Pancreastatin, A Novel Peptide Regulator of Glucose and Lipid Metabolism

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Technology description

Summary

It has been demonstrated that pancreastatin can inhibit glucose-induced insulin secretion from pancreas, insulin signaling in adipocytes and hepatic cells via activation of conventional PKC, and leptin and adiponectin production from adipose tissue, but stimulate nitric oxide production. It has been shown also that circulating pancreastatin levels in diabetic patients are higher than healthy subjects. However, it has not been shown whether pancreastatin regulates expression of genes responsible for glucose production and lipid metabolism and whether the effects on those gene expressions are PKC and nitric-oxide dependent or not.

Description

Scientists at UC San Diego have discovered a new biological effect for pancreastatin. Pacreastatin elevates glucose interleukin-6 production from adipose tissue and can cause pre-diabetic conditions by promoting glucose intolerance and inflammation. It can also modulate expressions of PEPCK, G6PASE, SREBP-1, ACOX, and CPT-I genes, and suppression of p-FOXO-1 signal leading to hyperglycemia, suppression of lipid production, and enhancement of fatty acid oxidation. The invention demonstrates that transcription activation by pancreastatin is conventional PKC and nitric oxide dependent. The invention also establishes that pancreastatin is a hyperglycemic and hypolipogenic hormone and that treatment with natural and synthetic variants of pancreastatin can improve insulin effects on hepatic gluconeogenic gene (G6Pase) expression.

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