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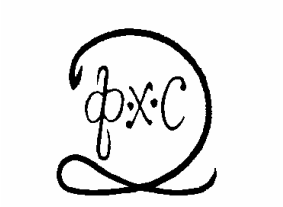

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CEREBRAL HYPOPERFUSION AND PROGESTERONE TREATMENT ALTER PARAMETERS OF OXIDATIVE STRESS AND ANTIOXIDANT DEFENCE IN MALE RATS

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ABSTRACT
Numerous natural compounds, like progesterone (P4), a sex steroid hormone, are highlighted as promising agents for treatment of different disorders including prolonged disturbance of blood flow. However, its action on several oxidative stress markers (pro/antioxidant balance, products of lipid peroxidation and phosphatidylcholine to lysophosphatidylcholine intensity ratio) and one of the major components of antioxidant system, superoxide dismutase in rat prefrontal cortex (PFC) following permanent bilateral occlusion of common carotid arteries (2VO) is not completely investigated. According to the obtained results, levels of investigated oxidative stress markers and SOD activity were altered in 2VO animals treated with vehicle, while P4 treatment returned them to control values. Overall, presented data indicate that P4 might manifest antioxidative features in PFC of 2VO rats.

INTRODUCTION
In pathophysiological conditions, including cerebrovascular insufficiency, the imbalance might be created between the generation of reactive species and their abolition by the components of antioxidant system, mostly by superoxide dismutase (SOD), catalase and glutathione peroxidase. Due to their inadequate removal, these over-synthesized highly toxic intermediates are capable of causing structural and functional cellular changes by oxidation of biomolecules, such as lipids, proteins and DNA that may initiate oxidative injury and even cell death in neurons. In the rat two vessel occlusion (2VO), model when both common carotid arteries are permanently ligated, the most prominent neuronal damage is detected in cortical pyramidal neurons in layer III and hippocampal CA1 neurons, as well as in the other vulnerable brain regions and cell types [1].

Different therapeutic strategies are proposed for protecting brain from the deleterious effects of restricted cerebral blood flow that causes limitation of tissue oxygen and nutrients supply. Their aim is to decrease the activation of toxic pathways and increase the activity of endogenous protective mechanisms. Recently, a few neuroprotectants are suggested, including sex steroid hormones, such as progesterone (P4) and its metabolites. Previous reports indicate that P4 treatment in various models of neuronal injury exhibits multiple neuroprotective outcomes by reducing blood-brain barrier leakage, cerebral edema, lesions' volume, thus promoting functional recovery, etc. This pleiotropic hormone might achieve its genomic and non-genomic actions via binding to the appropriate hormone receptors, controlling signalling cascades in neurons, astrocytes and microglia, modulating the inflammatory response, and regulating glutamate excitotoxicity [1, 2]. P4-mediated protection might be also related to membrane stabilization and reactive species scavenging, either of which might attenuate the damage provoked by oxidative stress [3]. However, the additional research is necessary to better analyse the antioxidant mechanisms underlying this hormone's neuroprotective effects in the state of cerebral hypoperfusion and allow its use in preclinical and clinical studies.

To addresses these disputable issues, in the present study, the rat 2VO model and P4 treatment were used to investigate the potential alterations of several parameters of oxidative stress (pro/antioxidative balance (PAB), products of lipid peroxidation (LPO products) and phosphatidylcholine (PC) to lysophosphatidylcholine (LPC) intensity ratio) and one of the major components of antioxidant system, superoxide dismutase (SOD). These changes were evaluated in prefrontal cortex (PFC), a brain region that might be directly affected by mild and prolonged disturbance of blood flow as well as hormone therapy.

METHODS

All research procedures were conducted on adult male Wistar rats (350 – 400 g) and approved by the Ethical Committee for the Use of Laboratory Animals of VINČA Institute of Nuclear Sciences – National institute of the Republic of Serbia, Belgrade (authorization numbers 02/11 and 323-07-04253/2016-05). The animals were randomly divided into three groups: controls subjected to sham operation and treated with vehicle (commercial flaxseed oil, dose 1 mg/kg/day, Sham + V) (n = 5); rats subjected to permanent common carotid artery ligation and vehicle treatment (commercial flaxseed oil, dose 1 ml/kg/day, 2VO + V) (n = 5); and animals with permanently occluded common carotid arteries treated with P4 dissolved in commercial flaxseed oil in dose 1.7 mg/kg/day (2VO + P4) (n = 5). The surgical procedures were conducted by neck ventral midline incision and exposition of both common carotid arteries, followed by their careful separation from carotid sheaths, cervical sympathetic and vagus nerves. In 2VO groups, both common carotid arteries were permanently double-ligated with 5-0 silk suture, while controls underwent the same surgical intervention but without actual occlusion of carotid arteries. The treatments in all experimental groups were administrated in the form of subcutaneous injections for seven consecutive days [2]. On the last day of the experiment, 4 h following the last injection, rats were decapitated with guillotine (Harvard Apparatus, Holliston, USA). PFCs were isolated on the ice, frozen in liquid nitrogen and stored at -70°C until processing.

All analyses were performed on samples homogenized in iced-cold medium (0.25 M sucrose, 1 mM Tris-HCl EDTA buffer pH 7.4) that were then centrifuged two times per 10 min, at 3000 rpm, 4°C (Beckman, Germany). The obtained supernatants were pooled, additionally centrifuged per 20 min, at 10000 rpm, 4°C (Beckman, Germany) and obtained pellets were resuspended in ice-cold 5 mM TRIS HCl pH 7.4 [4].

For PAB assay, as previously reported [5], 10 µl of sample/standard (dilution series of hydrogen peroxide)/blank (dH2O) were incubated with 200 µl of working solution (1 ml TMB (3,3’,5,5’-tetramethylbenzidine) cation solution with 10 ml TMB solution)in a dark place for 12 min, at 37°C. Then, the reaction was stopped by adding 100 µl of 2 N HCl. The absorbencies were read on microplate reader (WALLAC 1420-Victor2 Multilabel Counter, PerkinElmer, USA) at 450 nm.

To estimate the LPO levels, the methane sulfonic acid was added to the reaction mixtures containing samples/standards (dilution series of 10 mM TMOP (1,1,3,3,"-tetramethoxypropane))/blank (acetonitrile:methanol in the ratio 3:1) and working solution. Then the mixtures were heated at 45°C/60 min, centrifuged (15 min, 13000 rpm, 4°C) (Eppendorf 5417, Germany) and the absorbencies were measured at 580 nm in a microplate reader (WALLAC 1420-Victor2 Multilabel Counter, PerkinElmer, USA). The LPO levels were determined using the corresponding standard curve, as described earlier [5].

The activity of total SOD was evaluated by the adrenaline method of Misra and Fridovich, where one SOD unit (U) is defined as the amount of enzyme needed to exhibit 50 % dismutation of the superoxide radical at pH 10.2 [6].

The modified Folch procedure using a chloroform/methanol/water solvent system was used for total lipid extracts preparation. Obtained pallets were subsequently redissolved in matrix solution

(0.5 M DHB (2,5-dihydroxybenzoic acid, Merck Millipore, Germany) in methanol (Merck Millipore, Germany)), applied onto the stainless steel target plate and dried under warm steam of air [7]. All mass spectra were obtained in the reflector mode and "delayed extraction" conditions (delay time was approximately 130 ns) on a commercial matrix assisted laser desorption/ionization time of flight (MALDI-TOF) Voyager-DE PRO mass spectrometer (Sciex, USA).The raw data were processed with the "Data Explorer Software" version 4.9 (Applied Biosystems, USA).

Statistical analysis was performed by one-way analysis of variance (one-way ANOVA) followed by Tukey's *posthoc* test using GraphPad Prism 5 Software (USA). The significance level was $p < 0.05$, with values expressed as a percentage of the mean of the values in Sham + V group ± SEM (standard error of the mean).

RESULTS AND DISCUSSION

The brain tissue with its elevated metabolic activity, high oxygen consumption, lipid-rich content and low antioxidant capacity is highly prone to oxidative stress. A critically low oxygen supply to the brain in pathophysiological conditions is capable to modulate oxidative phosphorylation by mitochondria and considerably decrease cellular ATP production. As a result of rapid decline in cellular ATP to a level insufficient to sustain the activity of ion pumps, a prompt and widespread membrane depolarization of neurons and astrocytes arise. In parallel, mitochondria increase the production of highly reactive species that enhance lipid peroxidation and protein oxidation and induce the alteration of prooxidant/antioxidant balance in the cerebral tissues and lead to cell death. Although it is reported that reperfusion injury initiates intensive oxidative damage, there are also indications that enduring ischemic/oligemic condition provoked by 2VO creates mild, but permanent oxidative stress, which might be the cause of persistent and progressive neuronal damage [1]. These pathological processes might be prevented or even attenuated by different agents that are shown to exert protective effects in several experimental models, like those that mimic certain pathogenic features of brain dysfunction observed in advanced age- or age-related neurodegenerative diseases. Although P4 is reported to be protective against oxidative insults induced by glutamate, glucose deprivation, and $FeSO_4$/ amyloid β-peptide-provoked toxicity in primary hippocampal cultures [8], its effect in PFC in the state of cerebral hypoperfusion are still unclear. In current experimental setup, a relatively new but well defined assay for estimating the levels of PAB was used. As presented in **Figure 1.**, in 2VO group that received vehicle treatment, PAB level was augmented compared to the controls for 38 % ($p < 0.05$) and it was also increased ($p < 0.05$) regarding to P4 treatment. Previous study has shown a significant elevation of PAB level in acute cerebral ischemia and proposed that it could be used as a predictive marker of disease outcome [9]. To our knowledge, there are no available data about the effect of P4 on PAB levels in 2VO rat model, which makes them unique in the scientific literature.

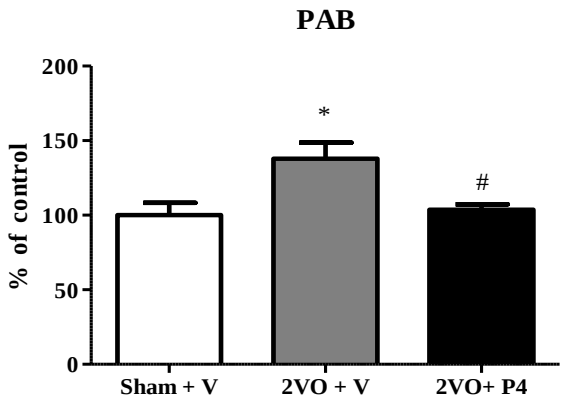

Figure 1. Levels of oxidative stress marker (pro/antioxidative balance (PAB) in prefrontal cortex of sham operated rats treated with vehicle, Sham + V, rats subjected to permanent ligation of common carotid arteries and either vehicle treatment, 2VO + V or P4 treatment, 2VO + P4. Data are presented as the mean ± SEM, whereas the values Sham + V are set as 100%. *$p < 0.05$, *vs.* control group; #$p < 0.05$ between 2VO groups.

Moreover, it was found that PAB status was positively correlated with other parameters of oxidative stress, such as LPO products [9]. Thus, in parallel with PAB levels, we estimated the level of LPO products. In comparison to Sham + V, in 2VO + V group the formation of LPO products was increased by 64 % ($p < 0.001$) (**Figure 2.**). Previous reports indicate that elevated levels of prooxidants lead to lipid peroxidation and formation of lipid radicals, like the aldehydes. These most abundant LPO products inactivate many cellular proteins and cause a rapid decrease and inhibition of the enzymatic and nonenzymic components of the antioxidative defense system. This provokes the overproduction of other reactive species and plays an important role in ischemic cell death, which might be associated with our previous findings [2]. Namely, previously we reported that prolonged reduction of cerebral blood flow alters the neuronal morphology, the amount of DNA fragmentation, along with the expression of progesterone receptors and the expression of the key elements of Akt/Erk/eNOS signal pathway. These modifications were attenuated by P4 treatment [2]. Although P4 does not have the characteristic chemical structure of an antioxidant, in the present study, it progressively modulated the levels of LPO products compared to vehicle treatment ($p < 0.01$) and returned them to values observed in controls (**Figure 2.**). This is consistent with literature data on P4 neuroprotective properties that are accomplished via its capacity to mitigate the lipid peroxidation by blocking the formation of free radicals and increasing the efficiency of their elimination by regulating antioxidant defense components [10]. These effects might be also achieved by controlling the isoprostanes production and pro-inflammatory genes activation [3] as detected in models of global cerebral ischemia, traumatic brain injury and subarachnoid hemorrhage [11]. Thus, the observed modulation of investigated parameters promoted by cerebral hypoperfusion might be indicative of a P4-induced reduction of oxidative stress and downregulation of proapoptotic signalling studied earlier [2].

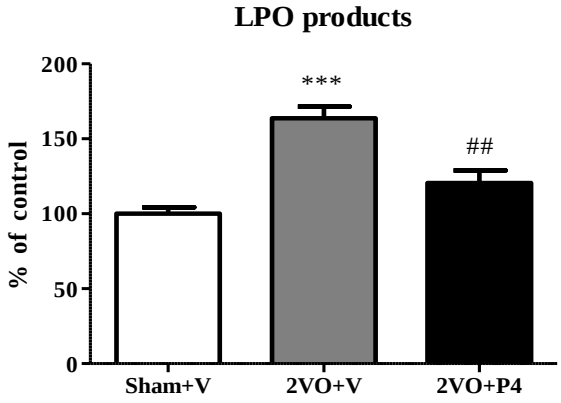

Figure 2. Levels of oxidative stress marker (products of lipid peroxidation (LPO products)) in prefrontal cortex of sham operated rats treated with vehicle, Sham + V, rats subjected to permanent ligation of common carotid arteries and either vehicle treatment, 2VO + V or P4 treatment, 2VO + P4. Data are presented as the mean ± SEM, whereas the values Sham + V are set as 100%. ***$p < 0.001$ *vs.* control group; ##$p < 0.01$ between 2VO groups.

PCs, the most abundant phospholipids, and PC-derived products, including LPCs (lipid species produced by spontaneous hydrolysis or enzymatic degradation of PCs) beside their structural functions and regulation of the physical properties of membranes, act as precursors of various lipid secondary messengers in diverse signalling cascades. The literature indicates that total concentrations of PCs and LPCs, as well as their intensity ratio, might be used as markers positively associated not only with normal aging but also with the onset and progression of different pathological conditions, including cerebrovascular and neurodegenerative diseases [7]. Since there are no available data about the lipid metabolism in PFCs' of 2VO rats treated either with vehicle or P4, this study attempted to add new knowledge about PC/LPC intensity ratio in both groups of hypoperfused animals in comparison to controls by employing MALDI TOF method followed by appropriate statistical analysis.

The observed PCs and LPCs peaks are listed in **Table 1**.

Table 1. List of phosphatidylcholine (PC) and lysophosphatidylcholine (LPC) species identified by MALDI TOF MS detected in positive ion mode.

Phospholipid Class	Adduct	m/z
LPC (16:0)	H	496.5
LPC (16:0)	Na	518.5
LPC (18:1)	H	522.5
LPC (18:0)	H	524.5
LPC (16:0)	K	534.5
LPC (18:1)	Na	544.3
LPC (18:0)	Na	546.4
LPC (22:6)	K	568.5
PC (32:0)	H	734.6
PC (32:0)	Na	756.6
PC (34:2)	H	758.6
PC (34:1)	H	760.6
PC (32:0)	K	772.6
PC (34:1)	Na	782.6
PC (36:4)	H	782.7
PC (36:3)	H	784.6
PC (36:2)	H	786.6
PC (36:1)	H	788.6
PC (34:1)	K	798.6
PC (36:4)	Na	804.6
PC (36:3)	Na	806.6
PC (36:2)	Na	808.6
PC (38:4)	H	810.6
PC (36:1)	Na	810.6
PC (36:4)	K	820.5
PC (36:1)	K	826.7
PC (38:4)	K	848.6

Figure 3. illustrates characteristic PC and LPC peaks observed in all three experimental groups, detected in positive ion MALDI spectra in the presence of DHB.

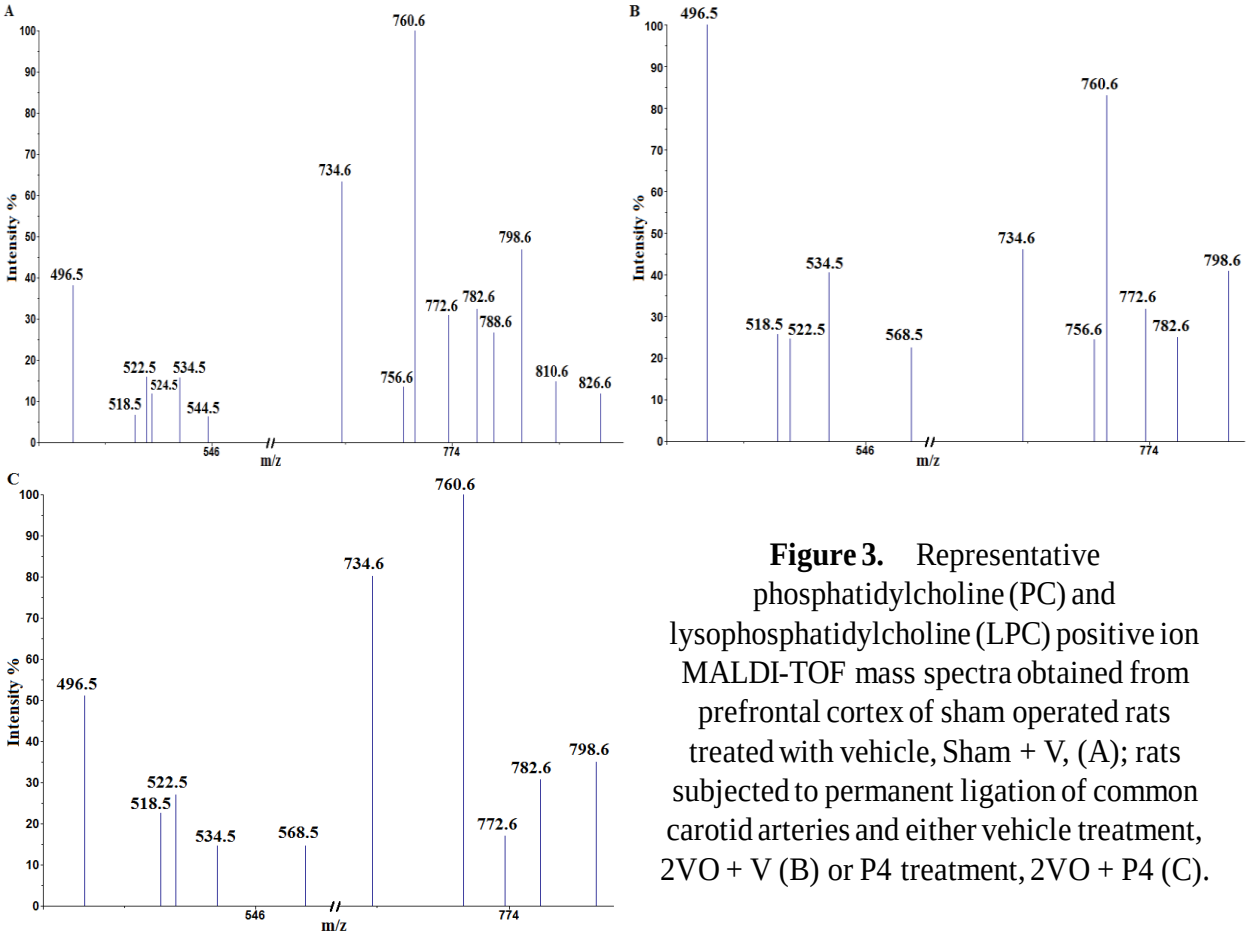

Figure 3. Representative phosphatidylcholine (PC) and lysophosphatidylcholine (LPC) positive ion MALDI-TOF mass spectra obtained from prefrontal cortex of sham operated rats treated with vehicle, Sham + V, (A); rats subjected to permanent ligation of common carotid arteries and either vehicle treatment, 2VO + V (B) or P4 treatment, 2VO + P4 (C).

The most intense peaks that correspond to PC 34:1 [M+H]+ (m/z 760.6), PC 34:1 [M+Na]+(m/z 782.6) and PC 34:1 [M+K]+(m/z 798.6); and LPC 16:0 [M+H]+ (m/z 496.5), [M+Na]+ (m/z 518.5) and LPC 16:0 [M+K]+ (m/z 534.5) were used to calculate the PC/LPC intensity ratio (**Figure 4.**). The other detected PC and LPC peaks detected in samples had much lower intensities and they were not used for further analyses.

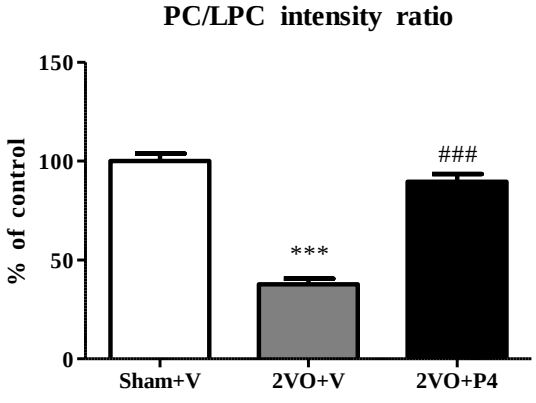

Figure 4. Phosphatidylcholine (PC) to lysophosphatidylcholine (LPC) (PC/LPC) intensity ratio calculated using most intense PC and LPC peaks (D). Intensities obtained from prefrontal cortex of sham operated rats treated with vehicle, Sham + V, rats subjected to permanent ligation of common carotid arteries and either vehicle treatment, 2VO + V or P4 treatment, 2VO + P4. Data are presented as the mean ± SEM, whereas values in Sham + V are set as 100%. ***$p < 0.001$ *vs.* control group; ###$p < 0.001$ between 2VO groups.

The observed decrease of PC/LPC ratio implies the abnormal phospholipid metabolism in 2VO + V group comparing to Sham + V ($p < 0.001$) which is most likely the result of increased activity of phospholipase A2 (PLA2). PLA2 is a lipolytic enzyme responsible for removal of the acyl group from the sn-2 position of PCs initiating the loss of vital membrane phospholipids and

generation of free fatty acids and LPCs. These species and products of their metabolism further react with other biomolecules and increase accumulation of lipid peroxides, alter membrane permeability, and ion homeostasis. Aforementioned processes, along with the loss of ATP and massive cellular calcium influx and calcium overload, may provoke the impairment of cellular components, trigger proapoptotic signalling and subsequent changes in neuronal morphology and even cell death [7] that were confirmed in our prior study [2]. Furthermore, in 2VO rats treated with P4, the PC/LPC ratio was increased in comparison to 2VO + V ($p < 0.001$) and returned to the values detected in controls (**Figure 4.**), indicating decline of the intensity of oxidative stress presented herein and amelioration of its downstream prosurvival signalling pathways revealed in our previous report [2].

A detoxification enzyme, SOD, catalyzes dismutation of the superoxide anion radical into less harmful oxygen and hydrogen peroxide. Thus, it contributes to the protection of the cells from detrimental agents that promote cell death [12]. In 2VO + V group the SOD activity was enhanced in comparison to controls ($p < 0.01$) (**Figure 5.**). The observed increased levels of proxidants (**Figure 1., Figure 2., Figure 3.**, and **Figure 4.**) along with previously reported apoptotic events [2], in current experimental setup however, indicate that investigated component of antioxidant system is not capable to completely compensate the overproduction of reactive species. These compounds further oxidize biomolecules, potentiate the accumulation of oxidative cell damage and finally, most likely provoke, at least partially, previously investigated cell death signalling pathway [2]. In contrast, as presented in **Figure 5.**, P4 decreased SOD activity in PFC regarding to vehicle treatment ($p < 0.05$) and returned it to basal values. The unchanged SOD activity along with no alteration of the other tested parameters when compared to controls, indicate that applied hormone might exert neuroprotective effect by attenuating 2VO-induced oxidative stress. The protective outcome in neuronal cells might also arise due to P4-binding to specific receptors in the nucleus and activation of gene transcription. In particular, SOD is one of the P4 target genes implicated in oxidative stress tolerance [11].

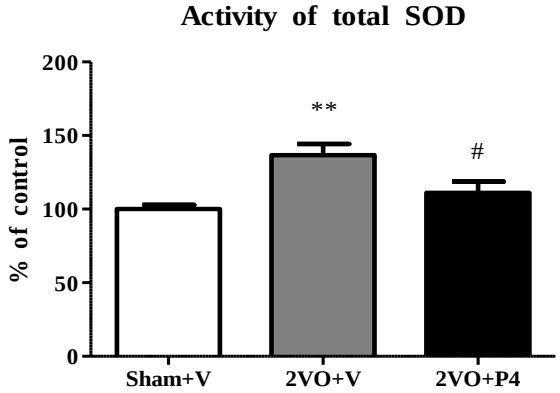

Figure 5. Activity of principal antioxidant marker (total superoxide dismutase (SOD) in prefrontal cortex of sham operated rats treated with vehicle, Sham + V; rats subjected to permanent ligation of common carotid arteries and either vehicle treatment, 2VO + V or progesterone (P4) treatment, 2VO + P4. Data are presented as the mean ± SEM, whereas the values of Sham + V are set as 100%. **$p < 0.01$ *vs.* control group; #$p < 0.05$ between 2VO groups.

CONCLUSION

Disturbed cognitive and behavioural functions observed in aging, senescence and/or patients with cerebrovascular diseases or cardiovascular pathologies are associated with cerebrovascular insufficiency/hypoperfusion. Even the pathophysiology of this type of brain injury is still ambiguous, the literature highlights that various pathways might be implicated, including the generation of reactive species, inflammation, cell death, and signaling pathway disturbance. Since there is a lack of efficient pharmacotherapy, cerebral hypoperfusion is still a challenging condition in clinical medicine. Among many proposed agents, those preventing the initiation of oxidative stress and targeting the components of antioxidant system might be the promising neurotherapeutic tools.

According to the presented results, hormone treatment modified 2VO-induced increase of investigated prooxidant markers, activity of main component of antioxidant defence system and

PC/LPC intensity ratio that were all restored to control values. Overall, repeated low-dose P4 treatment in PFC of animals subjected to permanent cerebral hypoperfusion, characteristic for elders and patients with cerebrovascular disorders, might exhibit neuroprotective outcome by stimulating its antioxidative capacity.

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