Proceedings of the Nutrition Society (2016), **75 (OCE3)**, E250 doi:10.1017/S0029665116002652

Summer Meeting, 11–14 July 2016, New technology in nutrition research and practice

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

E.L Feeney[1], B.A McNulty[1], J. Walton[2], A. Flynn[2] and E.R Gibney[1]
[1]*UCD Institute of Food and Health, University College Dublin, Dublin, Republic of Ireland and* [2]*School of Food & Nutritional Sciences, University College Cork, Republic of Ireland*

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 (Val296Ile) 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

BMI, adjusted for gender, energy intake and age) was significantly different across variants of *TMEM18* ($P < 0·005$). Energy intake was significantly different across genotypes of both SNPs, and ANCOVA revealed a significant interaction between the two with respect to energy intake (adjusted for age and BMI) ($p < 0·05$ for the interactive term).

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.

This work was supported by the Food Institutional Research Measure (FIRM) through the National Development Plan 2006–2011.

1. Maes HH, Neale MC, Eaves LJ (1997) *Behav Genet* **27**, 325–351.
2. Feeney E, O'Brien SA, Scannell AGM *et al.* (2011) *Proc Nut Soc*, **70**, 135–143.
3. Tepper BJ, Koelliker Y, Zhao L *et al.* (2008) *Obesity* **16**, 2289–2295.
4. Dotson CD, Shaw HL, Mitchel BD *et al.* (2010) *Appetite* **54**, 93–99.
5. Almen MS, Jacobsson JA, Shaik JHA *et al.* (2010) *BMC Med Genet* **11**, 58.
6. McCullough ML, Feskanich D, Stampfer MJ *et al.* (2002) *Am J Clin Nutr*, **76**, 1261–1271.