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MEDITATION, INFLAMMATION, AND TNF- α: A COMPREHENSIVE REVIEW OF CYTOKINE-MEDIATED EFFECTS

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Abstract:

Inflammation is a coordinated biological response involving immune, endocrine, and neural signalling, though persistent low-grade inflammation drives chronic diseases and neuroinflammation. Tumour necrosis factor-alpha (TNF-α), alongside interleukin-6 (IL-6) and interleukin-1β (IL-1β), acts as a primary pro-inflammatory cytokine. Mind–body modalities—including mindfulness meditation, Mindfulness-Based Stress Reduction (MBSR), Tai Chi, Qigong, and Yoga—are widely investigated as complementary therapies targeting stress-related inflammatory biomarkers. Current evidence suggests TNF-α levels may decline following mind–body interventions, though outcomes vary across studies. A 2026 dose-response meta-analysis demonstrated a directionally favourable yet statistically non-significant pooled reduction in TNF-α (SMD −0.33, 95% CI −0.71 to 0.05, P=0.09) alongside high heterogeneity (I^2=86.8%). Nevertheless, direct clinical trials show promise: a four-week randomized trial in 64 young women with depressive symptoms revealed significantly greater decreases in salivary TNF-α and IL-6 compared to controls, especially among those with higher baseline depression. Additional investigations have targeted mild cognitive impairment, Parkinson's disease, rheumatoid arthritis, and schizophrenia. Biologically, these practices may modulate autonomic, neuroendocrine, and NF-κB signalling to suppress cytokine production; however, a definitive human causal pathway remains unconfirmed. Ultimately, while meditation is an encouraging complementary regulator, definitive TNF-α conclusions require overcoming high heterogeneity, small samples, and variable measurement protocols.

Keywords: *Meditation, Mindfulness, Mindfulness-Based Stress Reduction, TNF-A, Tumour Necrosis Factor-Alpha, Inflammatory Cytokines, IL-6, IL-1β, IL-10, NF-K B, Neuro Inflammation, Chronic Inflammation, Mind–Body Medicine.*

1. Introduction

Chronic inflammation is characterized by prolonged immune activation and sustained production of inflammatory mediators. The supplied Tai Chi systematic review describes persistent secretion of TNF-α and IL-6 as an

important feature of chronic inflammatory states and identifies NF-κ B as a central inflammatory signalling pathway. [4]

TNF-α, IL-6 and IL-1β are commonly evaluated as pro-inflammatory biomarkers, whereas IL-10 represents an important anti-inflammatory cytokine. [1,2]

Neuro inflammation involves interactions between peripheral inflammatory signals and cells within the central nervous system, including microglia and astrocytes, and dysregulated inflammatory signalling has been discussed in relation to neuropsychiatric and neurodegenerative disorders. [2,14]

Meditation and related mind–body interventions are of interest because they may affect both psychological stress and physiological processes. The literature includes mindfulness meditation, MBSR, Mindful Awareness Practices, Mindfulness-Based Cognitive Therapy, Tai Chi, Qigong and Yoga. [1,3,5]

Importantly, these interventions are not biologically identical. Meditation may emphasize attention and awareness, whereas Tai Chi and Yoga may combine meditation with movement, breathing and physical activity. Therefore, evidence from the broader mind–body literature should be used as supporting evidence rather than as direct proof of a meditation-specific TNF-α effect. [1,5]

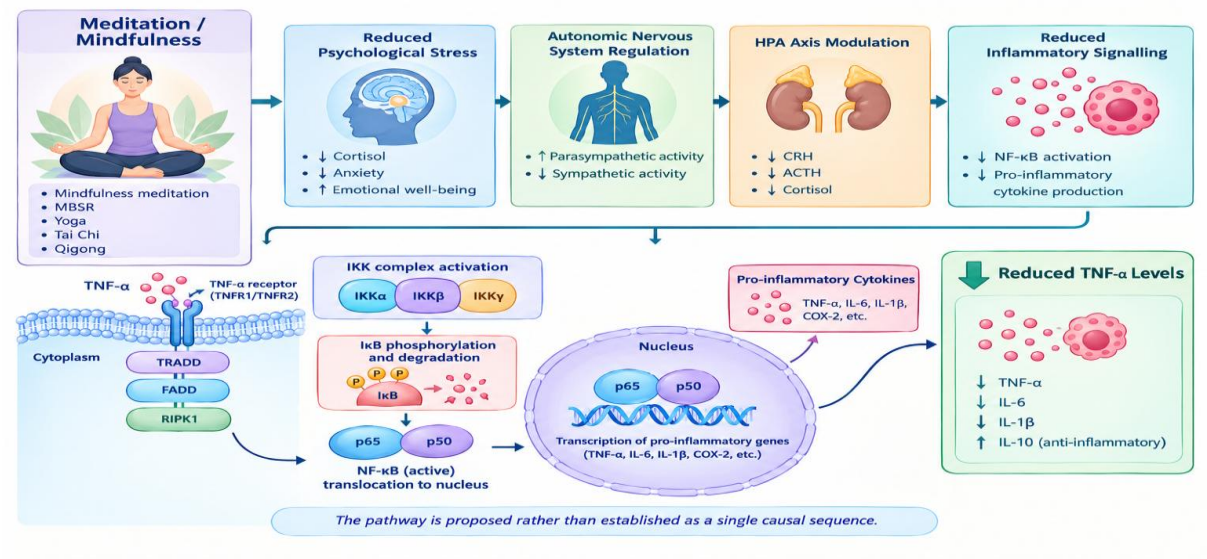

Figure 1: Simplified TNF-α/NF-κ B inflammatory signalling pathway. Redrawn and synthesized from the supplied literature describing TNF-α signalling, neuro inflammation and NF-κ B-related inflammatory pathways. [2,4,14]

2. TNF-α: Biological Role in Inflammation

2.1 TNF-α as a pro-inflammatory mediator

TNF-α is a central pro-inflammatory cytokine involved in immune-cell communication and inflammatory gene regulation. It is repeatedly examined together with IL-6 and IL-1β in mind–body intervention studies. [1,4]

Persistent activation of inflammatory pathways can maintain cytokine production and contribute to chronic inflammatory conditions. The 2026 Tai Chi review describes TNF-α and IL-1β as components of a positive feedback relationship with NF-κ B signalling. [4]

Because TNF-α belongs to a broader inflammatory network, a reduction in TNF-α should not be interpreted as complete suppression of inflammation. IL-6, IL-1β, IL-10, CRP and other biomarkers may change differently after the same intervention. [1,3]

2.2 TNF-α and NF-κ B signalling

In the canonical NF-κ B pathway described in the supplied literature, TNF-α and IL-1β can activate their receptor pathways, leading to IKK activation, I κ B degradation and nuclear translocation of NF-κ B transcriptional complexes. [4]

NF-κ B activation can promote transcription of inflammatory genes including TNF-α, IL-1β and IL-6, creating a self-amplifying inflammatory network. [4]

This feedback model gives a mechanistic rationale for studying interventions that may reduce upstream stress or inflammatory signalling, because dampening pathway activation could theoretically reduce transcription of several inflammatory mediators simultaneously. [4,13]

2.3 TNF-α in relation to IL-6, IL-1β and IL-10

IL-6 and IL-1β are frequently measured alongside TNF-α because they participate in overlapping inflammatory networks. The 2026 meta-analysis found significant pooled reductions in IL-6 and IL-1β even though the pooled TNF-α reduction did not reach statistical significance. [1]

IL-10 provides an important counter-regulatory marker because it is classified as anti-inflammatory in the supplied literature. The same meta-analysis found a significant increase in IL-10 after mind–body exercise. [1]

These results indicate at the biological effect of mind–body practice may involve broader immune regulation rather than a single cytokine-specific action. [1,3]

3. Meditation and Mind–Body Interventions

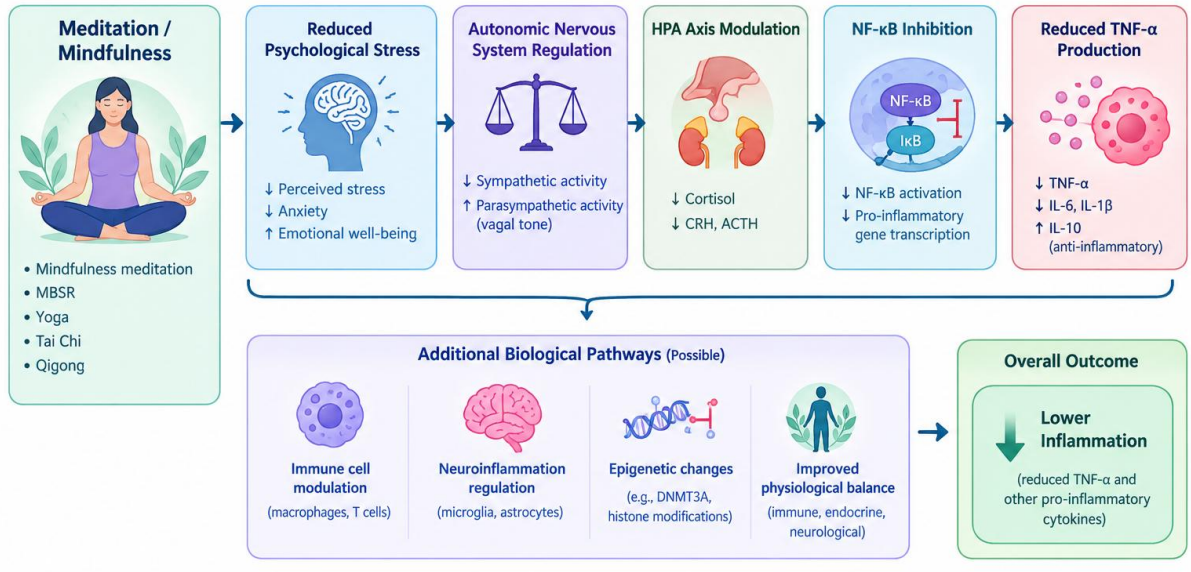

Figure 2: Proposed mechanism linking meditation/mindfulness with TNF-α regulation. Redrawn and synthesized from the supplied mindfulness, mind–body and stress-response literature. The pathway is proposed rather than established as a single causal sequence. [3,5,7,13,15,17]

MBSR was one of the intervention categories included in the 2026 systematic review, alongside Tai Chi, Qigong and Yoga. [1]

The intervention protocols included differed substantially in session duration, frequency and total intervention duration. Sessions ranged from approximately 15 to 180 minutes, and intervention frequency varied from one to seven sessions per week. [1]

Such differences are important because inflammatory responses may depend on intervention dose, duration, adherence, baseline inflammatory status and characteristics of the study population. [1,5]

Mindfulness-specific evidence is therefore most appropriate when answering the question of whether meditation affects TNF-α, while Tai Chi, Qigong and Yoga can be used to contextualize the broader mind–body inflammatory response. [1,5]

4. Direct Evidence for Meditation and TNF-α

4.1 Brief mindfulness intervention in depressive symptomatology

Walsh et al. conducted a four-week randomized study involving 64 young women with elevated depressive symptomatology. Participants were assigned to a mindfulness-based intervention or contact-control condition, and salivary IL-6 and TNF-α were measured before and after treatment. [7]

The mindfulness intervention predicted greater reductions in both IL-6 and TNF-α than the control condition. IL-6 reductions remained evident at the three-month follow-up. [7]

An important observation was that both groups showed similar reductions in self-reported depressive symptoms, meaning that the cytokine findings could not simply be explained by greater improvement in depression in the mindfulness group. [7]

The TNF-α response was moderated by baseline depressive symptom severity. Participants with higher baseline depressive symptoms demonstrated a stronger TNF-α reduction after mindfulness training, whereas the effect was not significant amin participants with lower baseline symptoms. [7]

This study provides one of the clearest direct pieces of evidence in the supplied literature for a relationship between a brief mindfulness intervention and reduced TNF-α. However, the population was limited to young women with depressive symptomatology, and the intervention was short. [7]

4.2 Mindfulness and inflammatory biomarkers in mild cognitive impairment

Ng et al. conducted a randomized controlled trial examining mindfulness and inflammatory biomarkers in older adults with mild cognitive impairment. The study is included in the supplied literature as evidence that mindfulness can influence inflammatory biomarker profiles in an older clinical population. [8]

Other MBSR research in mild cognitive impairment has also measured CRP, IL-6 and TNF-α, indicating that inflammatory regulation is a recurring biological outcome in this population. [9]

Because mild cognitive impairment is associated with age-related biological changes, findings from this population may be relevant to the hypothesis that mind–body practices influence inflammatory pathways involved in cognitive and neurological health. [8,9]

4.3 MBSR, stress physiology and inflammatory response

A randomized study of MBSR in generalized anxiety disorder measured cortisol, ACTH, IL-6 and TNF-α during a laboratory stress challenge. The MBSR group showed significant changes in ACTH, TNF-α and IL-6 area-under-the-curve concentrations compared with the control group. [13]

This finding is important because it links mindfulness training with inflammatory responses observed under experimentally induced stress rather than only resting biomarker levels. [13]

The result supports the possibility that stress-response regulation is one route through which mindfulness may influence inflammatory cytokines, although it does not prove that TNF-α changes are caused specifically by reduced sympathetic activity or NF-κ B signalling. [4,13]

4.4 Mindfulness in neurodegenerative disease

The supplied literature includes mindfulness and MBSR studies in Parkinson's disease, Alzheimer's disease and mild cognitive impairment, with inflammatory, endocrine and stress-related outcomes evaluated throughout various studies. [1,9,10]

These studies are relevant to neuro inflammation because inflammatory signalling has been implicated in neurological disorders, but the evidence does not establish that meditation directly treats neuro inflammation or reduces TNF-α within the brain. [2,14]

The distinction between peripheral blood or saliva biomarkers and central nervous system inflammation is therefore important when interpreting meditation studies. [2]

5.1 Tai Chi and TNF-α

Tai Chi is a mind–body exercise that unites physical movement with meditative and attentional components, making it relevant to the broader question of how mind–body practices may affect inflammation. [4]

A 2026 systematic review and meta-analysis of 20 studies found that Tai Chi significantly reduced NF-κB-driven downstream pro-inflammatory gene expression overall (SMD −0.48, 95% CI −0.76 to −0.19, P<0.01). IL-6 and IL-1β were significantly reduced, while TNF-α showed a downward but statistically non-significant trend (SMD −0.28, 95% CI −0.59 to 0.02, P=0.07). [4]

The strongest subgroup benefits in that analysis were observed in endocrine and respiratory diseases, with additional effects reported in neurological disease populations. [4]

The findings are important because they show a pattern like the broader 2026 mind–body meta-analysis: TNF-α tends to move downward, while the strongest statistical evidence may be observed for other inflammatory mediators. [1,4]

5.2 Tai Chi, oxidative stress and inflammatory signalling

The supplied Tai Chi review proposes an additional oxidative-stress mechanism. Reactive oxygen species can stimulate inflammatory transcriptional pathways, while Tai Chi has been associated with antioxidant responses involving NRF2-related signalling and changes in oxidative-stress markers. [4]

The review reports that increase in NRF2-associated activity and superoxide dismutase together with reductions in malondialdehyde were accompanied by lower IL-6, TNF-α and IL-1β levels in the included literature. [4]

This suggests that oxidative stress may interact with inflammatory signalling and represents a potential mechanistic bridge between mind–body exercise and cytokine regulation. However, these data are primarily derived from Tai Chi research and should not automatically be assigned to seated meditation. [4]

5.3 Qigong and TNF-α

Qigong has also been studied in relation to inflammatory status and TNF-α, including research in people with Parkinson's disease. [10,11]

The presence of TNF-α findings in Qigong studies strengthens the broader hypothesis that mind–body practices may influence inflammatory signalling, while also demonstrating the heterogeneity of intervention forms grouped under the mind–body category. [1,10,11]

5.4 Yoga and TNF-α

A systematic review of Yoga and inflammatory markers included 26 studies, of which 13 assessed TNF-α. Most included studies reported favourable inflammatory outcomes, but only two studies were judged to have low risk of bias, and the overall evidence quality was considered poor with considerable heterogeneity. [12]

The review found that pooled effects for IL-6, TNF-α and CRP favoured Yoga but did not reach statistical significance. [12]

Yoga interventions may include asanas, pranayama and meditation/dhyana, which makes them relevant to the broader mind–body literature but prevents attribution of the inflammatory effect to meditation alone. [12]

6. Quantitative Synthesis of TNF-α Evidence

In the 2026 systematic review and dose-response meta-analysis, 29 randomized controlled trials involving 2,253 participants were included across mind–body exercise categories. [1]

The pooled TNF-α effect was SMD −0.33 (95% CI −0.71 to 0.05; P=0.09), showing a numerical reduction but not a statistically significant pooled effect. The high heterogeneity (I^2=86.8%) suggests that the included studies differed substantially in ways that may influence the observed TNF-α response. [1]

Potential sources of heterogeneity include intervention type, disease population, intervention duration, session length, frequency, baseline inflammatory status, biomarker sampling and study quality. These factors should be considered when interpreting the pooled estimate. [1,5]

The dose-response analysis reported a greater TNF-α reduction in interventions with sessions longer than 60 minutes and identified approximately 600 MET-min/week as an estimated point of greatest TNF-α reduction. [1]

Because MET-min/week is closely related to physical activity expenditure, this finding should be interpreted cautiously when applying it to meditation that contains little or no physical movement. [1]

7. Comparative tables and figures

7.1 Comparative Cytokine Table

Table 1: Comparative summary of cytokine and biomarker responses to mind–body interventions

Biomarker	Direction	Pooled Effect Size (SMD)	Statistical Significance	Clinical Interpretation
TNF-α	Decrease	SMD −0.33 (95% CI −0.71 to 0.05)	P = 0.09	Favourable numerical trend, non-significant (1)
IL-6	Decrease	SMD −0.47	P = 0.02	Statistically pooled reduction (1)
IL-1β	Decrease	SMD −0.90	P < 0.0001	Strong significant reduction (1)
IL-10	Increase	SMD 0.87	P = 0.01	Significant increase anti-inflammatory increase (1)
BDNF	Increase	SMD 1.08	P = 0.0012	Significant increase in neurotrophic elevation (1)
CRP	Decrease	SMD −0.12	Non-significant	Small pooled trend (1)

7.2 Comparative Figures and Evidence Visualization

Figures 1–4 provide complementary visual summaries of the TNF-α/NF-κ B pathway, the proposed meditation-related inflammatory pathway, the TNF-α evidence pattern, and the overall evidence framework. The figures are redrawn/synthesized from the literature set used for this review and are intended to improve conceptual clarity rather than imply direct causality. [1–5,7–17]

8. Potential Mechanisms Relating Meditation to TNF-α

8.1 Psychological stress and autonomic regulation

Psychological stress can interact with autonomic and immune systems, and mind–body practices are proposed to modify this interaction through self-regulation and stress-response pathways. [3,5]

The supplied Tai Chi review describes sympathetic nervous system activation through β-adrenergic receptors as a pathway capable of promoting NF-κ B activation and increasing IL-6, TNF-α and IL-1β. It also proposes that regular mind–body practice may reduce sympathetic activity. [4]

This provides a plausible framework in which meditation could influence inflammation indirectly by changing the physiological response to stress rather than directly blocking TNF-α production. [3,4]

8.2 NF-κ B pathway

NF-κ B is a central transcriptional pathway connecting inflammatory receptors with expression of several pro-inflammatory genes. [4]

The supplied Tai Chi evidence indicates that TNF-α and IL-1β can participate in positive feedback with NF-κ B, while down-regulation of NF-κ B-related activity is associated with reductions in inflammatory gene expression. [4]

Although NF-κ B is a plausible mechanistic target, direct human evidence demonstrating the complete sequence meditation → reduced NF-κ B activity → reduced TNF-α is insufficient in the supplied literature. [1,4]

8.3 Oxidative stress and antioxidant pathways

Oxidative stress can activate inflammatory transcriptional pathways, and the supplied Tai Chi review describes antioxidant-related changes involving NRF2, superoxide dismutase and malondialdehyde together with reductions in inflammatory cytokines. This mechanism is potentially relevant to mind–body interventions that include physical activity, but evidence specifically demonstrating that seated meditation changes oxidative-stress pathways sufficiently to reduce TNF-α remains limited in the supplied material. [4]

8.4 Neuroendocrine and HPA-axis pathways

Mind–body interventions may influence inflammatory biology through interactions among the hypothalamic–pituitary–adrenal axis, cortisol, autonomic signalling and immune cytokines. [3,5,13]

The MBSR stress-challenge study measuring ACTH, cortisol, IL-6 and TNF-α provides experimental support for a relationship between mindfulness training and stress-related physiologic responses. [13]

However, endocrine changes should be interpreted as part of a multi-system mechanism rather than as evidence of a single direct pathway to TNF-α reduction. [3,13]

9. Disease-Specific Interpretation

9.1 Depression and depressive symptomatology

Inflammatory cytokines have been investigated as biological correlates of depression, making depressive populations an important setting for studying meditation and inflammatory biomarkers. [7,15]

The Walsh trial provides direct evidence that mindfulness reduced salivary TNF-α and IL-6 more than contact control, mainly within participants with higher baseline depressive symptoms. [7]

A broader 2025 systematic review of mind–body therapies in depression identified 12 RCTs and 1,058 participants, with 14 of 21 pieces of evidence supporting favourable effects on pro-inflammatory cytokines, although study quality ranged from low to high risk of bias. [15]

9.2 Mild cognitive impairment and neurodegeneration

Mindfulness and MBSR have been evaluated in older adults with mild cognitive impairment, including studies measuring TNF-α, IL-6 and other inflammatory biomarkers. [8,9]

These studies provide a basis for investigating whether inflammatory regulation may accompany cognitive and psychological benefits, but the supplied evidence does not establish that peripheral TNF-α changes translate directly into reduced central neuro inflammation. [2,8,9]

9.3 Parkinson's disease

Parkinson's disease appears repeatedly in the mind–body literature, with MBSR, Tai Chi and Qigong interventions evaluating inflammatory, stress-related or neurological outcomes. [1,10,11]

The 2026 Tai Chi review reported stronger reductions in NF-κ B-driven pro-inflammatory genes in neurological disease subgroups than in some other disease categories. [4]

These outcomes suggest that disease context may moderate inflammatory responses to mind–body interventions, but larger disease-specific trials are needed. [1,4]

9.4 Rheumatoid arthritis and other inflammatory conditions

Yoga and mindfulness interventions have been evaluated in inflammatory diseases such as rheumatoid arthritis, with studies measuring TNF-α, IL-6 and other biomarkers. The supplied RA studies by Ganesan et al. and Gautam et al. provide disease-specific evidence relevant to inflammatory and psycho-neuro-immune outcomes. [6,12,16]

Inflammatory disease populations may be particularly useful for testing the hypothesis that baseline inflammatory burden modifies the response to meditation, because intervention effects may be easier to detect when inflammatory biomarkers are elevated at baseline. [12]

10. Factors that may influence TNF-α response

Baseline inflammatory status may influence whether a measurable reduction in TNF-α occurs after a mind–body intervention. The supplied literature suggests that at-risk populations with higher inflammatory burden may show greater changes, although this hypothesis requires confirmation. [12]

Intervention duration may also matter. In one mindfulness study, TNF-α reductions were observed after four weeks, whereas another mindfulness study found clinically relevant inflammatory changes after a longer intervention period. [7,17]

Session duration may influence outcomes, with the 2026 dose-response analysis reporting greater TNF-α reduction for sessions longer than 60 minutes. [1]

Adherence is another important factor because the biological exposure to meditation depends on whether participants practice consistently and according to the prescribed protocol. [1,15]

Population characteristics, including age, disease status, psychological symptoms and baseline inflammatory state, may make further contributions to differences between study differences. [1,7,12]

11. Critical discussion of recent literature

11.1 Evidence supporting a TNF-α-modulatory effect

Across the supplied literature, the direction of effect generally favors lower inflammatory activity after meditation or related mind–body interventions, but the magnitude and statistical certainty of the TNF-α response vary. Direct mindfulness evidence includes reductions in salivary TNF-α in young women with depressive symptomatology and reductions in inflammatory biomarkers in other clinical populations. [7,13,15]

The broader mind–body literature strengthens biological plausibility. Tai Chi studies report reductions in inflammatory markers and changes in NF-κB-related signaling, while Yoga, Qigong and other mind–body approaches provide additional evidence of immune modulation. However, these interventions include movement, breathing and other components, so their findings cannot be treated as direct evidence for seated meditation. [1,4,5,6,10,11,12]

11.2 Conflicting and non-significant findings

A key critical finding is that TNF-α is not consistently statistically significant across pooled analyses. The 2026 meta-analysis reported a favorable numerical TNF-α effect but substantial heterogeneity, whereas IL-6 and IL-1β showed clearer pooled reductions. [1,4]

The contrast between direct mindfulness studies and pooled mind–body analyses is particularly important. Walsh et al. reported a reduction in salivary TNF-α after brief mindfulness training, whereas the broader 2026 pooled analysis found a smaller, statistically non-significant TNF-α effect. [1,7] This difference may reflect intervention type, participant characteristics, biomarker sampling, and the inclusion of movement-based practices in the pooled literature.

This pattern suggests that meditation-related inflammatory effects may be multidimensional rather than TNF-α-specific. A lack of statistical significance for TNF-α should not be interpreted as evidence of no biological effect, but neither should a numerical reduction be presented as definitive proof of efficacy. [1,5,12]

11.3 Comparison with broader mind–body interventions

The evidence is strongest when interventions are considered as a family of mind–body practices, but this broader category brings an important interpretive problem. Tai Chi and Yoga combine meditative attention with physical activity, breathing or postural practice, whereas mindfulness meditation can be delivered without substantial physical exertion. Consequently, inflammatory effects attributed to the broader category may partly reflect exercise-related or respiratory mechanisms. [1,4,10,12]

11.4 Clinical and biological relevance of TNF-α changes

TNF-α is an important component of inflammatory signaling, but it should not be considered a stand-alone measure of systemic or neuroinflammation. Changes in TNF-α need to be interpreted alongside IL-6, IL-1β, IL-10, CRP and, where appropriate, neurobiological or stress-related measures. [1,2,14,15]

The reviewed evidence does not support the interpretation that meditation acts as a pharmacological TNF-α inhibitor. Instead, meditation may influence upstream stress-related and immune-regulatory processes, with TNF-α serving as one possible downstream biomarker. [3,5,13,17]

11.5 Strengths and limitations of the evidence base

Strengths of the literature include randomized controlled trials, systematic reviews and meta-analyses, together with studies spanning psychiatric, neurological, renal and inflammatory conditions. [1,5,7,8,13,15]

The largest limitation is heterogeneity. The 2026 pooled TNF-α analysis had I^2=86.8%, indicating considerable variation between studies. [1]

Intervention heterogeneity is also important because meditation, MBSR, Tai Chi, Qigong and Yoga contain different combinations of meditation, movement, breathing and physical activity. [1,5]

Several individual studies are small or pilot trials, which limit statistical power and precision of cytokine estimates. [1,8,9,10]

Different biological samples are used across studies, including saliva, serum and plasma. This may complicate immediate comparison of cytokine concentrations across trials. [5,7]

Risk of bias is another concern. The Yoga systematic review found that only two of 26 studies had low risk of bias, while most had high risk, pointing out the need for better-designed trials. [12]

TNF-α is also not a standalone measure of inflammation. A study can show improvement in IL-6 or IL-1β without notable TNF-α change, as demonstrated in pooled analyses. [1,4]

Finally, most available evidence evaluates peripheral biomarkers instead of direct inflammatory activity within the brain. Therefore, claims about neuro inflammation should be made cautiously. [2]

12. Challenges and future perspectives

12.1 Heterogeneity of interventions and study populations

A major challenge is heterogeneity in intervention type, duration, session frequency, participant characteristics and baseline inflammatory status. This makes direct comparison of TNF-α responses difficult and contributes to between-study variability in pooled analyses. [1,5,12]

12.2 Standardization of TNF-α Measurement

Future studies should standardize biological sampling, assay methods, reporting units and timing of cytokine assessment. Consistent methodology would improve comparability across studies and help distinguish short-term biomarker fluctuations from sustained inflammatory changes. [1,5,12]

12.3 Distinguishing meditation from exercise and breathing effects

Studies should clearly distinguish meditation-only interventions from multimodal practices such as Tai Chi and Yoga. Factorial or adequately controlled designs could help determine whether TNF-α changes arise from attentional practice, physical activity, breathing regulation, or their combination. [1,4,10,12]

12.4 Mechanistic and long-term research needs

Mechanistic studies should examine inflammatory biomarkers together with autonomic, endocrine and molecular measures to test the proposed stress–immune pathway. Long-term follow-up is also needed to determine whether biomarker changes persist after supervised intervention ends. [3,5,13]

12.5 Future research directions

Future studies should isolate meditation or MBSR from movement-based practices when the research question specifically concerns meditation and TNF-α. [1,5]

TNF-α should be pre specified as a primary or key secondary biomarker, with standardized biological sampling, assay methods and reporting units. [1]

Future trials should simultaneously measure TNF-α, IL-6, IL-1β, IL-10 and CRP so that changes can be evaluated as a coordinated inflammatory profile rather than as isolated biomarkers. [1]

Mechanistic studies should combine cytokine measurement with autonomic, endocrine and NF-κB-related measures to test whether changes in stress physiology precede inflammatory changes. [3,4,13]

Longer follow-up is needed to determine whether TNF-α reductions persist after supervised meditation training ends. [1,7]

Future studies should report adherence and actual practice exposure, because prescribed intervention dose may differ from the amount participants complete. [1,15]

Large, adequately powered randomized controlled trials should stratify participants by baseline inflammatory status and disease category to determine whether populations benefit more strongly. [1,7,12]

13. Overall synthesis and review implications

The evidence can be summarized as a consistent biological direction but an inconsistent statistical effect for TNF-α. Several meditation and body studies report lower TNF-α after intervention, while pooled evidence does not yet show a statistically significant universal effect. [1,4,7]

In the 2026 pooled analysis, the clearest pooled changes were observed for IL-6 and IL-1β, while the TNF-α estimate did not reach conventional statistical significance. [1] This pattern argues against treating TNF-α as the sole indicator of response and supports evaluating a broader inflammatory profile.

Direct mindfulness studies are particularly important because broader mind–body interventions contain physical and breathing components that cannot be separated from meditation effects in pooled analyses. [1,5]

The current evidence therefore supports meditation as an encouraging complementary strategy for inflammatory regulation, but it does not justify describing meditation as a proven TNF-α-lowering treatment. [1,5]

The most defensible conclusion is that meditation may contribute to favorable inflammatory regulation in selected populations, with TNF-α representing a plausible but variable biomarker of response. [1,7,13]

14. Conclusion

TNF-α is an important pro-inflammatory cytokine within a broader network involving IL-6, IL-1β, IL-10 and NF-κB signaling. [1,4]

Direct human evidence indicates that mindfulness interventions can reduce TNF-α in some populations, including young women with depressive symptomatology, while other studies provide evidence for inflammatory changes in mild cognitive impairment, stress-related conditions and chronic disease. [7–10,13]

The broader Tai Chi, Qigong and Yoga literature supports the plausibility of mind–body modulation of inflammatory pathways, but these interventions must not be equated with meditation alone. [1,4,10–12]

The pooled TNF-α evidence remains statistically non-significant and highly heterogeneous, making it premature to conclude that meditation consistently reduces TNF-α across populations. [1]

Future high-quality randomized trials applying standardized TNF-α measurement, adequate sample sizes, clearly defined meditation protocols and mechanistic biomarkers are needed to determine whether meditation produces reproducible TNF-α reductions and to identify the populations most likely to benefit. [1,4,5]

Figure source and citation note: All figures are original redrawn/synthesized diagrams based only on the supplied 20-paper review set. No external figure source was used, and no source figure was reproduced verbatim. Each figure has an in-figure citation linked to the supplied reference numbering.

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Conflict of interest

The authors declare no conflict of interest.

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