

Effect of Selective Serotonin Reuptake Inhibitor, Escitalopram, on Adrenal Cell Steroidogenesis

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Depression is a highly prevalent psychiatric disorder worldwide and lowers the quality of life. Selective serotonin reuptake inhibitors (SSRIs) are the first line pharmacotherapeutic option for the treatment of depression and other psychiatric disorders. However, SSRIs are associated with a range of side-effects, many of them linked to the endocrine system e.g. hormonal imbalances, breast enlargement, sexual dysfunction, and menstrual cycle disorders. Thus, they affect both male and female sexual development and fertility. Five drugs of the SSRI family, namely citalopram, fluoxetine, fluvoxamine, paroxetine and sertraline, were previously shown to alter the steroid secretion by the H295R cells. However, the effect of Escitalopram, another drug in the SSRI family, on adrenal cell viability and steroidogenesis has not been studied. Hence, we aimed to study the effect of Escitalopram on the viability and steroid production of adrenal cells. Human adrenocortical carcinoma cell line, H295R, was grown in DMEM/F-12 medium supplemented with 7.5% FBS and 1% ITS+ Premix. Cells were seeded in 24-well plates or 96-well plates respectively for measurement of steroid production or cell viability. Cells were treated with different doses (0 – 400 μ M) of Escitalopram for 24 hours and spent culture media was harvested. Testosterone and progesterone were measured using Chemiluminescent microparticle immunoassay (CMIA) while androstenedione levels were measured by Enzyme-linked immunosorbent assay (ELISA). MTT assay was performed to assess the effect of Escitalopram on viability of the H295R cells. It was observed that production of progesterone was not affected by any of the drug doses. However, both the androgens measured showed reduced levels after treatment with Escitalopram. Significant decrease in the secretion of testosterone was seen only at higher doses of Escitalopram while androstenedione secretion was significantly decreased with all doses of Escitalopram. Interestingly, 100 – 400 μ M dose of

Escitalopram decreased cell viability also. Further experiments are required to decipher the molecular mechanisms underlying this negative impact of Escitalopram on adrenal cell steroidogenesis.