resent a useful pharmacological approach for cognitive enhancement.

Acknowledgements: This study was supported by the Statutory Activity of the Institute of Pharmacology, Polish Academy of Sciences, Krakow, Poland.

References

[1] Albuquerque, E.X., Pereira, E.F., Alkondon, M., Rogers, S.W., 2009. Mammalian nicotinic acetylcholine receptors: from structure to function. Physiol Rev 89, 73–120.
[2] Nikiforuk, A., 2016. Serotonergic and Cholinergic Strategies as Potential Targets for the Treatment of Schizophrenia. Curr Pharm Des 22, 2093–2116.
[3] Radek, R.J., Kohlhaas, K.L., Rueter, L.E., Mohler, E.G., 2010. Treating the cognitive deficits of schizophrenia with alpha4beta2 neuronal nicotinic receptor agonists. Curr Pharm Des. 16, 309–322.

doi: 10.1016/j.euroneuro.2018.11.429

P.316 Ketamine in a model of depression resistant to tricyclic antidepressants

V. Pesic*, A. Dobrosavljević, D. Stanić, B. Batinić, B. Plećaš

University of Belgrade- Faculty of Pharmacy, Department of Physiology, Belgrade, Serbia

Background: The emergence of rapid-acting antidepressants such as ketamine motivated a series of studies aiming to reveal the molecular mechanism of ketamine antidepressant effect and to enable the clinical application of rapid-acting antidepressants. We have focused on a model based on chronic administration of adrenocorticotropic hormone (ACTH) in rats, previously reported to induce depressive-like phenotype that resembles to treatment resistant depression (TRD) [1]. Another well-adopted model of depressive-like behavior in rats is a long-term corticosteroid application.
Objective: The aim of the study was to examine repeated application of ketamine in a TRD model induced by long-term ACTH treatment, along with a model induced by chronic corticosterone application.
Methods: In first experiment male Wistar rats, eight weeks old were used, divided into four groups: corticosterone (dissolved in drinking water 100mg/L, 21 days), ketamine (acute administration on 21st day of the experiment, i.p. 10mg/kgBW), Ket/Cort (chronic corticosterone treatment and acute administration of ketamine) and control group (acute saline i.p. on the last day of experiment) . At the end of a study a forced swimming test was performed. In the second experiment male Wistar rats were divided into 4 treatment groups: control (treated with saline for 3 weeks s.c.); ACTH (10 μg/400 μl/day s.c. 21 days); ACTH/KET1 (Treated with ACTH for 3 weeks and acutely with 10 mg/kg of ketamine); and ACTH/KET2 (ACTH and ketamine on 21st and 22nd day, 10 mg/kg). On the last day of treatments forced swim test was conducted. The analyzed parameters were time of immobility, latency to first immobility, struggle time, swim time and number of dives. Data were subjected to two-way ANOVA analysis, with post hoc Bonferroni testing.
Results: In animals chronically treated with corticosterone there was a significant increase of the time of immobility in comparison with control group ($p = 0.009$) while one acute administration of ketamine notably decreased this parameter compared to corticosterone group ($p = 0.011$). In the second experiment ACTH treatment significantly prolonged the time of immobility compared to the controls ($p = 0.032$), while ketamine applied once reduced this parameter only by a trend to significance ($p = 0.063$) relative to ACTH group. Second application of ketamine in ACTH treated rats reduced time of immobility significantly ($p = 0.040$). In both groups that received ketamine, time of swimming was increased compared to ACTH only treated group ($p = 0.035$ in ACTH/KET1 and $p = 0.059$ in ACTH/KET2). Also, ketamine significantly increased number of diving in ACTH/KET1 rats compared to ACTH group ($p = 0.022$).
Conclusion: In corticosterone-induced model of depression, one acute application of ketamine i.p. in rats exerts antidepressive-like effect. while in the animal model of ACTH-induced depression resistant to tricyclic antidepressants, the two applications of ketamine (24h apart) were needed to induce antidepressive-like effect. These results point to the necessity of examining endocrine status, particularly hormones of the hypothalamic-pituitary-adrenal axis, before considering introduction of the ketamine.

Reference

[1] Kitamura, Y., Araki, H., Gomita, Y., 2002. Influence of ACTH on the effects of imipramine, desipramine and lithium on duration of immobility of rats in the forced swim test. Pharmacol. Biochem. Behav. 71, 63–69.

doi: 10.1016/j.euroneuro.2018.11.430

P.317 Antidepressant effect of adipokinetic hormone/red pigment-concentrating hormone family of peptides in olfactory bulbectomy model of rats

O. Mutlu[1,*], L. Kleteckova[2], J. Horáček[3], K. Holubová[2], T. Páleníček[3], G. Ulak[1], C. Höschl[3], K. Vales[2]

[1] *Kocaeli University, Department of Pharmacology, Izmit-kocaeli, Turkey*
[2] *National Institute of Mental Health, Experimental Neurobiology, Topolova 748- 250 67 Klecany, Czech Republic*
[3] *National Institute of Mental Health, Psychiatry, Topolova 748- 250 67 Klecany, Czech Republic*

One of the major neuropeptide groups in insects is the adipokinetic and hypertrehalosemic peptide family, which belongs to the adipokinetic hormone/red pigment-concentrating hormone (AKH/RPCH) family of peptides. In our previous studies [1,2], we showed antidepressant effects of AKH after both acute and chronic injections in forced swimming test (FST) in mice. The aim of this study was to investigate the effects of Anax imperator AKH (Ani-AKH), Libellula auripennis AKH (Lia-AKH) and Phormia-Terra