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Original Article

Daegeum music therapy alleviates depression-like behaviors via the restoration of monoaminergic and neurotrophic systems in mice

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ABSTRACT

Objective: Chronic stress is a primary precipitating factor for depressive disorders, linked to central monoaminergic deficits, impaired neuroplasticity, and systemic inflammation. This study investigated the antidepressant-like efficacy and underlying molecular mechanisms of Daegeum music therapy (DMT), a traditional Korean bamboo flute acoustic intervention, in a mouse model of depression.

Materials and Methods: Mice were subjected to chronic stress and exposed to daily DMT for 3 weeks.

Results: Behavioral assessments demonstrated that DMT significantly reduced immobility times in the forced swimming test without altering general locomotor activity. Notably, this behavioral rescue was acoustically specific to DMT, as white noise and Buk music failed to alleviate behavioral despair. Mechanistically, DMT remarkably prevented stress-induced neurotransmitter deficits by restoring serotonin and dopamine levels within the hippocampus. Furthermore, Western blot analysis revealed that DMT robustly up-regulated hippocampal brain-derived neurotrophic factor expression and selectively promoted extracellular signal-regulated kinase phosphorylation, thereby stimulating neuroplastic signaling cascades. Beyond central remodeling, DMT exerted potent peripheral anti-inflammatory effects by down-regulating pro-inflammatory cytokines.

Conclusion: Taken together, these findings demonstrate that DMT effectively mitigates depressive-like behaviors by restoring hippocampal monoaminergic homeostasis, stimulating neuroplastic cascades, and suppressing systemic inflammatory pathways, highlighting its therapeutic potential as a non-pharmacological acoustic intervention.

Keywords DMT, depression, serotonin, neuroplasticity, BDNF, pro-inflammatory cytokines

INTRODUCTION

Chronic stress is an inevitable consequence of the modern world, precipitating a wide array of physical and psychological disorders. Depression remains a debilitating medical condition affecting a significant portion of the global population, with recent epidemiological data identifying it as a leading contributor to the global burden of disease.[1] Although conventional treatments primarily rely on a combination of chemical pharmacotherapy and psychiatric counseling, long-term pharmacological interventions often present compliance issues and adverse side effects.[2] Consequently, non-pharmacological modalities, such as structured music therapy, have recently gained substantial recognition as effective alternative strategies for modulating emotional states, regulating autonomic functions, and mitigating depressive symptoms.[3,4]

The pathophysiology of depression is heavily anchored in monoaminergic neurotransmission, where behavioral despair strongly correlates with deficiencies in serotonin (5-hydroxytryptamine, 5-HT) and dopamine (DA) within the central nervous system.[5] Central monoaminergic signaling acts as a vital upstream regulator of brain-derived neurotrophic factor (BDNF) expression, particularly within the hippocampus, which serves as a critical neural substrate for neuroplasticity, memory, and emotional processing.[6,7] This neurotrophic support activates downstream intracellular cascades, notably the extracellular signal-regulated kinase (ERK) pathway, driving synaptic

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remodeling essential for therapeutic recovery.[8] However, chronic stress triggers microglia-mediated neuroinflammatory responses, wherein elevated pro-inflammatory cytokines such as interleukin (IL)-1β, IL-6, and tumor necrosis factor-α disrupt and inhibit this critical hippocampal BDNF-ERK signaling axis.[9,10] Hence, restoring hippocampal monoaminergic homeostasis and rescuing neuroplastic signaling pathways from systemic inflammation represent core therapeutic targets in contemporary depression research.

The Daegeum, a traditional Korean transverse bamboo flute, possesses unique acoustic properties distinct from other wind instruments, primarily due to its resonant membrane called 'Cheong'. The vibration of the Cheong introduces rich higher-order harmonics, dynamic microtonal pitch variations, and a characteristic buzzy timbre.[11] Biophysically, these complex dynamic harmonic profiles and gentle acoustic fluctuations are known to facilitate neural entrainment, which aligns central neural oscillations with external auditory rhythms, thereby promoting autonomic balance and neural stabilization.[12] Despite these compelling acoustic mechanisms, the empirical efficacy and neurobiological substrates of Daegeum music therapy (DMT) in treating depressive disorders remain systematically unexplored.

In this study, we investigated the antidepressant-like efficacy of DMT utilizing a stress-induced mouse model. To validate the therapeutic potential of this acoustic intervention and its underlying biological substrates, we evaluated behavioral alterations through the forced swimming test (FST) and open field test (OFT). Furthermore, we analyzed the mechanistic capacity of DMT to regulate hippocampal monoamine levels, stimulate neuroplastic BDNF-ERK signaling cascades, and suppress systemic inflammatory cytokine profiles.

MATERIALS AND METHODS

Animals: Male ICR mice (3 weeks old) weighing 10-12 g were purchased from the Dae-Han Experimental Animal Center (Daejeon, Republic of Korea), and subsequently maintained at the College of Korean Medicine, Kyung Hee University. Animals were housed 5-10% in a laminar air-flow room maintained at a temperature of 22 ± 1℃ and a relative humidity of 55 ± 10% under a 12:12 L/D cycle light (on at 7:00 h) throughout the study. Food and water were provided ad libitum. All manipulations were carried out between 9:00 and 16:00 h and no animal was used more than once. The study protocol was approved beforehand by the institutional animal care use committee of Kyung Hee University (KHUASP(SE)-10-032).

Music treatment: ICR mice were randomly assigned to seven groups according to the experimental design: an untreated control group (Blank), a fluoxetine-treated group (positive control), a white noise group (WN), a Korean traditional drum music group (Buk), and three DMT groups with different exposure durations (10, 20, and 30 min). The background noise level inside the specially designed acoustic isolation chambers used for the Blank and fluoxetine groups was maintained at 10 - 40 dB SPL, while the sound pressure level for the WN, drum music, and DMT groups was maintained at 70 dB SPL (A-weighted). Mice were exposed daily to the designated acoustic stimuli for 3 weeks. All experiments were conducted in a quiet environment with minimal external acoustic interference. To ensure consistent delivery of the acoustic stimuli across all treatment groups, the daegeum performance by Professor Hyung-Min Kim was recorded using a high-resolution recording system and converted into high-quality digital audio files (.wav format, 24-bit/96 kHz). The daegeum sound primarily consisted of fundamental frequency components in the low-frequency range (approximately 250 Hz - 1 kHz), with higher-order harmonic components extending up to approximately 8 kHz. The recorded audio files were subsequently played back to each group of mice under strictly controlled experimental conditions. The acoustic stimuli were delivered through a full-range dynamic speaker (Britz International Co., Ltd., Paju, Republic of Korea) positioned 20 cm vertically above the wire mesh lid of each cage inside the acoustic isolation chamber. The sound intensity and delivery conditions were consistently maintained across all experimental groups to minimize variations in acoustic exposure.

FST: After measuring immobility times, 10 mice were allocated to each of the nine study groups in an immobility time-matched manner. Fluoxetine was dissolved in distilled water. Fluoxetine (10 mg/kg, a classical antidepressant) was orally administered to mice once daily for 3 weeks using an atraumatic feeding needle. The FST was performed at the end of the 3-week administration period. During the 6 min FST, immobility times were measured as described by Jeong et al.[13]. The FST apparatus consisted of two Plexiglas cylinders (height: 25 cm, diameter: 10 cm) placed side by side in a Makrolon cage filled with water at 23-25°C. Two mice were tested simultaneously for 6 min period inside the vertical Plexiglas cylinders; a nontransparent screen was placed between the two cylinders to prevent mice from seeing each other during the test. Total duration of immobility, during the 4 min period from 2 to 6 min was recorded. A mouse was considered immobile when it ceased struggling and remained floating motionless on the water, making only movements necessary to keep its head above water.

OFT: The OFT was conducted in clear black Plexiglas boxes (40 × 40 × 40 cm) equipped with the video-based Ethovision System (Nol-dus, Wageningen, The Netherlands), as described previously.[13] Mice were initially placed in the corner of the apparatus, and total distance moved was recorded for 30 min. The horizontal locomotor activity is expressed in terms of the total ambulatory distance (cm). Each box was cleaned with 70% ethanol between subjects.

Isolation of hippocampal tissues and protein extraction: Immediately following the FST, mice were sacrificed via cervical dislocation, and the brains were rapidly excised and chilled on an ice-cold brain matrix. To isolate the hippocampus, anatomical landmarks were utilized; the brain was micro dissected on a chilled stage, during which the cerebral cortex was

gently peeled back to expose the dorsal and ventral aspects of the hippocampus. The isolated hippocampal tissues were immediately snap-frozen in liquid nitrogen and stored at -80°C until further processing. For total protein extraction, the frozen hippocampal samples were thawed on ice and homogenized in an ice-cold lysis buffer supplemented with 0.2 mM dithiothreitol, 0.5 mM sodium vanadate, and a cocktail of protease and phosphatase inhibitors. To ensure complete nuclear and cellular lysis, NaCl was adjusted to a final concentration of 0.45 M. The resulting homogenates were centrifuged at 15,000 x g for 30 min at 4°C. The supernatants containing the soluble protein fraction were carefully collected and stored at -80°C for subsequent biochemical analyses

Hippocampal 5-HT and DA Assays: To quantify monoaminergic neurotransmitter levels within the emotional center of the brain, the extracted hippocampal samples were subjected to enzyme-linked immunosorbent assays (ELISA). 5-HT levels in the hippocampal homogenates were measured using a commercial 5-HT assay kit (MyBiosource, San Diego, CA, USA) according to the manufacturer's instructions. Concurrently, DA concentrations within the hippocampal tissues were quantified utilizing a specific DA ELISA kit (Genway Biotech Inc., San Diego, CA, USA).

Western Blot Analysis: For Western blot analysis, three biological replicates (n = 3 per group) were randomly selected from the original cohort (n = 3 per group) using a computer-generated randomization sequence. To prevent potential selection bias, the sample selection process was performed by an independent investigator blinded to the experimental group allocations. Equal amounts of hippocampal protein lysates were resolved by sodium dodecyl sulfate-polyacrylamide gel electrophoresis and transferred onto polyvinylidene difluoride membranes. The membranes were then subjected to Western blot analysis using specific primary antibodies (Santa Cruz, CA, USA) directed against BDNF (Santa Cruz Biotechnology, Cat# sc-546, 1:500 dilution), ERK (Santa Cruz Biotechnology, Cat# sc-271269, 1:500 dilution), and phosphorylated-ERK (Santa Cruz Biotechnology, Cat# sc-7383, 1:500 dilution). Bands were detected using enhanced chemiluminescence (GE Healthcare, Piscataway, NJ, USA). Glyceraldehyde 3-phosphate dehydrogenase (Santa Cruz Biotechnology, Cat# sc-32233, 1:500 dilution) was utilized as the internal loading control to normalize the expression levels of the target hippocampal proteins.

ELISA: Cytokine levels in serum were measured by ELISA kits (BD Biosciences Pharmingen, San Diego, CA, USA). The ELISA was performed by coating 96-well plates with 1 μg/well of capture antibodies for IL-1β, TNF-α, and IL-6. Before the subsequent steps in the assay, the coated plates were washed twice with phosphate-buffed saline with 0.05% Tween 20. All reagents and coated wells used in this assay were incubated for 2 h at room temperature. The standard curve was generated from known concentrations of cytokine, as provided by the manufacturer. After exposure to the medium, the assay plates were exposed sequentially to each of the biotin-conjugated secondary antibodies, and avidin-peroxidase. Finally, the substrate was added to each well, and the plate was read at 405 nm using a SpectraMax ABS ELISA microplate reader to measure the absorbance.

Statistical analysis: Results are presented as means ± SEMs. The independent t-test and ANOVA with Tukey's post hoc test were used to determine statistical significance. SPSS statistical software (version 12.0.1; IBM Corp., Armonk, NY, USA) was used throughout, and statistical significance was accepted for p values < 0.05.

RESULT

Effect of DMT on immobility and locomotor activity

To evaluate the antidepressant-like efficacy of DMT, we assessed behavioral changes in mice using the FST and the OFT. In the FST, the baseline (day 0) immobility times showed no significant differences among all experimental groups, confirming a homogenous baseline status across the cohorts (Fig.

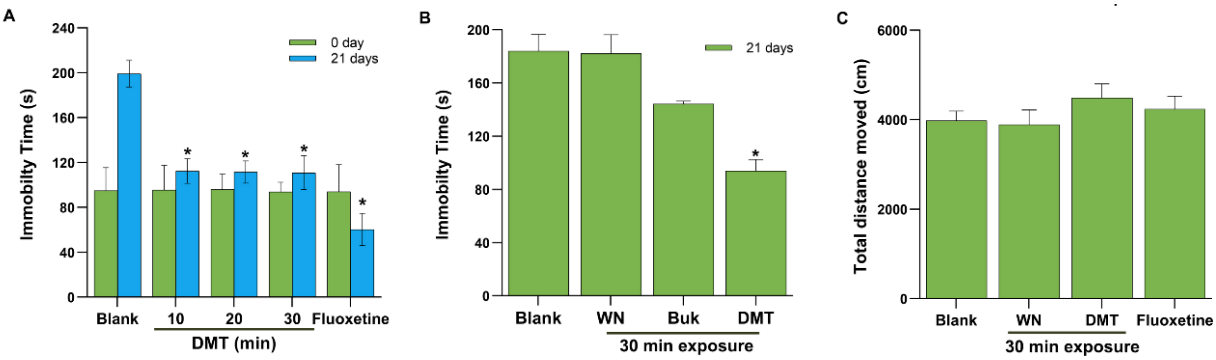

Fig. 1. Effects of DMT on immobility and locomotor activity in mice. Mice were exposed to DMT, WN, and Buk music, or administered fluoxetine daily for 3 weeks, and behavioral testing was performed 1h after the final treatment. (A-B) Immobility times measured during the FST. (C) Locomotor activity evaluated during the OFT for 30 min, represented as the total distance moved. Values are expressed as means ± SEMs (n = 5 per group). *p < 0.05 versus the blank group. WN, white noise; Buk, traditional drum music; DMT, Daegeum music therapy.

1A). Following the 3-week intervention period, however, the untreated control group exhibited a prominent increase in immobility time induced by repeated stress. In contrast, daily treatment with either fluoxetine or DMT significantly shortened the immobility duration compared to the untreated control group (Fig. 1A, $p < 0.05$), demonstrating a robust antidepressant-like effect.

To determine the acoustic specificity of this therapy, the effects of DMT were compared with other sound exposures (Fig. 1B). While WN and Buk groups failed to alter the immobility times, DMT treatment significantly reduced the immobility duration compared to both the untreated control and the WN group (Fig. 1B, $p < 0.05$).

Furthermore, to verify whether the observed reduction in immobility was secondary to a generalized psychostimulatory effect or hyperactivity, the OFT was conducted (Fig. 1C). There were no significant differences in the total distance moved among the DMT, fluoxetine, and WN groups (Fig. 1C, $p < 0.05$).

Effect of DMT on hippocampal 5-HT and DA levels

To determine whether the antidepressant-like effects of DMT are linked to the regulation of central monoaminergic neurotransmission, we analyzed 5-HT and DA levels in the mouse hippocampus following the FST. As shown in Figure 2A, daily intervention with DMT remarkably prevented this stress-induced neurotransmitter deficit, restoring 5-HT levels to 6.1 ± 0.7 ng/ml compared to the blank group (4.0 ± 0.8 ng/ml). Similarly, the positive control group treated with fluoxetine showed a robust elevation in hippocampal 5-HT levels (7.1 ± 1.4 ng/ml) compared to the blank group (Fig. 2A, $p < 0.05$).

In parallel, hippocampal DA levels exhibited a similar pattern of stress-induced reduction and therapeutic recovery (Fig. 2B). In the FST, DMT significantly attenuated behavioral despair, restoring hippocampal DA levels to 250 ± 50 (ng/ml) compared to the blank group (160 ± 40 ng/ml, Fig. 2B, $p < 0.05$). Furthermore, fluoxetine administration effectively maintained higher DA levels (275 ± 40 ng/ml) compared to the blank group (Fig. 2B, $p < 0.05$).

Stimulation of neuroplastic signaling cascades by DMT

To elucidate the molecular framework underlying the observed behavioral improvements, we focused on neurotrophic support and its downstream intracellular signaling essential for synaptic remodeling within the hippocampus. Western blot analysis demonstrated that the hippocampal expression of BDNF, a hallmark mediator of neuronal survival and synaptic plasticity, was robustly upregulated following chronic DMT exposure compared to both the blank and WN groups (Figs. 3A and 3B, $p < 0.05$). Notably, this neurotrophic enrichment in the DMT group was comparable to the pattern observed in the fluoxetine-administered positive control group (Figs. 3A and 3B, $p < 0.05$).

To map the precise signal transduction pathway activated by this elevated BDNF, we subsequently scrutinized the phosphorylation status of ERK in the hippocampus. Mice subjected to DMT exhibited a striking, selective promotion of hippocampal ERK phosphorylation (pERK), delivering a statistically significant increase compared to the vehicle-treated stressed cohort (Fig. 3C, $p < 0.05$). Crucially, the total pools of ERK protein remained completely unaffected and uniform across all experimental boundaries.

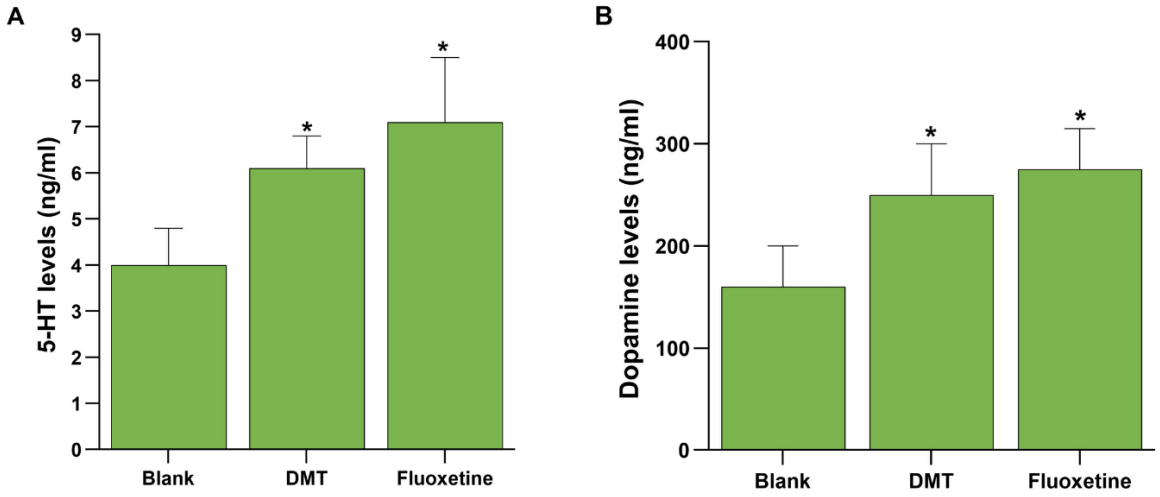

Fig. 2. Effect of DMT on hippocampal 5-HT and DA levels. DMT or fluoxetine was administered for 3 weeks and testing was conducted 1 h after final treatments. After the FST, (A) 5-HT and (B) dopamine levels in the hippocampus were measured using assay kits. Values are expressed as means ± SEMs (n = 5 per group). *$p < 0.05$ versus blank group. DMT, Daegeum music therapy.

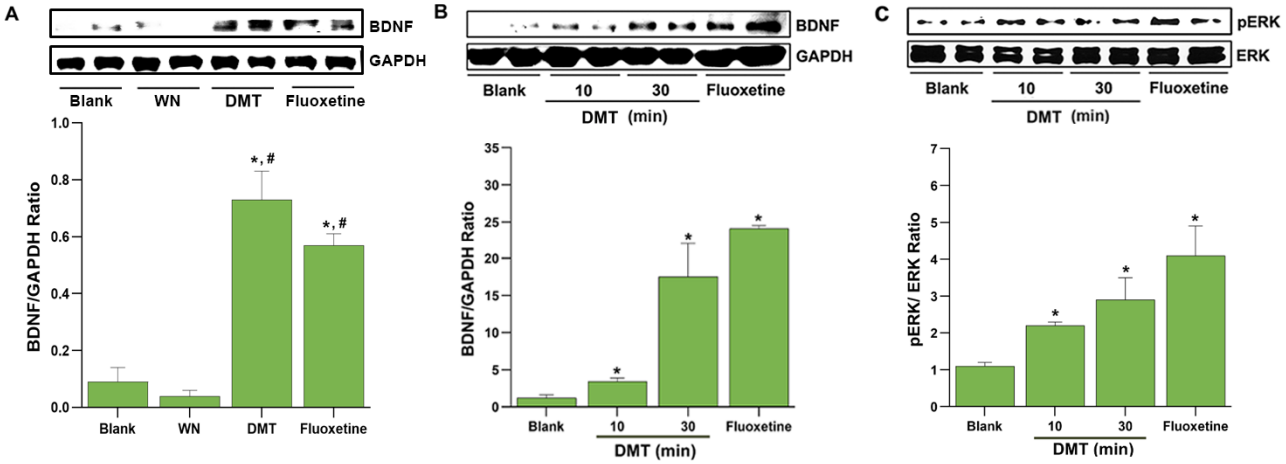

Fig. 3. Stimulation of hippocampal neuroplastic signaling cascades by DMT. (A and B) Expression of BDNF and (C) phosphorylation of ERK in the hippocampus were analyzed by Western blotting. The relative protein levels of (A and B, lower panel) BDNF and (C, lower panel) pERK were quantified by densitometry. Data are expressed as means ± SEMs (n = 3 per group). *$p < 0.05$ versus blank group. #$p < 0.05$ versus WN. WN, white noise; DMT, Daegeum music therapy.

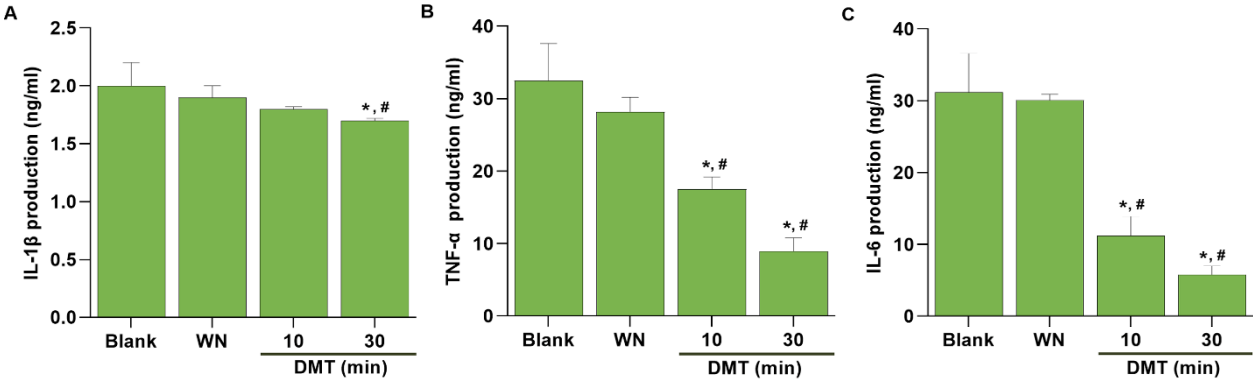

Fig. 4. Suppression of systemic inflammatory cascades by DMT. (A-C) Serum levels of IL-1β, TNF-α, and IL-6 in serum were measured using ELISA. Values are expressed as means ± SEMs (n = 5 per group). *$p < 0.05$ versus the blank group. #$p < 0.05$ versus WN. WN, white noise; DMT, Daegeum music therapy.

Suppression of systemic inflammatory cascades by DMT

To evaluate the systemic anti-inflammatory trajectory of DMT, we quantified the serum profiles of key pro-inflammatory cytokines, specifically IL-1β, TNF-α, and IL-6, following the chronic stress paradigm. Serum IL-1β dynamics revealed a conservative responsive threshold to the musical intervention (Fig. 4A). The baseline stress elevation in the blank (2.0 ± 0.20 ng/ml) and WN (1.9 ± 0.10 ng/ml) groups showed a subtle downward trend in the 10-min DMT group (1.8 ± 0.01 ng/ml), while a statistically prominent restriction was consolidated in the 30-min DMT treatment group (1.7 ± 0.01 ng/ml) (Fig. 4A, $p < 0.05$). As illustrated in Figure 4B, the blank group exhibited elevated serum TNF-α levels (32.5 ± 5.1 ng/ml), which remained largely unattenuated by WN exposure (28.2 ± 2.0 ng/ml). In contrast, intervention with DMT precipitated a robust, time-dependent down-regulation of TNF-α (Fig. 4B, $p < 0.05$). The serum concentration of TNF-α was diminished to 18.5 ± 3.1 ng/ml in the 10-min DMT group and further suppressed to 8.9 ± 1.9 ng/ml in the 30-min DMT group (Fig. 4B). A closely mirrored inhibitory pattern was observed in the serum IL-6 levels (Fig. 4C). High levels of circulating IL-6 induced by behavioral stress in the blank group (31.2 ± 5.4 ng/ml) and WN (30.1 ± 0.8 ng/ml) groups were markedly mitigated by the acoustic therapy. Specifically, DMT exposure for 10 and 30 min successfully drove down IL-6 expression to 11.2 ± 2.7 ng/ml and 5.8 ± 1.2 ng/ml, respectively (Fig. 4C, $p < 0.05$).

DISCUSSION

Music therapy has gained widespread recognition as an effective complementary intervention across various clinical domains, including psychiatry and neurology, owing to its non-invasive capacity to manipulate emotional states and alleviate depressive symptoms.[3,14] In preclinical research, the FST serves as a gold-standard behavioral paradigm for evaluating antidepressant-like efficacy, where a significant reduction in immobility directly reflects a mitigation of behavioral despair.[12,15] In the present study, we demonstrate for the first time

that DMT significantly decreases FST immobility durations in mice without disrupting baseline locomotor activity in the OFT. Crucially, this antidepressant-like effect was acoustically specific to DMT, as alternative sound exposures such as WN and Buk music failed to rescue the behavioral deficit, confirming the precise therapeutic utility of DMT's acoustic profile.

At the neurochemical level, classical and contemporary antidepressant strategies heavily target the restoration of monoaminergic homeostasis, specifically focusing on 5-HT and DA transmission within key limbic structures like the hippocampus.[16,17] Chronic stress is well-documented to impair the release and turnover of these essential neurotransmitters, leading to severe disruptions in emotional processing and structural neuroplasticity.[18] Our biochemical analyses revealed that chronic DMT exposure remarkably prevented this stress-induced deficit, successfully restoring hippocampal 5-HT and DA levels. Central monoaminergic signaling acts as a critical upstream driver of BDNF expression and its downstream intracellular cascades, notably the ERK pathway, which altogether govern synaptic remodeling and neuronal survival in the hippocampus.[8] Consistent with this molecular framework, DMT robustly up-regulated hippocampal BDNF expression and selectively promoted ERK phosphorylation, underscoring that the behavioral benefits of DMT are intimately linked to the activation of hippocampal neuroplastic cascades.

Beyond central remodeling, a growing body of evidence highlights the bidirectional link between peripheral inflammation and central neuroplasticity in the pathogenesis of depression.[10] Chronic stress provokes robust neuroinflammatory responses characterized by elevated systemic pro-inflammatory cytokines, such as IL-1β, IL-6, and TNF-α, which serve as critical physiological indicators of targeted neural dysfunction and psychological distress.[19] In our study, DMT precipitated a potent, time-dependent suppression of these circulating inflammatory cascades, with a 30-min exposure achieving the most prominent restriction of serum IL-1β, IL-6, and TNF-α levels. This dual action demonstrates that DMT operates by directly rescuing neuroplastic signaling within the brain while simultaneously reducing the systemic inflammatory load throughout the body.

A limitation of this study is that only male ICR mice were evaluated, leaving potential sex-specific differences in response to DMT unaddressed. Therefore, further studies involving female cohorts are necessary to confirm the broader applicability of these findings.

In conclusion, this study demonstrates that DMT exerts robust antidepressant-like effects by restoring hippocampal monoaminergic homeostasis, stimulating neuroplastic BDNF-ERK signaling cascades, and suppressing systemic inflammatory pathways. These findings provide compelling empirical evidence for the therapeutic potential of DMT as a viable, non-pharmacological acoustic modality for the management of depressive disorders.

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CONFLICT OF INTEREST

The authors have no conflicting financial interests

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