Gut bacteria may modify obesity risk

Obesity is a pervasive and potentially lethal public health concern. The concept of energy balance is central to our understanding of obesity: excessive calorie intake combined with minimal physical activity creates a surplus of energy that is stored in the body as fat. This caloric algebra may be accurate, but it is an incomplete description of the obesity problem, leaving many questions unanswered. Why are some individuals more likely than others to become obese? Are factors other than consumption and activity involved? What can be done to prevent or reverse excessive weight gain?

Recently, much attention has been focused on the community of bacteria living in our intestines. Results of nutritional and pathological studies suggest that this intestinal microflora may be critically important to our health. For example, some of these bacteria help to provide important nutrients (e.g., vitamin B-12) to their hosts. Coupled with data showing that obese individuals tend to have different intestinal microbial communities than lean individuals, these observations have led scientists to consider the possibility that our gut microflora might directly affect our risk of becoming obese.

To investigate this possibility, researchers Frank Duca, Yassine Sakar and Mihai Covasa from the French National Institute for Agricultural Research (Paris) designed a study to test whether altering an animal's bacterial profile could influence its likelihood of developing obesity. They collected gut bacteria from two strains of rats with different incidences of obesity and then transferred the bacteria into the intestinal tracts of germ-free mice, which had no gut microflora of their own. The mice were fed either a regular diet or a high-fat diet, and food intake and weight gain were monitored for 8 weeks. Intestinal samples were then collected for metabolic analysis.

Covasa presented the study results at the Experimental Biology 2012 conference in San Diego, CA, on 23 April. Mice that received intestinal bacteria from obesity-prone rats ate more food, gained more weight and became more obese than did mice that received bacteria from obesity-resistant rats and also showed changes in intestinal nutrient sensors and gut peptide levels, which could influence their responses to eating.

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The researchers believe that gut microflora may influence behavior and food intake, possibly by interfering with the ability to sense and respond to a meal, and may promote excessive weight gain in obese individuals given the opportunity to overeat. They hope to eventually find ways to manipulate intestinal bacterial communities of at-risk individuals to reduce the likelihood of obesity.

Monica Harrington

HIGH ANXIETY LINKED TO TUMOR GROWTH IN MICE

Researchers have long known that constant stress can impair the body's ability to fight diseases such as cancer. In order to examine this idea further, researchers investigated the endocrine, immune and tumor growth–promoting mechanisms mediating the effects of anxiety and stress on tumor development.

Using two common behavioral tests that measure anxiety based on avoidance of danger, a team of scientists led by Firdaus S. Dhabhar (Stanford University, Stanford, CA) first determined which of their mice had naturally high or low anxiety, dividing them into two groups. Next, both groups of mice were repeatedly exposed to ultraviolet B (UVB) radiation, which is carcinogenic and known to cause most non-melanoma skin cancers in humans, of which there are 2–3 million cases each year worldwide. Dhabhar explained in a press release, "This skin cancer model is really valuable because it closely mimics human skin cancer."

The mice that had showed higher anxiety on the behavioral tests grew more tumors after UVB exposure than those that had showed lower anxiety on the tests (*PLoS One* e33069; published online 25 April 2012). The high-anxiety mice also exhibited lower gene expression of factors that are protective against cancer, as well as higher expression of factors that are cancer-promoting, compared with the gene expression in low-anxiety mice. Furthermore, the ratio of immuno-protective to immuno-suppressive T cells in the high-anxiety mice was diminished. The highly anxious mice also had higher levels of a growth factor that is known to be associated with skin cancer in their skin, possibly underlying the increased tumor growth in these mice.

Next, the researchers wanted to determine how higher anxiety led to these physiological changes. They measured levels of corticosterone, the stress hormone, in the adrenal glands and found that high-anxiety mice had higher levels than low-anxiety mice. When the mice were restrained to induce stress, the mice that had shown higher anxiety on the earlier behavioral tests had higher levels of corticosterone in the blood than mice that had shown lower anxiety on the behavioral tests.

Long-term exposure to corticosterone is known to have deleterious effects on health. The researchers suggest that the high-anxiety mice may be more likely to become overly stressed in response to daily stressors, leading to chronically elevated levels of corticosterone. The next step will be for the researchers to determine whether treating chronic anxiety, such as with anti-anxiety medications, can enhance the benefits of cancer treatment.

Kara Rosania