

# Investigation of the Effect of Faradarmani Consciousness Field on *NF-κB* and Proinflammatory Factors Genes Expression Using Blood Samples of Patients with COVID-19

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## Abstract

The coronavirus disease 2019 (COVID-19) pandemic has become a worldwide challenge. Severe respiratory distress syndrome (ARDS) caused by coronavirus has often been associated with a cytokine storm. The Faradarmani Consciousness Field (CF), as one of many Taheri Consciousness Fields (TCFs) introduced by Mohammad Ali Taheri, is a novel field that is neither matter nor energy. Therefore, they are non-quantifiable and cannot be directly observed or measured; however, their effects can be demonstrated and measured through standard scientific experiments. The current study aimed to investigate the effect of Faradarmani CF on inflammatory factors in blood samples of patients with COVID-19. Blood samples were taken from 53 patients with COVID-19, and Faradarmani CF was applied to the blood samples of the treatment group. Then, the mRNA expression of *NF-κB*, *TNF-α*, *IL-1β*, and *IL-6* genes was examined in the treated and control (non-treated) blood samples. The results indicated that under the influence of Faradarmani CF, the expression of *NF-κB* and *IL-1β* genes was significantly decreased ( $p < 0.05$ ), the *TNF-α* gene expression was significantly increased ( $p < 0.05$ ), but the expression of *IL-6* did not show any significant change ( $p > 0.05$ ). We conclude that immunomodulation at the level of *NF-κB* and *IL-1β* by Faradarmani CF may inhibit the cytokine storm in COVID-19 patients. However, further studies are required to identify all effects of this T-Consciousness Field.

**Keywords:** COVID-19, Faradarmani, Taheri Consciousness Fields, T-Consciousness, Proinflammatory factors

## Introduction

COVID-19 is a newly emerged respiratory disease in which most patients display mild to moderate symptoms. However, about 15% of the patients develop severe pneumonia, and about 5% finally progress to acute respiratory distress syndrome (ARDS), septic shock, and multiple organ failure (Huang et al., 2020; Xu et al., 2020).

Symptom management and oxygen therapy with ventilators are the primary clinical treatments for cases of respiratory failure. Although numerous antiviral drugs have been actively explored, none has been approved as an effective cure for COVID-19. In addition to vaccine production and methods that target the virus directly or prevent its transmission, treatments that address the disease's immunopathology have received considerable attention. COVID-19 infection can stimulate adaptive and innate immune responses. Nevertheless, impaired adaptive immune responses and uncontrolled innate inflammatory responses may cause harmful tissue damage, both systemically and locally (Cao, 2020).

Activation of the transcription factor NF-kappa B (NF- $\kappa$ B) by COVID-19 leads to the production of TNF- $\alpha$ , IL-1, IL-2, IL-6, IL-12, GM-CSF, LT- $\alpha$ , LT- $\beta$ , and various chemokines (Hariharan et al., 2020). The mortality of COVID-19 patients has been linked to cytokine storms induced by the virus (Ragab et al., 2020). Some of the common clinical features of cytokine storm syndrome are splenomegaly, sustained fever, hepatomegaly, coagulopathy, skin rash, etc. (Shimizu, 2019). Another consequence of the cytokine storm is lung injury and multi-organ failure (Sun et al., 2020). Acute Respiratory Distress Syndrome (ARDS), which leads to low oxygen saturation levels, is the main cause of death in COVID-19. Although the precise mechanism of ARDS is not entirely understood, the overproduction of proinflammatory cytokines can be one of the most determining factors in the disease severity of patients with COVID-19 (Huang et al., 2020; Ragab et al., 2020; Lai et al., 2020).

According to Taheri's theory, there are various T-Consciousness Fields (TCFs) with different functions that are a subset of the Cosmic Consciousness Network (CCN). These fields are not material or energetic in nature; therefore, they cannot be measured using quantitative tools. It is possible to investigate the effects of these fields by designing experiments (Taheri, 2013).

In previous research, the effects of the TCFs on the MCF7 cancer cell line (Taheri et al., 2020a), spatial memory and avoidance behavior of a rat model of Alzheimer's disease (Taheri et al., 2021a), wheat plant (Torabi et al., 2020), bacterial population growth (Taheri et al., 2021b), and the electrical activity of the brain during Faradarmani Connection in the Faradarmangars population (Taheri et al., 2020b) have been investigated. In the present study, the effect of Faradarmani CF on the expression level of the *NF-kB* gene and pro-inflammatory factors *IL-1 $\beta$* , *IL-6*, and *TNF- $\alpha$*  in blood samples isolated from patients with COVID-19 infection was investigated.

## Material and Methods

### Application of Faradarmani Consciousness Field

Faradarmani CF was applied to the samples according to the protocols regulated by the COSMOintel research center ([www.COSMOintel.com](http://www.COSMOintel.com)). More details are provided in the Common Considerations section of this issue. In this study, two blood samples from each of 53 patients with COVID-19 were collected. All samples were stored at -72 °C until analysis. After collecting all samples, one group was treated with Faradarmani CF, and the other group was left untreated as a control. The RNA expression level of *NF-kB*, *TNF- $\alpha$* , *IL-1 $\beta$* , and *IL-6* genes was evaluated in the blood samples of Faradarmani CF and control groups.

## Preparation of cells and evaluation of expression levels of *NF-κB*, *TNF-α*, *IL-1β*, and *IL-6* genes

RNA extraction was performed using FavorPrep Blood/Cultured Cell Total RNA Mini Kit (Favorgen) according to the manufacturer's protocol. The concentration of RNA was measured using a NanoDrop spectrophotometer (Thermo Fisher).

A cDNA synthesis kit (BioFACT) was used to synthesize the cDNA. The reaction mixture was prepared by mixing 1,000 ng of the extracted RNA, 1 μL of Random Hexamer primer, 1 μL of oligo(dT), and 10 μL of reverse transcription (RT), and the total volume was made up to 20 μL with RNase-free water. According to the synthesis protocol, total solutions were

incubated at 95°C for 5 minutes, then cDNAs were prepared at 50°C for 40 minutes.

Real-time PCR was performed using a reaction mixture with a total volume of 15 μL consisting of 7.5 μL of 2× real-time PCR master mix (for SYBR-Green I; BioFACT), 1.5 μL of cDNA products, 0.6 μL of each forward and reverse primers, and 4.8 μL of sterile water to assess the RNA expression of the genes. The primer sequences used in this study are listed in Table 1.

The thermal cycling conditions were 95°C for 10 minutes followed by 40 cycles of 95°C for 15 seconds, annealing temperature for 25 seconds, and 72°C for 30 seconds. The values for the relative quantification were calculated via the  $2^{-\Delta\Delta C_q}$  expression formula.

Table 1. Sequences of the oligonucleotide primers used in this study

Genes	Primer sequences (5'→3')	Product length (bp)	Annealing temperature (°C)
<i>IL-1β</i>	F: CAGAAGTACCTGAGCTCGCC R: AGATTCGTAGCTGGATGCCG	153	55
<i>IL-6</i>	F: CTTTCGGTCCAGTTGCCTTCT R: GATGCCGTCGAGGATGTACC	169	55
<i>NF-κB</i>	F: ACCAGCCTCTGTGTTTGTCC R: CACTACCACCGCCGAAACTA	161	57
<i>TNF-α</i>	F: TCTCTCGAACCCCGAGTGA R: TATCTCTCAGCTCCACGCCA	126	61
<i>GAPDH</i>	F: GTGGTCTCCTCTGACTTCAAC R: GGAAATGAGCTTGACAAAGTGG	96	60

## Statistical analysis

Data are presented as mean and standard deviation. A Student t-test was used to compare differences between the Faradarmani CF and the control groups. Statistical analysis was performed via GraphPad Prism8, and a *p-value* of <0.05 was considered statistically significant.

## Results

In the present study, the effect of Faradarmani CF on the expression levels of *NF-κB* and proinflammatory factors (*IL-1β*, *IL-6*, and *TNF-α*) was assessed in isolated blood samples from patients with COVID-19 using real-time RT-PCR. The results revealed that the expression of *NF-κB* and *IL-1β* genes was significantly decreased ( $p < 0.05$ ), whereas *TNF-α* expression was significantly increased ( $p < 0.05$ ), but *IL-6* expression did not differ significantly ( $p > 0.05$ ) (Figure 1).

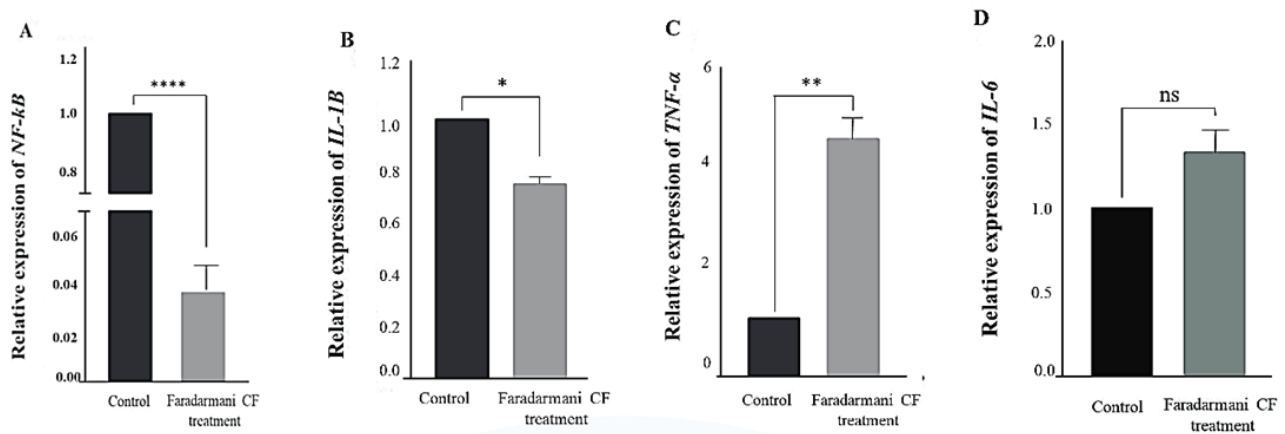


Figure 1. The expression level of evaluated genes in control and Faradarmani CF treatment on blood samples of patients with COVID-19 infection. A) *NF-κB*, B) *IL-1β*, C) *TNF-α*, and D) *IL-6* genes. The asterisk (\*) displays a significant difference ( $p < 0.05$ ) between the Faradarmani CF treatment and control groups.

## Discussion

The specific signaling pathways mediated during inflammatory responses in *HCoV*-infected patients have not been fully identified (Channappanavar et al., 2017; Battagello et al., 2020). However, several serum cytokines in COVID-19 patients have been reported to be higher than in healthy adults (Huang et al., 2020). A previous study suggested that among the signaling pathways activated by viruses, *NF-κB* plays an important role in inducing the expression of genes encoding cytokines and chemokines (Mogensen & Paludan, 2001). During the COVID-19 pandemic, research has associated the mortality rate among COVID-19 patients with cytokine storms (Mehta et al., 2020; Ruan et al., 2020). Anti-cytokine therapies, such as IL-6 receptor (Radbil et al., 2020; Xu et al., 2020) or IL-1 receptor antagonist (Cavalli et al., 2020; Conti et al., 2020), have been proposed for the treatment of COVID-19. The results of our research indicate that the expression of *NF-*

*κB* and *IL-1β* genes was significantly decreased under the influence of Faradarmani CF.

The results of the present study showed that the Faradarmani CF affects the expression of *NF-κB*, *TNF-α*, and *IL-1β*, proinflammatory genes, in isolated blood samples from COVID-19 patients. Further studies are recommended to explore the mechanism and extent of the favorable effect of this TCF in the treatment of COVID-19.

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## Conflicts of Interest

The authors declare no conflict of interest.

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