Insulin receptor mediated signaling

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references


Reactome database release: 79

This document contains 3 pathways (see Table of Contents)
Insulin receptor mediated signaling

Stable identifier: R-DME-110526

The insulin/IGF-like signaling (IIS) and TOR pathways are responsible for conveying nutrient signals and regulating growth in Drosophila. Activation of the IIS pathway occurs when insulin-like peptides bind the insulin receptor. Activation of the TOR signaling pathway occurs in the presence of amino acids via an unknown mechanism.

Together, these pathways coordinate cell-autonomous and non cell-autonomous growth responses to nutrients. The exact way these two signaling pathways are coordinated and their outputs balanced are not understood.

Editions

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Insulin signaling pathway

Location: Insulin receptor mediated signaling

Stable identifier: R-DME-110478

In Drosophila there is a single insulin/IGF receptor homologue (DIrn), seven insulin-like peptides (DILPs), and a single IRS homologue (Chico), in contrast to the insulin receptor, insulin and IRS entities, respectively, in mammals. DIrn and the DILPs interact to regulate growth, haemolymph carbohydrate levels, reproduction and ageing.

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The target of rapamycin (TOR) proteins are large protein kinases, evolutionarily conserved from yeast to humans. Under optimal nutritional conditions, TOR propagates the insulin-like signal to downstream effectors of cell growth such as D4EBP and DS6K. When nutrients are deficient, TOR transmits no signal and insulin signaling is inhibited.

**Editions**

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</table>
# Table of Contents

- Introduction  
  - Insulin receptor mediated signaling  
    - Insulin signaling pathway  
    - TOR signaling pathway  
- Table of Contents