Dysregulated Eating May Be Linked to Cortisol

BY KERRI WACHTER
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BALTIMORE — Children with greater food intake in the absence of hunger might have abnormal cortisol levels after stressful situations—a finding that could have implications for the development of obesity—data presented at the annual meeting of the American Psychosomatic Society show.

Lori A. Francis, Ph.D., of Pennsylvania State University, Hershey, and her colleagues looked for associations between cortisol levels in response to stress and evidence of dysregulated eating in 43 children aged 5-9 years.

They found that children with dysregulated eating (eating in the absence of hunger) had increased cortisol levels immediately after the stress test and increasing cortisol levels during a recovery period.

"We think there's some sort of blunted stress response there," Dr. Francis said.

For the study, the children had baseline saliva measurements 15 minutes and 35 minutes after arrival. An hour after arrival, they were submitted to a stress test. A saliva sample was collected after the stress test, and two more samples were collected during the recovery period. Two hours after arrival, the children were given a meal, and 30 minutes later the eating in the absence of hunger protocol was started.

The researchers used a stress test that mainly consists of public speaking and arithmetic tasks. The children were told that they must give a 4-minute speech that would be judged against the speeches of all of the other children.

The speech task was followed by either an arithmetic challenge (children aged 8-9 years) or a block design challenge (aged 5-7 years) for 4 minutes.

During the recovery period, the children completed questionnaires and participated in a craft project. After the recovery period, the children were given a standard meal and told to eat as much as they wanted.

The children were then given a small taste of each of 10 palatable snack foods. The children were then allowed free access to the snack foods and to a box of toys. Foods were weighed pre- and post access and caloric intake was calculated.

Two patterns of cortisol response were identified. Low reactors had cortisol levels that started out high but continued to decline. The high reactors started the stress period with lower cortisol levels that peaked right after the stress test and returned to baseline during the recovery period.

Dr. Francis said that in future studies, she and her colleagues hope to find that stress reactivity is an important marker in terms of the mechanism for developing overweight or obesity. ■

Genes May Make Some Vulnerable To Weight Gain

BALTIMORE — Genes appear to play a role not only in a child's vulnerability to weight gain but also in behaviors that can lead to weight gain, data presented at the annual meeting of the American Psychosomatic Society show.

Jane Wardle, Ph.D., director of the health behavior research center at University College London, and her colleagues analyzed data on 5,435 pairs of twins aged 8-11 years who are part of the Twins Early Development Study (TEDS). Parents rated each twin on satiety sensitivity and food cue responsiveness.

Using genetic model fitting, the researchers estimated that genes account for 63% of satiety sensitivity. Shared environment and nonshared environmental factors accounted for 21% and 16% of satiety sensitivity. The researchers also estimated that genes account for 75% of food cue responsiveness. Shared environment and nonshared environmental factors accounted for 10% and 15% of food cue responsiveness, respectively, she said.

The researchers also used genetic model fitting to estimate the influence of genes, shared environment, and nonshared environment on body mass index and waist circumference.

Genes account for roughly three-quarters of the variability in BMI and waist circumference in these children, shared environment accounts for about 10%, and nonshared environment accounts for roughly 15%.

—Kerri Wachter

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