Gender Differences in Response to Food Deprivation: Implications for Eating Disorders

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Title: GENDER DIFFERENCES IN RESPONSE TO FOOD DEPRIVATION: IMPLICATIONS FOR EATING DISORDERS

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Learning Outcome: Young women respond to food deprivation by decreasing their food intake. Young men respond to such deprivation by increasing their subsequent food intake. This difference may underlie the much higher prevalence of eating disorders in young women than young men.

Text: There is a clear gender bias in the prevalence of eating disorders, as young women are nine times more likely than young men to develop anorexia and bulimia. One possibility is that women are more likely to develop a complex psychiatric disorder that includes depression, anxiety, mania, and obsessive-compulsive disorder and disordered eating among its symptoms. Alternatively, eating disorders may arise in response to dieting, and the continued food deprivation may eventually evoke psychiatric symptoms as a response to semi-starvation. To test these differential hypotheses, we deprived 20 healthy young women and 20 healthy young men of their dinner and then monitored their food intake the next day. The men compensated for the food deprivation by increasing their food intake on the following day. On the other hand, the women actually decreased their food intake the next day. Young women may have a greater propensity to continue to sustain dietary deprivation than men, and there may be a small proportion of these women who can continue their restrictive response to food deprivation to the point that they develop anorexia or bulimia. The onset of these disorders therefore may be due t o an inability to recognize and respond to normal hunger and satiety cues. We have gone on to show that treating anorexics and bulimics by teaching them to recognize their hunger and satiety cues allows 90% to be significantly improved, with 75% in full remission and 90% remaining in remission over a 5-year follow-up.

Funding Disclosure: Mandometer, Inc.

Title: REAL-TIME QUANTITATIVE PCR DETECTION OF GENETICALLY MODIFIED CORN IN COMMONLY CONSUMED FOODS IN INFANTS AND TODDLERS

Author(s): S. E. Helm, J. Garneau-Fournier, M. Coelho; Nutritional Science, Pepperdine University, Malibu, CA

Learning Outcome: Participant will be able to read a Nutrition Label for infant and children's foodstuffs and determine if bioengineered or not. Participants will be able to diagram the process of creating a transgenic foodstuff. Participants will be able to explain the link of GMO and childhood allergies.

Text: An estimated 70% of food ingested by consumers is genetically altered and requires no label informing our public of this genetically modified (GM) distinction. Similarly, infant and toddler foodstuffs are not labeled GM. It has been postulated that increased intake of GM foods is associated or possibly the cause of an unprecedented rise in childhood allergies. Our overall purpose is to further study the relationship of GM food intake and childhood allergenicity. Presence or absence of transgenic DNA from genetically modified (GM) common food choices of infants and toddlers was determined using quantitative RT-PCR. In the 2002 Infants and Toddlers study, Ziegler and colleagues examined meal and snack intakes providing accurate data of common food choices. Of the 20 common food choices of infants and toddlers, presence of transgenic DNA was examined in 10 foodstuffs (containing corn) using maize and CAMV365 primers from Sigma-Genosys. Each sample was randomly selected from 12 samples purchased in different locations, with different lot numbers and expiration dates. DNA was extracted using DNeasy® Plant Tissue (QIAGEN) with concentration of total DNA assessed by using UV spectrophotometer, and detection of dsDNA analyzed using Brilliant®SYBR® Green (Stratagene Instruments). Presence of transgenic corn DNA was detected in 40% of the samples, 1 cracker, 1 cookie, and 2 cereals. We are currently investigating presence of transgenic soy and rice in the same samples randomly selected in this study. This study demonstrated diet-based origins of potential, inadvertent high quantities of novel proteins that may harm an infant or child.

Funding Disclosure: NSF Grant MSR-RUI 5-24557

Title: DIETARY INTAKE OF CHILDREN WITH SICKLE CELL DISEASE (SCD) COMPARED TO THE NATIONAL HEALTH AND NUTRITION EXAMINATION SURVEY (NHANES III)

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Learning Outcome: To compare the dietary intake of children with sickle cell disease with a contemporary sample of African-American children from NHANES III

Text: Children with SCD have poor growth and suboptimal dietary intake compared to Dietary Reference Intakes, particularly vitamins E, D, calcium and fiber. How the diet of children with SCD compares to that of healthy African-American children has not been investigated. A 24-hour recall was collected annually for four years and analyzed using Food Processor Plus. Nutrient intake was compared to age, gender and race matched median intake from NHANES III, (1988-1994) and %NHANES calculated by age range: 1-3.9, 4-8.9, 9-13.9, 14-18.9 years. Ninety-seven children (53 female) ages 1.5 to 18.7 (8.7 $\pm$4.7) years with SS-type SCD were evaluated at baseline. Overall energy intake was 83% NHANES III. The median intake of virtually all micronutrients in children $<$ 14 years was less than 100% NHANES and worse than their peers. Folate intake, an important nutrient supplemented in SCD, was 43% NHANES in the 1-3.9 year group. Although intake of some nutrients improved in the oldest age group, intake of vitamins C, D, E, calcium, fiber and selenium was still poor ($<$ 74%) relative to NHANES III in all age groups. Longitudinal evaluation of annual intake confirmed these findings of suboptimal intake of many micronutrients, and showed increased intake of folate and decreased intake of vitamin E relative to NHANES III over 3 years. Children with SCD $<$ 14 years have particularly poor dietary intake compared to NHANES III. Dietary intake in adolescents with SCD was comparable to the diets of healthy African-American children, but for many micronutrients remained inadequate.

Funding Disclosure: Comprehensive Sickle Cell grant NIH HL 38633, General Clinical Research Center M01RR00240 and Nutrition Center at Children's Hospital of Philadelphia

Title: EFFECTS OF ENHANCED MUSCLE GROWTH BY MYOSTATIN PROPEPTIDE TRANSGENE ON ADIPONECTIN AND ADIPONECTIN RECEPTORS GENE EXPRESSION

Author(s): S. T. N. Suzuki, B. Zhao, J. Yang; Human Nutrition, Food, and Animal Sciences, University of Hawaii, Honolulu, HI

Learning Outcome: Participants will be able to describe and discuss the biological functions of adiponectin and adiponectin receptors and how myostatin propeptide transgene and feeding of a high-fat diet affect the mRNA expression of these genes.

Text: Myostatin, a member of the transforming growth factor-β superfamily, is a highly conserved negative regulator of muscle growth which undergoes post-translational modification to yield the active form. We have demonstrated that transgenic over-expression of myostatin propeptide dramatically enhanced skeletal muscle development and decreased fat mass. By feeding the transgenic mice either a high-fat diet or normal-fat diet we found that transgenic, high-fat diet mice had improved insulin sensitivity, normal fat deposition, enhanced muscle growth, and significantly higher levels of circulating adiponectin compared to wild-type mice. Adiponectin is known to ameliorate insulin resistance and increase fatty acid oxidation. We theorized that the interaction between high-fat diet and myostatin propeptide would increase adiponectin mRNA expression in fat tissue depots and corresponding adiponectin receptor mRNA expression in muscle and liver. Results from real-time PCR analysis indicated transgenic mice fed a high-fat diet displayed increased adiponectin mRNA expression in epididymal fat by 5-fold over wild-type littermates. The transgenic mice fed a normal-fat diet expressed the most AdipoR1 and R2 in muscle tissue, 2.63-fold and 1.48-fold over the wild-type mice, respectively. The transgenic mice fed a high fat diet did not show increased mRNA level of either receptor in muscle or liver tissue. The increase in expression of adiponectin mRNA may partially explain why the high-fat diet did not cause obesity and insulin resistance in transgenic mice. Only normal-fat diet transgenic mice showed an increase in adiponectin receptors, indicating that high-fat diet may have inhibited expression of receptors in liver and muscle tissue.

Funding Disclosure: USDA Research Grant