Enhancing Pancreatic Islet Function in an Obese Mouse Model

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The genetic explanation of Type II Diabetes has been long explored in hopes of developing a therapeutic treatment. Recent developments have led to the discovery of Betatrophin, an adipokine secreted from adipose tissue, thought to regulate insulin secretion in humans. However, Betatrophin is new and its effects in humans have not been tested. Therefore, it has become increasingly clear that more research has to be done to understand the exact effects of Betatrophin and to discover the other forces present that might influence its effect. The purpose of this study is to expand upon prior knowledge through quantitative trait locus (QTL) analysis and by studying the correlation between adipocytes and Type II diabetes. Thereby addressing several questions: (1) how does Betatrophin function in obese models? (2) Are there loci on mouse chromosomes that influence Betatrophin? (3) Are there QTL that influence Betatrophin shared with any other phenotypes related to diabetes or obesity? (4) Are there genes that amplify the expression of Betatrophin? (5) Are other phenotypes affected by Betatrophin that are involved in the metabolic syndrome, and (6) can a causal model explain the expression of Betatrophin in relation to its QTL and other adipokines? Betatrophin did indeed increase insulin secretion in an obese model and there were loci on chromosomes 1,2, 6, and 10 that were shared among the adipokines and the phenotypes. Furthermore it was found that two other adipokines, apelin and adiponectin, influenced Betatrophin production and were involved in the causal model for Betatrophin production and regulation.

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