Effect of dietary sesame oil as antioxidant on brain hippocampus of rat in focal cerebral ischemia

Saif Ahmad a,⁎, Seema Yousuf a, Tauheed Ishrat a, M. Badruzzaman Khan a, Kanchan Bhatia b, Inayat Salem Fazli c, Jafar Salamat Khan d, Naseem Hasan Ansari e, Fakhrul Islam a,⁎

⁎ Corresponding authors. Tel.: +91 11 26059688; fax: +91 11 26059663.
E-mail addresses: saif_hamdard@yahoo.com (S. Ahmad), fislam2001@yahoo.co.in (F. Islam).

Neurotoxicology Laboratory, Department of Medical Elementology and Toxicology, Faculty of Science, Jamia Hamdard (Hamdard University), New Delhi 110 062, India
Department of Medical Elementology and Toxicology, Faculty of Science, Jamia Hamdard (Hamdard University), New Delhi 110 062, India
Department of Pediatrics, Baylor College of Medicine, Houston, Texas, USA
Council of Scientific and Industrial Research (CSIR), Ministry of Science and Technology, Govt. of India, Anusandhan Bhawan, Rafi Marg, New Delhi 110 001, India
Department of Biochemistry and Molecular Biology, University of Texas Medical Branch, Galveston, Texas 77555–0647, USA

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Abstract

Oxidative stress may be regarded as an imbalance between free radical production and opposing antioxidant defenses. Free radical oxidative stress is implicated in rat cerebral ischemia and naturaceutical antioxidants are dietary supplements that have been reported to have neuroprotective activity. Many studies have reported dietary sesame oil (SO) as an effective antioxidant. In the present study the neuroprotective effect of dietary SO was evaluated against middle cerebral artery occlusion (MCAO)-induced cerebral ischemia injury in rats. Rats were fed on diet (20% SO) for 15 days. The middle cerebral artery of adult male Wistar rat was occluded for 2 h and reperfused for 22 h. The antioxidant properties of brain were measured as levels of reduced glutathione (GSH), glutathione-S-transferase (GST), glutathione peroxide (GPx), glutathione reductase (GR), catalase (CAT), superoxide dismutase (SOD) and thiobarbituric acid reactive substance (TBARS). A decrease in the activity of all the enzymatic and non-enzymatic antioxidants was observed along with an increase in lipid peroxidation (LPO) in MCAO group. The neurobehavioral activity of rats was also observed by using videopath analyzer. Dietary SO improved the antioxidant status in MCAO + SO group when compared with MCAO group. The results of neurobehavioral activity also support our biochemical data. The results obtained suggest protective effect of SO against cerebral ischemia in rat brain through their antioxidant properties.
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Keywords: Dietary sesame oil (SO); Cerebral ischemia; Hippocampus; Oxidative stress; Antioxidant; Neurobehavioral activity

Introduction

In middle cerebral artery occlusion (MCAO)-induced cerebral ischemia, various biochemical events occur that cause intracellular calcium accumulation, depolarization, excessive release of excitatory amino acids, especially glutamate and inhibition of protein synthesis (Mies et al., 1993; Hossman, 1994). Cerebral ischemia is one of the leading causes for several neurological deficit and death (Chagnac-Amitai and Connors, 1989) and the causative mechanism suggested explaining this phenomenon is the involvement of reactive oxygen species (ROS) and oxidative stress (Traystman et al., 1991; Wang and Lo, 2003; Blomgren et al., 2003; Paradis et al., 2004). According to the literature, brain is very vulnerable to oxidative stress due to its high polyunsaturated fatty acids (PUFAs) content which are particularly susceptible to ROS damage (Cui et al., 2004; Halliwell, 2001). During ischemia, xanthine dehydrogenase undergoes irreversible proteolytic conversion to xanthine oxidase, producing superoxide and hydrogen peroxide in the presence of oxygen. Among the various free radicals produced, superoxide and hydroxyl radicals are potent in causing damage of cell membrane by inducing lipid peroxidation (Bromont et al., 1989). Although NO (nitric oxide) functions as an
important neural messenger in the normal brain (Dawson et al.,
1992) however, on reperfusion of the ischemic brain, it reacts with
superoxide to form peroxinitrite which is a source of highly
reactive hydroxyl and other radicals (Bondy, 1995), hence playing
an important role in neuronal loss. Natural antioxidants have been
reported to protect brain damage against neurological diseases
(Thiyagrajan and Sharma, 2004; Anbarasi et al., 2005a,b; Ahmad
et al., 2005a,b).

Various dietary oils are currently receiving considerable
attention across the world for their potential health benefits in
relation to neurological disorders. SO (Sesame oil) is a
component of the traditional health food in India as well as in
various other oriental countries. The oil is effective against
various diseases including atherosclerosis, hypertension and
anti-aging effects (Fukuda et al., 1985, 1994; Namiki, 1995).
Along with sesaminol, which is the principal antioxidant
component in SO (Osawa et al., 1990), the lignans present
contribute towards its antioxidant and antimutagenic properties
(Kang et al., 1998a,b). SO contains a good amount of phenol,
sesamin, sesamol and sesamolin and relatively small amounts of
tocopherol which contributes to its superior oxidative stability
(White, 1992). In an earlier study, it was reported that SO
showed good antioxidant activity as compared to canola oil
(Baba et al., 1998). Sesame oil, in comparison to other dietary
oils such as ground nut and sunflower, offers better protection
against increased blood pressure, hyperlipidemia and lipid
peroxidation by increasing enzymatic and non-enzymatic
antioxidants (Sankar et al., 2005). Studies have demonstrated
SO and its active ingredient sesamol to be a strong antitumor
promoting agent when compared with resveratrol and sunflower
oil (Kapadia et al., 2002). The present study was undertaken to
observe the protective effect of dietary sesame oil against
oxidative stress in MCAO-induced focal cerebral ischemia in
rats.

Material and methods

Chemicals and drugs

Glutathione (oxidized and reduced), nicotinamide adenine
dinucleotide phosphate reduced (NADPH), ATP, qubain, 1-
chloro-2,4-dinitrobenzene (CDNB), 5,5′-dithiobis-2-nitroben-
zonic acid (DTNB), thiobarbituric acid (TBA), 6-OHDA,
apomorphine hydrochloride, EDTA, NBT, 3,4-dihydroxyphe-
nyl acetic acid (DOPAC), 3,4-dihydroxybenzylamine (DHBA)
were purchased from Sigma Aldrich, USA, and other chemicals
were AR grade.

Dietary sesame seed oil

Sesame seed was provided by Grukul Kangari University,
Haridawar, Uttaranchal, India. SO was extracted from the
sesame seeds according to the method of Mohamed and Awatif
(1998). In brief, the sesame seeds were ground in a
disintegrator. The grounded seeds were extracted with n-hexane
for 48 h, and then filtered. This process was repeated three times
using fresh solvent each time to extract most of the oils from
ground seeds. Free fatty acids were determined by Gas Liquid
Chromatography (GLC) as per the protocol of Fazli et al. (2005)
and sesamin and sesamolin content were analyzed according to
the method of Budowski et al. (1950) (Table 1).

Preparation of diet

Fat-free diet was provided by Animal House, Jamia
Hamdard, New Delhi. Sesame seed oil was mixed in the diet
in appropriate amount (200 ml SO [20%] was mixed in 1 kg
diet). Small pellets were prepared manually and dried at room
temperature.

Animals

Male Wistar rats weighing 300–350 g were obtained from
the Central Animal House, Hamdard University, New Delhi.
Rats were housed in polypropylene cages in air-conditioned
room and were allowed free access to experimental and
pelletted diet (20% SO) and water ad libitum. The study was
approved by the Institutional Animal Ethics Committee (IAEC).

Experimental protocol

Animals were divided in four groups, each of six animals, for
15 days. The first group (sham) was fed pellet diet without
experimental diet; the second group was MCAO, i.e., ischemia
was induced for 2 h followed by reperfusion for 22 h; the third
and fourth groups were fed experimental diet for 15 days followed by
MCAO for 2 h and reperfusion for 22 h (MCAO + SO group);
and the fourth group was fed experimental diet alone (20% SO
in diet) for 15 days. After the completion of the reperfusion
period, the animals were assessed for their neurobehavioral
activity and were sacrificed thereafter. The brains were taken
out and hippocampus was dissected for estimation of biochem-
ical parameters.

Induction of focal cerebral ischemia in rats

The right MCAO was produced using an intraluminal
filament model as described by Salim et al. (2003). In brief, the

Table 1
Analysis of fatty acid profile and Sesamin and Sesamolin content in Sesame seed oil

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean±S.E.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatty acid (%)</td>
<td></td>
</tr>
<tr>
<td>Myristic acid C14:0</td>
<td>0.61±0.11</td>
</tr>
<tr>
<td>Palmitic acid C16:0</td>
<td>12.3±0.8</td>
</tr>
<tr>
<td>Stearic acid C18:0</td>
<td>3.9±0.2</td>
</tr>
<tr>
<td>Arachidic acid C20:0</td>
<td>0.23±0.07</td>
</tr>
<tr>
<td>Oleic acid C18:1</td>
<td>43.2±1.2</td>
</tr>
<tr>
<td>Linoleic acid C18:2</td>
<td>42.6±1.3</td>
</tr>
<tr>
<td>Linolenic acid C18:3</td>
<td>0.27±0.08</td>
</tr>
<tr>
<td>Sesamin (g/100 g)</td>
<td>0.8±0.06</td>
</tr>
<tr>
<td>Sesamolin (g/100 g)</td>
<td>0.5±0.04</td>
</tr>
</tbody>
</table>

Values are expressed as mean±S.E.
rats were anesthetized with chloral hydrate (400 mg/kg, i.p.), a 4–0 nylon monofilament was inserted into the external carotid artery and was advanced into the internal carotid artery. Two hours after the induction of ischemia, the filament was slowly withdrawn and the animals were put into their cages. In the sham-operated rats, the external carotid artery was surgically prepared for insertion of the filament but the filament was not inserted.

**Behavioral activity**

The neurobehavioral activity in MCAO model of focal cerebral ischemia was estimated by the slightly modified method of Yamamoto et al. (1988). The behavioral activity was monitored in video path analyzer (Coulbourn Instrument, USA) by measuring the locomotion, average speed, rest and distance travelled of the animal.

**Tissue preparation**

After producing MCAO and assessment of behavioral activity, the animals were sacrificed immediately and their brains were taken out to dissect the hippocampus. Postmitochondrial supernatant (PMS) obtained from 10% homogenate of tissue was used for the estimation of various parameters related to the oxidative stress.

**Biochemical estimation**

**Estimation of reduced glutathione (GSH).** Reduced glutathione was assayed by the method of Jollow et al. (1974). One milliliter PMS (10%) was precipitated with 1.0 ml sulfosalicylic acid (4%). The samples were kept at 4 °C for 1 h and then subjected to centrifugation at 1200 × g for 15 min at 4 °C. The assay mixture contained 0.1 ml filtered aliquot, 2.7 ml phosphate buffer (0.1 M, pH 7.4) and 0.2 ml DTNB (0.4% in phosphate buffer 0.4 M, pH 7.4) in a total volume of 3.0 ml. The yellow colour developed was read immediately at 412 nm. The rate of LPO was expressed as nmol TBARS formed/h/mg protein.

**Glutathione-S-transferase activity (GST).** Glutathione-S-transferase (GST) activity was measured by the method of Habig et al. (1974). The reaction mixture consisted of 1.425 ml phosphate buffer (0.1 M, pH 7.4), 0.2 ml reduced glutathione (1 mM), 0.025 ml CDNB (1 mM) and 0.3 ml PMS (10%) in a total volume of 2.0 ml. The changes in absorbance were recorded at 340 nm and enzyme activity was calculated as nmol CDNB conjugate formed/min/mg protein using a molar extinction coefficient of 9.6 × 10^3 M^−1 cm^−1.

**Glutathione peroxide activity (GPx).** GPx was estimated according to the procedure described by Mohandas et al. (1984). The reaction mixture consisted of 1.44 ml phosphate buffer (0.05 M, pH 7.0), 0.1 ml EDTA (1 mM), 0.1 ml of sodium azide (1 mM), 0.05 ml of glutathione reductase (1 EU/ml), 0.1 ml of glutathione (1 mM), 0.1 ml of NADPH (0.2 mM), 0.01 ml of hydrogen peroxide (0.25 mM) and 0.1 ml of PMS in the final volume of 2 ml. The disappearance of NADPH at 340 nm was recorded at room temperature. The enzyme activity was calculated as nmol NADPH oxidized/min/mg/protein by using molar extinction coefficient 6.22 × 10^3 M^−1 cm^−1.

**Glutathione reductase activity (GR).** Glutathione reductase activity was assayed by the method of Carlberg and Mannervik (1975), as modified by Mohandas et al. (1984). The assay mixture consisted of 1.65 ml phosphate buffer (0.1 M, pH 7.6), 0.1 ml NADPH (0.1 mM), 0.1 ml EDTA (0.5 mM) and 0.05 ml oxidized glutathione (1 mM) and 0.1 ml of PMS in total volume of 2 ml. The enzyme activity was quantitated at room temperature by measuring the vial of 20 ml capacity and incubated at 37 ± 1 °C in a metabolic shaker (120 rpm/min) for 60 min. Similarly, 1.0 ml of the same homogenate was pipetted in a centrifuge tube and incubated at 0 °C. After 1 h of incubation, 1.0 ml 5% chilled TCA was added followed by 1.0 ml of 0.67% TBA in each vial and proper mixing was done after each addition. The aliquot from each vial was transferred to a centrifuge tube and centrifuged at 3500 rpm for 15 min. Thereafter, supernatant was transferred to another tube and placed in the boiling water bath. After 10 min, the test tubes were cooled and the absorbance of the colour was read at 535 nm. The rate of LPO was expressed as nmol GSH/g tissue.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Sham</th>
<th>MCAO</th>
<th>MCAO+SO</th>
<th>SO alone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Locomotor time (s)</td>
<td>189.33±9.15</td>
<td>105.5±10.11** (44.27%)</td>
<td>144.83±8.92* (37.33%)b</td>
<td>194.0±12.21 (2.46%)a</td>
</tr>
<tr>
<td>Rest time (s)</td>
<td>150.67±10.68</td>
<td>222.83±10.97** (47.89%)</td>
<td>186.83±8.12** (16.15%)b</td>
<td>167.67±10.14 (11.28%)a</td>
</tr>
<tr>
<td>Distance travelled (cm)</td>
<td>3574.5±261.69</td>
<td>1701.83±240.13** (52.38%)</td>
<td>2775.5±209.3 (63.08%)b</td>
<td>3130.0±201.6 (12.43%)a</td>
</tr>
<tr>
<td>Average speed (cm/s)</td>
<td>604.5±40.4</td>
<td>342.83±31.8** (43.28%)</td>
<td>490.07±29.69 (42.94%)b</td>
<td>575.33±35.60 (4.82%)a</td>
</tr>
</tbody>
</table>

Values are expressed as mean±S.E.

a Values in parentheses indicate the percentage change vs. Sham.
b Values in parentheses indicate the percentage change vs. MCAO.
disappearance of NADPH at 340 nm and was calculated as nmol NADPH oxidized/min/mg protein using molar extinction coefficient of $6.22 \times 10^{-3} \text{ M}^{-1} \text{ cm}^{-1}$.

**Catalase activity (CAT).** Catalase activity was assayed by the method of Caliborne (1985). Briefly, the assay mixture consisted of 1.95 ml phosphate buffer (0.05 M, pH 7.0), 1.0 ml hydrogen peroxide (0.019 M) and 0.05 ml PMS in total volume of 3.0 ml. The change in absorbance was recorded at 240 nm. Catalase activity was calculated as nmol of $\text{H}_2\text{O}_2$ consumed/min/mg protein.

**Superoxide dismutase activity (SOD).** SOD activity was measured by the method of Beauchamp and Fridovich (1971). The reaction mixture of total volume 1.0 ml consisted of 0.5 M phosphate buffer pH 7.4, 0.1 ml PMS, 1.0 mM xanthine, 57 $\mu$M NBT. It was incubated for 15 min at room temperature and reaction was initiated by the addition of 50 mU xanthine oxidase. The rate of reaction was measured by recording change in the absorbance at 550 nm due to formation of formazan, a reduction product of NBT.

**Estimation of protein.** Protein will be estimated by the method of Lowry et al. (1951).

**Statistical analysis.** Results are expressed as mean±S.E. of six animals. Differences between the means of experimental and control groups were analysed statistically by using Student’s $t$-test.

**Results**

The fatty acid and antioxidant profile of sesame oil is discussed in Table 1. Similarly, the effect of MCAO and its protection by dietary SO on locomotion, rest, distance travelled and average speed is shown in Table 2. These neurobehavioral activities were significantly ($P<0.001$) decreased and rest time was significantly increased in MCAO group as compared to Sham group. The pretreatment with dietary SO in MCAO group (MCAO+SO group) increased the locomotor time and decreased the rest time as compared to MCAO group without
pretreatment with dietary SO. The Sham + SO group showed no significant difference as compared to Sham without SO group. The distance travelled (centimeters) was significantly depleted in MCAO group as compared to Sham group. The group Sham and Sham + SO showed no significant difference as compared to Sham without SO pretreatment with dietary SO. The Sham + SO group showed a significant difference as compared to Sham. The activities of these enzymes were found to be of the same order in SO alone group. No significant change was observed in Sham and Sham + SO group.

Figs. 1 and 2 show the effect of dietary SO on the TBARS and GSH content in MCAO-induced cerebral ischemia. The content of TBARS in hippocampus was significantly elevated \((P<0.001)\) in MCAO group as compared to Sham group and its content was significantly decreased in MCAO + SO group. The level of GSH content was significantly depleted \((P<0.001)\) in MCAO group as compared to Sham group and its content was significantly attenuated in MCAO + SO group when compared with MCAO group. No significant difference was observed in Sham group.

Figs. 3 and 4 show the effect of MCAO on the activity of SOD and CAT and its protection by dietary SO in cerebral ischemia. The activity of SOD and CAT were significantly decreased in MCAO group \((P<0.001)\) as compared to Sham group. These activities were observed to improve significantly in MCAO + SO group as compared to MCAO group. These activities were found to be of the same order in SO-alone group and Sham groups.

Table 3 also shows the effect of MCAO on the activity of GPx, GR and GST in hippocampus and protection offered by dietary SO. The activities of GPx, GR and GST were found to be significantly depleted in MCAO group \((P<0.001)\) as compared to Sham. The activities of these enzymes were significantly increased in MCAO + SO group as compared to MCAO group. No significant change in the activities of these enzymes was observed in the SO-alone group as compared to Sham group.

Discussion

As shown in earlier studies, oxidative stress promotes lipid peroxidation and alters the antioxidant defense system in brain tissue under ischemic conditions (Thiyagrajan and Sharma, 2004). Brain is highly susceptible to oxidative stress due to its enrichment with non-heme iron that is catalytically involved in the production of free radicals (Yousuf et al., 2005; Salim et al., 2003). The plant-based antioxidants and edible oils have shown to offer protection against human diseases as reported in many studies (Ahmad et al., 2005a,b; Russo and Borrelli, 2005). Neuroprotective effect of curcumin \((C.\) \textit{longa}) has been reported (Thiyagrajan and Sharma, 2004; Sharma et al., 2005). Cui et al. (2004) reported the antioxidant activity of plant derived active components \((c\text{curcumin, turmerin, } \beta\text{-carotene, lycopene and lutin})\) in \textit{in vitro} study. These antioxidant compounds were found protective against DNA damage. In another study, the memory enhancing and neuroprotective effect of \textit{Bacopa monniera} and its active ingredients bacoside A and bacoside B have been reported (Tripathi et al., 1996; Anbarasi et al., 2005a,b; Russo and Borrelli, 2005). Osawa et al. (1990) reported that free radical-induced oxidative damage can be effectively protected by use of various dietary antioxidants. However, not many studies focused on neuroprotective effect of dietary oils have been reported. In present study we used dietary SO (20%) in MCAO-induced rat cerebral ischemia injury to see its protective role and studied the antioxidant enzymatic and non-enzymatic activities in brain hippocampus along with behavioral activities. In our earlier study, the effect of dietary SO (20%) was observed in the frontal cortex of rat brain in MCAO-induced cerebral ischemia and significant enzymatic and non-enzymatic activities were noted in MCAO + SO group (Ahmad et al., 2005b).

The pretreatment with dietary SO before cerebral ischemia for 15 days has shown significant improvement in locomotor activity after 2 h of occlusion and 22 h of reperfusion \((P<0.001)\). In the present study, it has been observed that dietary SO significantly reduced lipid peroxidation level in brain hippocampus in MCAO + SO group as compared to MCAO group \((P<0.001)\) (Fig. 1). The involvement of ROS and oxidative stress has already been reported for damaging brain in MCAO-induced cerebral ischemia and may contribute to selective neuronal vulnerability of regions like hippocampus (Yousuf et al., 2005; Gutsaeva et al., 2006). Impaired balance between prooxidant and antioxidant mechanism results in lipid peroxidation \((LPO)\) which is a major cause for oxidative stress. The reduction of LPO in MCAO + SO group is attributed to the presence of lignans \((sesamin, sesamol and sesamolin)\) in SO (Cooney et al., 2001). Sesamin is reported to enhance hepatic detoxification, reduce occurrence of chemically induced tumor, and protect against oxidative stress (Akimoto et al., 1993; Hirose et al., 2003).
SO also contains vitamin E and is reported to increase glutathione (Sharma et al., 2000), a reservoir of the aldehyde binding compound cysteine. The presence of many antioxidant enzymes such as SOD, CAT, GPx, GR and GST in the brain prevents these tissues from the oxidative damage by free radical formation (Cui et al., 2004). SOD reacts with the superoxide radicals to form \( \text{H}_2\text{O}_2 \). CAT and GPx are involved in the formation of \( \text{H}_2\text{O}_2 \), both at high and low concentrations. In earlier studies, researchers have demonstrated that brain contains low CAT levels, and hence, GPx has a major role in quenching \( \text{H}_2\text{O}_2 \) and other peroxides which otherwise lead to production of hydroxyl and peroxyl radicals in the presence of iron (Bast and Barr, 1997; Gutteridge and Halliwell, 1994); however, oxidative stress lowers these enzyme activities (Ahmad et al., 2005a,b; Yousuf et al., 2005). GR is another important enzyme for the maintenance of intracellular concentration of reduced glutathione (GSH) (Gutteridge and Halliwell, 1994). Reduced glutathione serves as primary antioxidant defense of brain against prooxidant stress and plays an important role in behavioral activity (Cruz-Auguido et al., 2001). The level of brain GSH is significantly reduced after ischemic injury (Ravindranath and Reed, 1990). This depletion was directly associated with elevation in brain lipid peroxidation. The level of brain GSH has been attributed to offer protection against ROS generation by ischemic stress besides its consumption by the antioxidant enzymes, GPx and GST (Baskaran et al., 1999). GST catalyses the detoxification of oxidized metabolites of catecholamines (\( \text{o-quinone} \)) and may serve as antioxidant, preventing degenerative processes (Baez et al., 1997).

The SO mixed diet increased the antioxidant enzymes activity in brain hippocampus in MCAO+SO group as compared to MCAO group (Figs. 3, 4 and Table 3). This increased activation of the antioxidant enzymes due to the dietary SO could be due to the decreased utilization since the LPO levels are low in the SO group. Our results are in agreement with Prasanthi et al. (2005), as they have reported increased antioxidant enzyme activity in oxidative damage in rat tissue by using dietary SO. This is also supported by a wide range of studies, which have shown that naturally occurring antioxidants can prevent or slow down other diseases (Azuine and Bhide, 1994; Gordon, 1996).

The exact mechanism by which dietary SO reduces oxidative stress is not very clear from this study, but it is strongly believed that the protective effect is due to the presence of lignans (sesamin, sesamol and sesamolin) and vitamin E. The antioxidant properties of lignans have already been shown in several studies (Ogawa et al., 1995; Ide et al., 2001; Kapadia et al., 2002; Abe et al., 2005). It has also been reported earlier that vitamin E increases the glutathione (Sharma et al., 2000), a reservoir for the aldehyde binding compound cysteine, and significantly lowers blood pressure in rats (Newaz and Nawal, 1998). Vitamin E also lowers plasma lipid peroxides in diabetic patients (Sharma et al., 2000). Furthermore, sesamol has also been shown to be a classical inhibitor of LPO (Namiki, 1995; Uchida et al., 1996). This study was also supported by the significant improvement in behavioral activities in rats due to the pretreatment of SO. These results clearly indicate a correlation between the significant role of antioxidant enzymes and non-enzymes on behavioral activities of rats as the significant role of GSH has already been reported (Cruz-Auguido et al., 2001).

Conclusion

In conclusion, the findings of our study clearly indicate cerebral ischemia induced oxidative damage in brain in Male Wistar rats in terms of neurobehavioral activities, increased lipid peroxidation, depletion of glutathione level, and changes in various enzymatic antioxidants. Our findings further demonstrate that intake of dietary SO could effectively ameliorate the cerebral ischemia-induced oxidative damage which may be due to the presence of powerful antioxidant compounds in sesame seed.

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