Title: CAVEOLIN-1 K.O. MICE EXPOSED TO HIGH FAT DIET EXHIBIT A LIPOTOXIC MILIEU BUT LESS BETA PANCREATIC DAMAGE COMPARED WITH WILD TYPE MICE

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Introduction:

Background: Deleterious effects of high levels of free fatty acids lead to a phenomenon known as "lipotoxicity", associated with insulin resistance and beta pancreatic cell damage, key events in development of type 2 diabetes mellitus. Lipotoxicity has been associated with cellular oxidative stress and beta-cell apoptosis. Caveolin-1 is a membrane protein that has been associated with many cellular functions as cell signaling regulation and apoptosis, and it is normally present in beta pancreatic cells. We previously reported that the expression of the membrane protein Caveolin-1 promotes free fatty acids-induced apoptosis *in vitro* in a mouse beta cell line. Remains to be elucidated if this phenomenon is relevant in vivo. **Methods**: We used C57BL6J mice lacking expression of Caveolin-1 (CAV-1 K.O. mice). We evaluated free fatty acids and triglycerides levels in blood in fasting conditions, oral glucose tolerance test (OTTG), carbonylated proteins in serum and C-peptide in wild type (WT) and Caveolin-1 K.O. and wild type mice exposed to a high fat diet for three months. Also, the presence of apoptosis was evaluated by TUNEL staining in beta pancreatic islets. Results: We found that CAV-1 K.O. mice fed with high fat diet showed higher levels of triglycerides, cholesterol, free fatty acids and carbonylated proteins, although also a better response OGTT and C-peptide levels. Islets from K.O. mice showed lower levels of apoptosis. **Conclusion**: Although K.O. mice showed a lipotoxic profile, our results suggest that their pancreatic islets were more resistant to the high fat diet deleterious effects over beta cells.

Biography

Dr. Wehinger has completed his PhD in Biomedical Sciences from University of Chile in 2013 and actually is Associate Professor and Director of Magister in Biomedical Sciences of University of Talca in Chile. Dr. Wehinger had published papers in the field of metabolism and diabetes and he is currently investigating the molecular mechanisms involved in the cellular failure of the beta pancreatic islets, which is induced by elevated free fatty acids and oxidative stress levels, to elucidate how to prevent these processes.

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