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Exp Clin Endocrinol Diabetes 2001; 109(Suppl 2): S135-S148
DOI: 10.1055/s-2001-18576



Physiology - Metabolism - Pathophysiology

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Insulin resistance: definition and consequences

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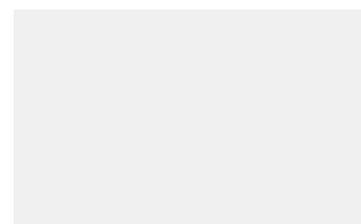
Abstract

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Summary:

Insulin resistance is defined clinically as the inability of a known quantity of exogenous or endogenous insulin to increase glucose uptake and utilization in an individual as much as it does in a normal population. Insulin action is the consequence of insulin binding to its plasma



membrane receptor and is transmitted through the cell by a series of protein-protein interactions. Two major cascades of protein-protein interactions mediate intracellular insulin action: one pathway is involved in regulating intermediary metabolism and the other plays a role in controlling growth processes and mitoses. The regulation of these two distinct pathways can be dissociated. Indeed, some data suggest that the pathway regulating intermediary metabolism is diminished in type 2 diabetes while that regulating growth processes and mitoses is normal. - Several mechanisms have been proposed as possible causes underlying the development of insulin resistance and the insulin resistance syndrome. These include: (1) genetic abnormalities of one or more proteins of the insulin action cascade (2) fetal malnutrition (3) increases in visceral adiposity. Insulin resistance occurs as part of a cluster of cardiovascular-metabolic abnormalities commonly referred to as “The Insulin Resistance Syndrome” or “The Metabolic Syndrome”. This cluster of abnormalities may lead to the development of type 2 diabetes, accelerated atherosclerosis, hypertension or polycystic ovarian syndrome depending on the genetic background of the individual developing the insulin resistance. - In this context, we need to consider whether insulin resistance should be defined as a disease entity which needs to be diagnosed and treated with specific drugs to improve insulin action.



Key words:

Insulin action - visceral obesity - fetal malnutrition - syndrome X - free fatty acids

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