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THE EFFECTS OF ESTROGEN ON THE MORPHOLOGY OF THE PYRAMIDAL NEURONS OF THE PARIETAL CORTEX OF FEMALE RATS

DREKIĆ D*, LOZANČE OLIVERA*, MILOVANOVIĆ N**, KERKEZ M***, STEFANOVIĆ D*** and ZORIĆ Z*

** Faculty of Veterinary Medicine, Belgrade; **Advanced Medical School, Ćuprija; *** Institute of Diseases for the Digestive System, Belgrade*

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In this study the effects of a neonatally (3rd day of life) administered single dose (1 mg) of estradiol dipropionate (E_2) on the parietal cortex of juvenile (16 days of life) female rats were investigated. The morphology, the volume of the soma and the thickness of the apical dendrite were studied in Golgi impregnated pyramidal neurons from both the external and internal pyramidal layers. In the treated female rats the volume of the soma and the thickness of the apical dendrite of pyramidal neurons was increased, surpassing the values in the corresponding controls. These findings indicated significant and prolonged effects of a single dose of estrogen, administered in the neonatal period, on the parietal neocortical pyramidal neurons of female rats.

Key words. estrogen, estradiol, rats, pyramidal neurons

INTRODUCTION

Many brain regions, particularly certain parts of the limbic system, have target neurons for sex steroids, which may induce significant changes (Mizukarni *et al.,* 1983; Rees *et al.,* 1980; Commins and Yahr, 1985; Phillis and O Regan,1988; MacLusky *et al*., 2004). Estrogen target cells have been found in the brain of many vertebrates including teleosts, amphibians, reptiles, birds and mammals (Rees *et al.,* 1980; Commins and Yahr 1985; Malobabić *et al.,* 2002; Drekić *et al.,* 2004). Estrogen stimulates dopamine release and neuronal excitability in the striatum (Chiodo and Cagginla, 1980; Becker and Ramirez, 1981), and alters receptor and synthetic degradative enzyme systems for amino acid neurotransmitters in extrahypothatamic areas (Maggi and Perez, 1985; Murphy *et al.,* 1998; Short *et al.,* 2004).

Estrogen exerts global activation effects on sensoromotor function in the rat and humans, enhancing seizure activity (Mattson and Cramer, 1985; Kretz *et al.,* 2005). Estrogen can influence nociceptive sensory processing, but the molecular mechanisms underlying sex differences in pain perception remain unclear. BDNF gene expression in certain brain structures is inhibited by inflammatory pain, yet estrogen may enhance central nervous system sensitization associated with sensory processing. Since alterations in BDNF gene expression in higher brain

centers may be relevant to cognitive changes that occur in recurrent depression, these results may provide insights into the coincidence of chronic pain and depression (Amy *et al.,* 2005), and spontaneous firing of neurons in the male rat cerebral cortex (Kelly *et al.,*1977), as well as other effects (Dennerstein *et al.,* 1984).

Different estrogen manipulations have effects on the expression of muscarinic acetylcholine receptors (mAChRs) in the adult female rat hippocampus. The hippocampus was obtained from rats in proestrus (control), ovariectomized at 2, 10 and 15 days of age, ovariectomized at 15 days of age and rats treated with 17-estradiol for 7 days, and rats treated with 17β-estradiol immediately after ovariectomy for 21 days. Rats estrogen status was monitored by measuring estradiol plasma levels and uterus relative weight. [^{3}H] quinuclidinyl benzilate ([^{3}H]QNB) binding studies indicated that ovariectomy time-dependently increases the number of mAChRs in the hippocampus when compared to those obtained from control rats (Valeria *et al.,* 2005). The role of high levels of dehydroepiandrosterone (DHEA) is to influence the ovarian function and embryonic resorption during early pregnancy in BALB/c mice. Pregnant animals were injected with DHEA following both the post-implantatory (DHEA-2) and peri-implantatory (DHEA-6) models. Morphological studies of implantation sites showed 40% of embryonic resorption in the DHEA-2 group while 100% of resorption was observed in the DHEA-6 group. The data presented here suggest that DHEA treatment during early pregnancy modulates the ovarian function and is responsible for embryonic resorption at different degrees, depending on the time of administeration (Valeria *et al.,* 2005; El-Bakri *et al*., 2004).

In this study we investigated the effects of a single dose of estrogen on the morphology of the parietal pyramidal neurons of female rats.

MATERIALS AND METHODS

The pyramidal neurons of the parietal cortex were investigated in 10 control and 10 female rats, neonatally (3 days old) treated with a single dose (1 mg) of (E_2) and sacrificed on the 16th day of life. After four weeks of fixation in 10 % neutral formalin, the brains were divided into smaller blocks which were impregnated by a modification of the Golgi-Kopsch method (Drekić and Malobabić, 1987).

After impregnation the tissue blocks were kept in 2% potassium dichromate solution at 37^{o}C for 1-2 days. The solution was changed daily, and prior to every change, the blocks were blotted gently in order to remove excessive dichromate precipitate. After dichromate, the tissue blocks were washed and then left for 3 days in fresh 2.5 % silver nitrate solution at 37^{o}C, and then in 96% ethanol for 24 hours. Dehydration was completed in 100 % ethanol in which the impregnated blocks were stored in the dark for a considerable time. After enhancing the blocks in liquid paraffin (56^{o}C) and leaving them for 10-20 minutes at room temperature, they were cut (140 μm thick sections) on the microtome. These frontal serial sections were dropped into xylol for at least 10 minutes.

The sections were mounted with Canada balsam and covered with a glass plate. Well impregated pyramidal neurons of the female rat parietal cortex (layers

III and V) were selected for this study. The thickness of the initial part of the apical dendrite and the perykaryon volume of the parietal pyramidal neurons were determined. In order to standardize the method, the initial part of each apical dendrite was measured at the distance of 10 μm from the point where its diameter became constant.

The ocular grid was used at the magnification of 190 x. The perykaryon volume of pyramidal neurons was calculated according to the formula for a rotatory elipsoid conus.

RESULTS AND DISCUSSION

Control animals

Morphology: The soma, the apical and basal dendrites of pyramidal neurons from both the external and internal pyramidal layers of the parietal cortex were well developed. In the control female rats, sacrificed in the early juvenile period, the initial part of the apical dendrites was without spines. These were present on segments of the basal dendrites which were far from the soma. The neuropil was rich in nerve fibers.

Morphometry: The volume of the soma of the external layer of pyramidal neurons was $2 \times 10^3 \pm 0.57\ \mu m^3$. The average neuron volume at the initial part of the apical dendrite was $3.9 \pm 0.2\ \mu m^3$. The volume of the soma in the internal layer of pyramidal neurons was $2.4 \times 10^3 \pm 0.74\ \mu m^3$. The neuron volume at the initial part of the apical dendrite was $5 \pm 0.2\ \mu m^3$.

Treated animals

Morphology: The shape of the soma of the parietal pyramidal neurons of both pyramidal layers was more pyramidal or pyriform than in neonatally treated female rats, sacrificed in the early juvenile period. These neurons were more darkly impregnated than the controls. The thinner and more elongated apical dendrite had numerous spines and contacts with other neurons. It appears that synaptogenesis increased in treated animals. The basal dendrites with numerous spines were more fat than the controls. The neuropil had a granular structure with numerous dendrites of other neurons.

Morphometry: In the treated rats, the volume of the soma of parietal pyramidal neurons in the external pyramidal layer was $4 \times 10^3 \pm 0.9\ \mu m^3$ which was significantly ($p<0.001$) less than the controls. The apical dendrite was significantly ($p<0.001$) bold (5.7 ± 0.4 μm) in treated rats compared to the corresponding controls. The volume of some of the internal pyramidal neurons of the parietal cortex in treated females was $6 \times 10^3 \pm 0.9\ \mu m^3$ which was significantly larger compared to the controls The apical dendrite was significantly ($p<0.001$) bold 7.7 ± 0.4 μm in treated rats, compared to the corresponding controls. In treated rats argyriophilia is more pronounced, with numerous synaptic contacts and dendrites.

Estrogens have both excitant and sedative effects on the central nervous system (Phillis *et al.,* 1988; Murphy *et al.,* 1998; Hao *et al*., 2003), significant

epileptogenic activity when applied directly to the cerebral cortex (Julian *et al.,* 1975) and they increase the spike frequency in epileptic patients (Logothetis *et al.,* 1995; Domingnez *et al.,* 2005). In rabbits, 17β-estradiol had a hypnogenic action when injected intracerbro- ventricularly (Paisley and Summerlee, 1984; Lee *et al*., 2004). Estradiol alters the concentration of brain imipramine binding sites (Ravizza *et al.,* 1985., Leuner and Shors, 2004), causing an increase in the B max and Kd of the imipramine binding sites to the frontal cortex of male rats (Wilson *et al.,* 1989). In our investigation, female rats treated with a single dose (1 mg of E_2) neonatally (3rd day of life) and sacrificed in the juvenile period (16th day of life), showed a highly significant increase in neuronal soma volume, as well as in the initial part of the apical dendrite both in external and internal pyramidal neurons. This suggests prominent sensitivity of the parietal neocortical pyramidal neurons to neonatally applied estrogen. Besides the expected reactivity of phylogenetically older parts, such as limbic system regions, the parietal neocortex is also sensitive to estrogen action, even though it is a phylogenetically newer structure (Drekić *et al*., 1990, 1995).

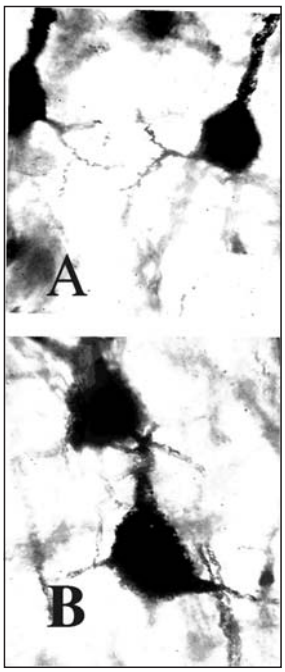

Figure 1. Internal pyramidal neurons in the parietal cortex of female rats. A. controls; B. treated. In treated rats argyrophilia is more pronounced with numeorus synaptic contacts and dendrites. The pyramidal neuronal shape is changed in to an oval or pyramidal one in treated animals (Golgi methods, modification Drekić and Malobabić; x 1000)

The decrease of pyramidal neuron perykaryon volume relates well to the greater density of cortical neurons in females (Haug, 1984) and in male rats (Drekić *et al.,* 1992). A generally accepted concept has been that the hormonal actions of 17β-estradiol are mediated via RNA-dependent protein synthesis (Phillis *et al.,* 1988; Kretz *et al*., 2005). This mechanism of action has been established for the uterus and was adopted for steroid action on hypothalamic neurons (McEwen *et al*., 1991; Lee *et al*., 2004; Li *et al*., 2004).

Estrogens have been demonstrated to rapidly modulate calcium levels in a variety of cell types. The relative importance of intra - and extracellular sources of calcium in estrogenic effects on neurons is also not well understood (Zhao *et al*., 2005). Previously, we had demonstrated that membrane-limited estrogens, such as E-BSA given before an administration of a 2-hour pulse of 17β-estradiol (E_2), could potentiate the transcription mediated by E_2 from a consensus estrogen response element (ERE)-driven reporter gene. Neither the L- nor P-type VGCCs

seem to play a role in estrogen action in these cells; while the N-type VGCCs are important in both the non-genomic and genomic modes of estrogen action (Zhao *et al*., 2005). Specific inhibitors also showed that the ryanodine receptor and the inositol trisphosphate receptor are important to E-BSA-mediated transcriptional potentiation (Zhao *et al.,* 2005). This report provides evidence that intracellular stores of calcium are required to couple non-genomic actions of estrogen initiated at the membrane to transcription in the nucleus. Extracellular sources of calcium are also important in both non-genomic and genomic actions depending on estrogens (Zhao *et al.,* 2005). Lee *et al*. (2005) indicate an effect of estrogen (E_2) on affect and cognition, which may be mediated by the cAMP response element-binding protein (CREB) pathway and CREB-related gene target brain-derived neurotrophic factor (BDNF). Lee *et al*. (2005) investigated the effect of E_2 on CREB expression and phosphorylation and BDNF expression in the amygdala and hippocampus, areas involved in emotional processing effect of E_2 on calcium/calmodulin kinase (CaMK IV) immunolabeling in the hippocampus. Estrogen (E_2) increased immunolabeling and mRNA levels of BDNF in the medial and basomedial amygdala and CA1 and CA3 regions of the hippocampus, but not in any other amygdaloid or hippocampal regions examined (Lee *et al.,* 2005).

Estrogen (E_2) increased immunolabeling of CREB and pCREB in the medial and basomedial, but not central or basolateral amygdala (Lee *et al.,* 2005). Estrogen (E_2) also increased CaMK IV and pCREB immunolabeling in the CA1 and CA3 regions, but not in the CA2 region or dentate gyrus, of the hippocampus. (Hao *et al.,* 2003). There was no change in immunolabeling of CREB in any hippocampal region. These data identify a signaling pathway through which E_2 increases BDNF expression that may underlie some actions of E_2 on affective behavior and indicate neuroanatomical heterogeneity in the E_2 effect within the amygdala and hippocampus (Murphy *et al*., 1998; Lee *et al.,* 2005).

Jontophoretic application of the hemisuccinate derivative of estrogen exerts effects on neuronal function which mimics those seen after i.v. administration of the steroid, but with a shorter latency (Smith *et al.,* 1988; El-Bakri *et al*., 2004). The observed steroid-induced potentiation of an excitatory neurotransmitter response parallels the reported activating effects of E_2 on seizure activity and sensoromotor function (Smith *et al.,* 1988; Zhao *et al*., 2005). During the development of various behavioral and physiological traits there are periods when presenting, or witholding specific types of stimulation, has profound consequences. Such sensitive periods are evident for language aquisition and sexual differentiation of normal sensory function (Shors *et al*., 2004). Most neurobiologists agree that these sensitive periods limited by the quantity of estrogen are periods when presenting or witholding specific types of stimulation shape the functional organization of the nervous system (Campbell *et al*., 2005). A sensitive period in the development of the mammalian visual system occurs when the terminals of the lateral geniculate neurons are segregating into cortical ocular dominance columns (Nordeen and Nordeen, 1990; Drekić *et al.,* 1992).

In a sexually dimorphic nucleus in the rodent spinal cord the sensitive period for sexual differentiation is limited by cell death and gonadal hormones that masculinize sexual function by enchancing the survival of a specific neuronal

population (Nordeen and Nordeen, 1990). Our study with the technique of Golgi silver impregnation, has revealed that neonatal estradiol treatment increases the number of dendritic spines in juvenile male rats, similar to the effects of estradiol seen in the hippocampus (Mc Ewen, 1991; Drekić *et al.,* 1992; Lee *et al*., 2004). Our finding of large sizes of apical dendrites and perykaryon volume are consistent with the observation that exogenous estrogen, given to adult animals, decreased the thickness of the cortex (Diamond, 1987; Drekić *et al.,* 1992). Thus, our data do not conflict with the hypothesis that one role of the cortical estrogen receptors is to aid in developing asymmetry (Diamond, 1987; Malobabić *et al.,* 2002; Drekić *et al.,* 2004). We believe that estrogen given to female rats in the early neonatal period, causes long lasting significant effects on neurogenesis, even in the phylogenetically youngest parts of the central nervous system, such as the neocortex (Drekić *et al.,* 1992).

The morphological and morphometric Golgi study of the pyramidal neurons indicate the sensitivity of the male rat (Drekić and Malobabić, 1987; Drekić *et al.,* 1992) and female rat parietal cortex to a single dose of estrogen, administered neonatally. The remarkable effect on these neocortical neurons is long lasting, because the decrase in male rats, and increase in female rats of the perykaryon volume and apical dendrite thickness is mainly expressed later, i.e. in the juvenile period.

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Address for correspondence:
Dmitar Drekić, DVM, PhD
Bulevar Oslobođenja 18
11000 Belgrade
Serbia &Montenegro
e-mail: dmitar@vet.bg.ac.yu

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DELOVANJE ESTROGENA NA MORFOLOGIJU PIRAMIDALNIH NEURONA PARIJETALNOG KORTEKSA KOD ŽENKI PACOVA

DREKIĆ D, LOZANČE OLIVERA, MILOVANOVIĆ N, KERKEZ M,
STEFANOVIĆ D i ZORIĆ Z

SADRŽAJ

Delovanje 1 doze (1 mg) estradiol dipropionata (E2) proučavano je na parietalnom korteksu ženki pacova u ranom juvenilnom periodu (16 dana života). Proučavana je morfologija, volumen some i debljina apikalnog dendrita kod neurona spoljašnjeg i unutrašnjeg piramidalnog sloja parietalnog korteksa impregni-

ranih pomoću Goldži metode (modifikovane po Drekiću i Malobabiću, 1987. godina).

U tretiranih ženki pacova, volumen some i debljina apikalnog dendrita piramidalnih neurona u oba piramidalna sloja parietalnog korteksa bio je značajno uvećan u poređenju sa odgovarajućim kontrolama iste starosti. Takođe je bio povećan i broj sinapsi na apikalnom i bazalnim dendritima u oba ispitivana sloja. Prema našim nalazima, postoji značajan produženi efekat jedne doze estrogena (date u neonatalnom periodu) na parietalne neokortikalne neurone ženki pacova proučavanih u ranom juvenilnom periodu.