Featured Publication

VANDERBILT UNIVERSITY labnodes

Reprogramming Cells: Challenges Remain

Type 1 diabetes results from the loss of insulin-producing beta cells in the pancreas. One potential way to cure the disease is to “reprogram” pancreatic acinar cells, which normally produce proteolytic enzymes, so that they start producing insulin instead.

This is easier said than done. In a report published last month in the journal *Cell Reports*, graduate student Hannah Clayton, Mark Magnuson, M.D., and colleagues describe how the conversion of acinar into beta cells into depends on both the concentration of a triad of reprogramming transcription factors and the level of inflammation.

Overly robust expression of these factors induces induce pancreatic inflammation, which blocks apartment reprogramming and results instead in production of duct-like cells. Only when inflammation is attenuated, by reducing transcription of factor expression or by depending inflammatory macrophages, does production of new beta-like cells occur.

For i*n vivo* beta-cell restorative therapy to become clinically feasible, it will necessary to understand more the fully the complex and coordinated series of events that modulate intercellular, the researchers concluded.

The Reporter: Vanderbilt University Medical Center’s Weekly Newspaper

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Bill Snyder

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Pancreatic Inflammation Redirects Acinar to ■ Cell Reprogramming.
Clayton HW, Osipovich AB, Stancill JS, Schneider JD, Vianna PG, Shanks CM, Yuan W, Gu G, Manduchi E, Stoeckert CJ, Magnuson MA (2016) *Cell Rep* 17(8): 2028-2041

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Hannah, a graduate student, is the first author of the Featured Publication.