Faradarmani Consciousness Field Suppresses Alzheimer's Disease Development in both *in vitro* and *in vivo* Models of the Disease

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ABSTRACT

Alzheimer's Disease (AD) is one of the most common causes of dementia, imposing large financial and psychological burdens on nations worldwide. Thus, we direly need new treatment strategies or drugs for this disease. The aim of this study is to investigate the effects of a novel non-pharmacological method in the treatment of Alzheimer's disease, based on employing Taheri Consciousness Fields. These fields function at the level of cellular and molecular processes. In this study, the effects of Faradarmani Consciousness Field (CF) on the AD mouse model (in vivo) and human neuron cell line in vitro were investigated. In this study, we established a human neuron cell culture as well as a traumatic brain injury (TBI) mouse model. We then measured changes in amyloidopathy, tau protein content, microtubule assembly, neuronal cell survival, and finally behavior of TBI mice in Elevated Plus Maze under treatment of the Faradarmani CF. According to the results, treatment of human neural cells and a mouse model of Alzheimer's disease by the Faradarmani CF leads to complete survival of neural cell models and elimination of amyloidopathy and tau protein, and remarkable behavioral improvement of the treated TBI mice model in the elevated plus-maze. Based on the results, Faradarmani CF treatment suppresses AD development in the laboratory models. In this regard, conducting a human clinical study with the aim of introducing a new global complementary and alternative medicine in AD treatment is highly recommended.

INTRODUCTION

Alzheimer's disease (AD)-related dementia is a critical public health issue in industrialized and non-industrialized nations [1]. According to the World Health Organization, AD and other forms of dementia ranked as the 7th leading cause of death in 2019, and globally 65% of deaths are women [2]. There are many diseases that lead to dementia, but AD is the most common dementing illness in the elderly, and it has been estimated that AD contributes to approximately 60-77% of cases [3]. It takes many years before symptoms of AD emerge from its onset. Therefore, preclinical studies of AD would provide a critical opportunity for developing therapeutic interventions [4]. Impaired recent memory is an initial symptom of AD and is sometimes associated with other cognitive deficits, like changes in attention and problem-solving abilities. As dementia progresses, other cognitive deficits, such as language dysfunction, visuospatial difficulty, and loss of insight, are frequently apparent [5]. In individuals, these cognitive impairments are usually accompanied by changes in personality, behavior, and uncharacteristic mood fluctuations such as agitation, impaired motivation, initiative, apathy, social withdrawal, etc. [6]. In order to manage the behavioral symptoms, psychological interventions, alternative therapies, as well as antidepressant treatments are employed [7].

Pathologically, AD is defined by the progressive accumulation of β-amyloid (Aβ) plaques and neurofibrillary tangles composed of hyperphosphorylated tau protein [8, 9, 10]. Accumulation of Aβ in the brain is the primary factor driving AD pathogenesis [11]. Therefore, mechanistic clearance of Aβ peptides becomes a potential target for drug development for AD [12]. Multiple enzymes are capable of degrading Aβ like neprilysin and insulin-degrading enzymes. Conversely, reductions in the activity of Aβ-degrading enzymes could contribute to AD itself [13,14]. The cysteine protease cathepsin B (Cat B) reduces levels of Aβ peptides, especially Aβ 1-42, through proteolytic cleavage [15]. In addition, it has been reported that the peripheral system is potent in clearing brain Aβ and preventing AD pathogenesis [16]. As it was mentioned before, tau accumulation in senile plaques is another hallmark of AD. In healthy subjects, tau proteins are abundant microtubule-associated proteins that have roles in maintaining the stability of microtubules in axons [17]. Microtubules maintain the architectural support for the elongated shape of growing axons and the development of neurons [18,19]. However, tau hyperphosphorylation could lead to the detachment of tau from microtubules and lead to the formation of Neurofibrillary tangles (NFTs) [20,21]

The risk of dementia is highest in people with a history of Traumatic Brain Injury (TBI) [22]. A study found that TBI history is associated with an earlier onset of AD-related cognitive decline [23,24]. They reported that before tauopathy, cis p-tau increase in sport- and military-related TBI in humans, also reflected in mice and stressed neurons. In that study, treating TBI mice with cis antibodies prevented the development of widespread tauopathy. Thus, cis p-tau could be an early biomarker for diagnosis and therapy. In addition, the cis pT231-tau isoform was introduced as a central mediator in TBI and neurodegeneration. Therefore, targeting cis pT231-tau could be a good candidate for immunotherapy for several tauopathy disorders, including AD [25].

The loss of cholinergic function in the central nervous system contributes to cognitive decline

and dementia [26], which has been the target of anti-AD drugs [27]. Pharmacologic treatments temporarily improve symptoms but can't slow or stop the degeneration of neurons [28].

The nature of consciousness and its place in science has received much attention in the current century. Many philosophical and scientific theories have been proposed in this area. In the1980s, Mohammad Ali Taheri introduced novel fields with a non-material/non-energetic nature named Taheri-Consciousness Fields (TCFs). In this perspective, T-Consciousness is one of the three existing elements of the universe apart from matter and energy. According to this theory, there are various TCFs with different functions, which are the subcategories of a networked universal internet called the Cosmic Consciousness Network (CCN). The major difference between the theory of TCFs and other theoretical concepts about consciousness is related to the practical application of the TCFs. TCFs can be applied to all living and non-living creatures, including plants, animals, microorganisms, materials, etc.

Mohammad Ali Taheri, the founder of Erfan Keyhani Halqeh, a school of thought, introduced a new science in 2020 as a branch of this school. He coined the term Sciencefact for this new science because it utilizes scientific investigations to prove the existence of T-Consciousness as an irrefutable phenomenon and a fact. Although science focuses solely on the study of matter and energy and Sciencefact, by contrast, explores the effects of the [non-material/non-energetic] TCFs, Sciencefact has provided a common ground between the two by conducting reproducible laboratory experiments in various scientific fields, and it has used the scientific approach in proving TCFs.

The influence of the TCFs begins with the Connection between CCN as the Whole Taheri Consciousness of the universe and the subjects of study as a part. This Connection called "Ettesal" is established by a Faradarmangar's mind (a certified and trained individual who has been entrusted with the TCFs). The human mind has an intermediary role (Announcer) which plays a part by fleeting attention to the subject of study and then the main achievement obtained as a result of the effects of the TCFs. These fields cannot be directly measured by science, but it is possible to investigate their effects on various subjects through reproducible laboratory experiments [29].

The research methodology in the study of T-Consciousness has been founded on the process of Assumption, Argument, and Proof, in which the basic Assumption is: The Cosmos was formed by a third element called T-Consciousness that is different from matter and energy.

The Argument: The existence of TCFs can be demonstrated by its effects on matter and energy (e.g., humans, animals, plants, microorganisms, cells, materials, etc.)

The Proof: is the scientific verification of the effects of TCFs on matter and energy (according to the Argument) through various reproducible scientific experiments.

Accordingly, to investigate and verify the existence, effects, and mechanisms of TCFs, the following five research phases (Phases 0 through 4), and the aims of each phase are outlined below.

Phase-0 studies aim to prove the existence of TCFs by observing their effects. The nature of T-Consciousness and what it is will not be addressed in this phase. Phase-1 explores the varied effects of different TCFs. Phase-2 examines the reason behind the varied effects of these fields. Phase-3 investigates the mechanism of TCFs ef-

fects on matter and energy. Finally, Phase-4 draws significant conclusions, particularly with regard to the mind and memory of matter and their relation to the T-Consciousness, etc. In previous investigations, we examined the effects of Faradarmani CF on the spatial memory and avoidance behavior of a rat model of AD [30]. The results showed that Faradarmani CF improved the learning and memory impairment induced by scopolamine in rats. In addition, remarkable restorative-enhancing effects were observed in their passive avoidance behavior. Changes in cancer cell growth [31], investigations on the electrical activity of the brain during Faradarmani CF Connection in Faradarmangar [32], and alleviative effects of Faradarmani CF on the wheat plant under salt stress [33] are other observations that have been used this method. The aim of the present study was to investigate the influence of the Faradarmani CF on AD cells and animal models.

MATERIALS AND METHODS

Faradarmani CF Application

The use of Faradarmani CF (and other TCFs) is possible by registering a request through the announcement section on the website for the research center of TCF (www.consmointel.com). For this purpose, each researcher, based on desired time and place and by introducing the generalities of the experiments, can allocate TCFs treatment for the subject of study. The Research and Development Department of the Sciencefact research center is staffed to allocate the TCFs treatments for studies under the direct supervision of Mr. Taheri, 24 hours a day, free of charge.

In this study, the FCF treatment was assigned to cellular and animal models of AD on a daily ba-

sis and for the entire duration of the experiment from the time the model was created to the ends of related assays. All executive processes and primary analyses of this study have been done by double-blind experts unfamiliar with the theory of how to use the Faradarmani CF.

Antibodies

The primary antibodies were cis pT231-tau mAbs (gift from KPL, Harvard), β-actin mAb (Sigma, St Louis, MO), Caspase-3 (Abcam, Cambridge, MA), Tau5 (Biosource Camarillo, CA), and oligomeric tau T22 polyclonal antibodies (EMD Millipore, Billerica, MA).

Generating human embryonic neural progenitor cells (hESC-NPCs)

Induced Pluripotent Stem Cells (iPSCs) from two late-onset AD patients and two healthy control age-matched subjects were donated from Royan Cell Bank, which were in turn generated from fibroblasts employing Yamanaka factors. The iPSCs were then differentiated to NPCs. The neuro-induction medium composed of DMEM/F12 medium (Gibco, 21331020) supplemented with 5% knockout serum (Gibco, 10828028), Glutamax (Gibco, 25030081), MEM-NEAA (Gibco, 11140050), 1% N2 (Gibco, 17502048), 3 µM SB431242 (Cyman, 13031), 5 µM Dorsomorphin (Stemgent, 04-0024), 3 µM CHIR99021 (Stemgent, 04-0004-10), and 0.5 µM SAG (Cayman, 912545-86-9). The rosette form structures were manually picked up after seven days of the induction. The NPCs were then re-plated on 1mg/mL laminin and 15mg/mL poly-l-ornithine–coated tissue culture dishes (Sigma-Aldrich) in the neural expansion medium included DMEM-F12 medium supplemented with 5% knockout serum replacement (KOSR), 1%

non-essential amino acid, 2 mM L-glutamine, 2% N2 (all from Invitrogen), 0.1 mM β-mercaptoethanol, 20 ng/ml basic fibroblast growth factor (bFGF, Royan Institute), 20 ng/ml additional epidermal growth factor (EGF, Sigma-Aldrich) and 0.2 mM ascorbic acid (Sigma-Aldrich). The medium was changed every other day for seven days. After the differentiation of neurons, the expansion medium was replaced with a differentiation medium by eliminating growth factors (bFGF) and adding hBDNF (PR-1113), hGDNF (pr-1107), and cAMP (Sigma, D0627) for 35 days. The differentiation process was tracked and confirmed with SOX2, PAX6, NESTIN, NCAM, GFAP, MAP2, GAD65, GABA, and S100 staining.

Nutritional starvation stress

We normally changed the culture medium every four days according to our differentiation protocol. However, in order to starve the cells, we didn't change the medium for additional 96 hours. We initially confirmed the tauopathy process timeline in our cell culture model and found prominent tauopathy after 96 hours of starvation; consistent with previous findings [24].

Live and dead cell assay

Fluorescein diacetate (FDA) and Propidium Iodide (PI) double staining were used for cell viability assessment. Aliquots of 20 μl of FDA stock solution and 50 μl of PI stock solution were diluted in 10 ml PBS [34]. The cells were initially washed with cold PBS and then the FDA/PI solution was added to the cells and was inspected by a fluorescent microscope after 5 min incubation at room temperature with the solution.

Cell staining

Cells were harvested at different time points and fixed with 4% paraformaldehyde (PFA) for 20 minutes in the dark.

They were either directly subjected to microscopic observations or stained with antibodies. In case of further processing, they were permeabilized with 0.2 % TritonX 100 for 20 minutes and stained with primary antibodies overnight at 4 ºC. They were then incubated with Alexa Fluor 488 or 568 conjugated secondary an-tibodies for one hour at room temperature. The samples were visualized with a Zeiss confocal mi-croscope (LSM 800).

Traumatic brain injury

The mouse TBI model was used as previous-ly described [24]. Briefly, 18 male C57BL/6 mice (2–3 months old), obtained from Royan Animal Facility, were randomized to undergo injury or sham-injury. The mice were anaesthetized for 45 seconds using 4% isoflurane in a 70:30 mixture of air: oxygen. Anaesthetized mice were placed on a delicate task wiper (Kimwipe, Kimberly-Clark, Ir-ving, TX) and positioned such that the head was placed directly under a hollow guide tube. The mouse's tail was grasped. A 54-gram metal bolt was used to deliver an impact to the dorsal aspect of the skull, resulting in rotational acceleration of the head through the Kimwipe. Mice underwent a single severe injury (ssTBI, 60-inch height). Sham-injured mice underwent anaesthesia but not concussive injury. All mice were recovered in room air. Anaesthesia exposure for each mouse was strictly controlled for 45 seconds. Subsequent behavioral and histopathological testing was con-ducted in a blinded manner.

Immunohistochemistry

Mice were perfused with 4% PFA at various time points after injury and brains were harvested for further analysis.

Serial 8 μm coronal sections from sham and injured brains were cut on a cry-ostat (Leica) and were collected on slides.

Immunoblotting analysis

Immunoblotting analysis was carried out as described [24]. Briefly, brain tissues or cultured cells were lysed in RIPA buffer (50 mM Tris-HCl, pH 7.4, 150 mM NaCl, 2 mM EDTA, 1% NP 40, 0.1% SDS, 0.5% Na-deoxycholate, 50 mM NaF) contain-ing protease and phosphatase inhibitors and then mixed with the SDS sample buffer and loaded onto a gel after boiling. The proteins were resolved by polyacrylamide gel electrophoresis and trans-ferred to PVDF membrane and block stained with 5% milk in TBST (10 mM Tris-HCl pH 7.6, 150 mM NaCl, 0.1% Tween 20) for 1 hour. The membrane was then incubated with primary antibodies in 5% milk in TBST overnight at 4 ºC. Then, the mem-branes were incubated with HRP-conjugated sec-ondary antibody in 5% milk in TBST. The signals were detected using a chemiluminescence reagent (Perkin Elmer, San Jose, CA). The membranes were washed six times with TBST after each step. Im-munoblotting results were quantified using Quan-tity One from BioRad.

Immunostaining analysis

Immunofluorescence staining of mice brains was done essentially as described [24]. After treatment with 0.3% hydrogen peroxide, slides were briefly boiled in 10 mM sodium citrate, pH 6.0, for antigen retrieval. The sections were incu-bated with primary antibodies overnight at 4 ºC. Then, the sections were incubated with an Alexa Fluor 488 or 568 conjugated secondary antibod-ies (Jackson ImmunoResearch, West Grove, PA)

for one hour at room temperature. The sections were washed three times with TBS after each step. The sections were visualized with a Zeiss confocal microscope (LSM 800). The gain of the confocal laser was set at the level where there are no flu-orescence signals, including autofluorescence, in sections without primary antibody but with sec-ondary antibody.

Elevated plus-maze

The elevated plus maze was used to assess anx-iety two months after injury and carried out as de-scribed. In brief, the maze consists of two closed and open arms (30 × 5 cm) extended out oppo-site each other from a central platform (decision zone). The entire apparatus is raised 100 cm above the floor. Mice were placed on the center platform of the maze, facing a closed arm, and allowed to explore the apparatus for 5 min. The maze was thoroughly cleaned between subjects with a weak ethanol solution. We track-recorded the total time spent in the open center (decision zone), the two closed arms, and the two open arms. The percent time spent in the open arms is presented as a sur-rogate measure of risk-taking behavior.

STATISTICAL ANALYSIS

Experiments were repeated at least three times. We didn't exclude any animals or samples from the analysis. For all behavioral tests, exper-imenters were blinded to injury and treatment status. The Hitmap data was collected with Python & OpenCV. All data are presented as mean ± stand-ard deviation (SD) followed by a two-way analysis of variance and multiple comparisons with a 95% confidence interval, and significant p-values less than 0.05. All other analyses were carried out with GraphPad Prism version 8.

RESULTS

In vitro assessments:

At this stage, the effect of the Faradarmani CF on tauopathy and amyloidopathy, as well as sur-vival and accumulation of microtubules structure of neurons under the aging stress has been inves-tigated. This part of the study was conducted in three groups: A, B and C. Group A is the control group (with no treatments), group B is a sham group (or a positive control group in which neu-ron cells are under aging stress without affecting the Faradarmani CF) and group C, is the sample group in which the effect of the Faradarmani CF on the parameters related to the Alzheimer's cel-lular model investigated in comparison with other groups.

Immunoflourescent intensity of cis p-tau pro-tein in three samples of the present study is shown in Figure 1. As can be seen in Figure 1, while there was a profound neurotoxic cis p-tau increase in the stressed-out culture neurons, the Faradarmani CF treatment eliminated p-tau from the cells. More-over, as can be seen in Figure 2, aging stress-in-duced significant amyloidopathy in cultured neu-rons and Faradarmani CF treatment blocked the pathogenic process.

On the other hand, changes in the survival of neurons and structural strength of the microtu-bules under aging stress in different cell groups of this study are shown in Figure 3. As can be seen in this figure, Faradarmani CF treatment prominent-ly suppressed neurodegeneration in cultured neu-rons upon aging stress. Also, there was a profound microtubule disruption in the stressed-out neu-rons, but the Faradarmani CF treatment healed the phenomena confirmed by immunofluorescence staining of the cells.
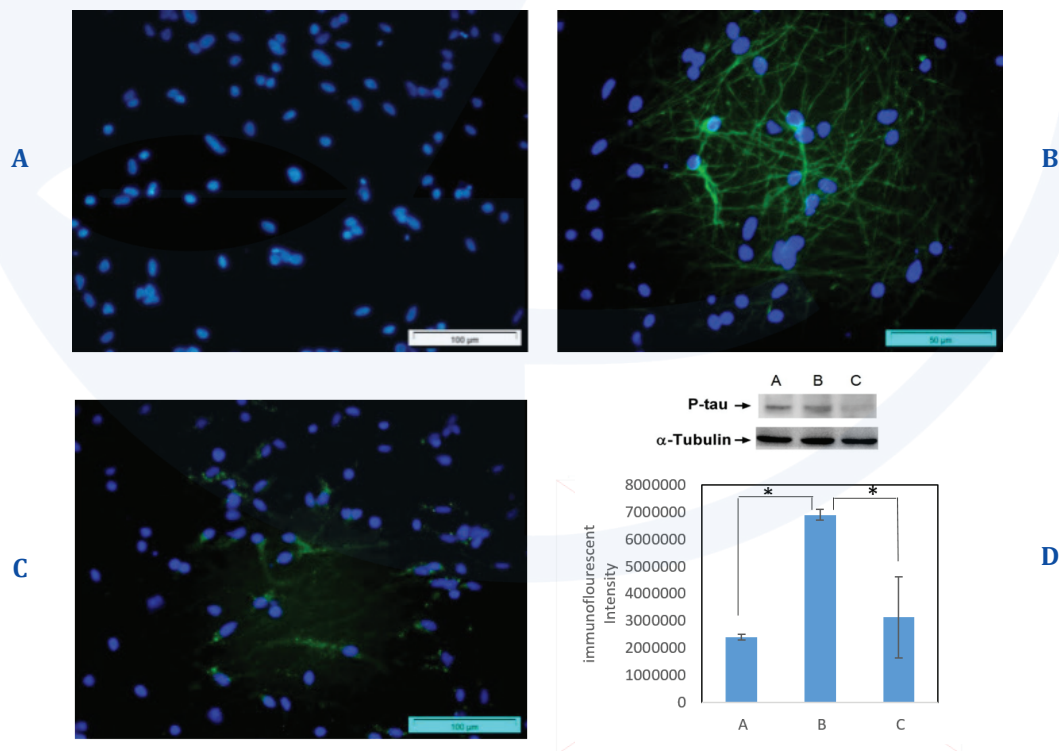

Figure 1. Immunostained cultured neurons with pathogenic p-tau under (A) Control, (B) Nutritional starved and (C) Stressed out neurons treated with Faradarmani CF. (D) Quantification representation of Immunoflourescent intensity of A, B, and C (*: p-value < 0.00l, **: p-value<0.0l; difference between A and C is not significant).
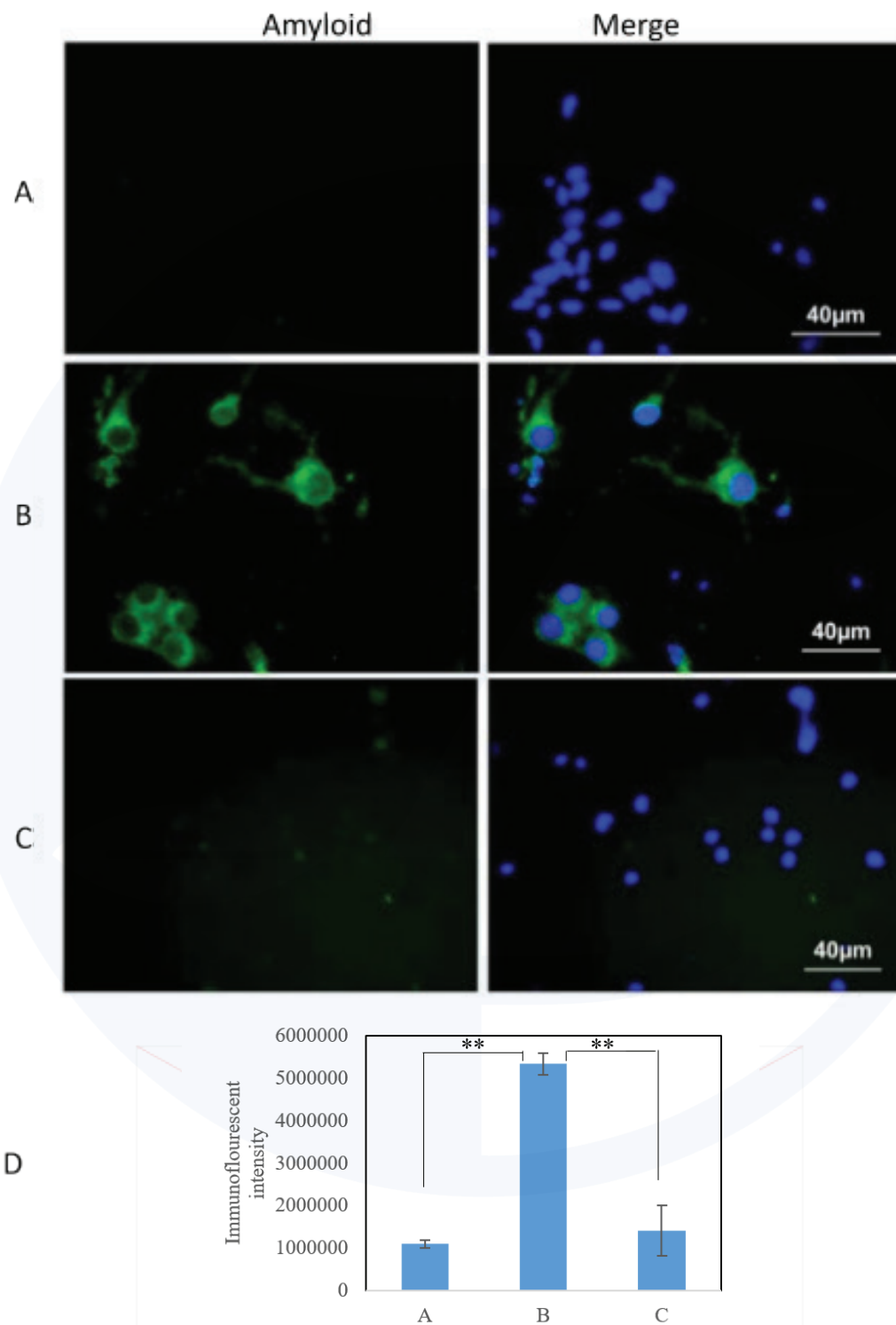
Figure 2. Immunostained cultured neurons with anti-amyloid antibody in (A) Control, (B) nutritional starved and, (C) Faradarmani CF (FCF) treated sample. (D) Quantification representation of A, B, and C (**: p-value<0.00l; the difference between A and C is not significant).
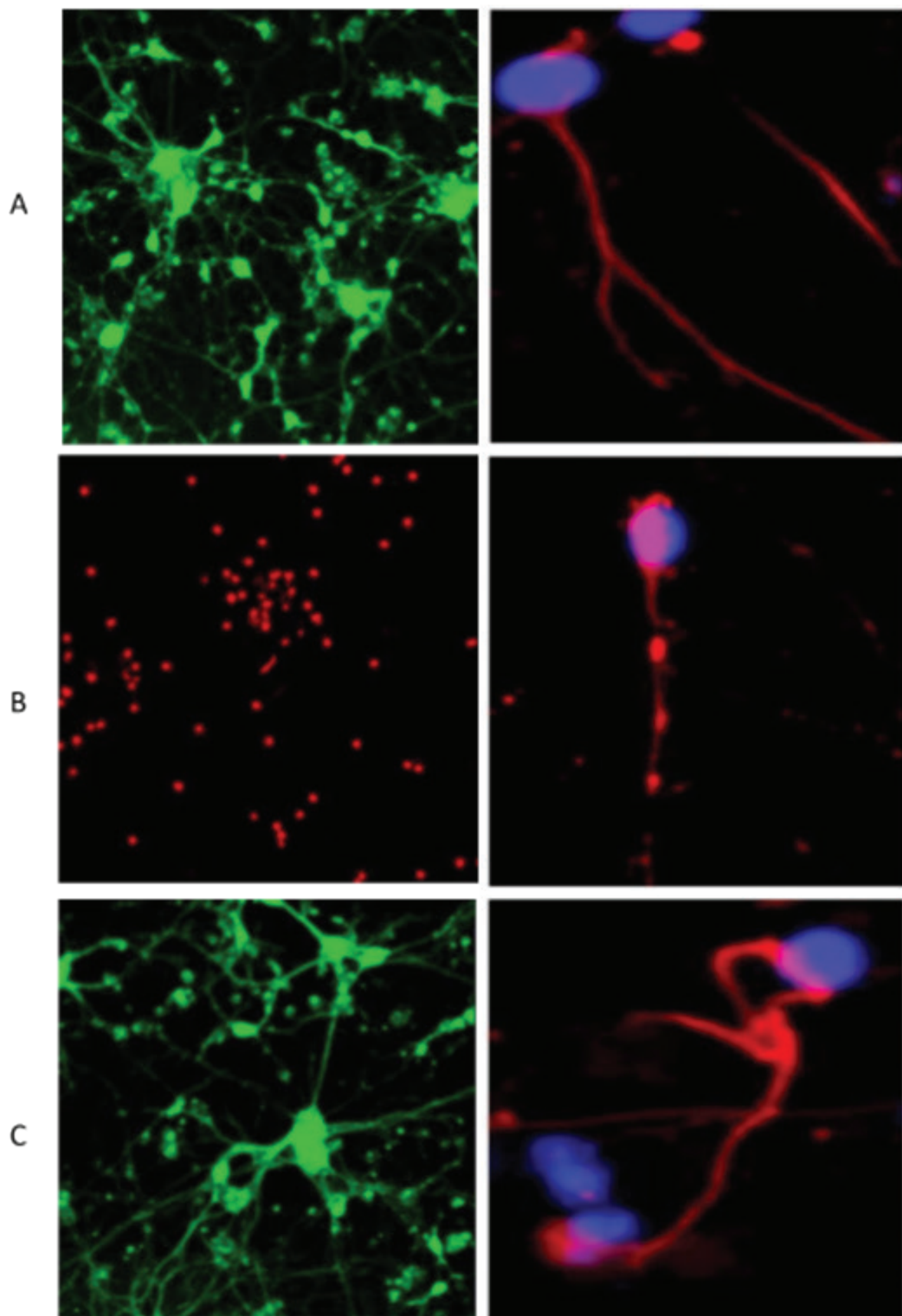
Figure 3. Live and dead cell assay and immunoblotting assay (Left) and Immunofluorescence staining of microtubules (Right) of stressed-out cultured neurons. (A) Control, (B) Sham and (C) Faradarmani CF (treated samples.

In vivo assessments:

In the in vivo assessment of mice AD models, the p-tau protein production (tauopathy) is evaluated in different samples of this study and is shown in Figures 4 and 5. As can be seen in these figures, while Traumatic Brain Injury induced prominent neurotoxic p-tau in the brain, the FCF treatment suppressed pathogenic p-tau accumulation. More-over, the use of elevated plus maze and the cognitive decline in TBI mice models have been investigated. As can be seen in Figure 6, the complete behavioral improvement in the FCF treated mice is such that even compared to the control group, they exhibit behaviors based on optimal brain function in relation to the memory, especially in the decision arm
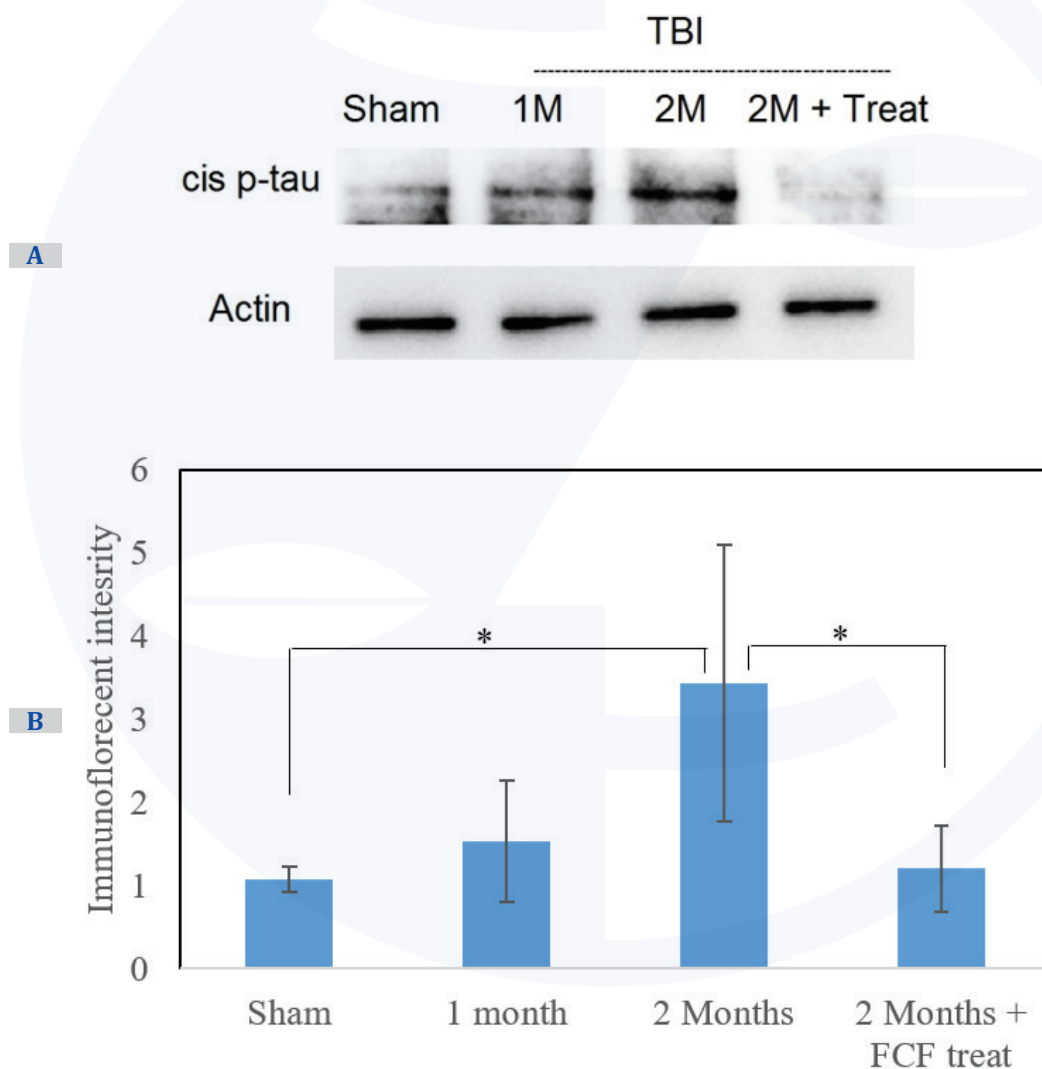
Figure 4. Immunoblotting (A) and Immunoflourescent (B) analysis of mouse brains stained with p–tau antibody upon various conditions. (C) Quantification representation of A and B (*: p–value<0.01).
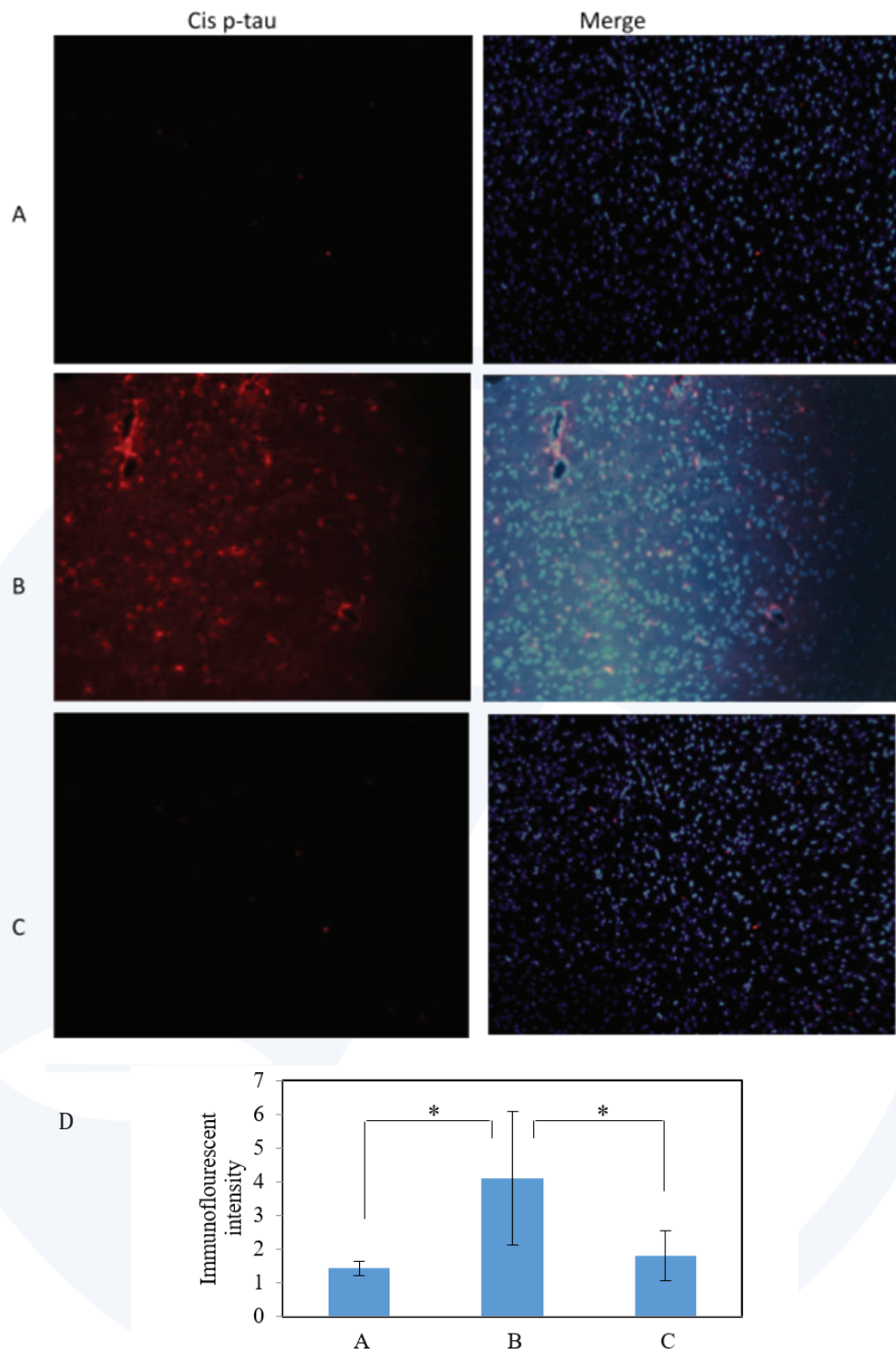
Figure 5. Immunoflourescent stained TBI mouse brains with p-tau antibody (A) Sham, (B) TBI, (C) FCF treated TBI mouse. (D) Quantification representation of A, B, and C (*: p-value<0.01).
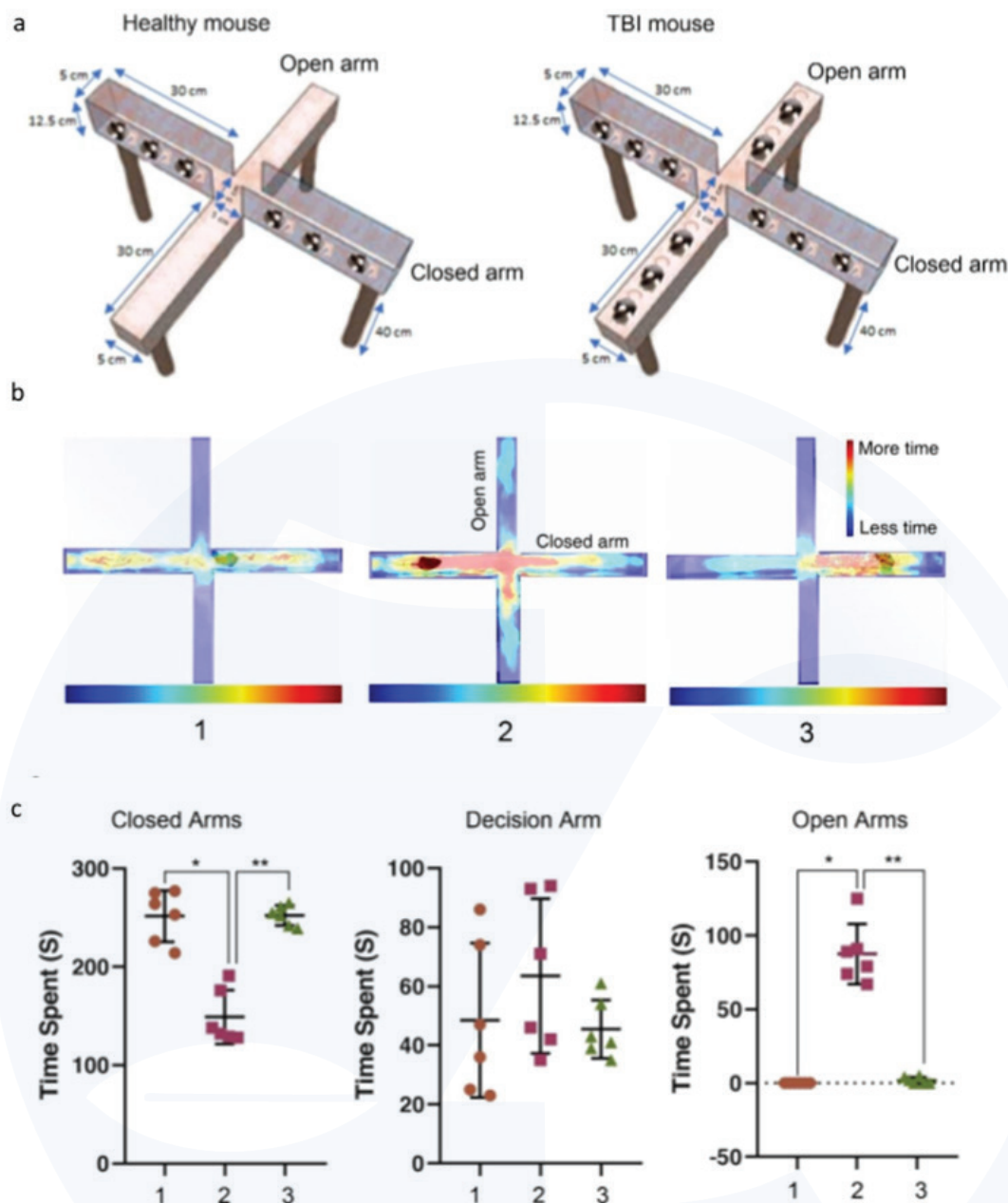
Figure 6. Behavioral analysis of TBI mouse models with the elevated plus-maze. (A) Schematic representation of healthy and TBI mice. (b) The hit map road of the mice in the different arms of the plus-maze. (c) Quantification of mice behavior based on the time spent in the different regions of the plus maze. (1) Healthy mice (Control), (2) Mice after 2-month TBI initiation, (3) Mice after 2-month TBI initiation with the Faradarmani CF (FCF) treatment (*: p-value <0.05, **: p-value <0.001).

DISCUSSION

This study is a continuation of assessments of Faradarmani CF on behavioral changes in AD mouse models, in addition to investigations on cell and molecular mechanisms involved in AD. The reduction in the Tau protein concentration in AD neuronal cells under stress, their survival, stabilization of microtubules, and reduction in the amyloidopathy are distinct differences when compared with the un-treated sham groups. AD is considered one of the most expensive diseases of old age and so the development of therapeutic

interventions to ameliorate or prevent the symptoms is of great interest. A therapeutic remedy still remains far-fetched, encouraging us to test the effects of the Faradarmani CF on AD animals and cell models. The use of Faradarmani CF as proposed by Taheri, has been considered in a variety of contexts in the past decade. As the TCFs are neither matter nor energy, we cannot measure them with scientific means. However, we can measure their effects on other objects or living organisms. We observe that Faradarmani CF does affect the subjects under the study at the cellular and molecular levels in addition to the whole organisms in a reproducible fashion. An appealing advantage of using Faradarmani CF is its non-pharmacological and interventional approach to producing results. This is particularly helpful in illnesses that have no pharmacological or other therapeutic forms of treatment in living systems. Since access to the Faradarmani CF is free and accessible to any researcher, we recommend its use for the assessment of TCFs' efficacy and specificity in similar or different systems. For instance, the efficacy of Faradarmani CF in the treatment of other neurological disorders such as Parkinson's disease, multiple sclerosis, or amyotrophic lateral sclerosis can be of particular interest.

CONCLUSION

Overall, in this study, by investigating the effects of a novel non-pharmacological method in the treatment of AD, disease development suppresses in the laboratory models. The results indicate that Faradarmani Consciousness Field is an effective factor in biological systems, especially at the level of the nervous system, and can be used as a powerful tool in the treatment of diseases and disorders.

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COMPETING INTERESTS

The authors declare that they have no competing interests.

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