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IN BRIEF

DIABETES

Sustained breastfeeding reduces risk of T2DM

Women with gestational diabetes mellitus are more likely to develop type 2 diabetes mellitus (T2DM) in the postpartum period. However, breastfeeding is known to reduce the risk of T2DM. In a new study in 304 women who had gestational diabetes mellitus, women who tested negative for islet autoantibodies and continued breastfeeding for >3 months had lower T2DM risk than those who did not breastfeed or who breastfed for ≤3 months. Additionally, women who breastfed had a median time to T2DM diagnosis of 12.3 years versus 2.3 years in those who did not breastfeed.

Original article Ziegler, A.-G. *et al.* Long-term protective effect of lactation on the development of type 2 diabetes in women with recent gestational diabetes mellitus. *Diabetes* doi:10.2337/db12-0393

METABOLISM

Gut-derived serotonin increases energy release?

Adipose tissue and liver are the main energy stores in the body. A murine study suggests that synthesis of serotonin in the gut is enhanced during fasting, increasing energy release from adipose tissue and liver. In adipocytes, gut-derived serotonin favours lipolysis through activation of hormone-sensitive lipase. Glucose uptake into hepatocytes is inhibited by gut-derived serotonin, which leads to increased serum glucose levels. Additionally, gut-derived serotonin enhances gluconeogenesis in hepatocytes. The researchers suggest inhibiting synthesis of gut-derived serotonin as a treatment for type 2 diabetes mellitus.

Original article Sumara, G. *et al.* Gut-derived serotonin is a multifactorial determinant to fasting adaptation. *Cell Met.* **16**, 1–13 (2012)

NEUROENDOCRINOLOGY

Leptin influences transporter expression in astrocytes

Leptin is known to affect the morphology of glial cells. A new study demonstrates that this hormone also influences the expression of glutamate and glucose transporters in these cells. In mice, elevated body weight increased the expression of these transporters in astrocytes. Therefore, the metabolic state alters hypothalamic glial cell activity and the researchers hypothesize that metabolic control by astrocytes is likely to be involved in the pathogenesis of obesity.

Original article Fuente-Martin, E. *et al.* Leptin regulates glutamate and glucose transporters in hypothalamic astrocytes. *J. Clin. Invest.* doi:10.1172/jci164102

THERAPY

Role for bone in adverse effects of glucocorticoid therapy?

Osteocalcin is secreted from osteoblasts and might affect metabolic regulation. A study in mice shows that disruption of glucocorticoid signalling specifically in osteoblasts prevents the development of insulin resistance, glucose intolerance and weight gain, which are common adverse consequences of glucocorticoid therapy. Additionally, following glucocorticoid treatment, these adverse effects were attenuated in mice expressing carboxylated and undercarboxylated osteocalcin in the liver. These results suggest that the adverse metabolic effects of glucocorticoid therapy are mediated via effects on the skeleton.

Original article Brennan-Speranza, T. C. *et al.* Osteoblasts mediate the adverse effects of glucocorticoids on fuel metabolism. *J. Clin. Invest.* doi:10.1172/jci63377