ORIGINAL ARTICLE

Ischemia/reperfusion in rat: Antioxidative effects of enoant on EEG, oxidative stress and inflammation

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Abstract
Primary objective: The present study was undertaken to evaluate whether enoant, which is rich in polyphenols, has any effect on electroencephalogram (EEG), oxidative stress and inflammation in ischemia/reperfusion (I/R) injury.

Methods: Ischemia was induced by 2-hour occlusion of bilateral common carotid artery. Animals orally received enoant. Group 1 was the ischemic control group. Group 2 was treated with enoant of 1.25 g kg⁻¹ per day for 15 days after I/R. Group 3 received the same concentration of enoant as in group 2 for 15 days before and after I/R. Group 4 was the sham operation group. EEG activities were recorded and the levels of TNF-α, IL-1β and IL-6, TBARS and GSH were measured in the whole brain homogenate.

Results: There were significant changes in EEG activity in groups treated with enoant either before or after ischemia when compared with their basal EEG values. TNF-α, IL-6 and IL-1β levels were significantly increased after I/R. GSH levels in group 3 treated with enoant in both pre- and post-ischemic periods were significantly increased and TBARS concentration was decreased compared with the ischemic group.

Conclusion: The findings support that both pre-ischemic and post-ischemic administrations of enoant might produce neuroprotective action against cerebral ischemia.

Keywords: EEG, ischemia/reperfusion, oxidative stress, inflammation, polyphenols

Abbreviations: EEG, electroencephalogram; GSH, glutathione; IL-1β, interleukin 1 beta; IL-6, interleukin 6; I/R, ischemia-reperfusion; ROS, Reactive oxygen species; TBARS, thiobarbituric reactive substance; TNF-α, tumour necrosis factor alpha.

Introduction

Reactive oxygen species (ROS) are important mediators of a variety of pathological processes, including oxidative stress and inflammation in ischemia/reperfusion (I/R) [1]. There are a number of potential sources for free radicals generation in ischemic brains [2]. These free radicals leak from mitochondrial respiratory chain; sequences catalysed by cyclooxygenase and lipoxygenase, peroxidation of lipid membrane, auto-oxidation of various small molecules, including catecholamine, by the microsomal cytochrome p450 reductase system and xanthine oxidase reaction [3]. Naturally, the brain is highly susceptible to oxidative stress because of its structural specificity. Although the brain consists of 2% of total mass of body, it consumes 20% of the total oxygen used by it. Since the brain is rich in polyunsaturated lipids and pro-oxidative metals and has very low antioxidant capacity [4], it is vulnerable to I/R-induced ROS which promote damage to lipids, DNA, carbohydrates and proteins and induce production of several inflammatory proteins which contribute to the process of neuronal death [5]. Thiobarbituric reactive substance (TBARS) is
the most frequently studied marker of oxidative tissue damage during I/R.

Recent studies of the mechanisms underlying neuronal death caused by cerebral ischemia have indicated the importance of immune/inflammatory cytokines [6, 7]. Injury to the central nervous system by ischemia elicits an inflammatory response involving several cytokines including tumour necrosis factor alpha (TNF-α), interleukin (IL)-1β and interleukin 6 (IL-6).

Oxidative stress is a function of balance between pro-oxidants such as ROS and antioxidants scavenging them [8]. Antioxidants have been evaluated as neuroprotective agents and are able to reduce cerebral damage in I/R [9, 10]. Polyphenolic compounds such as resveratrol, quercetin and catechin have been recently shown to have neuroprotective activity against oxidative stress [11]. Enoant contains grape polyphenoles both from seeds and skin in biologically accessible form.

The development of experimental models of cerebral ischemia has allowed for a better knowledge of its pathophysiology and for testing new therapeutic strategies. In this direction, diverse rodent models of brain ischemia/reperfusion have been proposed. The two-vessel occlusion model is easier to perform; the less-invasive surgical intervention allows greater scope for recovery experiments and widely used to investigate the effectiveness of potential therapeutic agents [12, 13].

In previous studies, it was shown that electroencephalogram (EEG) could be a suitable neurophysiologic testing tool in the model of global ischemia [14]. EEG permits prognostic information by monitoring the effects of pharmacological treatments that begun immediately after the onset of cerebral ischemia. Despite high technology instrumentation including positron emission tomography and functional magnetic resonance imaging used to clarify the complexity of cerebral blood flow and metabolism, the EEG retains a useful place in the evaluation of processes induced by cerebral ischemia, especially in experimental conditions [15]. In the light of these findings, the present study was undertaken to evaluate whether enoant, which is rich in polyphenols, has any effect on EEG, oxidative stress and inflammation in I/R injury.

Materials and methods

Experimental design

Male Wistar albino rats weighing 200–250 grams were used in this study. Animals were housed 3–4 per laboratory cage and maintained on a 12-hour light–dark cycle, with ad libitum access to water and standard dry rat diet. They were housed under standard laboratory conditions at least 1 week prior to the experiment. All animal studies carefully conformed to the guidelines outlined in Interdisciplinary Principles and Guidelines for the Use of Animals in Research and Education from the New York Academy of Sciences.

Electrode implantation

Animals \((n = 6)\) were anaesthetized with thiopental (50 mg kg\(^{-1}\), i.p.) and positioned in a stereotaxic apparatus. The EEG electrodes were constructed from insulated stainless steel wire (200 μm diameter) with insulation removed at the end to form the contact. Sixteen holes were drilled through the skull and electrodes were stereotaxically placed (Figure 1) at FP1-2, F1-4, C1-4, P1-4 and O1-2 locations referenced to an electrode implanted over the cerebellum. Electrodes were fixed to the skull with dental acrylic cement.

Administration of enoant

Rats were randomly divided into four groups: Group 1 \((n = 10)\), group 2 \((n = 11)\) and group 3 \((n = 10)\) and group 4 \((n = 9)\). Animals received enoant orally in fresh drinking water. The concentrations of enoant consumed each day by animals were determined. Group 1 was the ischemic control.
group treated only with water. Group 2 was treated with enoant of 1.25 g kg⁻¹ per day for 15 days after I/R. Group 3 received the same concentration of enoant as in group 2 for 15 days before and after I/R. Group 4 was the sham operation group.

Bilateral common carotid artery occlusion

Under thiopental anaesthesia, the two common carotid arteries were carefully isolated from the vagosympathetic trunks. Ischemia was induced by occlusion of bilateral common carotid artery in all animals. After 2 hours occlusion, reperfusion was allowed. After EEG recordings were taken, bilateral carotid arteries had been occluded. At the end of the 2-hour occlusion if 10–15% reduction was seen in amplitude of EEG frequency as compared with baseline EEG, it was thought that ischemia was induced and then the animals were used for experiment.

Recordings of EEG

Eight days after operation, the experiment was performed in freely moving rats that lived in a Plexiglas recording cage. Animals in the recording cage were placed in a Faraday cage and continuous EEG recordings were taken for 10 minutes with Neuroscan (SynAmps Model 5083, Charlotte, NC, USA) from all groups. The behaviour of the animals during the EEG recording session was observed. For group 1, basal EEG and EEG on the 15th day after I/R were recorded. For group 2, basal EEG and EEG recordings on the 15th day after enoant treatment were taken. For group 3, basal EEG, EEG after enoant treatment for 15 days just before I/R and EEG after enoant treatment for 15 days following I/R were recorded (Figure 2). EEG signals were recorded with a band pass of 0.3–70 Hz digitally with a sampling frequency of 1000 Hz. The frequency band of 0.5–4 Hz was expressed as delta, 4–8 Hz as theta, 8–11 Hz as alpha1, 11–14 Hz as alpha2, 14–25 Hz as beta1, 25–35 Hz as beta2 and 35–45 Hz as gamma. The respective grand averages of amplitude scores from all of the frequency bands were used to generate a topographic map using the Neuroscan EEG analysis software in all groups.

Preparation of brain tissue

The rats anaesthetized intraperitoneally with 50 mg kg⁻¹ of thiopental were transcardially perfused with heparinized 0.05 mol L⁻¹ phosphate-buffered saline and then sacrificed after 15 days of I/R. The normal control group was only used for histological examination. The brain was quickly removed and the hippocampus region was fixed in 4% paraformaldehyde at 4°C for histological examination. The remaining brain sample was stored at −80°C for biochemical analysis.

Verification of ischemia by histological examination

The hippocampus within fixative solution was embedded in paraffin for sectioning. Paraffin sections (5 μm) of tissues were taken using a microtome (Leica MR 2145, Heerbrugg, Switzerland). These sections were stained with haematoxylin and eosin for microscopic examination. Pyramidal neurons in the CA1 region were counted in four fields of each slide (eight slides).

Biochemical analysis

The levels of TNF-α, IL-1β and IL-6 were measured in whole brain homogenate by using ELISA kit according to the manufacturer’s instruction (Biosource Brand of Invitrogen, Paisley, UK). The concentrations of TNF-α, IL-1β and IL-6 were expressed as ng mg⁻¹ protein. Reduced Glutathione (GSH) was measured according to Beutler et al. [16], briefly whole brain tissue was homogenated in potassium chloride buffer and mixed with protein precipitation solution. After centrifugation, DTNB (Ellman’s reagent) and phosphate buffer were added to the removed supernatant. Optical density of yellow coloured complex was measured at 412 nm. TBARS of the whole brain levels were estimated according to Ohkawa et al. [17]; TBARS was used as an indicator of lipid peroxidation. Whole brain tissue homogenate was prepared in potassium phosphate buffer and then thiobarbituric acid and trichloroacetic acid added to the homogenate. After the mixture was boiled, the TBARS were extracted using n-butanol/pyridine (15 : 1 v/v). The absorbance of organic phase was measured at a wavelength of 535 nm. TBARS concentrations were determined using a standard curve generated from the hydrolysis of 1,1,3,3-tetraethoxypropane. The protein concentration of homogenate was estimated by the method of Bradford [18], using bovine serum albumin as the standard. Concentration of reduced GSH and TBARS levels were given as nmol mg⁻¹ protein.

Chemicals

Enoant was kindly supplied by Te-ha Cosmetic Company (Istanbul, Turkey). The content of polyphenols in enoant, extracted from skin and seeds of grape, was determined by HPLC as 1.47 mg ml⁻¹ catechin, 0.88 mg ml⁻¹ epicatechin, 130 μg ml⁻¹ quercetin and 23 μg ml⁻¹ resveratrol. Thiopental was obtained from Sigma (St. Louis, MO, USA). Thiopental was dissolved in saline and given intraperitoneally in a volume of 2 ml kg⁻¹. Potassium chloride (KCl, Merck, Germany), sodium chloride...
(NaCl, Merck), m-phosphoric acid (m-H₃PO₄, Merck), Ethylene diamine tetra acetic acid (EDTA, Merck), Trichloroacetic acid (TCA, Merck), n-butanol (Sigma, USA), 2-thiobarbituric acid (TBA, Sigma), 5,5'-dithio-bis (2-nitro benzoic acid, Sigma) (Ellman’s reagent), pyridine (Sigma), 1,1,3,3-tetraethoxypropane (Sigma), reduced GSH (GSH, Sigma), disodium hydrogen phosphate (Na₂HPO₄, Merck), potassium phosphate buffer (KH₂PO₄, K₂HPO₄, Merck) and Bovine serum albumin (Sigma).

**Statistical analysis**

Continuous EEG recordings were divided into 2-second epochs and epochs with movement artifacts (i.e. exceeding 500 µV) were extracted automatically. Then epochs were analysed by averaged
frequency spectra. The part from 0–48 Hz was normalized (was equalized to 100). Moving averages were calculated for five points. A repeated measures ANOVA followed by a series of point-to-point two-tail paired Student’s \( t \)-test was applied for each channel for the basal values and for the values that were recorded on the 15th day after ischemia. Kruskal Wallis and Mann-Whitney U-test were used to compare biochemical results. Histological analysis was carried out by one-way ANOVA. Data were expressed as mean ± SD. \( p < 0.05 \) was accepted to be statistically significant for all results.

Results

EEG results

The distributions of percentage ratios of delta, theta, alpha1, alpha2, beta1 and beta2 bands of basal EEGs were similar among all groups.

Figure 3 presents the topographic maps of the six EEG frequency bands from basal to 15 days post-I/R in group 1. The basal maps of group 1 are directly compared with the maps obtained on the 15th day after I/R. In group 1, delta, theta and alpha1 bands on the 15th day after I/R were decreased by 20%, 18.9% and 15.9%, respectively, compared with their basal values. In contrast, in the frequency spectra on 15 days after I/R, alpha2, beta1 and beta2 bands were increased by 14%, 34.7% and 42.6%, respectively, compared to the basal values.

Figure 4 shows the EEG recordings of group 2. In this group, delta band on the 15th day after I/R was decreased by 22.2%, whereas theta, alpha1, alpha2, beta1 and beta2 bands were increased by 7.9%, 10%, 18.9%, 18% and 20.6%, respectively, compared with their basal values.

Figure 5 presents the EEG findings of group 3. Delta band on the 15th day after enoant treatment was decreased by 28.8%, while theta, alpha1, alpha2, beta1 and beta2 bands were increased by 7.6%, 11%, 20%, 23% and 27.9%, respectively, compared to basal values. On the other hand, no differences were observed in the spectral power between pre- and post-I/R enoant treatments.

Regarding the frequency spectral analysis, all groups showed statistically significant differences with respect to each other group \((F(35, 90) = 1.824, p < 0.012 \) by RM-ANOVA).
Figure 6 presents the EEG findings in group 1. In order to show clearly the changes between basal and post-I/R data, differenced moving average amplitude spectra, i.e. post-I/R-basal, were used. While I/R induced reduction in the amplitudes of delta, theta and alpha1 frequency were observed in almost all localizations, the amplitudes of alpha2 frequencies were significantly increased only in FP1 and O2 localizations. The amplitudes of beta and gamma frequencies were significantly increased in FP1-2, F1-4, C1, P1, P4, O1 and O2 localizations and FP1, F2, F4, C1, P3, and O1 localizations, respectively.

Figure 7 demonstrates the EEG results of group 2. In animals treated with enoant after I/R, the amplitudes of delta frequency were significantly decreased almost in all localizations, while the amplitudes of alpha1 and alpha2 frequency bands were significantly increased in FP1-2, F1-4, C1, P1, P4, O1 and O2 localizations and FP1, F2, F4, C1, P3, and O1 localizations, respectively.

Figure 8 summarizes the EEG findings of group 3. In animals treated with enoant pre- and post-I/R, moving averaged amplitude spectra of pre-I/R values was compared with basal value. In the results of pre-I/R enoant treatment, the amplitudes of delta and theta frequencies were decreased almost in all localizations, while the amplitudes of alpha1 and alpha2 frequency bands were significantly increased in FP1-2, F1-4, C2-4, P1-2, P4, O1 and O2 localizations. The amplitudes of beta and gamma frequencies were significantly increased in FP1-2, F2, F4, C2-4, P1-2, and P4, O1 and O2 and in F2, C2-4, P1-4 and O2 localizations, respectively.

Figure 9 presents the EEG findings in group 3. In animals treated with enoant in pre- and post-I/R, the amplitudes of delta frequencies were significantly increased in FP1-2, F2-3, C1-3, P1-2 and P3 localizations. In contrast, the amplitudes of theta, alpha and both beta and gamma frequencies were significantly decreased in C2, P1 and P3 localizations and only in F2 localization, respectively.

**Evaluation of ischemic damage**

Ischemic damage was verified by histological examination although it was proved by EEG results.
Neuronal loss was evaluated by quantitative assessment of neuronal counts. Quantitative analysis was performed on histological sections stained with haematoxylin and eosin by the observer who was unaware of the identity of samples. The numbers of stained neurons in pyramidal CA1 sub-field area of brains were counted. The numbers of neurons counted in the ischemic group were expressed as the percentage of cells as compared with the control group. Quantitative analysis revealed that pyramidal CA1 neurons in the hippocampus of the ischemic group were significantly reduced as compared with control (22%, \( p < 0.01 \)) (Figure 10).

**Biochemical analysis**

Biochemical results are shown in Table I. The levels of TNF-\( \alpha \) and IL-6 in group 1 were significantly
increased when compared with group 2 and group 3 treated with enoant. The levels of TNF-α, IL-6 and IL-1β in groups 1, group 2 and group 3 were significantly increased as compared with group 4. GSH and TBARS were measured as markers of oxidant and antioxidant, respectively. GSH levels in group 3 were significantly increased when compared with group 1 and group 2. In contrast, TBARS concentrations were diminished in group 3 when compared with either group 1 or group 2. TBARS levels in group 2 were significantly higher than group 4 \((p < 0.05)\).

**Discussion**

EEG activity was monitored to evaluate the cerebral I/R damage resulting from oxidative stress and inflammation and also to test the neuroprotective effect of enoant on the brain. We measured TNF-α, IL-1β and IL-6 and GSH and TBARS as markers of inflammation and oxidative stress, respectively. To our knowledge, there is no report that describes the therapeutic effects of enoant, polyphenol compound, on oxidative damage, the inflammatory response and EEG recording in I/R insult. The change in the amplitude and frequencies of EEG activity during I/R is a sign for the presence of pathology [19–23]. In the present study, I/R induced decreases in delta, theta and alpha1 activities and increases in alpha2 and beta activities compared with their basal values. Enoant treatment after I/R prevented the decreases in theta and alpha1 resulting from I/R injury. Although enoant given before I/R had significant changes in EEG activity compared with basal, no significant changes were seen in the EEG activity between pre- and post-I/R enoant treatments in group 3. In studies concerning I/R, increases in delta and theta activities and reduction in beta activity were associated with the extent of I/R damage. In addition, in many studies, increased delta activity associated with brain damage was also indicated in adults [24].

The previous studies have been characterized by the slow-wave areas, especially with delta and lesser with theta activities in the ischemic areas. Recently, it was shown that delta and theta activities were increased with ischemic infarct size [25]. Mariucci
et al. [14] showed that ischemia produced a dramatic increase in delta activity and decrease in theta, beta and alpha activities derived from cortical areas and that EEG activity reverted to normal values after 6 days of reperfusion. There is no consistency between these data. The differences between those results might depend on the electrode localization, ischemia duration and time of data recording or species of animal used. In the literature, the authors could not find any data concerning EEG recordings 15 days following I/R. In group 1 and group 2, the delta activity was decreased but beta activity increased on the 15th day recording following I/R. If the EEG had been recorded on day 6 following I/R, this study would have obtained similar results to Mariucci et al. [14]. One can speculate that delta activity might increase and beta decrease in the first few days and these changes might return to normal on the 6th day, but delta continues to decrease and beta increases along 15 days. On the other hand, enoant given before I/R decreased delta activity and increased beta activity on the 15th day and those effects did not change on the 15th day with enoant treatment after I/R. The decreases in group 3 can be attributed to the pre-treatment of enoant in I/R. In other words, enoant given after I/R does not seem to be as effective as enoant given before I/R.

The biochemical results are consistent with EEG recordings. Several studies have indicated that TNF-α, together with other pro-inflammatory cytokines such as IL-1β and IL-6 may play a role in the development of central nervous system injury following I/R [26–28]. The levels of TNF-α, IL-6 and IL-1β were increased after I/R, but these values decreased with the treatment of enoant. Experimental studies demonstrated that TNF-α and IL-6 have detrimental effects on cerebral ischemia [29]. In the current study, inflammation might have an effect on the EEG activity. In animals treated with enoant in the pre-ischemic period, the amplitudes of slow-waves were decreased and the amplitudes of fast waves were increased, suggesting that enoant might improve metabolism of neuron. The changes seen in EEG findings after I/R might result from the elevated levels of TNF-α and IL-6. The mechanisms by which TNF-α could potentiate ischemic injury are probably multiple, since TNF-α increases capillary permeability [30], up-regulates expression of adhesion molecules [31], increases leukocytes adhesion to endothelium and their
leakage through blood vessel walls [30] and induces synthesis of prostaglandins [32]. IL-6 is a pivotal regulator of inflammatory and immune response [33]. IL-6 increases endothelial permeability by rearranging actin filaments [34]. Over-expression of IL-6 in astrocytes breaks down the blood–brain barrier in transgenic mice [35].

It was found that TBARS was markedly increased, while GSH, non-enzymatic scavenger, decreased following I/R, but this situation was reversed with enoant treatment. Lipid peroxidation is associated with progressive loss in membrane fluidity, reduction in membrane potential, increase in membrane permeability to ions and finally cell death [36]. In contrast, GSH is an essential tripeptide and endogenous antioxidant found in all animal cells. It reacts with free radicals and can protect cells from singlet oxygen, hydroxide radical and superoxide damage [37].

Some studies showed beneficial effects of antioxidants on I/R insult [38, 39]. In the present study, as seen in EEG, GSH and TBARS findings, oxidative stress were improved with treatment of enoant, but this improvement was more pronounced in group 3 treated with enoant in both pre- and post-I/R. When ginkgo biloba extract, a free radical scavenger, was administrated for 10 minutes, 15 minutes and 5 days before and 5 days after cerebral ischemia-reperfusion, the percentage of slow waves after reperfusion was decreased in the Ginkgo biloba group compared with the control group [40]. The results are in agreement with that study. In a previous study, resveratrol, an antioxidant compound found in enoant, was shown to reduce focal cerebral ischemic area. This effect of neuroprotection was reported depending on its anti-platelet aggregation, vasodilating and antioxidant effects [41]. This effect was reported depending on its anti-platelet aggregation, vasodilating and antioxidant effects. In addition, it was also reported that resveratrol provides neuroprotective effect by inhibiting K ion channels [42]. Quercetin, which is another antioxidant found in enoant, provides neuroprotective effects by inhibiting caspase activity [43].

A growing body of data indicates that polyphenols are the active ingredients in dietary plants and traditional medicines for treatment of disorders related to oxidative stress and inflammation [44–46]. The data have also suggested that...
treatment with enoant in the pre-ischemic period is an important factor to diminish I/R damage. Compounds such as resveratrol, quercetin and catechin enriched in vegetables, fruits, wine and tea have been shown to offer antioxidative and anti-inflammatory effects by inhibiting NF-kB [47]. NF-kB is a common regulator element in the promoter region of many pro-inflammatory cytokines [48]. In activated macrophages, NF-kB in synergy with other transcriptional activators plays a central role in coordinating the expression of genes encoding TNF-α, IL-1β and IL-6 levels [49]. Resveratrol has been shown to inhibit the release of TNF-α, IL-1β and IL-6 by lipopolysaccharide-activated mice monocytes [50, 51]. Activation of microglia is associated with increased phagocytosis and release of oxygen radicals, NO, proteases as well as pro-inflammatory cytokines. Prolonged and excessive stimulation of microglia initiates an inflammatory cascade in cerebral neuronal system that contributes pathogenesis of several neurodegenerative diseases [52, 53]. It is believed that agents that suppress microglial cell activation are beneficial for treatment of neurodegenerative diseases [51]. The beneficial effects of resveratrol, quercetin and catechin have separately been shown in numerous diseases, including cancer, stroke and cardiovascular diseases in vivo and in vitro studies [54–56].

Conclusions

This study showed that TNF-α, IL-6 and IL-1β pro-inflammatory cytokines correlate with the oxidative stress. EEG findings were consistent with biochemical results. Enoant has a neuroprotective effect on cerebral ischemia. The neuroprotective action of enoant could be mediated separately or synergistically through the following mechanisms: (1) Enoant might reduce the release of pro-inflammatory cytokines after I/R by inhibiting NF-kB activation; (2) Enoant may act as a free radical scavenger; (3) Enoant may improve GSH; and (4) Enoant may reduce generation of excitatory amino acids such as glutamate and aspartate.

In conclusion, the results reported here support the idea that pre-ischemic administration of enoant might produce a neuroprotective action against cerebral ischemia.
Acknowledgements

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Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Table I. Concentrations of TNF-α, IL-1β, IL-6, GSH and TBARS in Wistar albino rats.

<table>
<thead>
<tr>
<th>Groups [n]</th>
<th>TNF-α, ng mg⁻¹ (mean ± SD)</th>
<th>IL-1β, ng mg⁻¹ (mean ± SD)</th>
<th>IL-6, ng mg⁻¹ (mean ± SD)</th>
<th>GSH, nmol mg⁻¹ (mean ± SD)</th>
<th>TBARS, nmol mg⁻¹ (mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 [10]</td>
<td>0.32 ± 0.03</td>
<td>0.44 ± 0.08</td>
<td>0.51 ± 0.07</td>
<td>1.65 ± 0.51</td>
<td>22.0 ± 4.47</td>
</tr>
<tr>
<td>Group 2 [11]</td>
<td>0.24 ± 0.10ᵃ</td>
<td>0.37 ± 0.22</td>
<td>0.41 ± 0.07ᵃ</td>
<td>1.68 ± 0.39</td>
<td>25.3 ± 7.08</td>
</tr>
<tr>
<td>Group 3 [10]</td>
<td>0.22 ± 0.04ᶜ</td>
<td>0.46 ± 0.21</td>
<td>0.41 ± 0.05ᵇ</td>
<td>2.27 ± 0.46ᵇᵈ</td>
<td>18.2 ± 2.83ᵇᵈ</td>
</tr>
<tr>
<td>Group 4 [9]</td>
<td>0.12 ± 0.04⁵ᶜᵈᶠ</td>
<td>0.19 ± 0.035ᶜᵈᶠ</td>
<td>0.21 ± 0.11ᶜᵈᶠ</td>
<td>1.81 ± 0.39</td>
<td>19.9 ± 2.64ᶜ</td>
</tr>
</tbody>
</table>

[ⁿ] number of animals.
ᵃ p < 0.05, ᵇ p < 0.01, ᵇᵇ p < 0.001 compared to group 1; ᵇᵉ p < 0.01, ᵇᵉᵇ p < 0.05 compared to group 2; ᵇᵉᵖ < 0.001 compared to group 3.

References


