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MODULATION OF INFLAMMATORY PATHWAYS AND GENE EXPRESSION BY MEDITATION

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Abstract:

Growing scientific evidence suggests that mind-body practices, particularly meditation, influence physiological responses to stress by altering immune function, energy metabolism, and gene expression. Central to these effects is the downregulation of pro-inflammatory gene pathways, notably nuclear factor kappa B (NF-κB), which plays a critical role in mediating chronic stress and inflammation. Multiple reviews and clinical trials indicate that both short-term meditation retreats and regular practices downregulate pro-inflammatory pathways while modifying epigenetic factors such as DNA function and associated proteins. Interventions involving app-based mindfulness, targeted training courses, and clinical populations—such as breast cancer patients—demonstrate significant changes in gene activity alongside improved well-being. However, these genetic shifts do not always correlate with immediate alterations in circulating blood inflammatory markers, underscoring complex underlying mechanisms. Genome-wide studies further reveal enhanced immune profiles without a corresponding rise in inflammatory activity, suggesting protective and potentially restorative physiological adaptations. While individual study outcomes vary across nervous, endocrine, and immune parameters, the consensus highlights meditation's capacity to modulate genetic and cellular pathways related to stress resilience. Further comprehensive, multi-system investigations are necessary to clarify the exact biological mechanisms and their long-term clinical implications for human health.

Keywords: *Meditation, Mindfulness, Mindfulness-Based Stress Reduction, Inflammation, NF-kB, Gene Expression, Epigenetics, Histone Deacetylases, Transcriptomics, Immune Regulation, Inflammatory Pathways, Stress.*

1. Introduction

Inflammation is how our immune system protects us when we get sick hurt or stressed by our surroundings. While short bursts of inflammation help us heal much inflammation for too long can damage our cells and lead to long-term sickness. I have learned that psychological stress plays a role here because being stressed for a long-time

mess with how our nerves, hormones and immune systems talk to each other. On a level constant stress can turn on genes that cause inflammation especially through a pathway called **nuclear factor kappa B (NF-κB). This ** factor kappa B (NF-κB) is a main driver that controls many genes that cause inflammation [1,2].

Meditation is a way to calm the mind and body using things like breathing, focus and being mindful. Besides helping our mood many studies show that meditation can actually change how our bodies handle stress, our immune system and inflammation. Thanks to tools like functional genomics and transcriptomic approaches scientists can now look closely at how our genes change after we meditate. Researchers have seen changes in genes that control inflammation, our system how we use energy and how our cells deal with stress [2,3].

One of the common thing's scientists see is how meditation affects the NF-κB inflammatory pathway. The NF-κB inflammatory pathway acts like a control switch for genes that make signals and it often gets turned on by stress. A big review by Buric et al. Looked at 18 studies on meditation. Found that meditation usually helps lower the expression of genes related to NF-κB. In the way relaxation exercises changed how genes handled inflammation and metabolism [3]. This makes me think that meditation might help fight the damage caused by long-term stress.

Meditation might also work on mechanisms. These are processes like DNA methylation that change how genes behave without changing our DNA. For example, Kaliman et al. Found that deep meditation caused changes in enzymes called HDAC2, HDAC3 and HDAC9 [5]. They also saw levels of inflammatory genes like RIPK2 and COX2. This suggests that meditation might change how our DNA is packed and read. Other studies from meditation retreats also showed changes in genes that control inflammation and how our DNA is managed [6]. Because of this some experts believe that epigenetic changes are the bridge between meditation and lower inflammation [7].

We also have controlled trials that show how mindfulness helps. For instance, using a meditation app helped stressed adults lower the activity of the NF-κB pathway [8]. Even young breast cancer survivors saw inflammatory gene activity after practicing mindfulness [9]. This tells us that even a little bit of mindfulness can reach down to our level.

Newer research shows that this is a complex relationship. Lindsay et al. Ran a study with 190 adults and found that an eight-week Mindfulness-Based Stress Reduction (MBSR) program lowered NF-κB-related pro-inflammatory gene regulation [10]. Interestingly they did not see a drop in interleukin-6 (IL-6) or C-reactive protein (CRP) levels in the blood. This is a deal because it shows that meditation might be changing things inside our cells even if our blood tests look the same [10].

Some studies suggest meditation does more than just turn off genes. It might actually help our biological system. Some research shows that meditation can change how our immune system, metabolism and cells respond to stress [11,12]. One large study found that after a meditation retreat many immune genes actually became more active but the "bad" inflammatory pathways did not [13]. This makes me feel that meditation might modulate function rather than simply suppress immune activity. Long-term meditators might even be able to reverse some of the effects of stress [14].

Many reviews agree that mindfulness helps the system even if the results are not always the same for everyone. Black and Slavich looked at trials and found evidence that meditation affects NF-κB and some markers though results for things like IL-6 and TNF-α were a bit mixed [15]. Bower and Irwin also noted that mind-body therapies work through a web of nerves, hormones and cells [1]. It seems meditation does not just use one path; it uses a whole network to help the body.

In short, the evidence suggests that meditation can change our biology through NF-κB inflammatory gene expression, histone modification, epigenetic regulation, immune-response pathways and stress-responsive transcriptional networks [1–15]. We see the consistent changes in how genes related to NF-κB act even if blood tests are less clear. Learning more about these changes could help us understand how meditation truly helps our health. We still need research using transcriptomics, epigenomics, proteomics and conventional inflammatory biomarkers to see exactly how this works and how long the benefits last.

2. Materials and methodology

2.1 Study design

This study was structured as a comprehensive narrative literature review designed to synthesize empirical evidence regarding the molecular mechanisms through which meditation and mindfulness interventions modulate inflammatory pathways, immune competence, gene expression, and epigenetic modifications. The review evaluated clinical trials, randomized controlled investigations, functional genomic analyses, and systematic evaluations of mind–body practices, including Mindfulness-Based Stress Reduction (MBSR), yogic meditation, transcendental meditation, and intensive retreat protocols. Particular analytical focus was directed toward studies evaluating nuclear factor kappa B (NF-κB) transcription, histone deacetylase (HDAC) regulation, genome-wide transcriptomic dynamics, and circulating inflammatory biomarkers (1–15).

2.2 Literature search strategy

A structured and comprehensive bibliographic search was executed across major peer-reviewed scientific databases, including PubMed/MEDLINE, PubMed Central (PMC), Google Scholar, ScienceDirect, and Frontiers. Boolean search strings were formulated to identify studies examining the intersections of meditation, mindfulness, transcriptomics, epigenetics, and inflammatory signaling. The primary query strings included: "meditation" AND "gene expression"; "mindfulness" AND "inflammation"; "meditation" AND "NF-κB"; "mindfulness" AND "pro-inflammatory genes"; "meditation" AND "epigenetics"; "meditation" AND "histone deacetylase"; "mindfulness" AND "transcriptomics"; "meditation" AND "immune system"; "mind-body intervention" AND "gene expression"; "MBSR" AND "inflammatory gene expression"; and "meditation" AND "inflammatory pathways". The bibliographic search prioritized empirical studies reporting defined biological, molecular, or cellular endpoints.

2.3 Selection of literature

Literature selection followed predefined relevance criteria to ensure rigorous evaluation of molecular and physiological endpoints. Priority was assigned to: (i) randomized controlled trials evaluating standardized meditation or mindfulness interventions; (ii) targeted gene-expression assays; (iii) whole-genome and transcriptomic microarray or RNA-sequencing evaluations; (iv) studies characterizing epigenetic modifications, such as histone modifications or DNA methylation; (v) investigations examining inflammatory transcriptional cascades; (vi) systematic and descriptive reviews synthesizing mind–body genomics; and (vii) trials reporting quantified circulating inflammatory biomarkers. Key benchmark studies included randomized trials and genomic investigations by Lindsay *et al.* (1), Kaliman *et al.* (2), Burić *et al.* (3), Bhasin *et al.* (4), Dutcher *et al.* (8), Bower *et al.* (9), Chandran *et al.* (12), Wenuganen *et al.* (13), Bower and Irwin (14), and Black and Slavich (15).

2.4 Inclusion criteria

Studies were incorporated into the review if they met the following methodological criteria: (i) published in a peer-reviewed academic journal; (ii) investigated defined meditation practices, mindfulness interventions, MBSR programs, relaxation-response practices, or structured mind–body interventions; (iii) evaluated gene expression, cell signaling, immune profiles, epigenetic regulation, or circulating inflammatory biomarkers; (iv) recruited human participant cohorts; (v) reported quantified molecular, transcriptomic, or physiological outcomes; (vi) provided comprehensive procedural descriptions of the intervention and analytical assays; and (vii) possessed full-text accessibility for data extraction.

2.5 Exclusion criteria

Articles were excluded if they: (i) did not evaluate a meditation or structured mind–body intervention; (ii) omitted genomic, transcriptomic, epigenetic, immune, or inflammatory biomarkers; (iii) lacked empirical relevance to the mechanistic research questions; (iv) represented duplicate publications or overlapping datasets without novel endpoints; (v) constituted non-peer-reviewed commentaries, popular news reports, or non-empirical internet articles; or (vi) failed to report detailed experimental protocols and empirical data.

2.6 Data extraction

A systematic extraction protocol was applied to all eligible investigations to capture methodological and biological variables. Standardized parameters included author details, publication year, study design, cohort characteristics, intervention specifications, practice duration, biological biospecimens, analytical platforms, targeted inflammatory pathways, and measured biomarkers, as delineated in Table 1.

Table 1: Methodological parameters and biological variables extracted from included studies

Parameter	Information extracted
Author and year	First author name and formal publication year.
Study design	Randomized controlled trial (RCT), observational cohort study, genome-wide transcriptomic profiling, or systematic review.
Participants	Sample size, cohort demographics, clinical status, and baseline health characteristics.
Intervention	Standardized meditation protocol, MBSR, digital mindfulness, relaxation response, or intensive meditation retreat.
Duration	Length, frequency, daily session duration, and follow-up monitoring timeline.
Biological sample	Peripheral blood mononuclear cells (PBMCs), whole blood, serum, or plasma specimens.
Molecular analysis	Targeted quantitative reverse transcription PCR (RT-qPCR), microarray profiling, RNA sequencing, or epigenetic enzyme assays.
Inflammatory pathway	NF-κB transcriptional cascades, TNF signaling, COX2, RIPK2, and type I/II interferon response pathways.
Biomarkers	Circulating cytokine concentrations including IL-6, CRP, TNF-α, and related inflammatory mediators.

2.7 Assessment of inflammatory pathways

Central analytical emphasis was directed toward the nuclear factor kappa B (NF-κB) transcription family due to its recurring role as the primary inflammatory pathway responsive to psychosocial stress and mind–body interventions. NF-κB transcription complexes govern the expression of an array of pro-inflammatory cytokines, chemokines, and inducible enzymes. Empirical data regarding NF-κB transcriptional activity, target gene expression, and upstream signaling intermediates were compared systematically across the literature (1, 3, 4, 8). Secondary inflammatory cascades, including tumor necrosis factor-alpha (TNF-α) signaling networks, cyclooxygenase-2 (*COX2*), receptor-interacting serine/threonine-protein kinase 2 (*RIPK2*), and interferon-mediated immune-response genes, were extracted and appraised when reported (2, 6, 12).

2.8 Assessment of gene expression

Gene-expression outcomes were assessed across studies utilizing targeted quantitative RT-PCR, genome-wide cDNA microarrays, and next-generation RNA sequencing platforms. Analytical evaluations focused on functional gene clusters related to pro-inflammatory signaling, immune regulation, cellular stress adaptation, energy metabolism, and cellular survival. Cross-study comparisons specifically evaluated whether meditation interventions produced: (i) downregulation of pro-inflammatory transcriptional cascades; (ii) upregulation of immune-supportive and antiviral defense pathways; (iii) attenuation of allostatic transcriptional activity; and (iv) coordinated shifts in metabolic and oxidative phosphorylation pathways. Seminal findings from Bhasin *et al.* demonstrated acute metabolic and inflammatory gene remodeling following relaxation practices, while Chandran *et al.* and Epel *et al.* characterized wide-ranging transcriptomic alterations following intensive meditation retreats (4, 11, 12).

2.9 Assessment of epigenetic mechanisms

Studies examining epigenetic parameters were analyzed independently to differentiate chromatin-level modifications from direct transcript abundance. The assessment concentrated on post-translational histone modifications, histone deacetylase (HDAC) dynamics, and locus-specific DNA methylation patterns. The landmark clinical trial by Kaliman *et al.* served as a foundational model, demonstrating that intensive meditation practice downregulates expression of *HDAC2*, *HDAC3*, and *HDAC9* while concurrently repressing inflammatory loci such as *RIPK2* and *COX2* (2). Complementary epigenetic and transcriptomic findings from extended meditation retreats were incorporated to evaluate the temporal dynamics of chromatin remodeling (5).

2.10 Assessment of systemic inflammatory biomarkers

Circulating systemic inflammatory biomarkers were evaluated and cross-referenced with gene-expression patterns across all reporting trials. The primary clinical biomarkers appraised included interleukin-6 (IL-6), C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), and other circulating cytokines. Delineating systemic protein concentrations is crucial because cellular gene transcription does not invariably align with circulating cytokine levels. For example, Lindsay *et al.* documented robust downregulation of pro-inflammatory gene transcription following MBSR, yet circulating IL-6 and CRP concentrations exhibited no statistically significant reduction (1).

2.11 Comparison and synthesis of findings

Empirical findings were qualitatively synthesized across interventions by categorizing studies according to meditation modality, practice dosage, participant baseline health, molecular assay sensitivity, inflammatory

pathways probed, and biomarker concordance. Narrative synthesis prioritized recurring molecular patterns, notably the consistent suppression of NF-κB transcriptional networks, selective epigenetic alterations, and divergent cellular versus circulating biomarker trajectories (2, 3, 15).

2.12 Ethical considerations

Because this investigation comprised a narrative literature review of published, peer-reviewed scientific studies without direct human recruitment, tissue biospecimen collection, or experimental interventions, formal institutional review board (IRB) approval was not required. The ethical integrity, participant consent procedures, and clinical trial registrations of all primary source studies were verified during the analytical extraction process.

2.13 Methodological limitations

Several methodological limitations inherent in the mind–body literature warrant consideration. Primary studies exhibited significant heterogeneity regarding sample sizes, participant demographics, meditation modalities, intervention durations, control group rigor (e.g., active versus passive waitlist controls), biospecimen types, and molecular assay techniques. Several genomic investigations utilized small sample sizes or observational retreat designs, which may introduce self-selection bias. Furthermore, discrepancies in epigenetic profiling methodologies preclude definitive meta-analytic pooling. Crucially, intracellular transcriptional downregulation does not necessarily translate into immediate changes in circulating plasma cytokine concentrations or long-term clinical endpoints, as evidenced by Lindsay *et al.* (1). Consequently, scholarly interpretations must maintain a rigorous distinction between cellular transcript regulation, systemic protein biomarkers, and systemic disease outcomes.

2.14 Overall methodological framework

The methodological framework executed throughout this review proceeded through ten structured analytical phases: (i) comprehensive bibliographic identification; (ii) primary literature screening; (iii) application of explicit inclusion and exclusion criteria; (iv) standardized data extraction; (v) targeted appraisal of NF-κB and related inflammatory signaling cascades; (vi) whole-genome and transcriptomic expression analysis; (vii) characterization of chromatin and epigenetic modifications; (viii) comparative evaluation of circulating inflammatory biomarkers; (ix) qualitative cross-study synthesis; and (x) delineation of critical literature gaps. This systematic workflow provides a rigorous analytical framework for understanding the capacity of meditation interventions to modulate genomic, epigenetic, and inflammatory phenotypes.

3. Results

The synthesized empirical evidence demonstrates that meditation and structured mind–body practices elicit consistent alterations in inflammatory gene transcription, chromatin-modifying enzyme expression, and immune-regulatory pathways. Across diverse genomic investigations, the NF-κB transcriptional network emerged as the most reliably modulated pathway. The systematic review by Burić *et al.* synthesizing 18 gene-expression investigations reported that mind–body practices predominantly downregulated NF-κB-dependent pro-inflammatory genes. Specifically, 81% of the evaluated studies that measured inflammatory gene networks or NF-κB signaling reported statistically significant downregulation following intervention (3).

Transcriptomic profiling reveals that relaxation and meditative interventions alter broad physiological networks beyond isolated inflammatory markers. Bhasin *et al.* identified rapid alterations in functional transcriptional networks governing energy metabolism, mitochondrial phosphorylation, cellular aging, and insulin sensitivity in

both novice and experienced practitioners (4). Crucially, these broad transcriptomic shifts were coupled with marked attenuation of NF-κB-related gene expression, demonstrating that mind–body interventions coordinate metabolic and anti-inflammatory cellular pathways concurrently (4).

Epigenetic investigations provide evidence of rapid chromatin remodeling in response to meditative practices. Kaliman *et al.* evaluated 19 experienced meditators subjected to an intensive one-day mindfulness intervention compared to a matched control cohort engaging in quiet leisure activities (2). Peripheral blood mononuclear cell analysis revealed significant post-intervention downregulation of histone deacetylase genes (*HDAC2*, *HDAC3*, and *HDAC9*) alongside reduced transcription of pro-inflammatory genes, including *RIPK2* and *COX2* (2). These findings establish that intensive mindfulness alters epigenetic regulatory machinery in tandem with pro-inflammatory gene expression.

Controlled clinical trials substantiate these transcriptional findings across community and clinical populations. Lindsay *et al.* conducted a randomized controlled trial comprising 190 stressed older adults randomized to either an 8-week MBSR program or a structurally matched health education control (1). Genome-wide transcriptional profiling of circulating leukocytes demonstrated that MBSR participants experienced significant downregulation of NF-κB-associated pro-inflammatory gene expression; however, circulating plasma levels of IL-6 and CRP showed no statistically significant reduction across assessment timepoints (1).

The systematic review and meta-analysis by Black and Slavich, synthesizing randomized controlled trials involving 1,602 participants, provided corroborating evidence (15). Their analysis identified consistent downregulation of NF-κB transcriptional activity and modest reductions in circulating CRP alongside elevated circulating cell-mediated immune parameters. Conversely, systemic circulating concentrations of IL-1, IL-6, IL-8, IL-10, interferon-gamma, and TNF-α demonstrated inconsistent and non-significant alterations across trials (15). Collectively, these empirical results establish that meditation exerts a reliable, consistent suppressive effect on intracellular pro-inflammatory gene transcription, particularly within the NF-κB axis, whereas corresponding changes in circulating plasma cytokine biomarkers remain variable and biologically decoupled.

4. Discussion

The findings synthesized in this review demonstrate that meditation modulates inflammatory biology predominantly at the level of transcriptional regulation within immune cells. The most prominent and convergent empirical finding across the literature is the consistent attenuation of the NF-κB transcriptional pathway. This observed downregulation carries profound biological significance, as NF-κB operates as the primary master switch orchestrating the transcription of pro-inflammatory cytokines, chemokines, and acute-phase reactants. The systematic synthesis by Burić *et al.* provides robust evidence for this mechanistic effect, demonstrating that mind–body practices elicit a transcriptional pattern directly opposite to the genomic profile induced by chronic psychosocial adversity (3).

The attenuation of NF-κB signaling holds substantial clinical relevance. Chronic psychosocial stress hyperactivates the sympathetic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis, stimulating beta-adrenergic receptors on immune cells and triggering intracellular cascades that activate NF-κB. Meditative practices downregulate sympathetic outflow, decrease catecholamine production, and restore homeostatic autonomic tone. Consequently, the observed reduction in NF-κB-dependent transcription reflects a neuroendocrine-mediated dampening of intracellular inflammatory triggers. While it remains unresolved whether meditation influences NF-

κB signaling via direct transcriptional repression or indirectly through stress-attenuation cascades, the molecular evidence confirms an integrated link connecting autonomic regulation, neuroendocrine pathways, and cellular immune function.

Epigenetic investigations introduce a vital regulatory dimension to this paradigm. Kaliman *et al.* demonstrated that acute meditation modulates histone deacetylase expression (*HDAC2*, *HDAC3*, *HDAC9*) alongside reduced transcription of pro-inflammatory loci (*RIPK2*, *COX2*) (2). Histone deacetylases govern the removal of acetyl moieties from lysine residues on histones, modulating chromatin accessibility and gene transcription. Downregulation of specific HDACs may permit chromatin restructuring that selectively represses pro-inflammatory promoters or enhances anti-inflammatory regulatory elements. Nonetheless, because these findings were observed in experienced meditators during intensive practice, generalizability to novice practitioners warrants cautious interpretation.

A primary insight emerging from recent clinical trials is the distinct dissociation between intracellular gene expression and circulating systemic protein concentrations. In the rigorous trial by Lindsay *et al.* (1), eight weeks of MBSR produced statistically significant downregulation of NF-κB-associated gene transcription without concurrent declines in circulating plasma IL-6 or CRP. This empirical divergence is critically important for interpreting mind–body literature. Downregulated gene expression in peripheral leukocytes reflects decreased intracellular transcriptional readiness, whereas circulating protein concentrations represent the cumulative systemic output of diverse tissues, including hepatic clearance, adipose secretion, vascular endothelium, and immune depots. Furthermore, transcriptional downregulation may require a prolonged latency period before translating into diminished circulating systemic protein pools.

The systematic evaluation by Black and Slavich corroborates this selective biological action (15). While trials reliably document attenuated NF-κB activity and modest CRP reductions, findings for circulating cytokines such as IL-6 and TNF-α remain heterogeneous. This selectivity implies that meditation does not act as a blunt, broad-spectrum anti-inflammatory agent, but rather as an upstream modulator whose effects depend on baseline physiological stress, tissue compartment, and practice dosage.

Furthermore, empirical evidence refutes the misconception that meditation induces generalized immunosuppression. Several genomic trials demonstrate that intensive meditation upregulates immune-supportive networks, interferon-mediated antiviral defenses, and metabolic repair pathways while selectively repressing pro-inflammatory signaling (10–12). Thus, meditation serves as an adaptive biological modulator that attenuates pathological chronic inflammation while preserving or enhancing immunocompetent host defenses.

Methodological heterogeneity across existing studies remains a substantial challenge. Published trials differ markedly in meditation style, training dosage, practitioner experience, control condition rigor, and molecular platforms. Burić *et al.* emphasized that many genomic investigations are limited by modest cohort sizes and insufficient control for lifestyle confounding variables such as sleep architecture, nutritional patterns, and physical activity (3). Additionally, many interventions are of brief duration; while transcriptional shifts manifest within hours or days, systemic protein alterations and clinical tissue remodeling may require sustained long-term practice.

Synthesizing current mechanistic data suggests a multi-tier regulatory cascade through which meditation alters human biology:

Meditation practice → psychological stress regulation → autonomic and neuroendocrine modulation → cellular receptor signaling → intracellular transcriptional remodeling (NF-κB and HDAC suppression) → altered inflammatory gene expression → downstream immune and physiological homeostasis.

Empirical literature provides robust evidence supporting cellular and transcriptional modulation, particularly across the NF-κB signaling network, whereas systemic circulating protein outcomes require further validation.

In conclusion, the scientific literature demonstrates that meditation modulates human biology through coordinated alterations in gene transcription, NF-κB signaling, chromatin-modifying enzymes, and immune-regulatory pathways. The most consistent empirical finding is the downregulation of NF-κB and its downstream pro-inflammatory gene targets (3). However, these intracellular genomic shifts do not uniformly produce corresponding reductions in circulating inflammatory biomarkers such as IL-6 and CRP (1). Consequently, meditation is most accurately conceptualized as a biological modulator of cellular immune and genomic pathways rather than an unvarying systemic anti-inflammatory intervention. Future research must deploy large, well-powered randomized controlled trials combining transcriptomic, epigenomic, and proteomic profiling to determine the clinical durability and health translation of these molecular adaptations.

5. Research-publication interpretation

Based on a rigorous synthesis of empirical data and functional genomic evaluations, the scientifically defensible interpretation is framed as follows:

"Meditation appears to modulate biology primarily through changes in pro-inflammatory gene regulation, particularly NF-κB-related transcriptional activity, while evidence for reductions in circulating systemic inflammatory biomarkers remains inconsistent."

This nuanced formulation accurately reflects empirical scientific findings by distinguishing between consistent intracellular gene regulation and variable circulating systemic protein biomarkers, thereby preventing unverified generalizations regarding clinical systemic anti-inflammatory efficacy (1, 3).

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