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From Diabetes to Dementia

It may be a bit late for our residents, but you should be very interested in your hemoglobin A1c; so says the May 24th edition of *Diabetologia*. The journal just released the results of a prospective atherosclerosis trial. Subjects with prediabetes (A1c 5.7 – 6.4%) at the start of the trial were 12% more likely to develop dementia than those who began with normal A1c values.

The trial was large, enrolling 11,656 subjects who were followed for a median of 24.7 years. The average age at the outset was 58.6 years, and 19% went on to develop dementia. Early onset of prediabetes (before age 60) corresponded to a greater incidence of both diabetes and dementia down the road.

Prediabetics who became diabetic before age 60 were nearly three times as likely to develop dementia. Those developing diabetes from 60-69 were 1.73 times as likely to become demented, and those diagnosed with diabetes in their 70s experience dementia 1.23 times as often.

Vascular damage related to glucose toxicity is thought to be the principal mechanism responsible for neurological damage. Staving off diabetes for longer or preventing it altogether is likely to help prevent cognitive loss. Lifestyle modifications such as improved diet and increased exercise are a good start. BP, cholesterol, and weight control are also critical. It will be interesting to see if recommendations for early treatment with metformin, GLP-1 agonists (Ozempic®, etc.), or SGLT2 inhibitors (Jardiance®, etc.) emerge to prevent progression to diabetes then dementia via the A1c lowering action of these agents.

Cognitive A-Salt

We know that many roads lead to dementia. Many causes were listed in the previous article. Some other contributing factors include smoking, excessive drinking, and hearing loss. A previously unrecognized substance, salt, may be bad for our brains (in addition to our heart and blood vessels).

A prospective Chinese study (*J Am Medical Directors; Oct* 2022) divided 2,041 healthy community residents (60 years+) into four groups based on salt intake: low (<6g/d), mild (6-9g/d), moderate (9-12g/d), and high (>12g/d). To confirm the accuracy of the groupings each participant provided 7 consecutive days of 24-hour urine samples. Urinary salt output is the most reliable indicator of intake.

Cognitive function was assessed using Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), and the Dementia Rating Scale (DRS). Subjects were followed for an average of 11.4 years and cognitive function declined in all groups over this time. Compared to the low salt intake group, cognitive impairment risk was higher by 75% in the mild group, 180% in the moderate group, and 330% in the high intake group. All differences were statistically significant compared with low intake.

Not surprisingly, BP values were higher with increased salt consumption. The results were adjusted to eliminate BP as a factor, however. Consumption was substantial in the lowest groups and extreme in the high-intake groups. This may have exaggerated the results. The WHO recommends limiting dietary salt to just 5g/day. Vascular studies in mice show that high salt concentrations can damage cerebral endothelial cells. This may in turn cause damage to nerve cells leading to cognitive dysfunction. The adage, "everything in moderation" seems to apply to both sugar and salt.

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