Hunger Hormone Increases During Stress

It is becoming clearer why some people who are stressed or depressed overeat. Although levels of the so-called “hunger hormone” ghrelin increase when we do not eat, researchers from the University of Texas in Dallas suggest that the hormone might also help defend against symptoms of stress-induced depression and anxiety.

Dr. Michael Lutter, Instructor of Psychiatry at the university, said, “Our findings support the idea that these hunger hormones don’t do just one thing; rather, they coordinate an entire behavioral response to stress and probably affect mood, stress and energy levels.”

We know that fasting causes ghrelin to be produced in the gastrointestinal tract. The hormone then plays a role in sending hunger signals to the brain. Research groups, including Dr. Zigman’s, have suggested that blocking the body’s response to ghrelin signals might be one way to help control weight by decreasing food intake and increasing energy expenditure.

“However, this new research suggests that if you block ghrelin signaling, you might actually increase anxiety and depression, which would be bad,” Dr. Zigman said.

To determine how ghrelin affects mood, Dr. Zigman and his colleagues recruited 166 healthy female participants for 10 days. This caused ghrelin levels to quadruple.

Until modern times, the one common human experience was securing enough food to prevent starvation. Our ancestors needed to be calm when it was time to venture out in search of food or they risked becoming dinner themselves, Dr. Zigman said. He added that the anti-anxiety effects of hunger-induced ghrelin may have provided a survival advantage.

Dr. Lutter said the findings might be relevant in understanding conditions such as anorexia nervosa.

“We’re very interested to see whether ghrelin treatment could help people with anorexia nervosa, with the idea being that in a certain population, calorie restriction and weight loss could have an antidepressant effect and could be reinforcing for this illness,” he explained.

In future studies, the researchers hope to determine which area in the brain ghrelin may be acting on to cause these antidepressant-like effects.

(Source: Nature Neuroscience, 2008; 11:752-753.)

Coenzyme Q10 May Alleviate Pain

Drugs called statins may reduce cholesterol levels by inhibiting a signaling pathway that also involves coenzyme Q10 (ubiquinone). Some patients do not comply with statin therapy because of their intolerance to the medication, which can result in muscle symptoms. According to Dr. Giuseppe Caso and his colleagues from Stony Brook University in New York, coenzyme Q10 supplementation may reduce muscle pain that can result from taking statins.

Coenzyme Q10 is an essential part of adenosine triphosphate (ATP) generation in the mitochondria, and any decrease in its levels can impair energy production. Dr. Caso’s team hypothesized that this might underlie the myopathy and muscle pain that have been reported as a side effect of statin therapy.

In their pilot study, 32 patients who were treated with statins and who had myopathic symptoms were assigned to receive of 100 milligrams (mg.) of coenzyme Q10 supplements daily or placebo. After 30 days, pain severity had decreased by 40 percent in the patients taking coenzyme Q10, compared with a nonsignificant 9 percent increase in controls. Patients taking coenzyme Q10 also reported a significant 38 percent reduction in the impact of muscle pain on their daily lives, whereas those taking a placebo reported a nonsignificant 11 percent reduction.

The researchers concluded that there was insufficient evidence to prove the etiologic role of coenzyme Q10 deficiency in statin-associated myopathy. Therefore, the routine use of coenzyme Q10 cannot be recommended in statin-treated patients. Nevertheless, there are no known risks to this supplement, and there is some anecdotal evidence of its effectiveness.

The small number of subjects limited the study. Larger studies are warranted to confirm or refute these data. It is possible that coenzyme Q10 supplementation may still be beneficial in some patients, depending on their genetic susceptibility to metabolic myopathy, their age, and coexisting disease.

(Source: American Journal of Cardiology, 2007; 99:1409-1412.)

Obesity Linked To Infertility In Women

Obesity is a known risk factor for ovulation problems, but it also contributes to infertility in women who ovulate normally. In a new study, women who were severely obese were 43 percent less likely to achieve pregnancy than normal-weight women or women who were considered overweight but not obese during the year-long study.

Jan Willem van der Steeg, M.D., of Amsterdam’s Academic Medical Center, found that obesity was an additional risk factor for infertility in women who had regular menstrual cycles. This is important, given the increase in obesity worldwide. His team evaluated 3,029 couples who were having trouble conceiving on their own. Each couple had spent a year or more trying to conceive, and none had obvious reasons for fertility problems; the women were ovulating and had at least one functioning fallopian tube, and the men had normal semen analyses.

The couples were followed until pregnancy was achieved or until they started fertility treatments. Upon entry into the study, researchers noted the women’s fertility history, weight, height, and smoking status.

The women were classified as being underweight, of normal weight, overweight, or obese, on the basis of their body mass index (BMI). For instance, a 5-foot 6-inch woman who weighs 115 to 154 pounds is considered to be of normal weight (with a BMI of 18.5 to 24.9). If she weighs between 155 and 185 pounds, she is considered overweight (with a BMI of 25 to 29.9). At a weight of 186 or more, she would be considered obese (with a BMI of 30 or higher).

Most of the study participants (86 percent) were either of normal weight or overweight. An additional 10 percent were obese, with BMIs of 30 or more. These women had the most trouble conceiving during the year. For example, women with a BMI of 35 were 26 percent less likely to achieve a spontaneous pregnancy than normal-weight or overweight women but not obese women. Women with a BMI of 40 or more were 43 percent less likely to become pregnant.

It is not clear how obesity affects fertility in women who ovulate normally. Dr. van der Steeg suggested that disruptions in the hormone leptin, which regulates appetite and energy expenditure, may prevent successful fertilization.

Reproductive endocrinologist William Dodson, M.D., claimed that the role of obesity in reproduction is more complex than what was once thought.

“What we once held as dogma is now starting to fall apart. We thought that if a woman’s obesity was not affecting her ovulatory function, her fertility would be similar to a normal-weight woman’s. But this does not appear to be true.”

(Source: Human Reproduction, 2007; 23:324-328; and Fertility and Sterility, 2006; 86:642-646.)

Hope for Obesity?

Scientists at the Harvard School of Public Health have identified a newly discovered class of hormones—lipokines. Furthermore, they consider that the lipokine is a molecule that might stop or even reverse obesity-related conditions such as insulin resistance and “fatty liver.”

Lipokines are hormones made from fats (fats). All other known hormones—chemical signals secreted into the blood that regulate distant cells and organs—are steroid-based or protein-based. From previous experiments, researchers know that an unidentified factor in fat tissue sent signals to regulate metabolism in liver and muscle tissues.

After sifting through massive amounts of data, the scientists discovered an omega-7 fatty acid called C16:ln7-palmitoleate. This health-promoting hormone travels to the muscles and liver, where it improves cell sensitivity to insulin and blocks fat accumulation in the liver. The researchers observed that palmitoleate suppressed inflammation, previously identified as a primary factor leading to metabolic disease.

Palmitoleate is found in natural products, but it does not currently exist in a pure form.

(Source: Cell, 2008; 134:933-944.)