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EDITORIAL

In this issue: four outstanding scientific contributions to the journal in the form of review papers and original studies in experimental animal models

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In this second number of the first volume of the journal, we have selected two review papers and two original studies for publication.

Doxorubicin (DOX) is an anthracycline antibiotic discovered and isolated from *Streptomyces peucetius* several decades ago. It still remains as one of the most important antineoplastic agents used against a broad spectrum of cancer. The anticancer activity of DOX is attributable to its ability to bind and intercalate the DNA double strand causing a stereochemical disorder and thus inhibiting the synthesis of DNA, RNA, and proteins, eventually leading to cell death.

The review by PEREIRA & OLIVEIRA [1] summarized the current knowledge on the mitochondrial targets of DOX toxicity as well as the pharmacological strategies designed to decrease it. The authors reviewed the effects of DOX on mitochondrial physiology and bioenergetics as well as the consequences at the myocyte level, the non-pharmacological strategies already demonstrated to counteract DOX-induced cardiotoxicity, and the damage-preventing approaches involving mitochondria and their mechanism. The manuscript contains an in-depth analysis of the topic that the readers may find helpful for developing new strategies to counteract DOX toxicity focusing on the mitochondria as the target.

Persistent pulmonary hypertension of the neonate significantly contributes to an increase of neonatal morbidity and mortality, including asphyxia during delivery, meconium aspiration, and respiratory distress syndrome. In the review paper by GONZÁLEZ-LOZANO et al. [2], the evaluation of pulmonary vasodilators in animal models as well as in some clinical trials was reviewed. The authors concluded that experimental animal models are critical for examining, in depth, current pharmacological alternatives for treating human babies as well as for developing new therapeutic options. However, as with any experimental animal model, interspecies differences should be considered when evaluating the mechanisms involved in perinatal asphyxia, its consequences and treatment.

Regarding drug therapy, inhaled nitric oxide (iNO) remains as the pulmonary vasodilator of choice in neonates with pulmonary hypertension. However, sildenafil, an non-specific inhibitor of type-5 phosphodiesterases (5-PDE) that enhances the vasodilatory effects of nitric oxide by decreasing the biodegradation of a second-messenger GMPc, has

shown to be a good alternative both as a single drug or associated with iNO.

Cocaine addiction has been a serious medical and social problem in recent years and intensive research has been carried out in an attempt to explain the behavioral and neurobiological bases of its development. In this context, studies in experimental animal models to investigate the neurobehavioral consequences of cocaine discontinuation are fundamental to understand the mechanisms involved in addiction and craving.

GOMES SILVA et al. [3] evaluated animals treated once a day, during a week, with cocaine 20 mg kg^{-1} or saline (control), intraperitoneally. Following one or seven days of cocaine administration, animals were submitted to the forced swimming (FST) and open field tests (OFT), while another group of animals was decapitated and their brain striatum was removed for determination of dopamine (DA) and serotonin (5-HT) and their metabolites levels. Interestingly, the authors found evidence to indicate that one or seven days of cocaine administration in rats, at low doses, was not enough to produce symptoms of depression.

Ischemia of central nervous system is associated with increased extracellular concentrations of glutamate, which leads to uncontrolled activation of NMDA receptors and favors the influx of calcium. Glycine is an inhibitory neurotransmitter amino acid that acts as neuromodulator of NNMDA receptors, critically involved in the process of ischemic brain injury. URIBE-ESCAMILLA et al. [4] investigated the potential neuroprotective effects of glycine in an experimental animal model of ischemic brain damage.

The authors concluded that glycine administered i.p. to rats with permanent left carotid occlusion limited the ischemic brain damage, probably by increasing the neurological availability of glycine concentration enough to prevent the desensitization of NMDA receptors and consequently altering the cascade of events that lead to cellular death. However, levels of glycine have been associated with the severity of hypoxic encephalopathy-ischemic observed in human infants and further studies to clarify the neuroprotective role of glycine are warranted.

The issue of the journal is a continuation of our original plan to publish relevant studies and to be among the best options for journals covering the topics found in the *Journal of Theoretical & Experimental Pharmacology*. Although a substantial effort has been made to expedite the review and publication processes, we are still facing some tough challenges that have required the dedicated effort of numerous people. Although these challenges appear to be enormous, the support provided by the Editorial Board is encouraging and has facilitated the work of the supporting team.

As always, I would like to express my gratitude to the authors of the published manuscripts for giving us the opportunity to publish their studies in this journal, and to the readers for using the journal as a source of information.

The Editor

REFERENCES

[1] Pereira GC, Oliveira PJ. Pharmacological strategies to counteract doxorubicin-induced cardiotoxicity: the role of mitochondria. J Theor Exp Pharmacol 2010; 1: 39-53.

[2] González-Lozano M, Nava-Ocampo AA, Trujillo-Ortega ME, Alonso-Spilsbury M, Mota-Rojas D. A critical overview of clinical and experimental studies on pulmonary vasodilators in newborns. J Theor Exp Pharmacol 2010; 1: 54-61.

[3] Gomes Silva MI, De Oliveira Citó MC, Falcão do Amaral J, De Aquino Neto MR, Arcanjo Moura B, Freire de Vasconcelos P, et al. Behavioral and neurochemical evaluation in rats withdrawn from repeated cocaine treatment and exposed to the forced swimming and open field tests. J Theor Exp Pharmacol 2010; 1: 62-71.

[4] Uribe-Escamilla R, Padilla-Martín K, González-Maciel A, Arch-Tirado E, Nava-Ocampo AA, Alfaro-Rodríguez A. Neuroprotective effects of glycine in rats with permanent cerebral ischemia. J Theor Exp Pharmacol 2010; 1: 72-75.