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PHYSICAL ACTIVITY IN DEPRESSION MANAGEMENT: MECHANISMS, CLINICAL IMPLICATIONS, AND EXERCISE PRESCRIPTION - A NARRATIVE REVIEW

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ABSTRACT

Background: Depression is one of the top contributors to global disability and represents an important public health challenge. Studies show an increasing body of evidence that physical activity might be instrumental in both the prevention of and intervention with depressive disorders.
Purpose of the investigation: This narrative review intends to summarize and synthesize current evidence pertaining to the antidepressant effects of physical activity, considering suggested mechanisms, clinical results, and prescription advice of exercise.
Material and methodology: The review was compiled after conducting a systematic search of PubMed/MEDLINE, Embase and the Cochrane Library for peer-reviewed publications, stressing high-impact findings published between 2024–2026. Systematic reviews, meta-analyses and well-designed randomized controlled trials (RCTs) with large prospective cohorts were prioritized.
Results: The evidence base suggests that there is significant evidence that exercise is associated with decreases in depressive symptoms in varied populations. The proposed mechanisms are changes in monoaminergic neurotransmission, elevation of BDNF, normalization of HPA-axis function, and anti-inflammatory effects. Data support the dose-response relationship, with even small increases in activity positively correlated with mental health improvement.
Conclusions: The interventions provided by exercise are beneficial, reasonable and cheap, which is relevant to the prevention and treatment of depression. Studies should focus more on developing specific combinations of exercise recommendations and specific exercise regimens tailored for each patient cohort.

KEYWORDS

Physical Activity; Depression; Exercise; Antidepressant Effects; Exercise Prescription; BDNF

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Introduction

Depression is one of the most common and most significant causes of disability across the world, with more than 280 million people suffering from it and being a major public health issue [12]. It is a heterogeneous disorder that is characterized by a chronic low mood, diminished pleasure, cognitive declines, and impaired daily functioning. Despite the centrality of pharmacological treatments and psychotherapy, large numbers of patients fail to reach full remission, highlighting the necessity of adjunctive strategies [4]. Behavioral strategies such as movement, particularly physical activity, have received growing attention as preventive and therapeutic strategies for individuals suffering from depression. Exercise is cheap, readily available, provides general good physical health benefits and offers an appealing adjunctive therapy in a clinical environment [8, 18].

Regular physical activity is associated with clinically meaningful reductions in depressive symptoms. The mechanisms of action of these antidepressant effects are probably multifactorial [10, 33]. Exercise exerts effects in monoamine neurotransmitter systems, increases BDNF, reinforces neuroplasticity, regulates the HPA axis and helps with systemic inflammation. Furthermore, physical exercise can be beneficial for mental health effects at the macro level by enhancing psychological well-being as well as by fostering cognitive performance, sleep quality, social relationships, etc. [10, 27, 32]. Despite increasing support for exercise-based approaches, important questions remain: Which modalities, intensities and frequencies of workouts or their duration might offer the best therapeutic benefit? How to enhance adherence among people with depression? In this narrative review, we discuss the most recently available evidence on the involvement of physical activities on the management of depression including biological processes of the disease, clinical relevance, and clinical and therapeutic exercise prescription guidance. It also discusses exercise as a potential preventive measure against depression as well as how to individualize exercise for clients suffering from depressive disorders.

Methodology

The review included RCTs, systematic reviews, meta-analyses and observational studies of physical activity in depression and depressive symptoms. Most included studies were published between 2013 and 2025, with seminal earlier studies included where they provided critical biological or clinical context. Searches in PubMed/MEDLINE, Embase, and the Cochrane Library included relevant keywords including; physical activity; exercise; depression; depressive symptoms; aerobic exercise; resistance training; BDNF; exercise prescription; mental health. This ensured that the selection of studies was guided by relevance to the review objectives, methodological rigor, and availability in the literature; systematic reviews, meta-analyses, RCTs, and large cohort studies were the prominent ones. In addition to these, reference lists of the selected articles were screened in order to identify further appropriate studies. In total, 37 publications that met predefined criteria for eligibility were retained. We did not conduct a formal risk-of-bias assessment given that the review is narrative in nature.

Results

Aerobic exercise

Aerobic activity has been shown to be strongly positive and robustly associated with a decrease in depressive symptoms in cross-validation of many cohorts. Walking, jogging, and cycling have been associated with improved mood, diminished anxiety and better psychological health [18]. Meta-analyses have reported medium but clinically meaningful effect sizes for aerobic therapies, thus, indicating their therapeutic value [16, 17]. Aerobic exercise has emerged in recent studies as a possible complement to standard care and may also represent a possibility for some patients with mild to moderate depression in which pharmacotherapy is an effective adjuvant [13, 16]. A crucial distinction regarding this approach is that the effects on some symptom clusters of exercise might be selective and not present across depressive profiles. A pooled analysis of six cohort studies completed by over 28,000 individuals showed especially strong associations between low physical activity and symptoms of anhedonia, fatigue, low mood, and appetite changes [5]. It implies that exercise may exert only moderate effects on motivation and somatic symptoms.

Resistance training

The benefits of resistance training and resistance exercise show clinically significant antidepressant characteristics. Structured progressive strength programs are associated with reductions in depressive symptoms and improvements in mental states [19, 25]. Proposed mechanisms: Increased self-efficacy, body image, behavioral activation due to measurable improvements in physical ability. For people who like their training shorter, more focused and more structured, resistance training is sometimes more accessible than an aerobic program. Meta-analytic evidence provides evidence to support integrating resistance exercise into multimodal, exercise-based approaches to depression treatment.

The intensity and dosing response of physical exercise

The antidepressant effects of physical activity seem to depend on intensity, frequency, and dose of exercise level. As mentioned above, moderate intensity physical activity has consistently led to stable psychological benefits, but even higher quantities decrease symptoms, especially in formerly inactive subjects [1, 13]. Several studies corroborate a dose-response relationship that suggests higher levels of activity are overall associated with reduced depressive burden [15, 16]. But overly intense exercise may provide little further psychological benefit at all, and could harm long-term adherence. Indeed, there is a large population of individuals with major depressive disorder who have improved symptoms in the presence of aerobic, resistance, combined, and mind–body interventions [4]. Importantly, exercise duration and antidepressant response is also U-shaped: both high and low exercise levels may be less effective [4]. Greater responses were observed at ≈860 MET-minutes/week and clinical benefits were noted at a lower weekly volume (~320–480 MET-minutes/week). These trends highlight the need to balance exercise prescriptions with maintaining engagement.

Antidepressants and sedentary behaviour prevention

Antidepressants and sedentary behaviour aside from therapeutic impacts, physical activity is a major preventative factor. Epidemiological evidence is consistent with lower rates of depression in an active population [1, 24, 9]. These findings provide recommendations to enable public health approaches to encourage physical activity to decrease population-level mental health burden. In contrast, sedentary life has become a standalone independent risk factor for depressive symptoms. In fact, among those participating in well-organized exercise protocols, prolonged periods of sitting and lack of movement are both linked to a higher risk of depression [11]. Thus, reduced total inactivity should be incorporated with the recommendation to promote formal exercise.

Comparative efficacy and individualized exercise prescription

Recent (large) meta-analyses show that exercise-based interventions might be as effective as psychotherapy or pharmacotherapy in selected populations, including patients with mild to moderate depression [2]. Aerobic, resistance, and mind-body strategies have all been found to be effective, but the outcome is likely determined by compliance, individual preference, and baseline patient phenotypes [7,8]. Evidence points to the need for tailoring exercise prescriptions to individual patients' symptoms, age, baseline fitness and biological heterogeneity in response to treatment [7], as well as treatment variability. Precision approaches in exercise psychiatry may improve the efficacy of therapies by adapting individual interventions to characteristics specific to the patient.

Mechanisms contributing to activity-induced antidepressant effects of physical activity

The exercise effect of physical activity is mediated by different neurobiological, psychological, and behavioral mechanisms. Depression's pathophysiology includes a heterogeneous set of systems under the influence of several modulating factors through exercise: monoaminergic neurotransmission, neuroplasticity, HPA-axis, inflammatory dynamics, sleep, and psychosocial factors. The extent of these influences may help to explain the diversity in the phenotypes of depressive symptoms when physical activity is used.

Regulation of neurotransmitters by the neurotransmitter systems

Exercise directly affects serotonin, dopamine, and norepinephrine systems—core targets of traditional antidepressant remedies [10, 22]. Especially dopaminergic modulation is relevant for reward and motivation and plays a role for the relief of symptoms, such as anhedonia and fatigue, that seem to be particularly exercise responsive [5, 30]. Acute exercise leads to increased endorphin release and consequently temporary mood boost, although sustained antidepressant effects are probably mediated by more general neurobiological and psychosocial changes over and above transient endorphin peak [31].

Neuroplasticity - the role of Brain-derived Neurotrophic Factor (BDNF)

Dysregulation of neural circuits, especially with respect to hippocampal networks, is central to depression. Brain-derived neurotrophic factor (BDNF) is a master molecule contributing to synaptic plasticity, neuronal survival, and neurogenesis. Exercise increases BDNF expression and can reverse stress-associated neural disorder and enhance emotionality and cognition [20, 27, 32]. The neurotrophic hypothesis, supported by decreased BDNF signaling, explains depressive symptoms, while therapeutic interventions aimed at upregulation of BDNF, like exercise and antidepressant treatments, are the mainstay of therapeutic strategies [28, 29]. Other emerging data also suggest that genetic moderators (such as the BDNF Val66Met polymorphism) affect response to exercise, pointing towards the development of exercise-based personalized treatments [27].

Hypothalamic–pituitary–adrenal axis regulation

HPA axis dysregulation is a well-established biological aspect of depression. Persistently high stress elevates cortisol levels, leads to impaired coping mechanisms with depression, and induces neurobiological alterations associated with depressive experiences. Regular physical activity seems to enhance regulation of HPA-axis and resilience to stress. While episodes in acute exercise may temporarily increase cortisol elevation, regular physical exercise will help to normalize basal cortisol level and potentiate adaptive stress responses and may dampen an underlying biological disorder underlying depression [10, 33].

Aerobic-Exercise Protocols

The effective use of aerobic exercise in psychiatric care demands meticulous control of the F.I.T.T. parameters. Although moderate-intensity continuous training (MICT) has long been the normative protocol, HIIT is the approach with recent attention that provides enhanced cardiovascular fitness (VO_2 max). The recommended aerobic activity consists of 3–5 sessions per week at ~64–76% HRmax for 30–45 minutes per session, according to evidence. This intensity and duration can potentiate central neurotrophic activation whilst avoiding unfavorable physiological events that may jeopardize adherence [4].

Inflammatory processes

Low-grade chronic inflammation has been found to be increasingly linked with depression that increases pro-inflammatory cytokines like IL-6 and TNF-α in patients [8, 10]. In general, physical activity can have anti-inflammatory actions, such as changes in cytokine response, regulation of the immune response, and decreases in visceral adiposity. These processes could be underlying the anti-depression effects of exercise in the inflammatory model of depression [30].

Sleep - circadian regulation

Insomnia is prevalent with depression and its effects may worsen symptom intensity. Exercise may contribute to better sleep quality, increased sleep efficiency, circadian rhythm entrainment and enhanced nocturnal restorative processes, which can reduce depressive symptoms. Higher quality of sleep helps improve mood regulation and cognitive functioning and is a positive factor contributing to recovery [15, 33].

Psychological and behavioral mechanisms

Aside from a biological impact, exercise influences psychological and behavioral variables related to depression. Physical activity may enhance self-efficacy, raise self-esteem, and give a sense of achievement that counteracts negativity as a cognitive process. Group-based or socially inclined activities may reduce loneliness and strengthen social support. Furthermore, exercise acts as a method of behavioral activation—an evidence-based treatment approach—by training a person in highly structured, goal-directed activities. These psychosocial pathways probably interact with neurobiological systems to help the antidepressant effects of physical activity.

Integrated perspective

The antidepressant effects of physical activity may be multifactorial and cannot be accounted for by a single biological or psychological pathway. Instead, exercise seems to have synergistic effects on the neurobiological, behavioral, and social aspects simultaneously. This multidimensional process might be a unique facet of the benefit of physical activity as an intervention therapy, unlike interventions for specific neurochemical systems in isolation [30].

Table 1. Classification of Exercise Modalities, Prescriptive Variables (FITT), and Hypothesized Neurobiological and Clinical Outcomes in Depression Management.

Exercise Type	Recommended Protocol (FITT)	Primary Biological Mechanism	Impact on Depressive Symptoms
Aerobic (MICT)	3–4x per week, 30–45 min, 60–70% HRmax	HPA axis regulation, upregulation of hippocampal BDNF	Reduction of anxiety, mood enhancement, mitigation of somatic symptoms
Anaerobic (HIIT)	2x per week, intervals (e.g., 4x4 min at 85–95% HRmax)	Acute endorphin release, accelerated neuroplasticity	Rapid, short-term improvement in cognitive functions
Resistance Training	2–3x per week, 3 sets, 8–12 repetitions, 70% 1RM	Enhanced self-efficacy, muscle protein synthesis and myokine release	Significant reduction in anhedonia, improved sleep quality
Mind-Body (Yoga)	2–5x per week, 60 min, low intensity + breathwork	Vagus nerve stimulation (increased parasympathetic tone)	Reduction of rumination (intrusive thoughts), psychological stabilization

Note: MET = metabolic equivalent of task; HRmax = maximum heart rate; 1RM = one-repetition maximum; HPA = hypothalamic–pituitary–adrenal; BDNF = brain-derived neurotrophic factor.

Synthesis and clinical application

Table 1 indicates that there is a necessity to discard a one-size-fits-all exercise model for psychiatric practice. Rather than consider one method or another of physical activity to be a standardized treatment approach, suitable measures should be adapted to the corresponding patient phenotypic profile. For instance, it is suggested that severe vegetative symptoms (marked anhedonia and psychomotor retardation) may benefit more from programs of structured resistance or higher-intensity training that directly impact mesolimbic dopaminergic circuits and encourage self-efficacy. On the other hand, patients who have comorbid anxiety and compulsive rumination may respond better to mind-body approaches, or to the predictably interoceptive activity derived from MICT, that can increase parasympathetic tone.

The synergistic and dose-response continuum

Network meta-analytic work [4] suggests that while different exercise protocols may impact different physiologic pathways, adjunctive or synergistic treatments yield better results. The U-shaped dose response in the literature implies that therapeutic benefit does not need to be extreme; regular moderate-volume activity, for the most part, is most effective. Applying treatment at low volumes (~320 MET-minutes/week) can result in clinically useful treatment-related symptom change, specifically in patients who were sedentary and early dropout is more likely in the presence of motivational deficiencies related to depression.

Methodological problems in exercise psychiatry

The multiplicity of mechanisms involved in exercise responses might explain why a single biomarker of response has not been identified. The benefits depend on peripheral–central cross-talk (in the form of myokine release from skeletal muscle modulating central BDNF expression) and should be evaluated in conjunction. For precision exercise psychiatry, further studies should look at baseline biological markers—notably inflammatory cytokine signatures or genetic variants such as BDNF Val66Met—to predict which FITT parameters are the most likely to result in remission for individual patients [10, 27].

Future directions: Multimodal precision exercise psychiatry

The future field of depression care is towards multimodal precision exercise psychiatry. It combines neuroimaging, circulating "exerkines" and machine learning to tailor interventions. For example, preliminary fMRI studies show that the baseline amygdala connection can predict remission following aerobic exercise training (AUC = 0.93 in one exploratory report) [36]. These neural measures alongside longitudinal wearable-derived behavioral data examined through machine learning could pave the way for personalized, dynamic intervention. Identification of intensity-responsive exerkines (e.g., Lac-Phe) and the moderating role of BDNF Val66Met also provide support for classifying patients into genotype- or biomarker-guided treatment pathways resulting in a change between a generalised and a more data-based, individualized approach to dosing and modality selection [37].

Discussion

In this narrative overview, the evidence base between physical activity and improvements in depressive disorders is summarized. In general terms, the results indicate routine physical activity is linked to clinically meaningful symptom reduction in a variety of populations and exercise modalities among people in a variety of populations. Ongoing reviews and network meta-analyses confirm exercise can be added to the evidence-based approach to the treatment of depression [2,6]. The probability of response to treatment seems to depend on adherence, patient preference and sustained treatment adherence, rather than on a routine regimen [26].

However, there are various experimental settings, the heterogeneity in studies restrict the strength of the inference. Diversity in type of exercise, duration of intervention, participants type and outcome parameters limits direct comparisons and creation of generalised exercise guidelines. Blinding an exercise trial is challenging in and of itself, and confounding factors, particularly the contribution of expectancy and placebo effects.

A great number of the studies available have small samples and few follow-ups, which is important in the long-term follow-up and assessment of the strength of an antidepressant [12, 14, 21]. Practicing adherence was a prominent clinical challenge at this point with core depressive symptoms of fatigue, lack of motivation and anhedonia disrupting adherence. This emphasis underscores the importance of personalized, long-term exercise protocols backed by behavioral and motivational interventions. In the field of public health, increasing population-level physical exercise and reducing sedentary time could directly reduce depression burden.

Epidemiological studies show a strong correlation between more activity and lower levels of depressive disorders, or between lack of physical activity and poor mental health in prolonged periods, or periods in a lifetime [1,11]. Small increases in physical activity by sedentary individuals can yield clinically significant gains. Increasing research in precision exercise psychiatry aims to individualize treatment based on symptoms and biological profiles which might include biomarkers, including BDNF, inflammatory cytokines and stress-related physiological indices, to help predict treatment response. This is going to require large, well-controlled randomized, widely spread exercise prescriptions and long-follow-up follow-ups that measure optimal dose, modality and intensity across subgroups of patients [35]. These include the narrative design of the review, the absence of a systematic search and formal bias assessment, and heterogeneity in the included studies that potentially influences interpretation.

Conclusions

An accumulating body of literature suggests that physical activity is a worthwhile intervention for amelioration of depressive symptoms. Both aerobic and resistance training have shown clinically relevant antidepressant effects and work as adjuncts to standard treatment. Presumably, benefits may derive from a biologic- and psychosocial-based combination - of neurotransmitter, HPA-axis, anti-inflammatory effects and neuroplastic effects - or, on the contrary, exercise may have differential functions on symptom clusters such as anhedonia, tiredness and low mood. Regular exercise also seems to provide protection against early depression, thus indicating that it is protective as well in terms of the prevention of its presence. Clinically, exercise is also inexpensive, widely applicable, widely available, large scale and amenable, and possible to adopt in preventive and treatment programmes. However, more detailed high-quality randomized trials are needed to determine optimal treatment regimens, level, intensity, dose and duration. Further research should also look into the possibility of applying personalized exercise prescriptions, taking into account persons' symptom patterns and biological variability, to maximize therapeutic benefit.

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