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 is one of the most common causes of memory loss and imposes a substantial psychological and financial burden on societies worldwide. Therefore, there is a clear need for therapeutic strategies or treatments for this disease. The aim of this study was to investigate the effects of a novel non-pharmacological approach for the treatment of Alzheimer's disease based on the T-Consciousness Fields introduced by Mohammad Ali Taheri. This approach is proposed to act at the cellular and molecular levels. In this study, the effects of the Faradarmani Consciousness Field were evaluated in both an Alzheimer's disease mouse model (*in vivo*) and human neuronal cells (*in vitro*). For this purpose, human neuronal cell cultures and a traumatic brain injury (TBI) mouse model were used. Changes in amyloidopathy, tau protein content, microtubule aggregation, neuronal cell viability, and, finally, the behavior of TBI mice in the Elevated Plus Maze were assessed following Faradarmani treatment. According to the results obtained, treatment of human neuronal cells and the Alzheimer's disease mouse model with Faradarmani resulted in near-complete neuronal cell survival, elimination of amyloidopathy and tau protein accumulation, and significant behavioral improvement in TBI mice in the Elevated Plus Maze. Based on these findings, treatment with the Faradarmani Consciousness Field suppressed the progression of Alzheimer's disease in experimental models. Therefore, a human clinical study is proposed with the aim of introducing a new complementary medicine approach for the treatment of Alzheimer's disease.

Keywords: Alzheimer's disease, Faradarmani Consciousness Field, TBI, mouse model, neuronal cell model

Introduction

Memory loss associated with Alzheimer's disease is a major global health challenge in both industrialized and developing countries [1]. According to the World Health Organization, Alzheimer's disease and other forms of dementia were the seventh leading cause of death worldwide in 2019, and women accounted for 65% of these deaths [2]. Although various diseases can lead to dementia, Alzheimer's disease is the most common form among older adults and is estimated to account for 60–70% of all cases [3].

The disease begins years before the onset of clinical symptoms. Therefore, studies of preclinical Alzheimer's disease (AD) may provide valuable opportunities for therapeutic intervention [4]. Impairment of recent memory is typically the first symptom of AD and is sometimes accompanied by cognitive deficits such as changes in attention and problem-solving ability. As dementia progresses, other cognitive impairments, including language dysfunction, visuospatial difficulties, and loss of insight, frequently emerge [5]. These cognitive impairments are often accompanied by changes in personality and behavior, as well as mood disturbances such as irritability, impaired motivation and initiative, apathy, social withdrawal, and other related symptoms [6]. To manage these behavioral symptoms, psychological interventions, alternative therapies, and antidepressant treatments are commonly employed [7].

From a pathological perspective, AD is characterized by the progressive accumulation of amyloid-beta (Aβ) plaques and intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein [8, 9, 10]. The accumulation of Aβ in the brain is considered a primary factor in the development of Alzheimer's disease [11]; therefore, the clearance of amyloid-beta peptides has become a potential target for Alzheimer's drug development [12].

Various enzymes are capable of degrading Aβ, including neprilysin and insulin-degrading enzymes. Moreover, reduced activity of Aβ-degrading enzymes can itself contribute to the development of Alzheimer's disease [13, 14]. The cysteine protease cathepsin B (Cat B) reduces the levels of amyloid-beta peptides, particularly Aβ1–42, through proteolytic cleavage [15]. In addition, it has been reported that the peripheral system has the potential to clear amyloid-beta from the brain and help prevent Alzheimer's disease [16].

As mentioned earlier, tau accumulation within senile plaques is another hallmark of Alzheimer's disease. Under healthy conditions, tau proteins are abundantly associated with microtubules and are responsible for maintaining their stability in axons [17]. Microtubules provide structural support for the axons of growing and developing neurons [18, 19]. However, hyperphosphorylated tau can dissociate from microtubules and lead to the formation of intracellular neurofibrillary tangles (NFTs) [20, 21].

The risk of dementia is higher in individuals with a history of traumatic brain injury (TBI) [22]. One study has shown that a history of TBI is associated with an earlier onset of Alzheimer's disease-related cognitive decline [23, 24]. The authors reported that cis p-tau increases in TBI patients following sports-related injuries and military combat, as well as in stressed mice and neurons, prior to the development of tauopathy. In that study, treatment of TBI mice with a cis antibody prevented the progression of tauopathy. Therefore, cis p-tau may serve as an early biomarker for diagnosis and treatment. Furthermore, the cis pT231-tau isoform has been identified as a central mediator of TBI and neurodegeneration. Accordingly, targeting cis pT231-tau may represent a promising candidate for immunotherapy in tau-related disorders, including Alzheimer's disease (AD) [25].

The loss of cholinergic function in the central nervous system contributes to dementia and cognitive decline, and has therefore been a major target of anti-Alzheimer’s medications. [26, 27]. Although drug therapies may temporarily improve symptoms, they are unable to slow or halt neuronal degeneration [28]. In the present study, the effects of the Faradarmani Consciousness Field (FCF) on cellular and animal models of Alzheimer’s disease were investigated. According to Taheri’s theory, T-Consciousness is a fundamental constituent of the Universe with a non-material and non-energy nature. This theory also introduces various T-Consciousness Fields (TCFs),

which are non-frequency-based fields whose effects are initiated through a brief and instantaneous attention directed toward the subject under study. It is hypothesized that the information transmitted through these fields may produce changes in treated samples compared with controls. It should be noted that the FCF has been proposed as a complementary medicine approach [29].

Previous studies have reported the effects of the FCF on spatial memory and avoidance behavior in rat models of Alzheimer's disease [30]. The findings showed that the FCF improved scopolamine-induced impairments in memory and learning in rats. In addition, significant 'improving-enhancing' effects were observed in their passive avoidance behavior. Other studies that have used this approach include investigations of changes in cancer cell growth [31], the examination of brain electrical activity during Faradarmani connection in Faradarmangars (Faradarmani practitioners)[32] ,and the ameliorative effects of the FCF on wheat plants exposed to salt stress [33].

Materials and Methods

Application of the Faradarmani Consciousness Field

Faradarmani Consciousness Field was applied to the samples in accordance with the protocol established by the Cosmointel Research Center[1]. Further details are provided in the Common Considerations section of this issue. In this study, the Faradarmani Consciousness Field was applied daily to the AD animal and cellular models throughout the duration of the respective assessments. All animal experiments were approved by the Institutional Animal Care and Use Committee of the Royan Institute (Tehran, Iran) and were in accordance with the NIH Guide for the Care and Use of Laboratory Animals.

Antibodies

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

Generation of Human Embryonic Neural Progenitor Cells (hESC-NPCs)

Induced pluripotent stem cells (iPSCs) derived from two late-onset Alzheimer's disease patients and two age-matched healthy controls were obtained from the Royan Cell Bank. These iPSCs had originally been generated from fibroblasts using Yamanaka factors. The iPSCs were subsequently differentiated into neural progenitor cells (NPCs). The neuronal induction medium consisted of DMEM/F12 medium (21331020 Gibco) supplemented with 5% knockout serum replacement (Gibco, 10828028), Glutamax (25030081 Gibco), MEM-NEAA (11140050 Gibco), 1% N2 supplement (17502048 Gibco), 3 µM SB431242 (13031 Cayman), 5 µM Dorsomorphin (04-0024 Stemgent), 3 µM CHIR99021 (04-0004-10 Stemgent), and 0.5 µM SAG (912545-86-9 Cayman).

Rosette-like structures were manually isolated seven days after induction. The NPCs were then replated onto tissue culture dishes coated with 1 mg/mL laminin and 15 mg/mL poly-L-ornithine (Sigma-Aldrich) and cultured in a neural expansion medium consisting of DMEM/F12 supplemented with 5% knockout serum replacement (KOSR), 1% non-essential amino acids, 2 mM L-glutamine, and 2% supplement (all from Invitrogen), 0.1 mM β-mercaptoethanol, 20 ng/mL basic fibroblast growth factor (bFGF; Royan Institute), 20 ng/mL epidermal growth factor (EGF; Sigma-Aldrich), and 0.2 mM ascorbic acid (Sigma-Aldrich). The culture medium was replaced every other day for seven days. Following neuronal differentiation, the expansion medium was replaced with a differentiation medium by removing bFGF and supplementing with hBDNF (PR-1113), hGDNF (PR-1107), and cAMP (Sigma, D0627) for 35 days. The differentiation process was monitored and confirmed by SOX2, PAX6, NESTIN, NCAM, GFAP, MAP2, GAD65, GABA, and S100 staining.

1. www.cosmointel.com

Nutrient Deprivation Stress

Under standard conditions, the culture medium was changed every four days according to the differentiation protocol. To induce starvation or nutrient deprivation stress, the medium was not replaced for 96 hours. Tauopathy was initially observed in the cell culture during this period, and after 96 hours of nutrient deprivation, sustained tauopathy was detected, consistent with previous findings [24].

Live/Dead Cell Assay

Cell viability was assessed using fluorescein diacetate (FDA) and propidium iodide (PI) double staining. Stock solutions of 20 µL FDA and 50 µL PI were diluted in 10 mL PBS [34]. Cells were first washed with cold PBS, then incubated with the FDA/PI solution. After 5 minutes of incubation at room temperature, samples were examined using a fluorescence microscope.

Cell Staining

Cells were collected at different time points and fixed with 4% paraformaldehyde (PFA) for 20 minutes in the dark. Samples were either observed directly under a microscope or processed for immunostaining. For further processing, cells were permeabilized with 0.2% Triton X-100 for 20 minutes and incubated overnight at 4°C with primary antibodies. Subsequently, samples were incubated for One hour at room temperature with Alexa Fluor 488- or 568-conjugated secondary antibodies. Imaging was performed using a Zeiss LSM 800 confocal microscope.

Traumatic Brain Injury (TBI)

Based on previous studies in this field, a mouse model of TBI was used [24]. A total of 18 male C57BL/6 mice (2–3 months of age) were obtained from the Royan Animal Facility and randomly assigned to injury or sham groups. Mice were anesthetized for 45 seconds using 4% isoflurane in a 70:30 air/oxygen mixture. The anesthetized animals were placed on a Kimwipe (Kimberly-Clark, Irving, TX) and positioned so that the head was directly under a hollow guide tube. The tail was gently restrained. A 45 g metal weight was used to deliver a blow to the posterior skull, inducing rapid rotation. This procedure resulted in a single severe traumatic brain injury (ssTBI, 60-inch height). Sham-injured mice were anesthetized but did not receive any brain injury. All mice recovered in room air conditions. Each animal remained under anesthesia for 45 seconds. Subsequent behavioral and histological assessments were performed in a blinded manner.

Immunohistochemistry

Following injury, mice were perfused with 4% PFA at different time points, and brains were collected for further analysis. Serial 8 µm coronal sections of both sham and injured brains were cut using a cryostat (Leica) and mounted onto slides.

Immunoblotting Analysis

Immunoblotting was performed as previously described [24]. 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% sodium deoxycholate, 50 mM NaF) supplemented with protease and phosphatase inhibitors. Samples were mixed with SDS sample buffer, boiled, and loaded onto gels. Proteins were separated by polyacrylamide gel electrophoresis and transferred to PVDF membranes. Membranes were blocked with 5% milk in TBST (10 mM Tris-HCl, pH 7.6, 150 mM NaCl, 0.1% Tween 20) for 1 hour. Subsequently, membranes were incubated overnight at 4°C with primary antibodies diluted in 5% milk/TBST. After washing, membranes were incubated with HRP-conjugated secondary antibodies in 5% milk/TBST. Signals were detected using chemiluminescence (PerkinElmer, San Jose, CA). Membranes were washed six times with TBST between steps. Quantification of immunoblotting results was performed using Quantity One software (Bio-Rad).

Immunostaining Analysis

Immunofluorescent staining of mouse brain sections was performed as previously described [24]. Slides were treated with 0.3% hydrogen peroxide in 10

mM sodium citrate buffer (pH 6.0) and subjected to antigen retrieval by boiling. Sections were then incubated overnight at 4°C with primary antibodies. After washing, samples were incubated for 1 hour at room temperature with Alexa Fluor 488- or Alexa Fluor 568-conjugated secondary antibodies (Jackson ImmunoResearch, West Grove, PA). Slides were washed three times with TBS after each step and imaged using a Zeiss LSM 800 confocal microscope. Confocal laser settings were adjusted to eliminate fluorescence signals arising from secondary antibody–based autofluorescence in sections processed without primary antibodies.

Elevated Plus Maze

The elevated plus maze (EPM) test was used to assess anxiety-like behavior two months after injury. The maze consisted of two open arms and two closed arms (30 × 50 cm) arranged perpendicular to each other around a central platform (decision zone). The entire apparatus was elevated 100 cm above the floor. Mice were placed in the center of the maze facing a closed arm and allowed to explore freely for 5 minutes. The apparatus was cleaned with a diluted ethanol solution between trials. Time spent in the center zone, open arms, and closed arms was recorded. The percentage of time spent in open arms was used as an index of risk-taking behavior.

Statistical Analysis

All experiments were repeated at least three times. No samples or animals were excluded from the analyses. Heatmap data were collected using Python and OpenCV. Data are presented as mean ± standard deviation (SD). Statistical significance was assessed using two-way analysis of variance (ANOVA), followed by multiple-comparison tests with a 95% confidence interval. A p-value of less than 0.05 was considered statistically significant. All other statistical analyses were performed using GraphPad Prism version 8.

Results

***In Vitro* Assessments**

At this stage, the effects of the FCF on tauopathy, amyloidopathy, neuronal viability, and microtubule organization in neurons subjected to aging-related stress were evaluated. Three experiment groups were included: A, B, and C. Group A served as the control (without any treatment), Group B was the sham group (positive control; neurons exposed to aging stress without Faradarmani treatment), and Group C was the experiment group in which the effects of the FCF on parameters associated with the cellular model of Alzheimer's disease were evaluated and compared with the other groups.

The immunofluorescence intensity of cis p-tau protein in the three study groups is shown in Figure 1. As illustrated in Figure 1, cultured neurons exposed to stress exhibited a marked increase in cis p-tau levels, whereas Faradarmani treatment eliminated p-tau from the cells.

In addition, as shown in Figure 2, aging-related stress induced significant amyloidopathy in cultured neurons, while FCF treatment halted the progression of this pathological process.

Changes in neuronal viability and microtubule structural integrity under aging stress in the various cell groups of this study are presented in Figure 3. As shown, FCF treatment markedly suppressed neuronal degeneration in cultured cells exposed to aging stress. Furthermore, microtubule degradation was observed in stressed neurons, whereas FCF reversed this condition, which was confirmed by immunofluorescent staining of the cells.

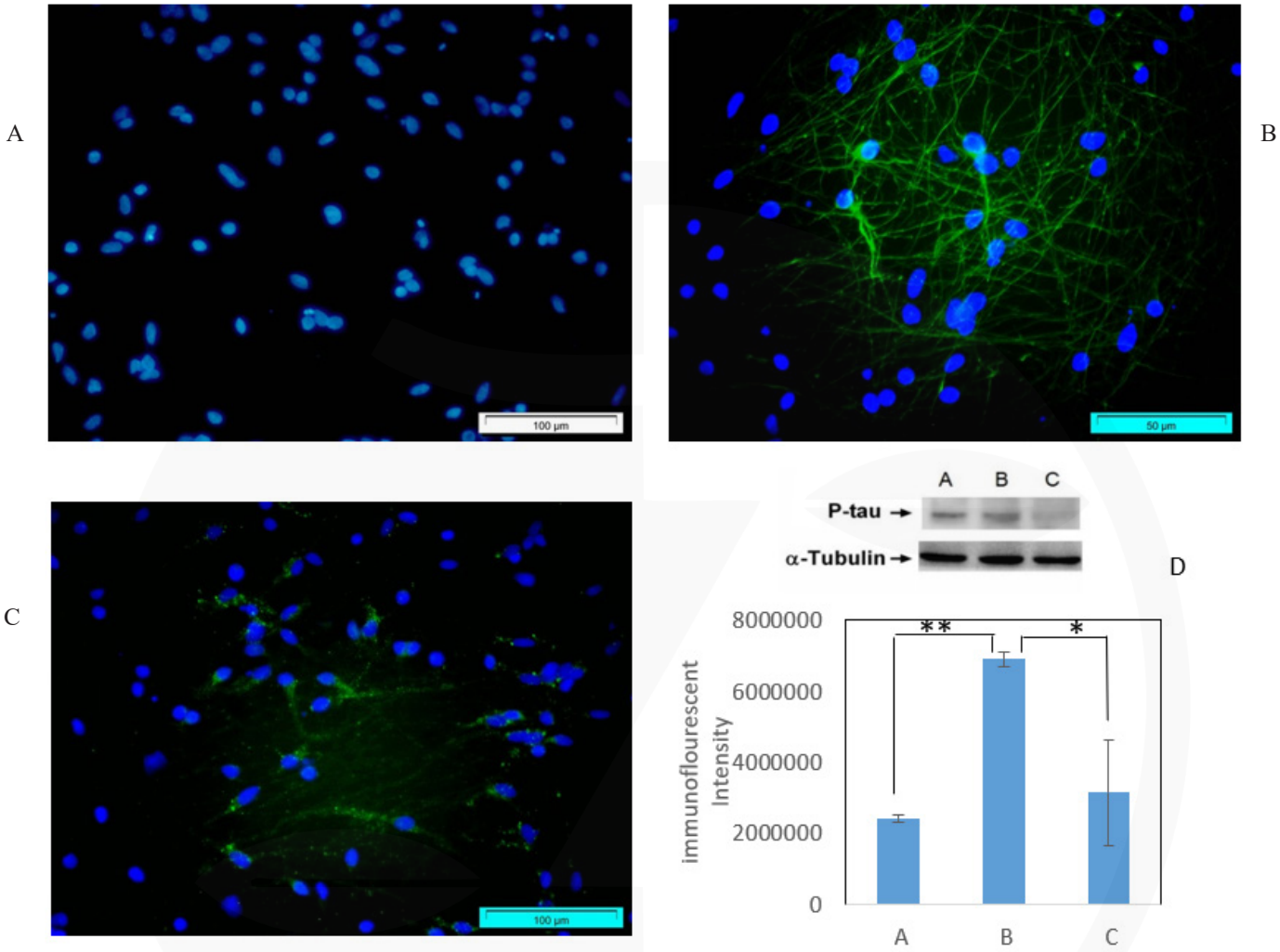


Figure 1. p-tau immunostaining in cultured neurons.
A: Control group. B: Nutrient-deprived group. C: Stressed neurons treated with the Faradarmani Consciousness Field. D: Quantification of immunofluorescence intensity for groups A, B, and C. *: $p < 0.01$; **: $p < 0.001$. The difference between groups A and C was not statistically significant.

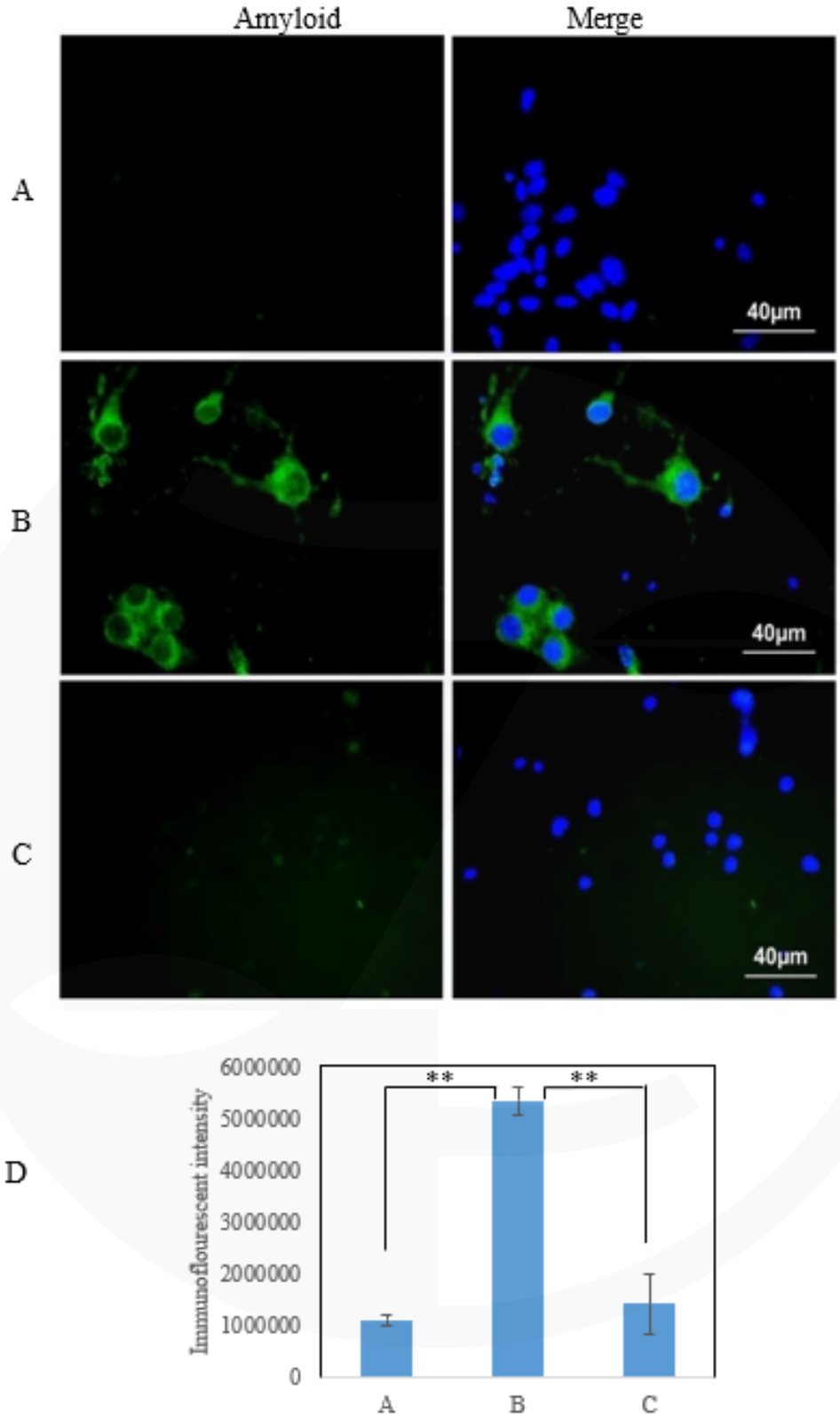


Figure 2. Neurons stained with anti-amyloid antibody. A: Control group. B: Nutrient-deprived group. C: Sample exposed to the Faradarmani Consciousness Field. D: Quantitative analysis of groups A, B, and C. **: $p < 0.001$. The difference between groups A and C was not statistically significant.

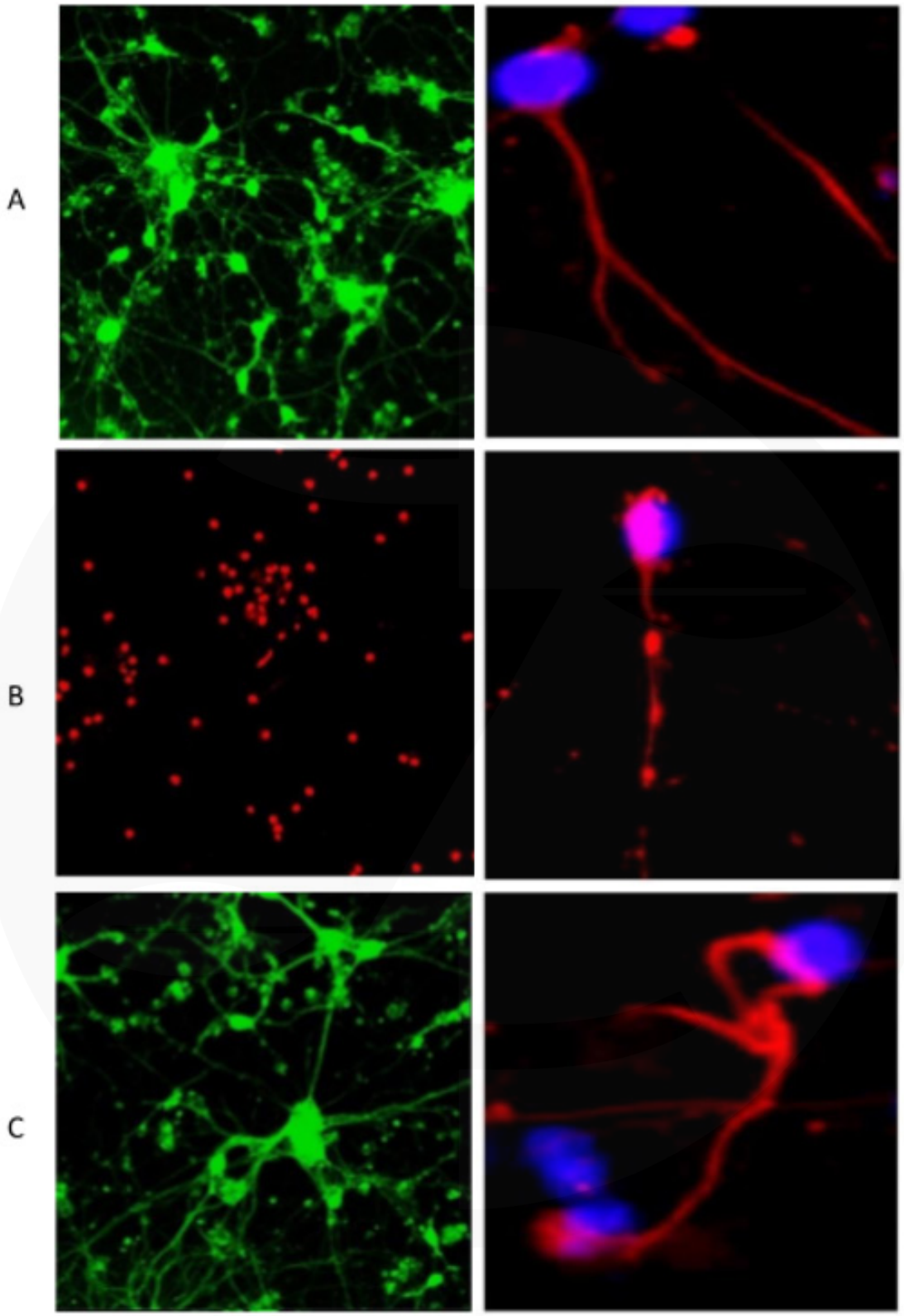


Figure 3. Live/dead cell assay and immunoblotting analysis (left), and immunofluorescence staining of neuronal microtubules (right) under stress conditions in culture. A: Control group. B: Sham group. C: Samples treated with the Faradarmani Consciousness Field.

***In vivo* Evaluations**

In the *in vivo* Alzheimer's disease (AD) mouse model, production of p-tau protein (tauopathy) was evaluated in different samples, as shown in Figures 4 and 5. As illustrated in these figures, while traumatic brain injury (TBI) induced neurotoxic accumulation of p-tau in the brain, treatment with the FCF prevented p-tau aggregation.

Cognitive decline in the TBI mouse model was also assessed using the elevated plus maze. As shown in Figure 6, a complete behavioral recovery was observed in the FCF-treated mice, which, compared to the control group, demonstrated optimal brain performance in memory-related functions, particularly in the decision-making arm.

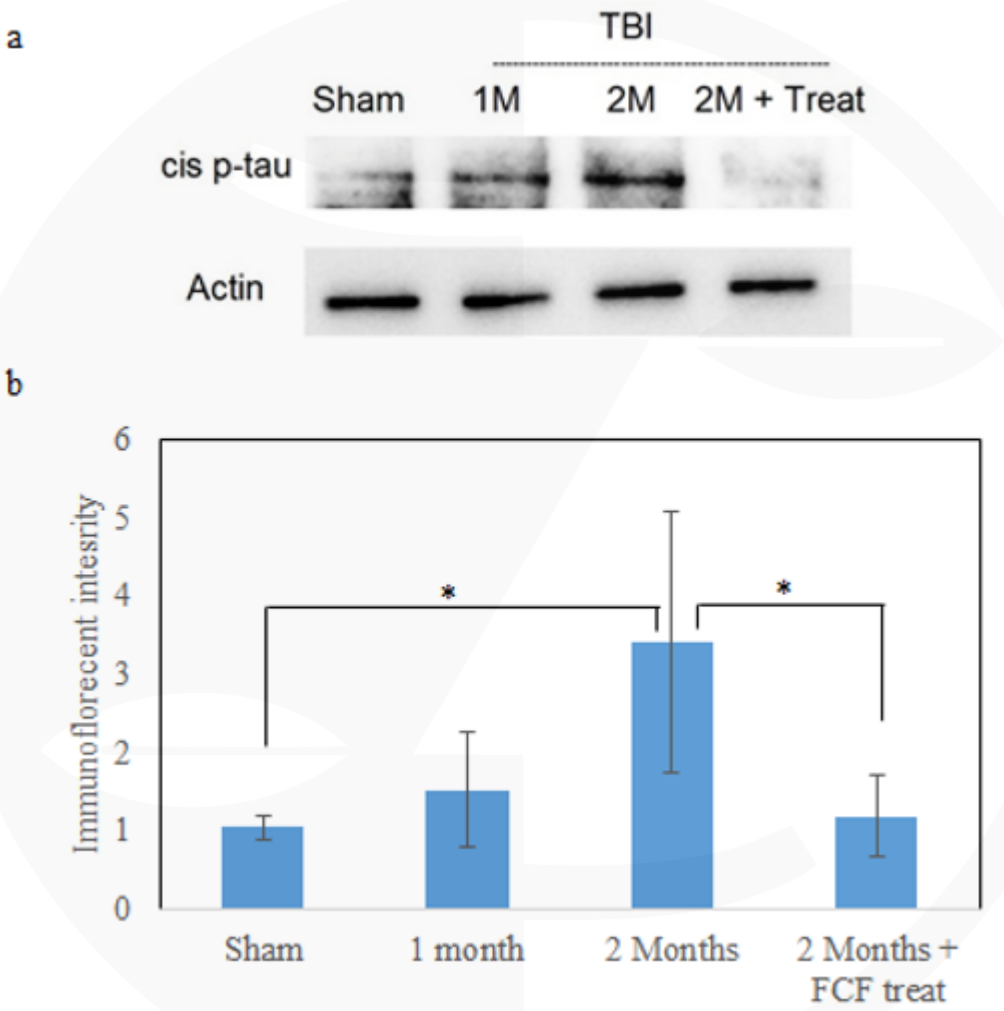


Figure 4. Immunoblotting (a) and immunofluorescence (b) analyses of mouse brain sections stained with anti–p-tau antibodies under different conditions. *: $p < 0.01$.

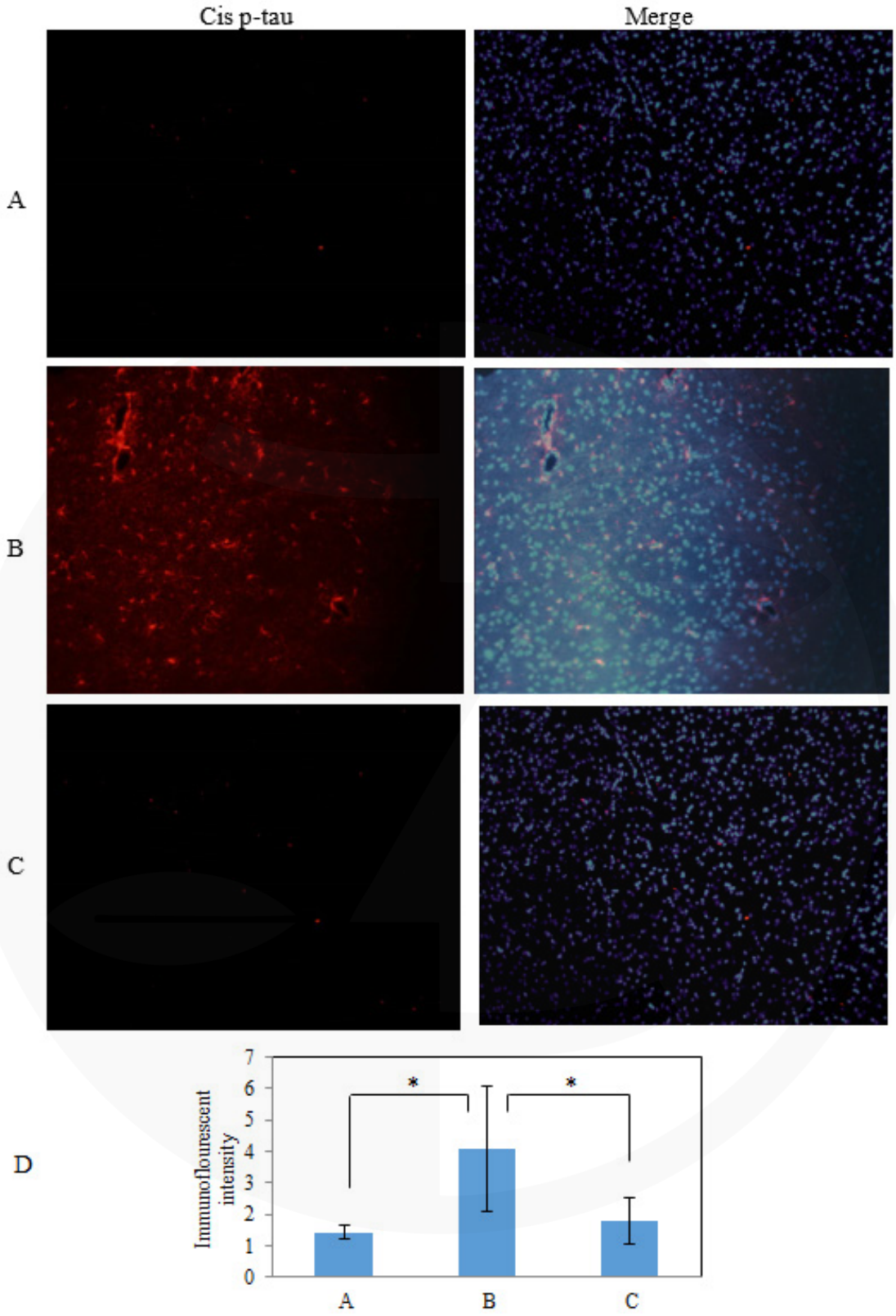


Figure 5. Immunofluorescence staining of TBI mouse brain sections using anti–p-tau antibody.
A: Sham; B: TBI; C: Faradarmani Consciousness Field–treated group; D: Quantitative analysis of the above conditions. * p-value < 0.01.

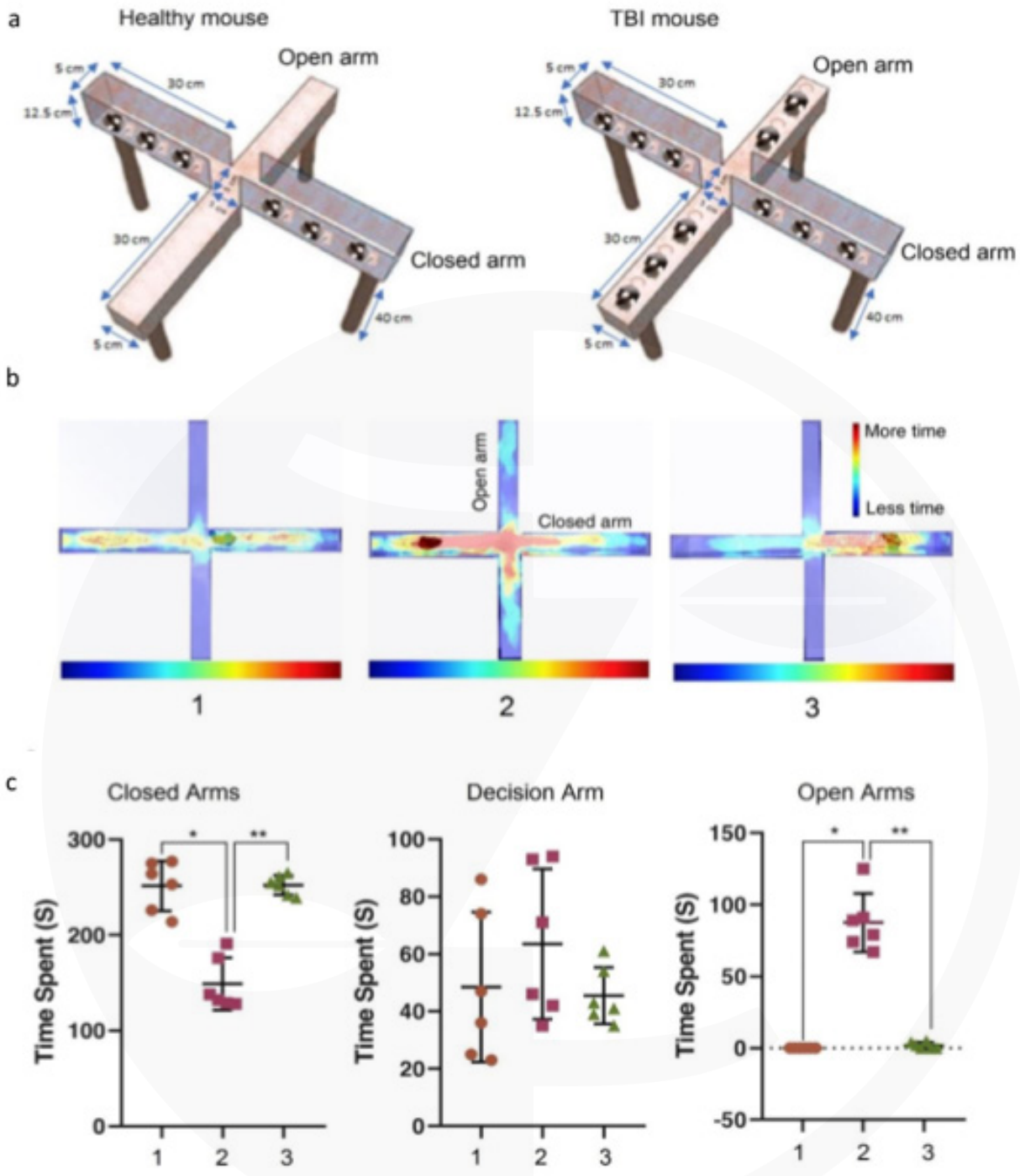


Figure 6. Behavioral analysis of TBI mouse models using the elevated plus maze. a: Schematic representation of healthy and TBI mice. b: Movement trajectories of mice in different arms of the elevated plus maze. c: Quantitative analysis based on time spent in different zones of the elevated plus maze. 1: Healthy mouse (control); 2: Mouse two months after TBI induction; 3: Mouse two months after TBI treated with Faradarmani Consciousness Field. * p-value < 0.05; ** p-value < 0.001

Discussion

This study examined the effect of the Faradarmani Consciousness Field (FCF) on cellular and animal models of Alzheimer's disease. As noted in the introduction, this 'field' has a non-frequency nature, and its effect is initiated without any physical intervention, through a brief and instantaneous attention directed by the 'Announcer' (*Faradarmangar*). A key distinction between this theory and other mind–body approaches, such as meditation and mindfulness, is that here the mind functions only as a mediator in initiating the influence of the T-Consciousness Fields. This feature makes it possible to design various experimental models, including animal and cellular systems, to record the effects of these fields.

Tau protein plays a key role in stabilizing microtubules, and it has been shown that neuronal degeneration and cognitive decline are often associated with the accumulation of tau proteins [35]. Accordingly, extensive research has focused on reducing tau accumulation and stabilizing microtubules in order to control and halt disease progression [36, 37]. In the present study, a reduction in p-tau accumulation was observed both in cells subjected to nutritional deprivation-induced tauopathy and in the brain injury mouse model. Behavioral assessment of mice in the elevated plus maze also indicated improved performance in the group exposed to the FCF.

Given that no material or energy intervention was applied to the experiment groups, the observed changes are attributed to the effects of this field. Although the exact mechanism of action remains unclear, it is hypothesized that information transmitted from T-Consciousness Fields may induce changes in treated samples compared with the control group. Further studies in this area, including evaluation of the effect of the FCF on a scopolamine-induced mouse model of Alzheimer's disease, as well as assessment of brain electrical activity in humans, are planned by the authors of this study.

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