Endometria from Obese PCOS Women with Hyperinsulinemia Exhibit Altered Adiponectin Signaling

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Resumen

Hyperandrogenemia, hyperinsulinemia, and obesity affect 60-70% of patients with Polycystic Ovarian Syndrome (PCOS), who exhibit an altered endometrial insulin signaling. The aim of the study was to evaluate whether hyperandrogenism, hyperinsulinism, and obesity present in PCOS patients impair the endometrial adiponectin signaling pathway. The ex vivo study was conducted on 27 samples from lean (n=9), obese (n=9), and obese-PCOS (n=9) patients. The in vitro assays were performed in immortalized human endometrial stromal cells stimulated with testosterone, insulin, or testosterone plus insulin. Serum steroid-hormones, adiponectin, glucose, and insulin; body mass index, free androgen index, ISI-Composite, and HOMA were evaluated in the 3 groups. Ex vivo and in vitro gene expression and protein content of adiponectin, AdipoR1, AdipoR2, and APPL1 were determined. Adiponectin serum levels were decreased in obese-PCOS patients compared to lean (78%) and obese (54%) controls ($p<0.05$). AdipoR1 protein and gene expression were increased in obese group vs. obese-PCOS and lean groups (2-fold, $p<0.05$). In turn, AdipoR2 protein and mRNA content was similar between the 3 groups. APPL1 protein levels were reduced in endometria from both obese groups, compared to lean group (6-fold, $p<0.05$). Testosterone plus insulin stimulation of T-HESC and St-T1b leads to a reduction of adiponectin, AdipoR1, AdipoR2, and APPL1 protein content in both endometrial cell lines ($p<0.05$), whereas, in the presence of testosterone or insulin alone, protein levels were similar to basal. Therefore, endometrial adiponectin-signaling pathway is impaired in hyperandrogenemic and hyperinsulinemic obese-PCOS patients, corroborated in the in vitro model, which could affect endometrial function and potentially the implantation process.

Palabras clave

Palabras clave de autor:adipokine; adiponectin receptors; APPL1; hyperandrogenemia
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