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IN BRIEF

THERAPY

Metformin reduces adverse effects of glucocorticoid treatment

Patients undergoing glucocorticoid treatment are at risk of developing metabolic adverse effects, such as the metabolic syndrome. Preclinical studies have suggested that metformin can prevent these effects; a new paper has investigated the use of metformin in a randomized controlled trial. In the trial, 29 patients who did not have diabetes mellitus were randomly assigned to receive a glucocorticoid (prednisone, prednisolone or methylprednisolone) with a placebo or metformin (850 mg once daily for 1 week followed by 850 mg twice daily for 3 weeks). After 4 weeks, the median glucose levels increased in the placebo group but were similar to the baseline levels in the group that received metformin. The authors suggest that metformin could, therefore, be used to prevent metabolic adverse effects in patients taking glucocorticoids.

ORIGINAL ARTICLE Seelig, E. *et al.* Metformin prevents metabolic side effects during systemic glucocorticoid treatment. *Eur. J. Endocrinol.* http://dx.doi.org/10.1530/EJE-16-0653 (2017)

STEM CELLS

Bone healing in diabetes mellitus

Bone fractures in patients with diabetes mellitus often do not heal as well as those in people without diabetes mellitus; however, the mechanisms underlying this impaired healing have been unclear. A recently published paper analysed the function of skeletal stem cells in mouse models of diabetes mellitus to try and determine what factors impair bone repair. The authors found that high serum levels of tumour necrosis factor in diabetic mice repressed the expression of Indian hedgehog (Ihh) in skeletal stem cells. Repressing hedgehog signalling during fracture repair suppressed the expansion of skeletal stem cells that usual happens after injury, which resulted in impaired healing. Importantly, the effects could be reversed with exogenous administration of Ihh.

ORIGINAL ARTICLE Tevlin, R. *et al.* Pharmacological rescue of diabetic skeletal stem cell niches. *Sci. Transl Med.* http://dx.doi.org/10.1126/scitranslmed.aag2809 (2017)

DIABETES

The role of adiponectin in gestational diabetes mellitus

Women with gestational diabetes mellitus often have hypoadiponectinaemia, but it is unknown whether the low levels of adiponectin are causal in the development of gestational diabetes mellitus. Now, researchers have used a mouse model in which the gene that encodes adiponectin is knocked out (*Adipoq*$^{-/-}$ mice) to demonstrate that adiponectin is involved in regulating the metabolic adaptations to pregnancy. Pregnant *Adipoq*$^{-/-}$ mice developed glucose intolerance and hyperlipidaemia in the late stages of pregnancy; this effect was not seen in knockout mice that were not pregnant. The fetuses of *Adipoq*$^{-/-}$ mice also had increased body weight and blood levels of glucose. The lipid profiles and blood glucose levels of *Adipoq*$^{-/-}$ mice returned to levels seen in wild-type mice after delivery. In addition, administering adiponectin *in vivo* reversed glucose intolerance and prevented high fetal body weight. The authors of the paper conclude that hypoadiponectinaemia could have an important role in the development of gestational diabetes mellitus.

ORIGINAL ARTICLE Qiao, L. *et al.* Adiponectin deficiency impairs maternal metabolic adaptation to pregnancy in mice. *Diabetes* http://doi.org/10.2337/db16-1096 (2017)