Obesity Risk Based on Genotype: Research Summary

"The FTO gene and measured food intake in children" investigated the impact of the FTO gene on children's eating behaviors. Specifically, the study examined the FTO gene as it relates to satiety and food intake among children (1). The researchers tested their hypothesis that "FTO genotype status" is correlated to a child's consumption of a palatable food item after already consuming a meal (1). It was predicted that a child with higher-risk FTO alleles (AA homozygotes or AT heterozygotes) would eat a larger portion of the palatable food item as compared to a child with the lower-risk FTO alleles (TT homozygotes) (1).

Participants of the investigation were between the ages of 4 and 5 years (1). The study included 131 children with known DNA genotype status (1). The children were grouped based on genotype: AA, AT, and TT (1). In a home visit, food intake was evaluated via the "'eating in the absence of hunger' (EAH) paradigm" (1). The children ate a typical home-cooked meal prepared by their mothers. Within the following hour, the children were offered a variety of biscuits, sweet and savory. Each child was prompted "to eat as much as they liked for 10 min" (1). The plate containing the biscuits was weighed prior to and succeeding consumption in order to measure the quantity of biscuits eaten.

Results of the study confirmed the researchers' hypothesis that having a higher-risk FTO genotype would result in a larger consumption of a highly palatable food item following a meal. Food intake evaluated via the EAH test differed significantly across the 3 groups of genotypes. Findings revealed that children with the highest-risk genotype (AA) ate 25% more than children with the lowest-risk genotype (TT) (1). Food intakes of AT-heterozygote children fell between the AA and TT groups (1). The authors note that this finding was statistically significant even

after controlling for the children's BMI status (1). There was no correlation between FTO genotype and physical activity, based on parental reports (1).

The authors attributed the differences in food intake between genotype groups to FTO's impact on "responsiveness to satiety signals" (1). The final conclusion was written as follows: "In environments with multiple opportunities to eat highly palatable foods, those with higher satiety responsiveness are likely to be relatively protected from overeating, whereas individuals with a less responsive satiety system will be more likely to overeat and will therefore be at higher risk for weight gain" (1).

Overall, this study provides insight into emergent theories of nutritional genomics that suggest there is a genetic link to obesity risk. Wardle et al. employed a strong study design that examined how presence of the FTO gene affects eating patterns; the methods were strong considering eating behaviors were investigated among young children. As individuals age, eating patterns can be influenced by emotions and societal pressures; by studying children, the researchers were able to draw conclusions based on authentic, unbiased results. The findings revealed that the FTO gene may influence one's ability to manage their food intake via the regulatory driver of satiety. As a Registered Dietitian, it will be valuable to understand that weight status can be caused by elements outside of the patient's control, including genetic predisposition.

Reference:

1. Wardle, J., Llewellyn, C., Sanderson, S. *et al.* The *FTO* gene and measured food intake in children. *Int J Obes* 33, 42–45 (2009). https://doi.org/10.1038/ijo.2008.174