**Sesame indicum, a Nutritional Supplement, Elicits Antiamnesic Effect via Cholinergic Pathway in Scopolamine Intoxicated Mice**

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ABSTRACT: Purpose: Present study was undertaken to evaluate the antiamnesic effect of *Sesamum indicum* (S. indicum) seeds (standardized for sesamin, a lignan, content) in scopolamine, a muscarinic antagonist intoxicated mice. Methods: Male Swiss albino mice (18–22 g bw) were pretreated with methanolic extract of sesame seeds (MSSE) (100 and 200 mg/kg/day, p.o) for a period of 14 days. Scopolamine (0.3 mg/kg, i.p.) was injected on day 14, 45 ± 10 min after MSSE administration. Antiamnesic effect of MSSE was evaluated using step-down latency (SDL) on passive avoidance apparatus and transfer latency (TL) on an elevated plus maze. To unravel the mechanism of action, we examined the effects of MSSE on the genes such as acetyl cholinesterase (AChE), muscarinic receptor M1 subtype (mAChR-M1), and brain derived neurotrophic factor (BDNF) expression within hippocampus of experimental mice. Further, its effects on bax and bcl-2 were also evaluated. Histopathological examination of hippocampal CA1 region was performed using cresyl violet staining. Results: MSSE treatment produced a significant and dose dependent increase in step down latency in passive avoidance test and decrease in transfer latency in elevated plus maze. To unravel the mechanism of action, we examined the effects of MSSE on the genes such as acetyl cholinesterase (AChE), muscarinic receptor M1 subtype (mAChR-M1), and brain derived neurotrophic factor (BDNF) expression within hippocampus of experimental mice. Further, its effects on bax and bcl-2 were also evaluated. Histopathological examination of hippocampal CA1 region was performed using cresyl violet staining. Results: MSSE treatment produced a significant and dose dependent increase in step down latency in passive avoidance test and decrease in transfer latency in elevated plus maze in scopolamine intoxicated injected mice. MSSE down-regulated AChE and mAChRM1 and up-regulated BDNF mRNA expression. Further, it significantly down-regulated the bax and caspase 3 and up-regulated bcl-2 expression in scopolamine intoxicated mice brains. Mice treated with MSSE showed increased neuronal counts in hippocampal CA1 region when compared with scopolamine-vehicle treated mice. Conclusion: Sesame seeds have the ability to interact with cholinergic components involved in memory function/restoration and also an interesting candidate to be considered for future cognitive research. © 2015 Wiley Periodicals, Inc. Environ Toxicol 00: 000–000, 2015.

Keywords: Sesamum indicum; memory; scopolamine; acetyl choline esterase; step down; latency; transfer latency

INTRODUCTION

In the modern era, herbs or herbal based products are gaining prominence as pharmaceuticals and nutraceuticals because...
of the lesser or no side effects and cost effectiveness (Noguchi, 2000). Sesamum indicum commonly known as sesame is one of the earliest and common condiment crops cultivated for its edible oil (Kotade, 2008). In South Asia, sesame products (seeds and oil) are being used as a nutrition rich component in the traditional culinary practices. Several reports revealed its rich antioxidant properties (Yamashita et al., 2003; Suja et al., 2004; Joshi et al., 2005) Lignans—sesamol, sesaminol, pinoresinol, and sesaminol and vitamin-E were indicated as bioactive compounds for the free radicals scavenging activity of sesame (Kang et al., 1999, 2000; Lemcke-Norojarvi et al., 2001). Further, lignans were reported to impart neuroprotective effect in a rat model of focal cerebral ischemia (Saif et al., 2006). However, sesame seeds remain to be evaluated for its effects against scopolamine induced anterograde amnesia.

Central cholinergic system plays a crucial role in learning and memory (Hlinak and Krejci, 1998). Loss of cholinergic neurons is implicated in cognitive decline associated with aging and other neurodegenerative diseases. The other major systems like glutameric, opioidergic neurotransmissions were also reported to be involved in learning and memory processes. Muscarinic receptor agonist and cholinesterase inhibitors were shown to improve memory both in animals and humans (Schwarz et al., 1999; Lyketsos et al., 1999). Dynorphin, an endogenous kappa opioid agonist shown to ameliorate scopolamine induced amnesia (Itoh et al., 1993) and CO induced delayed amnesia. Whilst multiple neurotransmitter systems are involved in learning and memory, still chemical entity (ies) that restores cholinergic function are of major interest in drug discovery (Hiramatsu et al., 1995, 1997; Van Beek and Claassen, 2001).

Scopolamine model of amnesia is one of the widely used pharmacological models of “cholinergic amnesia” which turn out to be very popular after the establishment of cholinergic hypothesis of geriatric memory dysfunction (Blake et al., 2011; Wang et al., 2013) In the present study, standardized extract of sesame was evaluated for its protective effect against scopolamine induced amnesia. Furthermore, its role on the memory related cholinergic genes such as acetylcholine esterase (AChE) and muscarinic receptor (mAChRM1) expression was also investigated.

**MATERIALS AND METHODS**

**Sesame Seeds, Chemicals, and Reagents**

Sesame seeds were procured from M/s Organic Farm, Chennai, India. Seeds were authenticated by Dr. P. Jayaraman, Director, National Institute of Herbal Science, Plant Anatomy Research Centre, Chennai.

Scopolamine hydrobromide, piracetam, and Tri reagent were purchased from Sigma Aldrich, USA. RT-PCR premix (2×) was purchased from Genet Bio, USA. Primers for RT-PCR were purchased from Eurofins Genomics, Bangalore. All other chemicals used were of analytical grade.

**Rationale for Selecting Methanol as Solvent for Extraction of Sesame**

Though it is understandable that in nutritional research, the test substance should be given to animals in the same form/manner as that of human exposure; but in order to obtain a reliable and reproducible result in the initial screening it was decided to perform using extract of sesame seeds. Methanolic sesame extract was shown to possess major lignans like sesamolin and sesamin (Sukumar et al., 2008) and minor lignans such as acuminatolide, piperitol, and pinoresinol (Kuo et al., 2011). Methanolic extract was also reported to possess rich antioxidant activity than n-hexane extract which was corroborated to high lignans content (Farhoosh and Sharif, 2013). Further, lignans were reported for neuroprotective effects (Park et al., 2010) Hence, we decided to use methanol as solvent to prepare the extract of sesame seeds.

**Preparation of Methanolic Extract of Sesame Seeds (MSSE)**

S indicum seeds were coarsely powdered and subjected to hot continuous extraction using methanol in a Soxhlet’s apparatus. Extracts were concentrated under vacuum in a rotary evaporator, dried and stored in vacuum desiccator until further analysis.

**Standardization of MSSE**

MSSE was standardized for sesamin content by high performance thin layer chromatography (HPTLC) following the method published earlier (Sukumar et al., 2008).

**Animals, Husbandry, and Ethical Approval**

Male Swiss albino mice (18–22 g b.w.) were obtained from Central Animal Facility, Sri Ramachandra University, Chennai, Tamil Nadu, India. Animals were housed in polypropylene colony cages (six animals/group) in a well-ventilated room (air cycles: 15 air exchanges per hour; recycle ratio: 70:30) under an ambient temperature of 22 ± 3°C and 50–70% relative humidity, with a 12-h light/dark artificial photo cycle. They were provided with extruded rodent feed (Provimi Animal Nutrition India Pvt. Ltd, India) and purified water *ad libitum*. Animals were acclimatized at least for 7 days to laboratory conditions before initiation of the experiment. Guidelines of “Guide for the Care and Use of Laboratory Animals” (Institute of Laboratory Animal Resources, National Academic Press 1996; NIH publication number #85-23, revised 1996) were strictly followed throughout the study. Institutional Animal Ethical Committee (IAEC), Sri Ramachandra University, Chennai, India approved the study protocol (IAEC/XXII/SRU/171/2011).
Dose for the present study was fixed based on the earlier published equivalent paper (Monteiro et al., 2004). It is in use since time immemorial which reveals its safety.

**Groupings and Test Drug Treatment Schedule**

Experiment animals were divided into five groups (six animals/group). Group I and II received 0.5% CMC (10 mL/kg b.w., p.o.; negative control), Group III received piracetam (250 mg/kg b.w., p.o.; reference control), Group IV and V received MSSE (100 and 200 mg/kg b.w., p.o. respectively). Animals were pretreated with vehicle/test drug for a period of 14 days. On day 13, 45 ± 10 min after appropriate treatment, animals were trained in passive avoidance (step down latency) and elevated plus maze (transfer latency) apparatus. On day 14, 45 ± 10 min after vehicle/test drug administration, scopolamine (0.3 mg/kg b.w., i.p.) was injected to experimental animals except Group I. Animals were subjected to memory tests 30 ± 5 min after scopolamine administration.

Sesame seeds forms integral diet in south Indian diet and it is in use since time immemorial which reveals its safety. Dose for the present study was fixed based on the earlier published equivalent paper (Monteiro et al., 2004).

**Memory Tests**

**Passive Avoidance Paradigm**

One-trial step-down type passive avoidance task was performed to examine the short-term working memory in mice. The apparatus consists of electric grid floor with centrally located wooden block which served as shock free zone (SFZ). During training period, mouse was placed on SFZ, as the mouse stepped down on the platform (placing all the four paws) a foot shock (20 mA) was delivered for a period of 15 s. Then mice were replaced to their respective home cage. Retention test was carried out 24 h (day 14) following training session (day 13). Latency to step down from SFZ was recorded. Escape latency (EL), time taken by the animal to get back to the SFZ was also recorded. A cut-off time of 180 s was fixed and animals not entering the closed arm within this period were assigned another 2 min and returned to its home cage. Retention of this learned task was examined 24 h after (day 14) the first day trial. A cut-off time of 180 s was fixed and animals not entering the closed arm within this period were assigned with retention latency of 180 s (Itoh et al., 1991)

**Transfer Latency Paradigm**

Transfer latency is used as an index of learning and memory processes. Elevated plus-maze consisted of two open arms (16 L × 5 W cm) and two closed arms (16 L × 5 W × 12 H cm). The arms extended from a central platform (5 × 5 cm) and the maze was elevated to a height of 25 cm from the floor. During training session (day 13), mice were kept at the end of open arm, facing away from central platform. Transfer latency (the time taken by the mice to move into any one of the closed arms), was recorded on the first day (day 13) for each animal. Mice were allowed to explore the maze for another 2 min and returned to its home cage. Retention of this learned task was examined 24 h after (day 14) the first day trial. A cut-off time of 180 s was fixed and animals not entering the closed arm within this period were assigned with retention latency of 180 s (Itoh et al., 1991)

**Determination of Acetylcholinesterase Activity**

Following memory tests, the animals were euthanized and brains were collected, hippocampal region was identified using mouse atlas, micro-dissected and processed for further analysis. Acetylcholinesterase activity was determined as per Ellman et al. method with minor modifications (Ellman et al., 1961). Briefly, 10% homogenate of the hippocampal tissues was prepared using 0.1M phosphate. To the homogenate, 0.5 mL of 0.01M 5-dithiobis-2-nitrobenzoic acid (DTNB) and 0.075 mL of 0.075M acetylthiocholine iodide were added. All the reagents except homogenate served as blank. Absorbance was read at 412 nm and acetylcholine esterase activity was expressed as mg of acetylcholine liberated/minute/mg protein.

**Total Protein**

Total protein content in the brain homogenate was estimated by Lowry et al. method (Lowry et al., 1951).

**mRNA Expression Analysis**

Reverse transcriptase polymer chain reaction (rtPCR) was performed as described previously (Shiao et al., 2013) using RT-PCR premix (2×) to determine the mRNA expression, gene and primer sequence are represented in Table I. Briefly, total RNA was extracted using TRIzol reagent. 1% of hippocampal brain regions were homogenized in TRI reagent, tubes were incubated for 10 min and centrifuged. Chloroform was added to the supernatant, allowed to incubate for 5 min at room temperature and centrifuged at 12,000 rcf.

### TABLE I. Represents the gene its forward and reverse primer sequence

<table>
<thead>
<tr>
<th>Gene</th>
<th>Forward Primer</th>
<th>Reverse Primer</th>
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<tbody>
<tr>
<td>AChE</td>
<td>5'-GCA GAA CTT CAC TGA CCA AAAG-3'</td>
<td>5'-TCA AAG GAG GGG GAC TCA TA-3'</td>
</tr>
<tr>
<td>mAChR M1</td>
<td>5'-CTG GTC AAG GAG AAG GCA GCT 3'</td>
<td>5'-GTC TCT CTG GCC TGTCCA GGA AGG-3'</td>
</tr>
<tr>
<td>BDNF</td>
<td>5'-CCA TAA GGA CGC GGA CTT GT-3'</td>
<td>5'-GAG GCT CCA AAAG GCA TTGA-3'</td>
</tr>
<tr>
<td>BAX</td>
<td>5'-GAG TGT CTC CGG CGA ATTG-3'</td>
<td>5'-TTG TGA CGG AGG CGG TGAG-3'</td>
</tr>
<tr>
<td>Caspase 3</td>
<td>5'-AGG GGT CAT TTA TGG GAC A-3'</td>
<td>5'-TAC ACG GGA TCT GTT TTG TG-3'</td>
</tr>
<tr>
<td>Bcl-2</td>
<td>5'-CGG GAG ATC GTG ATG AAGT-3'</td>
<td>5'-CCA CGG AAC TCA AAG AAGG-3'</td>
</tr>
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Then, isopropyl alcohol was added to precipitate the total RNA and centrifuged for 15 min, following the incubation period of 10 min. The supernatant was decanted, the pellet was washed thrice with 75% ethanol, centrifuged and the pellet was allowed to air dry. The pellet was resuspended in RNase free water and stored at 280°C until use. rtPCR was carried using PCR master cycler gradient (Eppendorf, Germany) and semiquantified using Bio1D software in gel documentation (Vilber Loumart, France).

Histopathological Examination

Experimental animals were euthanized and transcardially perfused with normal saline. Brains followed by perfusion were fixed in 10% neutral buffered formalin for a period of 48 h. Coronal sections of brain at hippocampus region were trimmed and subjected to paraffin embedding process. 5 μm thickness of brain tissues were sectioned and stained with cresyl violet for histopathological examination and examined under light microscopy (Motic DMB1–2MP, China) (Bancroft et al., 2008).

Data Analysis

Data were expressed as mean ± standard error mean (SEM). Mean difference between the groups were analyzed by one way ANOVA followed by Tukey’s multiple comparison as post hoc test. p value ≤0.05 was considered as significant. Statistical analysis was performed using GraphPad prism 5.0 (San Diego).

RESULTS

Sesamin Content

Sesamin, asarinin, and sesamolin are the major chemical constituents of sesame seeds. In particular, sesamin was shown to possess multiple pharmacological properties like neuroprotective (Cheng et al., 2006), antihypertensive (Lee et al., 2004; Nakano et al., 2008) and hypocholesteremic (Visavadiya and Narasimhacharya, 2008) effects. The present study being an initial attempt to study the nootropic ability of sesame seeds, it was standardized for its sesamin content using high performance thin layer chromatography technique. The sesamin content in seeds used for the present study was found to be 16.35 ± 0.09% w/w. HPTLC chromatogram is shown in Figure 1.

Passive Avoidance Test

A significant (p < 0.01) decrease in step down latency was recorded in the scopolamine-vehicle treated mice when compared with negative control group. Mice pretreated with MSSE showed a significant (p < 0.01) and dose dependent increase in the time taken to step down from the shock free zone when compared with the scopolamine-vehicle treated mice [Fig. 2(A)]. Similarly, the escape latency was also found to be significantly (p < 0.01) prolonged in scopolamine-vehicle treated group when compared with vehicle control group. Treatment with MSSE at 100 mg/kg b.w. (p < 0.05) and 200 mg/kg b.w. (p < 0.01) significantly decreased escape latency when compared with scopolamine-vehicle treated group. Results were comparable with piracetam [Fig. 2(B)].

Transfer Latency

Scopolamine-vehicle treated group showed significant increase in transfer latency and retention latency when compared with negative control group. MSSE at 200 mg/kg b.w. significantly decreased the transfer latency. Results were found to be comparable with that of reference drug, piracetam [Fig. 3(A,B)].

Determination of Acetylcholinesterase Activity

Acetylcholine esterase activity was significantly increased in scopolamine-vehicle treated group when compared with
negative control group. Pretreatment with MSSE decreased AChE activity significantly ($p < 0.05$) in a dose dependent manner (Fig. 4).

**mRNA Expression Analysis**

mRNA expression of AChE ($p < 0.01$) and mAChRM1 ($p < 0.01$) were significantly up-regulated and BDNF level was

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**Fig. 2.** Effect of MSSE on (A) step down latency (SDL) and (B) escape latency (EL) as evidenced by one-trial step-down type passive avoidance test. Data are expressed in mean $\pm$ SEM; $n = 6$; significance with Tukey's multiple comparison followed by one-way anova. ##$p < 0.01$ vs. vehicle control group; **$p < 0.01$ vs. scopolamine-vehicle treated group.

**Fig. 3.** Effect of MSSE on (A) transfer latency (TL) and (B) retention latency (RL) as evidenced by transfer latency. Data are expressed in mean $\pm$ SEM; $n = 6$; significance with Tukey's multiple comparison followed by one-way anova. ##$p < 0.01$ vs. vehicle control group; **$p < 0.01$ vs. scopolamine-vehicle treated group.
significantly \( (p < 0.01) \) down-regulated in scopolamine-vehicle treated group in comparison to negative control group. Bax and caspase 3 expression were significantly \( (p < 0.01) \) up-regulated and bcl-2 expression was down-regulated \( (p < 0.01) \) in scopolamine-vehicle treated group when compared with negative control group.

Treatment with MSSE at both dose levels significantly \( (p < 0.01) \) decreased hippocampal AChE expression when compared with scopolamine-vehicle treated group. MSSE at 200 mg/kg down-regulated mAChRM \(_1\) expression significantly \( (p < 0.01) \). However, MSSE at 100 mg/kg b.w. did not show any significant difference on mAChRM \(_1\) expression, compared with scopolamine-vehicle treated group.

Pretreatment with MSSE at 100 and 200 mg/kg b.w. significantly \( (p < 0.05 \text{ and } p < 0.01, \text{ respectively}) \) up-regulated BDNF expression in a dose dependent manner in comparison with the scopolamine-vehicle treated group. MSSE at 200 mg/kg b.w. significantly \( (p < 0.01) \) down-regulated bax and caspase 3 expression when compared with scopolamine vehicle treated group. There was a significant \( (p < 0.05) \) decrease in caspase 3 expression in MSSE at 100 mg/kg b.w. however, no significant change was observed in Bax expression when compared with scopolamine vehicle treated group. The results were comparable with that of reference drug, piracetam [Fig. 5(A–F)].

### Histopathology Analysis

Figure 6(A–E) shows the representative photographs of CA\(_1\) region stained with cresyl violet. In vehicle control group, hippocampal region revealed well rounded nuclei with presence of Nissl substances. Histopathology analysis of scopolamine-vehicle treated group revealed nuclear pyknosis with loss of intracytoplasmic Nissl substances. There was approximately 75\% neuronal loss with moderate degree of degeneration in CA\(_1\) region of hippocampus when compared with vehicle control group. MSSE at 100 mg/kg b.w. exhibited loss of intracytoplasmic Nissl substance with approximately 50\% neuronal loss. However, MSSE at 200 mg/kg b.w. showed significant decrease in neurodegeneration with approximately 35\% neuronal loss in hippocampus when compared with scopolamine-vehicle treated group.

**DISCUSSION**

Data from the present study reveals that the methanolic extract of sesame seeds have the ability to restore the molecular and functional properties of cholinergic components against scopolamine induced neurotoxicity. Scopolamine produces learning, consolidation, and retrieval deficits by precipitating cholinergic functions (Hodges et al., 2009; Klinkenberg and Blokland, 2010). Shortened step down latency and increased escape latency in passive avoidance and transfer latency period in elevated plus maze in the scopolamine-vehicle group reveals that scopolamine interfered in retrieval process in mice. Cholinergic enhancers were shown to improve memory in both passive avoidance and elevated plus maze paradigm (McNamara et al., 1996). In the present study, MSSE ameliorated the retrieval deficits in both passive avoidance and transfer latency tests which demonstrate its antiamaesic activity.

Bernard et al. (2003) demonstrated a correlation between AChE activity and mAChR expression wherein, AChE knock-out mice exhibit severe loss of mAChR binding sites at cell surfaces. In the present study, we observed an up-regulated mAChR expression which might be due to increased AChE activity in scopolamine injected mice. MSSE treated mice showed decreased AChE activity which could have played a significant role in improving memory in scopolamine injected mice.

Preclinical studies demonstrate that mAChR antagonist produces cognitive decline. In addition, mAChR activation was found to attenuate apoptosis induced by \( \beta \) amyloid in AD brain via PKC-mediated activation of Wnt/\( \beta \)-catenin signaling pathways (Fuentenalba et al., et al., 2004) In the present study, scopolamine increased proapoptotic and decreased the antiapoptotic and BDNF mRNA expression in mice brain. Increased expression of mAChR M\(_1\) by MSSE could be corroborated to the decrease in bax and caspase 3 and increase in bcl-2 expression.

Neurotrophic factors are largely associated with regulation of neurotransmitters synthesis. Neurotrophins activates and promotes cholinergic neuronal survival and intracerebral administration of neurotrophic factors prevents cholinergic
dysfunction and associated behavioral deficits. Cholinergic neurotransmitter play prominent role in release and synthesis of neurotrophic factors (Knipper et al., 1994). We observed an increased expression of BDNF in MSSE treated mice and this may be the underlying mechanism to the improved cholinergic function and in turn improved memory in scopolamine injected mice.

Apoptosis is considered to be one of the principal mediators of neurodegeneration. Activation of bax and caspase 3 are associated with neuronal loss in various neurodegenerative diseases (Mattson, 2000). There was a severe neurodegeneration in the CA1 region of scopolamine injected mice as evidenced by decreased Nissl substances (Figueiredo et al., 2011). Sesaminol glucosides, major sesame seed lignan was shown to inhibit the activation of caspase and elevate the bax/bcl ratio against Aβ-amyloid induced apoptosis in SK-N-SH cell lines (Um et al., 2012). Further, Ramachandran et al. (2012) showed that sesamol, a phenolic antioxidant contained only in processed sesame oil, upregulated Bcl2 and down-regulated p53, Bax, caspase 3, in human skin fibroblast, HDFa, cell lysates exposed to UVB irradiation. MSSE suppressed bax and caspase 3 and improved bcl-2 expression which reveal its anti-apoptotic effect in scopolamine intoxicated state and this could be the possible reason for the decreased in neuronal loss in histopathological examination. From the foregoing discussion, it is clear that sesame seeds can potential interacts with cholinergic system and possess neuroprotective activity.

Sesame has been reported to contain high percentage (around 40%) of PUFA (Ref: USDA nutrition database) that corroborates its beneficial effect on cognitive function with aging (Zhang et al., 2011). Similarly sesame has been found to be a rich source of fat soluble vitamins like vitamin E and K (Ref: USDA nutrition database) that has an ability to cross BBB and protect brain from oxidative and neuronal damage (Xiang et al., 2012). In recent days, the usage of sesame has been not only limited to cooking purpose but also for therapeutic use as an alternative medicine. Sesame seeds were reported for their high lignin content especially sesamin and sesamolin. Lignans, especially sesamin and sesamolin were identified as active principles in sesame seeds which were

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**Fig. 5.** Effect of MSSE on mRNA expression of (A) AchE, (B) mACHR, (C) BDNF, (D) Bax, (E) Caspase-3, and (F) Bcl-2 as evidenced by rtPCR in scopolamine induced mice. Data are expressed in mean ± SEM; n = 3; significance with Tukey’s multiple comparison followed by one-way anova. ##p < 0.01 vs. vehicle control group; *,**p < 0.05 and 0.01, respectively vs. scopolamine-vehicle treated group.
reported to be responsible for its potent neuroprotective effect. In the present study, the nootropic activity exerted by sesame seeds can be speculated at least in part, due to its rich lignans content.

CONCLUSION

To summarize, sesame as food or neutraceutical, it has an advantageous role in cognitive function and potency to exhibit neuroprotective activity.

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