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Cell hydration and insulin signalling

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Abstract

Changes in cell hydration are critically important for the signalling towards metabolic responses to hormones, substrates and reactive oxygen intermediates. In liver insulin-induced cell swelling is due to a net K(+)-uptake resulting from the concerted activation of Na(+)/K(+)/2Cl(-) cotransport, Na(+)/H(+) exchange and the Na(+)/K(+)-ATPase. Insulin-induced swelling is essential for generating the antiproteolytic response to the hormone, which depends on activation of the MAP-kinase p38. Recent investigations show, that cell swelling induced by either hypoosmolarity or insulin triggers the activation of signalling cascades. Cell swelling by insulin is Ptdins-3-kinase mediated and contributes to the activation of Erk- and p38-type MAP-kinases. Conditions dehydrating insulin target tissues such as hyperosmolarity or amino acid deprivation are frequently associated with insulin resistance. In liver, hyperosmolarity impairs the Ptdins-3-kinase-dependent K(+) uptake and cell swelling in response to insulin, leading to resistance of MAP-kinases and proteolysis to regulation by insulin. Likewise, a reduction of insulin-induced swelling by the loop diuretics furosemide and bumetanide cause insulin resistance shown by the levels of cell swelling, MAP-kinase activation and proteolysis control. Blockage of the cell volume response to insulin may be the common denominator in dehydration-induced insulin resistance found in clinical settings such as sepsis, burn injury and diabetes mellitus.

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