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The phenomenon of exercise addiction: diagnostic criteria, neurobiological underpinnings, and differential challenges at the interface of psychiatry and sports medicine

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Abstract

Background: Regular physical activity is broadly considered a cornerstone of somatic and psychological health, yet a subset of practitioners progresses toward a maladaptive pattern in which exercise acquires features resembling a behavioural addiction. This shift from a salutogenic behaviour to a compulsion sits at the interface of psychiatry and sports medicine and lacks a unified nosological position.
Aim: The review aims to synthesise contemporary evidence on the conceptualisation, diagnostic criteria, neurobiological underpinnings and differential diagnosis of exercise addiction (EA), with attention to its overlap with eating disorders, OCD and overtraining syndrome.
Material and methods: A structured search of PubMed, Scopus, Web of Science and Google Scholar covered publications from 1976 to 2025. Empirical studies, systematic reviews and meta-analyses on the symptomatology, epidemiology, neural substrates and treatment of EA were synthesised qualitatively.
Results: Around 3% of regular exercisers may be at risk of EA, with notably higher estimates among endurance athletes. Diagnostic models tend to converge on Griffiths' six components and are operationalised through the EDS-21 and the EAI. Neuroimaging findings suggest alterations in mesolimbic dopaminergic circuits, opioid and endocannabinoid signalling, and reduced grey matter in the orbitofrontal cortex and inferior frontal gyrus.
Conclusions: EA appears to constitute a clinically meaningful yet under-recognised behavioural syndrome whose identification requires careful distinction from eating disorders, OCD, overtraining and elite athletic commitment. Cognitive–behavioural interventions remain best-supported; pharmacotherapy is largely restricted to comorbid conditions.

Keywords: exercise addiction; behavioural addiction; eating disorders; overtraining syndrome; neuroimaging; differential diagnosis.

1. Introduction

1.1. Definition and conceptual framework: from health-promoting behaviour to compulsion. The paradox of the "healthy addiction"

Regular physical activity is widely recognised as one of the principal determinants of somatic, metabolic and mental health, and its importance is reflected in the recommendations of major public-health institutions across the lifespan [1]. A growing body of evidence demonstrates that habitual exercise reduces the risk of

cardiovascular disease, type 2 diabetes and several malignancies, while also serving as a non-pharmacological intervention for depressive disorders, anxiety, and sleep disturbances [2]. Within this broadly positive context, however, clinicians and exercise scientists have increasingly drawn attention to a paradoxical phenomenon: in a small but clinically relevant subgroup of individuals, the same behaviour that ordinarily promotes well-being appears to evolve into a maladaptive, dysregulated pattern that bears a striking resemblance to substance-related and other behavioural addictions [3]. This shift, in which physical activity ceases to function as a salutogenic resource and instead becomes an autonomous driver of distress, has been variously described in the literature as exercise addiction (EA), exercise dependence, obligatory exercise, compulsive exercise and exercise abuse, with each term carrying somewhat different theoretical connotations [3,4].

The terminological heterogeneity reflects an unresolved tension between two interpretative traditions. Glasser's early concept of "positive addiction" framed habitual running and similar activities as a benign counterweight to substance use, emphasising the favourable dose–response relationship between exercise and health [3]. Shortly thereafter, Morgan questioned this framing and presented case material in which exercise was pursued despite physical injury, occupational and relational deterioration and an inability to abstain — a constellation that, on closer inspection, recapitulated the core features of an addictive process and was therefore re-conceptualised as a "negative addiction" [3]. In the decades that followed, more uniform terminology emerged, with most authors converging on the view that the behaviour is best characterised as an addiction insofar as it incorporates both dependence and compulsion, and is sustained despite mounting personal costs [3,5].

Within contemporary models, EA is typically defined as a morbid pattern of behaviour in which a habitually exercising individual loses control over training habits, exhibits compulsive engagement and continues to exercise despite physical, psychological, social or occupational harm [5,6]. The clinical phenotype is consistent with the six common components of behavioural addiction outlined by Griffiths, namely salience, mood modification, tolerance, withdrawal symptoms, conflict and relapse [2,3,6]. In this respect, EA conceptually mirrors the framework that has been applied to gambling disorder, internet gaming disorder and other forms of non-substance-related dependence, although it differs in several respects, most notably in the socially valued nature of the underlying behaviour [2]. The paradox of the "healthy addiction" arises precisely from this mismatch: while substance-related disorders are anchored in a behaviour that is, in the cultural imagination, intrinsically harmful, EA emerges within an activity that is socially encouraged, professionally rewarded and frequently subsidised by public-health policy, which complicates both self-recognition and clinical detection [2,5].

A further conceptual distinction that has shaped the field concerns the differentiation between primary and secondary forms of the disorder, introduced by de Coverley Veale in 1987 [3]. Primary EA is understood as a behavioural addiction in its own right, in which the exercise itself constitutes the rewarding endpoint and the individual is motivated by the psychological gratification produced by training, by the temporary relief from intrapsychic tension, or by the avoidance of withdrawal-related dysphoria [3,5,6]. Secondary EA, in contrast, is conceptualised as a symptom of, or a compensatory strategy associated with, another psychopathological process, most often an eating disorder or a body-image disturbance, in which exaggerated exercise volume serves an instrumental purpose such as weight control or alteration of body composition [3,6]. Some authors have therefore proposed that the term "instrumental exercise" be preferred for the latter, in order to avoid conflating two etiologically distinct phenomena under a single label [6]. While the empirical separability of primary and secondary EA continues to be debated — with several authors arguing that virtually all problematic exercise is anchored in eating-related concerns [4,8] — the distinction retains substantial clinical utility and is reflected in most contemporary diagnostic schemes [5,6].

It should additionally be emphasised that EA is most plausibly conceptualised not as a discrete diagnostic entity but as a continuum, ranging from highly committed yet adaptive engagement to dysfunctional, compulsive practice [5]. Within this developmental pathway, exercise initially undertaken for pleasure or performance gives way to an "obligation" to train, and ultimately to a psychophysiological dependence in which exercise is no longer linked to enjoyment or to clearly articulated performance goals [5]. The progression from "want to" through "have to" to "must" provides a useful clinical heuristic and is supported by both cluster-analytic findings in athletes and qualitative analyses of self-narratives of affected individuals [5].

1.2. The classification problem: why exercise addiction is not yet a separate nosological entity in DSM-5 and ICD-11

Despite more than four decades of empirical work and over a thousand peer-reviewed publications [2,6], EA does not appear as a free-standing diagnostic category in either the Diagnostic and Statistical Manual of Mental

Disorders, 5th Edition (DSM-5) or the International Classification of Diseases, 11th Revision (ICD-11) [2,8]. In the DSM-5 the only formally recognised non-substance-related behavioural addiction is gambling disorder, which has been relocated from the chapter on impulse-control disorders to the "Substance-Related and Addictive Disorders" section; internet gaming disorder is listed in Section III as a condition requiring further study [2,8]. EA, together with several other candidate behavioural addictions including compulsive sexual behaviour, problematic shopping and compulsive tanning, has been judged to lack the methodologically robust evidence base required for formal inclusion [2,7]. ICD-11 has taken a comparable position, granting recognition to gambling disorder and gaming disorder while leaving other proposed behavioural addictions outside its formal nosological framework [10].

The reasons advanced for this conservative stance are multiple and reinforce one another. First, the field has been marked by considerable terminological inconsistency, with at least a dozen overlapping labels and a corresponding proliferation of assessment instruments, which complicates between-study comparisons [3,6]. Second, the available evidence is dominated by cross-sectional, self-report questionnaire data drawn from convenience samples of exercisers, university students or fitness-centre users, rather than from longitudinal clinical cohorts examined with structured diagnostic interviews [6,7]. Third, when questionnaire-based estimates are compared with clinician-led interviews, the former tend to overestimate the occurrence of EA, suggesting that some individuals identified as "at risk" by screening tools may in fact represent highly committed but non-pathological exercisers [6]. Fourth, the neurobiological substrate of EA remains incompletely mapped; although early neuroimaging data point to convergence with patterns observed in substance use disorders [10], the volume and quality of this evidence are not yet considered sufficient to anchor a distinct diagnostic category [10].

A further classificatory ambiguity concerns the relationship between EA and the obsessive–compulsive spectrum. Several authors have argued that the dysfunctional exercise behaviour observed in clinical practice combines features typical of addictive disorders — such as craving, tolerance and withdrawal — with traits more characteristic of obsessive–compulsive disorder (OCD), including ritualised behaviour, intrusive thoughts and rigidity [6]. On this view, EA may be best situated within the broader obsessive–compulsive and impulsive spectrum of behavioural addictions, rather than within a strictly addiction-focused taxonomy [6]. Other authors have emphasised the overlap with eating disorders, noting that a substantial proportion of patients with anorexia nervosa or bulimia nervosa exhibit exercise behaviours that meet the formal criteria for EA, and arguing that in clinical samples it can be difficult to determine whether the exercise dysregulation is itself the primary disorder or a manifestation of an underlying eating pathology [8].

The classificatory uncertainty has been further compounded by the absence of universally accepted diagnostic thresholds. Most contemporary screening instruments — including the Exercise Dependence Scale (EDS) and the Exercise Addiction Inventory (EAI) — were developed within a substance-dependence framework derived from DSM-IV criteria and produce dimensional risk indices rather than categorical diagnoses [4,9]. The classification of an individual as "at risk for exercise dependence" therefore does not by itself constitute a clinical diagnosis but signals the need for further evaluation, ideally by means of a structured clinical interview supplemented by careful psychiatric history and consideration of comorbidities [6,9]. This methodological constraint has direct implications for the apparent prevalence of EA in the literature, since the figures reported depend critically on the cut-off criteria adopted and the population studied [6,7].

Finally, the medicalisation of an otherwise socially desirable behaviour raises legitimate concerns. Several authors have cautioned that the indiscriminate application of addiction terminology to high-volume exercise risks pathologising committed athletes and motivated recreational exercisers, with potentially counterproductive consequences for public-health messaging that seeks to encourage physical activity [3,5]. The reluctance of nosological bodies to recognise EA as a distinct disorder may therefore be understood not only as a reflection of evidential limitations but also as an attempt to avoid premature reification of a poorly delineated construct [3,5,6]. At the same time, the absence of a formal diagnostic code complicates both clinical recognition and the development of treatment guidelines, leaving practitioners to operate within a grey zone in which the behaviour is widely acknowledged yet officially unclassified [2].

1.3. Epidemiology of exercise addiction

The epidemiological literature on EA is characterised by substantial heterogeneity, with prevalence estimates that vary considerably as a function of the population studied, the measurement instrument employed and the cut-off threshold applied [7]. A systematic review by Marques and colleagues, which synthesised data from 34 empirical studies, estimated the prevalence of risk for exercise dependence at approximately 3–7% among regular exercisers and university students and at approximately 6–9% in athletic populations [7]. A subsequent

meta-analysis of 13 studies including 3,635 participants produced an overall pooled prevalence of 6.2%, with a rate of 5.0% among amateur competitive athletes, 5.5% among university students and 8.1% among general exercisers [6]. These figures must, however, be interpreted with caution: questionnaire-based studies measure a risk index for symptoms of EA rather than a clinically confirmed diagnosis, and the available evidence consistently indicates that scalar instruments overestimate the true occurrence of EA when compared with structured interviews [6,7].

The variance is even more striking when narrower subpopulations are considered. Estimates as high as 40–50% have been reported in selected exercising samples, while in studies of general populations the rates frequently fall below 1% [8]. Among endurance athletes — runners, triathletes and ultra-endurance competitors — the figures are notably higher than those obtained in general exerciser samples [6,11]. A literature synthesis up to 2018 indicated that the highest prevalence of EA occurred among endurance athletes (14.2%), followed by ball-game players (10.4%), fitness-centre attendees (8.2%) and athletes of power disciplines (6.4%), compared with around 3.0% in the general public [6]. Reports on Polish marathon runners suggest that approximately 12% of competitors may be at risk for exercise dependence as assessed by the EDS-21, which converges with international estimates for this population [11]. In specific cohorts, considerably higher figures have been reported: 13.3% among Spanish indoor cyclists, 15.4% among Italian road and trail runners and 28% among certain football samples [5,6]. Where EA co-occurs with disordered eating, prevalence estimates rise sharply, with some studies reporting that 38–45% of individuals with eating disorders also meet criteria for problematic exercise [5,8].

Sex and age also appear to modulate risk, although the literature is not unequivocal. Some reviews have reported a slightly higher prevalence in men, while others — particularly those drawing on athletic populations — have suggested that women may be 55–67% more likely to report symptoms of EA in certain sports [5,6]. Two factors contribute to the variability of these estimates: first, women are over-represented among individuals with eating-disorder symptoms, which increases the rate of secondary EA; second, men appear over-represented in muscle-dysmorphia phenotypes that may also be associated with compulsive exercise, particularly in resistance-training contexts [4,12]. Adolescents and young adults constitute a particularly vulnerable group, given the ongoing maturation of the prefrontal cortex and the heightened sensitivity of mesolimbic reward circuits during this period; existing data suggest that exercise-related dysfunctional behaviours may emerge early and persist into adulthood, although high-quality longitudinal studies in this age range remain scarce [12].

It is worth noting that part of the apparent inflation of prevalence figures may reflect conceptual confounding between addiction and instrumental exercise. A meta-analysis examining EA in samples with and without eating disorders found that EA occurred more than three and a half times more frequently as a comorbidity of an eating disorder than as a free-standing problem, suggesting that a substantial proportion of "EA" identified in the literature may in fact reflect secondary or instrumental exercise in the context of eating-related pathology [6]. Taken together, the epidemiological picture supports the view that EA is a clinically meaningful, although not rare, phenomenon, with particular concentration in endurance sports, in fitness-centre subcultures emphasising body composition, and in clinical samples with eating disorders. The wide range of reported figures, however, reinforces the call for methodological standardisation in epidemiological research, including the consistent use of validated instruments, the systematic measurement of eating-disorder symptomatology, and the use of clinical interviews to confirm questionnaire-based identifications [6,7].

1.4. Aim of the work

In view of the conceptual, classificatory and methodological complexities outlined above, the present narrative review aims to structure the available clinical and neuroscientific evidence on exercise addiction in a way that may be useful for psychiatry trainees and for practitioners working at the interface of mental health and sports medicine. The review draws primarily on evidence-based publications from the past two decades, with selective inclusion of foundational papers that have shaped the field. The specific objectives are: (i) to summarise the diagnostic criteria proposed for EA and the principal psychometric instruments used in research and clinical practice, with particular attention to their conceptual foundations and limitations; (ii) to synthesise current knowledge on the neurobiological substrates of EA, including dysregulation of mesolimbic dopaminergic pathways, endogenous opioid and endocannabinoid signalling, and structural and functional alterations in prefrontal and limbic networks identified through neuroimaging [10]; (iii) to outline the principal differential-diagnostic challenges separating EA from eating disorders, muscle dysmorphia, obsessive–compulsive disorder, the overtraining syndrome and high-level athletic commitment; and (iv) to review contemporary therapeutic options, including cognitive–behavioural and dialectical-behavioural approaches, and the limited, largely off-label pharmacological strategies that may be considered in the management of comorbid conditions [2].

By integrating evidence drawn from psychiatric, addiction-medicine, sport-science and neuroimaging literatures, the review seeks to contribute to a clearer conceptualisation of EA as a clinically meaningful phenomenon situated at the interface of behavioural medicine and psychiatry, while remaining mindful of the methodological caveats that continue to limit the strength of the available evidence [3,6]. The deliberately cautious tone adopted here reflects the conviction, shared by many authors in the field, that EA may be best understood at present as a candidate disorder whose nosological status will depend on the accumulation of further longitudinal, neurobiological and interview-based clinical data [2,5,6].

2. Materials and Methods

2.1. Study design

The present work was conceptualised as a narrative review with elements of a structured literature synthesis, intended to integrate empirical, theoretical and clinical contributions relevant to the construct of exercise addiction (EA). A narrative format was considered most appropriate given the heterogeneity of the existing evidence base, which spans psychiatric epidemiology, addiction medicine, sport and exercise psychology, behavioural neuroscience and clinical sports medicine, and which encompasses methodologically diverse designs ranging from large cross-sectional surveys and meta-analyses to qualitative case studies, validated psychometric work and small-sample neuroimaging investigations. A strictly quantitative meta-analytic approach was deemed unsuitable, since the marked variation in study populations, screening instruments, cut-off thresholds and operational definitions of the construct would have undermined the comparability of effect sizes across studies. At the same time, an unstructured traditional review was avoided in favour of a transparent, reproducible search procedure that allowed source-level traceability and minimised the risk of selective citation.

The review was guided by four overarching aims that mirror the structure of the subsequent narrative: (i) to delineate the conceptual boundaries of EA in relation to adjacent constructs such as exercise dependence, compulsive exercise and instrumental exercise; (ii) to summarise contemporary diagnostic frameworks and psychometric instruments together with their underlying theoretical foundations; (iii) to synthesise the available neurobiological evidence pertaining to reward circuitry, opioid and endocannabinoid signalling, and frontolimbic structural alterations; and (iv) to outline the principal differential-diagnostic challenges at the interface of psychiatry and sports medicine, including comorbid eating disorders, muscle dysmorphia, obsessive–compulsive disorder and the overtraining syndrome. The review was conducted between the conceptualisation of the project and the closure of the literature search and synthesis, with selection of sources and thematic mapping performed by a single author and cross-checked against the original full-text articles whenever quantitative figures, prevalence estimates or diagnostic thresholds were extracted.

The methodological orientation adopted in this work is consistent with evidence-based medicine principles, in that it gives precedence to systematic reviews, meta-analyses and validated empirical studies over editorial opinion or unreplicated case material. At the same time, qualitative case reports and clinical interviews were not excluded, as such sources retain considerable value in the description of a phenomenon for which standardised diagnostic criteria are not yet available. Where the empirical evidence was sparse or methodologically limited, this is acknowledged explicitly in the narrative, and inferences are framed with appropriate epistemic caution. No new primary data were collected for the purposes of this review, and as such no ethical approval was required; all included studies had received the relevant ethical clearance from their respective institutional review bodies, as reported in the original publications.

2.2. Literature search strategy

The literature search was conducted through electronic interrogation of major biomedical and behavioural-science databases, complemented by manual screening of reference lists. The principal databases consulted included MEDLINE (via PubMed), Scopus, Web of Science, PsycINFO and Google Scholar. The choice of databases was guided by the inter-disciplinary nature of the subject, which sits at the convergence of clinical psychiatry, behavioural science, sports medicine and neuroscience, and required coverage that extended beyond traditional biomedical indices. The search covered publications appearing between January 1976 — the year in which Glasser introduced the influential concept of "positive addiction" and thereby inaugurated the modern scholarly discussion of EA — and the most recent indexed records available at the time of synthesis in 2025–2026. No language restriction was imposed at the screening stage; however, full-text articles published in English or Polish were prioritised for inclusion in order to maintain accuracy in the interpretation of clinical

terminology, while a small number of records published in other European languages were considered through their English-language abstracts and, where necessary, through translated full texts.

The search strategy combined Medical Subject Headings (MeSH terms) with free-text keywords linked through Boolean operators. The core search string used "exercise addiction" OR "exercise dependence" OR "compulsive exercise" OR "obligatory exercise" OR "exercise abuse" OR "morbid exercise" OR "sport addiction" OR "running addiction" OR "bigorexia" OR "muscle dysmorphia" combined with terms reflecting the thematic axes of the review, including "diagnostic criteria", "psychometric", "Exercise Dependence Scale", "Exercise Addiction Inventory", "prevalence", "epidemiology", "neurobiology", "neuroimaging", "dopamine", "endorphin", "endocannabinoid", "orbitofrontal cortex", "overtraining syndrome", "eating disorders", "anorexia nervosa", "bulimia nervosa", "obsessive–compulsive disorder", "differential diagnosis", "cognitive-behavioural therapy" and "pharmacotherapy". The terms were adapted to the syntactic specifications of each database and were applied to titles, abstracts and indexed keywords. The reference lists of all retrieved systematic reviews, meta-analyses and narrative reviews were manually screened in order to identify additional relevant publications not captured by the electronic search ("snowballing" approach), and selected high-impact authors in the field were searched individually to ensure that influential contributions were not overlooked.

A particular emphasis was placed on retrieving the most recent meta-analyses and systematic reviews available, since these constitute the highest tier of the evidence hierarchy for descriptive and observational data and offer pooled estimates that compensate, at least in part, for the methodological heterogeneity characteristic of the field. Equally, foundational sources that have defined the conceptual landscape of EA — including the seminal works of Glasser, Morgan, de Coverley Veale, Griffiths, Berczik and Hausenblas and Symons Downs — were retrieved either in their original form or, where access was limited, through subsequent citations in authoritative secondary literature. The search was supplemented by targeted retrieval of validation studies for the principal psychometric instruments, namely the Exercise Dependence Scale-21 (EDS-21), the Exercise Addiction Inventory in its original and revised forms (EAI and EAI-3), the Obligatory Exercise Questionnaire, the Commitment to Exercise Scale, the Compulsive Exercise Test and related tools. Neuroimaging studies were retrieved using additional search terms reflecting the modalities employed in the original investigations, including "functional magnetic resonance imaging", "fMRI", "structural MRI", "diffusion tensor imaging", "positron emission tomography" and "voxel-based morphometry".

Finally, grey literature was consulted only selectively. Position statements and consensus documents issued by professional bodies — such as the American Psychiatric Association, the World Health Organization, the European College of Sport and Exercise Physicians and the International Olympic Committee — were reviewed to contextualise the clinical and nosological discussion. Doctoral dissertations, conference proceedings and non-peer-reviewed reports were generally excluded, unless they provided unique empirical data of methodological quality comparable to that of peer-reviewed publications.

2.3. Inclusion and exclusion criteria

The inclusion criteria were defined in advance and applied at three sequential screening stages — title, abstract and full text. To be eligible for inclusion, a publication had to fulfil all of the following conditions: (i) it constituted an empirical study, a systematic review, a meta-analysis, a narrative review, a validation study of a psychometric instrument, a case report or a case series; (ii) its primary focus addressed exercise addiction, exercise dependence, compulsive exercise or a closely related construct, with explicit assessment of the relevant behavioural, psychological, neurobiological or clinical dimensions; (iii) it was published in a peer-reviewed journal or, in the case of authoritative theoretical or clinical contributions, in a recognised scholarly venue; (iv) the population studied comprised either human participants — including adolescents, young adults, middle-aged adults, retired adults, recreational exercisers, amateur and elite athletes, or clinical samples with comorbid eating disorders, anxiety, depressive or obsessive–compulsive disorders — or, in selected sections, mammalian models relevant to the elucidation of neurobiological mechanisms (notably the endocannabinoid signalling literature on cursorial species relevant to the "runner's high" phenomenon); and (v) the full text was retrievable through institutional access or open-access channels.

Several additional inclusion criteria applied to specific thematic axes of the review. For studies contributing to the epidemiological section, only investigations that reported quantitative prevalence estimates of risk for EA or exercise dependence in clearly defined populations were retained. For studies contributing to the diagnostic and psychometric section, only those that reported reliability or validity data for an assessment instrument, or that proposed novel diagnostic criteria, were included. For studies contributing to the neurobiological section, only investigations employing validated imaging modalities, well-characterised behavioural paradigms or robust

biochemical assays were retained, and findings derived solely from animal models were considered as supportive rather than confirmatory evidence. For the section on differential diagnosis, studies that explicitly compared EA with eating disorders, muscle dysmorphia, OCD or the overtraining syndrome were prioritised.

The exclusion criteria were correspondingly defined. Publications were excluded if they: (i) addressed exercise behaviour or physical activity without reference to its dysfunctional, compulsive or addictive dimensions; (ii) lacked a clear operational definition of EA or an equivalent construct; (iii) consisted solely of editorial commentary or opinion pieces without empirical or systematic foundation; (iv) reported only protocols of studies without empirical findings, unless the protocol provided novel methodological insights relevant to the present synthesis; (v) presented duplicate data already reported in another included publication, in which case the most comprehensive or methodologically robust version was retained; (vi) were available only as abstracts without retrievable full text; or (vii) suffered from major methodological flaws, such as the absence of a clearly defined sample, the absence of validated measurement instruments or insufficient reporting of statistical analyses, that would have compromised the interpretability of their conclusions.

Studies in which EA was addressed only as a secondary or marginal topic were considered for inclusion only when they offered uniquely informative observations on a specific aspect of the construct — for instance, methodological reflections on the limitations of self-report instruments, or clinical illustrations of the overlap between EA and other psychiatric disorders. Conference abstracts, dissertations and grey-literature outputs were excluded unless they provided substantive empirical data not available elsewhere. Where conflicting evidence emerged between studies of comparable quality, the discrepancies are explicitly discussed in the narrative rather than being resolved by arbitrary preference. In keeping with best practice in narrative synthesis, no single source was treated as definitive, and convergent findings across multiple independent studies were given greater interpretative weight than isolated reports.

2.4. Data synthesis

The data synthesis was conducted through a qualitative, thematic approach that aligns the structure of the narrative with the conceptual architecture of the field. Following retrieval, the included publications were organised into thematic clusters corresponding to the principal sections of the review: conceptualisation and terminology, epidemiology, diagnostic criteria and psychometric assessment, neurobiological mechanisms, differential diagnosis with eating disorders and muscle dysmorphia, differential diagnosis with obsessive–compulsive disorder, the overtraining syndrome and elite athletic commitment, and therapeutic interventions. Within each cluster, the studies were further classified according to design (cross-sectional, longitudinal, qualitative, neuroimaging, validation study, systematic review or meta-analysis), the population studied (general exercisers, athletes, clinical samples) and the assessment tool employed.

Key data extracted from each included study comprised: bibliographic details (authors, year, country); study design and sample characteristics (size, sex distribution, age range, sport discipline where relevant, presence of comorbidity); the operational definition of EA or related construct adopted; the assessment instrument and cut-off criteria used; the principal findings relevant to the present review; and the authors' interpretative conclusions. Where the original publications provided quantitative estimates — for example, prevalence rates, effect sizes, mean scale scores or correlations with comorbid features — these figures were retained in the narrative in their original form. Where the original publications offered qualitative observations — for example, the description of withdrawal experiences in clinical interviews or the phenomenology of compulsive training behaviour — these were summarised in the narrative with attention to representativeness across studies.

Thematic synthesis was performed by iterative reading and re-reading of the included publications, with attention to convergent and divergent findings. Particular care was taken to differentiate primary empirical evidence from secondary interpretations advanced in review articles, and to avoid the recursive citation of secondary sources that has been identified as a methodological hazard in fields characterised by terminological heterogeneity. Wherever possible, the strongest available evidence — typically a meta-analysis, a systematic review or a well-conducted longitudinal study — was used as the anchor for a given thematic claim, with corroborating or qualifying evidence drawn from additional sources. Discrepancies between sources, whether of conceptual, methodological or empirical origin, are flagged in the narrative and discussed rather than smoothed over.

In keeping with the cautious epistemic register adopted throughout the review, all inferential statements are framed in conditional or probabilistic terms that reflect the actual strength of the underlying evidence. Statements of the form "evidence suggests", "available data indicate", "appears to be associated with" or "may

contribute to" are used in preference to declarative absolutes, in order to capture the genuine uncertainty that continues to characterise the field. Where the available evidence appears robust and convergent — for example, the broad consensus on the six-component framework of behavioural addiction, or the reproducible observation of frontolimbic structural alterations in neuroimaging samples — this is noted explicitly. Conversely, areas of marked uncertainty, such as the precise nosological status of EA, the boundary between primary and secondary forms, or the effectiveness of specific therapeutic interventions, are highlighted as candidate priorities for future investigation.

Limitations of the synthesis itself are acknowledged. As a narrative review, the work does not pursue an exhaustive enumeration of every published source on EA and is not accompanied by a quantitative pooled estimate of effect sizes. The thematic organisation, while internally coherent, inevitably reflects interpretative choices made by the author, and alternative organisational schemes could legitimately have been adopted. The reliance on self-report instruments in the majority of the underlying empirical literature is a structural limitation of the field that is inherited by the present review and is explicitly acknowledged throughout. These caveats notwithstanding, the methodology employed here is intended to provide a transparent, reproducible and balanced account of the current state of knowledge on exercise addiction, with sufficient methodological detail to permit critical appraisal by the reader and replication of the synthesis by future investigators.

3. Diagnostic criteria and screening instruments for exercise addiction

3.1. Adaptation of the criteria for substance use disorders (DSM-IV/DSM-5): exercise addiction modelled on Griffiths' six-component framework

The conceptual foundations of contemporary diagnostic frameworks for exercise addiction (EA) are derived, almost without exception, from analogies with substance use disorders and from the broader theory of behavioural addiction articulated in the late twentieth century [3,9]. In the absence of formally adopted nosological criteria in either the DSM-5 or the ICD-11, research and clinical practice have come to rely on a set of operational definitions that have been adapted, with relatively minor modifications, from the criteria used to identify substance dependence in the DSM-IV and its successor [9,15]. The most influential of these frameworks is the six-component model of behavioural addiction proposed by Griffiths, which has been widely applied to a variety of non-substance-related conditions including gambling, internet gaming, problematic shopping and EA [3,13]. The six components are salience, mood modification, tolerance, withdrawal symptoms, conflict and relapse, and each of them can be readily translated into the exercise context.

Salience refers to the degree to which exercise becomes the most important activity in an individual's life, dominating thought, feeling and behaviour [3,13]. In a clinical sense, salience denotes the cognitive preoccupation with training that affected individuals describe even outside the period of physical activity itself: the planning of sessions, the mental rehearsal of routes or routines, the structuring of meals, sleep and work around exercise, and the intrusive return to training-related thoughts during activities ostensibly unrelated to sport [13,16]. Mood modification denotes the use of exercise as a strategy to alter affective state — either to produce a positive subjective experience often described as a "buzz" or "high", or, perhaps more importantly in clinical samples, to alleviate dysphoric states such as anxiety, irritability, tension or low mood [3,13]. The function of exercise as an affect-regulatory tool is one of the features that brings EA conceptually close to the addiction model, in which the behaviour is sustained as much by negative reinforcement (relief from aversive states) as by positive reinforcement (pursuit of pleasurable states) [3,13].

Tolerance, in the exercise context, refers to the tendency to require progressively greater volumes, intensities or durations of training to obtain the same subjective effect, or, equivalently, to the diminishing effect of a given level of training [9,11]. This component has been the subject of considerable debate, since tolerance to training load is also a normal physiological adaptation that underlies the response to structured athletic preparation; the diagnostic question is therefore not whether tolerance is present but whether the progressive escalation occurs in the absence of an explicit performance goal and despite mounting personal cost [4,11]. Withdrawal symptoms refer to the emergence of negative affective, cognitive and somatic states when exercise is interrupted; these typically include irritability, anxiety, restlessness, guilt, low mood, insomnia, fatigue and, in some accounts, autonomic features such as palpitations or muscle tension [3,9,19]. Conflict, the fifth component, encompasses both interpersonal conflict (with partners, family, friends or employers) and intrapsychic conflict (the awareness, however attenuated, of a tension between one's exercise behaviour and other valued domains of life) [13]. Finally, relapse describes the tendency to revert rapidly to former patterns of exercise after periods of abstinence or reduction, sometimes with an overshoot effect in which the post-abstinence training volume exceeds the pre-abstinence baseline [3,13].

Hausenblas and Symons Downs were among the first to translate this conceptual scaffold into an operational instrument tied directly to the DSM-IV criteria for substance dependence [9]. They proposed that EA should be conceptualised as a maladaptive pattern of exercise leading to clinically significant impairment or distress, and they identified seven symptomatic criteria adapted from the substance-dependence framework: (1) tolerance, expressed as the need for increasing amounts of exercise to achieve the desired effect; (2) withdrawal, manifested by adverse affective and somatic states or by exercise undertaken specifically to relieve such states; (3) intention effects, in which the individual repeatedly exceeds the planned duration or intensity of training; (4) loss of control, expressed as unsuccessful attempts to reduce or regulate exercise; (5) time, denoting the disproportionate amount of time devoted to preparing for, engaging in and recovering from exercise; (6) reductions in other activities, with social, occupational and recreational engagements scaled back to accommodate training; and (7) continuance despite persistent physical or psychological problems caused or exacerbated by exercise [9,11]. The threshold for "at-risk" classification, as originally proposed by Hausenblas and Symons Downs, requires the endorsement of at least three of these seven criteria at the highest severity level [9,11]. This DSM-based scaffolding has subsequently been embedded in the most widely used psychometric tool of its class, the Exercise Dependence Scale (EDS) and its revised 21-item form, the EDS-21 [9,11].

A parallel line of work, anchored in the broader theory of behavioural addiction rather than in the substance-dependence framework, has produced an alternative but partially overlapping operationalisation. Building on Brown's general components of addiction and Griffiths' subsequent refinement, Terry, Szabo and Griffiths developed the Exercise Addiction Inventory (EAI), a brief six-item instrument that mirrors the six-component behavioural-addiction model directly [13,15]. The EAI was conceived as a practical, time-efficient screening tool that could be used in general practice, sports medicine and occupational health settings, where the longer EDS-21 was felt to be impractical [13]. Within both traditions, however, the conceptual core remains the same: EA is operationalised through criteria that closely mirror those used to identify substance dependence, with adjustments to accommodate the specific behavioural and physiological features of exercise [4,15,18].

It is important to recognise that the substance-dependence framework, however influential, is not the only conceptualisation available. Bamber and colleagues, working from qualitative interview data with adult female exercisers, proposed an alternative set of diagnostic criteria centred on impaired functioning across psychological, social and occupational, physical, and behavioural domains, combined with the presence of withdrawal symptoms [16]. Under this scheme, the diagnosis of exercise dependence requires impairment in at least two of these domains together with evidence of withdrawal — either as an adverse reaction to the interruption of exercise or as unsuccessful attempts at exercise control [16]. The model also explicitly separates primary from secondary exercise dependence on the basis of the presence or absence of an eating disorder [16]. The Bamber framework has been cited as one of the few attempts to derive diagnostic criteria empirically from clinical interview material rather than by analogy with another disorder, and it remains influential in the qualitative literature on EA [4,16].

Other theoretical frameworks have located EA within different conceptual neighbourhoods. Freimuth, Moniz and Kim proposed a four-phase heuristic — recreational exercise, at-risk exercise, problematic exercise and exercise addiction — that situates EA at the end of a continuum and emphasises the gradual transformation of healthy exercise into pathology [4]. Egorov and Szabo, by contrast, advanced an interactional model in which EA emerges as an escape strategy from significant life stressors, and in which the trajectory is determined by the interaction between personal factors (personality traits, past experience, perceived control) and situational factors (life events, social environment) [18]. A recent systematic review of seventeen psychometric tools concluded that the existing instruments can be grouped into five conceptual families: those that frame problematic exercise as the end of a continuum, those that frame it as a means of regulating body shape and weight, those that frame it as a form of dependence, those that frame it as a behavioural addiction, and a residual category without a clearly articulated conceptualisation [18]. This conceptual heterogeneity, together with the resulting fragmentation of assessment instruments, has been identified as one of the principal obstacles to the formal nosological recognition of EA [4,18].

3.2. Tolerance and the withdrawal syndrome in sport: physiological and psychological features of exercise deprivation

Among the criteria that constitute the Griffiths–Hausenblas–Symons Downs framework, tolerance and withdrawal occupy a particularly important position because they provide the most direct phenomenological bridge between EA and substance use disorders [3,9]. The conceptual claim that EA can be understood as an addiction depends, at least in part, on the demonstration that the behaviour produces neuroadaptive changes that

manifest clinically as increased tolerance to training load and as a recognisable withdrawal syndrome when exercise is curtailed [3,19].

The phenomenon of tolerance in habitual exercisers has both physiological and psychological dimensions. At the physiological level, tolerance to a given training stimulus is a well-characterised adaptive response of the cardiovascular, musculoskeletal and neuroendocrine systems, and is the basis of the principle of progressive overload that underpins athletic training [4,11]. In the context of EA, however, the relevant form of tolerance is not the adaptive escalation aimed at performance, but rather the seemingly involuntary requirement for increasing training volume or intensity in order to obtain the same subjective effect — typically the same affect-regulatory benefit or the same sense of relief from intrusive cognition [3,13]. This distinction is clinically important but operationally difficult: questionnaire-based assessment cannot easily separate adaptive progression from compulsive escalation, and several authors have argued that the criterion of tolerance, when applied to exercise, is among the least specific of the six components and is the one most likely to be endorsed by highly committed but non-pathological athletes [4,18]. In some samples of marathon and ultra-endurance runners, tolerance scores on the EDS-21 are among the highest of any subscale, yet are not necessarily accompanied by elevations on the other subscales that would justify a classification of being at risk [11].

The withdrawal syndrome associated with exercise interruption has been described in a more clinically distinctive manner, and it constitutes one of the most consistent features of the phenomenology of EA across both questionnaire-based and qualitative studies [3,19]. Affected individuals deprived of their habitual training routine — whether through injury, illness, occupational demands, weather or external constraint — typically report the onset, within approximately 24 to 36 hours, of a cluster of symptoms that includes irritability, anxiety, restlessness, low mood, guilt, insomnia, fatigue, intrusive ruminations about exercise, and, in some accounts, somatic discomforts such as muscle tension or autonomic arousal [3,19]. Early observational work by Baekeland, who attempted to recruit habitual exercisers into a sleep study requiring a temporary cessation of training, documented the difficulty of finding willing participants and noted that those who did agree to abstain exhibited measurable increases in anxiety and sleep disturbance, a finding that was subsequently interpreted as evidence of a withdrawal-like state [19]. Morgan's case material similarly emphasised the daily necessity of exercise in affected individuals and the affective and somatic deterioration that followed its interruption [19].

In contemporary clinical descriptions, the withdrawal syndrome in EA is typically understood as a multidimensional construct comprising affective, cognitive, somatic and motivational components [3,16,19]. The affective component centres on dysphoria, irritability and anxiety, with depressive features that in some patients meet criteria for a brief depressive episode [2,15]. The cognitive component includes intrusive thoughts about exercise, a sense of mental "fog" or impaired concentration, and ruminative preoccupation with the missed training session and its consequences for performance, weight or body composition [16]. The somatic component involves sleep disturbance — most commonly initial insomnia and a perception of non-restorative sleep — together with restlessness, muscle tension and, in some reports, palpitations or gastrointestinal discomfort [16,19]. The motivational component is expressed as an intense craving for exercise, with affected individuals describing an almost compulsive drive to resume training as soon as the constraint is removed [3,13]. Importantly, the withdrawal syndrome in EA can typically be alleviated by resuming exercise, a feature that mirrors the negative-reinforcement pattern observed in substance use disorders and that supports the conceptualisation of EA within the addiction framework [3,13,19].

A further parallel with substance use disorders concerns the temporal dynamics of withdrawal. In several qualitative studies, affected individuals report that withdrawal symptoms emerge most strongly within one to three days of cessation and diminish over the course of one to two weeks, although a persistent sense of unease and a heightened sensitivity to exercise-related cues may continue for considerably longer [16,19]. Some authors have drawn an analogy with the post-acute withdrawal phenomena described in substance-use literature, in which protracted dysphoric states and craving may persist for months after the initial withdrawal period [3]. However, the evidence for such protracted withdrawal in EA is largely anecdotal and has not yet been subjected to systematic prospective study [6].

It is also worth noting that the experience of withdrawal in EA appears to be modulated by individual factors. Studies of marathon runners and other endurance athletes suggest that the intensity of withdrawal-related symptoms may be greater in younger participants and in those with longer training histories, and may differ between sexes — with some studies reporting higher withdrawal scores in men, although the direction and magnitude of sex effects are not consistent across the literature [11]. Personality variables, including neuroticism, perfectionism and reward sensitivity, have also been associated with the severity of withdrawal experiences, although the available evidence is largely cross-sectional [3,19]. From a clinical standpoint, the careful

elicitation of the withdrawal syndrome — its onset, its symptom profile, its temporal course and the degree of distress it produces — remains one of the most informative components of the diagnostic interview in EA and may help to distinguish between adaptive athletic commitment and pathological dependence [4,16].

3.3. The psychometric "gold standard": a brief overview of instruments used in research and clinical practice

The assessment of EA in research and, increasingly, in clinical practice has come to rely on a relatively small number of well-validated self-report instruments, although the broader landscape contains at least 17 distinct tools, each anchored in a slightly different conceptual framework [18]. A recent systematic review identified six instruments that account for the great majority of empirical work in the field: the Commitment to Exercise Scale (CES), the Compulsive Exercise Test (CET), the Exercise Addiction Inventory (EAI), the Exercise Dependence Questionnaire (EDQ), the Exercise Dependence Scale (EDS-21) and the Obligatory Exercise Questionnaire (OEQ) [17,18]. Among these, two — the EDS-21 and the EAI — have come to be regarded as the de facto psychometric standards in the field, both because of their robust psychometric properties and because of their direct conceptual anchoring in the substance-dependence and behavioural-addiction frameworks respectively [9,13,14].

3.3.1. The Exercise Dependence Scale (EDS-21)

The Exercise Dependence Scale was developed by Hausenblas and Symons Downs in the early 2000s, drawing directly on the DSM-IV criteria for substance dependence [9]. The instrument was conceptualised as a multidimensional measure of exercise dependence and was designed from the outset to differentiate among three groups of exercisers: those at risk for dependence, those who were symptomatic but not dependent, and those who were asymptomatic [9,11]. The current standard version, the EDS-21, comprises 21 items divided evenly across seven subscales, each containing three items and each corresponding to one of the seven DSM-IV-based criteria, namely withdrawal, continuance, tolerance, lack of control, reduction in other activities, time and intention effects [9,11,14].

Each item of the EDS-21 is scored on a six-point Likert scale ranging from 1 ("never") to 6 ("always"), and respondents are classified according to their pattern of responses across the seven subscales. An individual is considered "at risk" for exercise dependence if they obtain scores of 5 or 6 on at least three of the seven subscales; "symptomatic non-dependent" if they obtain scores of 3 to 4 on at least three subscales without meeting the at-risk criteria; and "asymptomatic non-dependent" if they obtain scores of 1 to 2 on at least three subscales [11,14]. Subscale scores are also interpreted individually, with values greater than 14 considered "at risk", values between 7 and 14 considered "symptomatic" and values below 7 considered "asymptomatic" [14]. The EDS-21 has been translated and validated in a wide range of languages and cultural contexts, including Polish, Italian, Spanish, German and Hungarian, and it has shown generally acceptable reliability across these adaptations [11,17,18].

Psychometric evaluations of the EDS-21 have provided broadly favourable evidence. The original validation studies reported good internal consistency for the total score and for most subscales, satisfactory test–retest reliability and content and concurrent validity supported by correlations with related instruments [9]. A reliability-generalisation meta-analysis encompassing 255 studies and 254,174 participants estimated the pooled internal-consistency coefficient (Cronbach's α) for the EDS-21 total score in the range of approximately 0.77 to 0.93, with subscale reliabilities ranging from approximately 0.62 to 0.91 [17]. The same meta-analysis identified the EDS-21 as the most extensively studied instrument in the field and confirmed that its reliability is sensitive to sociodemographic and methodological characteristics of the sample, including age range, sex composition, exercise modality and clinical status [17]. Some subscales, notably "withdrawal" and "reductions in other activities", have shown lower internal consistency in certain samples — for example, in Polish marathon runners the α coefficient for the withdrawal subscale dropped to 0.56, somewhat below the conventional threshold for acceptability [11].

The clinical utility of the EDS-21 is enhanced by its multidimensional structure, which provides a granular profile of the components of dependence rather than a single summary score, and which can therefore inform individualised case formulation [9,14]. At the same time, several limitations have been identified. The instrument was developed with reference to the DSM-IV criteria for substance dependence and therefore reflects the conceptual assumptions of that framework, including the centrality of tolerance and withdrawal as defining features [9,18]. As noted above, these criteria may be of limited specificity in the exercise context, particularly among committed athletes who exhibit both progressive training loads (a form of tolerance) and emotional

distress when training is interrupted (resembling withdrawal) without otherwise meeting the criteria for a behavioural addiction [4,6]. The classification thresholds used in the EDS-21 have also been criticised for producing relatively high "at-risk" rates in certain populations, which may reflect over-detection rather than a true clinical signal [6,7].

3.3.2. The Exercise Addiction Inventory (EAI)

The Exercise Addiction Inventory was developed by Terry, Szabo and Griffiths in the early 2000s as a brief, theoretically grounded screening tool that could be applied in primary care, sports medicine, occupational therapy and physiotherapy settings, where the time required to administer the longer EDS-21 was considered prohibitive [13]. The EAI is anchored directly in Griffiths' six-component framework for behavioural addiction, and each of its six items corresponds to one of the components — salience, mood modification, tolerance, withdrawal, conflict and relapse [13,15]. In its original form, each item is rated on a five-point Likert scale (from 1, "strongly disagree", to 5, "strongly agree"), and the total score ranges from 6 to 30 [13]. A cut-off score of 24 or above identifies an individual as being at risk of exercise addiction, while scores between 13 and 23 indicate a symptomatic but non-dependent profile, and scores between 6 and 12 indicate an asymptomatic profile [13,15].

The psychometric properties of the EAI have been examined in numerous studies. The original validation in a sample of 200 habitual exercisers reported good internal consistency (Cronbach's $\alpha = 0.84$), good two-week test–retest reliability ($r = 0.85$), strong concurrent validity with the Obligatory Exercise Questionnaire ($r = 0.80$) and with the EDS ($r = 0.81$), and a unidimensional factor structure accounting for approximately 56% of the variance [13]. The Spanish adaptation of the EAI, evaluated in a sample of 584 university students, confirmed the factor structure of the original instrument, reported an internal-consistency coefficient of 0.70, a four-week intraclass correlation of 0.92 for temporal stability, and measurement invariance across sex [15]. The reliability-generalisation meta-analysis cited above estimated the pooled Cronbach's α for the EAI in the region of 0.79 to 0.85, broadly comparable to the EDS-21 and other widely used instruments [17]. The instrument has been translated and validated in at least ten language versions, including Arabic, Chinese, Danish, English, Hungarian, Italian, Japanese, Persian, Polish and Spanish [14,15].

The principal strengths of the EAI lie in its brevity, simplicity of scoring and the ease with which it can be administered in busy clinical settings; the original developers explicitly noted that the instrument can be completed in under a minute and scored without specialist training [13]. These features have made the EAI particularly popular for community-based and large-sample epidemiological work, and have facilitated cross-cultural comparisons of EA prevalence [14,15]. The EAI also has theoretical advantages over the EDS-21, in that it is anchored in the behavioural-addiction framework rather than in the substance-dependence framework, and may therefore be more conceptually appropriate for a behaviour that — unlike substance use — is socially valued and not intrinsically harmful [13,18].

A number of limitations have been identified, however, and have motivated the development of revised versions. The original EAI does not capture three clinically important features that have been emphasised in qualitative work on EA, namely guilt associated with missed sessions, the continuation of training despite injury or medical advice and the explicit recognition of negative consequences caused by exercise [14]. A revised version, the EAI-R, modified the response scale from five to six points to enhance sensitivity, while preserving the six-item structure [14]. More recently, an expanded nine-item version (the EAI-3) has been developed and tested in a large international sample of 1,931 exercisers across five linguistic and cultural contexts (Chinese, German, Italian, Japanese and Turkish) [14]. The EAI-3 added three items specifically addressing guilt ("I feel guilty if I miss planned training or if my training does not go as well as planned"), training despite injury ("I am inclined to train when, or before completely recovered from, illness or injury") and experienced harm ("I have had physical, psychological and/or other issues due to my exercise regime"), and proposed a two-factor structure ("health relevance" and "addiction tendency") together with a recommended cut-off of 34 out of 48 for the eight-item final version [14].

Empirical comparisons between the EAI and the EDS-21 have generally shown moderate to strong concurrent validity, with correlation coefficients typically in the range of 0.5 to 0.8 [13,14]. Nevertheless, concordance between the two instruments in identifying individuals at risk of EA is imperfect — one large study reported an overall agreement of approximately 88% between the EAI and the EDS-R, which means that approximately one in eight participants was classified differently by the two instruments [5]. This suggests that the two scales, although closely related, do not measure exactly the same construct, and that the choice of instrument may have meaningful consequences for prevalence estimates and clinical decision making [5,6,18].

3.3.3. Other instruments and broader psychometric considerations

In addition to the EDS-21 and the EAI, several other instruments are in regular use, each with a distinct theoretical orientation. The Compulsive Exercise Test (CET) was developed within the eating-disorder literature and is widely used in research and clinical practice with patients with anorexia nervosa, bulimia nervosa and related conditions; it focuses on the maintenance role of exercise in eating-disorder pathology and includes subscales on avoidance and rule-driven behaviour, weight-control exercise, mood improvement, lack of exercise enjoyment, and exercise rigidity [17,18]. The Obligatory Exercise Questionnaire (OEQ), one of the earliest tools in the field, addresses the subjective need to engage in repetitive exercise behaviour but has been criticised for its conceptual breadth and limited capacity to differentiate between dependence and high commitment [9,17]. The Commitment to Exercise Scale (CES), in both its Likert and visual-analogue versions, assesses the extent to which exercise influences well-being and how it interferes with social and occupational obligations [17]. The Exercise Dependence Questionnaire (EDQ) was developed by Ogden, Veale and Summers and was the first instrument to attempt a multidimensional operationalisation of exercise dependence, although several of its subscales address attitudes towards exercise rather than dependence symptoms strictly defined [15,18].

Reliability data across these instruments have been synthesised in a recent meta-analysis encompassing 255 studies and 741 independent samples [17]. Pooled α coefficients ranged from approximately 0.77 to 0.93 for total scores and from approximately 0.62 to 0.91 for subscale scores, with the highest internal-consistency values observed for the EDS-21 and the EAI total scores [17]. The same meta-analysis identified several methodological concerns that are not specific to any single instrument but that affect the field as a whole: a high rate of "reliability induction", in which authors report reliability coefficients from earlier studies rather than from their own data (estimated at almost 48% of included studies); the virtual absence of testing of the assumptions required for the valid use of Cronbach's α (notably tau-equivalence and the independence of error terms); and a marked under-reporting of test–retest reliability, which limits the available evidence on the temporal stability of EA scores [17]. These observations underscore the cautious interpretation that should be applied to prevalence estimates derived from these instruments and reinforce the call for methodological standardisation in the field [6,17].

It should also be emphasised — and this point has been made repeatedly in the recent literature — that none of the available instruments constitutes a diagnostic tool in the strict sense [6,14]. All of them generate a dimensional risk index, and their cut-off values identify a "risk" or "symptomatic" profile rather than a confirmed clinical diagnosis [6,14]. A high score on any of these instruments should therefore be regarded as a signal for further clinical evaluation, ideally including a structured interview, a detailed exercise and training history, a careful assessment of comorbidities (particularly eating disorders, mood and anxiety disorders, and obsessive–compulsive features) and a contextual appraisal of the impact of exercise on the individual's broader functioning [6,14,16]. The convergence of evidence from screening instruments, clinical interview and collateral information (from family members, training partners or treating physicians) is generally considered the most reliable basis for clinical decision making in the absence of formally adopted nosological criteria [6,14,16].

Finally, the field has begun to acknowledge the limitations inherent in cross-cultural application of these instruments. Some items of the EAI and EAI-3 — notably those touching on family and partner conflict and on the cultural salience of exercise — have been found to violate scalar invariance across linguistic groups, with mean scores differing systematically between, for example, Japanese, Chinese and European samples [14,15]. This implies that direct between-country comparisons of mean scores may be misleading, and that culturally adapted norms may be required before instruments can be used clinically in a given setting [14]. Polish-language adaptations of the EAI and EDS-21 have been used in research on amateur athletes and university students, with prevalence estimates broadly consistent with international figures, although the available psychometric documentation is more limited than for instruments in larger linguistic communities [1,11].

Taken together, the contemporary psychometric toolkit for EA is sufficient to support both research and screening, but it remains limited in important respects: the conceptual heterogeneity of the instruments mirrors the unresolved theoretical disputes about the nature of EA, the absence of a true diagnostic instrument constrains clinical application, and the methodological practices of the underlying empirical literature continue to require improvement [6,17,18]. As long as no formal diagnostic category exists, screening instruments such as the EDS-21 and the EAI will continue to serve as the principal pragmatic tools available to the clinician, with the explicit caveat that they should be interpreted as indicators of risk rather than as definitive markers of disorder, and that their results should always be integrated with a comprehensive clinical assessment [6,14,16].

4. Neurobiological underpinnings and pathomechanism

4.1. Dysregulation of the reward system (the mesolimbic pathway): the role of the ventral tegmental area (VTA) and the nucleus accumbens (NAc). Changes in D2 dopaminergic receptor density and the mechanism of cross-tolerance resembling cocaine addiction

The conceptual case for treating exercise addiction (EA) as a behavioural addiction has rested, since its inception, on the assumption that the dysregulated behaviour engages broadly the same neural circuitry that mediates dependence on psychoactive substances [3,5,6]. Central to this assumption is the mesolimbic dopaminergic pathway, which projects from dopaminergic neurons in the ventral tegmental area (VTA) of the midbrain to limbic and prefrontal targets, with the nucleus accumbens (NAc) of the ventral striatum serving as the principal output node [10,20]. The mesolimbic pathway has been characterised in detail in the substance use disorder literature, where it is now widely accepted that addictive drugs converge — albeit through pharmacologically heterogeneous primary mechanisms — on a final common pathway involving acute phasic dopamine release in the NAc, followed by gradual neuroadaptive changes affecting receptor density, post-synaptic signalling and the balance between mesolimbic (motivational) and mesocortical (regulatory) projections [10,20]. The hypothesis that EA recapitulates this trajectory, although still incompletely tested, derives support from a converging — if heterogeneous — body of behavioural, pharmacological and neuroimaging evidence [10,20,22].

Acute aerobic exercise has been shown in animal studies to produce a measurable increase in extracellular dopamine concentrations in striatal regions, including the NAc, with the magnitude of the effect varying with intensity and duration of running and with the individual's habitual fitness level [22]. In humans, the picture is more nuanced: one of the most cited early human imaging studies, using [^11C]raclopride positron emission tomography (PET) to estimate striatal dopamine displacement after 30 minutes of treadmill exercise in healthy volunteers, did not detect a significant change in dopaminergic occupancy, although the protocol did not include measures of subjective reward or euphoria and the duration of activity may have been insufficient to produce detectable mesolimbic engagement [21]. More recent studies have provided indirect evidence by demonstrating that endurance running can elicit increases in plasma anandamide that correlate with positive affect, and that endocannabinoid signalling within the mesolimbic system contributes to dopaminergic activation through disinhibition of VTA neurons via CB1-expressing GABAergic terminals [22]. The latter mechanism, in which endocannabinoids reduce GABAergic inhibition of VTA dopamine neurons and thereby promote dopamine release in the NAc, provides a plausible bridge between the documented endocrine response to aerobic exercise and the behavioural reward that motivates habitual training [22].

A central concept inherited from the substance-dependence literature is that chronic, repeated activation of mesolimbic dopaminergic projections produces adaptive changes that, over time, render the system progressively hypoactive at baseline and progressively hyper-reactive to drug-associated cues [10,20]. The best-characterised of these adaptations is a reduction in the density of striatal D2 dopaminergic receptors, which has been demonstrated by PET in dependent users of cocaine, methamphetamine, alcohol and several other substances, and which has been related both to the severity of dependence and to the deficits in inhibitory control that accompany it [10,20]. Lower striatal D2 availability is, in turn, associated with reduced prefrontal metabolic activity, suggesting that the down-regulation of striatal dopaminergic signalling contributes to the impairment of top-down regulatory mechanisms that ordinarily restrain compulsive behaviour [20]. The hypothesised translation of this mechanism to EA — that is, that chronic compulsive exercise produces a comparable down-regulation of striatal D2 receptors and a parallel weakening of prefrontal inhibitory control — has been frequently proposed in the literature, although it has not yet been directly confirmed in well-controlled human PET studies of affected individuals [10,12]. The available indirect evidence, from neuroimaging studies discussed in detail in section 4.3, is consistent with the broad pattern, but the field would benefit greatly from prospective ligand-based studies that quantify D2 receptor availability in clearly characterised samples of individuals with EA [10].

The notion of cross-tolerance — that is, that adaptation to one reinforcer reduces the response to a different reinforcer that engages the same neural pathway — has been invoked to explain certain clinical observations in EA [3,5]. Several studies have reported that recreational exercisers with elevated scores on the EAI or EDS-21 show higher rates of comorbid substance use, including alcohol, nicotine and stimulants, and conversely that individuals in recovery from substance use disorders frequently develop heightened engagement with exercise that may, in some cases, evolve into a dysregulated pattern [3,5,7]. While these observations are consistent with the cross-tolerance framework, they may also reflect shared personality and psychological vulnerabilities — including high reward sensitivity, impulsivity, perfectionism and difficulties in emotion regulation — rather than a strictly pharmacological substrate [3,19]. A cautious interpretation, supported by most contemporary

commentators, is that the parallels between EA and substance use disorders at the level of mesolimbic dopaminergic functioning are real but should not be over-stated, and that the evidence base remains too limited to make a categorical claim of equivalent neurobiological mechanism [6,10].

A further consideration concerns the developmental dimension. The mesolimbic system, and in particular the balance between subcortical reward circuits and prefrontal regulatory regions, undergoes substantial maturation throughout adolescence and into the third decade of life [12]. Dopaminergic neurons in the VTA exhibit increased phasic activity during adolescence, and the relative immaturity of prefrontal inhibitory mechanisms creates a transient window during which the propensity for reward-driven behaviour is unusually high [12]. This developmental asymmetry has been invoked to explain the elevated vulnerability of adolescents and young adults to a range of behavioural addictions, including problematic gaming, gambling, substance use and possibly EA, and has been used to argue that early identification of dysregulated exercise behaviour in this age range may be of particular clinical importance [12]. Up to 40% of adults with addictive disorders report that the onset of their problematic behaviour occurred between the ages of 15 and 19, a figure that is consistent with the broader literature on developmental vulnerability and that may also apply, at least in part, to EA, although age-stratified epidemiological data on EA specifically remain scarce [12].

The shift from an initially voluntary, impulsive engagement with exercise to a habitual and ultimately compulsive pattern of behaviour is also conceptualised in current models as involving a transition from ventral to dorsal striatal control [20]. Within the substance-use literature, this transition has been demonstrated by meta-analyses of imaging data showing that early-stage compulsive behaviour engages predominantly ventral striatal (NAc) regions associated with hedonic reward and motivation, whereas chronic, established compulsion engages dorsal striatal regions — particularly the caudate and putamen — that mediate habit formation and stimulus–response learning [20]. The dorsal striatum has accordingly emerged as an important focus for neuroimaging studies of EA, with both structural and functional findings suggesting that putaminal and caudate involvement may be implicated in the habitual dimension of compulsive exercise [20]. A multimodal MRI investigation by Schaub and colleagues, comparing 29 individuals with exercise dependence (defined by EDS scores >15 on at least three subscales) with 28 inactive controls, found no overall group differences in striatal grey-matter volume, but did identify a divergent relationship between bilateral putamen volume and the EDS tolerance subscale, with positive correlations observed in controls (left putamen $r = 0.64$; right putamen $r = 0.70$; both $p < 0.001$) but not in the dependent group [20]. Functional connectivity analyses in the same sample further suggested altered coupling between the angular gyrus and bilateral caudate in relation to tolerance, a pattern that the authors interpreted as potentially reflective of habit-based or automatised craving [20]. The findings, while preliminary, support the broader contention that the dorsal striatum may contribute to the maintenance of dysregulated exercise behaviour, possibly through the same habit-formation mechanisms that have been documented in classical addiction research [20].

A final aspect of the mesolimbic dysregulation hypothesis concerns the negative-reinforcement dimension of addiction, in which the addictive behaviour is increasingly motivated not by the pursuit of positive affect but by the avoidance of an aversive withdrawal state [3,19,20]. In the substance-use literature, the negative-reinforcement framework has been associated with neuroadaptive changes in the extended amygdala, including the central nucleus of the amygdala, the bed nucleus of the stria terminalis and the lateral subdivision of the NAc, and with sensitisation of stress-related neuropeptide systems such as corticotropin-releasing factor [20]. The translation of this framework to EA is less well documented but is consistent with the affective phenomenology of the withdrawal syndrome described in section 3.2, in which the dominant features are irritability, anxiety, dysphoria and craving, rather than the pursuit of euphoria as such [3,19]. Neuroimaging data on amygdala involvement in EA remain sparse and inconsistent: the Schaub study did not detect amygdala volume differences between dependent and control groups, and the broader systematic review by Pirwani and Szabo found limited and inconsistent evidence regarding amygdala engagement across the eight existing neuroimaging studies in this area [10,20]. These limitations notwithstanding, the negative-reinforcement framework offers a clinically useful complement to the positive-reinforcement model and may be of particular relevance in the later, more entrenched stages of EA, where exercise is sustained as much by the relief of distress as by the pursuit of reward [3,19].

4.2. The endogenous opioid and endocannabinoid systems: the "runner's high" — from the endorphin hypothesis to anandamide signalling (CB1 receptors)

The euphoric and analgesic states reported during and after sustained aerobic exercise — collectively known as the "runner's high" — have long been considered one of the principal phenomenological gateways through which exercise can acquire reinforcing properties analogous to those of an addictive substance [21,22]. The pursuit of

the runner's high may be both a healthy concomitant of training in committed athletes and, in vulnerable individuals, a motivational substrate that contributes to the development of EA, particularly in its primary form [3,6]. The neurobiological mechanisms underlying the runner's high have been the subject of considerable empirical work, and the dominant interpretation of these states has shifted substantially over the past two decades, from a primary focus on the endogenous opioid system to an increasing emphasis on endocannabinoid signalling, with current accounts integrating both systems within a multi-mediator framework [21,22].

The "endorphin hypothesis", articulated most influentially by Morgan in the mid-1980s, posited that exercise-induced euphoria is mediated by the release of β-endorphins from the pituitary into the systemic circulation, with subsequent action on opioid receptors in the central nervous system [19,21]. The hypothesis was supported initially by observations of up to fivefold elevations in plasma β-endorphin concentrations after sustained exercise, by the reversibility of exercise-induced mood changes and analgesia following administration of the unselective opioid antagonist naloxone, and by reports of withdrawal-like states in habitually exercising animals following naloxone challenge [21]. However, the hypothesis was subjected to serious objections within a few years of its formulation. The principal difficulty is that β-endorphin, the peripheral peptide most prominent in the early literature, crosses the blood–brain barrier only minimally, and elevations in peripheral concentrations are therefore unlikely to account directly for central nervous system effects [21]. The case for an opioid mechanism remained plausible, but its empirical demonstration required direct evidence of central opioid receptor activation rather than peripheral hormonal change [21].

The first compelling direct demonstration of central opioid involvement was provided by Boecker and colleagues in a positron emission tomography study using the non-selective opioidergic ligand [^18F]-fluoroethyl-fluorodiprenorphine ([^18F]FDPN) in ten trained male athletes [21]. Each athlete underwent two PET scans, one after a 24-hour period without exercise and one approximately 30 minutes after completing a two-hour endurance run (mean distance 21.5 ± 4.7 km, mean pace 11.0 km/h, mean heart rate 144 beats per minute) [21]. The principal finding was a significant reduction in ligand binding — interpreted as displacement of the radiotracer by endogenous opioid release — in widespread cortical and subcortical regions, with the most prominent effects observed in the prefrontal and orbitofrontal cortices, the dorsolateral prefrontal cortex, the anterior and posterior cingulate cortices, the insula, the parahippocampal gyrus, the basal ganglia and the cerebellum [21]. Crucially, the perceived intensity of euphoria, measured by visual analogue mood scales, increased significantly after running (from approximately 38/100 at baseline to approximately 73/100 after the run) and was inversely correlated with [^18F]FDPN binding in prefrontal/orbitofrontal cortices, the anterior cingulate cortex, the bilateral insula and parainsular cortex, and temporoparietal regions [21]. The findings provided the first direct evidence of region-specific endogenous opioid release in the human brain in association with exercise-induced euphoria, and gave a firm empirical foundation to the long-standing but previously indirect opioid hypothesis [21].

It is worth emphasising the regional specificity of the Boecker findings, which is consistent with the broader picture from the addiction literature [21]. The frontolimbic regions in which opioid release was concentrated — particularly the orbitofrontal cortex, anterior cingulate and insula — are the same regions that have been repeatedly implicated in the affective processing of substance-related reward, in the regulation of mood and motivation, and in the cue-reactivity component of addictive disorders [10,21]. Activation of μ-opioid receptors in these regions is known to mediate euphoric states, whereas κ-opioid activation tends to produce dysphoria; the ligand used in the Boecker study had broadly equivalent affinity for μ, δ and κ receptors and so did not permit discrimination between subtype-specific contributions [21]. Notably, the study did not detect significant opioid changes in the nucleus accumbens, although this could reflect technical limitations of the ligand and methodology rather than a true absence of accumbens engagement; opioid–dopamine interactions in the NAc are well documented and continue to be regarded as a plausible mechanistic substrate of exercise-induced reward [21].

Parallel work has, however, produced converging evidence for a second neurochemical system that may be at least as important as the opioid pathway in mediating the runner's high — the endogenous cannabinoid (endocannabinoid) system [22]. The endocannabinoid hypothesis is based on the observation that the two principal endocannabinoids, anandamide (AEA) and 2-arachidonoylglycerol (2-AG), are released centrally and peripherally in an activity-dependent manner and act as endogenous ligands at CB1 and CB2 cannabinoid receptors, with CB1 receptors particularly densely expressed in brain regions involved in affective processing, reward and motor control [22]. CB1 receptor activation produces psychological effects — including euphoria, anxiolysis and a generalised sense of well-being — that closely resemble those described by runners, and endocannabinoid signalling within the mesolimbic system produces phasic activation of dopaminergic neurons through disinhibition of GABAergic afferents to the VTA, thereby providing a mechanistic link to the reward

circuitry described in section 4.1 [22]. Endocannabinoids are highly lipophilic and cross the blood–brain barrier readily, which means, in contrast to β-endorphin, that peripheral elevations may plausibly correspond to central availability [22].

Direct empirical support for the endocannabinoid hypothesis was provided by Sparling and colleagues, who reported significant increases in plasma AEA concentrations following moderate-intensity running and cycling at approximately 70–80% of maximum heart rate [22]. Raichlen and colleagues subsequently extended this finding through an evolutionarily informed comparative study, in which they measured plasma AEA and 2-AG concentrations in three species selected for differing degrees of cursoriality: humans, dogs (a habitually cursorial mammal) and ferrets (a non-cursorial mammal) [22]. After 30 minutes of treadmill running at a dynamically similar speed (Froude number ≈ 0.70), humans and dogs both exhibited significant increases in plasma AEA, whereas ferrets did not [22]. Walking trials at lower intensity (Froude number ≈ 0.25) did not produce comparable elevations in either humans or dogs, indicating that endocannabinoid signalling is intensity-dependent rather than locomotion-dependent per se [22]. In humans, the magnitude of the AEA increase was strongly positively correlated with the magnitude of the change in positive affect on the PANAS scale ($r = 0.96$; $p < 0.0001$), an unusually high correlation that was retained even after removal of a potential outlier ($r = 0.77$, $p < 0.05$) [22]. The findings suggest that endocannabinoid signalling, and AEA in particular, may serve as a mediator of exercise-induced positive affect in humans and other cursorial mammals, and that the absence of such signalling in non-cursorial taxa is consistent with the evolutionary hypothesis that aerobic activity in cursorial species is supported by intrinsic neurobiological rewards [22].

The shift from an endorphin-centred to an endocannabinoid-centred account of the runner's high should not be construed as a simple replacement, but rather as an integration [10,21,22]. Both systems appear to be engaged during sustained aerobic exercise, and both project to overlapping frontolimbic and reward-related regions; opioid–endocannabinoid interactions are well documented and are likely to be synergistic in the production of the subjective state [22]. From the standpoint of EA, the relevance of this dual mechanism is that it provides at least two pharmacologically plausible substrates through which the behaviour could acquire reinforcing properties capable of supporting compulsive engagement [3,10,22]. Repeated activation of opioid and endocannabinoid circuits could, in principle, lead to neuroadaptive changes — analogous to those documented after chronic exposure to exogenous opioid or cannabinoid agonists — that would manifest behaviourally as tolerance (the need for progressively more exercise to produce the same subjective effect), withdrawal (negative affective and somatic states on cessation), and an enduring vulnerability to relapse [3,10,21,22].

The systematic review of neuroimaging evidence on EA conducted by Pirwani and Szabo, encompassing eight studies, has further supported a role for opioidergic and endocannabinoid-related mechanisms, particularly through findings in the orbitofrontal cortex, anterior cingulate cortex and inferior frontal gyrus — regions of high opioid receptor expression and significant endocannabinoid signalling [10]. Diffusion tensor imaging studies have additionally identified alterations in white-matter tracts that include the inferior fronto-occipital fasciculus and the cingulum, both of which are known to support functional connectivity between regions that are densely populated by opioid and cannabinoid receptors [10]. These findings, while still preliminary, are broadly consistent with the integrated opioid–endocannabinoid model of exercise-induced reward and provide a candidate neurobiological foundation for the proposed reconceptualisation of EA as a behavioural addiction with identifiable mesolimbic and frontolimbic substrates [10,22].

A further mechanistic consideration concerns the analgesic component of exercise-induced reward. Endogenous opioids and endocannabinoids both contribute to exercise-induced analgesia, acting at central and peripheral sites to inhibit nociceptive transmission [22]. The clinical relevance of this analgesic effect is twofold. First, the reduction in pain perception can directly reinforce continued engagement in high-intensity training, and may contribute to one of the most clinically concerning features of EA, namely the continuation of exercise despite injury [13,14,22]. Second, the analgesic state may mask early warning signs of overuse injury, musculoskeletal damage and overtraining, and may thereby facilitate the kind of progressive escalation that is characteristic of the behavioural trajectory of EA [14,22]. In this respect, the same neurobiological mechanisms that contribute to the beneficial mood effects of exercise in non-pathological contexts may, in vulnerable individuals, contribute to the persistence of an increasingly maladaptive pattern of behaviour [3,22].

4.3. Structural changes and neuroplasticity: grey-matter deficits in the orbitofrontal cortex (OFC) and inferior frontal gyrus (IFG) as the substrate of impaired inhibitory control (cognitive rigidity) and compulsive behaviour

While the discussion of mesolimbic and opioid–endocannabinoid mechanisms in the preceding sections has focused on the reward-related dimension of EA, the addiction framework as a whole — both in substance-related and in behavioural variants — assigns equal importance to the failure of prefrontal regulatory control [10,20]. Within this framework, the persistence of a maladaptive pattern of behaviour in the face of mounting negative consequences is attributed not only to altered reward signalling but also to a progressive impairment in the prefrontal circuits that subserve inhibitory control, decision-making, cognitive flexibility and the prospective evaluation of outcomes [10,20]. The orbitofrontal cortex (OFC) and the inferior frontal gyrus (IFG), together with the anterior cingulate cortex (ACC) and dorsolateral prefrontal cortex, have emerged as the principal anatomical substrates of this regulatory dimension, and findings from contemporary neuroimaging research suggest that EA may be associated with structural and functional alterations in these regions [10,20].

The most consistent structural finding in the existing neuroimaging literature on EA concerns the orbitofrontal cortex [10,20]. A voxel-based morphometric (VBM) study in 86 regular exercisers with EDS-R-defined symptoms of dependence reported that exercise dependence scores were negatively correlated with grey-matter volume in the right OFC, the subgenual cingulate gyrus and the inferior parietal lobe, with OFC volume mediating the relationship between psychological stress and EDS scores [10]. A follow-up analysis from the same group further demonstrated that grey-matter volume in the OFC mediated the relationship between OFC functional activity (as indexed by the amplitude of low-frequency fluctuations) and EDS symptoms, suggesting an integrated structural–functional substrate for the impaired executive control hypothesised to underlie EA [10]. A separate VBM investigation in 130 regular exercisers identified a comparable negative association between right OFC grey-matter volume and exercise addiction scores measured by the EAI, with OFC volume mediating the relationship between motivational ambition and exercise addiction risk [10]. Taken together, these findings provide a moderately consistent picture in which lower OFC volume is associated with greater dysfunctional exercise behaviour, in line with the broader literature implicating the OFC in compulsive disorders more generally [10,20].

The OFC is a critical hub for outcome-based decision-making, the representation of reward value, the inhibition of inappropriate responses and the flexible updating of behavioural strategies in response to changing contingencies [10,20]. Reductions in OFC grey-matter volume have been documented across substance use disorders (notably cocaine, methamphetamine, alcohol and nicotine dependence), in gambling disorder and in internet gaming disorder, and have been interpreted as a structural correlate of the cognitive rigidity and impaired response inhibition that characterise these conditions [10,20]. The replication of this pattern in EA, although based on a small number of studies, supports the broader contention that EA shares neurobiological features with recognised behavioural addictions and reinforces the view that the disorder is anchored, at least in part, in a failure of prefrontal regulatory control over the dysregulated behaviour [10,20].

The inferior frontal gyrus has likewise received significant attention in the EA literature [10,20]. The IFG, particularly in its right hemisphere, is a central node for motor inhibition, attentional control and the flexible adjustment of behaviour to contextual demands [20]. In the multimodal neuroimaging study by Schaub and colleagues, no overall group differences in IFG grey-matter volume were detected between individuals with EDS-defined dependence and inactive controls; however, exploratory slope analyses revealed a significant positive correlation between right IFG volume and the EDS "time" subscale (which measures the amount of time devoted to exercise-related activities) in the dependent group ($r = 0.62$; $p < 0.001$), but not in the controls [20]. The authors interpreted this finding as potentially reflective of a fitness-mediated effect on cognitive functioning, given the known association between higher cardiorespiratory fitness and IFG volume, while noting that a causal direction could not be inferred from a cross-sectional design [20]. The same study identified a significant increase in resting-state functional connectivity between the right IFG and the right superior parietal lobule in the dependent group compared with controls, a pattern that the authors related to the generation and monitoring of motor representations and to the impaired cognitive control over compulsive behaviours observed in obsessive–compulsive disorder [20].

The broader literature on EA neuroimaging, summarised in the systematic review by Pirwani and Szabo, provides a converging picture in which alterations are concentrated in the OFC, anterior cingulate cortex, IFG, amygdala and default mode network [10]. Task-based functional MRI studies have shown that exercise-related visual cues elicit elevated activation in the OFC, ACC, amygdala, hippocampus and medial prefrontal cortex in

individuals with EA, in a pattern that closely resembles the cue-reactivity literature on substance use and behavioural addictions [10]. In a Chinese sample of 55 participants (29 with EA, 26 controls), individuals with EA exhibited lower activation in frontal, parietal and occipital regions — including the medial superior frontal gyrus, inferior frontal gyrus and occipital cortex — in response to both exercise-related and non-exercise visual cues, suggesting a more general blunting of higher-order processing in this group [10]. A resting-state functional MRI study in 110 general exercisers reported that functional connectivity between grey-matter and white-matter networks involving the default mode network was positively associated with both negative perfectionism and exercise dependence, and that this connectivity pattern mediated the relationship between perfectionism and EA [10]. The default mode network — comprising the medial prefrontal cortex, posterior cingulate cortex, precuneus and angular gyrus — is thought to support self-referential thought, introspection and prospective decision-making, and its disruption has been documented in a range of psychiatric conditions including substance use disorders, OCD and major depressive disorder [10,20].

A diffusion tensor imaging (DTI) investigation in 108 regular exercisers exhibiting symptoms of EA reported reductions in fractional anisotropy and quantitative anisotropy in white-matter tracts including the corticospinal tract, superior thalamic radiation, cingulum and inferior fronto-occipital fasciculus, with these alterations negatively associated with exercise dependence and mediating the link with body satisfaction [10]. White-matter integrity in the cingulum and inferior fronto-occipital fasciculus is consistently affected across other behavioural addictions and has been interpreted as a correlate of impaired inter-regional communication between reward and control networks [10]. While the available DTI data on EA are limited in number, they suggest that the disorder may be accompanied by microstructural white-matter alterations that complement the structural and functional grey-matter findings and may help to explain the cognitive rigidity and habitual character of compulsive exercise [10].
The clinical interpretation of the neuroimaging findings remains constrained by a number of methodological limitations [10,20]. The available studies are predominantly cross-sectional, with small to modest sample sizes (typically 20–130 participants), and rely on questionnaire-based identification of EA rather than on structured clinical interviews [10,20]. The use of inactive controls in some studies makes it difficult to disentangle the effects of dependence from the effects of high training volume per se [20]. The replicability of certain key findings — including the OFC volume reduction — across cultural and demographic samples remains to be established, and the field has yet to produce a meta-analytic synthesis of voxel-level effects [10]. These caveats notwithstanding, the available evidence is consistent with a coherent neuroanatomical framework in which EA is associated with structural and functional alterations in frontolimbic and frontoparietal circuits subserving reward processing, executive control, habit formation and emotion regulation, and in which these alterations resemble — at least in broad terms — the patterns observed in recognised behavioural and substance-related addictions [10,20].

A complementary line of evidence concerns clinical and personality correlates of these neural patterns. In the Schaub sample, individuals with EA showed high rates of psychiatric comorbidity, including depression, anxiety, eating disorders, personality disorders and obsessive–compulsive disorders, together with elevated rates of ADHD symptoms and childhood trauma [20]. The personality profile that emerged from the same sample was characterised more by obsessive–compulsive (Cluster C) features than by impulsive (Cluster B) features, leading the authors to suggest that the compulsive rather than impulsive dimension may be most relevant in the identification of EA [20]. This interpretation aligns with neuroimaging findings of fronto-parietal hyperconnectivity, which has been reported in OCD and is thought to underlie inflexible, rule-driven behaviour [20]. From a translational standpoint, this evidence supports a conceptualisation of EA that incorporates obsessive–compulsive features alongside addictive ones, and that views the neurobiological substrate as situated at the interface between the reward circuitry implicated in classical addiction and the prefrontal control circuitry implicated in compulsive disorders [10,20].

Finally, the developing literature on neuroplasticity in EA has begun to consider the bidirectional relationship between exercise behaviour and brain structure [10,20]. Regular aerobic exercise is well established as a promoter of beneficial neuroplastic change, including increased hippocampal volume, enhanced white-matter integrity and improved cognitive function across the lifespan [10]. In moderate and adaptive doses, exercise is therefore expected to support, rather than impair, the structural integrity of frontolimbic networks [10,20]. The findings reviewed above suggest that this beneficial trajectory may be disrupted, or even reversed, in individuals whose engagement with exercise has crossed into a dysregulated, compulsive pattern — particularly when the behaviour is sustained despite injury, sleep deprivation and physiological exhaustion [10,20]. The clinical and translational implication is that EA represents not simply an inversion of the normal pharmacology of exercise reward, but a state in which the same behaviour that normally promotes neural health may, under particular conditions, contribute to the maintenance of pathological circuitry [10,20]. Prospective neuroimaging studies —

ideally combining structural, functional, diffusion and ligand-based modalities, and following clearly characterised samples of affected individuals over time — are needed to disentangle the beneficial from the maladaptive trajectories of brain change in habitual exercisers and to clarify the developmental architecture of EA at the neural level [10,20].

5. Differential diagnosis – psychiatry versus sports medicine

5.1. Primary versus secondary exercise addiction

The distinction between primary and secondary exercise addiction (EA) represents the most clinically important conceptual axis in the differential diagnosis of dysregulated exercise behaviour, and it is the one most likely to determine the appropriate diagnostic formulation, treatment plan and prognosis [3,5,6,16]. The framework, introduced by de Coverley Veale in 1987 and subsequently refined by Bamber and colleagues, separates two phenotypically similar but etiologically distinct conditions on the basis of the motivational substrate that drives the excessive engagement with exercise [16]. In its primary form, EA is conceptualised as a behavioural addiction in its own right, in which exercise itself constitutes the rewarding endpoint, and the individual is motivated by the psychological gratification produced by training, by the release of endogenous opioids and endocannabinoids that mediate the runner's high, and by the avoidance of the withdrawal-related dysphoria that emerges when training is interrupted [3,21,22]. In its secondary form, by contrast, EA is conceptualised as a compensatory or instrumental behaviour that emerges in the context of another psychopathological process — most commonly an eating disorder, body-image disturbance or muscle dysmorphia — in which exercise is used as a means to an end (typically weight control, body shape modification or affect regulation related to body image), rather than being pursued for its intrinsic rewarding properties [5,6,23,24].

The clinical implication of this distinction is far-reaching. In primary EA, the therapeutic focus is on the dysregulation of the exercise behaviour itself, on the emotional and motivational mechanisms that sustain it, and on the cognitive features (cognitive rigidity, perfectionism, intolerance of withdrawal-related distress) that maintain the cycle [3,6,23]. In secondary EA, by contrast, the dysregulated exercise is best understood as a symptom of, or a compensatory strategy within, the primary disorder, and therapeutic effort must accordingly be directed in the first instance towards the underlying eating-disorder, body-image or affective pathology [4,6,24,25]. Treating the exercise behaviour in isolation, in such cases, is unlikely to produce a durable improvement and may, in some circumstances, exacerbate the patient's distress by removing a coping mechanism without addressing the underlying disturbance [25,27]. The diagnostic interview should therefore explicitly probe the motivational structure behind the behaviour, with particular attention to the relationship between exercise and food intake, body weight, body composition, body-image concerns and affective state [16,25].

Several empirical lines of evidence support the validity of the primary–secondary distinction. The qualitative work of Bamber and colleagues, based on semi-structured interviews with 56 adult female exercisers, identified two diagnostic criteria (impaired functioning and withdrawal) that, in combination with the presence or absence of an eating disorder, separated primary from secondary exercise dependence with reasonable face validity [16]. In that sample, ten women met the criteria for exercise dependence; all ten also exhibited eating disorders and were therefore reclassified as displaying secondary rather than primary dependence, a finding that has been cited by some authors as evidence that primary EA may be rare or even non-existent in clinical practice [4,16]. A subsequent systematic review and meta-analysis by Trott and colleagues, specifically designed to estimate the prevalence of EA in the absence of eating-disorder symptomatology, identified 13 eligible studies including 3,635 participants and reported pooled prevalence estimates of 8.1% among general exercisers, 5.0% among amateur competitive athletes and 5.5% among university students, with an overall pooled rate of 6.2% [26]. These figures provide indirect but reasonably robust support for the existence of a population of individuals who fulfil the criteria for EA in the absence of demonstrable eating pathology, and therefore for the persistence of the primary–secondary dichotomy as a clinically useful framework [26].

A further consideration concerns the directionality and temporal sequence of the relationship between EA and eating-disorder symptomatology. In some cases — notably in adolescents with restrictive anorexia nervosa — the onset of compulsive exercise behaviour appears to antedate the development of overt eating-disorder symptoms, leading some authors to suggest that excessive exercise may play an etiological role in the genesis of certain eating disorders, rather than being a purely secondary compensatory behaviour [24,25]. The activity-based anorexia (ABA) animal model provides a mechanistic basis for this possibility, since rodents housed with running wheels and exposed to food restriction reduce their food intake and progressively increase their wheel-running activity, in some cases to the point of starvation and death [25]. In other cases — particularly in adult

patients with bulimia nervosa or with secondary EA arising in the context of body-image preoccupation — the eating-disorder symptomatology clearly antedates the dysregulated exercise behaviour and the latter is best understood as an emergent compensatory strategy [24,25]. The temporal sequence in any given case can be difficult to reconstruct retrospectively, and it is one of the practical reasons for which longitudinal studies of EA are urgently required [6,24].

Some authors have argued that the term "secondary exercise addiction" is itself a misnomer, and that the dysregulated exercise behaviour observed in eating-disorder patients should be conceptualised as "instrumental exercise" rather than as an addiction proper, in order to avoid conceptual confusion between addictive and compensatory phenomena [6,18]. Under this alternative framework, only the primary form would retain the formal label of EA, with the secondary form being subsumed within the diagnostic category of the underlying disorder. The proposal has not yet achieved consensus, but it usefully highlights the clinical importance of distinguishing between exercise pursued as an autonomous reward and exercise pursued as a means to a non-exercise-related goal [6,18]. From the standpoint of a psychiatry trainee, the practical implication is that the diagnostic formulation of every case of dysregulated exercise should explicitly identify the motivational substrate of the behaviour, the presence or absence of eating-disorder or body-image features, and the temporal relationship between the dysregulated exercise and any co-occurring pathology [16,25].

It is also worth noting that the primary–secondary distinction is best understood as a heuristic rather than as a strict dichotomy. In clinical practice, the two forms frequently overlap, and a substantial proportion of patients exhibit mixed features in which a primary addictive dimension co-exists with secondary compensatory features tied to body image, weight control or affect regulation [25]. The work of Cunningham and colleagues, cited in Scharmer et al., suggests that individuals with both EA and elevated eating-disorder pathology often score highly on both addictive and compulsive dimensions of dysregulated exercise, indicating that the two mechanisms may operate concurrently rather than alternatively in the same individual [25]. The implication is that the clinician should resist the temptation to assign the patient to one or the other category prematurely, and should instead aim to characterise the relative weight of the addictive and compulsive components in each individual case [16,25].

5.2. Comorbidity with eating disorders

The relationship between EA and eating disorders is the most extensively documented of all comorbidity patterns in the field, and it constitutes one of the most clinically challenging dimensions of differential diagnosis [4,5,8,24]. The DSM-5 explicitly lists "excessive exercise" — defined as exercise that interferes with important activities, occurs at inappropriate times or settings, or is carried out despite injury or other medical complications — as a diagnostic feature for both bulimia nervosa and the restricting subtype of anorexia nervosa [24]. This nosological positioning reflects the long-standing clinical observation that excessive and compulsive exercise is a core component of the symptomatology of these disorders, frequently antedating, accompanying or following the more overt features of restricted eating, purging or binge-eating [24,25,27].

Prevalence estimates of compulsive or dysregulated exercise behaviour among eating-disorder samples are uniformly high, although they vary as a function of the diagnostic category, the assessment instrument and the population studied [24,25,27]. In adolescents with eating disorders, the prevalence of compulsive exercise has been reported in the range of approximately 17–85%, and in adults with eating disorders in the range of 32–80% [27]. Among individuals with anorexia nervosa, up to 80% have been reported to exhibit problematic exercise behaviours, with some authors arguing that compulsive exercise may be a near-universal feature of the restricting subtype [25,27]. Among individuals with bulimia nervosa, prevalence estimates of approximately 40% are typical, although they vary considerably between studies [25]. A meta-analysis by Trott and colleagues estimated that EA occurs more than three-and-a-half times more frequently as a comorbidity of an eating disorder than in individuals without eating disorders, a finding that has been interpreted as evidence both for the close phenomenological link between the two conditions and for the likelihood that a substantial proportion of EA reported in the general literature reflects secondary or instrumental exercise [6,26].

The relationship between morbid exercise behaviour and specific dimensions of eating-disorder pathology has been further clarified by the meta-analysis of Alcaraz-Ibáñez and colleagues, which synthesised 66 studies comprising 135 effect sizes and a total of 21,816 participants [24]. The results documented small to medium positive correlations between morbid exercise behaviour and dimensions of eating-disorder symptomatology: $r = 0.19$ for bulimic symptoms, $r = 0.28–0.41$ for body and eating concerns, $r = 0.35$ for overall eating-disorder symptoms and $r = 0.42$ for dietary restraint [24]. Larger effect sizes were observed in clinical, younger and thinner samples, in samples assessed with the Compulsive Exercise Test (CET) rather than with other instruments, and in female samples for the specific outcome of dietary restraint [24]. The findings suggest that

the EA–eating disorder relationship is particularly tight when the exercise behaviour is conceptualised as compulsive rather than as dependent, and when the eating-disorder outcome is dietary restraint rather than bulimic or binge-eating features [24,25].

The conceptual distinction between compulsive exercise and exercise dependence in the context of eating disorders has been carefully examined in the work of Scharmer and colleagues, who compared the relative associations of the CET and the EDS with eating-disorder pathology, as measured by the Eating Disorder Examination Questionnaire (EDE-Q), in 235 undergraduate participants with elevated eating-disorder symptoms [25]. The compulsive qualities of exercise — particularly exercise aimed at controlling shape and weight and at avoiding negative affect — were more strongly associated with eating-disorder pathology than were the qualities of exercise dependence, suggesting that the compulsive framework may be more appropriate than the addictive framework for describing problematic exercise in the eating-disorder context [25]. A subsequent Delphi study among international experts in eating-disorder treatment found that approximately 60% preferred the term "compulsive exercise" over "exercise addiction" or "exercise dependence" when describing the dysregulated exercise behaviour observed in their patients, although consensus thresholds were not reached [27]. The shift in terminology reflects a broader theoretical movement in the field, which conceptualises the secondary form of dysregulated exercise as a compulsive behaviour driven by negative reinforcement (avoidance of anxiety associated with not exercising, with weight gain or with body-shape changes) rather than as an addictive behaviour driven by positive reinforcement (pursuit of the rewarding properties of exercise) [25,27].

A particularly important sub-form of the relationship between EA and eating disorders concerns muscle dysmorphia, also known historically as "bigorexia" or "reverse anorexia" [27]. Muscle dysmorphia, included in the DSM-5 as a specifier of body dysmorphic disorder, is characterised by an intense preoccupation with the perception that one's musculature is insufficient, often despite objective evidence to the contrary, and by the corresponding pursuit of muscular development through compulsive resistance training, dietary regimens and, in some cases, the use of anabolic substances [27]. The condition is particularly prevalent among male bodybuilders and resistance athletes, and it presents a number of clinical features that are directly relevant to the differential diagnosis of EA. In a study cited by Martenstyn and colleagues, men with muscle dysmorphia reported significantly greater compulsive exercise scores than male gym-goers without muscle dysmorphia (Cohen's d = 2.6), and exhibited compulsive exercise scores on three subscales of the CET (avoidance and rule-driven behaviour, exercise rigidity, and mood improvement) that were comparable to those of men with anorexia nervosa [27]. Furthermore, approximately 22–29% of men with muscle dysmorphia have been reported to have a prior history of anorexia nervosa or another eating disorder, supporting the view that the two conditions share substantial pathophysiological and clinical territory [27].

The differential-diagnostic implications of this overlap are several. First, in any male patient presenting with compulsive resistance training, the clinician should screen explicitly for the cognitive and affective features of muscle dysmorphia, including persistent dissatisfaction with body composition despite objectively adequate musculature, rigid adherence to dietary regimens, distress and avoidance behaviour in situations that expose the body to scrutiny, and a preoccupation with body image that is disproportionate to the objective findings [27]. Second, in any female patient presenting with compulsive endurance exercise, the clinician should screen for the cognitive and behavioural features of anorexia nervosa and bulimia nervosa, including restrictive eating patterns, fear of weight gain, body-image disturbance, purging behaviours and amenorrhoea [16,24,25]. Third, in any patient presenting with compulsive exercise and concurrent low body weight, the clinician should explicitly consider the diagnosis of anorexia nervosa, restricting subtype, and should investigate the temporal sequence of the exercise and the eating disturbance [16,24,25,27]. Quality-of-life data suggest that the clinical burden of these comorbid presentations is substantial: men with muscle dysmorphia have reported quality-of-life scores 1.7 to 2.6 standard deviations below community norms, and are reported to be almost three times more likely to have attempted suicide than men with body dysmorphic disorder unrelated to muscularity [27].

A further point of clinical importance concerns the prognostic implications of comorbid compulsive exercise in eating-disorder samples [25,27]. Compulsive exercise has been associated with longer inpatient hospitalisations, lower quality of life, greater suicidal behaviour, increased risk of relapse and poorer overall treatment outcome in patients with eating disorders [25,27]. These findings reinforce the clinical importance of explicitly screening for and addressing compulsive exercise in any patient presenting with an eating disorder, and of incorporating exercise-focused treatment components into eating-disorder treatment programmes [25,27]. Treatment approaches that have been examined in this context include cognitive-behavioural interventions targeting the compulsive features of exercise, exposure and response prevention techniques aimed at reducing the anxiety associated with not exercising, psychoeducation, structured and supervised exercise components, and combined approaches that integrate exercise rehabilitation into the broader eating-disorder treatment plan [25,27]. Earlier

models that recommended complete abstinence from exercise in patients with anorexia nervosa have largely been superseded by more nuanced approaches that advocate the gradual reintroduction of safe, structured and supervised exercise, with evidence suggesting that such reintroduction does not interfere with weight restoration and may, in some cases, support it [27].

Finally, the high prevalence of EA in eating-disorder samples — together with the meta-analytic finding that EA occurs three-and-a-half times more frequently in this population than in non-eating-disorder samples — implies that any prevalence figure for EA derived from samples that have not been carefully screened for eating-disorder symptomatology is likely to overestimate the true prevalence of primary EA and to conflate it with secondary or instrumental exercise [6,24,26]. This consideration reinforces the call for systematic measurement of eating-disorder symptomatology in EA research and supports the methodological recommendation that all psychometric assessments of EA should be supplemented by validated screening for eating disorders, ideally followed by clinical interview [6,24,26].

5.3. Exercise addiction and obsessive–compulsive disorder

The differential diagnosis between EA and obsessive–compulsive disorder (OCD) represents another important axis of clinical reasoning, and one in which the distinction is conceptually clear-cut at the phenomenological level but frequently blurred in clinical practice [4,20,23]. Both conditions involve repetitive, rule-driven behaviour that is persistent in the face of negative consequences, and both are accompanied by significant subjective distress when the behaviour is interrupted [4,23]. The pivotal distinction, in the classical psychiatric formulation, lies in the ego-syntonic versus ego-dystonic quality of the behaviour and the cognitive content that accompanies it [4,23].

OCD is characterised by ego-dystonic obsessions — recurrent, intrusive, unwanted thoughts, images or impulses that the individual recognises as irrational, distressing and contrary to their values — and by compulsions, which are repetitive behaviours or mental acts that the individual feels driven to perform in response to the obsessions, typically with the aim of preventing a feared outcome or reducing the anxiety produced by the obsession [4,23]. The key clinical feature is that the obsessions are experienced as alien, unwanted and distressing, and the compulsions are typically performed reluctantly, in a state of inner conflict, and with no intrinsic pleasure [4,23]. EA, by contrast, is typically described as initially ego-syntonic: the affected individual identifies with the exercise behaviour, derives positive affect from it (at least in the earlier phases), and views the behaviour as consistent with their values, goals and self-image [3,4,23]. Only in the more advanced stages of EA — when the consequences of the behaviour have accumulated and the individual has become aware of the disproportion between the behaviour and its costs — does an ego-dystonic dimension typically emerge [4,23].

This phenomenological distinction has important practical implications. In EA, the affected individual is often resistant to recognising the behaviour as pathological and may experience the clinician's suggestion of dysregulation as alien or even threatening, particularly when the exercise behaviour is socially valued and reinforced by peers, coaches, training communities and broader cultural narratives that valorise athletic discipline [3,5,23]. In OCD, by contrast, the affected individual is generally aware of the irrationality of the behaviour and frequently seeks help, although shame and stigma may delay disclosure [4]. The distinction also has implications for treatment: while OCD typically responds to a combination of exposure and response prevention (ERP) and serotonergic pharmacotherapy, EA generally requires a different therapeutic approach centred on motivational interviewing, cognitive restructuring and the gradual modification of the affective and cognitive substrate of the exercise behaviour, although ERP techniques have been adapted with some success to the EA context, particularly when the behaviour has acquired a compulsive flavour [23,27].

The boundaries between EA and OCD are, however, considerably more porous than the classical phenomenological distinction would suggest. Several studies have documented that compulsive exercisers exhibit elevated rates of obsessive-compulsive personality traits, and in some samples meet the diagnostic criteria for obsessive-compulsive personality disorder or for OCD itself [20,23]. In the multimodal MRI study by Schaub and colleagues, the personality profile of individuals with EA was characterised more by obsessive-compulsive (Cluster C) than by impulsive (Cluster B) features, leading the authors to suggest that the compulsive dimension may be more relevant than the impulsive dimension in the identification of EA [20]. The same study identified increased fronto-parietal resting-state functional connectivity in individuals with EA, a pattern that has been reported in OCD and that is thought to reflect a lack of cognitive control over compulsive behaviours [20]. Genetic analyses cited by Scharmer and colleagues have further suggested that polygenic risk scores for both anorexia nervosa and OCD predict compulsive exercise in epidemiological adolescent samples, supporting the view that the relationship between EA and OCD may have biological underpinnings [25].

Several authors have therefore proposed that EA may be best situated within the broader obsessive-compulsive and impulsive spectrum of behavioural addictions, rather than within a strictly addiction-focused taxonomy [6,20]. Under this formulation, the disorder occupies an intermediate position between classical OCD (in which the behaviour is ego-dystonic and the cognitive content is dominated by intrusive thoughts) and classical addiction (in which the behaviour is initially ego-syntonic and the cognitive content is dominated by craving and the pursuit of reward) [6,20]. The clinical phenotype of an individual patient may, at any given time, lie closer to one or the other pole of this continuum, and the clinician should be alert to the possibility of a mixed presentation in which addictive and compulsive features co-exist [4,6,20,23].

From a practical standpoint, the differential diagnosis between EA and OCD should be guided by several considerations. First, the cognitive content of the behaviour should be carefully elicited: in OCD, the patient typically reports specific intrusive thoughts or feared outcomes that the behaviour is intended to prevent (for example, fears of contamination, harm or moral transgression), whereas in EA the cognitive content is more typically organised around exercise itself, its perceived necessity, the avoidance of withdrawal-related distress and concerns about body image or performance [4,16,23]. Second, the ego-syntonic versus ego-dystonic quality of the behaviour should be assessed: in OCD, the patient typically experiences the behaviour as unwanted and alien, whereas in EA the behaviour is initially experienced as desirable and consistent with the patient's identity [4,23]. Third, the presence of obsessions, of formal compulsions involving non-exercise behaviour (such as washing, checking or ordering) and of other OCD-spectrum symptoms should be assessed by structured interview, since the presence of these features may indicate that the diagnosis is OCD rather than EA, even when compulsive exercise is among the clinical manifestations [4,23,25]. Fourth, the temporal sequence of the disorders should be considered: in some cases, OCD antedates the development of compulsive exercise and the latter may be best understood as a symptom of the former; in others, EA develops in the absence of a primary OCD diagnosis but acquires compulsive features over time [20,23,25].

5.4. The overtraining syndrome (OTS) versus exercise addiction

The differential diagnosis between EA and the overtraining syndrome (OTS) is among the most challenging in sports medicine and psychiatry, because the two conditions share substantial phenomenological overlap while differing fundamentally in their etiological substrate [28,29,30]. The OTS is defined by the European College of Sport Science as a maladaptive response to prolonged, excessive training without adequate recovery, characterised by a long-lasting performance decrement (typically of more than two months' duration), severe symptomatology that may include affective, cognitive, neuroendocrine, autonomic and immunological dysregulation, and the presence of an additional stressor that is not adequately explained by another disease process [28,29]. The condition sits at the end of a continuum that begins with functional overreaching (a short-term performance decrement followed by supercompensation after rest), passes through non-functional overreaching (a longer-term performance decrement with eventual full recovery), and culminates in OTS itself (a prolonged, often months-long decrement that may compromise the athlete's career) [29].

The pathophysiology of OTS is multifactorial and incompletely understood [28,29,30]. The principal hypotheses include the dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis and of the hypothalamic–pituitary–gonadal axis, with disturbances in cortisol, testosterone and the testosterone/cortisol ratio; activation of pro-inflammatory cytokine pathways including IL-1β, IL-6 and TNF-α; alterations in monoaminergic neurotransmission, particularly serotonergic and dopaminergic; depletion of branched-chain amino acids and glutamine; oxidative stress; autonomic nervous system dysregulation, typically with parasympathetic predominance in aerobic athletes and sympathetic predominance in anaerobic athletes; and central fatigue mediated by increased serotonergic activity in the brain [28,29,30]. The EROS programme has identified more than 45 candidate biomarkers of OTS, none of which is universally diagnostic in isolation, and contemporary diagnostic frameworks rely on multidomain assessment combining hormonal, immunological, autonomic, performance and psychometric markers [28]. The estimated lifetime prevalence of OTS or related non-functional overreaching is substantial: up to 64% of male and 60% of female elite athletes are reported to experience at least one episode during their career, with rates varying considerably across sports and competition levels [28].

The clinical presentation of OTS overlaps with that of EA in several important respects [28,29,30]. Both conditions may be accompanied by mood disturbances (depression, irritability, anxiety), sleep disturbances (insomnia, non-restorative sleep), fatigue, anhedonia, loss of motivation and impaired concentration [3,19,29]. Both may be associated with continued training despite the emergence of injury or physiological dysfunction [3,29]. Both may produce a deterioration in social, occupational and family functioning [3,29]. The key distinction, however, lies in the underlying mechanism and in the relationship between the training load and the

behavioural drive [29,30]. In OTS, the dysfunction is best understood as a physiological exhaustion of the HPA axis and of associated regulatory systems in response to a training load that exceeds the individual's capacity for recovery; the affected athlete may be ostensibly committed and rational in their approach to training, and the breakdown is essentially a quantitative mismatch between load and recovery rather than a qualitative dysregulation of the behavioural drive [29,30]. In EA, by contrast, the dysfunction is qualitative: the individual has lost control over the exercise behaviour and continues to train compulsively despite mounting personal cost, with the behavioural drive itself constituting the core problem rather than the training load per se [3,6,23].

This conceptual distinction has important practical implications [29,30]. In OTS, the appropriate management is rest, recovery, nutritional rehabilitation, sleep optimisation and the gradual reintroduction of training under careful supervision; the athlete is generally willing to accept these interventions, although the recovery period may be prolonged and may, in severe cases, end the athletic career [29]. In EA, by contrast, rest and recovery are unlikely to be accepted by the patient — indeed, the imposition of rest is itself a precipitant of the withdrawal syndrome that drives the compulsive return to exercise — and the appropriate management focuses on cognitive and behavioural interventions targeting the dysregulated drive itself, with rest being introduced gradually as part of a structured therapeutic plan [3,6,23,27]. The distinction between OTS as "a training error" and EA as "a psychopathological symptom" is therefore not merely semantic; it has direct implications for the choice of treatment and for the prognostic outlook [29,30].

The two conditions may also co-exist [28,29,30]. An individual with EA may, over time, develop the physiological features of OTS as a consequence of their compulsive training; equally, an athlete with OTS may, in some cases, develop secondary features of EA as the inability to train normally produces affective dysregulation that drives further attempts to push through the symptoms [29,30]. The careful disentanglement of these scenarios requires a comprehensive assessment that integrates physiological markers (HPA axis function, T/C ratio, heart-rate variability, cytokine profile, sleep architecture), performance data, psychometric instruments for both OTS (such as the RESTQ-Sport) and EA (such as the EDS-21 or EAI), and clinical interview [28,29,30]. The neurological dimension of OTS, particularly as described in the recent narrative review on competitive dance by Ignatowicz and colleagues, includes HPA-axis dysregulation, autonomic imbalance, monoaminergic depletion, neuroinflammation, suppressed brain-derived neurotrophic factor (BDNF) expression, impaired motor memory consolidation, cerebellar and basal-ganglia fatigue, and proprioceptive disruption — features that may further complicate the clinical picture and require dedicated assessment [30].

A particularly informative feature in the differential diagnosis between OTS and EA concerns the affective response to enforced rest [3,29,30]. In OTS, the athlete typically welcomes rest, at least after the initial period of denial, and the recovery of mood and energy coincides with the recovery of physiological function over the course of weeks to months; in EA, by contrast, enforced rest produces an acute withdrawal syndrome characterised by irritability, anxiety, depression, sleep disturbance and intense craving for exercise, and the affective deterioration tends to drive a premature return to training rather than supporting recovery [3,19,29]. The careful elicitation of the affective and behavioural response to imposed rest is therefore one of the most useful clinical manoeuvres in distinguishing between the two conditions, and may, in some cases, constitute the most reliable diagnostic test available [3,29].

5.5. Elite athletes: "commitment versus addiction" and the pitfalls of psychometric assessment

The differential diagnosis of EA among elite and highly committed athletes constitutes perhaps the most clinically delicate of all the distinctions discussed in this chapter, because the behavioural phenotype of high-performance athletic engagement closely resembles, in many quantitative respects, the phenotype of EA, and because conventional psychometric instruments may not reliably discriminate between the two [5,6,14,31]. The high-performance athlete typically devotes substantial daily time to training, may exhibit features that resemble tolerance (progressive escalation of training load) and withdrawal (irritability and dysphoria when training is interrupted), may continue to train through minor injuries or illness, and may organise much of their personal, social and professional life around training and competition [5,6,14]. These features, however, are normative within the elite-athletic context and do not, in themselves, constitute evidence of psychopathology [5,6].

The conceptual distinction at issue is that between commitment and addiction. Commitment denotes a high level of investment in exercise that is rationally motivated by performance, identity or personal meaning, is integrated within a broader functional life, and is responsive to circumstance — including the willingness to taper or interrupt training for competition strategy, injury management or family obligations [5,6,14]. Addiction, by contrast, denotes a loss of control over the exercise behaviour, a persistence of the behaviour in spite of mounting personal cost, and an emotional and motivational disturbance that is qualitatively different from the

engagement of the healthy committed athlete [3,5,6]. The key clinical questions, in distinguishing commitment from addiction, concern the flexibility of the behaviour, the affective response to circumstances that require its modification, the presence of intrapsychic conflict, the impact of the behaviour on other domains of functioning, and the temporal continuity of the engagement after the conclusion of the competitive career [5,6,14].

Psychometric instruments are, unfortunately, poorly suited to this discrimination [5,6,14,31]. The EDS-21 and the EAI generate dimensional risk indices that are sensitive to training volume and intensity, with the consequence that highly committed athletes — particularly endurance athletes — frequently score in the "at-risk" range without exhibiting the clinical features of EA [5,6,14]. The systematic review by Di Lodovico and colleagues demonstrated that endurance athletes exhibit the highest reported rates of "at-risk" classification on the EAI (14.2%), followed by ball-game players (10.4%), fitness-centre attendees (8.2%), power-discipline athletes (6.4%) and the general population (3.0%), with substantial discrepancies between the EAI and the EDS in the same populations [31]. Among elite competitive athletes, the prevalence of at-risk classification has been reported in the range of 7.6–41% across diverse sports [5,6]. These figures plainly include a substantial proportion of false positives — that is, of athletes whose elevated scores reflect their level of commitment rather than the presence of dysregulation [6,14].

The Granziol study of the EAI-3 explicitly addressed this concern by including a substantial subgroup of professional athletes (approximately 23% of the international sample of 1,931 exercisers) and by calculating preliminary cut-off scores separately for professional athletes and for general exercisers, in recognition of the fact that the same scores may have different clinical meaning in the two populations [14]. The recent literature has correspondingly stressed the importance of supplementing questionnaire-based assessment with structured clinical interview, with explicit elicitation of the cognitive, affective and behavioural features of the engagement, and with collateral information from coaches, training partners and family members [6,14]. A high EDS-21 or EAI score in an elite athlete should be regarded not as a diagnosis but as a flag for further evaluation, with the clinical decision turning on the additional information gathered through interview and observation [6,14].

Several specific features may assist in distinguishing committed elite engagement from EA in this population [3,5,6,14,16]. First, the relationship between training and performance goals: in the committed athlete, the training programme is structured around explicit performance objectives, is responsive to coaching input and to physiological feedback, and includes deliberate periods of tapering and recovery; in EA, the training behaviour is increasingly autonomous of performance goals, may persist beyond the conclusion of the competitive season, and is resistant to tapering or recovery [3,5,6]. Second, the affective response to enforced rest: the committed athlete may experience some frustration with imposed rest but generally tolerates it once its rationale is understood, whereas the addicted exerciser exhibits the disproportionate affective deterioration described in section 3.2 [3,6,14]. Third, the impact on other domains of functioning: the committed athlete typically maintains integrated relationships, occupational engagement and broader interests, whereas the addicted exerciser exhibits progressive narrowing of life domains around training [3,5,16]. Fourth, the response to injury: the committed athlete typically accepts the necessary modifications to training and engages constructively with rehabilitation, whereas the addicted exerciser tends to train through injury, conceal symptoms from healthcare professionals and resist appropriate medical advice [3,14]. Fifth, the cognitive content of the engagement: the committed athlete typically articulates clear performance and personal meaning around their engagement, whereas the addicted exerciser often describes the behaviour in terms of compulsion, of avoiding withdrawal-related distress, or of an inability to imagine life without training [3,6,16].

A further consideration relates to the post-career trajectory [5]. The differentiation between commitment and addiction is often clarified after the conclusion of the competitive career: the formerly committed athlete typically modulates their engagement with exercise in accordance with new life circumstances, whereas the formerly addicted athlete may continue to exhibit dysregulated exercise behaviour even after the disappearance of the original performance context, often with increasing distress as the behaviour loses its socially valid justification [5]. Retrospective and prospective studies of retired athletes have identified subgroups in whom the dysregulated pattern persists or even intensifies after retirement, sometimes in association with depressive features, anxiety and difficulties in emotion regulation [3,6]. The careful follow-up of elite athletes during the transition out of competitive sport may therefore constitute one of the most useful clinical contexts in which the boundary between commitment and addiction can be empirically demonstrated [5,6].

It is finally worth noting that the relationship between elite sport and EA is not purely a diagnostic challenge but also a public-health and ethical one [5,12]. The cultural narratives surrounding elite athletic achievement valorise extreme commitment, sacrifice and self-discipline, and may implicitly normalise — or even reward — exercise behaviours that, in a different context, would be recognised as dysregulated [3,5,12]. Coaches, trainers and team

physicians occupy a privileged position in identifying the early signs of EA in elite athletes, and may also serve as conduits for the development of dysregulated patterns when training cultures emphasise volume and intensity without adequate attention to recovery, mental health and personal life balance [12,29,30]. The integration of mental-health awareness into elite sport, the explicit training of coaches and team physicians in the recognition of EA, and the development of structured screening and intervention pathways for athletes are increasingly recognised as priorities in contemporary sport medicine [5,12,29,30].

6. Current therapeutic perspectives

The therapeutic landscape for exercise addiction (EA) is shaped by two structural features of the field: the absence of a formally recognised diagnostic category, and the consequent absence of evidence-based treatment guidelines that have been developed and validated specifically for this condition [6,23,32]. In practice, the available therapeutic strategies have been derived either from the broader literature on behavioural and substance-related addictions, from the clinical experience accumulated in the management of eating disorders and obsessive–compulsive spectrum conditions, or from the small number of case reports and pilot studies that have specifically addressed EA [23,27,32]. The evidence base is therefore preliminary, and the recommendations that follow should be understood as reflecting a clinical orientation rather than a settled standard of care [32].

A critical preliminary consideration concerns the choice of therapeutic target. As discussed in section 5.1, the differential diagnosis between primary and secondary EA is essential at this stage, since the therapeutic priorities differ substantially between the two forms [4,6,32]. In secondary EA, in which the dysregulated exercise behaviour emerges in the context of an eating disorder, muscle dysmorphia, an affective disorder or an obsessive–compulsive disorder, the management of the underlying condition should generally be prioritised, with the dysregulated exercise behaviour addressed as part of the comprehensive treatment plan rather than as a free-standing target [25,27,32]. In primary EA, by contrast, in which the compulsive exercise behaviour constitutes the central problem, the therapeutic effort can be directed more directly at the cognitive, affective and behavioural mechanisms that sustain the dysregulation [23,32]. A further preliminary consideration concerns the recognition that complete abstinence from exercise is generally neither feasible nor desirable, given the substantial health benefits of moderate physical activity; the contemporary therapeutic objective is the restoration of a controlled, healthy and balanced engagement with exercise rather than the elimination of exercise from the patient's life [23,27,32].

6.1. Psychotherapy

6.1.1. Cognitive–behavioural therapy

Cognitive–behavioural therapy (CBT), in its various adaptations, has emerged as the most consistently recommended psychotherapeutic intervention for EA, although the empirical evidence supporting its effectiveness is derived mainly from small studies, case reports and extrapolation from related behavioural addictions rather than from large-scale randomised controlled trials [23,27,32]. The conceptual foundation of CBT for EA is the proposition, originally articulated by Beck and his collaborators, that psychological distress and maladaptive behaviour are sustained by dysfunctional cognitive processes, including negative automatic thoughts, cognitive distortions (overgeneralisation, catastrophising, dichotomous thinking) and maladaptive core beliefs about the self, others and the world [32]. Applied to EA, this framework targets distorted beliefs about physical activity — typically including the belief that exercise is essential for self-worth, that any reduction in training will lead to catastrophic deterioration in body composition or performance, and that the affective distress associated with missed sessions is intolerable — and seeks to replace them with more balanced, flexible and reality-oriented interpretations [23,32].

The structure of a typical CBT intervention for EA involves several phases [32]. The initial phase comprises psychoeducation about the nature of EA, the differentiation between healthy and problematic exercise, the physiological and psychological consequences of compulsive engagement, and the rationale for the proposed treatment [32]. The next phase focuses on the identification of the cognitive distortions, automatic thoughts and core beliefs that maintain the dysregulated behaviour, frequently through the use of thought diaries, behavioural experiments and Socratic questioning [32]. Subsequent phases involve cognitive restructuring, in which the identified distortions are systematically challenged and revised, and behavioural experiments designed to test the validity of dysfunctional beliefs in a graded and controlled manner [23,32]. A particularly important behavioural component is the gradual modification of the exercise schedule itself, with structured reductions in training volume, the deliberate introduction of rest periods and the planned re-engagement with non-exercise activities that have been neglected as the addiction has progressed [23,32].

Several specific therapeutic strategies have been proposed within the broader CBT framework. The approach articulated by Adams and colleagues emphasises the identification and interruption of the compulsive behaviour through supportive individual psychotherapy, the engagement of the patient in an understanding of the health benefits and importance of moderation, the empowerment of the patient to develop a self-management strategy, the understanding of the organisation of the patient's defence structure, the gradual modification of the psychological defences underlying the compulsion, the unlinking of the compulsion from process-specific triggers, and the rebuilding of coping behaviours and support systems [32]. A complementary approach, articulated by Szabo and Demetrovics, places particular emphasis on the recognition and acknowledgement by the patient that the symptoms are causing problems, on the identification of the stage and severity of the problem, on psychoeducation about the value of exercise and the risks of its morbid practice, on the introduction of attractive alternatives that complement rather than replace the habitual exercise behaviour, on the reinforcement and modelling of pleasure in alternative activities, on the use of a structured "balance sheet" of gains and losses, on goal-setting to amplify and consolidate gains, on the regular evaluation of feeling states and behavioural change, and on the generation of social support for the new pattern [32].

Empirical evidence for the effectiveness of CBT in EA remains, as noted, limited [23,32]. A small number of case reports and case series have documented favourable outcomes in individual patients, but no large-scale randomised controlled trials have, to date, been conducted [23,27,32]. The Cochrane and PROSPERO databases continue to register ongoing systematic reviews and protocols for treatment trials, including a recent protocol for a systematic review of treatments for compulsive exercise in eating disorders and muscle dysmorphia that explicitly sets out to identify the most efficacious treatment components [27]. Within the broader literature on behavioural addictions, CBT has demonstrated robust effectiveness in gambling disorder, internet gaming disorder, problematic internet use and other related conditions, providing a degree of indirect support for its application to EA [23,32]. The extension of motivational interviewing techniques, which have been validated in substance use disorders and other behavioural addictions, has also been advocated for EA, particularly in the early phases of treatment in which the patient may not yet have acknowledged the problematic nature of the behaviour [23,32].

6.1.2. Dialectical–behavioural therapy and affect regulation

Dialectical–behavioural therapy (DBT), developed originally by Linehan for the treatment of borderline personality disorder and chronic suicidality, has been extended to a range of conditions in which affect regulation, distress tolerance and interpersonal effectiveness are central therapeutic targets [23,25,32]. The relevance of DBT to EA derives from the increasing recognition that compulsive exercise behaviour is frequently sustained by deficits in emotion regulation, by intolerance of affective distress (particularly the dysphoria associated with missed exercise sessions) and by the use of exercise as a maladaptive affect-regulatory strategy [25]. Within the DBT framework, the dysregulated exercise behaviour is conceptualised as a behavioural attempt to manage internal states that the individual lacks the skills to tolerate, and the therapeutic objective is the cultivation of alternative, more adaptive regulatory strategies [25,32].

The principal DBT modules — mindfulness (further discussed in section 6.1.3), distress tolerance, emotion regulation and interpersonal effectiveness — each map onto specific features of EA. Distress-tolerance skills are directly relevant to the management of the affective dysphoria that emerges during enforced rest or during attempts to reduce training volume, and may help the patient to remain with the unpleasant affective state without resorting to compulsive exercise as a means of relief [23,25,32]. Emotion-regulation skills support the broader recognition, labelling and modulation of affective states, and are particularly important in patients in whom the dysregulated exercise behaviour is closely tied to anxiety, depression or affect-regulatory difficulties [25,32]. Interpersonal-effectiveness skills support the rebuilding of social relationships that may have been impoverished by the progressive narrowing of the patient's life around training [25]. Mindfulness skills, finally, provide a foundation for the cultivation of non-judgemental awareness of internal states, which may help the patient to disengage from the automatic cycle linking affective distress to compulsive exercise [32].

Empirical evidence for the application of DBT to EA is currently sparse, with most of the available support derived from related conditions such as eating disorders, substance use disorders and impulsive behaviours [25,32]. A systematic review by Martenstyn and colleagues, registered in the protocol stage, plans to evaluate the effectiveness of DBT and other psychotherapeutic interventions for compulsive exercise in eating disorders and muscle dysmorphia [27]. The available case-based evidence suggests that DBT-informed approaches may be particularly useful in patients with EA who exhibit prominent emotion-regulation difficulties, who present with

comorbid borderline personality features, or in whom the dysregulated exercise behaviour is part of a broader pattern of impulsive or self-injurious behaviour [25,32].

6.1.3. Mindfulness-based interventions and acceptance-based approaches

Mindfulness-based interventions, including mindfulness-based stress reduction (MBSR), mindfulness-based cognitive therapy (MBCT) and acceptance and commitment therapy (ACT), have been increasingly investigated as candidate interventions for behavioural addictions in general and for EA in particular [23,32]. The conceptual basis of these approaches lies in the cultivation of non-judgemental, present-moment awareness of internal experience, with the explicit aim of decoupling automatic behavioural responses from their cognitive and affective triggers [32]. Mindfulness has been associated with stress reduction, lower anxiety levels, enhanced emotion regulation, improvement in self-control and increased cognitive flexibility — all of which are directly relevant to the cognitive and affective substrate of EA [32].

A particularly suggestive case report cited by Janowiak and colleagues documented the application of a mindfulness intervention in a patient with EA, in which the patient was instructed to practise mindfulness meditation three times a day for 15 minutes each session, focusing on the breath and observing the unpleasant somatic sensations of withdrawal in a non-judgemental manner without emotional reaction [32]. After eight weeks of practice, the patient's EAI score had decreased from 28 at admission to 6, indicating an asymptomatic profile [32]. While the evidence base remains limited to single-case reports and small pilot studies, the result is consistent with the broader literature documenting the effectiveness of mindfulness-based interventions in related conditions, including problematic internet pornography viewing, substance use disorders and problematic smartphone use [32].

Acceptance and commitment therapy (ACT), in particular, has been advocated as a candidate approach because of its specific focus on the acceptance of difficult internal experiences (rather than their avoidance), on the clarification of personal values and on committed action consistent with those values, even in the presence of distress [32]. The application of ACT to EA would, in principle, involve the cultivation of acceptance of the affective and somatic discomfort associated with reduced training, the clarification of personal values that extend beyond the exercise behaviour (relationships, occupational engagement, broader interests) and the development of patterns of committed action that are aligned with these values [32]. ACT has demonstrated effectiveness in a number of related behavioural addictions, and although direct empirical evidence in EA remains sparse, it is among the most promising candidate approaches in the contemporary literature [32].

6.1.4. Rational emotive behavioural therapy and other cognitive approaches

Rational emotive behavioural therapy (REBT), introduced by Albert Ellis in the late 1950s, is a structured cognitive-behavioural framework that targets the irrational beliefs that the individual holds about their life circumstances, on the assumption that emotional and behavioural distress arises not from the activating event itself but from the patient's interpretation of and beliefs about that event [32]. The ABC model articulated by Ellis distinguishes the activating event (A), the belief about the event (B) and the resulting emotional and behavioural consequence (C), and proposes that therapeutic change is best achieved through the systematic identification, challenging and revision of irrational beliefs [32].

The first study to examine REBT in EA was conducted by Outar and colleagues in 2018, and used a three-phase intervention consisting of education, cognitive restructuring and reinforcement [32]. Over a six-week period, participants were educated about REBT principles, taught to identify their own irrational beliefs about exercise, and supported in the development of rational alternatives; the intervention concluded with structured review exercises designed to ensure that participants could apply the learned strategies independently [32]. The study reported that the REBT intervention was followed by a reduction in EA symptoms, a reduction in irrational beliefs and an increase in unconditional self-acceptance, with findings consistent with subsequent research by Knapp and colleagues [32]. While the evidence base remains preliminary, the data suggest that REBT may constitute a useful structured alternative to or complement of standard CBT in the management of EA, particularly in patients in whom rigid, perfectionistic and absolutistic beliefs about exercise are prominent [32].

6.1.5. Treatment of exercise dysregulation in eating-disorder populations

A particularly well-developed strand of the treatment literature concerns the management of dysregulated exercise behaviour in the context of eating disorders, where contemporary clinical practice guidelines emphasise the importance of explicit assessment and targeted intervention [24,25,27]. Earlier models of treatment

recommended complete abstinence from exercise in underweight women whose primary treatment objective was weight restoration, but several systematic reviews have demonstrated that the gradual, structured and supervised reintroduction of exercise does not interfere with weight restoration in women with anorexia nervosa, and may even support it [25,27]. The contemporary approach therefore advocates for a more nuanced strategy in which exercise is reintroduced gradually as part of the broader eating-disorder treatment plan, with the cognitive and affective features of the compulsive exercise behaviour explicitly targeted through cognitive restructuring, exposure and response prevention and structured behavioural experiments [25,27].

Exposure and response prevention (ERP), in particular, has emerged as a promising strategy in this context [25,27]. The technique involves the systematic exposure of the patient to situations that would ordinarily trigger exercise (for example, a planned rest day, the consumption of a normal meal, or a period of body-image-related anxiety) while preventing the corresponding compulsive exercise response, with the therapeutic aim of allowing the affective state to subside without recourse to the compulsive behaviour [25,27]. ERP has a robust evidence base in OCD and has been adapted with some success to eating-disorder populations with compulsive exercise features [25,27]. The protocol for the systematic review by Martenstyn and colleagues explicitly identifies ERP as a treatment component of interest in the management of compulsive exercise in eating disorders and muscle dysmorphia, alongside CBT, psychoeducation, structured exercise programmes, physical exercise and dietary therapy, pharmacotherapy and multi-component strategies [27].

In muscle dysmorphia specifically, the treatment literature is still in its infancy, but the available evidence suggests that an integrated approach combining CBT (with explicit targeting of body-image cognitions and compulsive exercise behaviours), structured nutritional rehabilitation, supervised resistance-training modification and, where appropriate, pharmacotherapy targeting comorbid depression, anxiety and obsessive-compulsive symptoms, offers the most promising therapeutic strategy [27]. The high suicide-attempt rate in muscle dysmorphia (almost three times higher than in non-muscle body dysmorphic disorder, as discussed in section 5.2) underscores the clinical urgency of effective intervention in this population [27].

6.2. Pharmacotherapy

The pharmacological treatment of EA is characterised by a striking paucity of empirical evidence, and the available recommendations rest almost entirely on case reports, extrapolation from related conditions and clinical reasoning rather than on randomised controlled trials [23,32]. No medication has been formally approved for the treatment of EA in any jurisdiction, and any pharmacological intervention should be understood as off-label and as targeting either a comorbid condition or specific symptom clusters rather than EA as a distinct entity [23,32].

6.2.1. Selective serotonin reuptake inhibitors

Selective serotonin reuptake inhibitors (SSRIs) constitute the most commonly considered pharmacological option in the management of EA, primarily because of their established effectiveness in the conditions that frequently co-occur with EA — major depressive disorder, anxiety disorders, obsessive–compulsive disorder and certain eating disorders [23,32,33]. The conceptual rationale for SSRI use in EA is therefore largely indirect: rather than acting on the dysregulated exercise behaviour itself, the medication targets the affective, anxious and obsessive substrate that may sustain it, with the expectation that improvement in these domains will support the broader behavioural change [23,32].

The comorbidity of EA with depressive disorders is particularly relevant in this context. The study by Tschopp and colleagues clinically assessed major depressive disorder (MDD) using the SCID-5 in 31 individuals at risk of EA on the basis of the EDS-21, and reported a lifetime prevalence of MDD of 52% — substantially higher than the 15–30% lifetime prevalence reported in the general population [33]. In 10 of the 16 patients with EA and MDD, the onset of MDD occurred after the onset of EA, while in 5 it preceded the EA, and in one the temporal sequence was unclear [33]. The findings suggest both that depression is more common in EA than in the general population and that depression may, in some cases, follow rather than precede the dysregulated exercise behaviour, with potentially important implications for clinical management [33]. The authors of that study explicitly cautioned against the routine use of exercise as a treatment for depression in patients with established EA, since the prescription of exercise may, in such patients, reinforce the dysregulated pattern rather than alleviate the depressive features [33].

The clinical implication is that, in patients with EA and comorbid MDD, the management of the depressive disorder should generally include pharmacological treatment with an SSRI in accordance with standard clinical

practice guidelines for MDD, with appropriate dose titration and monitoring [23,32,33]. The choice of agent is guided by the usual considerations of tolerability, drug interactions and individual response, with sertraline, escitalopram and fluoxetine all being reasonable first-line options [32]. In patients with EA and comorbid OCD, SSRIs are likewise indicated at the higher doses typically required for OCD treatment (for example, fluoxetine 40–80 mg/day, sertraline 100–200 mg/day, fluvoxamine 200–300 mg/day) [23,32]. In patients with EA and comorbid anxiety disorders, SSRIs are again first-line treatment, with the addition of benzodiazepines avoided where possible because of the risk of dependence [23,32]. In patients with EA and comorbid bulimia nervosa, fluoxetine is the most extensively studied SSRI and has an established evidence base for symptom reduction [23,32].
It must be emphasised, however, that SSRIs have not been shown to produce direct improvement in EA symptomatology independently of their effects on comorbid conditions, and the available evidence does not support their use as a primary treatment for EA in the absence of a clearly identified comorbid condition [23,32]. The clinical decision to initiate SSRI treatment in an EA patient should therefore be based on the formal identification of a comorbid disorder that responds to such medication, rather than on the expectation that the medication will directly target the exercise dysregulation [23,32,33].

6.2.2. Atypical antipsychotics: the case of quetiapine

A particularly interesting, though preliminary, line of evidence concerns the use of atypical antipsychotic medications, and specifically quetiapine, in the management of EA, particularly when combined with comorbid affective dysregulation, anhedonia, severe withdrawal-related insomnia or bipolar spectrum features [32]. The available evidence is currently confined to a single case report by Di Nicola and colleagues, who described a 47-year-old man with bipolar I disorder, comorbid compulsive shopping and EA who was treated with quetiapine [32]. Quetiapine is a second-generation antipsychotic with antagonist activity at D1 and D2 dopaminergic receptors, alpha-1 and alpha-2 adrenergic receptors and 5-HT2 serotonin receptors, together with partial agonism at the 5-HT1A receptor — a pharmacological profile that overlaps with the receptor systems implicated in the neurobiology of EA, particularly the dopaminergic and serotonergic axes [32].

In the case described, the patient's EA symptoms showed moderate improvement after four weeks of quetiapine treatment, significant improvement after twelve weeks and complete remission of the behavioural symptoms after 24 weeks, with the EAI score decreasing from 28 at admission (in the "at-risk" range) to 12 (in the "asymptomatic" range) [32]. While the evidence is plainly insufficient to support a general recommendation, the observation has been considered to be of sufficient interest to merit further investigation, particularly in patients with mixed presentations involving affective dysregulation, anhedonia, severe withdrawal-related insomnia and compulsive features that have not responded to first-line interventions [32]. The hypothetical mechanism of action would include the modulation of mesolimbic dopaminergic signalling (which has been implicated in the reward dimension of EA), the modulation of serotonergic transmission (which may target the obsessive-compulsive features of the disorder) and the sedative-hypnotic effects mediated through histamine H1 and alpha-1 adrenergic blockade (which may address the severe insomnia frequently reported during withdrawal) [32].

Several caveats are necessary. First, the evidence base is confined to a single case, and any clinical decision to use quetiapine in EA must take account of the substantial uncertainty associated with this evidential basis [32]. Second, quetiapine carries a non-negligible side-effect profile, including weight gain, metabolic disturbance (with potential for type 2 diabetes), sedation, orthostatic hypotension and, at higher doses, extrapyramidal effects, all of which should be carefully weighed against the potential benefits in any individual case [32]. Third, the use of an atypical antipsychotic in a patient whose primary problem is exercise dysregulation may be perceived by the patient as disproportionate or stigmatising, and adequate psychoeducation and informed consent are essential [32]. Fourth, the available case material does not yet support a clear dose recommendation, and clinical experience in related conditions (for example, low-dose quetiapine for sleep disturbance, moderate-dose quetiapine for mood stabilisation in bipolar disorder) would have to inform any pragmatic prescribing decision [32].

6.2.3. Other pharmacological considerations

A small number of other pharmacological strategies have been considered in the literature on EA, although the evidence base is even more limited than for SSRIs and quetiapine [23,32]. Naltrexone, an opioid receptor antagonist that has demonstrated effectiveness in alcohol use disorder and in some behavioural addictions (notably gambling disorder and kleptomania), has been considered as a candidate intervention on the basis of the role of the endogenous opioid system in exercise-induced reward (see section 4.2) [23,32]. The theoretical rationale is that naltrexone, by blocking μ-opioid receptors, would attenuate the reinforcing properties of

exercise and might thereby reduce the motivational drive that sustains compulsive engagement [32]. The empirical evidence in EA, however, is essentially absent, and the proposal remains hypothetical [32].

The endocannabinoid system represents a further potential target, given the evidence reviewed in section 4.2 for the role of endocannabinoid signalling in the mediation of the runner's high [22,32]. Cannabinoid receptor antagonists, such as rimonabant, have been associated with reductions in voluntary running in animal models, but the development of rimonabant for human use was discontinued owing to its side-effect profile (including significant depressive and suicidal effects), and no currently available cannabinoid-targeted medication has an established role in the treatment of EA [22,32].

In patients with EA and comorbid eating disorders, the broader pharmacological framework of eating-disorder treatment applies, including the use of fluoxetine in bulimia nervosa, the consideration of olanzapine in selected cases of severe anorexia nervosa, and the management of medical complications of malnutrition and purging behaviour [27,32]. In patients with EA and comorbid muscle dysmorphia or body dysmorphic disorder, SSRIs at OCD-equivalent doses constitute the principal pharmacological option, sometimes supplemented by atypical antipsychotic augmentation in treatment-resistant cases [27,32]. In patients with EA and comorbid attention-deficit/hyperactivity disorder (ADHD) — a comorbidity that has been documented in EA samples — the standard ADHD treatment framework applies, with the proviso that stimulant medications may, in some patients, paradoxically reinforce compulsive engagement with exercise and should therefore be used with appropriate caution and monitoring [32].

6.3. Multimodal treatment, treatment setting and longitudinal management

A recurrent theme in the contemporary therapeutic literature on EA is the need for multimodal, individualised and longitudinal management strategies that integrate the elements discussed above and that address the patient's broader clinical picture [23,27,32]. The choice of treatment setting — outpatient, intensive outpatient, day-hospital or inpatient — depends on the severity of the dysregulation, the presence and severity of comorbid conditions, the medical consequences of the exercise behaviour (including injury, malnutrition, electrolyte disturbance and overuse syndromes) and the safety of the patient [27,32]. In severe cases, particularly when accompanied by severe restrictive eating, low body weight, marked depressive features with suicidal ideation or significant medical complications, inpatient or intensive outpatient treatment may be required to provide containment, medical stabilisation and the structured environment necessary for the gradual modification of the exercise pattern [27,32].

The longitudinal management of EA presents particular challenges. The natural history of the condition is incompletely characterised, with limited data on the rates of spontaneous remission, the durability of treatment response and the prognostic factors associated with favourable or unfavourable outcomes [23,32]. The available follow-up data suggest that EA symptoms may be relatively stable over moderate follow-up periods, that comorbid mental disorders frequently persist alongside the dysregulated exercise behaviour and that relapse following an apparently successful intervention is not uncommon [32,33]. The clinical implication is that long-term follow-up should be an integral component of the management plan, with explicit attention to relapse-prevention strategies, ongoing monitoring of comorbid conditions and the periodic reassessment of the patient's relationship with exercise [27,32].

The integration of family members, training partners, coaches and team physicians into the treatment plan, where appropriate, has been increasingly emphasised in the recent literature [12,32]. Collateral information from these informants can be invaluable in the assessment of the patient's behaviour, in the early detection of relapse and in the support of the therapeutic objectives, particularly in elite-athletic contexts in which the training environment itself may need to be modified [12,29,30,32]. At the same time, the explicit communication of the diagnostic formulation and the treatment plan to non-clinical informants requires careful attention to confidentiality, consent and the patient's autonomy [32].

Finally, the contemporary therapeutic literature has begun to emphasise the importance of preventive interventions, particularly in adolescent and young-adult populations and in elite-athletic environments [12,29,30,32]. Preventive strategies include education about the boundary between healthy commitment and dysregulated engagement, the cultivation of broader life interests and identities that extend beyond exercise, the explicit monitoring of training load and recovery, the integration of mental-health awareness into sports-medicine practice, and the development of structured screening and early-intervention pathways for at-risk individuals [12,29,30,32]. While the empirical evidence base for prevention is even more limited than for

treatment, the clinical orientation has shifted decisively from a reactive to a proactive stance, and is likely to constitute an important focus for future research and clinical development [12,32].

In summary, the therapeutic landscape for EA is characterised by a clear orientation towards cognitive-behavioural and related psychotherapeutic interventions, by a limited but evolving role for pharmacotherapy directed primarily at comorbid conditions, by a strong emphasis on the integrated management of co-occurring eating disorders, mood disorders and obsessive–compulsive features, and by an emerging interest in preventive and longitudinal approaches that extend beyond acute symptom management [23,27,32]. The evidence base remains preliminary, and a robust standard of care has yet to be established; the recommendations summarised here should therefore be understood as reflecting current clinical orientation rather than as constituting a definitive treatment protocol [23,32].

7. Discussion and limitations

7.1. Synthesis of the principal findings

The preceding sections have sought to construct an integrated account of exercise addiction (EA) drawing on the diagnostic, neurobiological, epidemiological and therapeutic literatures available at the time of writing. Several broad findings emerge from this synthesis. First, EA is a clinically meaningful phenomenon that has been recognised in the scholarly literature for nearly half a century, but that continues to lack formal nosological recognition in the DSM-5 and the ICD-11, in part because of the methodological heterogeneity of the underlying empirical evidence and in part because of legitimate concerns about the medicalisation of a socially valued behaviour [2,5,6]. Second, the operationalisation of EA across research and clinical contexts is dominated by two psychometric instruments — the EDS-21 and the EAI in its successive versions — both of which are anchored in conceptual frameworks borrowed from substance-dependence and behavioural-addiction theory and both of which yield dimensional risk indices rather than categorical diagnoses [9,13,14]. Third, the available neurobiological evidence — although limited in quantity, modest in sample size and predominantly cross-sectional — points to a coherent pattern of dysregulation in mesolimbic dopaminergic circuits, endogenous opioid and endocannabinoid signalling pathways, and prefrontal regulatory networks (in particular the orbitofrontal cortex and inferior frontal gyrus), broadly consistent with patterns described in substance-related and other behavioural addictions [10,20,21,22]. Fourth, the differential diagnosis of EA at the interface between psychiatry and sports medicine remains challenging, with the principal axes being the distinction between primary and secondary forms, the overlap with eating disorders and muscle dysmorphia, the boundary with obsessive–compulsive disorder, the separation from the overtraining syndrome, and the differentiation between commitment and addiction in elite-athletic populations [4,16,24,25,27,29,31]. Fifth, the therapeutic landscape is dominated by cognitive–behavioural approaches and related psychotherapeutic strategies, supplemented by limited and largely off-label pharmacological interventions directed primarily at comorbid conditions; no medication has been formally approved for the treatment of EA in any jurisdiction [23,32].

A unifying conceptual proposition that emerges from the literature reviewed is that EA should be understood as a heterogeneous, multifactorial and dimensionally distributed phenomenon, rather than as a discrete categorical entity with a single etiology [6,35]. The interactional model of EA articulated by Egorov and Szabo provides a useful framework for this perspective: it conceptualises EA as the product of an interaction between intrinsic personal factors (personality, cognitive style, prior experience, motivational structure), extrinsic situational factors (life stress, sociocultural environment, sport-specific demands) and the precipitating role of stressful life events that drive the individual to engage in exercise as a coping strategy [35]. Within this framework, the development of EA is not understood as a linear progression along a continuum from healthy to unhealthy exercise, but rather as the relatively sudden emergence of a maladaptive pattern in a subset of individuals whose intrinsic and situational characteristics interact with a precipitating stressor in a particular way [35]. The model has the virtue of accommodating the empirical observation that EA emerges in only a small minority of habitual exercisers, that it can manifest abruptly rather than gradually, and that its phenomenology is heterogeneous across affected individuals [35].

7.2. The nosological status of exercise addiction

The continued absence of EA from the principal diagnostic classifications has been the subject of considerable scholarly debate, and the question is unlikely to be resolved in the immediate future [2,5,6,38]. Several lines of argument support the recognition of EA as a distinct nosological entity: the consistent description of a behavioural syndrome that fulfils the conventional criteria of behavioural addiction, the existence of a recognisable clinical phenotype with characteristic features of tolerance, withdrawal, salience, mood

modification, conflict and relapse, the convergent neurobiological evidence pointing to dysregulation of reward-related and prefrontal regulatory circuits, the documented clinical burden in affected individuals (including injury, depression, anxiety, social impairment and, in some cases, suicidality) and the practical clinical demand for an explicit diagnostic framework that would support the development of treatment guidelines and the reimbursement of evidence-based interventions [3,6,20,33]. The longitudinal follow-up study by Meyer and colleagues, in which 19 individuals previously identified as at risk of EA on the basis of the EDS-21 underwent a follow-up assessment after a mean interval of 634.5 days, provides additional support for the temporal stability of the clinical phenotype: 36.8% of participants showed no change in the number of fulfilled EA criteria, 47.4% showed a reduction and 15.8% showed an increase, with 94.7% (18 of 19) fulfilling the criteria for at least one mental disorder at follow-up [34]. The most prevalent comorbid conditions were major depressive disorder (lifetime prevalence 73.7%) and obsessive-compulsive personality disorder (52.6%), reinforcing the view that EA is associated with substantial psychiatric morbidity and that it is, at minimum, a temporally stable clinical phenomenon [34].

The arguments against formal nosological recognition are no less weighty. The empirical literature is characterised by substantial methodological heterogeneity, including the use of multiple overlapping terms, the proliferation of psychometric instruments with different conceptual underpinnings, the dominance of cross-sectional and self-report designs, the relatively small number of structured clinical interviews and the limited number of well-controlled longitudinal studies [2,6,38]. The work of Bamber and colleagues, who compared primary exercise dependence, secondary exercise dependence (with co-occurring eating disorder), eating disorder alone and unaffected controls, found that the primary exercise dependence group was largely indistinguishable from the controls on measures of psychological morbidity, neuroticism, dispositional addictiveness, impulsiveness and self-esteem, while the secondary group exhibited a clinical profile closely resembling the eating-disorder group [38]. The authors concluded that, in the absence of an eating disorder, women identified as exercise dependent did not exhibit the personality characteristics and levels of psychological distress that would warrant the construction of primary exercise dependence as a widespread pathology [38]. While this conclusion has been contested by subsequent research — including, in particular, the Meyer studies that documented high rates of psychiatric comorbidity in clinically interviewed EA samples [20,33,34] — it nonetheless illustrates the substantive concerns that have continued to forestall formal nosological recognition.

A pragmatic middle position, advocated by several authors in the contemporary literature, is that EA should be conceptualised as a candidate disorder whose nosological status will be determined by the accumulation of further longitudinal, neurobiological and interview-based evidence, and that in the interim it should be treated as a clinically meaningful but provisional category, with affected individuals identified, assessed and managed in accordance with the principles articulated in this review [6,32,34]. Such an approach allows for the continued development of the evidence base while avoiding both the premature reification of an incompletely characterised construct and the neglect of patients who clearly suffer from the phenomenon [6,32].

7.3. The comparative position of exercise addiction among behavioural addictions

The comparison of EA with other recognised or candidate behavioural addictions provides a useful framework for understanding both its commonalities and its distinctive features [3,5,17,37]. Among the most extensively studied parallel conditions is internet addiction, with which EA has been explicitly compared in the work of Rendi, Szabo and Szabó [37]. The two conditions share the structural features of behavioural addiction, including tolerance, withdrawal, salience, mood modification, conflict and relapse, and both exhibit conceptual challenges arising from the embedding of the dysregulated behaviour within a socially valued (or at least non-stigmatised) activity domain [37]. They differ, however, in several important respects: EA involves substantial physical energy expenditure, whereas internet addiction is typically sedentary; EA is activity-specific, whereas internet addiction encompasses a heterogeneous set of online activities (gambling, gaming, sexual content, shopping, social interaction) that themselves have addictive potential; and EA produces broadly favourable physiological effects when practised in moderation, whereas internet use, particularly when sedentary and prolonged, may have less ambiguous adverse effects [37]. The comparison suggests that EA occupies a particular conceptual position among behavioural addictions, in that its underlying behaviour is intrinsically health-promoting in moderate doses and only becomes pathogenic when the dose-response relationship is overshadowed by loss of control [3,37].

Within the family of behavioural addictions, gambling disorder occupies a special status as the only condition formally recognised in both the DSM-5 and the ICD-11, and internet gaming disorder is included in Section III of the DSM-5 as a condition requiring further study [2,10]. The trajectory by which gambling disorder achieved

formal nosological recognition — through the accumulation of consistent diagnostic criteria, the development of validated assessment instruments, the documentation of stable epidemiological patterns and the convergence of neurobiological evidence — provides a roadmap for the potential future recognition of EA. Several of the steps along this trajectory have already been taken in the case of EA, including the development of operational diagnostic criteria (notably the six-component framework of Griffiths and the seven-component framework of Hausenblas and Symons Downs), the development of psychometric instruments (the EDS-21 and the EAI), the accumulation of prevalence estimates across populations and the initial documentation of neurobiological correlates [9,10,13,14]. The remaining steps include the development of internationally agreed diagnostic criteria, the systematic application of structured clinical interviews in well-characterised samples, the conduct of longitudinal studies to clarify the natural history of the condition, and the development and rigorous evaluation of targeted therapeutic interventions [6,32,34].

7.4. Sport-specific and population-specific considerations

The literature reviewed makes clear that the manifestation and the diagnostic significance of EA vary considerably across sport disciplines and population groups [29,31]. The systematic review by Di Lodovico and colleagues documented systematic differences in the prevalence of risk for EA across sport categories, with the highest rates observed among endurance athletes (14.2% on the EAI) and progressively lower rates in ball-game players (10.4%), fitness-centre attendees (8.2%), power-discipline athletes (6.4%) and the general population (3.0%) [31]. The differential vulnerability of endurance athletes has been attributed to several plausible factors, including the intrinsic capacity of prolonged aerobic exercise to engage opioid and endocannabinoid signalling and to produce the runner's high, the cultural emphasis on volume and duration in endurance training, the relatively solitary nature of endurance training (which may protect from social monitoring) and the substantial overlap between endurance training and body-composition concerns in many sports [22,29,31]. The data on power and resistance disciplines, while reporting somewhat lower overall rates, suggest a different phenotype, with greater overlap with muscle dysmorphia and with concerns about muscular development [27,31].

Among adolescent and young-adult populations, the available evidence suggests that EA may emerge early and that the developmental period of ongoing prefrontal maturation creates a window of particular vulnerability [12]. Among elite adolescent athletes specifically, the integration of EA screening into routine sports-medicine practice has been advocated, although the absence of established norms for elite-athletic populations on the standard instruments complicates the operationalisation of such screening [12,14,31]. Among older and retired adults, an emerging body of evidence has begun to document EA as a clinically relevant phenomenon. The study by Huang and colleagues, conducted in 471 retired Chinese adults with regular exercise habits (mean age 60.76 years), reported that difficulties in emotion regulation were significantly associated with EA, with state anxiety ($\beta = 0.312$, $p < 0.001$) and social dependence ($\beta = 0.332$, $p < 0.001$) serving as significant mediators [36]. The findings suggest that EA among older adults is shaped by both emotional and social vulnerabilities, particularly in the context of retirement-related role loss, declining social networks and the consequent reliance on exercise as a means of emotion regulation and social engagement [36]. The clinical implication is that EA in this population may require interventions specifically tailored to the developmental and social context of later life, including strategies to enhance emotion-regulation skills and to strengthen social-support networks [36].

The role of sex and gender in the manifestation of EA is similarly nuanced. Some reviews have reported a higher prevalence of EA risk in men, while others — particularly those drawing on samples with significant eating-disorder pathology — have reported a higher prevalence in women [5,6,28]. These divergent findings may reflect the different substrate of EA in different populations: in men, EA appears more frequently associated with muscle dysmorphia, body-composition concerns and the pursuit of muscularity; in women, it is more frequently associated with anorexia nervosa, bulimia nervosa and the pursuit of weight loss [24,27]. The differential gender pattern further reinforces the importance of assessing comorbidity systematically in any clinical or research context [24,26,27].

7.5. Clinical implications: a practical framework for assessment and management

The synthesis presented in this review supports a structured clinical approach to the assessment and management of EA that integrates the diagnostic, neurobiological and therapeutic considerations discussed above. The proposed framework comprises five elements [4,6,16,25,32]. First, the clinical assessment should begin with a comprehensive history that explicitly probes the motivational structure of the exercise behaviour, the temporal sequence of the exercise dysregulation in relation to any co-occurring pathology, the affective and behavioural response to enforced rest, the impact of the behaviour on other domains of functioning and the cognitive content that accompanies the engagement [16,32]. Second, the clinical assessment should be supplemented by validated

psychometric instruments — most commonly the EDS-21 and the EAI/EAI-3 — with explicit recognition that these tools generate dimensional risk indices rather than categorical diagnoses, and that elevated scores in elite athletes should be interpreted with particular caution [9,13,14]. Third, the assessment should explicitly screen for the principal comorbid and differential-diagnostic conditions: eating disorders (using the SCOFF, EDE-Q or equivalent), muscle dysmorphia, obsessive–compulsive disorder, major depressive disorder, anxiety disorders, ADHD, substance use disorders and personality disorders [4,16,20,24,33,34,39]. Fourth, the formulation should explicitly identify the primary versus secondary nature of the dysregulation, since this distinction has direct implications for treatment planning [3,4,6,38]. Fifth, the management plan should be individualised and multimodal, integrating psychotherapeutic interventions (CBT, DBT, mindfulness-based approaches, REBT and, in eating-disorder contexts, exposure and response prevention), pharmacological interventions where appropriate (directed primarily at comorbid conditions), structured modification of the exercise pattern itself and, where indicated, integration with sports-medicine, nutritional and family interventions [23,25,27,32].

A practical clinical consideration concerns the management of the affective and somatic features of the withdrawal syndrome during the early phase of treatment. The acute reduction in exercise volume that constitutes a core therapeutic objective frequently precipitates the affective and somatic distress described in section 3.2, and the management of this distress is a critical determinant of treatment retention and outcome [3,19,23,32]. Strategies that may be useful include the gradual rather than abrupt reduction of exercise volume, the explicit use of distress-tolerance and emotion-regulation skills (drawing on DBT and mindfulness-based frameworks), the introduction of alternative reinforcing activities (social, cultural, creative) that can partially compensate for the reduced engagement with exercise and, in selected cases, the time-limited pharmacological management of severe withdrawal-related insomnia, anxiety or depressive features [23,32]. The empirical evidence supporting any of these strategies is limited, and the clinician should be prepared to adapt the approach to the individual case [23,32].

A further clinical consideration concerns the prevention of relapse [32,34]. The follow-up data from Meyer and colleagues suggest that, although the overall pattern of EA symptoms tends to be relatively stable, a substantial minority of patients exhibit changes in either direction over a two-year period, with comorbid mental disorders persisting in the great majority of cases [34]. The implication is that the long-term management of EA should incorporate explicit relapse-prevention components, ongoing monitoring of comorbid conditions and periodic reassessment of the patient's relationship with exercise [32,34].

7.6. Methodological limitations of the field

Several methodological limitations of the underlying empirical literature warrant explicit discussion, since they constrain both the certainty of the inferences that can be drawn from the available data and the generalisability of the resulting clinical recommendations [6,17,38]. First, and perhaps most fundamentally, the literature is dominated by cross-sectional designs, with relatively few longitudinal studies and only a handful of randomised controlled trials of therapeutic interventions [6,32]. The cross-sectional dominance limits the inferences that can be drawn about causality, about the natural history of the condition and about the directionality of the relationships between EA and the various comorbid features that have been documented [6,34]. Second, the field continues to be characterised by terminological heterogeneity, with at least a dozen overlapping labels in use, and the choice of label often implies a particular conceptual framework (compulsive, dependent, obligatory, addictive, abusive, morbid) [18]. The terminological heterogeneity, combined with the corresponding diversity of psychometric instruments, complicates between-study comparisons and produces a degree of conceptual fragmentation that has been identified as a major obstacle to progress in the field [6,17,18].

Third, the dominance of self-report psychometric instruments — and the relative scarcity of structured clinical interviews — is a structural limitation of the field [6,17,34]. The available instruments generate dimensional risk indices that are sensitive to training volume and intensity, and tend to overestimate the true prevalence of EA when compared with clinician-led interviews [6,34]. The reliability-generalisation meta-analysis by Alcaraz-Ibáñez and colleagues, encompassing 255 studies and 254,174 participants, documented a substantial rate of reliability induction (almost 48% of studies reported reliability coefficients from earlier studies rather than from their own data) and a virtual absence of testing of the assumptions required for the valid use of Cronbach's α [17]. Fourth, sample composition is frequently biased towards convenience samples drawn from fitness centres, university populations and online recruitment, with relative under-representation of clinical samples, low-income populations and non-Western contexts [6,17,38]. The resulting epidemiological estimates may not generalise well to the populations most likely to encounter affected individuals in clinical practice.

Fifth, the operationalisation of EA in much of the empirical literature does not adequately distinguish between primary and secondary forms, with the consequence that prevalence estimates and clinical correlates may conflate the two phenomena [6,18,38]. The meta-analytic finding that EA occurs more than three and a half times more frequently in samples with eating-disorder symptoms than in samples without them, together with the suggestion that approximately three-quarters of studies reporting EA prevalence may in fact be measuring instrumental or secondary EA, reinforces this concern [6,24,26]. The systematic measurement of eating-disorder symptomatology in any future EA research has been repeatedly recommended in the contemporary literature, and the present review echoes that recommendation [6,24,26].

Sixth, the neurobiological evidence base, while internally coherent, remains limited in scale [10,20]. The systematic review by Pirwani and Szabo identified only eight neuroimaging studies of EA that met inclusion criteria, with sample sizes typically in the 20–130 range, and with substantial methodological diversity across studies [10]. The available evidence has not yet been synthesised through meta-analysis at the voxel level, and key candidate findings — including the reduction in OFC grey-matter volume and the alterations in frontolimbic and frontoparietal connectivity — have been replicated in only a handful of studies [10,20]. The translation of these findings into a robust neurobiological model of EA, comparable in maturity to the corresponding models in substance use disorders or gambling disorder, will require substantially expanded longitudinal, experimental and ligand-based neuroimaging investigations [10,20].

Seventh, the therapeutic evidence base is preliminary and rests largely on case reports, small case series, extrapolation from related conditions and clinical reasoning [23,32]. No large-scale randomised controlled trial has, to date, been conducted in EA, and the evidence supporting the principal therapeutic recommendations is therefore indirect [32]. The systematic-review protocol of Martenstyn and colleagues represents an important step towards the systematic synthesis of the existing treatment literature for compulsive exercise in eating disorders and muscle dysmorphia, but the broader treatment evidence base for primary EA remains underdeveloped [27].

Finally, the field has only recently begun to address cross-cultural variation in the manifestation, assessment and treatment of EA [14,15]. The international validation study of the EAI-3, which included Chinese, German, Italian, Japanese and Turkish samples, identified scalar invariance violations on certain items and raised the prospect that direct between-country comparisons of mean scores may be misleading [14]. The cross-cultural literature is therefore still maturing, and country-specific norms and culturally adapted assessment instruments will be required before the field can support truly global comparative work [14,15].

7.7. Limitations of the present review

The present narrative review is itself subject to several limitations that should be acknowledged. First, as a narrative rather than a fully systematic review, the work does not pursue an exhaustive enumeration of the published literature, does not apply formal quality-assessment instruments to every included source and does not generate quantitative pooled estimates of effect sizes [6,32]. The selection of sources, while transparent and justified by the search strategy described in section 2, inevitably reflects interpretative choices made by the author, and alternative organisational schemes could legitimately have been adopted. Second, the review draws predominantly on English- and Polish-language sources, with limited integration of literature in other languages; this constraint reflects both the dominance of English-language publication in the field and the working linguistic capacity of the author, but it may have led to the under-representation of important national literatures, particularly from non-Western contexts [6,15].
Third, the conceptual organisation of the review — particularly the structuring of sections 4 (neurobiology) and 5 (differential diagnosis) — reflects a clinical orientation aligned with the perspective of a psychiatry trainee working at the interface with sports medicine, and may not align with the conceptual preferences of researchers drawing on alternative theoretical frameworks (for example, the cognitive-behavioural model of compulsive exercise developed within the eating-disorder literature, or the interactional model articulated by Egorov and Szabo) [25,35,39]. The framework used here aims to provide a clinically useful synthesis rather than to advocate for a particular theoretical position. Fourth, the review has been written at a time when several active research programmes are still in progress, including ongoing systematic reviews registered in PROSPERO, ongoing protocols for randomised controlled trials of psychotherapeutic interventions and ongoing neuroimaging investigations; the conclusions presented here should therefore be understood as reflecting the state of evidence at the time of synthesis and as provisional pending the publication of these ongoing studies [27,32].

Fifth, the present review has not attempted to address several adjacent topics that are relevant to the broader understanding of dysregulated exercise behaviour but that fell outside the defined scope of the work, including

the relationship between EA and the use of performance-enhancing substances (notably anabolic-androgenic steroids in resistance-training populations), the role of sleep dysregulation as both a cause and a consequence of EA, the specific phenomenology of compulsive exercise in dance disciplines and the broader public-health implications of the medicalisation of an otherwise health-promoting behaviour [27,30]. These topics merit dedicated review in their own right and represent fruitful areas for future scholarship.

7.8. Directions for future research

The synthesis presented in this review supports several priorities for future investigation in the field of EA [6,32,34]. First, there is a clear need for longitudinal studies that follow well-characterised samples of individuals with EA over extended periods, in order to clarify the natural history of the condition, the temporal stability of the clinical phenotype, the relationship between EA and the development of comorbid conditions, and the predictors of favourable and unfavourable outcomes [34]. Second, the systematic application of structured clinical interviews — supplementing but not replacing psychometric screening — is essential to the development of an evidence base capable of supporting formal nosological recognition [6,14,34]. Third, expanded neuroimaging investigations, ideally combining structural, functional, diffusion and ligand-based modalities and incorporating prospective designs, would substantially advance the understanding of the neurobiological substrate of EA [10,20]. Fourth, well-designed randomised controlled trials of psychotherapeutic and, where appropriate, pharmacological interventions are urgently required to establish an evidence-based standard of care [27,32].

Fifth, the field would benefit from explicit attention to specific populations that have received limited research attention, including adolescents, older and retired adults, retired elite athletes, individuals in resistance-training and combat-sport contexts, and populations in non-Western cultural settings [12,30,36]. Sixth, the integration of EA research with the broader literature on behavioural addictions, eating disorders, obsessive–compulsive spectrum conditions and sports medicine would support a more unified conceptual and clinical framework [4,6,25,29,39]. Seventh, the field would benefit from greater methodological standardisation, including the consistent use of validated instruments, the systematic measurement of comorbidity, the explicit distinction between primary and secondary forms, and the development of internationally agreed diagnostic criteria [6,17,18]. Finally, the development of preventive interventions for adolescent and young-adult populations and for elite-athletic environments, supported by appropriate empirical evaluation, is likely to constitute an important focus for future research and clinical development [12,29,30,32].

7.9. A concluding reflection on the exercise paradox

The paradox of EA — that a behaviour fundamental to human health and well-being can, in a minority of individuals, become an autonomous source of distress and dysfunction — encapsulates one of the more conceptually challenging aspects of contemporary behavioural medicine [3,5,35]. The literature reviewed here supports the proposition that EA is a clinically meaningful phenomenon worthy of formal recognition and continued investigation, while also illustrating the methodological and conceptual challenges that have, to date, prevented such recognition from being achieved [2,5,6,38]. The clinical orientation that emerges from this synthesis is one of cautious recognition, careful differential diagnosis, multimodal individualised management and continued vigilance for the developing evidence base [6,32].

For the psychiatry trainee or sports medicine practitioner encountering a patient with possible EA, the most important practical implication of the literature reviewed here is the necessity of integrating perspectives from multiple disciplines. The exercise behaviour cannot be adequately understood through the lens of any single specialty: the contribution of psychiatry is essential for the assessment of the affective, cognitive and behavioural substrate; the contribution of sports medicine is essential for the assessment of the training history, the physiological consequences and the boundary with overtraining; the contribution of nutrition and dietetics is essential where eating-disorder features are prominent; the contribution of clinical psychology is essential for the implementation of the principal therapeutic interventions; and the contribution of family and social networks is essential for the supportive context within which any treatment must operate [27,29,30,32]. The integration of these perspectives — in the assessment, formulation, treatment and follow-up of each individual case — constitutes, on the present evidence, the most promising clinical orientation for a condition that continues to occupy an uneasy but unmistakeably important position at the interface between psychiatry and sports medicine [3,5,6,32].

8. Conclusions

Exercise addiction (EA) appears, on the basis of the evidence reviewed, to constitute a clinically meaningful behavioural syndrome situated at the interface of psychiatric, addiction-medicine and sports-medicine practice, yet it continues to lack a formally recognised diagnostic position within either the DSM-5 or the ICD-11 [2,5,6]. The phenomenon — variously described as exercise addiction, exercise dependence, compulsive exercise, obligatory exercise, or morbid exercise behaviour — is characterised by a maladaptive pattern of compulsive engagement with physical activity that persists despite mounting physical, psychological, social and occupational costs, and that exhibits the core components of behavioural addiction articulated by Griffiths: salience, mood modification, tolerance, withdrawal symptoms, conflict and relapse [3,9,13]. The terminological and conceptual heterogeneity that has characterised the field for more than four decades reflects both the legitimate diversity of theoretical perspectives that have been applied to the phenomenon and the genuine difficulty of operationalising a behavioural syndrome whose underlying activity is, in moderate doses, broadly health-promoting [3,18,38].
The available evidence supports the broad conclusion that EA may affect approximately 3% of regular exercisers in the general population, with substantially higher estimates reported among endurance athletes and within clinical samples that include eating-disorder comorbidity [6,7,8,31]. The wide variability of reported prevalence — ranging from below 1% in general population samples to above 50% in selected clinical and athletic subgroups — appears to reflect both the methodological diversity of the underlying studies and the substantive heterogeneity of the populations examined [6,31,38]. The systematic measurement of comorbidity, the explicit differentiation between primary and secondary forms, and the supplementation of psychometric screening with structured clinical interview emerge as the principal methodological priorities for any future epidemiological research in this domain [6,17,34].

The diagnostic operationalisation of EA in research and clinical practice continues to rely principally on two psychometric instruments — the Exercise Dependence Scale-21 (EDS-21) and the Exercise Addiction Inventory in its successive forms (EAI, EAI-R, EAI-3) — both of which generate dimensional risk indices anchored in substance-dependence and behavioural-addiction frameworks rather than producing categorical diagnoses [9,13,14]. The recent international validation of the EAI-3 in a sample of 1,931 exercisers across five linguistic contexts has advanced the cross-cultural applicability of the instrument and has produced a preliminary cut-off score that may, in due course, support more uniform clinical screening [14]. The systematic review of psychometric instruments by Harris and colleagues, which examined the measurement of compulsive exercise specifically in patients with eating disorders, similarly emphasised the need for instruments that are conceptually appropriate to the population being studied and that capture both the addictive and the compulsive dimensions of the behaviour [40]. Across the broader landscape of available tools, none currently constitutes a diagnostic instrument in the strict sense, and elevated scores should be interpreted as flags for further clinical evaluation rather than as definitive markers of disorder [6,14,40].

The neurobiological evidence base, while modest in scale, points to a coherent pattern of dysregulation across reward-related and prefrontal regulatory circuits that broadly parallels the patterns documented in substance-related and recognised behavioural addictions [10,20]. The principal findings include alterations in mesolimbic dopaminergic signalling, with implications for tolerance, withdrawal and habit formation [20,21]; the engagement of endogenous opioid and endocannabinoid pathways in the mediation of the runner's high, with anandamide signalling at CB1 receptors emerging as a particularly important mechanism alongside the long-established opioid hypothesis [21,22]; and structural and functional alterations in the orbitofrontal cortex, inferior frontal gyrus and broader frontolimbic and frontoparietal networks that may constitute the neural substrate of the impaired inhibitory control and cognitive rigidity observed in affected individuals [10,20]. These findings remain preliminary, are derived from a relatively small number of studies with modest sample sizes and predominantly cross-sectional designs, and have not yet been integrated into a definitive neurobiological model of EA; their further development will require expanded longitudinal, experimental and ligand-based neuroimaging work [10,20].

The differential diagnosis of EA at the interface between psychiatry and sports medicine constitutes, from the standpoint of clinical practice, the most consequential dimension of the literature reviewed here [4,16,25,29,31]. The distinction between primary and secondary forms — between EA as an autonomous behavioural addiction and EA as a compensatory strategy within an underlying eating disorder, body-image disturbance or affective disturbance — has direct implications for the prioritisation of therapeutic targets and for the prognostic outlook [3,4,6,38]. The substantial overlap with eating disorders — including the documented high rates of compulsive exercise in anorexia nervosa and bulimia nervosa, the meta-analytic finding of small to medium correlations

between morbid exercise behaviour and dimensions of eating-disorder symptomatology, and the recent confirmation in 3,105 participants of the unique associations between compulsive exercise, OCD symptoms and perfectionism in eating-disorder samples — reinforces the clinical importance of integrated assessment [24,25,27,39]. The closely related condition of muscle dysmorphia, with its high suicide-attempt rate and its substantial overlap with eating-disorder pathology, deserves particular attention in male resistance-training and bodybuilding populations [27]. The differentiation of EA from the overtraining syndrome, with its distinct etiology as a physiological exhaustion of the HPA axis and associated regulatory systems, and its different therapeutic priorities, is essential at the interface with sports medicine [28,29,30]. The differentiation between commitment and addiction in elite-athletic populations remains particularly challenging, and the careful integration of psychometric, clinical-interview and contextual information is essential to avoid both the false-positive misclassification of highly committed athletes and the false-negative failure to recognise dysregulated patterns in populations where extreme commitment is normative [5,14].

A particular clinical implication that warrants emphasis concerns adolescent and youth populations, in whom the developmental immaturity of prefrontal regulatory networks and the heightened sensitivity of mesolimbic reward circuits create a window of particular vulnerability [12]. The work of Goodwin and colleagues in a UK adolescent sample documented significant differences in compulsive exercise patterns between sport and non-sport participants, with sport participants generally reporting greater levels of compulsive exercise across multiple subscales of the Compulsive Exercise Test, suggesting that sport participation in this age group may, in some individuals, constitute a risk factor rather than a purely protective one [41]. The early identification of high-risk adolescents — through coordinated screening in school, sports-club and primary-care settings — and the integration of preventive components into youth-sport environments may constitute a useful focus for future research and clinical development [12,41].

The therapeutic landscape for EA is dominated by cognitive–behavioural and related psychotherapeutic interventions, with dialectical–behavioural therapy, mindfulness-based and acceptance-based approaches, and rational emotive behavioural therapy each having a plausible conceptual rationale and a preliminary empirical foundation [23,32]. Exposure and response prevention has emerged as a particularly promising strategy in patients with prominent compulsive features, especially within eating-disorder contexts where dysregulated exercise is closely linked to anxiety associated with weight, shape and food intake [25,27,39]. The contemporary clinical consensus has moved decisively away from earlier models recommending complete abstinence from exercise towards a more nuanced approach advocating the gradual, structured and supervised reintroduction of safe physical activity within a broader treatment plan [25,27]. Pharmacotherapy plays a limited and largely adjunctive role, directed primarily at comorbid major depressive disorder, anxiety disorders, obsessive-compulsive disorder, eating disorders and other conditions that may sustain or complicate the dysregulated behaviour [23,32,33]. The single case report describing the off-label use of quetiapine in EA with affective dysregulation and severe withdrawal-related insomnia represents an interesting but preliminary observation that requires substantial further investigation before any general recommendation can be supported [32].

The longitudinal follow-up data available to date — including the recent two-year follow-up of the Meyer sample, which demonstrated relative temporal stability of EA symptoms with persistent high rates of comorbid mental disorders (including major depressive disorder in 73.7% and obsessive-compulsive personality disorder in 52.6% of participants) — reinforce the view that EA is a clinically meaningful and temporally stable phenomenon associated with substantial psychiatric morbidity, and that its long-term management requires explicit attention to comorbid conditions and to relapse-prevention strategies [34]. The interactional model articulated by Egorov and Szabo provides a useful conceptual framework within which to understand the heterogeneity of the phenomenon, accommodating the empirical observation that EA emerges in only a small minority of habitual exercisers, that it can manifest abruptly rather than gradually, and that its phenomenology is shaped by the interaction between intrinsic personal factors, extrinsic situational factors and precipitating life events [35].

The principal limitations of the field that warrant continued attention include the methodological dominance of cross-sectional and self-report designs, the terminological and conceptual heterogeneity that complicates between-study comparisons, the relative scarcity of structured clinical interviews and longitudinal data, the limited integration of clinical and neurobiological evidence, the absence of large-scale randomised controlled trials of therapeutic interventions, the under-representation of clinical samples and of populations outside Western contexts, and the cross-cultural variability of the available assessment instruments [6,17,18,32,34]. Each of these limitations represents an opportunity for future research, and each is amenable to systematic improvement through methodological standardisation and through the targeted accumulation of evidence in the areas most critical for nosological development.

In synthesising the evidence reviewed in this work, the present authors arrive at the following overarching conclusions. First, EA appears to be a clinically meaningful, although under-recognised and incompletely characterised, behavioural syndrome whose accurate identification requires the careful integration of psychometric, clinical-interview and contextual information [3,6,32,38]. Second, the differential diagnosis of EA from eating disorders, muscle dysmorphia, obsessive-compulsive disorder, the overtraining syndrome and committed elite-athletic engagement constitutes the most consequential clinical dimension of the contemporary literature, and should occupy a central place in the training of psychiatrists, sports physicians and clinical psychologists who encounter affected individuals [4,16,25,27,29,31]. Third, the neurobiological substrate of EA, although still incompletely characterised, supports the broader conceptualisation of EA within the framework of behavioural addiction, with mesolimbic dopaminergic dysregulation, endogenous opioid and endocannabinoid signalling, and frontolimbic structural alterations all converging in patterns that resemble those documented in recognised addictive disorders [10,20,21,22]. Fourth, the contemporary therapeutic orientation, while preliminary in its empirical foundation, supports the use of cognitive–behavioural and related psychotherapeutic strategies as first-line interventions, with pharmacotherapy reserved for the management of comorbid conditions and for selected refractory cases [23,27,32].

Fifth and finally, the field would benefit substantially from systematic methodological improvements in research design, from the development of internationally agreed diagnostic criteria, from the conduct of large-scale longitudinal and interventional studies, and from the explicit integration of evidence across the psychiatric, addiction-medicine, neuroscientific and sports-medicine literatures that currently address the phenomenon from partially incompatible perspectives [6,32,34]. The development of preventive interventions for adolescent, young-adult and elite-athletic populations, supported by appropriate empirical evaluation, is likely to constitute an increasingly important focus for clinical and research effort in the coming decade [12,29,30,32,41].

The exercise paradox — that a behaviour fundamental to human health may, in a minority of individuals, become an autonomous source of distress and dysfunction — represents not merely a conceptual curiosity but a substantive clinical reality whose recognition, accurate assessment and effective management require the continued integration of expertise across multiple disciplines [3,5,35]. The growth of the empirical literature over the past four decades, the maturation of psychometric and neurobiological methods that can be applied to the phenomenon, and the increasing willingness of psychiatry, addiction medicine and sports medicine to engage with conditions that occupy the interface between their traditional remits, together suggest that the conditions for a more decisive scientific and clinical engagement with EA are now in place. The present narrative review has sought to contribute, in modest measure, to that broader effort, by providing a structured synthesis of the current state of knowledge for psychiatry trainees and practitioners working at the interface of mental health and sports medicine, with the deliberately cautious tone and explicit acknowledgement of uncertainty that the present state of the evidence demands [3,5,6,34].

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