Vitamin B1 deficiency a key factor in the development of alcohol-related dementia

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A common consequence of chronically high alcohol consumption is a decline in cognitive function, which can even progress to full-blown dementia. However, we do not yet fully understand how alcohol damages

the brain. A research group led by Stephan Listabarth from MedUni Vienna's Department of Psychiatry and Psychotherapy, Division of Social Psychiatry, has now developed a hypothesis whereby iron deposits in the brain—resulting from alcohol-induced vitamin B1 deficiency—can be regarded as key factors in cognitive decline. The work has now been published in the leading journal *Alzheimer's and Dementia*.

In Austria, around 5% of the population are alcohol dependent from the age of 15 onwards. This means that approximately 365,000 people are affected by the dangerous health consequences associated with high alcohol consumption. One of these consequences is a decline in cognitive function, especially memory and abstraction. This is then referred to as alcohol-related dementia. However, researchers do not yet fully understand the exact pathomechanism by which the brain is damaged by alcohol.

Researchers Stephan Listabarth, Daniel König and Benjamin Vyssoki from the Department of Psychiatry and Psychotherapy, Division of Social Psychiatry at MedUni Vienna and Simon Hametner from MedUni Vienna's Department of Neurology, Division of Neuropathology and Neurochemistry, have now advanced a plausible hypothesis to explain alcohol-induced brain damage: Cognitive deterioration is caused by iron deposits in the brain, but the administration of vitamin B1 could protect the brain from these deposits.

We know from neurodegenerative diseases that iron deposits in the brain are responsible for nerve tissue damage. These deposits can also be detected in specific regions of the brain (including the basal ganglia) in people who drink a lot of alcohol. The hypothesis advanced by the study authors now also offers an explanation as to why iron deposits are so prevalent in this patient group: High alcohol consumption results in elevated iron levels in the blood and vitamin B1 (thiamine) deficiency,

which, among other things, is important for maintaining the blood-brain barrier. If these two situations coincide, more iron will be deposited inside the brain, ultimately leading to oxidative tissue damage.

This newly described role of vitamin B1 in this process could represent a huge step forward in our understanding of the development of alcohol-related neurological damage and, in particular, could offer a new point of attack for preventive and therapeutic approaches. It would then be conceivable to give continuous vitamin B1 substitution in the future as a preventive measure.

The researchers believe it would also be useful to evaluate the use of drugs to reduce iron levels (e.g. chelators), as is already done in other neurodegenerative diseases. The authors of the current work have already started planning a prospective clinical study to validate the above-mentioned relationship between alcohol dependency, vitamin B1 deficiency and cerebral iron deposits and to provide a basis for further research in the field of alcohol-related dementia in the future.

More information: Stephan Listabarth et al. Does thiamine protect the brain from iron overload and alcohol-related dementia?, *Alzheimer's & Dementia* (2020). DOI: 10.1002/alz.12146

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