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Effects of estrogen on GLUT4 and IRS1 expression in SGBS-adipocytes

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Context: New findings show that estrogen therapy, via estrogen receptor alpha (ERÎ±) 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 ErÎ± 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 17Î²-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 ERÎ± 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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