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IN BRIEF

DIABETES

Enhancing β-cell mass in diabetes mellitus

A new study has demonstrated that serpinB1, a liver-derived protease inhibitor, can promote β-cell proliferation in mice, zebrafish and humans. The researchers found that mice lacking serpinB1 had reduced β-cell compensation in response to insulin resistance. Furthermore, treating islets with serpinB1 *in vitro* resulted in inhibition of elastase and activation of growth factor and survival-factor signalling pathways. Although these findings are an important step in achieving regeneration of functional β cells, more work is needed to identify other factors involved and to determine whether this approach can be used safely in humans.

ORIGINAL ARTICLE El Ouaamari, A. *et al.* SerpinB1 promotes pancreatic β cell proliferation. *Cell Metab.* http://dx.doi.org/10.1016/j.cmet.2015.12.001

OBESITY

Importance of genetic background

A comprehensive metabolic characterization of mice fed a chow diet or a high-fat diet for 6 weeks has determined the effects of a genetically altered immune system. Nonobese diabetic (NOD) mice have reduced fat mass and increased insulin sensitivity compared with C57BL/6 wild-type mice. SCID mice also have increased insulin sensitivity, along with increased glucose metabolism in muscle and resistance to diet-induced obesity as a result of increased energy expenditure and physical activity. NSG mice (which lack some immune cells and have deficient cytokine signalling) do not develop diet-induced obesity or insulin resistance. These findings demonstrate the importance of genetic background, lymphocytes and cytokine signalling in diet-induced obesity and insulin resistance.

ORIGINAL ARTICLE Friedline, R. H. *et al.* Genetic ablation of lymphocytes and cytokine signaling in nonobese diabetic mice prevents diet-induced obesity and insulin resistance. *FASEB J.* http://dx.doi.org/10.1096/fj.15-280610 fj.15-280610

THERAPY

Effects of anti-B-cell therapy

Rituximab, which depletes B cells, preserves β-cell function in patients with type 1 diabetes mellitus, but how this affect is achieved is not known. A new study has shown that rituximab does not alter the frequencies of autoreactive and polyreactive B cells in patients with type 1 diabetes mellitus 52 weeks after treatment. The researchers suggest that rituximab temporarily dampens autoimmune processes by depleting B cells, but that newly generated B cells replace those that are lost (rather than B cells that were not destroyed repopulating the periphery). This observation could explain why many patients relapse after anti-B-cell therapy.

ORIGINAL ARTICLE Chamberlain, N. *et al.* Rituximab does not reset defective early B cell tolerance checkpoints. *J. Clin. Invest.* http://dx.doi.org/10.1172/JCI83840