

17th World Congress of the Academy of Human Reproduction

15–18 March 2017 Rome, Italy

TITLE

CHARACTERIZATION OF SEX HORMONE EFFECT ON HUMAN PREADIPOCYTES DIFFERENTIATION

AUTHOR/S

Shortrede J E (IT) [1], Montt-Guevara (IT) [2], Finiguerra M (IT) [3], Petrucci E (IT) [4], Simoncini T (IT) [5]

ABSTRACT

Context: Human Simpson-Golabi-Behmel syndrome (SGBS) preadipocytes cells are a unique and useful tool for studies human adipocyte biology. The cells originate from an adipose tissue specimen of a patient with SGBS. Adipose tissues are important contributors to the pathophysiology of insulin resistance, atherosclerosis, diabetes mellitus and metabolic syndrome. There is evidence that suggest an important role of sex steroid hormones in the regulation of localization and fat accumulation.

Objective: Study the effect of sex steroid hormones on SGBS cell differentiation by lipid droplets quantification.

Methods:

Cell culture and adipogenic differentiation: SGBS cells were cultured and differentiated to adipocyte as described by Fischer-Posovszky et al., 2008, in absence and presence of E2 10-9 M, P4 10-8 M, T 10-9 M or with their respective chemical inhibitors.

Lipid droplets quantification: Cells were fixed with paraformaldehyde 2% for 20 minutes at 0, 4, 8, 12, 15 differentiation days. Formation of lipid droplets was observed by fluorescence microscopy Olympus vx41 after staining with Oil red-O. Nuclei were stained with DAPI. Using Image J software Feret diameters and number of lipid droplets were analyzed.

Western blot: After differentiation without hormones, cells were cultured in high and low glucose medium in presence and absence of insulin 100 μ M and E2 10-9 M for 24 h. Expression of p-IRS1, IRS1, Glut1, p-Glut4 and Glut4 were analyzed.

Results: Sex hormone modulates SGBS cells differentiation increasing the number of lipid droplets and Feret diameter. Insulin and E2 treatment increased IRS1 and Glut4 phosphorylation.

Conclusions: Sex hormone modulates time differentiation of SGBS cells. In addition, insulin and E2 stimulate the activation of IRS1 and Glut4.

INSTITLITE