"Aggressive drunk" gene may protect carriers from obesity and associated risks

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A genetic mutation which makes its bearers more likely to behave impulsively while intoxicated may shield them from obesity and change the way testosterone impacts insulin resistance, indicates a study conducted at the University of Helsinki.

University of Helsinki researchers have previously demonstrated that a point mutation in a gene of serotonin 2B receptor can render the carrier prone to impulsive behaviour, particularly when drunk. Now the research group has established that the same mutation may shield its bearers from obesity and insulin resistance, both of which are associated with type 2 diabetes.

Published in a recent issue of the *Journal of Psychiatric Research*, the study focused on the insulin sensitivity, beta cell activity and BMI of 98 Finnish men between the ages of 25 and 30, all of whom had been diagnosed with antisocial personality disorder. The results indicate that carriers of a point mutation in a gene of serotonin 2B receptor had a lower BMI and higher insulin sensitivity than persons without the mutation. Normally, men with low testosterone levels are more susceptible to metabolic disorders, but among carriers of the point mutation, this tendency was reversed – lower levels of testosterone increased insulin sensitivity.

The results also suggest that men in their thirties with antisocial personalities may constitute a risk group for insulin resistance, and consequently type 2 diabetes later in life.

"It is fascinating to think that this receptor mutation which has been passed through the chain of evolution would impact both the brain as impulsive behaviour and energy metabolism," says psychiatrist, Dr Roope Tikkanen from the University of Helsinki, who led the study.

"We could speculate that the compound effect the mutation and testosterone have on energy metabolism may have been beneficial in the cool, nutrition-poor environment after the Ice Age, particularly for men with a high physiological level of testosterone – they would have survived with a lower calorie intake.

Simultaneously, the aggression associated with high levels of testosterone may have helped them compete for food."

In our modern society with ample food, the carriers of the mutation who have normal or low levels of testosterone may be better protected from metabolic illnesses relating to obesity, such as type 2 diabetes.

"One would assume that the effect would be particularly pronounced in women, who naturally have lower levels of testosterone than men," Tikkanen points out.

Over 100,000 Finns and more than 1,000 Finnish infants born every year are carriers of the point mutation in the serotonin 2B receptor. The intention is to study the national health implications of the results from the extensive FINRISKI research material through cooperation between Finnish, Swedish and American researchers.

"Our results will further highlight the importance of Finnish diabetes research," Tikkanen states.

More information: Roope Tikkanen et al. The effects of a HTR2B stop codon and testosterone on energy metabolism and beta cell function among antisocial Finnish males, *Journal of Psychiatric Research* (2016). DOI: 10.1016/j.jpsychires.2016.06.019

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