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TITLE

ESTROGEN ENHANCEMENT OF GLUCOSE TRANSPORTER 4 AND INSULIN RECEPTOR SUBSTRATE 1 EXPRESSION AT BOTH LOW AND HIGH GLUCOSE CONCENTRATIONS IN SGBS-ADIPOCYTES

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ABSTRACT

Context: New findings show that estrogen therapy, via estrogen receptor alpha (ER?) activation, may influence the metabolic homeostasis of insulin-dependent tissues and modulate the expression of the proteins involved in insulin signaling. However, it is not clear which proteins partake in this activity and how they are expressed. A possible crosstalk between Er? and the insulin receptor could help increase glucose uptake and promote energy advantage for the cell. Objective: This study aimed at determining the effects of estrogen on insulin signaling pathway activation. Methods: Following adipogenic differentiation, the SGBS-adipocytes were treated with glucose (1.000 mg/L and 1.800 mg/L), insulin (20 nM), and 17?-estradiol (10-8). After 24 hours of treatment, protein expression detection of glucose transporter 4 (GLUT4) and insulin substrate 1 (IRS-1) was performed by Western Blot analysis. Results: We here show that SGBS-adipocytes treated with estrogen and exposed to low (1,000 mg/L) and high (1,800 mg/L) concentrations of glucose, with or without insulin, display an elevated GLUT4 expression. Furthermore, the concomitant administration of estrogen and insulin enhance the expression of ER? and IRS1 both at low and high glucose concentrations. Conclusions: Estrogen therapy may regulate the expression of GLUT4 and IRS1, which are involved in the insulin signaling pathway. Although these findings broaden the understanding of estrogen action on SGBS-adipocytes, further studies addressing glucose uptake are needed to better comprehend the hormone's action on insulin-dependent cells.

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