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Brain scans reveal a reproducible pattern linked to alcohol use severity

Across roughly 53,000 people, the MRI signal replicated in two cohorts but remained too weak for diagnosis

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A large brain imaging study has identified a reproducible structural pattern linked to the severity of alcohol use, but the effect was small and not strong enough to support individual diagnosis or prediction.

The research, published in Translational Psychiatry, analyzed MRI scans and alcohol-use data from three cohorts in Germany and the United Kingdom. The main discovery sample came from the German National Cohort, known as NAKO, with about 28,000 participants. The researchers then tested the same approach in the UK Biobank, with about 25,000 people, and in a separate German clinical cohort called FOR2107, which included about 1,900 participants.

The team used self-supervised deep learning to extract patterns from gray-matter volume maps in structural MRI scans. They then applied ridge regression to estimate alcohol consumption severity based on the Alcohol Use Disorders Identification Test-Consumption, or AUDIT-C, a widely used questionnaire that measures drinking behavior. The goal was to see whether brain-wide structural information could capture a stable signal related to alcohol use across large populations.

In the NAKO cohort, the MRI-derived score showed a statistically significant but weak correlation with measured AUDIT-C severity, with a correlation coefficient of 0.11. In the UK Biobank, the association was similar, with a correlation of 0.09. The researchers said that this replication across two very large population datasets suggests the signal is real, but modest. The average prediction error also remained substantial, which the authors said limits the score's usefulness for individual-level decisions.

The study found that the brain-derived score also lined up with several external markers associated with alcohol-related burden. Higher scores were linked to higher gamma-glutamyl transferase, a liver-related blood marker, and higher mean corpuscular volume, a blood measure that can rise with sustained heavy drinking. Higher scores were also associated with slower reaction time, a sign of weaker cognitive performance in this analysis.

The researchers also examined diagnosed alcohol use disorder in the UK Biobank using ICD-10 records. People in the highest fifth of the brain-score distribution had 2.28 times the odds of alcohol use disorder compared with those in the lowest fifth. That result suggests the structural pattern has clinical relevance at the group level, even though it is not accurate enough to classify individuals on its own.

The authors compared the alcohol-related brain axis with a similar model for depressive symptoms. They reported that the alcohol signal transferred across cohorts better than the depression signal under the same framework, which they said may reflect the cumulative biological effects of alcohol on brain structure. Even so, the study did not show that alcohol caused the brain differences. It identified an association, not a direct cause-and-effect relationship.

Several limitations could affect the findings. In the UK Biobank, questionnaire data and MRI scans were often collected far apart in time, with a median gap much larger than in NAKO. That timing mismatch may have weakened the link between reported drinking and brain structure. The clinical FOR2107 cohort also used a different alcohol questionnaire, the full AUDIT rather than AUDIT-C, making that part of the validation more exploratory. In addition, blood markers such as gamma-glutamyl transferase and mean corpuscular volume are not specific to alcohol use alone.

The paper adds to a growing body of evidence that alcohol-related effects on the brain are distributed rather than confined to a few isolated regions. The authors said classical region-by-region approaches may miss these subtle patterns, while whole-brain representation learning may be better suited to detect them in very large datasets. Still, they stressed that the observed associations were weak and that structural MRI should not be treated as a stand-alone tool for screening or diagnosing alcohol problems in individual patients.

That caution is likely to matter beyond clinical research. For producers and sellers of beer, wine and spirits, the study adds population-level evidence that alcohol use may leave detectable traces in brain structure, which could shape future public health debates, risk communication and product policy. At the same time, the findings do not justify using brain scans to make claims about any one consumer, and they do not support simple causal conclusions about moderate or occasional drinking based on an individual MRI.