BIOCELL 43 (suppl. 4), 2019
ISSN 0327- 9545
ISSN 1667-5746 (online version)

ABSTRACTS BOOK

XXXVI Scientific Meeting of the Cuyo Biology Society

Mendoza, Argentina
6-7 December 2018

A84
TRIIODOTHYRONINE INDUCES CHANGES IN THE PROTEOME AND TRANSCRIPTOME OF ECC1 CELL LINE

Rinaldini E [1], Callegari EA [2], Paez MD [2], Troncoso ME [1,3], Gamarra Luques C [1,4], Hapon MB [1,3]
[1]IMBECU CCT Mendoza CONICET; [2]University of South Dakota; [3]Facultad de Ciencias Exactas y Naturales. UNCuyo; [4]Facultad de Ciencias Médicas. UNCuyo. E-mail: estefirinaldini@gmail.com; erinaldini@mendoza-conicet.gob.ar

During women´s reproductive life, steroid hormones govern the periodic changes of the endometrium. Estradiol (E2) and progesterone (P4) are essential to regenerate and differentiate the endometrium after menses, in order to provide an adequate milieu for embryo implantation. Both implantation and maintenance of pregnancy depend on the correct function and interaction between the corpus luteum, the receptivity and functionality of the uterus, and the action of thyroid hormones (THs). THs dysfunction causes irregularities during the menstrual cycle, failure of implantation and early pregnancy loss. Therefore, we hypothesize that interaction between THs, E2, and P4 in the endometrium are fundamental for development and differentiation, providing the right environment for implantation. The goal of this study was to investigate how endometrial cells respond to HTs-controlled signaling, through the analysis of the genomic and proteomic expression pattern. For this purpose, the human endometrial ECC1 cell line was used to determine the expression of E2 (ERS1 and ERS2), P4 (PGR) and TH (TRα and TRβ) receptors and also how it responds to E2, P4, and triiodothyronine (T3) stimulation. Besides, the pattern of protein and genomic expression in response to the combination of E2 and P4, with or without T3 was analyzed. The protein identification was performed by LC and tandem mass spectrometry. The data were analyzed using Mascot server, followed by PROTEOIQ (Premier Biosoft) and Functional Enrichment analysis tool (FunRich) software. The relative expression of mRNA was analyzed by RT-qPCR. The mRNA analysis confirmed the expression of PGR, ERS1, ERS2 and TRα, TRβ in ECC1 endometrial cells. The mRNA expression of PGR increased significantly ($p < 0.05$) with T3. The proteomic analysis showed that cells treated with E2 and P4 present a greater amount of proteins involved in biological processes such as energy metabolism; protein transport; and cell growth. Whereas the combination with T3 expressed a greater amount of proteins involved in immune response and signal transduction. This work elucidates that ECC1 cells respond differentially to the hormonal treatments with E2, P4, and T3, showing that these cells are good model for the study of the interaction between steroid and THs. On the other hand, the proteomic analysis allowed us to infer that T3 induces changes in the relative expression of proteins when it interacts with maternal hormones. Altogether, the changes observed, demonstrated that the presence of THs is essential to provide an optimal environment for embryo implantation and growth.

A85
NEUROBEHAVIORAL DEVELOPMENT OF RAT OFFSPRINGS EXPOSED TO AT2 RECEPTOR BLOCKAGE.

Soler García F[1,2], Sanchez S[1], Fuentes L[1].
[1]Laboratorio de Farmacología - Facultad de Química, Bióquimica y Farmacia - Universidad Nacional de San Luis; [2]IMIBIO-SL, CONICET. Ejército de los Andes 950, CP 5700. E-mail: fsolergarcia@gmail.com

Renin-Angiotensin system is known primarily for its effects on blood pressure and electrolyte homeostasis. Several studies suggestthat may play a role in the regulation of growth, maturation and neuronal functions. The brain has two major angiotensin II receptors: type 1 (AT_1) and type 2 (AT_2). A high expression of AT_2receptors has been found on rat embryonic and postnatal phases, and it is rapidly down-regulated in adult tissues. In young rats, the AT_2 receptor expression was determined in areas involved in learning, control of motor activity, sensory areas, and selective limbic system structures. Adult knockout AT_2 receptor mice presented a phenotype with a selective reduction of exploratory behavior but not locomotors activities.The aim of this study was to test the effects of prenatal AT_2 inhibition on pup's neurobehavioral development. Pregnant Wistar rats (200g) on the 13th day of pregnancy wereimplanted mini-osmotic pumps subcutaneously withAT_2 antagonist (PD123319, 1mg/kg/day, n=6) and saline solution (SF, n=6). The effects of prenatal blockage of AT_2 receptor were evaluated on physical landmarks and behavioral indicators at different postnatal ages (n=18/each test). Treated pups didn't show any change on somatic growth or developmental landmarks in comparison with control pups. Neurobehavioral tests shown significant differences in righting reflex (PD: 11.62±1.10 sec vs SF:8.13±1.17 sec, $p<0.05$) and negative geotaxis performances during first postnatal week (PD:15.49±1.70 sec vs SF:11.09±0.72 sec $p<0.05$). Rota-Rod test result in delay latency to fall of PD123319 treated pups at 12 postnatal day (PD:1.38±0.11 sec vs SF:0.84±0.12sec, $p<0.01$). Open field test shown decrease motion time (PD:86.3±4.1% vs SF: 67.7±5.1%, $p<0.05$), latency to start moving (PD:0.5±0.0 sec vs SF: 0.2±0.0sec, $p<0.05$) and exploratory activity (PD: 31.43±3.20 % vs SF:41.15±3.89 %, $p<0.05$) and significant increase on locomotor speed(PD:9.2±0.8 cm/sec vs SF: 7.6±0.7cm/sec, $p<0.05$).Rota-Rod test result in delay latency to fall of PD123319 treated pups on 12 postnatal day (PD:1.38±0.11 secvs SF:0.84±0.12 sec, $p<0.05$). At postnatal day 12, treated pups spend more time on transition areas (intermediates) of the open field (PD:35.6±4.5% vs SF: 15.7±2.0%, $p<0.05$) and control pups remains on the center areas (PD:12.8±2.6% vs SF: 21.9±5.0%, $p<0.01$).These results indicate that AT_2 receptorof the central nervous system could be involved on locomotorsestablishment, spontaneous activity and anxiety behavior during postnatal development.