Abstract
Growth factors, insulin signaling and nutrients are important regulators of \( \beta \)-cell mass and function. The events linking these signals to regulation of \( \beta \)-cell mass are not completely understood. Recent findings indicate that mTOR pathway integrates signals from growth factors and nutrients with transcription, translation, cell size, cytoskeleton remodeling and mitochondrial metabolism. mTOR is a part of two distinct complexes; mTORC1 and mTORC2. The mammalian TORC1 is sensitive to rapamycin and contains Raptor, deptror, PRAS40 and the G protein \( \beta \)-subunit-like protein (G\( \beta \)L). mTORC1 activates key regulators of protein translation; ribosomal S6 kinase (S6K) and eukaryote initiation factor 4E-binding protein 1. This review summarizes current findings about the role of AKT/mTORC1 signaling in regulation of pancreatic \( \beta \) cell mass and proliferation. mTORC1 is a major regulator of \( \beta \)-cell cycle progression by modulation of cyclins D2, D3 and cdk4/cyclin D activity. These studies uncovered key novel pathways controlling cell cycle progression in \( \beta \)-cells in vivo. This information can be used to develop alternative approaches to expand \( \beta \)-cell mass in vivo and in vitro without the risk of oncogenic transformation. The acquisition of such knowledge is critical for the design of improved therapeutic strategies for the treatment and cure of diabetes as well as to understand the effects of mTOR inhibitors in b-cell function.

Keywords
Diabetes Mellitus, type 2, Islets of Langerhans, Signal Transductions, Cell proliferation, AKT/PKB/mTORC1 signaling pathway, Cell cycle.