
Buddhist Mindfulness and Anti-Inflammatory Medication in Stress-Related Inflammation

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Abstract

This qualitative documentary study compares Buddhist mindfulness practice and anti-inflammatory medication regarding stress-related inflammation. It addresses three objectives: (1) to analyze the reported outcomes and proposed mechanisms of Buddhist mindfulness practice; (2) to examine the outcomes, mechanisms, and limitations of major anti-inflammatory drug classes; and (3) to compare the two modalities within a psychoneuroimmunological framework. Primary Buddhist sources were examined alongside peer-reviewed biomedical and mindfulness literature, with recent empirical studies prioritized and foundational sources retained where necessary.

The review suggests that mindfulness-based interventions may influence stress-related inflammatory processes by changing attention, emotional regulation, autonomic activity, and hypothalamic-pituitary-adrenal axis function. Evidence suggests possible reductions in selected markers such as cortisol, C-reactive protein, interleukin-6, and tumor necrosis factor-alpha, although findings are heterogeneous and depend on population, intervention, and study design. Anti-inflammatory medications act more directly on inflammatory pathways and remain essential for clinically indicated inflammatory conditions, but their benefits and risks vary substantially by drug class, dose, duration, and patient characteristics. Therefore, the comparison does not support simple equivalence between mindfulness and pharmacotherapy. Instead, mindfulness is best understood as a potentially useful complementary strategy for stress regulation, whereas medication should be prescribed according to established clinical indications. The proposed integrative framework is conceptual and requires direct empirical testing before it can guide clinical treatment.

Keywords: Buddhist mindfulness; stress-related inflammation; psychoneuroimmunology; anti-inflammatory medication; HPA axis; cytokines; integrative health.

Introduction

Psychological stress can influence immune and endocrine activity, and persistent stress exposure has been associated with dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, sympathetic activation, and changes in inflammatory signaling. Researchers frequently examine biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- α) in stress and inflammation research. However, these associations are complex and bidirectional, and stress should not be treated as the sole cause of inflammatory disease (Black & Slavich, 2016; Pascoe et al., 2017).

Biomedical management of inflammatory conditions includes several drug classes, among them nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, conventional disease-modifying antirheumatic drugs, targeted small-molecule agents, and biologics. These treatments differ substantially in indication, mechanism, speed of action, and adverse-effect profile. NSAIDs primarily inhibit cyclooxygenase pathways, corticosteroids regulate broad inflammatory gene expression, and targeted agents act on selected cytokine or signaling pathways (Barnes, 2006; Brunton et al., 2018; Díaz-González & Sánchez-Madrid, 2015; Roskoski, 2016). Their clinical role cannot therefore be reduced to a single category of ‘symptomatic treatment.’

Buddhist mindfulness originates in a different conceptual and practical context. In the Satipaṭṭhāna tradition, mindfulness (sati) is cultivated through systematic contemplation of body, feelings, mind, and dhammas as part of the path toward insight and liberation (Bodhi Bhikkhu, 1994). Modern clinical programs such as Mindfulness-Based Stress Reduction (MBSR) adapt selected contemplative practices for health-related settings (Kabat-Zinn, 1990). These programs should not be equated with the full soteriological aims of Theravāda practice, but they provide a bridge through which attentional and affective processes associated with mindfulness can be investigated empirically.

Research has increasingly examined whether mindfulness-based interventions influence inflammatory and neuroendocrine markers. Systematic reviews and clinical studies suggest potential effects on stress physiology and immune regulation, but effect sizes and biomarkers vary across studies, and many trials have small samples or heterogeneous interventions (Black & Slavich, 2016; González-Moret et al., 2020; Gardi et al., 2022; Alhawtmeh et al., 2024). These findings support cautious interest rather than a conclusion that mindfulness is an anti-inflammatory treatment equivalent to medication.

The Buddhist conceptual contribution requires similar caution. Abhidhamma discussions of mind-conditioned material phenomena (*cittaja-rūpa*) articulate a doctrinal account of mind-body dependence (Ācariya Anuruddha, 1993). This concept may offer a useful comparative lens for contemporary mind-body research, but it should not be presented as a premodern description of cytokines, cortisol, or neural circuitry. Doctrinal categories and biomedical mechanisms operate within different explanatory systems and should be compared analogically rather than identified as scientifically equivalent.

A clear research gap therefore remains. Mindfulness and anti-inflammatory medication are often discussed in separate literatures, and few reviews compare them using common dimensions such as level of action, time course, evidence base, safety, and appropriate role in care. The present study addresses this gap through a structured documentary comparison. Rather than asking which modality is ‘better,’ it examines what each is designed to do, what evidence supports its effects, where the mechanisms differ, and where a complementary relationship may be conceptually plausible.

Research Objectives

1. To analyze the reported outcomes and proposed mechanisms of Buddhist mindfulness practice in relation to stress-related inflammation.
2. To analyze the outcomes, mechanisms, and limitations of anti-inflammatory medications relevant to inflammatory regulation.
3. To compare the two modalities in terms of mechanisms, evidence, safety, time course, and potential complementary roles.

Literature Review

The literature relevant to this study falls into three overlapping areas: Buddhist and clinical mindfulness, psychoneuroimmunology, and anti-inflammatory pharmacology. Buddhist sources frame mindfulness as part of a wider ethical and contemplative path, whereas contemporary clinical programs operationalize mindfulness primarily as attentional and emotional training. Gunaratana (2011) offers a modern Theravāda-oriented presentation of mindfulness practice, while Kabat-Zinn (1990) provides the foundational clinical formulation of MBSR. These traditions overlap in technique but differ in purpose, terminology, and scope.

Psychoneuroimmunological research provides a scientific framework for examining pathways through which psychological processes may influence inflammatory activity. Black and Slavich (2016) reviewed randomized controlled trials and found that mindfulness meditation may affect selected immune parameters, although the evidence was heterogeneous. Pascoe et al. (2017) likewise reported changes in stress-related physiological measures across mindfulness-based and related interventions. More recent clinical studies have reported changes in CRP, cytokines, cortisol, and other inflammatory markers in specific populations, including those with inflammatory bowel disease, renal disease, and COVID-19 (González-Moret et al., 2020; Subramanian et al., 2022; Gardi et al., 2022; Alhawatmeh et al., 2024).

The pharmacological literature is more mechanistically specific because it defines drug targets more directly. NSAIDs inhibit cyclooxygenase-mediated prostaglandin synthesis; corticosteroids alter transcription of multiple inflammatory genes; and targeted agents such as JAK inhibitors and cytokine-directed biologics act on selected immune pathways (Barnes, 2006; Brunton et al., 2018; Díaz-González & Sánchez-Madrid, 2015; Roskoski, 2016). Efficacy and toxicity vary by drug class and clinical indication, which makes broad comparisons between ‘medication’ and ‘mindfulness’ potentially misleading unless the level of comparison is clearly specified.

Additional literature concerns inflammation as a treatment target in chronic disease. For example, Ridker et al. (2017) showed that targeted anti-inflammatory therapy with canakinumab reduced recurrent cardiovascular events in selected patients with prior myocardial infarction and elevated inflammatory risk. This finding confirms the clinical importance of inflammatory pathways but does not show that such therapy treats psychological stress. Conversely, mindfulness trials may target stress and quality of life while measuring inflammatory biomarkers only secondarily.

The literature therefore supports a functional, rather than equivalence-based, framework. Mindfulness may influence upstream psychological and neuroendocrine processes associated with stress reactivity, whereas anti-inflammatory drugs act more directly on inflammatory mediators or immune pathways. The evidence does not justify describing all mindfulness effects as ‘etiological’ or all medication effects as merely ‘symptomatic.’ A stronger scientific comparison must distinguish conceptual level, clinical indication, biomarker outcomes, and evidentiary strength.

Conceptual Framework

The conceptual framework compares Buddhist mindfulness practice and anti-inflammatory medication across five dimensions: intended target, proposed mechanism, measurable outcomes, time course, and safety/limitations. The comparison is interpretive rather than a claim of therapeutic equivalence. Mindfulness is examined primarily as a stress-regulation approach that may influence neuroendocrine and immune processes, whereas medications are examined according to their established pharmacological targets and clinical uses.

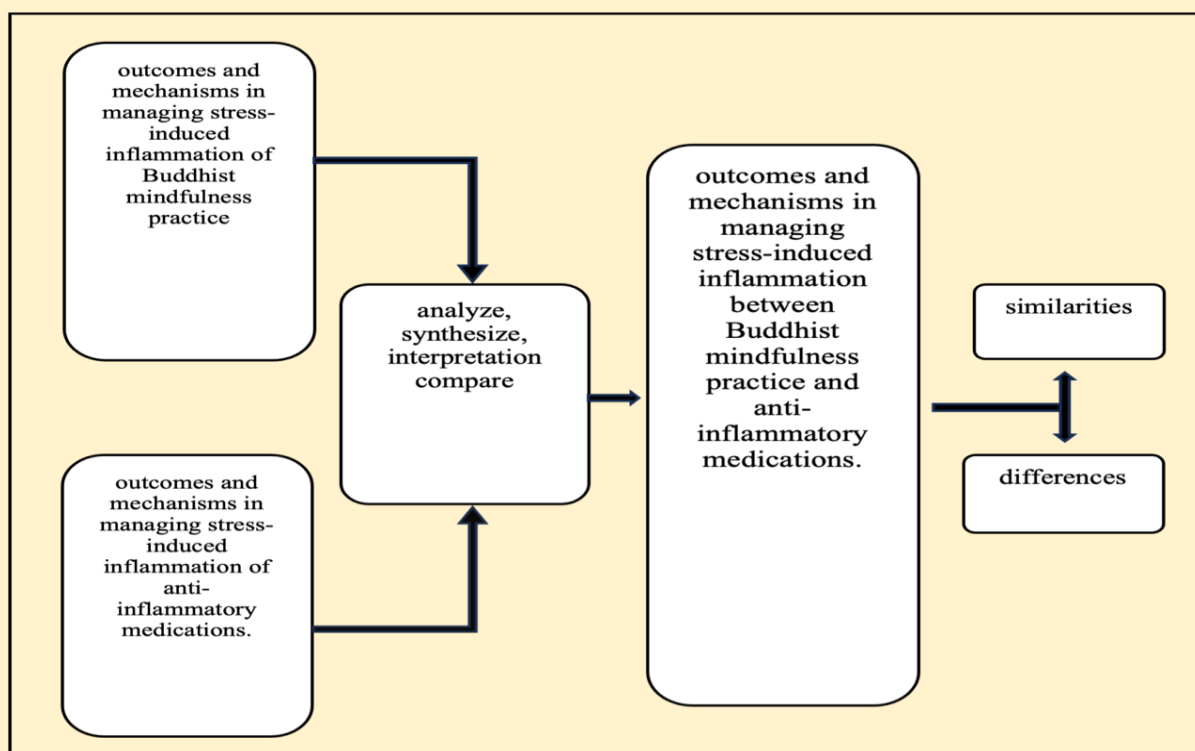


Figure 1. Conceptual Framework

Research Methodology

This study used a qualitative documentary-review design. It did not involve patients, clinical treatment, or experimental comparison. The purpose was to synthesize Buddhist doctrinal sources and biomedical literature in order to compare conceptual mechanisms, reported outcomes, and limitations.

Source selection followed two principles. First, we prioritized recent peer-reviewed empirical studies published mainly between 2020 and 2025 that made claims about mindfulness, inflammatory biomarkers, and clinical outcomes.

Second, we retained earlier foundational sources where they remain central to Buddhist doctrine, pharmacology, or the development of mindfulness-based clinical programs. These included the *Satipaṭṭhāna* and *Ānāpānasati* traditions, Abhidhamma summaries, Kabat-Zinn's MBSR formulation, and established pharmacological references.

Sources were included when they addressed at least one of the following: (1) mindfulness or meditation and stress-related physiological or inflammatory outcomes; (2) mechanisms or clinical effects of anti-inflammatory medications; (3) psychoneuroimmunological links among stress, endocrine regulation, and inflammation; or (4) Buddhist accounts of mindfulness and mind-body relations relevant to the conceptual comparison. We excluded non-scholarly sources and materials without a clear connection to the research objectives.

Analysis proceeded in three stages. First, we analyzed the mindfulness and pharmacological literature separately to identify proposed mechanisms, outcome measures, benefits, risks, and evidentiary limitations. Second, we organized findings in a comparison matrix using shared dimensions: level of action, onset and duration, biomarker effects, clinical indication, safety, and role in long-term care. Third, Buddhist concepts were interpreted alongside, but not equated with, biomedical mechanisms. This distinction avoided treating doctrinal categories such as *taṇhā*, *papañca*, or *cittaja-rūpa* as direct biological variables.

Because the reviewed literature identified no head-to-head trials directly comparing mindfulness with anti-inflammatory drug therapy, the study does not estimate comparative effect sizes or claim superiority of one modality. Any integrative framework proposed below is therefore conceptual and hypothesis-generating rather than a validated clinical protocol.

Research Results

Objective 1: Outcomes and Mechanisms of Buddhist Mindfulness Practice

The findings indicate that Buddhist mindfulness approaches stress and suffering primarily through the regulation of mental processes rather than through direct intervention in inflammatory pathways. Within the framework of the Four Noble Truths, *dukkha* encompasses multiple dimensions of suffering associated with embodied existence, illness, unwanted experience, and psychological distress, while *taṇhā* is identified as a central condition contributing to the arising and continuation of suffering (Bodhi Bhikkhu, 2000). Mindfulness (*sati*),

together with clear comprehension (*sampajañña*) and other wholesome mental qualities, is cultivated as part of a broader path concerned with understanding and transforming patterns of reactivity.

The Abhidhamma provides an additional conceptual account of mind–body interdependence through the notion of *cittaja-rūpa*, or material phenomena conditioned by mind (Ācariya Anuruddha, 1993). This doctrinal category suggests that Buddhist analysis does not treat mental and physical processes as entirely independent. However, *cittaja-rūpa* should not be interpreted as a premodern biomedical account of cortisol, cytokines, neural circuits, or inflammatory disease. Its relevance to the present study is therefore comparative and conceptual rather than a claim of direct scientific equivalence.

Contemporary empirical research provides a different but potentially complementary explanatory framework. Mindfulness-based and related meditation interventions have been associated with changes in physiological stress measures, including cortisol, autonomic activity, and selected inflammatory biomarkers. Meta-analytic and systematic-review evidence suggests possible reductions in CRP, TNF- α , cortisol, blood pressure, and other stress-related measures, although findings vary according to population, intervention type, duration, and study design (Black & Slavich, 2016; Pascoe et al., 2017). Accordingly, the evidence supports a potential regulatory effect rather than a uniform anti-inflammatory response.

From a psychoneuroimmunological perspective, mindfulness may operate predominantly through a top-down pathway. Training attention and reducing habitual emotional reactivity may influence neural and neuroendocrine systems involved in stress regulation, which in turn may affect autonomic and immune processes. Proposed mechanisms include reduced stress reactivity, altered HPA-axis activity, improved autonomic regulation, and downstream modulation of inflammatory signaling. Some studies also suggest that contemplative interventions may influence transcriptional pathways associated with inflammation, although this evidence remains developing and should not be interpreted as demonstrating a consistent shift of immune cells from pro-inflammatory to regulatory states across populations (Black & Slavich, 2016; Gardi et al., 2022).

From a Buddhist interpretive perspective, cultivation of *sati*, *sampajañña*, and *upekkhā* may reduce patterns of cognitive and emotional reactivity that contribute to sustained psychological stress. The interruption of repetitive mental

proliferation (*papañca*) provides a useful conceptual parallel to contemporary models in which persistent cognitive-emotional activation contributes to prolonged stress-system activation. The Buddhist and biomedical accounts remain epistemologically distinct, but both support the proposition that changes in mental regulation may have consequences for embodied stress responses.

Objective 2: Outcomes and Mechanisms of Anti-Inflammatory Medications

The findings indicate that anti-inflammatory medications act more directly on biochemical and immune pathways than mindfulness-based interventions. This study considers major pharmacological categories, including nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, disease-modifying antirheumatic drugs (DMARDs), targeted small-molecule agents, and biological therapies. These drug classes differ substantially in mechanism, clinical indication, speed of action, efficacy, and adverse-effect profile (Brunton et al., 2018).

NSAIDs, including aspirin, ibuprofen, naproxen, and selective COX-2 inhibitors such as celecoxib, primarily inhibit cyclooxygenase enzymes and thereby reduce the synthesis of prostaglandins involved in pain, fever, and inflammation (Díaz-González & Sánchez-Madrid, 2015). Their effects are relatively direct but vary with drug selectivity, dose, duration of treatment, and patient characteristics.

Corticosteroids such as prednisolone and dexamethasone exert broad anti-inflammatory and immunomodulatory effects through glucocorticoid receptors. After receptor binding, glucocorticoid signaling influences the transcription of numerous inflammatory genes and interferes with transcription factors such as NF- κ B and AP-1. These mechanisms can suppress inflammatory mediator production, including several cytokines and chemokines (Barnes, 2006). Their clinical effects may be substantial, but responsiveness varies by disease and patient, and prolonged exposure is associated with important adverse effects.

DMARDs and targeted immunomodulatory agents operate through additional mechanisms. JAK inhibitors such as tofacitinib and baricitinib interfere with intracellular JAK–STAT signaling, whereas biological agents may target specific cytokines or their receptors, including TNF- α and IL-6 pathways (Roskoski, 2016). Unlike purely symptomatic treatments, several of these therapies modify inflammatory processes central to disease activity and, under appropriate conditions, can alter disease progression.

The benefits of pharmacological treatment must nevertheless be balanced against class-specific risks. NSAIDs may increase gastrointestinal, renal, and cardiovascular risks, particularly with prolonged use or in vulnerable patients (Aronson, 2009). Long-term systemic corticosteroid exposure may contribute to adrenal suppression, metabolic complications, osteoporosis, infection risk, and other adverse effects (Barnes, 2006). Targeted immunomodulatory drugs also require condition-specific screening and monitoring, particularly because interfering with immune pathways can increase infection susceptibility and cause other adverse effects.

The findings therefore do not support characterizing all anti-inflammatory medication as merely symptomatic. Instead, pharmacological therapies act primarily at molecular or cellular levels and may suppress, regulate, or modify inflammatory pathways depending on the drug class and the disease being treated. At the same time, these medications are not generally designed to address psychological stress, cognitive reactivity, or behavioral patterns that may contribute to stress-related physiological activation.

Objective 3: Comparative Results

Similarities

The comparison identifies several areas of overlap, while also demonstrating important differences in evidentiary strength and mechanism. Both mindfulness-based interventions and anti-inflammatory pharmacotherapy may influence inflammatory outcomes, but through different pathways and with varying degrees of directness.

Mindfulness and meditation studies have reported changes in selected inflammatory and stress-related biomarkers, including CRP, TNF- α , cortisol, and other physiological indicators, although the findings remain heterogeneous (Black & Slavich, 2016; Pascoe et al., 2017). Pharmacological agents can produce substantially more direct and predictable effects on inflammatory mediators when used for appropriate clinical indications.

Both modalities may also influence interconnected regulatory systems. Mindfulness may affect inflammatory activity indirectly through changes in psychological stress, neuroendocrine signaling, autonomic regulation, and possibly inflammatory gene-expression pathways. Pharmacological therapies act more directly on enzymes, receptors, transcriptional pathways, cytokines, or intracellular signaling systems. Thus, convergence may occur at the level of

inflammatory outcomes even though the initiating mechanisms differ considerably.

Some evidence further suggests that mindfulness-based interventions may influence transcriptional pathways associated with inflammatory regulation, including NF- κ B-related processes, while corticosteroids and several other anti-inflammatory therapies directly interfere with inflammatory transcription and signaling mechanisms (Black & Slavich, 2016; Barnes, 2006). This similarity concerns the eventual biological pathways affected rather than equivalence in mechanism, magnitude, or clinical efficacy.

Differences

The principal difference identified in this study concerns the level at which the two modalities primarily intervene. Mindfulness can be described heuristically as predominantly top-down: intervention begins with attention, awareness, emotional regulation, and patterns of psychological reactivity, with possible downstream effects on neural, endocrine, autonomic, and immune processes. Anti-inflammatory medications operate more directly at biochemical, cellular, or immune levels by modifying inflammatory mediators and signaling pathways.

This distinction should not, however, be interpreted as an opposition between an “etiological” mindfulness treatment and a merely “symptomatic” pharmacological treatment. Stress-related inflammation is multifactorial, and psychological processes are only one contributing factor. Likewise, several pharmacological therapies modify mechanisms central to inflammatory disease rather than simply suppressing symptoms. The distinction is therefore better described as one of the primary level and mode of intervention.

A further difference concerns time course. Mindfulness generally requires repeated training and sustained practice, and its physiological effects, where present, are likely to develop through changes in stress regulation over time. It may therefore contribute to resilience, coping, and reduced stress reactivity. Pharmacological agents often produce faster effects on inflammatory activity, although onset varies substantially by drug class. Medications are not necessarily restricted to reactive use: some are prescribed chronically to prevent disease activity, relapse, or inflammatory progression.

The modalities also differ in scope. Mindfulness-based interventions may influence multiple interconnected aspects of functioning, including perceived stress, emotional regulation, sleep, pain experience, autonomic activity, and

quality of life. These broader effects are consistent with the fact that mindfulness primarily targets patterns of experience and self-regulation rather than a single molecular pathway. Buddhist accounts of mind–body interdependence provide a conceptual context for interpreting this multidimensional orientation, although they should not be equated directly with contemporary physiological models.

Pharmacological treatments are generally more pathway-specific. This specificity can be clinically advantageous because it allows treatment to target known disease mechanisms. However, specificity also means that different inflammatory diseases may require different therapeutic targets, combinations, or treatment strategies, with benefits and risks evaluated individually.

The comparison also indicates different relationships with endogenous regulation. Mindfulness may support the individual's capacity for psychological and physiological self-regulation by reducing stress reactivity and improving coping. Some studies report changes in cortisol, autonomic measures, and inflammatory biomarkers consistent with improved regulation, but the evidence does not support claims of complete restoration of homeostasis or immune function (Gardi et al., 2022; Alhawtmeh et al., 2024). Pharmacological treatment, by contrast, modifies physiological processes through externally administered agents; depending on the medication, prolonged use may also influence endogenous feedback systems, as occurs with HPA-axis suppression during chronic systemic corticosteroid therapy (Barnes, 2006).

Taken together, the results do not establish the superiority of one modality over the other. Pharmacotherapy has a substantially stronger evidence base for treating diagnosed inflammatory diseases, whereas mindfulness appears more appropriately understood as a behavioral and contemplative intervention that may support stress regulation, coping, quality of life, and selected physiological outcomes. Their most defensible relationship is therefore complementary rather than competitive. For patients with clinically significant inflammatory disease, mindfulness should not replace indicated pharmacological treatment but may serve as an adjunctive strategy where appropriate.

Discussions

The findings clarify that Buddhist mindfulness practice and anti-inflammatory medication operate at different but potentially complementary levels. Rather than supporting a binary distinction between “root-cause mindfulness” and “symptomatic medication,” the results indicate that

mindfulness primarily engages psychological and behavioral processes related to stress regulation, whereas pharmacological treatments act more directly on biochemical, cellular, and immune pathways.

For Objective 1, the findings align with prior research suggesting that mindfulness-based interventions may influence stress physiology and select inflammatory markers. Systematic and meta-analytic evidence reports changes in cortisol, CRP, TNF- α , and other stress-related measures, though effects remain heterogeneous across populations and intervention designs (Black & Slavich, 2016; Pascoe et al., 2017). More recent studies likewise suggest possible biomarker changes in specific clinical populations (González-Moret et al., 2020; Gardi et al., 2022; Alhawtmeh et al., 2024). These findings support plausible psychoneuroimmunological pathways, but they do not establish mindfulness as a direct anti-inflammatory treatment equivalent to pharmacotherapy.

The Buddhist contribution is primarily conceptual. Practices involving *sati*, *sampajañña*, and *upekkhā* may reduce habitual cognitive and emotional reactivity, while concepts such as *taṇhā*, *dosa*, and *papañca* help explain how mental patterns can sustain suffering and stress. The Abhidhamma concept of *cittaja-rūpa* further expresses mind-body interdependence (Ācariya Anuruddha, 1993). However, these doctrinal concepts should not be equated directly with the HPA axis, cytokines, or neural mechanisms. Their value lies in offering a distinct interpretive framework that can enter into dialogue with contemporary mind-body research.

For Objective 2, the findings confirm that anti-inflammatory medications have greater mechanistic specificity. NSAIDs inhibit cyclooxygenase pathways, corticosteroids influence inflammatory gene transcription, and JAK inhibitors and biologic agents target specific immune signaling pathways (Barnes, 2006; Brunton et al., 2018; Roskoski, 2016). This evidence also shows why pharmacotherapy should not be described as merely symptomatic; some treatments modify pathways central to disease activity. However, their use must be balanced against class-specific risks.

Objective 3 shows that the most defensible comparison concerns level and direction of action. Mindfulness may be described heuristically as predominantly top-down, beginning with attention and emotional regulation and potentially influencing downstream physiological systems. Medications act more directly at molecular or cellular levels. Neither pathway is exclusively one-directional, and neither should be treated as inherently superior.

The study therefore supports a complementary rather than competitive model. Pharmacotherapy remains essential when clinically indicated, while mindfulness may serve as an adjunctive strategy for stress regulation, coping, quality of life, and selected physiological outcomes. Because no direct head-to-head trials were identified, this framework remains conceptual and should be tested in future empirical research.

Knowledge from Research

The study's principal contribution is a comparative framework that distinguishes Buddhist mindfulness practice and anti-inflammatory medication by their primary levels of intervention, rather than treating them as competing or equivalent therapies. Mindfulness primarily engages attention, emotional regulation, cognitive reactivity, and stress-related self-regulation, with possible downstream effects on neuroendocrine, autonomic, and immune processes. Anti-inflammatory medications, by contrast, act more directly on molecular, cellular, and immune pathways through pharmacologically defined mechanisms.

A second contribution clarifies the relationship between Buddhist doctrine and biomedical science. Concepts such as *taṇhā*, *dosa*, *papañca*, and *cittaja-rūpa* provide a Buddhist framework for understanding suffering, mental reactivity, and mind-body interdependence. However, these concepts should not be interpreted as direct equivalents of cortisol regulation, cytokine signaling, neural circuitry, or inflammatory gene expression. Their value lies in offering a distinct interpretive framework that can contribute to interdisciplinary dialogue without collapsing Buddhist and biomedical explanatory systems into one another.

A third contribution is the rejection of the simplified distinction between “root-cause mindfulness” and “symptomatic medication.” The findings show that several pharmacological therapies modify mechanisms central to inflammatory disease, while mindfulness may influence important psychological and physiological processes without directly treating inflammatory pathology. The more defensible distinction therefore concerns level, direction, and mode of action rather than causal superiority.

Based on these findings, the study proposes a complementary model in which clinically indicated pharmacotherapy addresses inflammatory disease through established biomedical mechanisms, while mindfulness may support stress regulation, coping, quality of life, and selected physiological outcomes.

This model is conceptual rather than prescriptive and requires empirical testing through comparative and adjunctive clinical research.

Conclusion

This study compared Buddhist mindfulness practice and anti-inflammatory medication in relation to stress-related inflammation. The findings indicate that the two modalities operate primarily at different levels. Mindfulness begins with attention, emotional regulation, and patterns of psychological reactivity and may influence downstream neuroendocrine, autonomic, and immune processes. Anti-inflammatory medications act more directly on biochemical and immune pathways and remain essential for clinically indicated inflammatory conditions.

The study also demonstrates the importance of maintaining conceptual boundaries between Buddhist doctrine and biomedical science. Buddhist concepts such as *taṇhā*, *papañca*, and *cittaja-rūpa* offer a valuable account of suffering, mental reactivity, and mind-body interdependence, but they should not be equated directly with contemporary physiological mechanisms.

The evidence therefore does not support a binary opposition between an etiological mindfulness approach and merely symptomatic pharmacological treatment. Instead, the two approaches address different dimensions of stress-related and inflammatory processes. Pharmacotherapy has a stronger evidence base for treating diagnosed inflammatory disease, while mindfulness may support stress regulation, coping, quality of life, and select physiological outcomes.

The most defensible conclusion, therefore, is a complementary model rather than therapeutic substitution. Mindfulness should not replace indicated medical treatment, but it may serve as an adjunctive strategy in appropriate contexts. Because direct head-to-head evidence remains limited, the proposed framework should be regarded as hypothesis-generating and tested through future clinical and interdisciplinary research.

Suggestions

1. Suggestions for Clinical Practice

Mindfulness should be presented as an adjunctive stress-management option rather than as a replacement for prescribed anti-inflammatory treatment. Clinicians who incorporate mindfulness should consider patient preference, psychological readiness, disease severity, and the feasibility of sustained practice.

Any medication adjustment or tapering should remain under the supervision of the prescribing clinician and should not be inferred from mindfulness participation alone.

Where stress appears to exacerbate symptoms or impair coping, clinicians may consider evidence-based psychological or mindfulness-based programs as part of multidisciplinary care. Such integration should include appropriate screening, informed consent, realistic expectations, and monitoring for adverse psychological responses. The purpose is to support coping and self-regulation, not to reclassify meditation as a pharmacological substitute.

2. Suggestions for Further Research

Future research should prioritize direct comparative and adjunctive designs. A clinically meaningful trial could compare standard treatment alone with standard treatment plus a standardized mindfulness intervention while measuring identical inflammatory biomarkers, symptom outcomes, quality of life, medication use, adherence, and adverse events over sufficient follow-up. A three-arm design including mindfulness alone would be appropriate only where withholding standard treatment is ethically and medically acceptable.

Further studies should distinguish among mindfulness techniques, intervention intensity, teacher training, participant diagnosis, and baseline inflammatory status. Mechanistic research should avoid assuming that all contemplative practices act through the same pathways. Longitudinal studies are also needed to determine whether any biomarker changes are durable and whether they translate into clinically meaningful outcomes.

Sutta Piṭaka

MN Majjhima Nikāya

DN Dīgha Nikāya

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