Does adaptive thermogenesis contribute to risk of weight regain?

Sustained weight loss may not result in a substantial, disproportionately low Resting Energy Expenditure. Weight loss is best achieved by reducing calorie intake and increasing physical activity. Although adherence to this life-style based strategy typically helps people achieve their desired weight loss goals, long-term weight loss and weight maintenance is often more challenging. In fact, regaining weight after successful weight loss is the common occurrence. National data (NHANES; 1999-2006) show that the vast majority of overweight and obese adults are unable to maintain long-term weight loss. Scientists have speculated that, in response to weight loss, metabolic adaptations serve to conserve energy and prevent further weight loss. One component of this process is called adaptive thermogenesis, defined as decreased resting energy expenditure that influences risk of weight regain. Studies have demonstrated that weight reduction in rodents is accompanied by a suppression of resting energy expenditure beyond what is predicted from changes in body mass and composition. This compensatory response to weight loss may contribute to subsequent weight regain. Studies have demonstrated that weight reduction in rodents is accompanied by a suppression of resting energy expenditure that influences risk of weight regain. However, evidence of this metabolic adaptive response in humans is inconclusive. In a recent study published in the October 2018 issue, Ostendorf et al., investigated the complex relationship between resting energy expenditure and long-term successful weight loss in humans.


Can genetic variation explain physiological response to starchy foods?

The number of copies of the salivary amylase gene influences the rate of digestion of starchy foods. This gene is involved in the hydrolysis of starch, which is a primary energy source for human beings. Studies have shown that individuals with more copies of the salivary amylase gene have faster starch digestion rates, which can affect blood glucose levels and overall energy intake. This genetic variation can explain why some individuals may have a higher risk of weight gain or obesity compared to others. Further research is needed to fully understand the role of genetic variation in metabolic responses to starchy foods.
Starch digestion and glycemic responses after consumption of starchy foods.

Genetic studies show that dietary changes have played an important role in human evolution. In some cases, these genetic adaptations have proven beneficial to human health, whereas others appear to increase disease risk susceptibility. For example, acquired nutrient tolerances and intolerances have the potential to alter metabolic pathways that can result in serious health consequences. Genetic variants, called single nucleotide polymorphisms, are the most common type of genetic variation. However, these are not the only form of genetic alterations. Copy number variants, which are structural variations involving the number of copies of specific regions of DNA, can also contribute to genetic variability. In humans, the salivary α-amylase gene shows extensive variation in copy number, but the physiologic significance is unclear. However, it is reasonable to speculate that individuals with a higher salivary α-amylase gene copy number might digest starchy foods faster, have greater glucose absorption (lower breath hydrogen excretion), and have higher postprandial responses (glycemia and insulinemia) compared with those with a low salivary α-amylase gene copy number. In an article published in the October 2018 issue, Atkinson et al. conducted four linked studies to better understand the physiological significance of salivary α-amylase gene copy number.


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Dairy fat consumed in the matrix of cheese does not appear to adversely affect blood lipoprotein cholesterol.

Fats sequestered within the matrix of cheese can significantly lower plasma cholesterol levels compared with the same components eaten within different food matrices.

Dairy fat contains a broad range of fatty acids, approximately 60% of which are saturated fatty acids. Saturated fats are known to increase LDL-cholesterol and subsequent cardiovascular risk. Therefore, current dietary guidelines suggest that less than 10% of total energy intake come from saturated fat. Given the high proportion of saturated fat in dairy products, healthy eating guidelines, such as the Dietary Guidelines for Americans, recommend low-fat dairy alternatives. However, the assumption that all saturated fats influence blood lipids in a consistent manner is now being challenged. Researchers recognize that in addition to the specific types of fatty acids in foods, the food matrix itself might be of particular importance. For example, previous studies of cheese consumption indicate that changes in blood lipids could be due to fat interaction with other nutrients (i.e., calcium or casein proteins) or to the cheese matrix itself. In an article published in the October 2018 issue, Feeney et al. investigated whether dairy fat consumed in different matrices would have differing effects on LDL cholesterol and other markers of metabolic health.


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Autistic traits, autism spectrum disorder, and food neophobia: Is there an association?

Early emergence of Food Neophobia behaviors and their linkage to autistic traits may impact health outcomes such as body weight.

Autism spectrum disorder (ASD) is a complex neurodevelopmental condition with early developmental origins. Individuals with ASD can have difficulty with social communications and social interactions and exhibit restricted, repetitive behaviors. In addition, many people with ASD experience sensory sensitivities and subsequently have adverse responses to the textures, colors, and smells of certain foods. Therefore, it is not surprising to learn that
ASD has been linked to atypical eating- and feeding-related problems including food neophobia (an unwillingness to try new foods). It has been estimated that as many as 90% of children with ASD exhibit unusual feeding behaviors. What remains unclear, however, is the extent to which associations between autistic traits and food neophobia (FN) extend into the broader population of children (without an ASD diagnosis) and the potential impact on health. More specifically, children who are highly fussy about eating more nutrient-dense and less palatable foods (e.g., fruits and vegetables) may actually be at increased risk of becoming overweight or obese compared to those who have a more varied diet. In a recent study published in the October 2018 issue, Wallace et al. examined ASD-control group differences in FN for children with ASD versus non-ASD children and associations between autistic traits and FN in a large community-based sample of children. Furthermore, the ability of FN and autistic traits to predict health-related outcomes such as body mass index (BMI) was also examined.


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