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HASIL PENILAIAN SEJAWAT SEBIDANG ATAU *PEER REVIEW*
KARYA ILMIAH : JURNAL ILMIAH

Judul Karya Ilmiah (Artikel) : Activation of MIP-2 and MCP-5 Expression in Methylmercury-Exposed Mice and Their Suppression by N-Acetyl-LCysteine

Jumlah Penulis : 6 Orang

Status Pengusul : Muflihatul Muniroh, Ainun Rahmasari Gumay, Darmawati Ayu Indraswari, Yuriz Bahtiar, Hardian Hardian, Saekhol Bakri.

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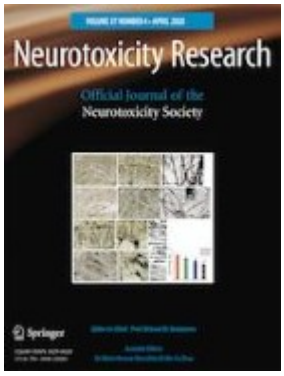
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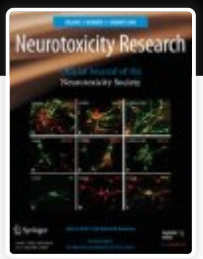

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Volume 37, issue 4, April 2020

25 articles in this issue

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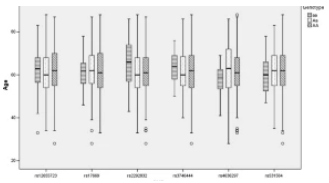

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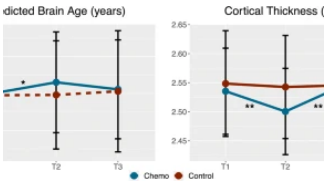

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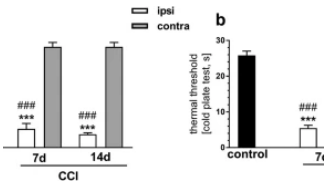

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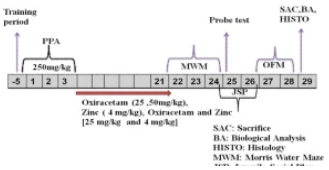

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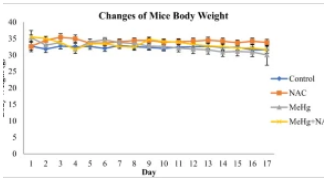

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ORIGINAL ARTICLE

Oxiracetam and Zinc Ameliorates Autism-Like Symptoms in Propionic Acid Model of Rats

Raju Paudel[1] · Khadga Raj[1] · Y.K. Gupta[2] · Shamsher Singh[1]

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Abstract

Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder characterized by restrictive behaviour, deficit in social skills and interaction. The multifactorial etiology, complex pathophysiology and different combination of symptoms (unusual speech patterns, frequent repetition of phrases) make it difficult to treat. Thus, present study aimed to find the protective effects of oxiracetam alone and in combination with zinc on brain behavioral, biochemical, pro-inflammatory cytokines and neurotransmitters level. Rats were administered with propionic acid (250 mg/kg p.o.) for 3 days and immediately on next day treatment were given with oxiracetam (25, 50 mg/kg i.p), zinc (4 mg/kg) as well as oxiracetam (25 mg/kg i.p) in combination with zinc (4 mg/kg p.o). Behavioral parameters were performed from 22th to 28th day. On 29th day, all the animals were sacrificed by cervical dislocation and the brain was preserved for biochemical (LPO, GSH, nitrite, mitochondrial complex I, IV and cAMP), neuroinflammatory (TNF-α, IL-1β, IL-6) and neurotransmitters (5-HT, GABA, glutamate and acetylcholine) analysis. The propionic acid administration showed memory impairment, restrictive behavior, increased proinflammatory cytokines level, biochemical and neurotransmitters alteration. However, treatment with oxiracetam alone and in combination with zinc significantly attenuated behavioral, biochemical, inflammatory cytokines and restored neurotransmitters level. The finding of present study demonstrated that oxiracetam alone and in combination with zinc afforded superior anti-autistic effect through antioxidant, anti-inflammatory and anti-excitotoxic mechanisms and could serve as attractive strategy in managing autism.

Keywords Autism spectrum disorders (ASD) · Propionic acid · Oxiracetam · Zinc · Neuroinflammatory · Neurotransmitters

Abbreviations

ASD	Autism spectrum disorder
PPA	Propionic acid
AMPAr	α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor
NGF	Nerve growth factor
NMDAr	N-Methyl-D-aspartic acid receptors
ORC	Oxiracetam
Zn	Zinc

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Introduction

Autism spectrum disorders (ASD) are the complex neurodevelopmental enduring disorder commonly reflected in early years of life with deficits in social skills and interaction, language impairments as well as presence of restricted interest and behavior (Livingston and Happé, 2017). ASD affects 1–2% of the population and is five times more common in males than in females. According to the neurobiologist at Bangalore's National Council of Biological Sciences (NCBS), about 10 million children likely to suffer from autism that is 1–1.5% or 1 in 66 children below ages 2 and 9 are affected in India (Baio et al. 2018).

The pathological aspect of ASD includes down-regulation of Co-enzyme Q10 and reduced mitochondrial complex activity leads to ATP deficit (Valenti et al. 2018). Excessive glutamate-dependent calcium discharge and excitotoxicity cause neuronal cell death is responsible for cognitive and behavioral abnormalities (Verkhratsky and Parpura 2016). The behavioral deficits in neuropsychiatric disorders like autism

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ORIGINAL ARTICLE

Aluminum-induced "mixed" cell death in mice cerebral tissue and potential intervention

Yan-xia Hao[1,2] · Mei-qin Li[1] · Jing-si Zhang[1] · Qin-li Zhang[1,3] · Xia Jiao[1] · Xiu-liang Ji[1] · Huan Li[1] · Qiao Niu[1]

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Abstract

The brain is one of organs vulnerable to aluminum insult. Aluminum toxicity is involved in neurobehavioral deficit, neuronal cell dysfunction, and death. The aim of this study are as follows: (1) to evaluate the repairing efficiency of Necrostatin-1 (Nec-1), a cell death inhibitor, and Z-VAD-FMK, a pan-caspase inhibitor, on Al-induced neurobehavioral deficit and neuronal cell death, in order to evidence the cell death inducing ability of aluminum, and (2) to primarily explore the possibility of treating neuronal cell loss–related disease, such as Alzheimer's disease, with Nec-1 and Z-VAD in Al-induced dementia animal model. We found Nec-1 and Z-VAD-FMK alone or in combination could reduce aluminum-induced learning and memory impairment in mice. Pathohistological results indicated that Nec-1 and Z-VAD-FMK can decrease Al-induced neuronal death cell. In addition, some cell death–associated proteins in cell death signal pathway were inhibited by Nec-1 and Z-VAD-FMK in Al-exposed mice. In conclusions, Nec-1 and Z-VAD-FMK can repair the injury of learning and memory induced by aluminum in mice. Furthermore, Nec-1 was more obvious to repair the injury of learning and memory function compared with Z-VAD-FMK. Nec-1 and Z-VAD-FMK can repair the Al-induced morphological injury of cell and reduce the amounts of dead cell, and repairing effects were more significant at higher doses. The effect of Nec-1 was stronger than Z-VAD-FMK, though their mechanism was different. The combination of them had the strongest effect. Our study evidenced Al-induced neuronal necroptosis and apoptosis existing in animal model and suggested potential therapeutic effects of Nec-1 and Z-VAD-FMK on neuronal cell death in neurodegenerative diseases.

Keywords Necrostatin-1 · Necroptosis · Apoptosis · Cell death · Aluminum

Introduction

Aluminum products are closely related to our life. It is used in industry, water treatment, food additives, and medicines such as antacids and vaccine adjuvant. Aluminum exists in the form of aluminum ions, which form insoluble compounds when absorb in living organism (Exley 2013, Walton 2014). Provided that it accumulates too much in the body, it will produce chronic toxicity and damage the body's kidney, bone, brain, and other organs (Esparza et al. 2018). More importantly, the intake of aluminum in the brain is more than the elimination, resulting in an increase in the net content of aluminum, and this process is related to the increase of age (Walton 2014). At present, aluminum is believed to have obvious neurotoxicity, and it is suspected to have a relationship with varieties of neurodegenerative diseases, for example Alzheimer disease (AD) (Neha Singla 2013), amyotrophic lateral sclerosis, and Parkinson's dementia of Guam (Bondy 2014, Esparza et al. 2018). AD is a progressive neurodegenerative disease with clinical manifestations of deteriorating cognitive and memory functions, and various neuropsychiatric and behavioral disorders (Liu-Seifert et al. 2015). Neuropathologic features of Alzheimer's disease caused by aluminum accumulation in neurons included neurofibrillary tangles (NFTs) (Walton 2010), Aβ plaque formation (Yumoto et al. 2009), and reduced connections between nerve cells (Yang et al.

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