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EVALUATE THE EFFECT OF *BACOPA MONNIERI, EVOLVULUS ALSINOIDES* AND *TINOSPORA CORDIFOLIA* ON BIOCHEMICAL PARAMETERS IN RATS BRAIN

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ABSTRACT: *Bacopa monnieri*, *Tinospora cordifolia*, and *Evolvulus alsinoides* are conventional Ayurvedic floras that are useful in the various neuropharmacological ailment. This research focused on the effect of ethanolic extract of *Bacopa monnieri* (BME), *Tinospora cordifolia* (TCE), and *Evolvulus alsinoides* (EAE) were evaluated alone and in a combination of extracts (CEPs) by using scopolamine-induced oxidative stress in rats. Tissue homogenates were prepared, an indicator of Superoxide Dismutase (SOD), Glutathione (GSH), Lipid peroxidation (LPO), Catalase (CAT) and Acetylcholinesterase (AChE) enzyme activities were estimated in the rat brain. Due to scopolamine treatment, enhanced LPO and AChE level but reduced SOD, CAT, and GSH levels in rats brain were observed. Rats treated with BME, TCE, EAE, and their CEPs [CEP-1 (EAE and BME), CEP-2 (TCE and BME), CEP-3 (TCE and EAE) and CEP-4 (BME, TCE, and EAE)] at 200 mg/kg, provided significant effect against scopolamine because these extracts decreases LPO, AChE level and restored SOD, GSH, and Catalase levels in rats brain. The CEPs produced significant effects in comparison to BME, TCE, and EAE. Thus, these results demonstrate that BME, TCE, and EAE in CEPs provide significant protective potential against Scopolamine induced oxidative stress.

INTRODUCTION: Deterioration of the cerebral neurons is a universal and essential cause of cognitive dysfunctions observed in older aged [1]. Dementia is an intellectual disorder that leads to loss of brain capability and memory. The main reason for dementia is known to be a progressive neurodegenerative ailment, which is Alzheimer's disease (AD) related to the neuronal loss in a cord and brain region [2,] and it is the main regular appearance of the beginning of dementia in adult and awareness insufficiency diseases [3].

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Losses of memory with lesser retention are general problems now a day. Strain and age are the most important circumstances that may escort to loss of memory and dementia [4]. Scopolamine is a muscarinic receptor antagonist, has found to create the loss of memory in assured investigational animals, and is considered diverse as a useful experimental approach to study the mechanism responsible for cognitive impairment in Alzheimer's disease [5]. Scopolamine also mediates the cholinergic discrepancy during oxidative strain [6]. Since oxidative strain has also been studied to cause Alzheimer's disease, thus several antioxidants have been used to reduce oxidative strain in AD [7, 8].

Natural products may be a good alternative as neuropsychotropic drugs [9] to cure Alzheimer's disease [10, 11]. *Bacopa monnieri* acts as a key ingredient and a memory enhancer of Ayurvedic

medicines of India [12, 13]. Complete flora of *E. alsinoides* used traditionally as medicine for immunomodulatory, nervous weakness and treatment of stress induced-neurodegenerative disease [14]. A rejuvenating method recommends by the Ayurveda where *Tinospora cordifolia* acts as an essential constituent to improve memory function [15]. *B. monnieri, E. alsinoides, T. cordifolia* and their combinations provided significant result against Scopolamine [16].

In Alzheimer's disease the level of the biochemical parameter changes in rat brain. Therefore, this research was focused to evaluate the effect of BME, TCE, and EAE on biochemical parameters (SOD, LPO, GSH, Catalase, and AChE) in rat's brain.

MATERIALS AND METHODS:

Plant Material: Plants were bought from and authenticated at Dr. Yashwant Singh Parmar University, Solan, Himachal Pradesh, India (Authentication reference No. UHF/FP/Herbarium/ 1113).

Extraction and Preparation of Combinations: Half kg of each plant material was processed and dried in shadow. Plant materials were trodden and after defatting with pet ether (40:60) these materials extracted in the Soxhlet apparatus by using ethanol as solvent. Using a rotary vacuum evaporator ethanolic extracts were dried (40°C) and reserved in a hermetic container till supplement use. Ethanolic extract of *Bacopa monnieri* (BME), *Tinospora cordifolia* (TCE), and *Evolvulus alsinoides* (EAE) were properly mixed and formulate four combinations of extracts (CEPs). CEP-1 (EAE and BME) in a ratio of 1:1, CEP-2 (TCE and BME) in a ratio of 1:1, CEP-3 (TCE and EAE) in a ratio of 1:1, and CEP-4 (BME, TCE, and EAE) in a ratio of 1:1:1.

Animals Housing and Ethics Clearance: The study was performed on Wistar rats. All rats were kept in under controlled situations (temperature 22 ± 2 °C). All rats were given regularly a benchmark diet (Golden feed, New Delhi) and water. The research protocol was approved by the Institutional Animal Ethics Committee, Bhupal Nobles' College of Pharmacy, Udaipur, Rajasthan (Reg. no. 870/ PO/Re/S/05/CPCSEA). Protocol Approval Ref. no. 14/BNCP/IAEC/2018.

Treatments and Experimental Design: Further rats were separated into 10 groups, each group having 6 rats. Group I- vehicle (0.1% CMC); Group II- scopolamine (Control); Group III- Piracetam (Standard) 100 mg/kg, scopolamine; Group IV- BME, scopolamine; Group V- EAE, scopolamine; Group VI- TCE, scopolamine; Group VII- CEP-1, scopolamine; Group VIII- CEP-2, scopolamine; Group IX- CEP-3, scopolamine; Group X- CEP-4, scopolamine. The dose of extracts in group IV to X was 200 mg/kg whereas for scopolamine the dose was 0.3 mg/kg.

Estimation of Biochemical Parameters: The ether anesthetized rats were sacrificed by cervical displacement after probe trial and the brain was dissected on the ice-cold surface. Tissue homogenates were prepared; an indicator of SOD, LPO, GSH, CAT, and AChE enzyme activities was estimated in the rat's brain.

Preparation of Tissue Homogenate: Tissue samples of rat's brains were weighed and in each sample, 10% w/v of pH 7.4 tris HCl (0.15 M) buffer was added. A mixture of each sample was homogenized under a chilled condition using a motor-driven Glass-Col® homogenizer, and tissue homogenate was collected.

Superoxide Dismutase (SOD) Assay: In clean test tubes tissue homogenate (0.1 ml) was taken, and to this Sodium pyrophosphate buffer 1.2 ml (pH 8.3, 0.052 M), 0.3 ml (300 µM) Nitroblutetrazolium, 0.1 ml (186 µM) Phenazine methosulphate and 0.2 ml (750 µM) NADH is added. Samples were mixed properly and incubate at 30 ºC for ninety seconds. After incubation, glacial acetic acid was mixed with 0.1 ml and stirred with n-butanol (4.0 ml). The sample was permitted to set for ten min, at 2000 rpm centrifuged the samples for ten min. Butanol film was taken out, and its color intensity was determined from a UV-visible spectrophotometer at 560 nm against butanol as blank. Control consists of a similar reaction mixture except for tissue homogenate sample, instead of which equal amount of buffer (Sodium pyrophosphate buffer pH 8**.**3) was taken [17].

Lipid Peroxidation (LPO) Assay: LPO of different extracts was projected by measuring the amounts of malondialdehyde (MDA). In the test

tubes, the tissue homogenate was taken exactly 0.2 ml, and 1.5 ml tertiary-butyl alcohol, 0.2 ml of 8.1% sodium dodecyl sulfate, and 20% acetic acids (1.5 ml) were added. With the use of distilled water, the volume of the assortment was prepared to 4 ml and after that heated in a water bath (95 ºC) for sixty minutes using glass balls as a condenser. Further, in-room temperature, the samples were cooled and volume adjusted with the use of distilled water, make up to 5 ml. Butanol: pyridine (15:1, 5 ml) combination was mixed, and the contented was vortexed thoroughly for two minutes. At 3000 rpm each mixture was centrifuged for ten minutes. The separated out an upper organic layer. The organic layer reads absorbance at 532 nm operating UV-visible spectrophotometer against butanol: pyridine (15:1) solution as a blank. This absorbance was measured and lipid peroxidation activity was calculated from the extermination Coefficient of malondialdehyde (MDA) formed which was compared with the standard graph [18].

Reduced Glutathione (GSH) Assay: In test tubes, 0.2 ml tissue homogenate was taken to this 0.2 ml of 20% TCA (trichloroacetic acid) and 1 mM EDTA was added. Permitted the solution to set for 5 minutes. For 10 min every one test tube was centrifuged at 2000 rpm. 200 µl supernatant was transferred in a fresh set of experimental tubes and 1.8 ml of prepared Ellman's reagent was added. Distilled water used to prepared the volume up to 2 ml. The absorbance of each sample was taken at 412 nm with a UV-visible spectrophotometer against water as the blank. Results were interpreted in mmol/gm tissue [19, 20].

Catalase (CAT) Assay: Exactly 100 µl of supernatant was taken in a quartz cuvette, phosphate buffer 1.9 ml (50 mM, pH 7.0) and freshly ready hydrogen peroxide (H_2O_2) 1.0 ml (30 mM) were mixed. At 240 nm absorbance of every sample was recorded for 3 min with an interval of 30 sec with an initial delay of 3 sec. A total of six readings were taken for each sample. As a reference phosphate buffer (pH 7.0, 50 mM) was used [21].

Acetylcholinesterase (AChE) Assay: In a cuvette which was already contained 2.6 ml phosphate buffer (0.1 M, pH 8) and 100µl of DTNB, 0.4 ml aliquot part of the homogenate was mixed. Cuvette contents were assorted systematically by bubbling air, and in a spectrophotometer, absorbance was calculated at 412 nm. When absorbance achieves a constant value, basal reading was recorded. 20µl of the substrate like ACh was mixed and alter in absorbance was noted for ten minutes at intermission of two minutes. Per-minute alteration in the absorbance was determined [22].

Intraperitoneal (I.P.) Injection of Scopolamine in Rats: Memory impairment was produced by I.P. administration of scopolamine (0.1 ml, 0.3 mg/kg), injected at a stat [23].

Statistical Analysis: The experimental values were expressed as Mean ± Standard error of the mean (SEM). Statistical analyses were performed by one-way ANOVA followed by the Bonferroni t-test.

RESULTS:

Effect on SOD Assay: After the administration of scopolamine significant decrease in the SOD level was observed in **Fig. 1**. In all treatment groups, the SOD level was found significant ($p<0.001$) increased in comparison to scopolamine. The effect of CEPs was found significant ($p<0.05$) in comparison with BME, TCE, and EAE. Further intercomparison between all CEP; CEP-4 showed a significant ($p<0.05$) effect in comparison to other CEP **Fig. 1**.

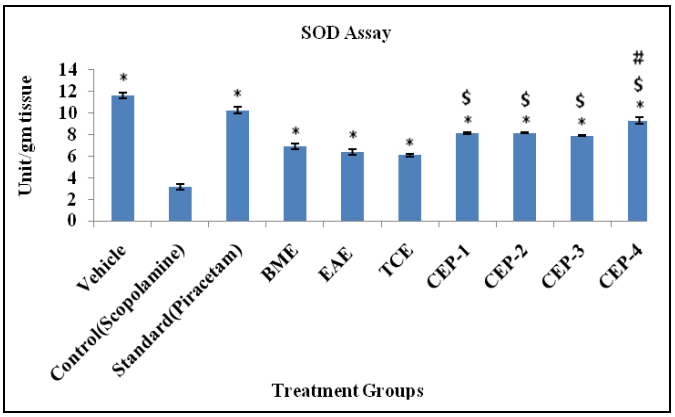

FIG. 1: EFFECT ON SOD ASSAY. Values are in Mean ± SEM (N=6). $^{*}p<0.001$ *vs.* Scopolamine, $^{\$}p<0.05$ *vs.* BME, TCE and EAE, $^{\#}p<0.05$ *vs.* CEP-1, CEP-2 and CEP-3

Effect on LPO Assay: After administration of scopolamine, significant enhanced in LPO level was observed in **Fig. 2**. In all treatment groups, the level of LPO was found significant ($p<0.05$) decreased in comparison to scopolamine.

Intercomparison between BME, TCE, and EAE; BME was found significant (p<0.001) in compare-son with TCE and EAE. The effect of CEPs was found significant (p<0.001) in comparison with BME, TCE, and EAE. Comparison between CEP-1, CEP-2, and CEP-3; CEP-1 was established significant (p<0.05) result in the comparison of CEP-3. Afterward, intercomparison between all CEP; CEP-4 showed a significant result (p<0.05, p<0.001) in comparison of CEP-2, CEP-3 respectively **Fig. 2**.

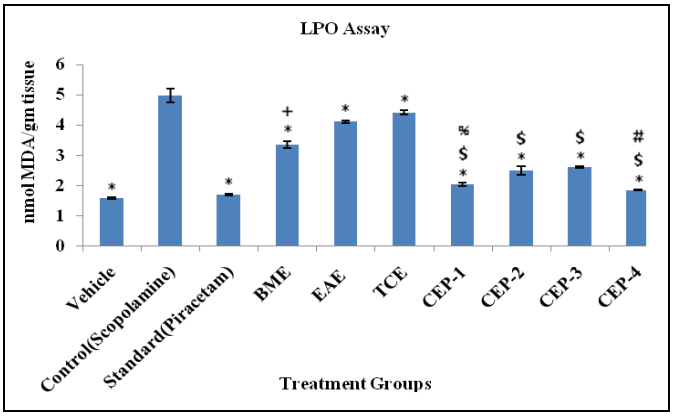

FIG. 2: EFFECT ON LPO ASSAY. Values are in Mean ± SEM (N=6). *p<0.05 *vs.* Scopolamine, +p<0.001 *vs.* TCE and EAE, $p<0.001 *vs.* BME, TCE and EAE, %p<0.05 *vs.* CEP-3, #p<0.05, p<0.001 *vs.* CEP-2 and CEP-3 respectively.

Effect on GSH Assay: After the administration of scopolamine significant decreased in GSH level was observed **Fig. 3**.

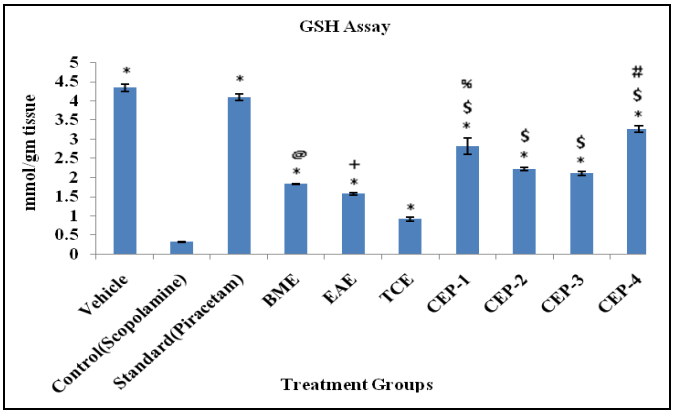

FIG. 3: EFFECT ON GSH ASSAY. Values are in Mean ± SEM (N=6). *p<0.001 *vs.* Scopolamine, @, +p<0.001 *vs.* TCE, $p<0.05 vs. BME, TCE and EAE, %p<0.05, p<0.001 *vs.* CEP-2 and CEP-3 respectively, #p<0.05, p<0.001, p<0.001 *vs.* CEP-1, CEP-2 and CEP-3 respectively.

In every treatment group; the level of GSH was increased significantly (p<0.001) in comparison to scopolamine. Intercomparison between BME, TCE, and EAE; BME and EAE showed a significant difference (p<0.001) in comparison to TCE. The result of CEPs was found significant (p<0.05) in comparison with BME, TCE, and EAE. Comparison between CEP-1, CEP-2, and CEP-3; CEP-1 was established significant (p<0.05, p<0.001) result in a comparison of CEP-2 and CEP-3, respectively. Afterward, intercomparison between all CEP; CEP-4 showed a significant result (p<0.05, p<0.001, p<0.001) in comparison of CEP-1, CEP-2, and CEP-3 respectively **Fig. 3**.

Effect on Catalase Assay: After the administration of scopolamine significant decrease in the Catalase level was observed in **Fig. 4**. In all treatment groups, the Catalase level was increased signify-cantly (p<0.001) in comparison to scopolamine. Intercomparison between BME, TCE, and EAE; BME was found significant (p<0.001) in com-parison to TCE and EAE. The result of CEPs was established significant (p<0.001) in comparison with BME, TCE, and EAE. Comparison between CEP-1, CEP-2, and CEP-3; CEP-1 was established significant (p<0.001) result in a comparison of CEP-3. Afterward, inter-comparison between all CEP; CEP-4 showed significant (p<0.05, p<0.001, p<0.001) effect in comparison of CEP-1, CEP-2, and CEP-3, respectively **Fig. 4**.

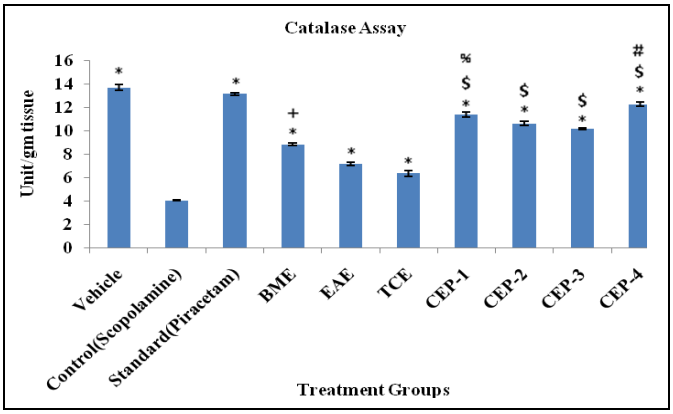

FIG. 4: EFFECT ON CATALASE ASSAY. Values are in Mean ± SEM (N=6). *p<0.001 *vs.* Scopolamine, +p<0.001 *vs.* TCE and EAE, $p<0.001 *vs.* BME, TCE and EAE, %p<0.001 *vs.* CEP-3, #p<0.05, p<0.001, p<0.001 *vs.* CEP-1, CEP-2 and CEP-3 respectively

Effect on AChE Assay: After the administration of scopolamine significant increase in AChE level was observed. **Fig. 5**. In all treatment groups, the level of AChE was significantly (p<0.001) decreased in comparison to scopolamine. Intercomparison between BME, TCE, and EAE; BME and EAE showed a significant difference (p<0.05) in

comparison to TCE. The result of CEPs was established significant ($p<0.001$) in comparison with BME, TCE, and EAE. Comparison between CEP-1, CEP-2, and CEP-3; CEP-1 was established significant ($p<0.05$) result in a comparison of CEP-3. Afterward, inter-comparison between all CEP; CEP-4 showed significant ($p<0.001$) effect in comparison to other CEP **Fig. 5**.

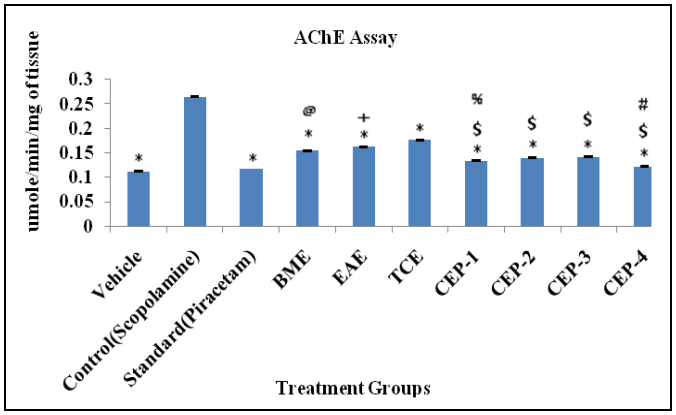

FIG. 5: EFFECT ON AChE ASSAY. Values are in Mean ± SEM (N=6). $^{*}p<0.001$ *vs.* Scopolamine, $^{@,\ +}p<0.05$ *vs.* TCE, $^{\$}p<0.001$ *vs.* BME, TCE and EAE, $^{\%}p<0.05$ *vs.* CEP-3, $^{\#}p<0.001$ *vs.* CEP-1, CEP-2 and CEP-3

DISCUSSION: In this research, evaluate the effect of BME, TCE, and EAE on various biochemical parameters were ascertained. On Scopolamine treatment; Scopolamine produces significant oxidative stress [16, 24, 25]. This increases oxidative stress, enhanced LPO and reduced GSH [25-27] and SOD level in the brain [26]. The relationship of oxidative strain with amnesia could be proved by the conclusion from the previous studies. Streptozotocin injection is given by Intracerebroventricular (ICV) and colchicine produced the destruction of cognition through an enhanced oxidative strain in rat brain [28, 29]. Scopolamine also significantly increased the AChE level in the brain [30, 31]. Scopolamine antagonizes the muscarinic (M1) receptor. It enhanced the AChE level and produced memory impairment in animals. It leads to reduce the ACh level at cholinergic synapses [24]. Also, Scopolamine increased AChE level by the decrease of hippocampal cholinergic neurons, as demonstrated by reduced choline acetyltransferase [30]. In the brain, Catalase extent was also decreased by Scopolamine treatment [31]. After administration of scopolamine; BME, TCE, EAE, and their CEPs were administered orally.

BME decreased AChE level [32,] and also TCE and EAE decreased AChE level in rat brain. In the case of GSH, SOD, LPO, and Catalase; BME, TCE, and EAE decreased the LPO level, but increase GSH, Catalase and SOD levels in rat brain.

In single extract treatment groups, BME was found to be most effective on LPO, Catalase, and AChE in comparison to TCE and EAE. Also, BME showed a significant difference in GSH levels in comparison to TCE. Even EAE was also having a significant effect on GSH and AChE levels in comparison to TCE. The CEPs was produced significant results on all these biochemical parameters in comparison with BME, TCE, and EAE.

Comparison between CEP-1, CEP-2, and CEP-3; CEP-1 showed significant results on LPO, Catalase, GSH, and AChE levels in comparison of CEP-3. Also, CEP-1 was established significant effects on GSH and AChE levels in comparison to CEP-2 treated groups of rats. These results showed that CEP-1 established significant effects in comparison with CEP-2 and CEP-3. Afterward, intercomparison between all CEP; CEP-4 showed significant results on SOD, Catalase, GSH, and AChE levels in comparison to other CEP. Also, CEP-4 has produced a significant effect on the LPO level in comparison with CEP-2, CEP-3. So, on the basis of results, probable synergic combinations of CEPs may improve cognition by the decrease of LPO and AChE level but increase SOD, GSH, and Catalase levels in rat's brains.

CONCLUSION: On the basis of significant results on biochemical parameters; probably it can say that the combinations of BME, TCE, and EAE provides a synergistic protective outcome by decreasing of LPO and AChE level but increase in SOD, GSH, and Catalase levels in rats brain. Further work may be carried out on a molecular level to know the detailed mechanism.

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CONFLICTS OF INTEREST: The authors do not have any conflicts of interest.

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