Variation of FTO gene linked to weight gain and obesity in children

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Researchers have discovered that children who do not have obesity, but who are at risk for the chronic disease due to a common genetic variant eat more, according to a new study to be published in the June 2019

issue of *Obesity*.

"Early identification of the physiology and behaviors that constitute early risk factors for subsequent weight gain will help inform best practices for intervention and prevention of obesity in children," said study author Michael Rosenbaum, MD, of Columbia University Irving Medical Center's Department of Pediatrics Division of Molecular Genetics. Rosenbaum is professor of pediatrics and medicine with appointments in both departments.

Rosenbaum and colleagues explain that many existing studies of children at risk for increased adiposity include those who already have obesity. The diversity of body fatness in such studies makes it extremely difficult to distinguish findings that are premonitory as opposed to the consequence of weight gain (i.e., cause vs. effect). "This study shows that even before the development of an obese phenotype, children at risk, in this case by virtue of a common genetic variant, exhibit increased food intake," said Rosenbaum.

Study organizers recruited 122 participants with the use of print and online advertisements directed towards parents of 5-10 year old children in the greater New York City metropolitan area. Children identified as having a body mass index at or below the 95th percentile were invited for further screenings. Children with medical conditions known to affect eating behaviors such as diabetes or eating disorders, youth taking medications or with severe food allergies were excluded from the study.

Parents of the participants completed a screening questionnaire and participated in an in-person evaluation. Researchers collected data on the height, weight and body composition of each child. Information was collected regarding medical, psychiatric and dietary (food preferences, consumption, and restrictions) histories for each child, as well as detailed family histories of body weight and its associated comorbidities. The

children were also asked to provide verbal liking ratings for a variety of foods to ensure that selections offered during the test meals would be acceptable.

Children were offered lollipops and asked to spit into a tube. The saliva was then analyzed for the number of doses of specific single nucleotide polymorphism of a gene called *FTO*. Approximately 70 percent of the population carries at least one copy of this polymorphism and it is known to be associated with an increased risk of excess adiposity. Researchers then tried to determine whether the presence of this common risk factor can be translated into a behavior that may be predictive of subsequent weight gain in children who had not developed obesity.

On the day of the study, parents were advised not to have their children eat or drink anything except for water after 10:00 p.m. the previous night. After researchers obtained measurements of height and weight, the children were provided a breakfast consisting of Cheerios or Kellogg's Cornflakes cereal and whole milk. Portion sizes were calculated based on calculated estimates of energy expenditure based on age, height, and weight.

Three and a half hours later, the children were offered a buffet lunch consisting of 28 food and beverage items such as items for making a sandwich, chicken nuggets, fruits and vegetables, salty snacks and desserts. The outcome variables were how much they ate and what they ate.

Researchers found that while there was no significant difference in body fatness between groups (0, 1 or 2 doses of the at-risk gene) each copy of the gene was associated with approximately 65 additional calories consumed in a single meal corrected for body weight. "Even though 65 calories is not a lot *per se*, if this pattern generalized to multiple meals

per week or day, this increased caloric intake can add up over time and may contribute to gaining excess weight," said Rosenbaum.

Tanja Kral, Ph.D., chair of the TOS Pediatric Obesity Section and associate professor in the School of Nursing and the Perelman School of Medicine at the University of Pennsylvania in Philadelphia was not part of the research but emphasized the importance of the study, stating "that the research sheds light into possible behavioral phenotypes for childhood obesity *before* obesity has developed. Once identified, it will enable us to design personalized behavioral interventions that target the individual components of the phenotype."

The study's authors added the report suggests a research model that could be used to study children at risk for other reasons. Rosenbaum and colleagues noted that further research is needed to address questions such as what behaviors are premonitory of subsequent weight gain and if methods exist to identify children at risk with the hope of targeted intervention to reduce the likelihood of subsequent obesity.

"The ultimate goal is to prevent the at-risk child or the child who has obesity from becoming an adult with obesity," concluded Rosenbaum.

More information: "The FTO Gene and Measured Food Intake in 5-10 Year Old Children Who Are Not Obese," *Obesity*, onlinelibrary.wiley.com/doi/10.1002/oby.22464

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