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PHYSICAL ACTIVITY AND ANXIETY DISORDERS: NEUROBIOLOGICAL MECHANISMS AND THERAPEUTIC EVIDENCE — NARRATIVE REVIEW

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ABSTRACT

Introduction: Anxiety disorders affect approximately 7.3% of the global population and remain undertreated. This narrative review synthesizes evidence on neurobiological mechanisms and therapeutic efficacy of physical activity (PA) in GAD, panic disorder, and PTSD.

Materials and Methods: A narrative review was conducted using PubMed, Scopus, and Google Scholar (2005–2026). Boolean strategies: ("physical activity" OR "exercise") AND ("anxiety disorder" OR "GAD" OR "panic disorder" OR "PTSD"); AND ("BDNF" OR "endocannabinoids" OR "neuroplasticity"); AND ("HPA axis" OR "cortisol" OR "serotonin") AND ("anxiety" OR "stress").

Results: Regular PA exerts anxiolytic effects through BDNF upregulation, HPA axis normalization, endocannabinoid system activation, and serotonergic adaptation. Aerobic exercise reduces anxiety symptoms (ES 0.34–0.66). Resistance training achieves GAD remission (NNT=3). HIIT produces rapid symptom reduction. Aerobic exercise attenuates CCK-4-induced panic attacks. Yoga demonstrates the greatest cortisol reduction among exercise modalities.

Conclusions: PA is an effective adjunct intervention for anxiety disorders, supporting integration into clinical protocols alongside pharmacotherapy and psychotherapy. Exercise prescription should consider modality, intensity, and patient characteristics.

KEYWORDS

Anxiety Disorders, Physical Activity, Exercise Therapy, Neurobiological Mechanisms, BDNF, Endocannabinoids, HPA Axis, Panic Disorder, PTSD, GAD

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1. Introduction

Anxiety disorders are, a public health problem of considerable scale. A cross-sectional WHO analysis of 147,261 adults from 26 countries placed the lifetime prevalence of DSM-5 GAD at 3.7%, with 12-month prevalence of 1.8% (Ruscio et al., 2017). When the lens widens to include all anxiety disorder subtypes, a meta-regression of 87 studies across 44 countries arrives at a global current prevalence of 7.3%, with substantial variation between regions — 5.3% in African cultures versus 10.4% in Euro/Anglo cultures (Baxter et al., 2013). Functional impairment is substantial: roughly half of individuals with 12-month GAD report severe disability in daily role performance (Ruscio et al., 2017). Epidemiological data further suggest that prevalence has been rising steadily in Western countries throughout the 21st century, a trend that correlates with increasing sedentary behavior and urbanization (Bandelow & Michaelis, 2015).

Treatment options are well-established on paper: SSRIs and benzodiazepines on the pharmacological side, cognitive-behavioral therapy (CBT) as the psychotherapeutic backbone. In practice, however, the picture is messier. Response rates for first-line pharmacotherapy in GAD hover around 40–60%, and a substantial portion of patients discontinue treatment due to side effects — weight gain, sexual dysfunction, dependency risk. This gap between guideline-recommended care and real-world outcomes has prompted growing interest in adjunctive strategies, and physical activity has emerged as one of the more consistently supported among them (Jakubowska et al., 2024; Woźniak et al., 2024; Myśliwiec et al., 2025; Wachowska et al., 2025; Romaniuk et al., 2025; Kurek et al., 2026).

WHO defines physical activity as any bodily movement produced by skeletal muscles that requires energy expenditure — a category that extends well beyond the gym to include walking, cycling, and recreational sport (Bull et al., 2020). Adults are recommended to accumulate at least 150–300 minutes of moderate-intensity aerobic activity per week, or 75–150 minutes at vigorous intensity, complemented by muscle-strengthening work. One-third of Europeans fall short, and activity rates continue declining among adolescents and working-age adults (Myśliwiec et al., 2025).

Why exercise works for anxiety is not a single-mechanism story. The pathways are multiple and, importantly, complementary: HPA axis regulation, serotonergic and dopaminergic modulation, upregulation of brain-derived neurotrophic factor (BDNF), endocannabinoid system activation, and broader psychosocial gains — improved self-efficacy, sleep quality, social connectedness — all appear to contribute (Myśliwiec et al., 2025; Li et al., 2025). This review attempts to map that evidence.

The aim of this narrative review is to synthesize the current evidence on four questions: which neurobiological mechanisms explain the anxiolytic effect of exercise; what the clinical data show for GAD, panic disorder, and PTSD specifically; how that evidence translates into prescription practice; and where the field's most pressing gaps remain.

2. Methodology

A narrative review was conducted in accordance with the methodological principles applicable to this study type. A comprehensive literature search was performed across PubMed, Scopus, and Google Scholar, covering publications from 2005 to 2026. The search was additionally supported by AI-assisted literature screening (Claude, Anthropic), which was used to identify relevant studies across the included source pool; all retrieved content was subsequently verified by the authors against primary sources. The following Boolean search strategies were applied: ("physical activity" OR "exercise") AND ("anxiety disorder" OR "generalized anxiety disorder" OR "panic disorder" OR "PTSD"); ("physical activity" OR "exercise") AND ("BDNF" OR "neuroplasticity" OR "endocannabinoids" OR "runner's high"); ("exercise" OR "physical activity") AND ("HPA axis" OR "cortisol" OR "serotonin") AND ("anxiety" OR "stress"). Both clinical studies (randomized controlled trials, cohort studies) and preclinical investigations were considered. Priority was given to systematic reviews, meta-analyses, and randomized controlled trials (RCTs).

Inclusion criteria consisted of: studies examining the relationship between physical activity or structured exercise and anxiety-related outcomes; studies involving human participants or, for mechanistic sections, animal models where the mechanistic rationale was supported by human data; English-language publications. Exclusion criteria included: studies with exclusively pediatric populations (under 18 years), non-peer-reviewed sources, and studies lacking a defined exercise intervention or validated anxiety outcome measure. The final pool consisted of 28 primary sources covering epidemiology, neurobiological mechanisms, and clinical evidence across GAD, panic disorder, and PTSD. No formal quality assessment or meta-analysis was performed due to the narrative nature of the review.

3. Results

3.1. Neurobiological mechanisms linking physical activity to anxiety reduction

3.1.1. Brain-Derived Neurotrophic Factor (BDNF) and neuroplasticity

BDNF is a neurotrophic protein that essentially keeps the brain plastic — capable of reorganizing, forming new connections, and recovering from stress. Aerobic exercise is among the most potent stimuli for BDNF secretion, supporting neurogenesis and synaptic strengthening in ways that translate into measurable gains in cognitive resilience (Myśliwiec et al., 2025). Reduced BDNF levels have been documented in anxiety disorders, including panic disorder (Ströhle et al., 2010).

A meta-analysis of 29 RCTs pooling 910 participants found that exercise training significantly elevated resting peripheral BDNF (SMD = 0.39, 95% CI: 0.17–0.60, $p < 0.001$) (Dinoff et al., 2016). The effect was driven almost entirely by aerobic modalities: SMD = 0.66 (95% CI: 0.33–0.99, $p < 0.001$) for aerobic exercise, versus SMD = 0.07 (95% CI: –0.15–0.30, $p = 0.52$) for resistance training — the latter statistically indistinguishable from zero. Notably, the BDNF response to exercise is not purely a chronic adaptation. Liang et al. (2023), in a meta-analysis of 8 RCTs, showed that a single endurance bout suffices to significantly raise BDNF (SMD = 0.30, 95% CI: 0.08–0.52, $p = 0.008$), with serum proving more sensitive than plasma as a readout. The effect held across age groups.

What makes the Ströhle et al. (2010) study particularly interesting is its selectivity: acute aerobic exercise (30 min at 70% VO₂max) raised BDNF in panic disorder patients but not in healthy controls. The implication for practice is worth naming directly: CBT for anxiety works through extinction learning, a process that depends on BDNF-mediated plasticity in the prefrontal cortex and hippocampus. If exercise reliably boosts BDNF in this patient group, combining exercise with exposure-based therapy might make neurobiological sense, not just additive clinical sense.

3.1.2. The endocannabinoid system and the runner's high

The runner's high — euphoria, reduced anxiety, hypoalgesia, and a particular sedative quality familiar to endurance athletes — was for decades attributed to endorphins. This turned out to be physiologically implausible: endorphins are hydrophilic and cannot cross the blood-brain barrier, and pharmacological opioid blockade leaves the subjective experience of exercise intact (Siebers et al., 2022). The endocannabinoid hypothesis offers a more credible explanation. The eCB system comprises CB1R and CB2R receptors and lipid ligands — anandamide (AEA) and 2-arachidonoylglycerol (2-AG). eCBs are lipophilic and cross the blood-brain barrier readily, and CB1 receptors are densely expressed in precisely the limbic regions implicated in anxiety and fear regulation (Maldonado et al., 2020).

Siebers et al. (2021) put the opioid hypothesis to a direct test: in a double-blind, placebo-controlled RCT of 63 participants, naltrexone was administered before 45 minutes of moderate treadmill running. The runner's high persisted regardless of opioid blockade — and plasma AEA and 2-AG rose in both conditions. This is about as clean a dissociation as exercise neuroscience offers. A subsequent review concluded that AEA, acting via CB1 receptors in the prefrontal cortex, amygdala, and hippocampus, is the most plausible substrate for exercise-induced anxiolysis (Siebers et al., 2022). One important caveat: not all exercise produces eCB release. Moderate-intensity endurance work appears most reliably effective.

3.1.3. The HPA axis and cortisol regulation

The hypothalamic-pituitary-adrenal (HPA) axis governs the body's physiological stress response. Chronic dysregulation — manifesting as elevated cortisol, blunted diurnal variation, and impaired negative feedback — is implicated in the pathophysiology of anxiety disorders. Exercise has been shown to normalize HPA axis activity, contributing to its protective and therapeutic effects (Myśliwiec et al., 2025).

A network meta-analysis of 44 RCTs encompassing 3,284 participants provides the most comprehensive picture available (Li et al., 2025). Five modalities were compared head-to-head. Yoga came out ahead on cortisol reduction (SMD = -0.59; 95% CI = -0.90 to -0.28; SUCRA = 93%), followed by qigong and multicomponent protocols. HIIT trended toward cortisol elevation — a counterintuitive finding, though wide confidence intervals warrant caution in interpretation. The dose-response relationship was non-linear: an inverted U-shape with the peak effect at roughly 530 MET-min/week, corresponding to 90–150 minutes of moderate exercise weekly. Above this threshold, additional volume does not appear to yield proportional benefit. Effects were largest for stress outcomes (SMD = 0.90), followed by anxiety (SMD = 0.74) and depression (SMD = 0.54). Intervention duration predicted outcome: each additional week of training corresponded to incrementally greater cortisol reduction ($\beta = -0.03$, $p = 0.04$).

3.1.4. Serotonergic, GABAergic, and autonomic mechanisms

The neurochemical abnormalities underlying anxiety disorders are well-mapped in vivo. Imaging data from 504 patients across anxiety disorder subtypes show reduced SERT binding (-13%), lower 5-HT1A receptor density (-18%), and frontocortical and temporocortical GABAA deficits (-13% in panic disorder, -16% in GAD) (Nikolaus et al., 2010). These targets overlap substantially with what exercise appears to modulate. In rodent models, voluntary wheel running produced time-dependent changes in serotonin transporter mRNA and 5-HT1A/1B receptor expression in the raphe nuclei — a pattern consistent with reduced serotonergic stress reactivity over weeks of training (Greenwood et al., 2005). Direct translation to humans requires caution given the preclinical source, but the directional consistency with clinical observations is notable. Beyond serotonin, regular PA stimulates dopamine and norepinephrine release (Myśliwiec et al., 2025), while yoga specifically appears to augment GABA tone via parasympathetic mechanisms (Li et al., 2025). Autonomic effects — improved heart rate variability, lower resting heart rate, reduced sympathetic dominance — likely compound these central adaptations.

Table 1. Neurobiological mechanisms linking physical activity to anxiolytic effects.

Mechanism	Target System	Effect on Anxiety	Key Evidence
BDNF upregulation	Hippocampus, PFC	Neuroplasticity; facilitates fear extinction; corrects BDNF deficit in PD	Aerobic: SMD = 0.66 (CI 0.33–0.99) for resting BDNF (Dinoff et al., 2016); single-bout: SMD = 0.30 (Liang et al., 2023)
HPA axis normalization	Hypothalamus, pituitary, adrenal cortex	Reduced basal cortisol; restored diurnal rhythm; blunted stress reactivity	Yoga SMD = –0.59, SUCRA 93%; optimal dose ~530 MET-min/week (Li et al., 2025)
Endocannabinoid activation	Amygdala, PFC, hippocampus (CB1R)	Anxiolysis, euphoria; eCBs — not opioids — mediate runner's high	Naltrexone RCT N=63: AEA/2-AG elevated regardless of opioid blockade (Siebers et al., 2021)
Serotonergic adaptation	Dorsal and median raphe nuclei	↓ SERT mRNA; ↑ 5-HT1A sensitivity; lower serotonergic stress reactivity	3 weeks: ↓ SERT mRNA; 6 weeks: ↑ 5-HT1A mRNA (Greenwood et al., 2005)
GABAergic modulation	Frontocortical and temporocortical regions	Increased GABA tone; reduced hyperarousal; anxiolysis	Yoga enhances GABA via parasympathetic activation (Li et al., 2025; Nikolaus et al., 2010)
Autonomic rebalancing	ANS: sympathetic/parasympathetic	↑ Heart rate variability; ↓ resting HR; reduced anxiety sensitivity	Consistent across modalities (Li et al., 2025; Myśliwiec et al., 2025)

Abbreviations: BDNF — brain-derived neurotrophic factor; PFC — prefrontal cortex; HPA — hypothalamic-pituitary-adrenal; eCB — endocannabinoid; AEA — anandamide; CB1R — cannabinoid receptor type 1; SERT — serotonin transporter; ANS — autonomic nervous system; SMD — standardized mean difference; SUCRA — surface under the cumulative ranking curve; PD — panic disorder; HR — heart rate; HRV — heart rate variability.

3.2. Clinical evidence: exercise in specific anxiety disorders

3.2.1. Generalized Anxiety Disorder

GAD presents a particular clinical challenge — the worry is pervasive, not event-triggered, and the somatic dimension (muscle tension, fatigue, disrupted sleep) is often as debilitating as the cognitive one. Comorbidity with depression and other anxiety disorders is the rule rather than the exception. Against this background, a systematic review by Aylett & Small (2018) found aerobic exercise effective versus waitlist control (ES = –0.41, 95% CI: –0.70 to –0.12), with higher-intensity programs outperforming lower-intensity ones. This is an encouraging starting point.

The headline finding from Herring et al. (2012) is worth dwelling on. In an RCT of 30 sedentary women with clinician-diagnosed GAD, six weeks of resistance training produced a remission rate of 60% — versus 30% for waitlist (NNT = 3, 95% CI 2–56). Aerobic training reached 40%. The finding that resistance work outperformed aerobic exercise runs counter to the intuitive assumption that cardio is the primary vehicle for anxiolytic benefit. The same group followed up on sleep (Herring et al., 2015): both modalities reduced sleep onset latency and time in bed relative to waitlist, with resistance training producing the larger weekend gains. The correlation between GAD severity improvement and sleep efficiency in the RET group raises the interesting possibility that some of the anxiolytic effect is mediated through sleep — a question that remains open.

Plag et al. (2020) asked whether intensity matters — and the answer appears to be yes. In 33 GAD patients randomized to 12 days of HIIT or lower-intensity training, both groups improved on PSWQ, HAM-A, and depression measures, but HIIT effects were roughly twice the magnitude. What's particularly interesting is the mechanism: changes in perceived control over anxiety-related stimuli predicted worry reduction in the HIIT group but not in the LIT group, suggesting the high-intensity condition activated a psychological pathway — perhaps mastery or self-efficacy — that lower loads do not reliably engage. Twelve days to significant symptom change is also noteworthy for clinical contexts where rapid response matters. Ewuzie et al. (2024) provide a useful integrative summary: across aerobic exercise, resistance training, Pilates, and walking, anxiolytic effects are consistently present, mediated by overlapping but distinct neurobiological routes.

3.2.2. Panic Disorder

Panic disorder presents a particular irony for exercise-based treatment: the physiological sensations of moderate-to-vigorous exercise — racing heart, shortness of breath, sweating — overlap substantially with the somatic signature of a panic attack. This is actually the basis for one of the more intriguing therapeutic rationales. Exercise, deliberately used to produce these sensations in a controlled and safe context, resembles interoceptive exposure — a technique already incorporated into established CBT protocols for PD. Beyond this psychological framing, the anxiolytic and anti-panic properties of aerobic training and the BDNF-correcting effects documented in this patient group (Ströhle et al., 2010) provide additional mechanistic support.

Ströhle (2009) reviewed the evidence and noted that while acute exercise may transiently worsen somatic symptoms in PD patients — again, consistent with interoceptive triggering — regular training produces clear anti-panic effects. The most direct evidence comes from a crossover RCT where 24 subjects underwent a CCK-4 panic challenge both following exercise (30 min at 70% VO₂max) and following rest (Ströhle et al., 2009b). The results were striking: panic attacks occurred in 5 of 24 subjects post-exercise (20.8%) versus 15 of 24 post-rest (62.5%). API anxiety subscores were significantly lower after exercise. Crucially, somatic symptoms were elevated during exercise — patients did feel the physical sensations — but cognitive anxiety was not, providing a clean empirical dissociation that mirrors the theoretical logic of interoceptive exposure. Chronic effects were examined in a pilot RCT of 10 PD patients, where 12 sessions of aerobic training produced significant reductions in BAI and BDI-II scores accompanied by EEG frontal asymmetry changes suggestive of underlying neurophysiological reorganization (Lattari et al., 2018).

3.2.3. Post-Traumatic Stress Disorder

PTSD sits somewhat apart from GAD and panic disorder in its treatment landscape. The first-line therapies — prolonged exposure, EMDR — are well-validated but demanding. Response rates run at 50–60%, and dropout is a persistent problem; many patients cannot sustain the distress that evidence-based exposure protocols require. Physical activity enters this picture through several distinct channels, and the rationale is stronger than it might initially appear.

The network meta-analysis by Li et al. (2025) gives yoga the strongest effect on stress regulation across five modalities (SMD = 0.90, SUCRA = 93%) — a result that aligns intuitively with PTSD's HPA-axis dysregulation and chronic hyperarousal profile. Yoga's particular value here may lie in what it does not require: recounting traumatic events, direct confrontation with feared stimuli, or performance of any kind. Parasympathetic activation, breath-body coupling, and the predictability of a structured movement practice offer a lower-threshold entry point for a population where avoidance is a defining symptom. Aerobic modalities offer complementary mechanistic value: BDNF upregulation and endocannabinoid release both facilitate fear extinction in preclinical models (Maldonado et al., 2020), raising the hypothesis that exercise timed around exposure therapy sessions could augment consolidation of extinction memory. Whether this translates to superior clinical outcomes remains to be tested directly.

Table 2. Summary of clinical evidence for exercise interventions across anxiety disorder subtypes.

Disorder	Modality	Key Finding	Outcome	Reference
GAD	Aerobic	Effective vs. waitlist	ES = -0.41 (CI -0.70 to -0.12)	Aylett & Small (2018)
GAD	Resistance (RET)	60% remission vs. 30% waitlist	NNT = 3	Herring et al. (2012)
GAD	RET + AET	Both reduced sleep onset latency and time in bed vs. waitlist	↑ Sleep efficiency (weekend)	Herring et al. (2015)
GAD	HIIT	Effects ~2× lower-intensity; onset within 12 days	HAM-A, PSWQ — large effects	Plag et al. (2020)
Panic disorder	Aerobic (acute)	CCK-4 panic: 20.8% post-exercise vs. 62.5% post-rest	↓ API score and anxiety subscore	Ströhle et al. (2009b)
Panic disorder	Aerobic (chronic)	↓ BAI and BDI-II after 12 sessions; EEG changes	Neurophysiological improvement	Lattari et al. (2018)
PTSD	Yoga/qigong	Greatest stress regulation in network meta-analysis	SMD = 0.90, SUCRA 93%	Li et al. (2025)

Abbreviations: GAD — generalized anxiety disorder; PTSD — post-traumatic stress disorder; RET — resistance exercise training; AET — aerobic exercise training; HIIT — high-intensity interval training; CCK-4 — cholecystikinin tetrapeptide; API — Acute Panic Inventory; BAI — Beck Anxiety Inventory; BDI-II — Beck Depression Inventory-II; HAM-A — Hamilton Anxiety Rating Scale; PSWQ — Penn State Worry Questionnaire; NNT — number needed to treat; ES — effect size; SMD — standardized mean difference.

3.3. Epidemiological perspectives and prevention

The prevention angle is worth considering separately from treatment. Higher physical activity levels are associated with lower odds of developing anxiety disorders, and multiple reviews confirm that even modest activity gains yield mental health returns (Jakubowska et al., 2024; Woźniak et al., 2024; Wachowska et al., 2025; Romaniuk et al., 2025; Kurek et al., 2026). Set against a background where anxiety disorders affect 7.3% of the population currently and lifetime rates approach 30% (Ruscio et al., 2017; Baxter et al., 2013), and where GAD is more prevalent precisely in high-income, high-sedentary societies (Bandelow & Michaelis, 2015), the population-level arithmetic becomes relevant: one-third of Europeans fail WHO activity targets, and more than 45% of girls fall below recommended thresholds (Myśliwiec et al., 2025). Whether this inactivity causally contributes to anxiety disorder prevalence or simply co-occurs with other risk factors is not fully resolved — but as a modifiable exposure, it represents an accessible point of intervention.

4. Discussion

Taken together, the evidence reviewed here makes a reasonably consistent case for physical activity as an adjunct intervention for anxiety disorders. The mechanisms are numerous enough that non-specific effects alone are an implausible explanation: BDNF plasticity, eCB release, HPA normalization, serotonergic adaptation, GABAergic upregulation, autonomic rebalancing — these are independent pathways, each with its own evidence base, each pointing in the same clinical direction.

Across disorder subtypes, the picture looks different in texture but consistent in direction. For GAD, NNT = 3 for resistance training (Herring et al., 2012) is a number that would be competitive with many pharmacological comparators, and HIIT adds the option of rapid onset within 12 days (Plag et al., 2020) — useful when the clinical situation calls for speed. For panic disorder, the CCK-4 data (Ströhle et al., 2009b) demonstrate acute anti-panic effects with a magnitude that is hard to dismiss, and the interoceptive exposure framework gives exercise a specific therapeutic role within the CBT structure rather than as a generic add-on. PTSD presents differently again: the strongest effect is from mind-body practices on stress regulation (Li et al., 2025), but the mechanistic rationale for pairing aerobic exercise with exposure sessions — via BDNF and eCB pathways relevant to extinction consolidation (Maldonado et al., 2020) — has sufficient biological plausibility to warrant direct clinical testing.

Effect sizes for aerobic exercise across meta-analyses of anxiety outcomes range from approximately 0.34 to 0.66 (Wegner et al., 2014) — modest to moderate by conventional benchmarks, but comparable to what is reported for many pharmacological comparators at equivalent severity levels. The comparison is imperfect, but it is worth making. Exercise also carries advantages that pharmacotherapy does not: no dependence risk, no withdrawal syndrome, and a side-effect profile that is almost entirely positive — cardiovascular fitness, metabolic regulation, bone density, improvements in sleep. These are not trivial considerations for a patient population whose physical health often deteriorates in the context of chronic psychiatric illness. The mental health benefits extend beyond anxiety as well, with consistent evidence across depression, schizophrenia, ADHD, and substance use disorders (Jakubowska et al., 2024; Woźniak et al., 2024; Myśliwiec et al., 2025; Wachowska et al., 2025; Romaniuk et al., 2025; Kurek et al., 2026).

One point worth emphasizing for clinical practice: the dose-response data suggest that more is not always better. Cortisol reduction peaks at around 530 MET-min/week — roughly 90–150 minutes of moderate exercise — and additional volume does not appear to proportionally add benefit (Li et al., 2025). This threshold sits comfortably within WHO recommendations (Bull et al., 2020) and is achievable without specialist facilities or high baseline fitness. For a patient group where motivation and adherence are already barriers, this is a clinically useful message.

5. Practical Considerations for Exercise Prescription

The evidence reviewed permits some specific prescriptive conclusions — though it is worth stating clearly that the RCT base is relatively small, and recommendations should be held proportionally to that caveat.

For GAD, resistance and aerobic training are both defensible first-line choices. The Herring et al. (2012) data give a slight edge to resistance training on remission (NNT = 3), but both modalities improve core GAD symptoms. When speed of response is a clinical priority, HIIT offers demonstrably faster onset — significant improvement within 12 days (Plag et al., 2020) — and may also engage a psychological mechanism (improved perceived control) that lower-intensity protocols do not reliably activate. Practically: a minimum of three sessions per week for at least four weeks appears to be the threshold for consistent efficacy in the reviewed literature.

For panic disorder, the prescription has an additional psychoeducational component that should not be skipped. Patients need to know before starting that their heart rate will climb, they may feel short of breath, and they may sweat — and that none of this is dangerous. Without that framing, the first moderate-intensity session risks becoming a self-confirming panic trigger and may end the intervention. With it, the same session functions as interoceptive exposure. The Ströhle et al. (2009b) data showing that 30 minutes at 70% VO₂max reduces CCK-4-induced panic attack rates from 62.5% to 20.8% also suggest a role for acute pre-session exercise as an adjunct to formal exposure therapy.

For PTSD, the data most strongly support yoga and qigong — both for their effect size on stress outcomes (Li et al., 2025) and for practical reasons specific to this population. These modalities involve no performance pressure, no need to recount experiences, and active attention to breath and body in a non-threatening frame. Trauma-informed instruction is not optional — it is a component of delivery that affects whether the intervention is safe. Aerobic exercise is a reasonable complementary prescription, with the specific rationale that BDNF and eCB pathways relevant to extinction learning (Maldonado et al., 2020) may be upregulated by moderate-intensity aerobic work. The timing question — whether to exercise before or after an exposure session — remains unanswered by current evidence.

Across all three subtypes, a basic clinical truth applies: the best exercise modality is the one the patient will actually do. Preference, accessibility, social format, and prior exercise experience are the variables that determine long-term adherence, and they should weigh at least as heavily as the modality-specific RCT data when making an initial recommendation. The dose ceiling of ~530 MET-min/week (Li et al., 2025) is a helpful anchor: it keeps the target achievable and aligned with existing public health guidance (Bull et al., 2020) without requiring patients to become athletes.

Table 3. Evidence-based exercise prescription recommendations for anxiety disorder subtypes.

Disorder	Recommended modality	Dose / frequency	Special considerations	Evidence quality
GAD	RET or aerobic (first-line); HIIT (augmentation)	≥150 min/week moderate; HIIT: ≥2×/week	RET may outperform AET; HIIT produces rapid effects within 12 days	Moderate–high (RCTs)
Panic disorder	Aerobic (moderate intensity)	30 min sessions, ≥3×/week	Educate patient about expected somatic symptoms; use as interoceptive exposure	Moderate (RCTs + crossover)
PTSD	Yoga / qigong (first-line); aerobic (adjunct)	~530 MET-min/week; trauma-sensitive pacing	Parasympathetic focus; avoid forced intensity; trauma-informed instruction	Moderate (network meta-analysis)
All anxiety disorders	Any modality matching patient preference	Minimum: 530 MET-min/week	Adherence > optimal modality; social formats improve compliance	Consistent across reviews

Abbreviations: GAD — generalized anxiety disorder; PTSD — post-traumatic stress disorder; RET — resistance exercise training; AET — aerobic exercise training; HIIT — high-intensity interval training; MET — metabolic equivalent of task; RCT — randomized controlled trial.

6. Conclusions

The case for physical activity as an adjunct intervention for anxiety disorders rests on converging evidence from multiple levels of analysis. Mechanistically, BDNF plasticity, endocannabinoid release, HPA axis normalization, serotonergic adaptation, and autonomic rebalancing are not competing explanations — they are complementary processes that may each contribute something. Clinically, effect sizes across GAD, panic disorder, and PTSD are meaningful, with resistance training achieving $NNT = 3$ in the best available GAD trial.

Exercise prescription belongs in the standard toolkit for anxiety disorder management — not as a replacement for established pharmacological or psychotherapeutic treatment, but as a structurally distinct adjunct with its own mechanistic rationale, minimal side effects, and physical health benefits that extend beyond psychiatry. Starting at WHO-recommended doses is a defensible and achievable target; HIIT can be layered in when rapid response is needed; yoga warrants preferential consideration in PTSD. The most pressing research gaps are long-term maintenance data, the optimal integration of exercise with exposure-based therapies, and tools for predicting who responds best to which modality.

Limitations

The limitations of this evidence base are worth stating plainly. Sample sizes are small across most clinical trials — the GAD RCTs reviewed enrolled 10–33 participants. Intervention durations rarely exceed 12 weeks, leaving questions about whether effects persist, attenuate, or build over time. Exercise protocols are sufficiently heterogeneous that direct cross-study comparison is treacherous, and few studies control adequately for social contact as a confound — a non-trivial issue when group exercise formats are compared against individual waitlist control. The peripheral biomarkers used to index central neurobiological change (plasma BDNF, circulating AEA) are reasonable proxies but imperfect ones. The rodent serotonin data (Greenwood et al., 2005) offer mechanistic plausibility, not human confirmation. And this review, being narrative in structure, necessarily reflects choices about which sources to include and how to weight them — choices that are not fully transparent and that should temper confidence in the conclusions drawn.

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