus a modifiable risk factor for dementia and should be a target for prevention strategies. Public health efforts should focus on populations with the lowest rates of controlled hypertension: older adults and Black, indigenous, and other people of color. There is substantial epidemiologic evidence for a link between long term hypertension and ADRD incidence and therefore an additional target population 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 quickly reduce the risk of cognitive dysfunction.

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 is an effective means to reducing the risk of cognitive dysfunction in older age. Importantly, there are many existing treatments for doing so. However, despite the wide availability of such antihypertensive 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 target 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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Exercise as Medicine to Prevent Cognitive Decline and Dementia:

Is it Worth the Sweat?

What Is Already Known

The U.S. 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 also become clear that high-intensity aerobic exercise improves not just the function of the body but also function of the brain. Based on research to date, the potential benefits of physical exercise on cognitive health cannot be overstated. The World Health Organization included physical activity as a top priority in its recent review of 12 non-pharmacological interventions with the potential to reduce the risk of cognitive decline and dementia.

Background and Evidence Base

More than two decades of research in both animal and human studies strongly supports a relationship between aerobic exercise and beneficial effects on the brain. A 2017 review summarized findings from the human and animal literature on the positive effects of physical exercise on the brain and highlighted the vascular links underlying those benefits. The review reported that exercise was associated with increased neurorepair, increased clearance of hyperphosphorylated tau, improved glucose metabolism, reduced inflammation, reduced β-amyloid plaques in the brain, and reduced oxidative stress. 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. Findings from randomized controlled trials are less consistent, but preliminary evidence suggests that aerobic exercise results in positive effects on cognition, brain structure and function, and Alzheimer's disease biomarkers.

A 2018 meta-analysis of 19 studies (17 of which were randomized controlled trials) involving a total of 1,145 older adults concluded that exercise improves cognition among individuals with mild cognitive impairment. There was a 1.3-fold higher effect when the analysis examined only the effects of aerobic exercise (excluding light physical activity).

A hallmark study in 2006 showed that, among cognitively normal older adults, six months of high-intensity aerobic exercise increased volumes in parts of the brain associated with executive function. This study provided the inspiration to examine exercise as a potential therapeutic intervention in adults with mild cognitive impairment. The Piedmont Aging, Cognition, and Exercise (PACE) study is one such randomized controlled trial. The intervention included six months of aerobic exercise compared with a control group that received six months of stretching and balance activities. Participants completed glucose tolerance tests, 400-meter walk tests, lumbar puncture (for evaluations of Alzheimer's disease biomarkers in cerebrospinal fluid), magnetic resonance imaging of the brain (to evaluate brain volumes and blood flow), and various standardized cognitive assessments. The results indicated that, compared with baseline levels, participants in the aerobic exercise group experienced significant improvement in executive function, while participants in the stretching group experienced a significant decline in this cognitive ability. (In essence, because only cognitively impaired individuals were enrolled in the trial, the control group's decline in cognitive ability indicates the stretching and balance activities had no effect). Additionally, aerobic exercise was shown to increase overall blood flow in the brain and in regions associated with executive function that included the right and left prefrontal, right and left posterior parietal, and right and left cingulate cortices.

The potential therapeutic effects of aerobic activity among individuals with Alzheimer's-type dementia have also been examined. The Alzheimer's Disease Exercise Program Trial (ADEPT) tested the effects of a six-month intervention of aerobic exercise in adults with early-stage Alzheimer's dementia, with stretching used as the control intervention. Cognitive, functional, cardiorespiratory fitness, and brain imaging outcomes were evaluated. Results showed that the six-month program of aerobic exercise was associated with a modest gain in functional ability. Secondary analyses revealed that improvements in cardiorespiratory fitness over time were positively correlated with improved memory performance and increased bilateral hippocampal volume.

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, frequency, and overall duration of interventions. Health-restoring effects of exercise take time; trials with interventions lasting less than six months rarely show cognitive benefits. Longer trials more commonly show benefits on executive function, but those with interventions lasting less than 12 months rarely show benefits on memory. Thus, interventions need to be of an appropriate intensity and an appropriate duration for individuals to experience benefits.

Additional challenges with respect to recommendations arise because many trials have historically enrolled cohorts that are not demographically representative of the overall population. Existing trials are more likely to have greater diversity, include more appropriate representation in their recruited cohorts, and ensure sustainability within the community if the trial results are positive. For example, in the Exercise in Adults with Mild Memory Problems (EXERT) study, nearly 20% of the recruited 300-person sample represents communities of color, and the intervention is delivered in the community at YMCAs under the supervision of study-certified YMCA trainers.

Implications for Public Health

The growing evidence for a relationship between physical activity and long-term preservation of cognitive function supports the need to promote community exercise participation. In addition, it is important to implement and assess new avenues for exercise participation in diverse communities and across all age groups, which will serve to strengthen the message that greater physical activity and exercise is a helpful pathway for reducing the burden of cognitive impairment and dementia in society. However, the success of intervention strategies depends on access and sustainability of delivery within the community.

Discussion

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. 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, this type of an intervention that is designed to protect cognitive function in older adults will not likely succeed. Because research studies have funding and project duration limits and thus cannot provide ongoing support for interventions shown to be successful, 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 promote "exercise as medicine" to their patients and should provide appropriate referrals to evidence-based community programs that can properly and effectively assist and support individuals.

There is increasing evidence that physical exercise, and aerobic training in particular, has favorable effects on multiple health outcomes, including reduced risk against cognitive decline and possibly 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 all adults should be encouraged to talk to their health care providers about participating in vigorous aerobic activity in line with Department of Health and Human Services guidelines.

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Diabetes, Obesity, and Cognition:

What We Do Now Affects How We Think Later

What Is Already Known

Rates of obesity and diabetes are rising steeply 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% have Type 2 diabetes (T2DM), including 25% of those aged 65 years or older. These conditions often co-occur and both increase the risks for cognitive decline and dementia as well as other health problems such as heart disease and disability.

Background and Evidence Base

The adverse consequences of obesity begin early, placing children at increased risk for obesity and diabetes as adults. Diabetes and midlife obesity result in increased risk for late-life dementia. Obesity is strongly linked to insulin resistance and elevated blood glucose which, in turn, adversely 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 diabetes adversely affect brain health, and many of these are driven by increases in inflammation and impairments in metabolism.

The Lancet Commission concluded that prevention of obesity and T2DM is a promising pathway to reduce the risk of cognitive decline. The most effective approach for this may be through the adoption of healthy lifestyles that include better diets with fewer calories and less processed foods and increased physical activity. Both the Finnish Diabetes Prevention Study and the Diabetes Prevention Program have developed interventions that have been proven to lower risks for T2DM.

Compared with preventing obesity as a means of reducing risk for dementia, the evidence is less compelling that treating midlife obesity will subsequently reduce the elevated risk. There is also less evidence that treatment of later-life obesity is effective in reducing risk for dementia. Therefore, all the evidence thus far points to prevention of obesity at younger ages, rather than treatment after it occurs, as holding the most promise for reducing the risk for dementia later in life.

While better control of T2DM is often associated with better cognitive functioning, it is not clear that treatment of T2DM through medical management or behavioral intervention reduces the risks it conveys for dementia. As for obesity, it is best to prevent the occurrence of T2DM to reduce risks of dementia.

Implications for Public Health

Providing communities with greater awareness through education and promoting balanced diet and greater physical activity will potentially prevent obesity and may have a positive benefit on cognition. 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 obesity and unrecognized or untreated T2DM — could yield benefits in terms of reduced burden of cognitive impairment and dementia across communities as a whole and also provide benefits for other comorbidities and health-related consequences of obesity and T2DM.

Discussion

Opportunities to prevent obesity, and thus lower risks of cognitive problems, have primarily targeted children and young to middle-aged adults. These include programs focused on behavior changes in children, especially through parental counseling and school-based programs. They also include individual- and group-based behavioral interventions targeting adults, and community-based strategies to increase physical activity and improve access to higher-quality diets. Opportunities to prevent T2DM as a means to reduce risk of cognitive problems have focused on weight loss and increased physical activity. For approaches to be successful in preventing obesity and T2DM, it is critical that they are sensitive to and effectively target the needs of individuals and local communities.

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References available upon request. Please contact us at CenterOfExcellence@alz.org.

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TBI and Cognitive Decline:

Traumatic Brain Injury Increases Risks

What Is Already Known

There are an estimated 2.5 to 4 million cases of traumatic brain injury (TBI) per year, with a bimodal distribution based on age; the oldest and youngest individuals account for the greatest number of cases. The reasons for these injuries also vary by age group. 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, and for older adolescents to middle-aged adults, injuries are generally related to sports, motor vehicle accidents, and military service. Other causes of TBI at younger ages include adverse childhood experiences (ACEs) — potentially traumatic events that occur during childhood — and violence. Examples of ACEs include witnessing or experiencing violence, abuse, or neglect in the home or community. Greater exposure to ACEs earlier in life increases risk for future adverse experiences that place an individual at further risk for TBI, such as violence (e.g., domestic/intimate partner abuse and elder abuse). Regardless of when across the lifespan TBI events occur, they result in poorer overall health and can increase risk of experiencing cognitive decline or dementia in later life.

Background and Evidence Base

Evidence of an association between traumatic brain injuries and cognitive decline

TBI is typically associated with executive function, processing speed, and learning/memory-related functions. Injuries occurring in people younger than their early 20s affect the brain while it is still developing and therefore can potentially interfere with normal development, while older adults who experience injuries are less likely to recover fully from them. Additionally, it is not uncommon for people to experience more than one TBI. In fact, one of the greatest risk factors for TBI is history of a prior TBI. Depending on the severity of the TBI, risk for dementia is significantly increased later in life. The associations between TBI and the risk of specific dementia subtypes or neuropathological outcomes vary, but TBI occurring in older adults is associated with increased risk of dementia. While not a perfect dose-response relationship, this elevated risk for dementia increases as the number of TBIs a person experiences increases.

The mechanisms underlying the relationship between TBI and later-life cognitive decline are not well understood. Leading potential mechanisms linking TBI and susceptibility to later-life cognitive decline 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

There is evidence to suggest that mild TBI (loss of consciousness or post-traumatic amnesia lasting 30 minutes or less) in early life may increase a person's risk of cognitive decline and dementia in later life, making further inquiry into such associations justifiable. Variations of the *APOE* gene have also been evaluated to assess how a genetic trait may relate to TBI and cognitive decline. So far, the evidence is mixed, although the presence of the ε4 allele may contribute in some populations to the risk of long-term symptomatology after TBI, especially cognitive impairment and fatigue. The *BDNF* gene (which involves the production of a protein that promotes the growth and maintenance of neurons) has been associated with various types of neurodegeneration. One variant of the gene (the $BDNF^{Val66Met}$ allele) has been linked to an increased risk of experiencing TBI and is also being investigated for its association with memory deficits after TBI. In a study of a small group of military members, carriers of a different *BDNF* variant were found to have atrophy and less functional connectivity in the hippocampus after experiencing TBI compared with those who were homozygous for the typical form of *BDNF*.

Potential causes of traumatic brain injuries

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). More than 60% of adults have reported experiencing at least one type of ACE, and 1 in 6 have reported experiencing four or more types. Compared with other

groups, women and those from underrepresented racial/ethnic groups have a greater risk of experiencing four or more types of ACEs. ACEs can disrupt neurodevelopment and have a profound impact on health and well-being throughout the lifespan. They are associated with poor health outcomes, including heart disease, cancer, and diabetes. Some outcomes associated with ACEs, such as mental illness, homelessness, substance abuse, and violence, can increase a person's risk of experiencing TBI and thereby increase their risk of cognitive decline. Other outcomes, such as an increased risk of HIV/AIDS, toxic stress, and chronic health conditions, can directly increase a person's risk of cognitive decline in later life and potentially compound the adverse neurobiological effects of TBI.

Violence

TBI can also occur through physical violence to the head. In the United States, abusive head trauma is a leading cause of child abuse deaths in children under the age of 5. Domestic violence, intimate partner violence, and elder abuse are other forms of violence that can result in TBI, as the use of physical force can cause injury to the victim's head, neck (including through strangulation), and face. One study reported that 30% of domestic violence survivors reported losing consciousness at least once, and 67% reported residual problems that were potentially related to a head injury. Loss of consciousness can be associated with hypoxia and anoxia, potentially causing ischemic brain injury, as well as the release of brain-specific and neuroinflammatory molecules into the bloodstream, reflecting multiple brain injury processes. TBI may be underdiagnosed among survivors of domestic violence because symptoms can mimic those associated with mental illness, such as depression or anxiety. Additionally, survivors of domestic violence and TBI may appear to have behavioral issues, such as problems keeping appointments, and poor health.

Falls

People over the age of 75 are about twice as likely as people under the age of 75 to experience falls and associated complications. Additionally, about 10% to 25% of falls in this population cause fractures, with hip fractures being the most common. Individuals in this age group are more likely 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 aftereffects can be achieved by targeting the circumstances precipitating a TBI event. Such prevention should be encouraged to preserve brain health in addition to overall general health. Targets must include children, young and middle-aged adults, older adults, and at-risk communities. Social-ecological models and approaches are needed for prevention to be effective in the public health space. Treatment for risk factors associated with TBI will have a large and compounding effect on preventing cognitive decline and impairment in older adulthood.

Discussion

TBI can occur in individuals of any age, and it can increase the risk for cognitive decline in older adulthood. Therefore, interventions to reduce the risk of cognitive decline associated with TBI should be focused on all age groups, with interventions targeted at the leading causes of TBI by age group. For example, to prevent injuries from accidents in children, playground surfaces should be safe, soft, and composed of appropriate materials (such as sand or wood chips). Children should also 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.

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Tobacco and Alcohol and the Risk of Cognitive Decline and Dementia:

Choices Make a Difference

What Is Already Known

A relationship between tobacco use and an increased risk of cognitive decline is well established. There is evidence that current smoking increases the risk of cognitive decline and dementia, quitting smoking may reduce a person's risk of cognitive decline to levels comparable to those of people who have never smoked, and heavy smoking in middle age may as much as double a person's risk of dementia in later life. However, the relationship between alcohol consumption and the risk of cognitive decline is less clear. The relationship may differ in people at different life stages, and the amount of alcohol a person typically consumes may also factor into the relationship. Thus, recommendations for people who drink will not be as straightforward as those for people who currently smoke.

Background and Evidence Base

Evidence for a relationship between tobacco use and cognitive decline

Results of a longitudinal study published in 2018 support a relationship between tobacco use and cognitive decline, and also show that long-term smoking cessation can reverse a person's risk of developing cognitive decline. In the study, when compared with continual smokers, long-term quitters and never smokers had a decreased risk of both overall dementia and vascular dementia. Never smokers additionally had a decreased risk of Alzheimer's disease compared with the risk for continual smokers.

Another study that examined the relationships among smoking, dementia, and death reported that current smoking increased the risks of both dementia and death and that some of the increased risk of death was attributable to dementia. The authors concluded that smoking cessation at any age might reduce these risks. Finally, it has been shown that even environmental exposure to tobacco smoke can adversely affect cognitive abilities. A study that used data from the Third National Health and Nutrition Examination Survey (NHANES III) reported an inverse association between environmental tobacco smoke exposure and cognitive deficits among children, even at extremely low levels of exposure.

Evidence for a relationship between alcohol consumption and cognitive decline

There is a more nuanced relationship between alcohol consumption and cognitive health than exists for tobacco use and cognitive health. Relationships vary depending on a person's age and the amount of alcohol a person typically consumes. The Lancet Commission reported that people who consumed more than two alcoholic beverages per day in midlife had an increased risk for later dementia, slower reaction times, and greater brain atrophy. Furthermore, a 2013 review concluded that excessive and prolonged use of alcohol may lead to permanent structural and functional damage. It also reported that abstinence after alcohol abuse may lead to recovery of some lost cognitive ability, perhaps due to remodeling in the brain that may overcome some of the structural damage caused by alcohol abuse.

In contrast, a 2009 systematic review and meta-analysis reported that individuals with light to moderate alcohol consumption had reduced risk of dementia and cognitive decline compared with both nondrinkers and heavy drinkers. Similarly, participants in a randomized trial who consumed a Mediterranean diet with an optional glass of wine per day showed small but statistically significant improvements in some measures of cognitive performance.

Although the relationship between alcohol use and cognitive health in adults remains unclear in some ways, adolescents are a group for whom the effects of alcohol use, and particularly of heavy alcohol use, are more straightforward. The Lancet Commission reported that alcohol abuse during adolescence was associated with particularly negative cognitive consequences, including brain structure abnormalities, deficits in memory and learning, poor academic performance, and disruption of brain maturation and plasticity. Furthermore, alcohol abuse in adolescence has been found to predict an increased risk of alcohol abuse later in life.

Implications for Public Health

Tobacco use and alcohol consumption are modifiable risk factors associated with the development of cognitive decline and dementia. With respect to tobacco use, it is clear that smoking increases the risk of dementia and

that smoking cessation can reduce this risk. Indeed, stopping smoking may be one of the best ways to reduce the risk of dementia in later life. Thus, current smokers comprise a group that could be targeted for smoking cessation interventions to reduce the risk of cognitive decline. People at any age who smoke, and especially those with young children in their household, should be provided with smoking cessation interventions. Smoking cessation provides many health benefits in addition to those for cognitive health.

With respect to alcohol consumption, the current evidence suggests that both a person's age and the amount of alcohol a person typically consumes affect the risks of cognitive decline and dementia. Adolescents who drink alcohol, and particularly those who abuse it, are a group that should be targeted for intervention, as alcohol abuse is particularly detrimental in this age group. Individuals at any life stage who habitually consume an excessive amount of alcohol should also be targeted for intervention, as alcohol abuse is strongly associated with cognitive decline. However, among adults at midlife and older, light to moderate drinking may protect against later cognitive decline. Therefore, while this group may not need intervention to reduce alcohol consumption, it may be appropriate to warn them of the risks associated with excessive drinking, emphasizing the importance of maintaining alcohol consumption in a beneficial range so that it does not develop into a problematic level of drinking.

Discussion

The evidence for a negative impact of tobacco on cognitive health, both among those who smoke tobacco and among those with environmental exposure to tobacco smoke, is strong. Because the relationship between cognitive health and alcohol consumption is less clear, whether intervention to reduce alcohol consumption is recommended should be considered in the context of a person's age and the amount of alcohol he or she typically consumes.

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Diet, Nutrition, and Cognition:

Seeing Food as Medicine

What Is Already Known

For overall health and well-being, the American Heart Association and the U.S. government's Dietary Guidelines for Americans recommend a diet that emphasizes plant-based foods, such as fruits, vegetables, whole grains, nuts, and seeds, as well as low-fat dairy, fish, and lean meats. Red meats, sodium, saturated fats, sugars, and highly processed foods should be limited or avoided. This pattern of eating is similar to the recommendations of both the "Mediterranean-style" and "Scandinavian-style (Nordic)" diets (although specific aspects differ) and is also associated with cognitive health. The World Health Organization (WHO) recently recommended a Mediterranean-style diet for all adults with normal cognition or mild cognitive impairment as one factor that may reduce the risk of cognitive decline or dementia.

Background and Evidence Base

Diet may have direct and/or indirect effects on cognitive health. Nutrients like vitamins, fiber, antioxidants, salt, fat, and fiber could directly affect cognitive health through effects on antioxidation, anti-inflammation, and endothelial and mitochondrial function. These nutrients may also indirectly affect cognitive health through cardiovascular-related effects from diabetes, dyslipidemia, hypertension, obesity, and/or homocysteine levels.

However, the effects of diet on overall health can be difficult to study. A person's diet consists of the individual foods eaten in a given day, along with the individual nutrients in each food and the interactions of these components. But, diet also comprises current dietary patterns against the background of one's dietary pattern history, sociocultural identity, and demographic characteristics. Thus, it is difficult to isolate a specific component of diet that is associated with a specific outcome. Because of methodological issues, randomized controlled trials with dietary interventions are difficult to perform. Accordingly, while there have been some randomized controlled trials, most studies on the relationship between nutrition and cognition have been observational.

Research related to nutrition and cognition generally includes studies of single nutrients, studies of food and beverage groups, and studies of overall dietary patterns.

A recent meta-analysis examined evidence from 15 randomized controlled trials that assessed the effect of interventions with a major dietary component on cognitive function or incident dementia in cognitively normal adults. The results showed that intervention with a major dietary component improved cognitive function.

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

Another recent review summarized research studies that explored the effects of diet on cognition. The review included an examination of studies on the effects of specific nutrients, such as vitamins and minerals, on cognitive health. It concluded that most studies showed no effect, although some reported protective effects of certain vitamins, antioxidants, and macronutrients (e.g., vitamins from the B group, vitamin E, and fatty acids). It is important to note, however, that current guidelines recommend getting nutrients from whole food sources, *not* from dietary supplements. The WHO guidelines strongly recommend *against* the use of supplementation as a means of reducing risk of cognitive decline because there is no evidence of benefit. However, multivitamins may be more promising than individual vitamin supplements. The review also noted positive associations between cognitive health and consumption of specific groups of foods and beverages, such as red wine (in moderate amounts), coffee, tea, fish/seafood, some fruits, and vegetables.

Evidence for an association between eating patterns and cognition

Findings from many studies support the recommendation of a balanced pattern of eating, such as a Mediterranean-style diet, for cognitive and overall health. Although a recent review reported some positive effects of individual nutrients or food groups on cognitive health, it concluded that an association between nutrition and cognitive outcomes was stronger for balanced dietary patterns than for individual nutrients and food groups, possibly because of the cumulative beneficial effects of the ingredients in these diets. The authors of the review highlighted the positive effects of a Mediterranean-style diet on cognitive health and also noted benefits associated with the Dietary Approaches to Stop Hypertension

(DASH) and Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) diets.

Evidence for an effect of multi-domain interventions on cognitive health

The Finnish Geriatric Intervention Study to Prevent Cognitive Impairment and Disability (FINGER) investigated whether multi-domain interventions that targeted several risk factors and mechanisms simultaneously would have a significantly positive effect on cognitive health. The study included 1,260 older adults at risk of cognitive decline. The study's interventions included exercise, cognitive training, social activities, diet, and vascular risk monitoring compared with a control intervention of standard health advice. The trial emphasized using small but effective changes, setting personal goals, and group sessions that covered topics such as diet and the brain, cooking, and grocery shopping. The dietary intervention was a Scandinavian-style diet that recommended reducing meat consumption, eating fish 2 to 3 times per week, and eating at least 500 grams of local fruits and vegetables per day. Results from the FINGER trial showed that people's baseline diet, which represented their general diet over a period of many years, predicted change in cognition in both the intervention and control groups over time. Importantly, however, dietary changes made during the study were associated with cognitive changes, and dietary improvement was significantly associated with beneficial changes in executive function. These findings show that when dietary changes are made along with other risk reduction strategies —and when people receive ongoing support related to these changes — people can make dietary changes that significantly benefit their cognitive health.

Implications for Public Health

The study results noted above highlight the health benefits of a "generally healthy" or Mediterranean-style diet for all adults, so changing to a healthier or Mediterranean-style diet should be recommended to anyone whose diet consists largely of items that should be avoided. However, while it is clear that food plays an integral role in a person's health, diet is just as integrally connected with a person's social and cultural experience. It is important to consider a person's existing diet and recommend manageable but important changes to bring the diet closer to the guidelines and make it easier for a person to adhere to the changes. Additionally, as with other risk reduction strategies, many people may need ongoing support to help maintain dietary changes. Therefore, there is a need for culturally-sensitive, community-based programs that offer ongoing support to help people sustain healthy changes to their diets.

Discussion

Research to date shows that what people eat has a significant effect on their cognitive health and that a Mediterranean-style or Scandinavian-style diet may benefit cognitive health. Current guidelines recommend these diets for all adults in general and specifically for adults at risk for, or with, cognitive decline. However, research on diet and cognition can be challenging because of multiple methodological issues, such as how best to measure nutrient levels, how to control for a person's baseline diet, and how to determine exactly what a person has eaten. Additionally, there are still many areas for which current information is not sufficient to make strong conclusions, such as how long dietary interventions need to last in order to be effective, whether there is a certain stage of life in which dietary intervention is most important, and whether nutrition from supplements (in contrast to nutrition from whole foods) can benefit people with nutritional deficiencies.

Finally, dietary interventions should be sustainable both for the individual and for the environment. They should be adapted to be culturally relevant and culturally appropriate for diverse communities. And attention must be paid to addressing disparate access to appropriate foods.

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DEMENTIA RISK REDUCTION

Sleep and Dementia:

Sleep is Important for a Healthy Brain

What Is Already Known

As people age, sleep disturbances increase and sleep quality decreases. 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). Also, less efficient sleep and taking longer to fall asleep have been associated with an increased risk of mild cognitive impairment and dementia. And, a study of adults with European ancestry who did not have dementia showed that sleep duration was significantly reduced (by 1.9 hours) in those with a genetic predisposition to Alzheimer's disease, suggesting that sleep duration might be useful as a marker for future cognitive decline.

Findings from animal studies suggest a potential mechanism for explaining the relationship between sleep and cognitive function. Two studies in mice showed that, during sleep, the brain cleared harmful substances (such as amyloid-β, a peptide found in the brains of people with Alzheimer's disease) through the lymphatic system. 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 awake mice did. Together, these findings suggest that when sleep is poor, the brain is not able to clear accumulated toxins that have been associated with 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 shown to be related to cognitive decline and dementia risk. Sleep-disordered breathing (e.g., sleep apnea) increases with age, and estimates suggest about 25% of older adults will experience it by the age of 75. 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 examined the effect of treating sleep disruptions as a means of reducing a person's risk of developing cognitive impairment. A study that evaluated the effect of continuous positive airway pressure (CPAP) therapy on cognitive function found no difference in cognitive function after six months between the group that used CPAP therapy and the group that used a fake CPAP therapy. In contrast, a study that included one group receiving six weeks of CPAP therapy and another group receiving three weeks of CPAP therapy showed improvement in both groups on tests of cognitive

function after three weeks of CPAP therapy, suggesting that improvement can be seen in a very short period of time. A small study showed that CPAP therapy reduced amyloid-β accumulation. Other potential therapies for improving sleep (such as cognitive-behavioral therapy and light therapy) have been insufficiently studied to assess any effects on cognitive outcomes. Thus, 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

There is currently a lack of strong evidence that treating sleep issues can reduce the risk of cognitive decline and dementia. However, it is known that improving sleep has beneficial effects on other health outcomes (including mortality, cardiovascular disease, inflammation, obesity, and others), some of which are themselves risk factors for cognitive decline and dementia. Standard sleep hygiene practices should be recommended for individuals wanting to improve their sleep. Such practices include daytime exercise; avoiding afternoon caffeine intake; avoiding 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 focused on behavioral interventions 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.

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 is difficult, as it cannot rely on self-report but requires performance-based measurement of sleep. New technology-based tools permit more precise measurement of sleep quality outside of the laboratory setting which may improve the identification of individuals more likely to 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. This research is examining whether improved treatments for the sleep-related disorders associated with these underlying conditions may secondarily reduce the risk for dementia.

Until the evidence is clearer on whether targeting sleep disruptions is effective at reducing the risk of cognitive impairment, individuals experiencing sleep issues should be encouraged to use standard sleep hygiene practices and/or current mobile health technologies (such as smart phone apps), as these practices are noninvasive and inexpensive. While it is not clear if improving sleep quality affects the risk of cognitive decline, improving sleep has been shown to improve other health outcomes, such as cardiovascular diseases, mortality, and inflammation.

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DEMENTIA RISK REDUCTION

Sensory Impairments and Cognition:

Does Intervening on One Improve the Other?

What Is Already Known

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. Age-related dysfunctions in vision, hearing, and smell have each been linked separately to cognitive impairment and cognitive decline. 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 — the reduction in function of one or more of your senses — is a highly prevalent age-related condition. A recent study showed that deficits in multiple senses are prevalent among older Americans, supporting the idea that a common process may underlie the aging process of all five senses. Age-related hearing impairment most commonly occurs through nerve degeneration or damage in the inner ear, but damage to other parts of the auditory system can lead to hearing impairment as well. Similarly, vision has multiple components, which are generally assessed separately when vision is tested. The five most common causes of vision impairment in older adults are age-related macular degeneration, glaucoma, cataract, diabetic retinopathy, and refractive error. Olfactory impairments are also common in older adults; as many as half of older adults have some loss of smell, and the prevalence increases with age.

The mechanisms that link sensory impairment to cognitive decline are likely complex. Some processes, such as vascular disease, may concurrently damage both the brain and sensory organs. Additionally, aging may contribute to a bidirectional relationship between peripheral sensory function and central cognitive function: age-related changes in sensory function may worsen cognition, while age-related changes in brain function may alter how sensory input is perceived. Finally, sensory impairments may lead to cognitive and psychological outcomes, such as excess cognitive load (when your working memory receives more information than it can handle), impairments in brain structure and function, social isolation, depression, and reduced activity – all of which in turn can result in 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.

Vision: Two recent meta-analyses provide strong 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.

Hearing: Hearing loss has also been strongly 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.

Smell: A 2016 longitudinal cohort study of more than 1,400 cognitively normal adults linked olfactory impairment to incident amnestic mild cognitive impairment and with 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.

There is currently insufficient evidence to determine whether preventing disease or injury to the sensory organs can improve cognitive trajectories or prevent/delay dementia. There is more information on whether treatment to improve or accommodate already-occurring impairments in vision or hearing has an effect on cognitive outcomes. A recent 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 in the years after they first began using hearing aids relative to their rate of memory decline in the years before they first used hearing aids. 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

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 were recently updated to 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.

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 evidence overall is mixed, and stronger evidence is needed before concluding that sensory interventions improve cognitive outcomes in people with dementia. 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.

Many Americans do not receive appropriate sensory care, regardless of their dementia status. 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 group.

Discussion

There are many areas in which additional research is needed on understanding 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. Current randomized clinical trials are seeking to determine whether specific interventions (such as hearing aids and cataract surgery) can improve cognitive outcomes in adults with possible 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 targeted at populations at greatest risk of sensory and cognitive impairment. Screening for loss of smell may help identify individuals at risk of cognitive impairment, but such screening is less common, and many older individuals are unaware of losses in this ability.

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DEMENTIA RISK REDUCTION

Social Engagement and Cognition:

A Key Component of Successful Aging

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 pertaining to a common interest, activity, or goal. There is no obligation, and no one is paid for these interactions. Surgeon General Vivek Murthy has focused on the deleterious impacts of loneliness — a related construct to social engagement — on health, including among older adults. Research suggests that loneliness can shorten a person's life by an estimated 15 years — the equivalent of smoking 15 cigarettes per day.

Others have explored whether midlife social engagement may help reduce risk for later-life dementia. Social engagement may protect cognitive function and reduce the risk of cognitive decline throughout the life course and may provide benefits to many individuals, including persons with dementia. Among older individuals with a genetic risk for dementia through APOE-ε4 (an APOE gene allele associated with increased risk for dementia in some populations), rates of dementia were lower among those who frequently engaged in social activities.

Background and Evidence Base

Social engagement is considered a key component of successful aging and an integral part of overall health. Although it may have less enduring impacts in midlife, social engagement appears to minimize dementia risk among older adults.

One possible mechanism by which social engagement may facilitate cognitive function and protect against cognitive impairment is through a buildup of cognitive reserve, which refers to increased neuronal connections and more efficient processing in the brain that enable a person to continue to carry out cognitive tasks despite brain changes. Greater cognitive reserve may allow the brain to maintain function and cope with conditions associated with cognitive impairment, such as cerebrovascular disease and atrophy. It evolves over the lifespan and theoretically maybe promoted by interventions at all stages of life.

Of note, social engagement and loneliness can coexist. While loneliness and social isolation (the lack of social connections) are both associated with increased rates of cognitive decline, social engagement is the converse of social isolation.

Implications for Public Health

Promotion of social engagement must be culturally compatible and specific. Priority populations or groups that may be at increased risk for social isolation/lower levels of social engagement include:

- Women, who tend to live longer than men, and are more likely to be caregivers or widows.
- Members of underrepresented racial and ethnic communities, who may be at increased risk of social isolation due to the impact of migration and immigration on social ties and subsequent engagement, as well as the effects of discrimination across the lifespan. They also have higher rates of engaging in risky behaviors during social engagement (e.g., smoking).
- Rural residents may be more likely to experience social isolation stemming from poor transportation infrastructure and subpar digital connectivity.
- Lesbian, Gay, Bisexual, and Transgender (LGBT) individuals may be subjected to stigma and/or lack a family support network, leading to less social engagement.

It remains important to understand intersectional populations in relation to social engagement, such as older Black and other men, as they have been largely underincluded in related studies. Also still needed are multi-site collaborations to examine and scale up existing community-based strategies to facilitate and foster social engagement, especially among priority populations (e.g., leveraging the efforts of Outreach, Recruitment, and Engagement Cores in the NIA-funded Alzheimer's Disease Research Centers). Once priority populations are identified, strategies and interventions may be further developed, tailored, and tested accordingly.

For strategies and interventions designed and implemented in the post-COVID era, hybrid formats (i.e., social engagement activities with both an in-person and virtual option) are likely needed for the foreseeable future. In

addition, as this issue has gained traction internationally, programs from other countries are a potential source for adaptation and testing in the United States for evidence of efficacy.

Discussion

Few randomized clinical trials or intervention studies exist that specifically tease out social engagement as an independent variable related to cognitive function/impairment. Such studies are needed to address directionality of impact (e.g., does social engagement affect cognitive function, or does cognitive impairment affect social engagement), the distinct role of social engagement and its collective impact with physical and cognitive activity, and the importance of social engagement in midlife and from mid- to late-life.

Studies are still needed to determine 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. 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 designed.

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