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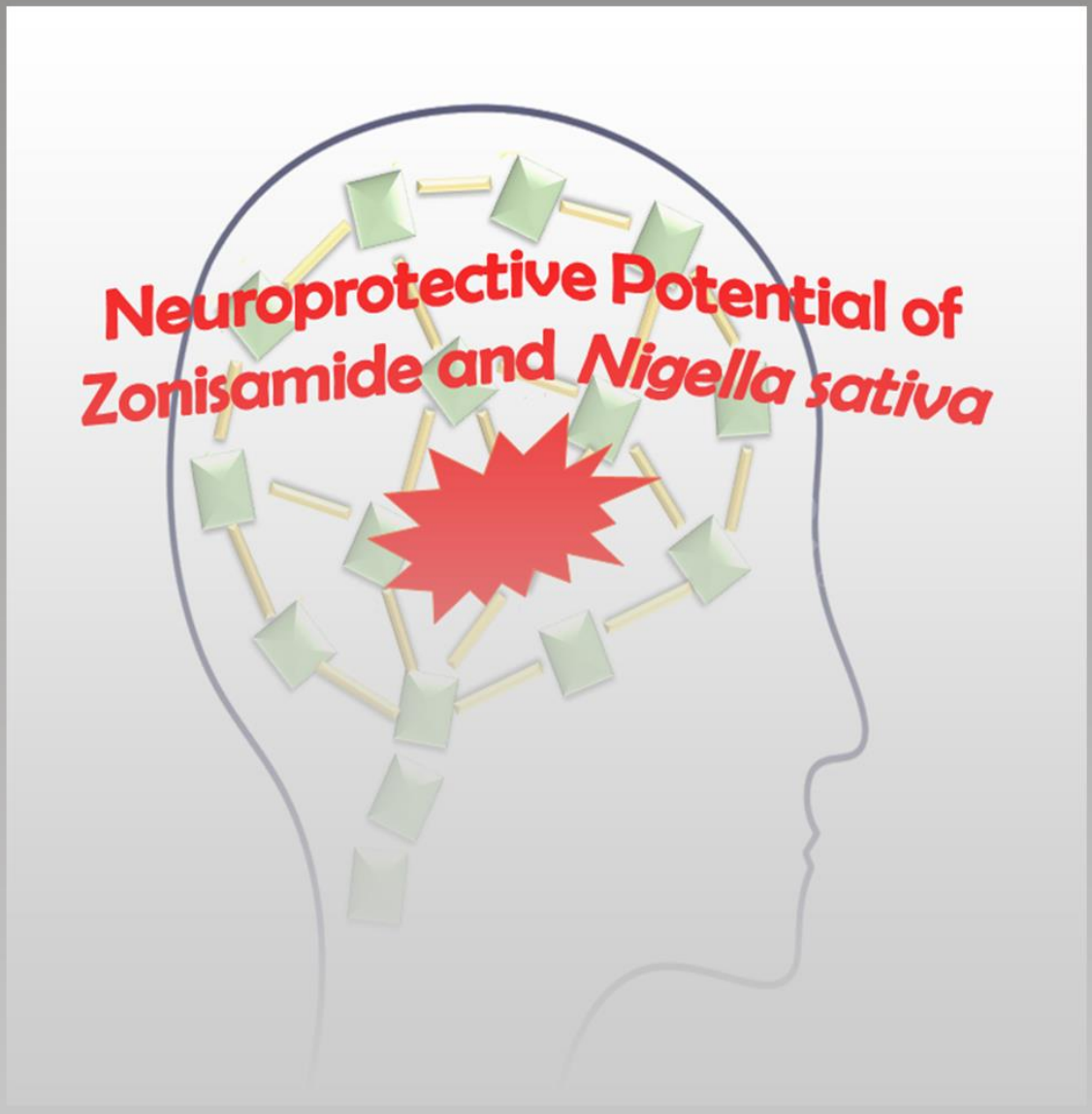

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From the Publisher's Desk

Welcome to Biotechnology Kiosk!

This issue includes a research article presenting animal studies on neuroprotective properties of drugs in treating traumatic brain injury and editor picks.

We hope our readers will enjoy reading these news and views on the current cutting-edge topics that include latest research breakthroughs in different areas of medicine and biotechnology.

We look forward to receiving your feedback. We do hope that you will enjoy reading this issue of Biotechnology Kiosk. Please do write to us with your comments. Your suggestions are always appreciated.

Dr. Megha Agrawal & Dr. Shyamasri Biswas.

Editors-in-Chief, Biotechnology Kiosk

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Volume 3, Issue 8 Aug 2021

Research Article

Neuroprotective Potential of Zonisamide and *Nigella sativa* on Traumatic Brain Injury-Induced Oxidative Stress in Mice

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Abstract

Traumatic brain injury (TBI) is a primary public health concern that has caused millions of deaths and disabilities around the world. Numerous medications are available to relieve TBI-associated complications. However, these medications do not prevent further harm from occurring. Therefore, the unmet need is the development of novel therapeutic medicines that protect against neuronal damage as a result of trauma and its repercussions, especially from secondary injury. We present a study using Swiss albino mice (25-30 g) of either sex to address the therapeutic issues concerning TBI. In our study, TBI was induced by the weight-drop method. Oxidative stress parameters were observed following the administration of zonisamide (100 mg/kg) and *Nigella sativa* (NS) (300 mg/kg) per se and in combination. The levels of glutathione (GSH), superoxide dismutase (SOD), and catalase (CAT) were observed to be significantly enhanced, but the levels of malondialdehyde (MDA) and nitric oxide (NO) were significantly reduced by treatment with the mentioned dugs. Our findings affirmed the potential function of both drugs in preventing TBI-induced oxidative damage.

Keywords: *Nigella sativa, Oxidative stress, Traumatic brain injury, Weight drop model, Zonisamide*

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INTRODUCTION

Traumatic brain injury (TBI) is considered a global health threat that kills and disables millions of people every year [1, 2]. Estimated 64 to 74 million new TBI cases occur every year, with the highest prevalence in the United States, Canada, and Europe [3], with around 53,000 casualties and 283,000 hospitalizations. Furthermore, on a larger scale, the predicted existence of TBI in youngsters aged 0-14 is about 475,000 [4]. TBI also has a substantial financial cost to societies, with an estimated $400 billion healthcare cost that is spent annually to manage TBI [5].

One of the aspects that influences TBI outcomes are biochemical cascades that emerge following primary and secondary injury. TBI creates a metabolic and ionic imbalance, resulting in an overabundance of reactive oxygen species (ROS) production and oxidative stress leading to brain malfunction and mortality [6, 7]. After TBI, various oxidative stress markers, including reactive oxygen species (ROS), are formed in the brain. At the same time, the level of anti-oxidant enzymes decreases. As a result, developing anti-oxidant techniques are a top priority in current attempts to treat trauma-induced brain injuries. It is to be noted that the US food and drug administration (FDA) has yet to approve a medicine for TBI treatment [8].

Nonetheless, it is expected that focusing on the pathological mechanisms might help to mitigate TBI's effects. Anti-oxidants have been investigated as a therapy option for TBI and have been demonstrated to have a neuroprotective impact. Excitatory amino acids like glutamate are released into the synapse after mechanical stress, overstimulating N-methyl-D-aspartate (NMDA) receptors. As a result, there is a Ca2+ overload and enhanced depolarization due to ionic imbalance. Excessive formation of ROS and, as an outcome, oxidative stress is caused by an excess of intracellular Ca2+ and excitotoxicity [9].

Zonisamide is a novel broad-spectrum antiepileptic drug that works well for refractory partial seizures [10–12]. Zonisamide has a structural, molecular, and pharmacokinetic profile apart from other antiepileptic drugs [10]. Zonisamide is considered to help dopaminergic and serotonergic neurotransmission by blocking voltage-dependent Na+ channels, lowering voltage-dependent T-type inward Ca2+ currents, binding to the GABA–benzodiazepine receptor complex, and blocking voltage-dependent Na+ channels. Zonisamide reduces Ca2+-dependent K+ evoked extracellular glutamate release [14, 15].

Recently, plants and herbs' low toxicity and cost-effectiveness have gained attention to investigate potential pharmacological activities. Nigella sativa (NS) is an herb belonging to the family Ranunculaceae, and its seeds are conventionally used to treat various diseases around the globe [16]. NS and its active component, thymoquinone have been shown in several human and animal investigations to have antioxidative properties [17]. Immunomodulatory [18], neuroprotective [19], antibacterial [20], hypertensive [21, 22],

and hypoglycemic [23] effects. The protective effect of NS to demolish oxidative stress plays a pivotal role in treating various diseases such as cancer, arteriosclerosis, and ischemia, in which free radical generation is involved [24, 25]. The usage of NS extract and thymoquinone also results in morphologic improvement and apoptosis, thus suggesting NS therapy could be helpful in the prevention of neurodegeneration.

We have presented an animal study on neuroprotective potential of Zonisamide and Nigella sativa on traumatic brain injury-induced oxidative stress.

MATERIALS AND METHODS

Animal

The study used Swiss albino mice (25-30g) of either sex, kept under natural day and night cycles in polypropylene cages with rodent food and water *ad libitum*. The Institutional Animal Ethics Committee of Maharshi Dayanand University Rohtak, Haryana, India, authorized all experimental protocols.

Induction of TBI by weight drop method

Mice were given a 2% isoflurane anaesthetic and permitted to breathe normally without tracheal intubation. After that, mice were placed on a sponge using surgical tape, and a small longitudinal incision was given to the overhead of mice exposed to the skull. The metallic disc was centrally fixed on the exposed skull and adequately placed the mice under the metallic pipe. Then the metallic spherical weight (60g) freely falls through the metallic pipe over the head of the mice. After that metallic disc was removed, the exposed skull was sutured. Finally, neosporin powder was spread over the surgery site and returned to its home case for recovery.

Treatment schedule

The experimental study was designed to determine oxidative parameters at 3, 6, 24, and 72h. The zonisamide and NS were administered per se and in combination 30 min after the induction of TBI. Mice were divided into five groups, each consisting of 6 mice. Group 1, the control group, did not receive any injury or drug treatment, whereas TBI was induced in all other four groups then Group 2 received vehicle, Group 3 received zonisamide (100mg/kg), Group 4 received NS (300mg/kg) and Group 5 received combination of both zonisamide (100mg/kg) and NS (300mg/kg).

Oxidative parameters

Tissue preparation: After cervical dislocation at 3, 6, 24, and 72h, the animal's brain was removed quickly and dipped in ice-cold normal saline (0.9% NaCl); after that, brain tissue was homogenized immediately in an ice-cold pH 7.4 phosphate buffer with 10 times its weight. The brain homogenates were centrifuged for 15 minutes at 3000 rpm. Aliquots of supernatants and cell pellets were separated and kept at -20°C in deep freeze (Blue Star Pvt. Ltd., India) until further oxidative parameters estimations were processed.

Superoxide dismutase (SOD) estimation:

In this method, 0.15ml of hydroxylamine hydrochloride was added to a reaction mixture containing 75µl nitro blue tetrazolium (NBT), 1.9ml distilled water, 0.15ml Triton-X100, and 0.1ml supernatant. The inhibition

of reduction of NBT by superoxide dismutase (SOD) present in the supernatant was then estimated by measuring the absorbance of the mixture at 560nm using a UV–visible spectrophotometer (Shimadzu UV-1800) against a blank, and the concentration of SOD was measured as unit/min/mg of protein [26].

Catalase (CAT) estimation: 0.1ml brain supernatant was combined with 0.1ml phosphate buffer (0.01M, pH 7.4) and 0.4ml distilled water. Then 0.5ml solution of 2M hydrogen peroxide was added to start the reaction. The potassium dichromate acetic acid reagent was later added in a volume of 2ml, and after 15 minutes in a boiling water bath, the reaction was allowed to cool. The green color solution was then measured at 570nm using a UV–visible spectrophotometer against a blank, and CAT concentration was expressed as µmol/mg protein [27].

Malondialdehyde (MDA) estimation: In this method, 0.1ml of the supernatant was mixed with 1.5ml of acetic acid (20%, pH 3.5), 1.5 ml of thiobarbituric acid (0.8%), and 0.2ml of sodium lauryl sulfate (8.1%). After that, the mixture was heated for 60 min. at 100°C and chilled with running water. After that 5ml of n-butanol-pyridine (15:1v/v) and 1ml of distilled water was added. It was shaken energetically for 10min, then centrifuged at 4000rpm. The pink color organic layer produced was measured at 532nm using spectrophotometer against a blank solution. The results were expressed as nmol/mg protein [28].

Measurement of reduced glutathione (GSH): The processed sample was mixed with an equal volume of 10% trichloroacetic acid. At 4°C, the sample was centrifuged for 10minutes at 2000rpm. 0.1ml supernatant, 2ml pH 8.4 buffer, 0.5ml 5-nitrobenzoic acid, and 0.4ml water was added to the mixture. At 412nm after 15minutes, the absorbance was then measured, and the results were expressed as µg/mg protein [29].

Measurement of nitric oxide (NO) content: The supernatant (1ml) was combined with equivalent measurements of Greiss reagent (0.1 % sulphanilamide in 2.5 % phosphoric acid and 0.1 percent N-(1-naphthyl) ethylenediaminedihydrochloride) and left at room temperature for 10min, and the absorbance was measured at 540nm against a blank sample using a spectrophotometer and NO content was expressed as µmol/mg protein [30].

Statistical analysis:

One-way ANOVA was used to examine the data, followed by Bonferroni's multiple comparison post hoc test. Data was denoted as mean ±SEM and statistical significance denoted as *, **, or *** for $p < 0.05$, 0.01 and 0.001, respectively. P values were shown with '#' representing $p < 0.05$ when compared with vehicle.

RESULTS

Effect of zonisamide and NS on the activity of SOD

The level of SOD was observed at 3, 6, 24, and 72h, and the results are described in figures 1(a), 1(b), 1(c), and 1(d), respectively. The level of SOD significantly improved zonisamide (100mg/kg) and NS (300mg/kg) compared to the vehicle-treated group at different time intervals. Still, the combination

showed a more significant amplification in the SOD level. Although, at 72h, the SOD level of the co-administered group showed no significant difference compared to the control group.

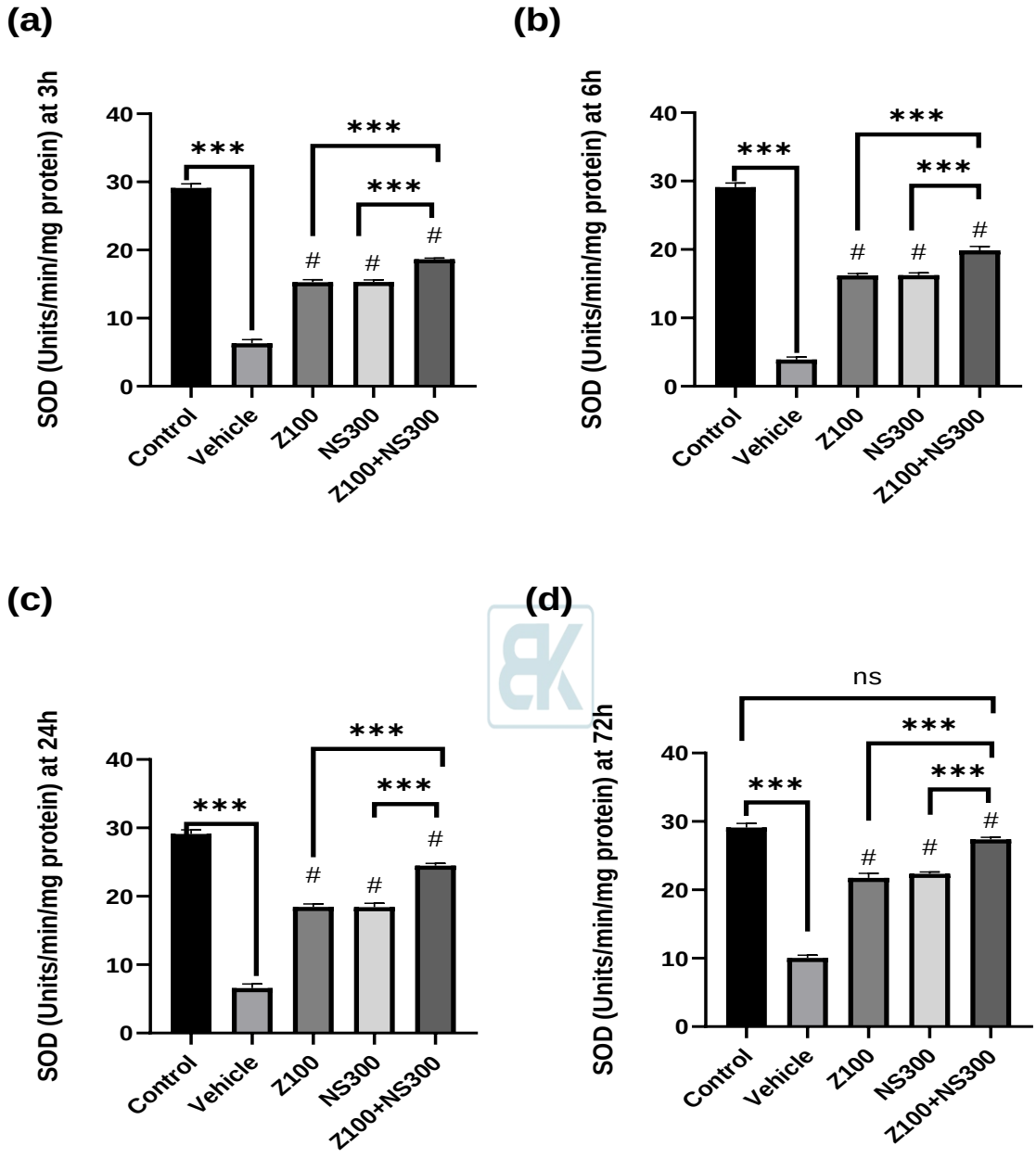

Figure 1: *Effect of zonisamide, NS and its combination on SOD levels at (a) 3h, (b) 6h, (c) 24h, (d) 72h in mice brain.*

Effect of zonisamide and NS on Catalase level The level of CAT was observed at 3,6,24 and 72h, and the result is described in figures 2(a), 2(b), 2(c), and 2(d), respectively. The level of CAT significantly improved in zonisamide (100mg/kg) and NS (300mg/kg) compared to the vehicle-treated group at different time intervals. Still, the combination

showed a more significant amplification in the CAT level. Although, at 72h, in compared to the control group, the co-administered group's CAT level exhibited no significant difference.

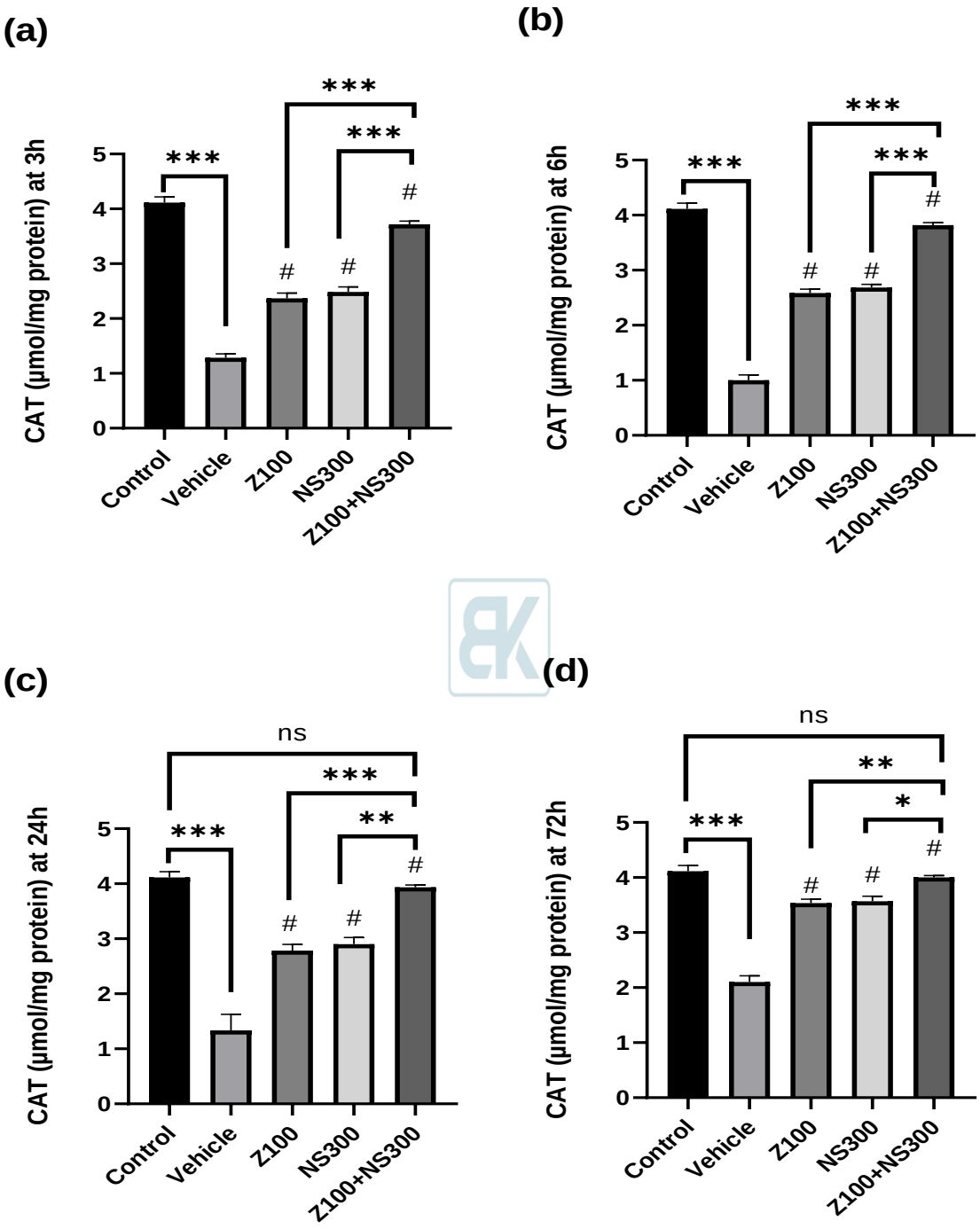

Figure 2: *Effect of zonisamide, NS and its combination on CAT levels at (a) 3h, (b) 6h, (c) 24h, (d) 72h in mice brain.*

Effect of zonisamide and NS on MDA level

The level of MDA was observed at 3,6,24 and 72h, and the result is described in figures 3(a), 3(b), 3(c), and 3(d), respectively. The MDA level was drastically increased in the vehicle-treated group. However, co-administration of both zonisamide (100mg/kg) and NS (300 mg/kg) showed a more considerable reduction in the MDA level than in vehicle-treated groups.

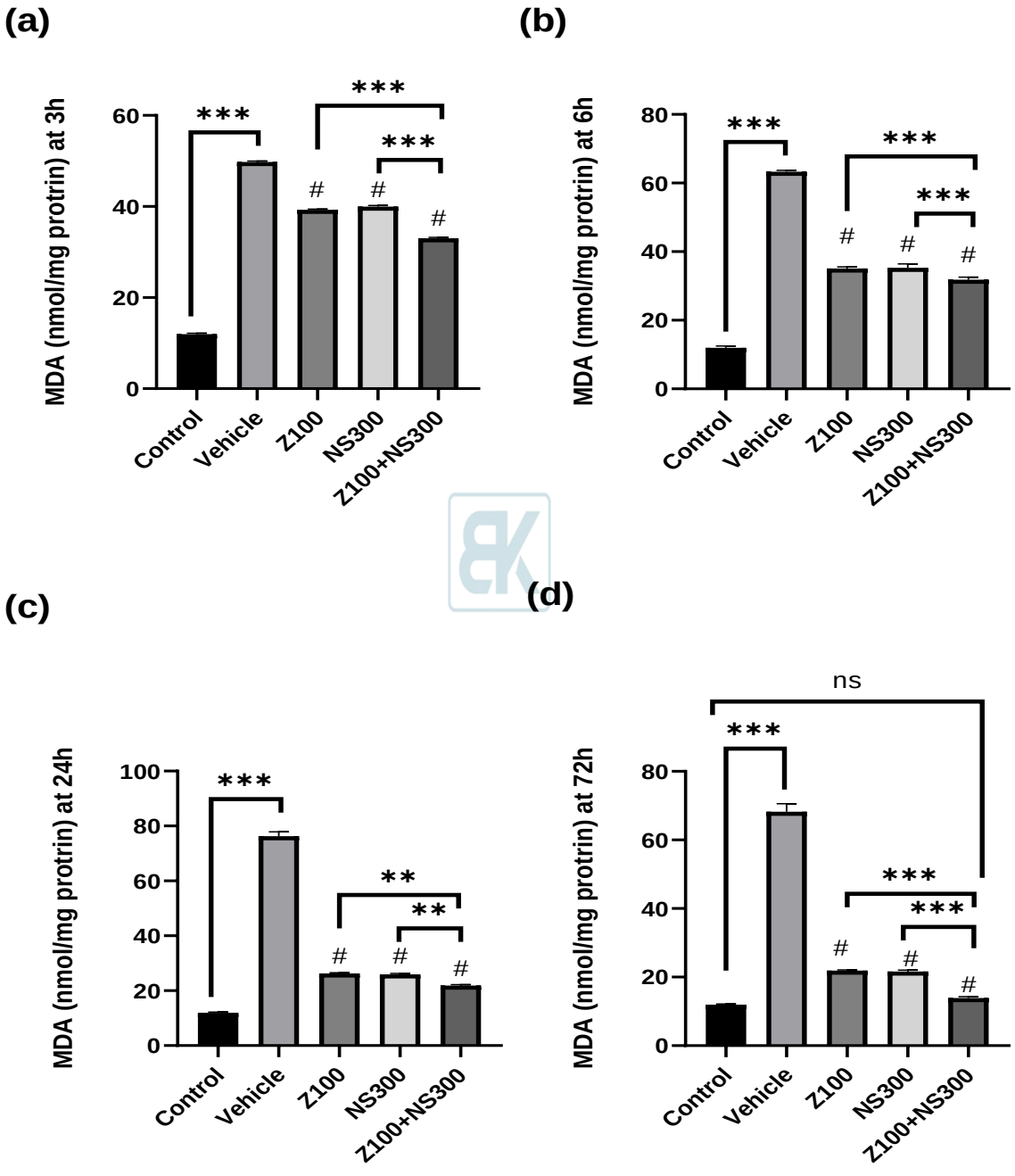

Figure 3: *Effect of zonisamide, NS and its combination on MDA levels at (a) 3h, (b) 6h, (c) 24h, (d) 72h in mice brain.*

Effect of zonisamide and NS on GSH level

The level of GSH was observed at 3,6,24 and 72h, and the result is described in figures 4(a), 4(b), 4(c), and 4(d), respectively. The GSH level significantly diminished in the vehicle compared to the control. On the other hand, the administration of zonisamide (100mg/kg) and NS (300mg/kg) extract per se remarkably increased the level of GSH. The combination exhibited a more significant improvement in the GSH level than the vehicle-treated group at all time intervals. Although, at 72h, the GSH level of the co-administered group showed no remarkable difference compared to the control group.

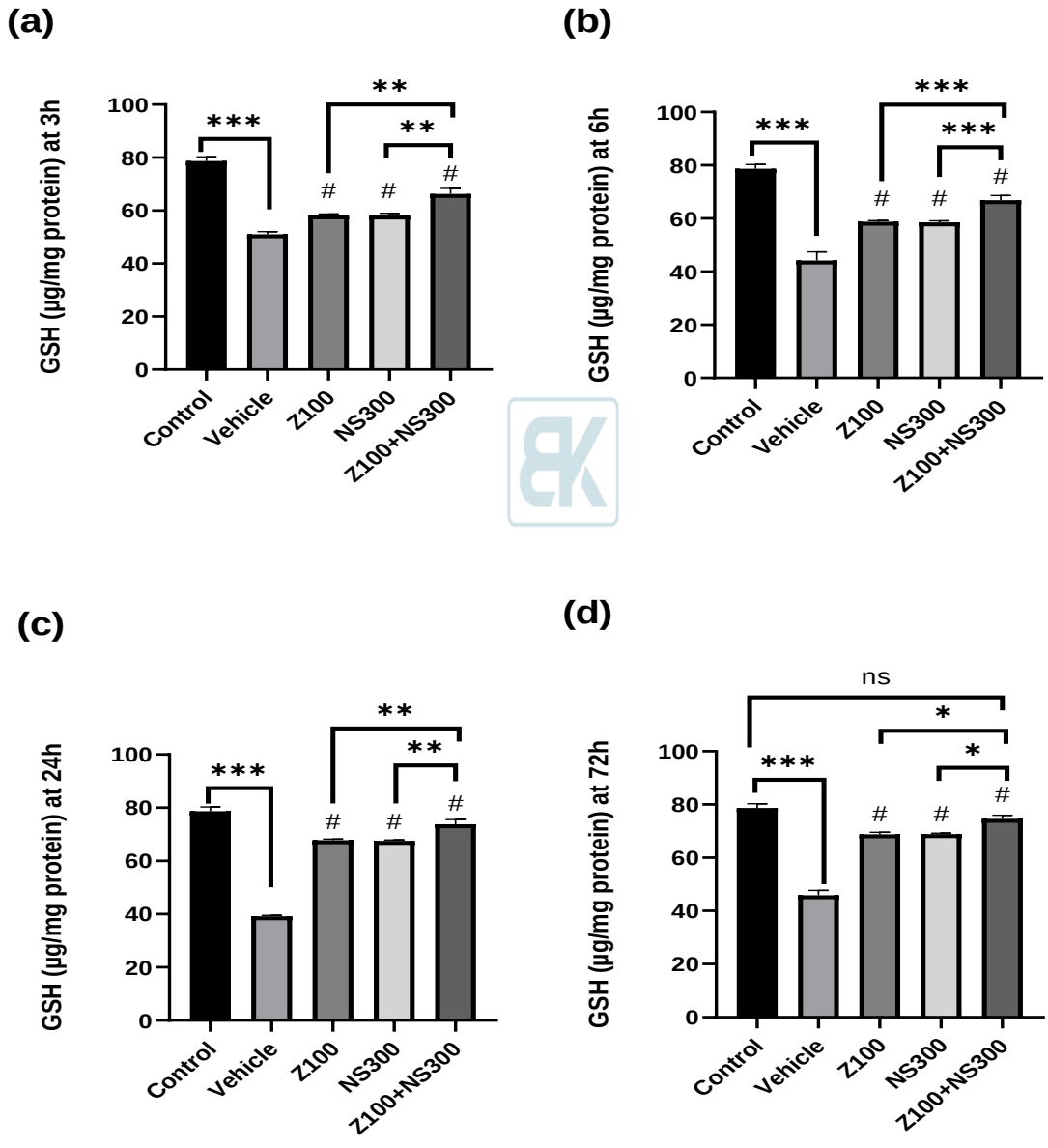

Figure 4: *Effect of zonisamide, NS and its combination on GSH levels at (a) 3h, (b) 6h, (c) 24h, (d) 72h in mice brain.*

Effect of zonisamide and NS on NO level

The level of NO was observed at 3,6,24 and 72h, and the result is described in Figures 5(a), 5(b), 5(c), and 5(d), respectively. The level of NO significantly elevated after the injury, but the treatment with zonisamide and NS extract significantly decreased its levels. However, the co-administration of both zonisamide (100mg/kg) and NS (300mg/kg) demonstrated a more considerable MDA reduction than vehicle-treated groups. Although, at 72h, the NO level of in comparison to the control group, the co-administered group showed no significant differences.

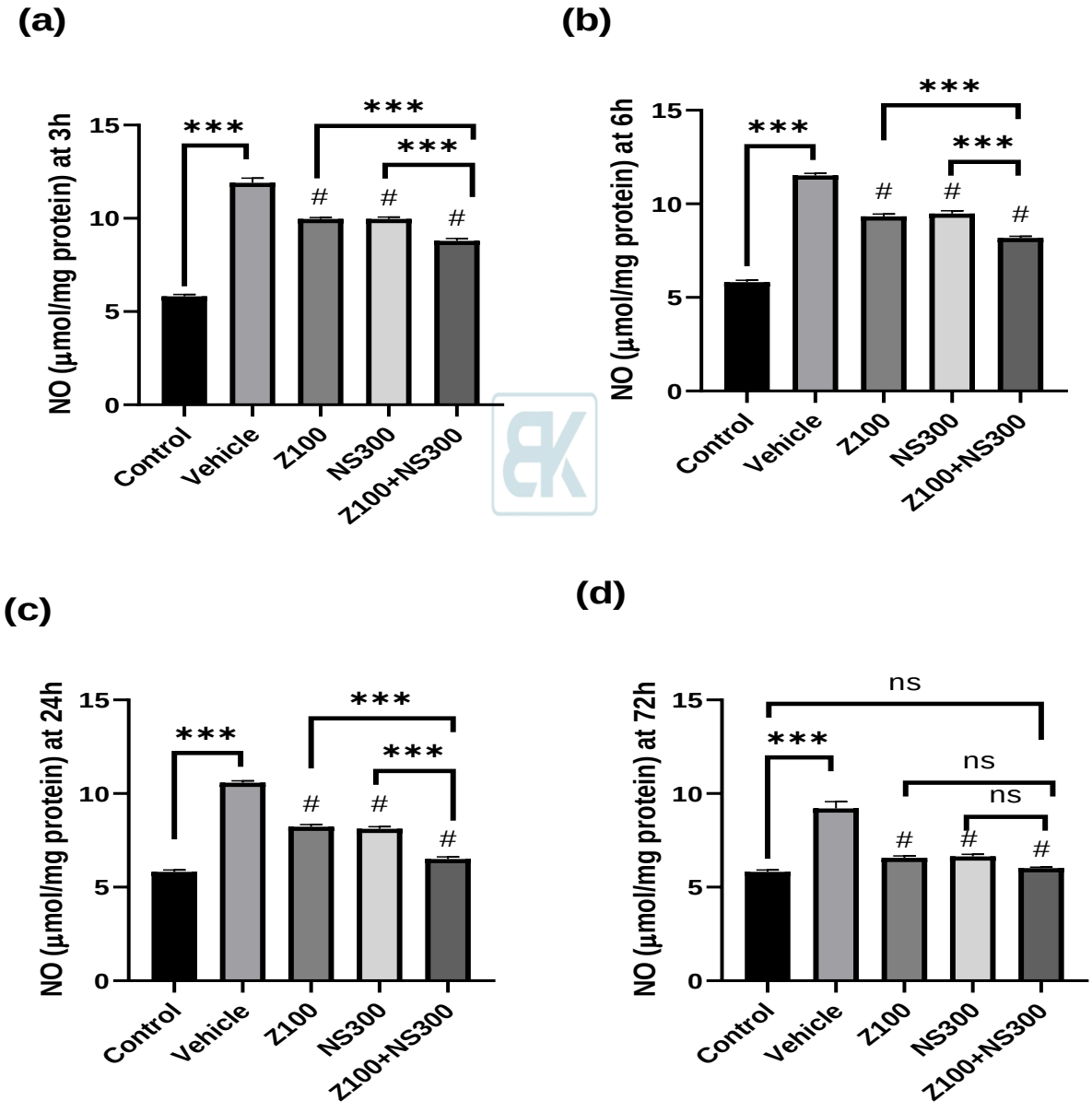

Figure 5: *Effect of zonisamide, NS and its combination on NO levels at (a) 3h, (b) 6h, (c) 24h, (d) 72h in mice brain.*

DISCUSSION

TBI is still a primary clinical and socioeconomic concern, with few therapeutic options tailored to its clinical and neurobiological trajectory. The pharmacological potential of zonisamide and NS alone and in combination with a weight drop model against TBI in mice was explored in the present experiment. The goal of anti-oxidant neuroprotective drug development for acute TBI has always been to stop the subsequent injury cascade by pharmacologically addressing the oxidative damage mechanism. Oxidative stress occurs when the scavenging anti-oxidant system is overwhelmed by oxygen levels and oxygen-derived free radicals. On the other hand, the neuronal loss after hours and days is driven by secondary injury from inflammation, excitotoxicity of neurotransmitters, cerebral ischemia, blood-brain barrier disruption, and neurological deficits [31, 32]. One of the essential aspects of TBI's pathophysiology is oxidative stress. Many studies confirm that traumatic brain injury directly amplifies oxidative stress [33]. In the current investigation, the vehicle-treated group displayed more oxidative stress. Intervention with zonisamide (100mg/kg) and NS (300mg/kg) per se and their combination eliminate that oxidative stress by augmenting the level of SOD, CAT, and GSH. Still, the levels of NO and MDA significantly decrease. Zonisamide and NS reduced oxidative stress, resulting in neuroprotection by preventing secondary injury. Additionally, neuroprotection is more significant in the co-administered group, which could be attributable to many targets in the pathophysiological cascade of secondary damage following TBI.

CONCLUSION

According to the above observation, it was concluded that co-administration of zonisamide and NS revealed neuroprotective effects in mice against TBI-induced neurodegeneration by reducing oxidative stress. These anti-oxidant and neuroprotective properties could aid in developing these medications as a therapy for neuronal death caused by traumatic injuries.

CONFLICT OF INTEREST

The author declares no conflict of interest.

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Biotechnology Advances around the World

Editor's Picks

Every issue of Biotechnology Kiosk presents select latest research news picked by the editors-in-chief on significant research breakthroughs in different areas of biotechnology around the world. The aim is to promote further R&D in all of these cutting-edge areas of biotechnology. The editors have compiled and included the following innovations and breakthroughs to highlight the latest biotechnology advances.

Dr. Megha Agrawal
Co Editor-in-Chief

Dr. Shyamasri Biswas
Co Editor-in-Chief

Fermented Food and Microbiome

A diet rich in fermented food can increase microbiome diversity and lower inflammation

Studies have suggested that diet can shape the gut microbiome that can positively impact the immune system and overall health. Especially, it has been suggested that microbiota-targeted diets can potentially change immune status that can provide a new therapeutic avenue for decreasing inflammation in healthy adults.

Recently researchers in the US conducted a clinical trial in 36 healthy adults. They were randomly assigned to a 10-week diet that included either fermented or high-fiber foods. Researchers obtained interesting results and the two different diets resulted in different effects on the gut microbiome and the immune system. These results were published in Cell (Gut-microbiota-targeted diets modulate human immune status. Cell, 2021; DOI: 10.1016/j.cell.2021.06.019).

Researchers found that eating foods such as yogurt, kefir, fermented cottage cheese, kimchi and other fermented vegetables, vegetable brine drinks, and kombucha tea led to an increase in overall microbial diversity, with stronger effects from larger servings. In their study, the researchers focused on fiber and fermented foods to study their potential health benefits. High-fiber diets were found to be associated with lower rates of mortality. And the consumption of fermented foods was found to be helpful in weight maintenance along with decrease the risk of diabetes, cancer and cardiovascular disease.

Analysis of blood and stool samples collected during a three-week pre-trial period was conducted that included the 10 weeks of the diet, and a four-week period after the diet when the participants ate as they chose.

The novel findings suggested influence of diet on gut microbes and immune status. These data showed increasing the consumption of fermented foods had similar effects on their microbiome diversity and inflammatory markers, consistent with prior research. This suggested that that short-term changes in diet can quickly alter the gut microbiome. However, the limited change in the microbiome within the high-fiber group indicated a general resilience of the human microbiome over short time periods.

Tumor Detection

Rapid detection of lung tumor

There are various types lung tumor such as small cell lung cancer, adenocarcinoma and squamous cell carcinoma. Accordingly, the prognosis and effective therapies differ based on the type of lung cancer. In addition, many rare tumor types and sub-types also exist. This diversity of tumor poses a serious challenge in developing reliable rapid diagnostic methods that can be employed in everyday clinical practice. In addition to histological typing, the tumor samples also need to be comprehensively examined for certain changes at a DNA level. Especially, detecting one of these mutations is key information that influences both the prognosis and subsequent therapeutic interventions.

Recently, a research team in Germany was able to reliably perform this determination in just one step using a combination of quantum cascade laser-based infrared microscopy and artificial intelligence. For the first time, they were able to identify spectral markers that allowed for a spatially resolved distinction between various molecular conditions in lung tumors.

They published their work in The American Journal of Pathology (Quantum Cascade Laser-Based Infrared Imaging as a Label-Free and Automated Approach to Determine Mutations in Lung Adenocarcinoma. The American Journal of Pathology, 2021; 191 (7): 1269 DOI: 10.1016/j.ajpath.2021.04.013). The researchers were able to verify the procedure on samples from over 200 lung cancer patients in their work. They showed that the most common genetic mutations can be determined with a sensitivity and specificity of 95 per cent compared to laborious genetic analysis with the developed detection method.

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