OMICS International
conferenceseries.com

Endocrinol Metab Syndr 2015, 4:4
http://dx.doi.org/10.4172/2161-1017.C1.012

Polycystic Ovarian Syndrome Conference

November 16-18, 2015 Seattle, USA

Glycolysis, TCA cycle, glycosylation, polycomb and trithorax complexes-related gene expression in the brain of fat tissue implanted polycystic ovarian syndrome mice

Silvana Aparecida Alves Correa
Federal University of Sao Paulo, Brazil

Polycystic Ovary Syndrome (PCOS) is the most common androgenic disorder in women during reproductive life. PCOS may also be accompanied by metabolic syndrome and recent studies point to leptin as playing a role in disrupting infertility. Previously, our group demonstrated the effect of gonadal white adipose tissue transplantation from wild-type lean and fertile female mice to isogenic obese anovulatory ob/ob mice. These complex metabolic interrelationships between obesity and PCOS have yet to be fully understood. The leptin treated mice show a decrease in the glucose metabolism. These confirm the ability of the adipose tissue-derived hormone leptin to regulate early crucial genes that are related to glycolysis mechanisms and to the TCA cycle. Besides that, quite relevant seems to be the responses triggered by the adipose tissue hormone, leptin, on the glycosylation in the brain molecules of obese mice. These mechanisms are markedly suppressed after treatment. These changes, caused by the rise of this hormone, induce the treated mice brain to display a generic profile similar to those of the normal weight. Ultimately, underlying neuronal changes caused by leptin in obese mice brain, there is an important role also being played by the histone code. Here there is evidence that leptin drives the chromatin packing to a more condensed pattern. In conclusion, leptin seems to change molecularly the expression of genes related to these signaling pathways. This can help us to better understand the neuronal mechanisms underlying the reversion of PCOS.

silaac@globo.com