Decreased phosphorylation of Y14caveolin-1 in endometrial tissue of polycystic ovary syndrome patients may be related with an insulin resistant state in this tissue

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Endometrial tissue of patients with polycystic ovary syndrome (PCOS) shows an impaired expression of insulin signaling molecules. Tyrosine phosphorylation of the insulin receptor (IR) by insulin promotes glucose uptake by activating the PI3K/Akt pathway. IR stability and function depend on the presence of the protein caveolin-1. Activation of IR increases phosphorylation of Y 14caveolin-1. Since the endometrium of PCOS patients is proposed to be insulin resistant, we evaluated the phosphorylation of IR and caveolin-1 in endometria of patients with insulin resistance (PCOSE-IR) compared to controls (CE). To explore the mechanism associated with this condition, cultured endometrial cells (T-HESC) were exposed to high glucose (25 mM, 24 h), an experimental condition that leads to insulin resistance in other cell types. Endometrial protein levels of phospho-Y972IR, phospho-Y 14caveolin-1 and caveolin-1 were determined by Western blotting. In cultured cells, protein levels of caveolin-1, IR