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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.