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RNase L mediates the insulin signaling pathway

College of Sciences and Health Professions

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Abstract

Diabetes is characterized by hyperglycemia mainly due to defect in insulin secretion and/or action. Regulation of glucose transport and use by insulin is central to the maintenance of whole-body glucose homeostasis. One of the potential mechanisms associated with insulin sensitivity is the activation of insulin receptor (IR) and subsequently transduces the signal through phosphorylation of insulin receptor substrate1 (IRS1) and activation of the PI-3K/Akt pathway. RNase L, an interferon (IFN)-inducible enzyme, plays an important role in IFN functions against viral infection and cell proliferation. However, a direct link between RNase L and insulin sensitivity has yet to be clearly established. In this study, we found that RNase L plays an important role in glucose homeostasis through impacting IR which is a trans-membrane receptor activated by insulin. The phosphorylation status of IR was significantly reduced in the cells deficient RNase L. As a result, activation of IRS1, the downstream substrate of IR, and the PI3K/AKT pathway was significantly inhibited in RNase L^{-/-} cells. Further investigation of the molecular mechanism underlying the role of RNase L in mediating the activation of IR revealed that RNase L might regulate the cleavage of the precursor of IR via the ubiquitin/ proteasome system. Our results suggest that RNase L may be a novel target in the design of therapeutic strategies for diabetes.