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IN BRIEF

BASIC RESEARCH

Leptin selectively regulates sweet taste receptors in mice

Leptin suppresses the response of taste cells to sweet compounds according to new data published in *Diabetes*. In mouse taste cells, the leptin receptor is expressed in ~75–80% of cells expressing taste receptor type 1 member 3 (also known as sweet taste receptor T1R3). In isolated taste cells, leptin supressed the ability of T1R3 to sense sweet compounds (in this case, saccharine), but did not modulate the response of cells expressing receptors for bitter or sour tastes. Interestingly, this response was absent in taste cells isolated from mice with diet-induced obesity (which develop leptin resistance), or leptin receptor-deficient *db/db* mice. In pancreatic β cells, leptin is known as activate K_{ATP} channels, which are also expressed in T1R3-positive taste cells. Treating T1R3-expressing taste cells with the K_{ATP} channel blocker glibenclamide prevented the leptin-mediated suppression response to saccharine.

Original article Yoshida, R. *et al.* Leptin suppresses mouse taste cell responses to sweet compounds. *Diabetes* doi:10.2337/db14-1462

ADRENAL GLAND

Increased CVD and metabolic morbidity in patients with CAH

Patients with congenital adrenal hyperplasia (CAH) have an increased risk of developing cardiovascular disease (CVD) and metabolic-associated disorder according to new research conducted by investigators in Sweden. The team compared 588 patients with CAH (resulting from 21-hydroxylase deficiency) with healthy individuals (matched for sex, age and birth place; $n = 58{,}800$). Overall participants with CAH were nearly four-times more likely to develop a cardiovascular or metabolic disorder (OR 3.9, 95% CI 3.1–5.0), and almost three-times more likely to develop CVD (OR 2.7, 95% CI 1.9–3.9) than healthy patients. The increased risk was similar when the patients were stratified according to sex or age group. This Swedish study is the first to investigate CVD itself, rather than CVD risk factors, in patients with CAH.

Original article Falhammar, H. *et al.* Increased cardiovascular and metabolic morbidity in patients with 21-hydroxylase deficiency: a Swedish population-based national cohort study. *J. Clin. Endocrinol. Metab.* doi:101210/JC.2015-2093

DIABETES

Cold therapy—a potential new approach to treat type 2 diabetes mellitus?

In a new study, published in *Nature Medicine*, exposing patients with type 2 diabetes mellitus (T2DM) to cold conditions improved insulin sensitivity. The patients ($n = 8$) were acclimatized to a cold environment (14–15 °C) over a 10-day period. This treatment led to an ~43% increase in peripheral insulin sensitivity. Although cold exposure can enhance brown adipose tissue (BAT) thermogenesis and might improve insulin sensitivity, the team found that glucose uptake was actually enhanced in skeletal muscle, with BAT activity remaining low. The investigators found that glucose transporter GLUT4 accumulated on the cell membranes of skeletal muscle during cold acclimatization. This effect was independent of insulin signalling and suggests a new mechanism by which cold acclimatization might improve the metabolic health of patients with T2DM.

Original article Hanssen, M. J. W. *et al.* Short-term cold acclimation improves insulin sensitivity in patients with type 2 diabetes mellitus. *Nat. Med.* doi:10.1038/nm.3891