PUBLIC HEALTH CENTER OF EXCELLENCE

ALZHEIMER'S ASSOCIATION

DEMENTIA RISK REDUCTION

DIABETES, OBESITY AND COGNITION:

WHAT WE DO NOW AFFECTS HOW WE THINK LATER

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

ALZHEIMER'S® ASSOCIATION

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

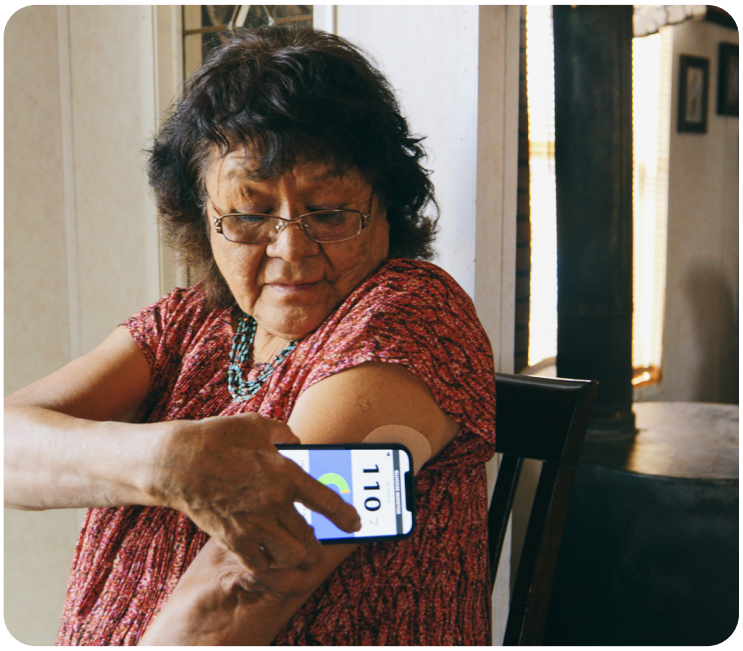
Providing communities with greater awareness of the need to intervene and prevent obesity through education and community-based programs promoting better diet and greater physical activity is likely to result in long-term preservation of memory and other cognitive functions. Greater efforts to target 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.

DIABETES, OBESITY AND COGNITION

Website: **alz.org/riskreduction**
Email: **CenterofExcellence@alz.org**

Selected References

1. Shalev D, Arbuckle MR. Metabolism and memory: obesity, diabetes, and dementia. Biol Psychiatry 2017;82: e81-e83.
2. Livingston G, Huntley J, Liu KY, et al. Dementia prevention, intervention, and care: 2024 report of the Lancet standing Commission. Lancet 2024;404:572-628.
3. Kim KY, Ha J, Lee J-Y, Kim E. Weight loss and risk of dementia in individuals with versus without obesity. Alzheimer's Dement 2023;19:5471-5481.
4. Lee M, Whitsel E, Avery C, et al. Variation in population attributable fraction of dementia associated with potentially modifiable risk factors by race and ethnicity in the US. JAMA Netw Open 2022;5(7):e2219672.
5. Walsh S, Govia I, Wallace L, Richard E, Peters R, Anstey KJ, Brayne C. A whole-population approach is required for dementia risk reduction. Lancet Healthy Longev 2022;3(1):e6-e8.
6. Leshner AI, Landis S, Albert M, Barnes LL, Blazer DG, Espeland MA, Harden JT, Kawas CH, Laird NM, Langa KM, Larson EB, Luchsinger JA, Petersen RC, Sacco RL, Seshadri S, Snyder LB, Yaffe K. Preventing Cognitive Decline and Dementia: A Way Forward. Washington, DC: The National Academies Press, 2017.

PUBLIC HEALTH CENTER OF EXCELLENCE
ALZHEIMER'S ASSOCIATION
DEMENTIA RISK REDUCTION

ALZHEIMER'S® ASSOCIATION

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.

For the full list of references, please contact us at CenterOfExcellence@alz.org.