Effects of Faradarmani Consciousness Field on Hippocampal Structure/Function and Pancreatic Beta Cells in STZ-Induced Diabetic Rats

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Abstract

Diabetes mellitus (DM) is a growing health issue that harms brain structure and function, particularly in the hippocampus, which is crucial for learning and memory and is affected by disrupted glucose homeostasis. There are limited non-invasive treatments specifically targeting CNS impairments associated with diabetes. T-Consciousness Fields (TCFs) with non-physical entities have been introduced by Taheri. The influence of these fields can be investigated on various subjects, including living organisms and inanimate materials. The purpose of this study is to investigate the effect of a type of TCFs named the Faradarmani Consciousness Field on hippocampus structure and function in diabetic rats. The male Wistar rats (n=48, 150 ± 10 g weight, 8 weeks age) were categorized into four groups (n=12) including control (C), Faradarmani Consciousness Field (T), Diabetic control (D), Faradaemani Consciousness Field+diabetic (DT). Faradarmani was applied over eight weeks to both Diabetic (DT) and T groups. Peritoneal injection of STZ solution (50 mg/kg) was used to induce diabetes. Cognitive functions were assessed using the Morris water maze. At the end of the experiment, rats were sacrificed, and their brains were processed for cresyl violet staining to quantify surviving neurons in the hippocampus. H&E staining showed significant disorganization and cell loss in brain regions of the diabetic group. According to cresyl staining, in diabetic groups, Faradarmani treatment significantly reduced the death of cells in the CA3 and DG areas of hippocampus. Additionally, the rate of beta cell destruction and cellular apoptosis, as graded, decreased in the DT group but not significantly. Data from the Morris water maze revealed that diabetic groups exhibited notably impaired cognitive performance relative to the non-diabetic group. The T group spent significantly more time in the target zone across all days, while the DT group outperformed the D group on the second day. Although the distance traveled was significantly higher in the Faradarmani groups, the time spent in target zone for the T and DT groups suggests a possible improvement in memory retention. In conclusion, this experiment provides evidence of the effects of Faradarmani on structure and function of hippocampus in a rat model

Keywords: Taheri Consciousness Fields, Faradarmani, Cognitive function, Diabetes mellitus, Cosmic Consciousness Network.

Introduction

Diabetes mellitus is considered a serious metabolic problem in the world. Different types of diabetes are characterized by hyperglycemia, insulin resistance, and relative insulin deficiency (American Diabetes Association, 2010). Studies have shown that lifestyle changes including weight loss, increased mobility, and lower consumption of glucose and high-calorie sources can prevent the disease and its progression (Baghianimoghadam et al., 2011).

Based on the results of recent studies, high levels of glucose are regarded as the main reason for damage to the nervous system (Marissal-Arvy & Moisan, 2022). The hippocampal formation is a part of the limbic system which is a key brain area for many forms of learning and memory and is particularly sensitive to changes in glucose homeostasis. Its neurons are extremely vulnerable to diabetes (Marissal-Arvy & Moisan, 2022). Lower hippocampal and brain volumes are likely to happen in diabetes (Ho et al., 2013).

Beta cell destruction is a pivotal factor in impaired insulin production in both Type 1 Diabetes (T1D) and advanced stages of Type 2 Diabetes (T2D). In T1D, an autoimmune response targets and erroneously destroys beta cells, resulting in a significant loss of insulin production. This compromises the regulation of blood glucose levels, necessitating exogenous insulin administration. In T2D, chronic exposure to elevated blood glucose and metabolic factors leads to gradual beta cell dysfunction and destruction. The diminished responsiveness of beta cells to glucose, coupled with insulin resistance in peripheral tissues, contributes to inadequate insulin secretion, exacerbating hyperglycemia over time (Kroon et al., 2008).

Various studies have shown that diabetes is mainly associated with degenerative and functional disorders of the central nervous system, and there is ample evidence that the pathway that begins through the neurodegenerative effects of diabetes is oxidative stress. Reactive oxygen species (ROS), which are composed of oxygen free radicals and other chemical compounds, can promote oxidative stress in the body (Montilla et al., 2005) and eventually cause neuronal death, leading to diabetes-related neuropathology, which has a direct impact on the memory and learning of diabetics (Kashihara et al., 2010).

Several methods have been proposed for the treatment and control of diabetes, including traditional medicine, different types of exercise, drug treatments, stem cells, Complementary and alternative medicine (CAM), etc. Due to the side effects of drug treatments and the reduction in their effectiveness over time, researchers are motivated to explore new methods and discover improved ways to manage diabetes and its complications (Burcelin et al., 1999). So far, the effect of more than 1,200 herbs in reducing blood sugar or reducing its side effects is known. Over the past 10-20 years, numerous laboratory and clinical studies have been performed on medicinal plants used in the treatment of diabetes, some of which have significant effects on lowering blood sugar in diabetic patients (Marles & Farnsworth, 1995).

The examination of consciousness and its position within the scientific discourse has attracted considerable scholarly attention in the contemporary era (Taheri, Modarresi-Asem, et al., 2022; Taheri, Payervand, et al., 2022). The aim of this study is to investigate the potential of Faradarmani Consciousness Field in alleviating diabetes-related consequences in the hippocampus and pancreas.

Material and Methods

Animals

To this aim, 48 male Wistar rats (150 ± 10 gr, 8 weeks age) were selected from Pastoor Institute in Iran. The rats were categorized into four groups including control (C), Faradarmani Consciousness Field (T) Diabetic control (D), and Faradarmani Consciousness Field +diabetic (DT). The study begins with one-week acclimatization to allow the rats to

become familiar with their new surroundings which were environmentally controlled (55 ± 5 humidity, 20 ± 5 °C temperature), 12-12 H light-dark cycle (lights on 6:00–18:00 hours).

Induction of Type 2 Diabetes

For obesity, diabetic rats were exposed to a high-fat diet for the first 4 weeks including 22% fat, 48% carbohydrates, and 20% protein from the Razi Institute of Dermatology in Iran (Zhang et al., 2008). Type 2 diabetes mellitus was induced by dissolving STZ (50 mg/kg, i.p.; Sigma) in a citrate buffer (pH 4.5) and injected in each rat. Then, the blood samples from the tail were obtained 24 hours after the injection. The blood glucose meter (Beurer GL42, Germany) measured the glucose levels to determine whether the rats were diabetic (>250 mg/d) (Islam, 2013).

Faradarmani Consciousness Field application protocol

Faradarmani was applied to two groups and to ensure that the control groups are not affected by Faradarmani, we separated Fardarmani groups into separate room in the lab. The treatment duration was 8 weeks, with the treated groups receiving Faradarmani for 10 minutes every day, six days a week. Those samples without Faradarmani treatments are considered as controls.

Cognitive tests

These tests were performed four weeks after induction of diabetes to allow time for the development of the diabetic-associated behavioral changes and then repeated at the end of the study after 8 weeks to assess the effect of the different treatment protocols.

The Morris water maze test

Morris water maze is a circular pool (150 cm diameter, 60 cm height) which was divided into four quadrants including North East (NE), North West (NW), South East (SE), and South West (SW), and was filled with water (20±1 °C, 55±5% humidity), escape platform, and camera which was suspended above the maze and recorded escape latency, distance to reach the platform, and the percentage of time which rat spent in target. Then, the surface of the escape platform was covered in a fixed position of 1 cm under the water. The MWM was in the laboratory surrounded by various signs and colors (circle, triangle, and square), which was considered a training protocol identified as a very good and relevant test for evaluating the effect of exercise on learning and memory. Each rat was given eight trials per day for three consecutive days. The interval between trials was three minutes. Each release point was randomly changed for every trial. To be accustomed to the maze, the mice were placed in water without a platform for three minutes to swim three hours before training. Then, each rat was placed slowly from the tail zone and faced the wall of the pool to avoid stress at different starting points, and then was allowed to swim to find the hidden platform. Each trial lasted until the rat discovered the hidden platform or for a maximum duration of 60 seconds. Further, it was allowed to rest on it for 30 seconds. Each rat that failed to find the platform within the allocated time was picked up and placed on the platform by the experimenter. After the last trial, the rats were dried with a towel and returned to the home cage. To assess memory retention, a spatial probe test was performed one day after the last acquisition trial, and the platform was removed from the maze. Each rat spent 60 seconds searching for the water maze.

Long-term memory

After three days' rest, a hidden platform was placed in the SW quadrant, and each rat performed one trial similar to the acquisition sessions to evaluate long-term memory. The spatial learning and memory of rats were tested according to the method of R. Morris. A Morris water maze with a submerged platform and a video tracking system (ANY-maze™ Video Tracking System; version 4.72-Stoelting Co.) were used.

The Morris water maze consisted of a circular tank, (diameter: 120 cm, height: 30 cm) filled to a depth of 24 cm. The water temperature was 26°C and a 10 cm clear circular platform was submerged 1 cm below the water level in the northwest quadrant of the maze.

Cue discrimination. a visible platform test was performed to exclude drug or experimental manipulation-induced changes in visual acuity. The video tracker system was not used and only a stopwatch was used in this test. Habituation to the pool was done by permitting the rats to swim freely for 30 seconds and giving them four trials (from four different directions) to climb to the platform that had been extended 1 cm above the water level. The rats then had 15 trials of cue training in 3 block intervals, each including five trials; the intervals (intertrial and interlock) were approximately 10 minutes. During this stage, we didn't provide the rats with cues except for the platform.

During spatial discrimination, the hidden platform was placed 1.5 cm below the water level changing the area of the pool from that used during cue discrimination training. We added powdered milk to make the pool water opaque, rendering the platform 'invisible'. The platform location had been fixed relative to the distal cues. Rats had trained in eighteen trials in the form of six blocks (three trials per block) and the intertrial intervals were about 10 minutes, after every trial, we stirred the water to avoid the effect of odor trails as unwanted cues. Rats were allowed to start swimming in each trial from one of four locations (north, south, east, and west); the choice of the location was random for each rat and each trial. The rat should escape to the platform within 60 seconds and if that didn't occur, we guided them gently toward the hidden platform where they remained for 10 seconds. The rats were dried with a towel and returned to their cage after every trial. The parameters recorded in these training blocks were: latency to reach the platform, distance travelled in the maze till reaching the platform and proximity (% of time spent within the quadrant where the platform was placed).

Probe trial

In the probe trial (the immediate probe trial) we removed the platform from the swimming pool and allowed the rat to swim for 60 seconds. The probe trial was given after the fifth training block and the rats then had the sixth block of training that was not included in the cognitive assessment. To assess 24-hour retention, rats were given another probe trial 24 hours later (the platform was removed from the pool) (Vorhees & Williams, 2006).

Tissue preparation and sectioning

Tissues were placed in 10% formaldehyde for two hours, then were removed and placed in a new formaldehyde solution for 24 hours before being dehydrated using ethanol (70% for 24 h, 90% for 1 h and 100% for 1 h) and then cleaned in xylene and embedded in paraffin. Coronal sections were cut with a microtome (Leica RM 2025, Germany) at 5 µm thicknesses, mounted on glass slides and underwent different staining methods (Theory and Practice of Histological Techniques, 6th Edition | Journal of Neuropathology & Experimental Neurology | Oxford Academic, n.d.).

Histological staining

Hematoxylin and eosin (H&E) staining

The left hippocampus and pancreas were fixed with 10% neutral paraformaldehyde, dehydrated through ascending concentrations of ethyl alcohol, cleared in xylene, embedded in paraffin, and then cut manually using a microtome to obtain 5 mm thick sections. The sections were deparaffinized and rehydrated through descending concentrations of ethyl alcohol and stained with hematoxylin and eosin (H&E). The stained tissues were dehydrated in 80% alcohol followed by 95% ethyl alcohol, placed in two changes of 100% ethyl alcohol, and cleansed with two changes of xylene. Histopathological examinations were carried out by using a phase contrast microscope with an attached camera (Nikon H600L, Tokyo, Japan).

The pancreas was harvested from the sacrificed rats after dissection and was weighed and washed with saline. The specimens were stretched on filter paper and fixed in 10% buffered formalin (pH 7.4). The fixed specimens were sliced, processed, and embedded into paraffin blocks. The blocks were cut into 4 μm paraffin sections by a rotator microtome. The sections were stained with Hematoxylin and Eosin (H&E) and with Masson trichrome stains

Fuchsin staining for pancreas beta cells

The pancreas was dissected out carefully and fixed in 10% formal saline for 48 hours and thereafter processed for paraffin blocks. Sections of 5-micron thickness were taken and stained with Haematoxylin and Eosin as well as Gomori's Modified Aldehyde Fuchsin stain and observed under a light microscope (Greenwell et al., 1983).

Nissl staining for hippocampus neurons

Nissl staining was used to assess neuronal damage in the hippocampus (Nobakht et al., 2011). Brain sections were immersed in xylene solution for 15 minutes and then slides were immersed in ascending grades of alcohol in the following order, absolute alcohol - 1 minute, 90 % alcohol - 2 minutes, 70 % alcohol - 2 minutes, 50 % alcohol - 2 minutes. After processing through alcohol, slides were immersed in distilled water for 10 minutes and were stained for 25–30 minutes at a temperature of 60° in 0.1 % cresyl violet stain and then allowed to cool at room temperature. Stained sections were again immersed in distilled water for 5 minutes and in ascending grades of alcohols (70 %, 90 %) for 2 minutes. Finally, sections were dipped in xylene for clearing and mounted with DPX. The brain sections were incubated with a 5% toluidine blue solution at room temperature for 15 min. Following rinses with tap water, the sections were dehydrated and mounted.

Statistical analysis

The results were analyzed using SPSS. Data were presented as mean ± SD. Comparison of quantitative variables between the studied groups was done using the analysis of variance One-Way (ANOVA) test with the Bonferroni post hoc test or Kruskal Wallis test with Wilcoxon signed rank test depending on the result of the Shapiro-Wilk test for normality of distribution which determined if data was parametric or non-parametric. Results were considered statistically significant at $P \leq 0.05$.

Results

Spatial learning and memory

The Morris Water Maze (MWM) test was performed after six weeks in four groups. The results showed that the T group spent the most time in the target zone across all test days compared to the other groups. The difference in time spent in the target area was significantly increased in the T group compared to the C group. Meanwhile, the DT group spent a similar amount of time in the target zone as the D group on the test day. In the final test, the T group spent the most time in the target quadrant overall. Spending more time in the target zone during the MWM test typically indicates better spatial learning and memory performance in rodents. However, in the long-term memory test, the DT group did not show significant differences in time spent in the target zone compared to the other groups, suggesting no changes in long-term memory.

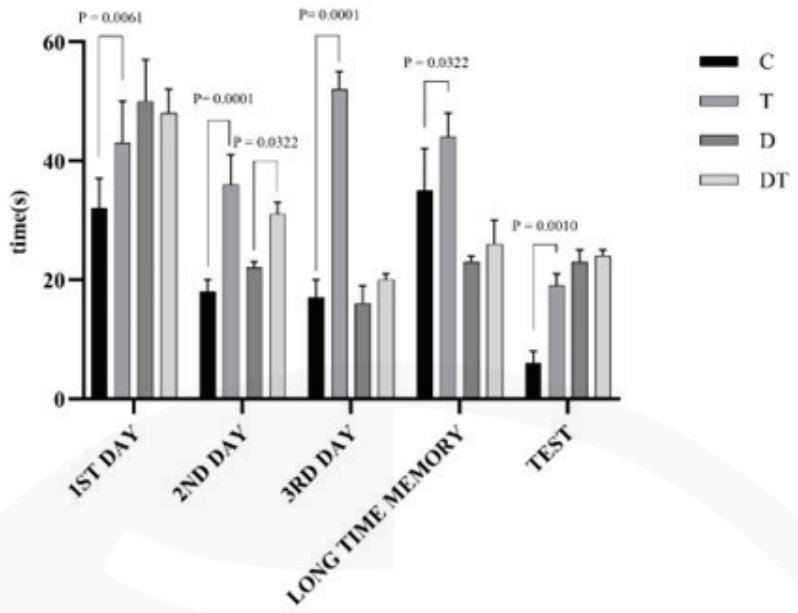

Figure 1. MWM test (time) in experimental groups, including control (C), Faradarmani Consciousness Field (T) Diabetic control (D), and Faradarmani Consciousness Field+diabetic (DT). As presented in Figure 1, the T group showed significantly higher time spent in the target zone on all days compared to the control group ($P \leq 0.05$). On the second day, the DT group also demonstrated a significant difference in time spent compared to the D group ($P = 0.0322$).

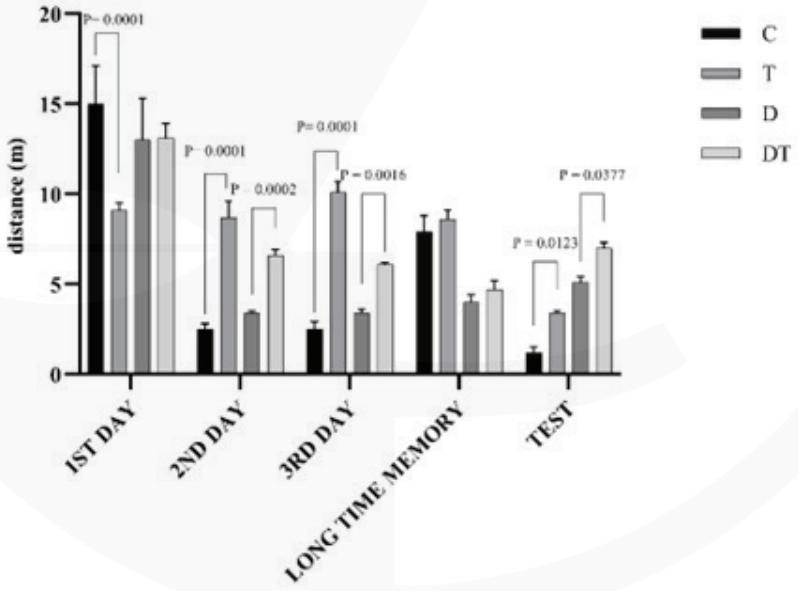

Figure 2: MWM test (distance) in experimental groups, including control (C), Faradarmani Consciousness Field (T) Diabetic control (D), and Faradarmani Consciousness Field+diabetic (DT). As presented in Figure 2, both healthy and diabetic Faradarmani group rats showed longer distance in the target range compared to the respective control groups ($P<0.05$). The difference in travel distance among the groups was not significant in the long-term memory test.

Pancreas tissue H&E staining

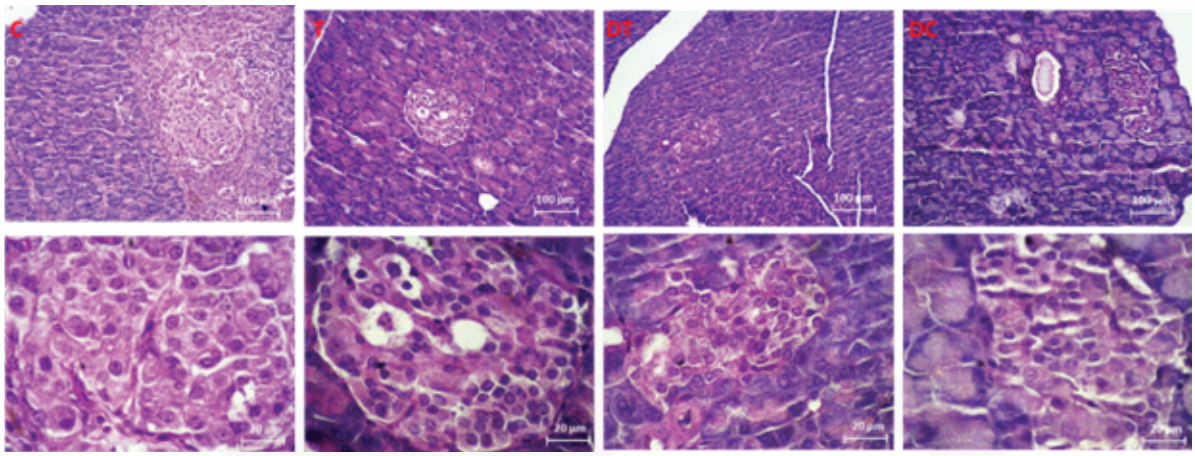

Figure 3. H&E staining of pancreas tissue in control (C), Faradarmani (T), diabetic + Faradarmani (DT), and diabetic control (D).

Images in groups C and T show the nucleus of beta cells in bold blue, while alpha cells are pink and scattered, mostly on the margins of islands and between beta cells (magnification x400). Based on the observed results, the rate of beta cell destruction and cellular apoptosis based on grading in control and T groups are 1% and 5%, respectively (Figure 5).

Images from the D and DT groups show a decrease in cell density. Degraded cells with nuclear contraction and pyknosis are more commonly seen in the center of the islands. Alpha cells with a pale pink nucleus are located on the islands and beta cells with an euchromatin nucleus are in the center of the island (magnification x400). According to the results observed in these groups, the rate of beta cell destruction and cellular apoptosis based on grading in the DT group was 50%, and in the D group, it was 70%, but the difference was not significant.

Fuchsin staining of pancreatic beta cells

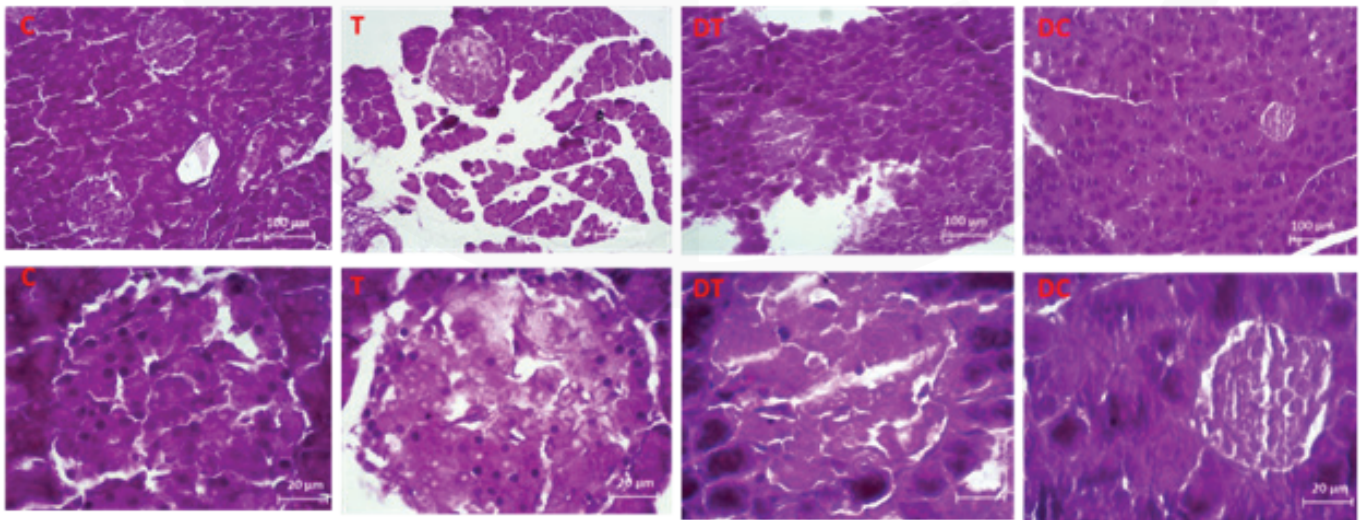

Figure 4. Fuchsin staining of pancreas tissue in control (C), Faradarmani (T), diabetic + Faradarmani (DT), and diabetic control (D)

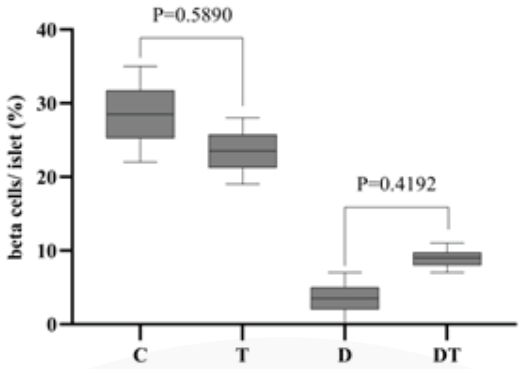

Figure 5. The number of beta cells per each islet in control (C), Faradarmani (T), diabetic + Faradarmani (DT), and diabetic control (D).

Based on the beta cell count in each area, the extent of pancreatic damage was estimated. Notably, the Faradarmani diabetic group exhibited a higher number of remaining healthy cells compared to the control diabetic group, although the difference was not statistically significant (P = 0.419). This suggests that the Faradarmani treatment may contribute to lower levels of destruction, potentially due to the effects of this field.

Hippocampus tissue H&E staining

Quantification of hippocampal damage across research groups revealed that the diabetic control group exhibited significantly higher proportions of cell death, exceeding 40% (P<0.05). The healthy control and Faradarmani control groups exhibited the lowest levels of damage (less than 20%) across all three areas of the hippocampus, with no significant difference between these groups. In the CA3 and DG regions of the hippocampus, the percentage of dead cells exhibited a significant decrease in the Faradarmani diabetic group compared to the diabetic control group, indicating the effectiveness of Faradarmani treatment in reducing cell death (DT: 25% and D: 45%) (P < 0.05) (Figures 6-14).

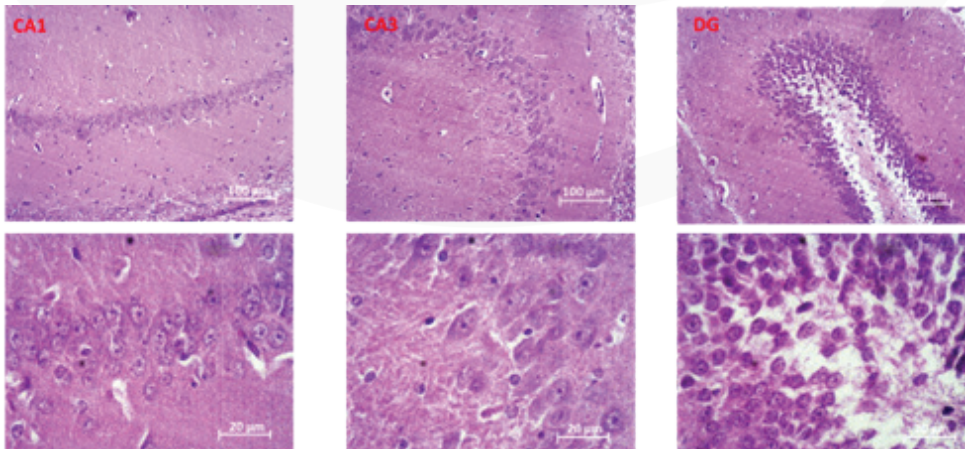

Figure 6. H&E staining of hippocampal tissue in three regions of CA1, CA3, and DG hippocampus of control group (C).

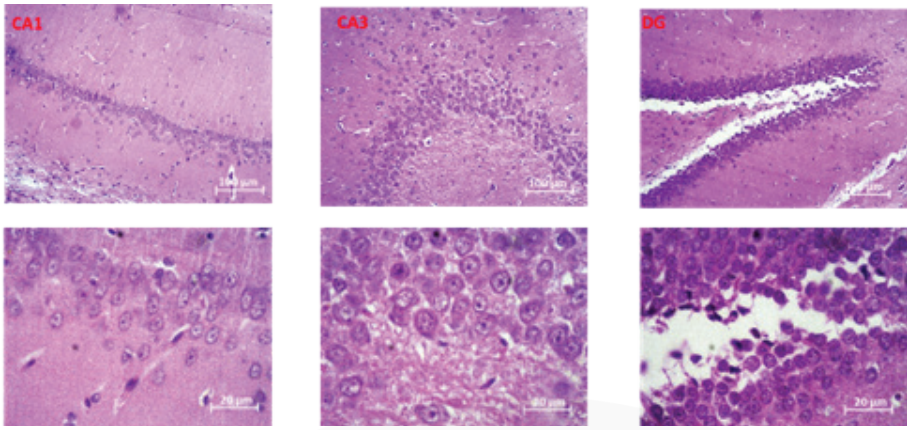

Figure 7. H&E staining of hippocampal tissue in three regions of CA1, CA3, and DG hippocampus of the Faradarmani group (T).

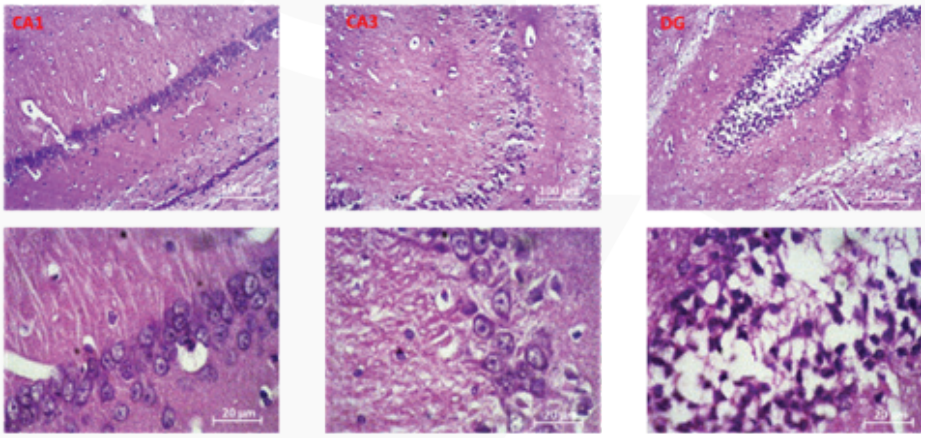

Figure 8. H&E staining of hippocampal tissue in the CA1, CA3, and DG regions of the hippocampus of the diabetic control group (D).

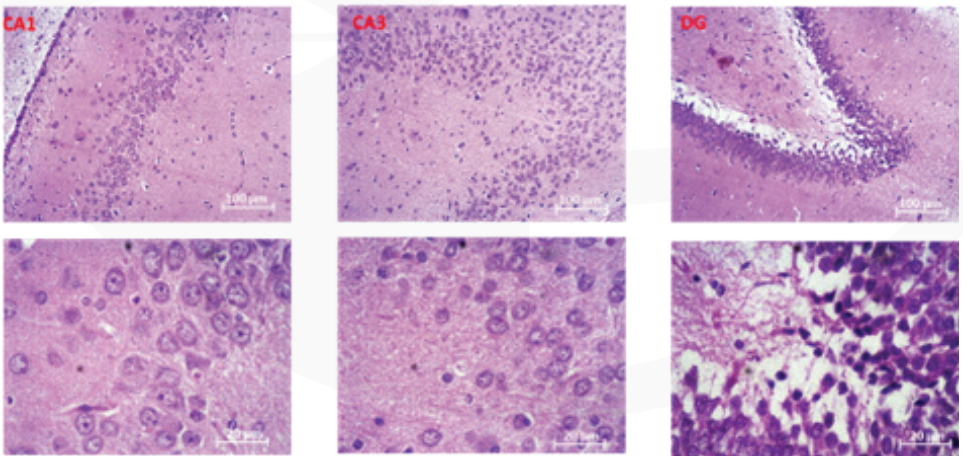

Figure 9. H&E staining of hippocampal tissue in the CA1, CA3, and DG regions of the hippocampus of the diabetic (DT) group

Hippocampus tissue Nissl staining

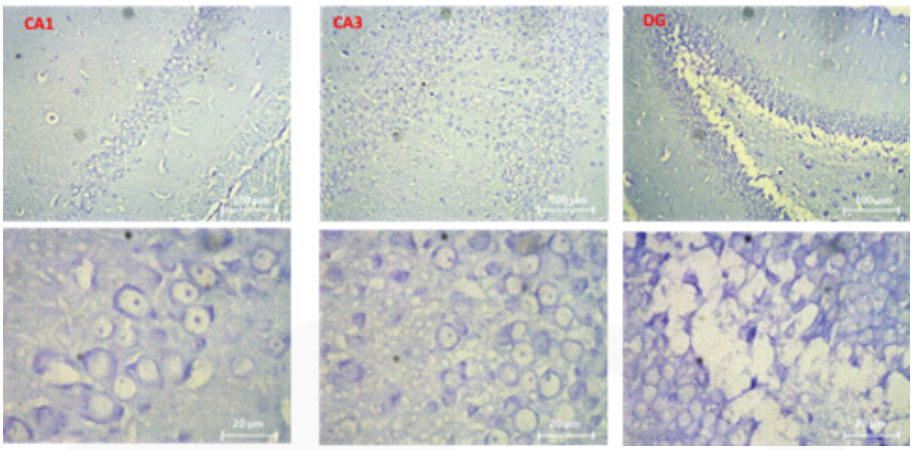

Figure 10. Nissel staining of hippocampal tissue in three regions of CA1, CA3, and DG hippocampus of control group (C).

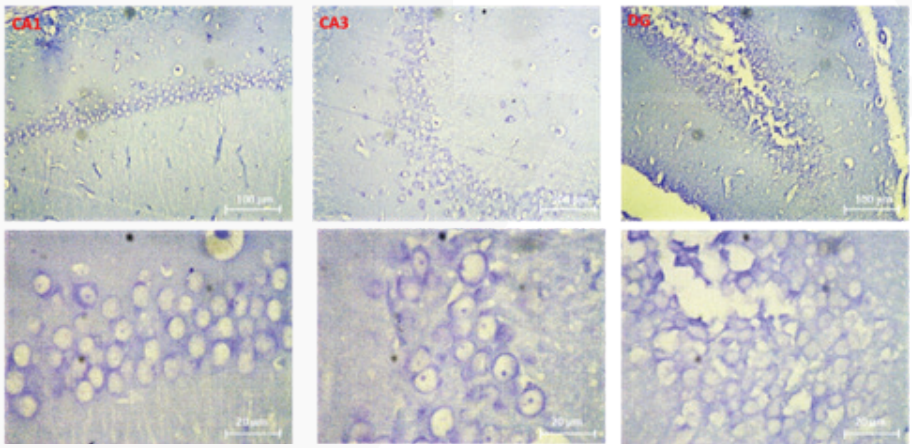

Figure 11. Nissel staining of hippocampal tissue in three regions of CA1, CA3, and DG hippocampus of Faradarmani group (T).

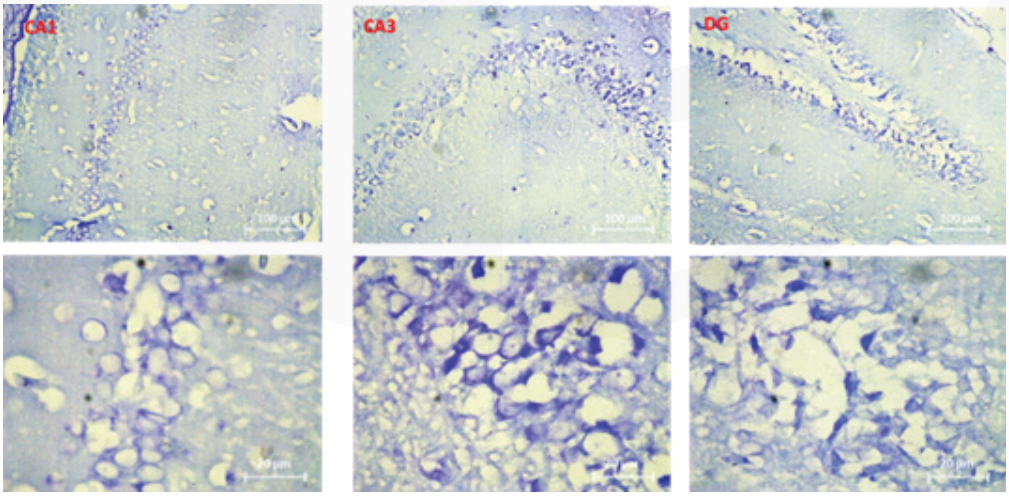

Figure 12. Nissel staining of hippocampal tissue in three regions of CA1, CA3, and DG hippocampus of the diabetic control group (D).

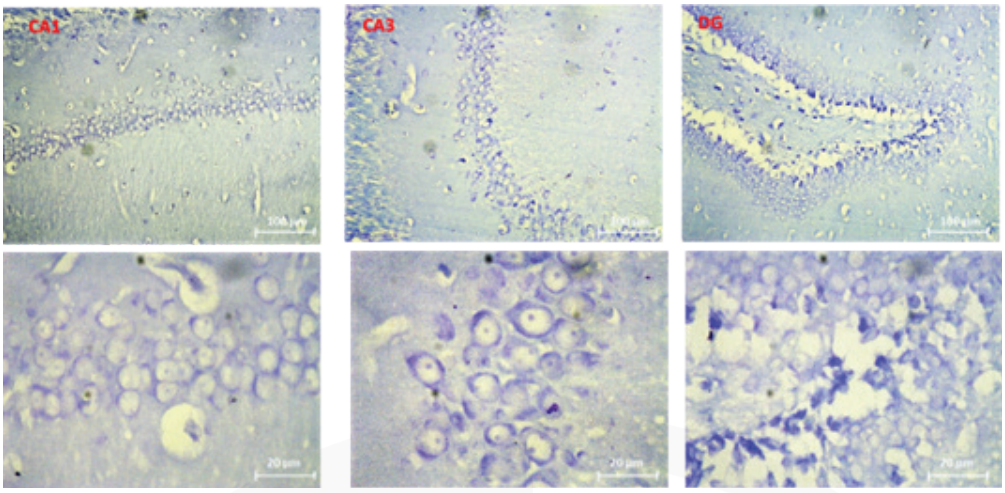

Figure 13. Nissel staining of hippocampal tissue in three regions of CA1, CA3, and DG of the hippocampus of the diabetic (DT) group.

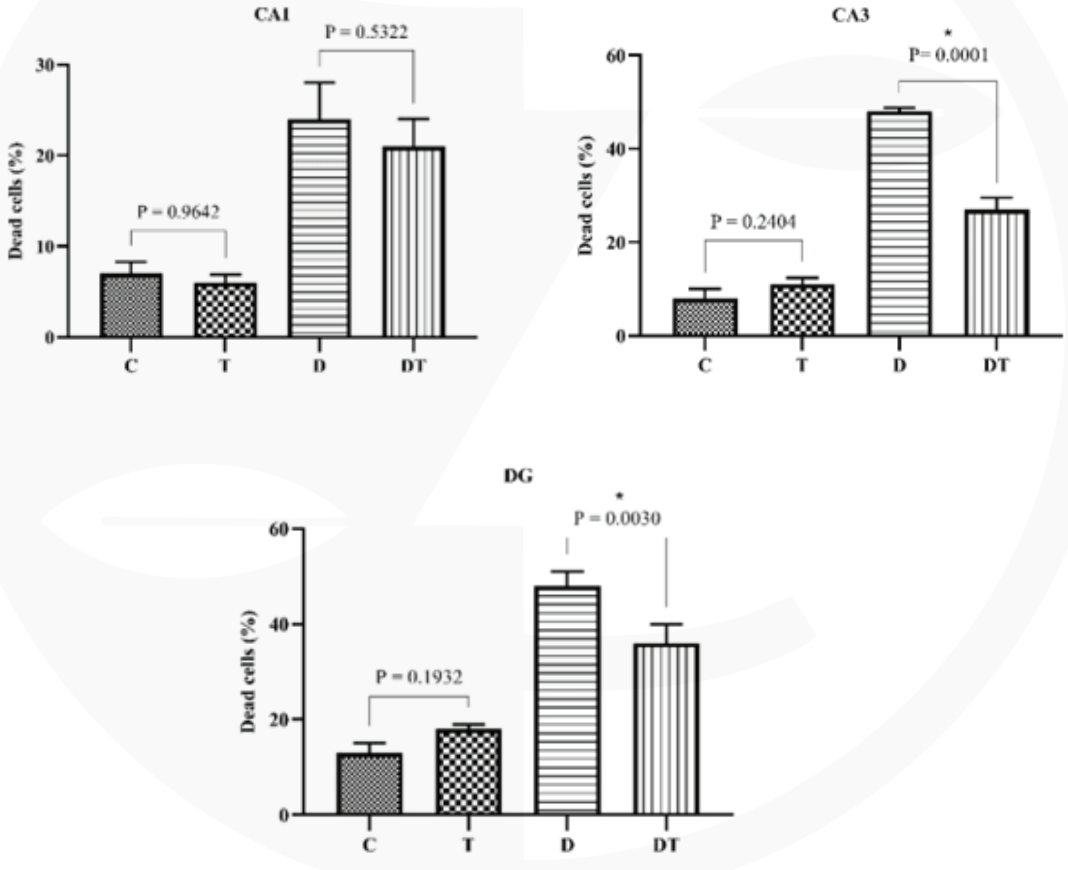

Figure 14. Percentage of dead cells in hippocampal tissue of CA1, CA3, and DG regions in control (C), Faradarmani (T), control diabetic (D), and diabetic Faradarmani (DT) groups.

The Figure 14 shows hippocampal damage between the diabetic control group (45%) and the diabetic Faradarmani group (25%), with a significant difference observed. There is a significant difference in the CA3 (P=0.001) and DG (P=0.030) areas, but not in CA1 (P=0.53). The difference between the control and treatment group (<20%) is not significant in the three areas of the brain (P>0.05).

Discussion

To investigate the effects of Faradarmani Consciousness Field on the modulation of diabetes consequences in the hippocampus, we considered three main assessments to discover cognitive, pancreatic and hippocampal changes among four experimental groups. At the structural level, when examining changes in cytoplasmic unspecified proteins with the H&E method, we observed insignificant changes in pancreatic beta cells and significant changes in hippocampal nerve cells in the diabetic group exposed to Faradarmani. β-cell regeneration can occur through two main mechanisms. The first involves stimulating the division of existing β-cells, but it is crucial to induce this proliferation in a cell-specific manner to prevent oncogenic transformation. The second mechanism involves cellular reprogramming, which can arise from stem-cell-like populations (directed differentiation) or from other terminally differentiated cell types (transdifferentiating) (Vetere et al., 2014).

Additionally, the number of hippocampal neuronal dead cells in the DG, CA3, and CA1 regions changed in diabetic rats exposed to Faradarmani. The results showed a significant decrease in dead cells in the DG and CA3 regions of the diabetic group compared to the control group. The healthy control and Faradarmani groups showed similar levels of dead cells (Figures 6-14). Treatment with Faradarmani caused improvement in the form of preservation of small pyramidal cells and markedly decreased apoptosis of large cells. However, the results showed that the treatment group did not exhibit significant changes in short and long-term memory compared to the control groups. After six weeks, the T group showed superior initial performance in the Morris Water Maze, spending more time in the target zone. In the final test, the T group also demonstrated prolonged time in the target zone, indicating enhanced spatial learning and memory but not significantly (Figure 1). However, the significant increase in the distance traveled by the T and DT groups, which is a marker of slow pace or inefficiency, was observed across all days (Figure 2).

The results of the spatial memory were in consistency with Taheri et al.'s (2021) studies in rat models of Alzheimer's disease. They found that treatment with scopolamine disrupted long-term memory, while treatment with Faradarmani CF improved memory function, similar to untreated controls. Both normal rats and AD models showed improved training and memory, enabling them to return to the maze platform. Additionally, rats treated with Faradarmani exhibited a decrease in swimming speed, indicating reduced overall stress levels (Taheri et al., 2021). A relevant review emphasizes that antidiabetic regimens can have beneficial effects on cognitive decline and memory impairment associated with diabetes. Treated groups may retain more information, potentially allowing them to navigate more effectively, even if they travel more slowly (Xourgia et al., 2019). This concept aligns with the idea that slower, more deliberate movement may facilitate better memory encoding and retrieval; however, this specific conclusion requires further empirical support from relevant studies. The observation of reduced travel speeds and extended durations among the Faradarmani groups may suggest enhanced memory encoding processes. However, additional research is required to validate this hypothesis.

The results of various studies have shown that diabetes causes structural and functional changes in the central and peripheral nervous system, including slowing down the conduction of nerve messages, disruption in the process of regeneration of peripheral nerves in the body and deformation of nerve fibers (Jackson-Guilford et al., 2000). It has also been shown that diabetes is one of the causes of memory impairment, which is one of the symptoms of Alzheimer's disease (Biessels et al., 2006).

The improvements in neural function observed in the DG and CA3 regions following treatment in diabetes could be attributed to several potential mechanisms (Figure 14). One possible

factor is the reduction of oxidative stress, as oxidative damage to neurons is known to contribute to cognitive impairment in diabetes (Kuhad et al., 2008). Additionally, the anti-inflammatory effects of the treatment may play a role, as chronic inflammation is a hallmark of diabetes-related cognitive decline (Lee & Jun, 2016). Furthermore, the treatment might enhance neurotrophic support, as neurotrophic factors are crucial for neuronal survival and function, particularly in the hippocampus (Eyileten et al., 2017). These mechanisms, either independently or in combination, could contribute to the observed improvements in neural function in the DG and CA3 regions in diabetic individuals following treatment. However, Further experiments are required to explore how Faradarmani may directly or indirectly influence these mechanisms, as suggested by our results.

The damage observed included hippocampal regions or beta cells destruction and necrosis, which is consistent with previous studies (Eizirik et al., 2020; Mohajeri et al., n.d.; Wang et al., 2021). The effects of TCFs on molecular and cellular reprogramming in different living subjects have been proven in different studies (Taheri et al., 2020; Taheri, Modarresi-Asem, et al., 2022; Taheri, Payervand, et al., 2022). These results are consistent with the study conducted by Taheri *et al.* (2020) to investigate the effects of Faradarmani on breast cancer cells. They showed the rate of cell death and apoptosis molecular markers decreased after TCFs treatment (Taheri et al., 2020).

According to the theory of TCFs, the mechanism of Faradarmani application can facilitate the reprogramming and regeneration process of impaired cells by establishing a connection between the subjects under study and the Cosmic Consciousness Network (CCN). Through this connection, required data and information are transmitted from the CCN to the impaired organs or cells of the subjects. As a result, structural or functional improvements can be observed, in accordance with the general rules of the ecosystem.

In conclusion, this study provides evidence suggesting that Faradarmani, as a type of TCFs introduced by Taheri, may have beneficial effects on hippocampal and pancreatic health in diabetic individuals. The observed improvements in neural function in the DG and CA3 regions, along with the regeneration of pancreatic beta cells, indicate the potential therapeutic benefits of Faradarmani. These effects may be mediated through mechanisms such as cellular reprogramming and neurotrophic support. However, further research, including long-term studies and clinical trials, is warranted to fully elucidate the mechanisms and long-term effects of Faradarmani in diabetes management. Overall, these findings contribute to our understanding of TCFs treatment and their potential role in improving health outcomes in diabetic individuals.

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