Type 2 diabetes and neurodegenerative diseases are increasingly prevalent worldwide, particularly in our Pacific rim neighborhood. There is an as yet unexplained shared risk for these diseases with cellular pathology that is remarkably similar. Despite major investments in research by the NIH and the pharmaceutical industry, there has been little advance in modifying the course of these diseases. We hypothesize that this might be because the impacted cells in both type 2 diabetes and neurodegenerative diseases manifest shared abnormalities that have been presumed to be targets but are actually pro-survival adaptations in response to the underlying injury. Further, we hypothesize that because the impacted beta cells and neurons are adapted to survive at the expense of function, if the proximal injury can be identified and targeted, then the pro-survival changes may be reversed to restore cellular function and favorably modify the course of these diseases.

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