



How we can stop stress from making us obese

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New findings on the mechanisms that trigger stress-induced obesity, published today in Nature Medicine online, could offer hope to millions.

Professor Herbert Herzog, Director of the Neuroscience Research Program at the Garvan Institute of Medical Research, together with scientists from the US and Slovakia, have shown that neuropeptide Y (NPY), a molecule the body releases when stressed, can 'unlock' Y2 receptors in the body's fat cells, stimulating the cells to grow in size and number. By blocking those receptors, it may be possible to prevent fat growth, or make fat cells die.

"We have known for over a decade that there is a connection between chronic stress and obesity," said Professor Herzog. "We also know that NPY plays a major role in other chronic stress-induced conditions, such as susceptibility to infection. Now we have identified the exact pathway, or chain of molecular events, that links chronic stress with obesity."

"There is not much we can do about the increased levels of NPY caused by stress, but we can do something about the damage it causes. If we can interfere before it causes fat to amass, it could have a major impact on cardiovascular disease, diabetes, and cancer (which all have links with obesity)."

"Basically, when we have a stress reaction, NPY levels rise in our bodies, causing our heart rate and blood pressure to go up, among other things. Stress reactions are normal, unavoidable, and generally serve a useful purpose in life. It's when stress is chronic that its effects become damaging."

Scientists at Georgetown University (Washington D.C), part of this collaborative study, have found a direct connection between stress, a high calorie diet and unexpectedly high weight gain. Stressed and unstressed mice were fed normal diets and high calorie (high fat and high sugar, or so called 'comfort food') diets. The mice on normal diets did not become obese. However, stressed mice on high calorie diets gained twice as much fat as unstressed mice on the same diet. The novel and unexpected finding was that when stressed and non-stressed animals ate the same high calorie foods, the stressed animals utilised and stored fat differently.

"Our findings suggest that we may be able to reverse or prevent obesity caused by stress and diet, including the worst kind of obesity; the apple-shaped type, which makes people more susceptible to heart disease and diabetes," says senior author of the Nature Medicine paper, Professor Zofia Zukowska of Georgetown University. "Using animal models, in which we have either blocked the Y2 receptor, or selectively removed the gene from the abdominal fat cells, we have shown that stressed mice on high calorie diets do not become obese. "Even more surprisingly, in addition to having flatter bellies, adverse metabolic changes tinked to stress and diet, which include glucose intolerance and fatty liver, became markedly reduced. We do not know yet exactly how that happens, but the effect was remarkable," she said.

Professor Herzog believes that these research findings will have a profound effect on the way society will deal with the obesity epidemic. "There are millions of people around the world who have lived with high levels of stress for so long their bodies think it's 'normal'. If these people also eat a high fat and high sugar diet, which is what many do as a way to reduce their stress, they will become obese."

"Until now, the pharmaceutical industry has focused on appetite suppressants with only moderate success. Our hope is that in the near future pharmaceutical companies, using the results of our research, will develop antagonists against the Y2 receptor that will bring about a reduction in fat cells."

Notes to editors:

Stress-activated adipogenic pathway in fat tissue exaggerates diet-induced obesity and metabolic syndrome. Kuo, L.E., Kitlinska, J.B., Tilan, J.U., Li, L., Baker, S.B., Johnson, M.D., Lee, E.W., Burnett, M.S., Fricke, S.T., Kvetnansky, R.K., Herzog, H. & Zukowska, Z. *Nature Medicine* advance online publication, 1 July 2007

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ABOUT GARVAN

The Garvan Institute of Medical Research was founded in 1963. Initially a research department of St Vincent's Hospital in Sydney, it is now one of Australia's largest medical research institutions with approximately 400 scientists, students and support staff. Garvan's main research programs are: Cancer, Diabetes & Obesity, Arthritis & Immunology, Osteoporosis, and Neuroscience. The Garvan's mission is to make significant contributions to medical science that will change the directions of science and medicine and have major impacts on human health. The outcome of Garvan's discoveries is the development of better methods of diagnosis, treatment, and ultimately, prevention of disease.

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