

The role of beta cell regeneration in type 2 diabetes

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The World Health Organization (WHO) has declared type 2 diabetes as the epidemic of the 21st century. A study is focusing on understanding the mechanisms underlying insulin resistance and the role of beta-cell regeneration.

Type 2 diabetes develops through a step-wise process, starting by the inability of the body to use insulin effectively which causes [pancreatic beta cells](#) to produce more insulin in an attempt to compensate for this phenomenon. Eventually, the beta cells become exhausted and cannot produce enough insulin to overcome [insulin resistance](#) and at this point,

this condition can progress to diabetes.

A substantial decrease in the number of beta cells due to extensive cell death or decreased proliferation, and/or an impairment of [insulin secretion](#) are considered the most likely mechanisms underlying type 2 diabetes. The European BCELL-T2D is aiming to delineate these mechanisms and study the potential of beta-[cell regeneration](#) in therapy.

To achieve this, scientists have characterised a mouse model carrying a mutation in the receptor that binds the fat cell-specific hormone leptin. These animals are prone to obesity and type 2 diabetes.

Results so far indicate that insulin resistance starts at 4–9 weeks of age, progresses to frank diabetes (10–18 weeks of age) and finally to advanced diabetes and its complications (after 19 weeks of age). Assessment of the beta-cell mass at each stage has shown an initial expansion in the insulin compensatory phase that disappears along the evolution of the disease.

As a therapeutic intervention, scientists are looking to overcome the proliferation shortage of beta cells by providing cyclin C, a protein that controls cell cycle entry. Ongoing research will determine if sustained beta-[cell proliferation](#) has the potential to prevent diabetes debut.

By studying the type 2 diabetes mouse model, scientists have revealed novel insight into the disease development and pathophysiology. Project findings hold great potential of being translated into new therapies in order to prevent and treat this devastating disease and its complications.

Provided by CORDIS

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