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Available FREE at MacUpdate. 2016 Update to Robot Structural Analysis Professional 2013 x64 & x32. Autodesk Super DX 2012 x64 64 Bit - Autodesk Super DX 2012 x32.Atg7 Deficiency Causes Hyperinsulinemia by Reducing β Cell Mass, Mitochondrial Function and Glucose Sensing in Mice. Autophagy is important for protein quality control, and there is emerging evidence that autophagy also regulates β cell function. However, the role of autophagy in β cell function remains controversial. To explore the role of autophagy in β cell function, we generated mice that were deficient in Atg7, a crucial autophagy protein, specifically in β cells by crossing Atg7flox/flox mice with Ins2-Cre mice. Atg7 deletion in β cells of these mice was confirmed by immunoblotting analysis. Sucrose density gradient and electron microscopy analysis showed that the number of autophagosomes was reduced and mitochondrial mass was increased in β cells of these mice. Glucose tolerance test results showed that Atg7 deficiency in β cells increased the blood glucose level during glucose tolerance test. Atg7 deficiency in β cells improved the post-prandial insulin levels, but in terms of β cell function, Atg7 deficiency decreased first-phase insulin secretion and increased second-phase insulin secretion. Next, to identify the defects in β cells upon Atg7 deficiency, we analyzed β cell mass by measuring β cell volume, β cell proliferation, apoptosis, and β cell mass in these mice. The size of β cells was reduced, and the number of EdU-positive cells was also reduced in Atg7-deficient mice. Furthermore, we detected the expression of the glucose transporter 2 (Glut2) and the glucose-sensing protein glucose-sensing receptor (GPR19), which may be the initial molecule for β cell function. However, neither Glut2 nor GPR19 expression was influenced by Atg7 deletion. Surprisingly, expression of the GPR19 co-receptor GPR99, which is important for GPR19 binding, was also decreased in these mice. Furthermore, analysis of the GPR99 gene promoter region showed that Atg7 may regulate the expression of GPR99 in mouse β cells. Our results indicate that autophagy regulates β cell function, even though β cell growth and function are negatively correlated and loss of β cell mass is related to 648931e174

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