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IN VITRO EVALUATION OF ERK1/2 PATHWAY ACTIVATION BY *BACOPA MONNIERI* EXTRACT COMPARED WITH *BACOPA MONNIERI* EASI™ IN A DIFFERENTIATED SH-SY5Y CELL LINE

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ABSTRACT

Neuropathic injury is a pathological condition that occurs due to progressive neurodegeneration driven by oxidative stress, resulting in suppression of cellular survival cascade like Extracellular Signal-Regulated Kinase 1/2 (ERK1/2) pathway. The present study aims to evaluate the neuroprotective effects of *Bacopa monnieri* extracts in regulation of the ERK1/2 pathway in a differentiated human neuroblastoma SH-SY5Y cell model of hydrogen peroxide (H_2O_2) induced neuropathic injury. The cells were exposed to two-step differentiation process to achieve the mature neuronal cells characteristics followed by treatment with 100 µM H_2O_2 to induce neuronal damage and then administered with safe, non-cytotoxic concentrations (62.5 µg/mL and 125 µg/mL) of standard *Bacopa monnieri* extract and *Bacopa monnieri* EASI™ (Hyssop QA™) an Energized Active Supplement Ingredient, both prepared to comprise 50% bacopasides. Hydrogen peroxide exposure significantly reduced the ERK1/2 expression in the differentiated cells, however co-treatment with the samples successfully restored the ERK1/2 levels in concentration dependent manner. The energized form of *Bacopa monnieri* extract (*Bacopa monnieri* EASI ™, Greenspace) showed the maximum ERK1/2 activation, making it a highly optimized therapeutic candidate for mitigating oxidative-stress induced neuropathic damage.

KEYWORDS: *Bacopa monnieri*, Energized Active Supplement Ingredient, Neuropathic injury, Extracellular Signal-Regulated Kinase 1/2 (ERK1/2) pathway, oxidative stress, Neuroprotective.

INTRODUCTION

Neuropathic injury is a condition that arises from damage, compression or disease affecting the somatosensory nervous system. This condition leads to multiple complications at cellular and molecular levels resulting in persistent pain, sensory abnormalities and neuronal dysfunction thereby compromising the quality of life.[1] Oxidative stress is considered to be one of the primary causes of neuronal damage, which results from the imbalance between generation of reactive oxygen species (ROS) and the cellular antioxidant defense system. Accumulation of ROS disrupts mitochondrial function, damages nucleic acids, and proteins ultimately triggering neuronal apoptosis. The pathophysiology of neuropathic injury involves the combination of neuronal and nonneuronal interactions. Neuronal mechanism includes peripheral sensitization wherein sensory neurons undergo state of hyperexcitability recruiting the proinflammatory mediators (TNF-α, NGF, bradykinin, and serotonin) to the local tissue environment altering the expression of neuronal ion channels and central sensitization which results in enhanced excitatory transmission and diminished inhibition in the spinal cord dorsal horn.[2] Wallerian degeneration, a process where Schwann cells are activated to clear the axonal/myelin debris and neuroimmune interactions where immune and stromal cells act as active drivers of neuropathic pain covers the non-neuronal mechanism.[1,3]

The proinflammatory mediators that are released have specific binding site at specific receptors on the surface of neurons, Schwann cells, and surrounding immune cells. These cytokines trigger multiple signaling cascades, including the mitogen-activated protein kinase (MAPK), nuclear factor-kappa B (NF-κB), Janus kinase/signal transducer and activator of transcription (JAK/STAT), phosphoinositide 3-kinase/protein kinase B (PI3K/Akt), and nuclear factor erythroid 2-related factor 2 (Nrf2) pathways. Among these signaling networks, the MAPK pathway, particularly the ERK1/2 signaling cascade, acts as one of the principal regulators of oxidative stress-induced neuronal injury.[4,5] Persistent ERK1/2 activation sustains neuroinflammation by upregulating pro-inflammatory mediators and promotes oxidative stress, neuronal hyperexcitability, central sensitization, and maladaptive synaptic plasticity through the regulation of BDNF, CREB, and c-Fos.[6]

Bacopa monnieri (Brahmi) extract is extensively studied for brain health. Research highlights improvements in logical memory, processing speed, and a reduction in the rate of memory loss than active learning. It is also recognized for its antioxidant and anxiolytic (anti-anxiety) properties Its neurorestorative properties serving as potent solution for neuropathic injury is also reported.[7,8] *Bacopa monnieri* shows potential neuroprotective effect by preventing oxidative cell death through MEK/ERK1/2 and PI3K/Akt pathway activation. Bioactive compounds, the triterpenoid saponins specifically Bacopaside I, Bacopaside II, and Bacoside A trigger these signaling pathways.[9] Therefore, the present study aimed to evaluate the activation of the ERK1/2 signaling pathway by standard *Bacopa monnieri* extract and *Bacopa monnieri* extract EASI™ (Hyssop QA™;Hirehal Greenspace Herbs) in a differentiated SH-SY5Y cell model of hydrogen peroxide-induced neuropathic injury.

In *In-Vitro* conditions hydrogen peroxide (H_2O_2) is widely used to stimulate oxidative stress-induced neuronal injury, triggering the rapid production of ROS and inducing apoptosis of neural cells. The human neuroblastoma SH-SY5Y cell line is highly validated as *In-Vitro* model for investigating neurodegeneration and cellular injury, especially when differentiated using agents such as Retinoic acid along with Brain Derived Neurotrophic Factor (BDNF).[10,11] The differentiated SH-SY5Y cells exhibit vigorous activation of MAPK signaling pathway, making them ideal for investigating ERK 1/2 -mediated neuroprotective mechanisms.

MATERIALS AND METHODS

Chemicals and reagents

The human neuroblastoma cells SH-SY5Y used for the study were procured from the National Centre for Cell Science (NCCS), Pune, India. The major components required for cell culture like Ham's F12 medium, Dulbecco's Phosphate Buffer Saline (DPBS), Trypsin-EDTA antibiotics (Penicillin, Streptomycin, Amphotericin B), and MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) reagent, were sourced from HiMedia, India. Fetal Bovine Serum (FBS) was procured from Gibco, USA, and Dimethyl Sulfoxide (DMSO) from SRL, India. For cellular differentiation and injury modeling, Retinoic Acid was obtained from Ottokemi, India, and Brain-Derived Neurotrophic Factor (BDNF) from Elabscience, USA. The ERK1/2 pathway ELISA kit was acquired from BT-lab, China.

Test samples

The samples evaluated in the current study were *Bacopa monnieri* extract and *Bacopa monnieri* EASI™ which is an energized active supplement ingredient developed by Hirehal Greenspace herbs, Bengaluru, India using a proprietary energization process. The preparation of *Bacopa monnieri* EASI™ involves controlled exposure to low-intensity pulsed electromagnetic and acoustic fields (Radiation Frequency Range:80MHz to 1 GHz at a field strength of 10 V/m; Pulsed Electromagnetic Field: 60 A/m [75 μT] at 50 Hz).

Preparation of test solution

Stock solution of test samples with concentration 10mg/ml was prepared by dissolving the samples in 100 µL of DMSO and 900 µL of Ham's F12 medium supplemented with 2% inactivated FBS. For cytotoxicity screening, serial two-fold dilutions were carried out to create a concentration gradient ranging from 1000 µg/mL down to 7.8 µg/mL. Non-toxic concentrations were further prepared from the stock solution.

Cell cytotoxicity determination by MTT assay

To determine the safe concentration of the drug, MTT assay was carried out. The SH-SY5Y cell suspension was adjusted to 100,000 cells/mL, using Ham's F12 media containing 10% FBS and 0.1 mL of these diluted cells suspension was seeded into each well of a 96-well microtiter plate. The cells were incubated for 24-hour allowing for partial monolayer formation, the supernatant was then discarded, and cells were washed with DPBS.

The cells were treated with varying concentrations of the test samples (7.8 to 1000 µg/mL) and incubated for another 24 hours at 37°C in 5% CO_2, with untreated cells serving as the experimental control. Post-treatment, the medium was removed, and 100 µL of MTT reagent (diluted with DPBS) was added to each well for a 3-hour incubation period. The supernatant was subsequently removed, and 100 µL of DMSO was added to solubilize the formazan crystals. Absorbance was quantified at a wavelength of 570 nm using an automated microplate reader (Biotek, USA) to determine the percentage of cell viability.

Cell culture and neuronal differentiation

SH-SY5Y cells stock solution was cultured in 25 cm^2 culture flasks using Ham's F12 media supplemented with 2% inactivated fetal bovine serum (FBS), Penicillin (100 IU/mL), Streptomycin (100 µg/mL) and Amphotericin B

(5 µg/mL) in a humidified atmosphere of 5% CO_2 at 37°C until confluent. Dissociation of the adherent cells were carried out by trypsinization using 0.25% trypsin, 0.02% EDTA in Hanks balanced salt solution. Two-step differentiation process was carried to achieve the neuronal characteristics for the neuropathic injury model. First differentiation was induced by 10 µM retinoic acid, by seeding cells on 6-well plates and culturing for 5–7 days in medium containing 2 % FBS. Second round of differentiation was carried with 50 ng/mL Brain-Derived Neurotrophic Factor (BDNF) in 1% FBS for 24 hours to enhance neurite growth.

Induction of Neuropathic Injury

The differentiated cell was exposed to hydrogen peroxide (100 µM) to stimulate an oxidative stress environment mimicking a neuropathic-like injury, parallelly the test samples of safe concentrations which gave 80% cell viability that is 62.5 and 125 µg/mL, were added and incubated for 24 hours. After the incubation, cells were washed with ice cold PBS and treated with lysis buffer (100–200 µL per well), the cells were scraped and transferred to centrifuge and further incubated on ice for 30mins. The lysate was then centrifuged 12,000–14,000 × g for 15 min at 4°C. supernatant was collected in a new tube. ERK1/2 pathway analysis was performed according to the manufacturer's protocol provided with the ELISA kit.

RESULTS

***In Vitro* Cytotoxicity**

The quantitative determination of biocompatibility of the *Bacopa monnieri* samples - *Bacopa monnieri* extract and *Bacopa monnieri* EASI™ was carried out using MTT assay on SH-SY5Y cell line. The samples demonstrated a dose-dependent effect on cell viability, with significant cytotoxicity observed at the highest concentration (1000 µg/ml), yielding cell viabilities of 10.19 ± 0.29% and 9.51 ± 0.08%, respectively.

The half-maximal cytotoxicity concentrations (CTC_{50}) determined for *Bacopa monnieri* extract and *Bacopa monnieri* EASI™ was 405.65 µg/ml, 383.97 µg/ml respectively. On the other hand, at lowest concentration 7.8 µg/ml, the cell viability observed was 99.03 ± 0.79% and 102.27 ± 3.91 indicating that the test items did not exhibit cytotoxicity at this dose. Based on these results the concentration of the samples that exhibited 80% cell viability was chosen for performing the study, which is specifically 62.5 µg/ml and 125 µg/ml.

Table 1: In vitro cytotoxicity of test items in terms of percentage cell viability against SHSY-5Y cell line by MTT assay.

Concentration (µg/mL)	*Bacopa monnieri* extract (% Cell Viability)	*Bacopa monnieri* EASI™ (% Cell Viability)
1000	10.19 ± 0.29	9.51 ± 0.08
500	11.65 ± 0.68	10.52 ± 0.32
250	67.73 ± 2.02	51.07 ± 2.06
125	80.32 ± 0.87	81.94 ± 0.42
62.5	86.99 ± 0.55	86.34 ± 0.88
31.25	90.74 ± 1.05	91.72 ± 0.88
15.625	96.08 ± 1.21	96.73 ± 0.30
7.8	99.03 ± 0.79	102.27 ± 3.91
CTC_{50} (µg/mL)	**405.65**	**383.97**

Values are expressed as Mean ± SD

Cell control | **1000 µg/mL** | **125 µg/mL**

Figure 1: Cell line images of *in vitro* cytotoxicity of *Bacopa monnieri* extract in terms of percentage cell toxicity at 24 hrs. against SHSY-5Y cell line.

Cell control	1000 µg/ml	125 µg/ml

Figure 2: Cell line images of *in vitro* cytotoxicity of *Bacopa monnieri* EASI™ in terms of percentage cell toxicity at 24 hrs. against SHSY-5Y cell line.

Upregulation of ERK1/2 Activation

The differentiation of the SH -SY5Y cells were confirmed by comparing the ERK1/2 levels before and after cellular differentiation. Undifferentiated cells exhibited low baseline ERK1/2 expression that is 125.50 ± 5.0 ng/L, treatment with retinoic acid and Brain Derived Neurotrophic Factor (BDNF) increased the basal levels to 920.50 ± 15.0 ng/L.

Exposure to H_2 O_2 mediated oxidative stress for simulating neuropathic injury resulted in drop in the level of ERK 1/2 to 638.00 ± 7.5 ng/L. Treatment of the injured differentiated SH-SY5Y cells with the *Bacopa monnieri* samples- *Bacopa monnieri* extract and *Bacopa monnieri* EASI™ elevated the ERK1/2 levels to 1236.75 ± 46.25 ng/L and 1364.25 ± 48.75 ng/L, respectively, at 125 µg/mL concentration. This clearly indicates the effectiveness of the Bacopa samples in counteracting the oxidative stress induced neuronal dysfunction. The *Bacopa monnieri* EASI™ is relatively more efficient compared to the *Bacopa monnieri* extract, showing around 21.3% greater restoration in ERK1/2 levels in H_2O_2 induced cells.

Table 2: The quantitative level of ERK1/2 by ELISA.

Sl. No.	Treatment Group	Concentration	ERK1/2 (ng/L)
1	Cell Control	—	125.50 ± 5.00
2	Differentiated Cell Control	—	920.50 ± 15.00
3	H_2O_2	100 µM	638.00 ± 7.50
4	*Bacopa monnieri* extract	125 µg/mL	1236.75 ± 46.25
		62.5 µg/mL	1174.25 ± 16.25
5	*Bacopa monnieri* EASI™	125 µg/mL	1364.25 ± 48.75
		62.5 µg/mL	1264.25 ± 73.75

Values are expressed as Mean ± SD

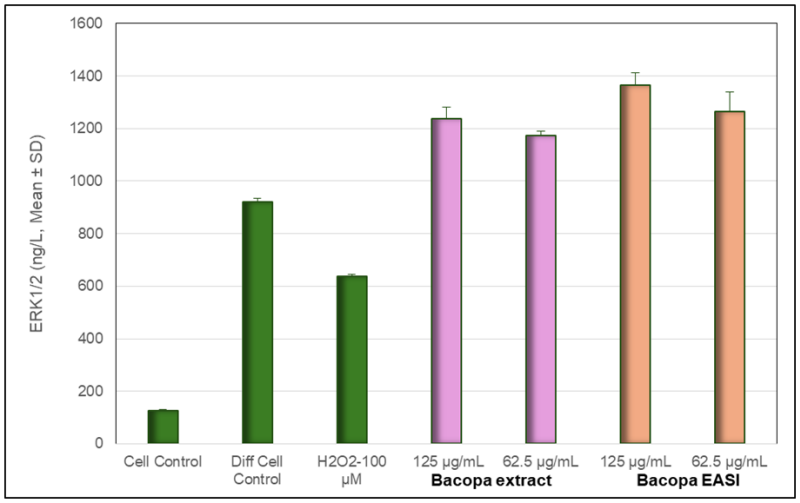

Figure 3: Graphical representation of level of ERK1/2 production in extracts differentiated SHSY5Y cells.

DISCUSSION

Bacopa monnieri has long been used for memory enhancement, mental health and as an antioxidant because of its multiple therapeutic benefits in curing neurodegenerative diseases, it is referred to as Medhya Rasayana (Brain tonic) in ayurveda.[12] This herb is constantly investigated for its potential to treat many medical conditions such as gastric ulcers and Alzheimer's disease.[7]

Bacopa monnieri has shown potent neuroprotective activity against neuronal injury which occurs due to destruction or degeneration of nerve cells in the brain, spinal cord and peripheral nervous system. The neuronal injury leads to severe condition like neuronal atrophy, synaptic loss, and in severe cases, neuronal and glial cell death.[13] The neuroprotective action of *Bacopa monnieri* include targeting multiple pathways involved in neuronal injury, such as glutamate receptors activation (NMDA, AMPA), GABA receptors inhibition, enhancement of BDNF-mediated neurotrophic signaling, and activation of the MAPK/ERK1/2–CREB pathway.

Thereby, it leads to reduction of proinflammatory cytokines such as TNF-α, IL-6, IL-17A, and CCL5, suppression of NF-κB activation, enhancement in CREB phosphorylation, and stimulation of kinase activity involved in neuronal repair and synaptic plasticity.[8,14] *Bacopa monnieri* not only protects neuron but also promotes neuronal regeneration as proven by an *In-Vitro* study conducted on Neuronal Stem Cells (NSC) derived from rat's brain opening the possibility for its use in treating neuronal injury by stimulating structural repair of damaged neuron.[15]

The current study validates the neuroprotective capabilities of *Bacopa monnieri*, emphasizing its ability to reverse the suppression of ERK1/2 pathway (Extracellular signal-regulated kinase 1/2 pathway) in a differentiated SH-SY5Y cell model of hydrogen peroxide-induced neuropathic injury. Both the *Bacopa monnieri* extracts samples restored the ERK1/2 levels more than that of differentiated control SH-SY5Y cell.

However, the efficacy of *Bacopa monnieri* EASI™ in activation or restoration of ERK 1/2 was found to be 21.31% higher compared to the *Bacopa monnieri* extract. This can be attributed to its unique formulation strategy via proprietary energization process, classifying it as Energized Active Supplement Ingredient (EASI™). The energization process may have an influence in bioavailability enhancement of bacopa further allowing for maximal cellular uptake and profound intracellular kinase stimulation. The activation of ERK1/2 is clinically significant because it is the central regulator of the neuronal differentiation, synaptic plasticity, cell survival and BDNF-associated neurotrophic signaling. The result of the current study is comparable with previous study conducted using *Bacopa monnieri* extract which showed an elevation in ERK1/2 levels up to 86.97% in *tert*-Butyl hydroperoxide (TBHP)-induced oxidative damage in SH-SY5Y cells at the concentration of 250 µg/mL, which is higher than the concentration used in the current study.[16]

While the current study aligns with the previous research work on *Bacopa monnieri's* neuroprotective mechanism by ERK1/2 pathway activation to mitigate oxidative stress and prevent neuronal apoptosis, it exhibits more pharmaceutically relevant advancements such as usage of two-step differentiated neuroblastoma cells instead of undifferentiated cells.[17–19] Several other medicinal herbs such as *Achyranthes bidentata* and *Codonopsis pilosula* also demonstrate ERK1/2 activation as mechanism for neural regrowth by promoting the neural outgrowth and schwann cell migration.[20] Unlike them the *Bacopa monnieri* EASI™ utilizes the ERK1/2 activation as a vigorous survival and anti-apoptotic signal to rescue the neuron for the oxidative stress induced damage of the neuron. Further clinical trials and pharmacokinetic studies may be essential to prove the efficacy of *Bacopa monnieri* EASI™ in humans and to determine if ERK1/2 activation correlates with improvements in neuronal recovery and cognitive function.

CONCLUSION

The current study demonstrated that *Bacopa monnieri* serves as efficient neuroprotective agent by restoring the differentiated SH-SY5Y cells from hydrogen peroxide induced neuropathic injury. The *Bacopa monnieri* EASI™ formulation showed the relatively higher restoration of ERK1/2 levels than the *Bacopa monnieri* extract, proving its effectiveness in combating neuronal injury. This can be attributed to the proprietary energization process utilized for its synthesis. The robust kinase stimulation translates directly into a profound anti-apoptotic and neurorescue effect, mitigating the oxidative tissue damage and neuronal dysfunction characteristic of neuropathic injuries.

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DISCLAIMER

Hyssop QA™ is the tradename for *Bacopa monnieri* an energized supplement ingredient developed by Hirehal Greenspace herbs. Complete details of the energization process are proprietary. However, additional information can be made available upon reasonable request.

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CONFLICT OF INTEREST

The authors declare no competing interests.

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