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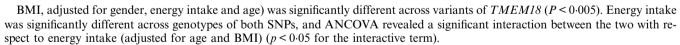
Interaction between bitter taste receptor gene TAS2R38 and obesity-related gene TMEM with respect to energy intake in an adult population

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Obesity is a multifactorial condition that arises from positive energy balance. Although low levels of physical activity in conjunction with excess energy intake are known contributors, other heritable factors may contribute to these⁽¹⁾. Variation in bitter taste receptor TAS2R38 has previously been associated with increased BMI, mainly in older females, in a number of independent studies^(2,3,4), potentially via increased preference for energy-dense foods. The gene TMEM18 also associates with increased BMI and body weight in other studies⁽⁵⁾, and is thought to act via impaired energy homeostasis⁽⁵⁾.

The aim of this analysis was to examine interactions between known SNPs within these genes, and reported nutrient intakes. Using data from the National Adult Nutrition Survey (NANS), approved by the University College Cork Clinical Research Ethics Committee of the Cork Teaching Hospitals, mean daily intake intakes were calculated for n 1500. Nutrient data were used to create a Healthy Eating Index (HEI) score, as per previously published methods⁽⁶⁾. Genetic data was available for a subset, and analysis was completed on n = 1083 with both nutrient intake and genetic data. Associations between genotypes in the aforementioned genes (rs10246939 in TAS2R38 and rs6548238 TMEM18) and anthropometric measures (BMI, % body fat) and dietary intakes (reported mean daily intakes) were examined via ANCOVA using SPSS v20 for Mac (IBM).

TAS2R38 (Val296lle) rs10246939	TMEM18 (rs6548238)	Mean Daily Energy /kcal		
		Mean	SD	N
T:T	C:C	2355-8	591-1	134
	T:C	2264.4	471.5	71
	T:T	3275.7	979.2	3
T:C	C:C	2320-1	584.4	214
	T:C	2308-1	559.4	98
	T:T	2397.7	712.6	12
C:C	C:C	2319-6	529.3	109
	T:C	2354-1	562.9	33
	T:T	2923-4	616-8	3



Mean HEI score, a measure of the overall quality of the diet, was 25·3 (9·5 SD) in the overall cohort. The mean HEI score was lower in the 'at-risk' individuals (24·2, 13·9 SD) compared to the lower-risk individuals (30·7, 13·9 SD), but not significantly so (P > 0.01). These data, although limited, support the idea that heritable variation in BMI and energy intake may be linked to differences in food choices. Considering that as much as 70 % of BMI variation may be heritable⁽¹⁾, improving our understanding of the role of genetic variation on food choice, and the identification of higher-risk individuals based on multiple gene-gene interactions, at earlier life stages may be key developments in the combat of obesity.

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