

Scientists untangle links between our genes and intake of alcohol and of salt

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Scientists have shed light on the complicated relationship between the makeup of our DNA and how much alcohol we drink.

In another study, they have explored the links between our genes and our intake of salt.

Genetic markers linked with alcohol intake

In the first study, published in *Nature Human Behaviour*, the international team, led by Imperial College London, identified new [genetic markers](#) associated with alcohol intake.

After analyzing data from around 500,000 people—most aged between 40-69—the team identified 46 new genetic markers linked to how much alcohol people drink.

The researchers found these genetic factors could account for seven percent of the variation in people's total alcohol intake. Those with the lowest alcohol-related genetic risk drank about one third less of a standard drink per day (2.6g of alcohol) compared with those with the highest.

The team also identified genetic pathways shared between alcohol intake and brain networks, in particular networks associated with psychiatric disorders such as schizophrenia. This could suggest that a person's alcohol intake and their risk of schizophrenia may be influenced by some of the same genes.

Professor Paul Elliott, lead author of the study from Imperial's School of Public Health, said: "This study suggests the amount we drink is not just socially determined, but also has a biological basis."

He continued: "Although we already knew there was an association between schizophrenia and alcohol drinking, this research suggests there may be some joint genetic mechanism that leads a person to drink more, as well as increase their risk of schizophrenia."

The paper also revealed one particular genetic variant was linked to size of a brain region called the putamen, which was in turn linked to alcohol intake.

Professor Elliott added that the research was based on data from people with European ancestry, and so data from other ethnicities should also be investigated. But the research furthers our understanding of our complicated relationship with alcohol:

"Excessive alcohol consumption is a major public health problem, and associated with around one in 20 deaths worldwide. If we understood more about the biology of why we drink alcohol, we may be able to understand more on how to effectively deal with alcohol issues."

Genes associated with salt intake

In another study published today in the journal *Nature Communications*, the same team investigated the genes linked to people's intake of sodium, the main component of salt, and potassium.

The research team investigated data from nearly 500,000 people, and assessed genes associated with sodium and potassium urinary excretion, which are measures of the amount of sodium and potassium in the diet.

Both sodium and potassium are crucial to many processes in the body, but excess sodium is linked to an increased risk of heart attack and stroke.

The team found 59 genetic markers linked to either sodium or potassium intake or both. The team found that many of these variants were also linked to lifestyle-related variables such as dietary habits, smoking, coffee and [alcohol](#) drinking..

The research also reported that genetic variants linked to sodium were linked to gene variants associated with obesity, lipid levels, high blood pressure and cardiovascular disease, and raised the possibility that sodium genetic variants are involved in temperature pathways in the regulation of blood pressure.

Professor Abbas Dehghan, lead author of the research from the School of Public Health, said: "Our study is the first to identify genetic variants linked to sodium and potassium in urine, which reflect dietary intakes. Our work gives us more insights into the link between [sodium](#) intake, [high blood pressure](#) and [cardiovascular disease](#) and reinforces [public health](#) messages of reducing salt intake to reduce cardiovascular risk."

More information: Evangelos Evangelou et al. New alcohol-related genes suggest shared genetic mechanisms with neuropsychiatric disorders, *Nature Human Behaviour* (2019). [DOI: 10.1038/s41562-019-0653-z](#)

Provided by Imperial College London

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