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IN BRIEF

INFLAMMATION

Leptin deficiency protects against SLE

Although patients with systemic lupus erythematosus (SLE) are known to have elevated circulating levels of leptin, the role of leptin in the pathogenesis of the disease has been unclear. Now, in new research, genetic deficiency of leptin is shown to protect mice against the development of pristane-induced SLE manifestations (autoantibody production and renal disease). Conversely, in a mouse model of spontaneous SLE, leptin administration accelerated the disease. The finding of a promotional role for leptin in SLE raises the possibility that leptin-targeted therapies could be used to treat the disease.

ORIGINAL ARTICLE Lourenço, E. V. *et al.* Leptin promotes systematic lupus erythematosus by increasing autoantibody production and inhibiting immune regulation. *Proc. Natl Acad. Sci. USA* http://dx.doi.org/10.1073/pnas.1607101113 (2016)

ADIPOSE TISSUE

Key role for FGF21 in GLP1-mediated weight loss

A population of T cells mediates weight loss via an FGF21-dependent mechanism, according to new data. In obese mice, in which invariant natural killer T (iNKT) cells were activated with α-galactosylceramide, weight loss was accompanied by increases in body temperature, fatty acid oxidation, thermogenic browning of white adipose tissue and serum levels of FGF21. Weight loss following activation of iNKT cells was markedly reduced in similarly treated FGF21-deficient mice, confirming the involvement of FGF21. As the glucagon-like peptide 1 agonist liraglutide also activates iNKT cells to induce weight loss, targeting the iNKT cell–FGF21 axis could be a promising approach to regulate weight.

ORIGINAL ARTICLE Lynch, L *et al.* iNKT cells induce FGF21 for thermogenesis and are required for maximal weight loss in GLP1 therapy. *Cell Metab.* http://dx.doi.org/10.1016/j.cmet.2016.08.003 (2016)

BONE

Dual mode of action of SSRIs on bone remodelling

New research shows that selective serotonin-reuptake inhibitors (SSRIs) affect bone remodelling via two distinct mechanisms. In the short term (3 weeks), fluoxetine (the active compound in Prozac), has local anti-resorptive properties and increases bone volume. However, long-term treatment (6 weeks) offset the initial anti-resorptive effect and resulted in bone loss. Importantly, co-treatment with the β-blocker propranolol prevented the long-term effects of fluoxetine and, thus, bone loss. The findings highlight a possible strategy to counteract the increased risk of fractures with SSRI use.

ORIGINAL ARTICLE Ortuño, M. J. *et al.* Serotonin-reuptake inhibitors act centrally to cause bone loss in mice by counteracting a local anti-resorptive effect. *Nat. Med.* http://dx.doi.org/10.1038/nm.4166 (2016)

ENDOCRINE DISRUPTORS

Air pollution linked to insulin resistance

A new study has shown that long-term exposure to air pollution is associated with insulin resistance in the general population. Analysis of almost 3,000 individuals showed that each 7.9 μg/m^3 increase in particulate matter was associated with an increased HOMA index (15.6%) and levels of insulin (14.5%). Effects were greater in individuals with prediabetes than in nondiabetics. The findings indicate that air pollution is a risk factor for type 2 diabetes mellitus, particularly in prediabetic individuals.

ORIGINAL ARTICLE Wolf, K *et al.* Association between long-term exposure to air pollution and biomarkers related to insulin resistance, subclinical inflammation and adipokines. *Diabetes* http://dx.doi.org/10.2337/db15-1567 (2016)