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Psychosocial etiology of maladaptive exercise and its role in eating disorders: A systematic review

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Abstract

Objective: Although maladaptive exercise (ME) is widely recognized as a clinical feature in transdiagnostic eating disorders, less is known about psychosocial factors that give rise to and perpetuate this behavior. This systematic review aimed to examine the empirical status of this association.

Method: We reviewed 46 full text articles examining longitudinal associations between psychosocial variables and ME.

Results: Eighteen studies met full inclusion criteria. Based on our qualitative synthesis, evidence suggests reasonably consistent associations between early concern with weight and shape, and negative affect on later development of ME.

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AUTHOR CONTRIBUTIONS

Potential sources were initially screened by Sasha Gorrell and Rachael E. Flatt. Authors Sasha Gorrell, Rachael E. Flatt, Cynthia M. Bulik, and Daniel Le Grange all contributed to the design, review, and writing of the manuscript. All authors have approved the final manuscript.

CONFLICT OF INTEREST

Dr. Le Grange has received royalties from Guilford Press and Routledge and payments from the Training Institute for Child and Adolescent Eating Disorders, LLC. All authors report no other biomedical financial interests or potential conflicts of interest. Dr. Bulik reports: Shire (grant recipient, Scientific Advisory Board member); Pearson (author, royalty recipient).

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

Discussion: Inconsistent and insufficient assessment of ME across a majority of studies underscores caution in interpretation of results, but guides important discussion for future clinical and research efforts.

Resumen

Aunque el ejercicio desadaptativo (ME, por sus siglas en inglés) es ampliamente reconocido como una característica clínica en el transdiagnóstico de los trastornos alimentarios, se sabe menos acerca de los factores psicosociales que dan lugar a este comportamiento y lo perpetúan. Esta revisión sistemática tuvo como objetivo examinar el estado empírico de esta asociación.

Revisamos cuarenta y seis artículos de texto completo que examinaban las asociaciones longitudinales entre las variables psicosociales y el ME.

Dieciocho estudios cumplieron todos los criterios de inclusión. Con base en nuestra síntesis cualitativa, la evidencia sugiere asociaciones razonablemente consistentes entre la preocupación temprana por el peso y la forma, y el afecto negativo sobre el desarrollo posterior de ME.

La evaluación inconsistente e insuficiente del ME en la mayoría de los estudios subraya la cautela en la interpretación de los resultados, pero guía una discusión importante para futuros esfuerzos clínicos y de investigación.

Keywords

compulsive exercise; eating disorder; exercise; maladaptive exercise

1 | INTRODUCTION

Eating disorders (EDs) are pernicious psychiatric illnesses that are associated with increased mortality and high treatment costs (Van Hoeken & Hoek, 2020), and significant global disease burden (Erskine, Whiteford, & Pike, 2016). At best, ED treatments for anorexia nervosa (AN) and bulimia nervosa (BN) in adolescents achieve full remission in only about 40% of cases who complete treatment (Lock & Le Grange, 2018), and effective treatments for adults with AN remain limited (Bulik, Berkman, Brownley, Sedway, & Lohr, 2007; Frank & Shott, 2016). Improving treatment outcome for a majority of patients with EDs depends on clarifying the etiology of key symptoms that maintain these life-interrupting disorders.

Maladaptive exercise (ME) is a hallmark ED-related behavior reported in up to 80% of those with AN (Dalle Grave, Calugi, & Marchesini, 2008), 73% of those with atypical AN (Sawyer, Whitelaw, Le Grange, Yeo, & Hughes, 2016), and 40% of those with BN (Shroff et al., 2006). A widely accepted definition of ME has yet to be harmonized across the literature, but a type of ME commonly identified among patients with EDs is referred to as “compulsive” (Adkins & Keel, 2005). Building on this construct, several criteria have been proposed that capture the compulsive nature of some ME (Dittmer, Jacobi, & Voderholzer, 2018), and a recent review of the assessment of exercise in ED patients (Harris, Hay, & Touyz, 2020) concluded that component characteristics across definitions of ME overlapped most closely with the American Psychiatric Association's account of compulsivity, defined by “repetitive behaviors that the person feels driven to perform” that

are “aimed at preventing or reducing distress” (APA, 2013). This exercise behavior may be excessive with regards to intensity, duration, and frequency (Solenberger, 2001), and individuals with ME may feel compelled to engage in physical activity to control weight or shape; to prevent or reduce negative affect; or to avoid adverse health effects of a sedentary lifestyle (Dittmer et al., 2018). Furthermore, individuals with EDs often engage in ME despite pain or injury, or significant adverse medical, psychological, social, or occupational consequences (Noetel, Dawson, Hay, & Touyz, 2017). Although ME has been assessed with considerable variability across the ED literature (Rizk, Lalanne, Berthoz, Kern, & Godart, 2015), this behavior pattern consistently marks increased risk for suicidal behavior (Smith et al., 2013), poorer treatment outcomes (Dalle Grave et al., 2008; Solenberger, 2001), increased ED and related psychopathology (Brewerton, Stellefson, Hibbs, Hodges, & Cochrane, 1995; Davis & Kaptein, 2006; Holtkamp, Hebebrand, & Herpertz-Dahlmann, 2004; Shroff et al., 2006), and ED relapse (Steinhausen, Grigoroiu-Serbanescu, Boyadjieva, Neumärker, & Winkler Metzke, 2008). The elevated prevalence and negative sequelae associated with this behavior suggest that a better understanding of ME onset in the context of EDs is critical to delineating factors that contribute to, and maintain this behavior. Identification of these precipitating factors may then be used to inform intervention targets and contribute to improved clinical outcomes. While complete cessation of exercise is typically recommended in the face of medical acuity, a historically cautionary approach to exercise among patients with restrictive EDs has inadvertently contributed to a lack of focus on ME, both in empirical ED intervention and research in this domain (Cook et al., 2016).

More recently, ME has received increased focus in ED research, including attempts to target the compulsive nature of this behavior (Dittmer et al., 2020). A restless urge to move among some patients with AN is thought to reflect involuntary neurobiological processes, secondary to weight loss (Casper, 2006, 2018). ME may also be conceptualized as a cognitively and/or emotionally motivated behavior, related to weight and shape concerns (Meyer, Taranis, Goodwin, & Haycraft, 2011), and driven by primary and/or secondary reward processing (Bamber, Cockerill, Rodgers, & Carroll, 2003; Cunningham, Pearman, & Brewerton, 2016). ME has been characterized independently of psychological processes in a rodent model of activity-based anorexia (ABA) (Epling & Pierce, 1989), which supports a link between increased activity and subsequent food restriction. In humans, evidence also supports a positive genetic correlation between AN and measured physical activity (Watson et al., 2019). Across both animal and human research, ME may thus be characterized by an intersection of involuntary and voluntary behavior patterns.

Historically, ME was characterized by its excessive nature (Davis & Fox, 1993), whereas more recent research supports the notion that *motivation* for exercise (e.g., weight control; regulating negative affect) is more problematic than simply the frequency or duration of activity (Adkins & Keel, 2005; Meyer et al., 2011; Mond & Calogero, 2009; Seigel & Hetta, 2001). Specifically, detrimental associations with exercise (e.g., lower remission rates in ED treatment; Monell, Levallius, Forsén Mantilla, et al., 2018) exist in ED samples, despite research that suggests exercise dependence, not exercise behavior, mediates the relationship between exercise and eating pathology (Cook, Hausenblas, Crosby, Cao, & Wonderlich, 2015). Therefore, given that psychological motivation for exercise within ED populations (e.g., Hausenblas & Downs, 2002a), rather than exercise behavior itself (i.e.,

exercise frequency, duration, type, and intensity), may be the pivotal component to impact clinical outcomes within ED populations (e.g., Young et al., 2018), it is critical to consider ME not alone, but within the context of eating pathology.

Conceptualizations of ME have been formalized with various psychosocial models that draw upon principles of positive and negative reinforcement to explain the maintenance of this behavior (Hausenblas & Downs, 2002b; Meyer et al., 2011). Two of the most prominent psychosocial models of ME (i.e., characterizing this behavior either as "exercise dependence" (Hausenblas & Downs, 2002a) or as "compulsive exercise" [Meyer et al., 2011]) agree on momentary affect regulation as the main psychological outcome of exercise behavior in the context of EDs. As such, an overarching theory of ME that is rooted in affect-regulation aligns well with etiological models that conceptualize ED behaviors more broadly as maladaptive attempts to regulate negative affect induced by ED-specific events and cognitions (Haynos & Fruzzetti, 2011). In alignment with reinforcement theory, the potential short-term affect-regulation effects of ED behaviors such as ME (i.e., decrease in negative affect, increase in positive affect) can be considered the primary motivators (and maintenance mechanisms) for maladaptive behavior (Swerdlow, Pearlstein, Sandel, Mauss, & Johnson, 2020).

Even within this affect-regulation framework, as a field, we remain without consensus regarding for whom, why, and how ME may arise and be maintained in the context of eating pathology. One barrier to consensus has been the considerable number of terms used to define the construct of ME, which vary in both the degree to which they quantify exercise (i.e., excessive nature) or in their qualitative nature (i.e., motivation for engaging in exercise behavior). Some of these constructs include "compulsive" (Meyer et al., 2011); "exercise dependence" (Hausenblas & Downs, 2002b); "obligatory" (Pritchard & Beaver, 2012), "addictive" (Adams & Kirkby, 2002), and "driven" (Stiles-Shields et al., 2015), among others. ME has also been defined as either a *primary* disorder (functioning to reduce negative affect) or a *secondary* symptom of an ED (functioning to control weight and shape) (Bamber et al., 2003), and research distinguishing these forms of problematic exercise typically qualifies primary pathological exercise as addictive and secondary pathological exercise as compulsive (Cunningham et al., 2016). Recent cross-sectional research suggests that those with ME and ED pathology experience greater levels of both the addictive and compulsive qualities of exercise than individuals without EDs (Cook et al., 2013; Cunningham et al., 2016). In acknowledgement of these myriad terms and their respective theoretical underpinnings, a decision was made among the authors for the current review to refer to this behavior broadly as ME. Of note, given the lack of consensus on an operational definition of exercise behavior across the ED literature, it is possible that our use of the term "ME" here may in fact capture several different but related constructs. Further, understanding ME in the context of eating pathology has been limited by nearly exclusive use of self-report methodology within cross-sectional designs. While numerous studies have characterized individuals who experience concurrent eating and exercise pathology (e.g., Dalle Grave et al., 2008; Mond & Calogero, 2009; Schaal et al., 2011), a paucity of studies that employ longitudinal analyses has limited our ability to evaluate the temporal nature of ME onset within EDs.

Given associations between ME and poorer prognosis (e.g., Monell et al., 2018; Solenberger, 2001), there has been a recent upsurge in the number of treatment interventions for exercise behavior in EDs (e.g., Dittmer et al., 2020; Hay et al., 2018). However, to date, targeting ME in ED treatment has been limited by lack of consensus in a "best practices" approach (e.g., Cook & Leininger, 2017; Danielsen, Rø, & Bjørnelv, 2018; Quesnel et al., 2018), suggesting that a better understanding of psychological factors that contribute to, and maintain ME, are indicated and necessary to guide future research and intervention efforts for ME in the context of EDs. Towards this end, increased experimental study of ME may lend support for specific affect-regulatory mechanisms that contribute to ME in the context of eating pathology, and ultimately lead to a more comprehensive model of this behavior (Kolar & Gorrell, 2020). To date, models of ME (e.g., "compulsive" exercise) propose several domains of psychosocial variables that are posited to contribute to ME including, but not limited to, weight and shape concerns, negative affect (and its regulation), compulsivity, rigidity and perfectionism (Meyer et al., 2011).

Accordingly, our primary aim with this study was to provide a systematic review of psychosocial variables that are longitudinally associated with ME in the context of EDs. In addition, we strove to a) determine, where possible, limitations of the current literature base, and b) provide commentary on important future lines of scientific inquiry.

2 | METHODS

2.1 | Data sources and search strategy

This review was registered on the International Prospective Register of Ongoing Systematic Reviews (PROSPERO; Booth et al., 2012) and conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al., 2009). The primary search strategy involved six online databases: PubMed, PsycINFO, MEDLINE, Web of Science, Scopus, and SPORTDiscus, and was not restricted to a given timeframe. Unpublished studies were solicited through targeted inquiry (e.g., ED-specific listserv and private communication) but none were provided. Search parameters with key terms (e.g., "eating disorders" AND "compulsive exercise") are presented in Supporting Information.

2.2 | Eligibility criteria

Articles were screened at two stages based first on title and abstract, and then on full text. To be included in the review, articles were required to have met the following criteria: (i) sufficient data from at least two time points (i.e., longitudinal or prospective study design); (ii) valid assessment of ME, and assessment of eating pathology. Articles were excluded if they (i) only collected data through an objective measure of exercise (e.g., accelerometer derived step-counts) without a secondary qualifying feature (e.g., motivation for activity); (ii) were interventions designed to target and reduce ME; (iii) were in languages other than English; or (iv) were published abstracts, reviews, book chapters, editorials and commentaries.

2.3 | Study selection and data abstraction

Two research team members (REF/SG) conducted independent dual review of study titles and abstracts. Studies were considered for full-text review if they met the predefined eligibility inclusion and exclusion criteria; a study was excluded at this stage if both reviewers agreed that it did not meet eligibility criteria. If reviewers did not agree on inclusion, they resolved the disagreement through discussion or with help of a third author. Papers meeting criteria for full-text review were read in their entirety by two authors (REF/SG) who abstracted information on study characteristics, methods, and results (Table 1); each researcher's findings were independently checked by the other.

2.4 | Data synthesis

All but one study eligible for inclusion in the review used a design in which a designated variable of interest was entered into an analytic model as an independent variable at Time 1 (T1) to predict the dependent variable of ME at Time 2 (T2). As an exception to this format, one study examined bidirectional covariation between eating pathology and ME among elementary school youth (Davis et al., 2018). As predictors emerged during the course of data abstraction, they were individually classed into common themes by two authors (REF/SG), with any differences in themes resolved by discussion across all authors. Statistical significance was relied upon to determine reported associations between predictor variables and ME, and effect sizes were considered when available.

2.5 | Quality rating

To critically appraise the quality of evidence provided by the included studies, we used the Quality Assessment Tool for Quantitative Studies (QATQS) developed by the Effective Public Health Practice Project (Thomas, Ciliska, Dobbins, & Micucci, 2004). Similar to study selection and data abstraction, two authors (REF/SG) conducted an independent dual review using the QATQS, and resolved discrepancies through discussion or consultation with a third author. A rating of *strong, moderate*, or *weak* was assigned for each of six domains: (1) selection bias, (2) study design, (3) confounders, (4) blinding, (5) data collection methods, and (6) withdrawals and dropouts. A global quality rating was then made according to the ratings from the six domains based on a scoring system recommended by Thomas et al. (2004); a *strong* global rating was made for studies that did not receive any weak ratings for each of the six domains. A *moderate* rating was made for studies that only received one weak rating across the six domains. A *weak* rating was made when a study received ≥ two weak ratings across the six domains. Consistent with previous reviews that have used the QATQS (Godfrey, Gallo, & Afari, 2015; Linardon, Gleeson, Yap, Murphy, & Brennan, 2019), we did not exclude any studies based on their quality rating. Full details of our quality assessment procedure are available in Supporting Information.

3 | RESULTS

3.1 | Characteristics of included studies

Titles and abstracts of 2,789 papers were scanned and full texts of 46 identified articles were reviewed (see Figure 1 for a PRISMA diagram flow of the literature search process).

Based on our inclusion and exclusion criteria, final review included 18 publications (Table 1). Eligible studies ($N = 18$) included youth or adolescent populations ($n = 11$); college or transition age youth populations ($n = 6$); and adult populations ($n = 1$). Studies represented populations from Australia ($n = 5$), Italy ($n = 1$), United Kingdom ($n = 6$), and the United States ($n = 6$). Nine studies did not report on socioeconomic status; some reported on parental education as a proxy for socioeconomic status, with a majority of studies reporting varied income levels across participants. Five studies did not report on race or ethnicity; across all studies that did report on race/ethnic identification, a majority of participants were White. Available study details are presented in Table 1.

3.2 | Key findings

Only one study examined bidirectional relations between eating behavior and ME (Davis et al., 2018). Although the researchers additionally examined purging and binge-eating behavior, significant effects in a cross-lagged model were only evidenced for T1 fasting, which predicted T2 ME, and T1 ME, which predicted T2 fasting; the duration of time in this bidirectional association was 6 months. Of note, in this study, ME demonstrated a linear decline over the 5 study years (with assessments over nine time points), and overall findings were invariant across gender.

Below, further results are organized according to the type of psychosocial variable that was assessed for its unidirectional association with ME. Patterns emerged across the literature, which we summarize below in light of their strength of evidence as assessed by the QATQS ratings. Specifically, findings from variables investigated in multiple studies with strong to moderate quality of evidence ratings (i.e., weight and shape concerns; thinness and eating expectancies; affect; and obsessive–compulsive [OC] traits) are highlighted in Table 2 and in the text. Variables represented by only one study with strong-moderate quality of evidence rating are also briefly described in the text. A majority of work defined ME based on its presence v. absence, according to a definition determined by the respective authors; we note exceptions to this approach when relevant.

3.2.1 | Weight and shape—Elevated body dissatisfaction and self-objectification predicted ME among college men (Dakanalis et al., 2016), with similar findings among adolescent girls (McCabe & Ricciardelli, 2004, 2006), but not among boys (McCabe & Ricciardelli, 2004, 2006, 2009). Distinct from body dissatisfaction, body importance (i.e., the degree to which body weight and shape reportedly impact self-identity and/or mood) predicted ME for both adolescent girls and boys (McCabe & Ricciardelli, 2006, 2009). Given further evidence of associations between strategies to increase muscularity and ME for some boys, and for girls who reported a focus on athletics (McCabe & Ricciardelli, 2006), it seems plausible that future research might benefit from assessing a broader and more comprehensive definition of body dissatisfaction and/or body importance when investigating factors that may increase risk for ME, across gender identity, and athletic status.

Of these studies, one qualified ME as those who “engaged in more intense exercise” (regardless of duration/frequency of exercise) “specifically to counteract the effects of

overeating" (Dakanalis et al., 2016), whereas the three other studies assessed ME as a form of "exercise dependence" (McCabe & Ricciardelli, 2004, 2006, 2009).

3.2.2 | Thinness and eating expectancies—In middle school boys and girls, greater thinness expectancies and eating expectancies were linked with the presence of ME (Davis et al., 2016a, 2016b). Despite some indication among college women that both athletic and thin-ideal internalization may impact ME, neither evidenced a significant unique contribution to ME when tested in one model (Homan, 2010). Although the directionality of these findings suggests positive associations with ME, the group of studies defined ME differently, with the former two studies defining ME by report of exercise as related to weight and shape (e.g., "a way to control your weight or because you ate a lot") (Davis et al., 2016a, 2016b), whereas the latter study defining ME by guilt or depression associated with missed exercise (e.g., "When I miss a scheduled exercise session, I may feel tense, irritable, or depressed") (Homan, 2010).

3.2.3 | Affect—Depressive symptoms or negative affect showed direct positive associations with ME in middle school boys (Davis et al., 2016a), middle school girls (Davis et al., 2016b), adolescent boys and girls (McCabe & Ricciardelli, 2009), late adolescent boys (Allen et al., 2013), and college men (Dakanalis et al., 2016). As a slight departure, one study found a negative effect of *positive* mood on ME for adolescent boys and girls, and a *negative* effect of depression on ME for adolescent girls, but not for boys (McCabe & Ricciardelli, 2006). Goodwin et al. (2014c) investigated the impact of different emotion regulation styles on the development of ME. The only unique significant predictor of ME for both boys and girls was "Internal Dysfunction," an emotion regulation style that describes the extent to which an adolescent copes with a situation by themselves (internally), incorporating such features as rumination (Phillips & Power, 2007).

Although these findings are generally consistent and together represent strong-moderate strength of evidence, definitions of ME had varying time frames of assessment (range 9 months to 6 years) and definition. Two studies evaluated ME based on Exercise Dependence Scale scores (Hausenblas & Downs, 2002a; McCabe & Ricciardelli, 2006, 2009), one study evaluated ME based on Compulsive Exercise Test scores (Goodwin et al., 2014c; Taranis, Touyz, & Meyer, 2011), and the remaining work defined ME as the presence or absence of exercising for weight control in the past month (Allen et al., 2013), three months (Dakanalis et al., 2016) or 2 weeks (Davis et al., 2016a, 2016b).

3.2.4 | Compulsivity—Providing some support for a compulsive characterization of ME (Meyer et al., 2011), and validation for similar cross-sectional work (Goodwin, Haycraft, Willis, & Meyer, 2011), within a community sample of adolescents, higher levels of OC and self-perfectionism (i.e., imposing an unrealistic desire to be perfect on oneself) predicted greater ME 2 years later among boys, but not girls (Goodwin et al., 2014b). This study defined ME using scores on the Compulsive Exercise Test (Taranis et al., 2011), which assesses an individual's compulsivity towards exercise (e.g., "I feel extremely guilty if I miss an exercise session").

In summary, the psychosocial variables that demonstrated associations with later ME broadly comprise body concern, affect regulation, and compulsivity. Together, these domains support a definition of ME as “compulsive” (Harris et al., 2020); features of a “compulsive” psychosocial model of ME (Meyer et al., 2011); and an overarching frame-work from which we might experimentally examine other potential affect-regulatory mechanisms that may maintain this behavior (Kolar & Gorrell, 2020).

4 | DISCUSSION

The current systematic review summarized existing evidence regarding psychosocial factors that appear to longitudinally contribute to the development of ME. In light of both the relative amount and strength of evidence, the domains of weight and shape concern and negative affect appear to be reliably supported in their association with ME. We detail below how these results improve our understanding of causes and maintaining factors of ME, and may inform both intervention strategies for clinicians treating EDs and research efforts.

4.1 | Clinical implications of findings

4.1.1 | Weight and shape, and Thinness expectancies—

Items in widely-used ED measures (e.g., Eating Disorder Examination; Fairburn, Cooper, & O'Connor, 1993) typically describe ME as a behavior that is intended to manipulate weight or shape. Therefore, it is notable that factors that do not appear to impact engagement in ME are pubertal timing (McCabe & Ricciardelli, 2004, 2006, 2009), and BMI or weight status (McCabe & Ricciardelli, 2006, 2009). However, greater risk for ME may result from thinness expectancies among middle school children (Davis et al., 2016a, 2016b) and among adolescents, from body dissatisfaction among girls (McCabe & Ricciardelli, 2004, 2006), and body importance, across gender (McCabe & Ricciardelli, 2006, 2009). In young adulthood, ME demonstrated associations with body dissatisfaction and weight and shape concerns for male undergraduates (Dakanalis et al., 2016), and with both athletic- and thin-ideal internalization among college women (Homan, 2010). Taken together, the evidence strongly suggests that across age and gender, risk for ME may be linked with a desire to manipulate body weight and shape.

Only one study explicitly evaluated drive for muscularity, but its findings suggest that strategies to build muscle may contribute to the presence of ME in adolescence (McCabe & Ricciardelli, 2006). In future work, measures should be selected for use that are sensitive to characterizing the pursuit of muscularity via weight-lifting/muscle-building activities, which may be more relevant or prevalent in men (Lavender, Brown, & Murray, 2017) or in female athletes (Negrin, Skemp, & Baumann, 2018) than a description of ME that promotes weight loss.

ME and dietary restriction are both means of influencing weight; a compelling finding emerged among studies directly investigating covarying relations between ME and ED symptoms, specific to fasting. Davis et al. (2018) found that fasting predicted ME and that ME predicted fasting; in earlier work, restriction and ME covaried over time in middle school children, with no difference across gender (Davis et al., 2016a, 2016b). These findings align with the rodent model of ABA (Epling & Pierce, 1989). That is, while

the ABA model cannot be expected to fully model human AN (Chowdhury, Chen, & Aoki, 2015), theoretical links between activity and energy depletion (Klok, Jakobsdottir, & Drent, 2007) suggest that biological mechanisms in energy regulation (e.g., leptin, ghrelin, dopamine, and endocannabinoids) should be considered alongside psychosocial models of ME in humans.

4.1.2 | Affect and compulsivity—With one exception (i.e., McCabe & Ricciardelli, 2006), the current review found a consistent role for negative affect in contributing to the development of ME, with a moderate to strong quality of evidence for these findings. In regard to study design, two studies defined ME by exercise dependence (McCabe & Ricciardelli, 2006, 2009), one defined ME by compulsivity (Goodwin et al., 2014c), and the remaining four defined ME by the presence of exercising for weight control (Allen et al., 2013; Dakanalis et al., 2016; Davis et al., 2016a, 2016b). Despite the differences in definitions, the consistency in these findings suggests that the association between negative affect and ME is robust to possible variations in the motivation for ME.

Of note, although one study reported a *negative* effect of anxiety on ME among adolescent boys, the authors also found a positive effect of OC traits and self-perfectionism on ME (Goodwin et al., 2014b), with further evidence that greater internalizing (i.e., ruminating) contributed to ME in a community sample of both boys and girls (Goodwin et al., 2014c). Both of these studies defined ME by scores on the Compulsive Exercise Test (Taranis et al., 2011); future work might investigate relations between OC traits and a measure of ME that includes different variants of ME (e.g., motivated by drive for muscularity or exclusively for weight control). While the strength of evidence in these studies is moderate, it aligns with consistent evidence from clinical ED research where lifetime comorbidity of AN and OCD is considerably elevated (35–44%; LaSalle et al., 2004; Pinto, Mancebo, Eisen, Pagano, & Rasmussen, 2006). Given that childhood OC symptoms and traits have been implicated in later risk for AN (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003; Micali et al., 2011) and recent work found OCD polygenic risk scores to predict ME in an epidemiological sample of adolescents (Yilmaz et al., 2020), longitudinal associations between OC features and ME warrant further inquiry.

Together, these findings underscore the importance of distinguishing between ME and adaptive exercise, in that while negative affect generally appears to increase risk for ME, burgeoning research indicates benefits of exercise to improve negative affect in samples with depression and anxiety (Asmundson, Fetzner, DeBoer, Otto, & Smits, 2013; Carek, Laibstain, & Carek, 2011; Heyman et al., 2012), which may have important implications for clinical ED samples. Specifically, given high prevalence rates of comorbid psychiatric disorders with EDs (Bühren et al., 2014; Salbach-Andrae et al., 2008; Udo & Grilo, 2019), as well as acknowledged benefit of physical activity in some ED samples (Achamrah, Coëffier, & Déchelotte, 2016; Calogero & Pedrotty, 2004; Carei, Fyfe-Johnson, Breuner, & Brown, 2010; Hausenblas, Cook, & Chittester, 2008), careful titration of *adaptive* exercise within the context of ED treatment may be therapeutically indicated (Cook et al., 2016; Cook & Leininger, 2017; Hausenblas et al., 2008).

Considering all findings in light of their strength of evidence, future screening, and research efforts should be oriented toward the following psychosocial domains and their links with ME. First, across age and gender, evidence supports associations between increased desire to manipulate body weight and shape and later engagement in ME. Some findings support the role of body importance (McCabe & Ricciardelli, 2006; McCabe & Ricciardelli, 2009), thinness and eating expectancies (Davis et al., 2016a, 2016b), thin- and athletic-ideal internalization (Homan, 2010), and muscularity concerns (McCabe & Ricciardelli, 2006), suggesting that assessment of the motivation for ME must include broader features that lie beyond "body dissatisfaction" or a desire for weight loss. In addition, depression and OC features may be particularly implicated in later onset of ME, for which increased screening in community samples, across gender, is warranted.

4.2 | Limitations in the evidence base

Findings are limited in their external validity as few studies reported extensively on participant characteristics that may impact both ME and ED behavior (e.g., socioeconomic status), and while over half of the studies included information on race and ethnicity, a majority of studies were comprised of White participants. There are also multiple instances where author groups are represented several times in different articles (i.e., using study design, assessment methods, and participant populations that were similar to one another). In particular, partial inclusion of a broader sample in two different studies, while unique in analyses and research question, are limited in their overall generalizability. Longitudinal studies are naturally vulnerable to attrition, and many authors included data only for participants for whom all assessment time points were available. While these analytic decisions were appropriately adjusted for (e.g., by using imputation methods for missing data), we should exercise caution in interpretating selectively reported data.

The limitation most germane to this review is the heterogeneity in how ME was assessed, quantified, and conceptualized, and the resulting challenge in drawing meaningful conclusions across studies. Consensus does not exist across the extant literature in determining differences between normative and problematic physical activity. Although most researchers and clinicians who focus their work on individuals with EDs would report that they assess for "problematic exercise behavior," the current review reveals how disparate and inconsistent the measures and definitions for ME are, and how difficult it is to integrate and interpret findings. For instance, roughly half of the studies relied on single binary items to determine whether participants endorsed ME. Four studies (~20%) earned a global rating of "strong" strength of evidence based on methodological rigor, but these studies used items only, and did not use a complete measure of ME. Of the remaining studies that used full measures or an adaptation, only four used a measure with psychometric validity in ED samples (e.g., CET; Harris et al., 2020; Meyer et al., 2016; Taranis et al., 2011). This limitation may be less important, as few samples in the current review were comprised of individuals with EDs; however, we are then limited in our ability to extend our findings across a broader conceptualization of ME within the context of more severe eating pathology. We acknowledge an additional challenge in synthesizing this literature, namely that engaging in ME "to manipulate body weight or shape" may have been included in both the assessment of the behavior (i.e., a criterion for endorsement) as well as an outcome (i.e.,

a stated reason for engaging in ME). This may have introduced a tautology in some of the included studies.

Although a majority of studies included covariates in their reported statistical models, only 10 studies explicitly report that T1 ME had been specifically accounted for in longitudinal analyses. Further, although a majority of studies defined group comparisons by the presence or absence of ME, the definition of ME across studies varied enormously. For example, some work defined a categorical group with ME as those who reported exercise as a means to control weight and shape (e.g., Davis et al., 2016a, 2016b); in contrast, other work defined a categorical group with ME as those who reported feelings of guilt or depression related to a missed exercise session (e.g., Homan, 2010). As such, both the heterogeneity and lack of quality in assessment of ME did not justify meta-analytic evaluation in the current review; quantitative synthesis of disparate and/or biased findings would likely yield more hindrance than benefit in interpretation. It is essential that moving forward, empirical longitudinal study of ME not rely on single items, but rather, use complete and validated measures. Further, to build cohesive understanding of ME in the literature, it is critical that, as an improved biobehavioral model of ME is established, we begin to minimize the number of validated self-report measures of ME employed across samples. Finally, only 10 of the 18 studies specifically stated that they included accounted for T1 ME in statistical analyses. With this limitation, and other methodological issues, less than a quarter of the studies met criteria for strong quality of evidence, and almost half were determined to have weak quality of evidence. Therefore, although we have made an effort to highlight the most robust evidence among studies included in the current review, we must also consider the evidence in light of the problems of definition and rigor that beset this field of inquiry.

4.3 | Implications for future research

We have highlighted, both in the results and noted limitations, areas that warrant further inquiry and methodological improvement. Several additional areas of research suggested by the current review are important to mention. For one, studies that include predictors of ME that are not psychosocial are not included in this review. For example, recent work prospectively exploring childhood neurocognitive profiles found that better scores on working memory tasks in childhood were associated with decreased risk of fasting and increased risk of ME in adolescence (Schaumberg et al., 2020). This line of inquiry is nascent, but highlights a need for future work investigating potential neurobiological mechanisms related to learning and reinforcement that may serve to initiate and perpetuate ME.

Such investigation would inform proposed psychosocial models of ME, and a broader understanding of how physical activity is both maintained within EDs, and also contributes to the severity of the ED itself. Although evidence suggests that the compulsive nature of ME aligns more with ED pathology than its addictive qualities (Scharmer, Gorrell, Schaumberg, & Anderson, 2020), the current review does little to support a model of exercise dependence (Bamber et al., 2003; Hausenblas & Downs, 2002b) or a continuum model for obligatory exercise (Elbourne & Chen, 2007). Evidence supporting negative affect avoidance and OC features in the current review does offer support for domains

within a model of compulsive exercise (Meyer et al., 2011); however, no longitudinal study to date has fully tested all elements in confirmation of this model. The current review only provides cursory information regarding the etiology, and function of ME. The reinforcing aspects of exercise, including its anxiolytic (Favaro, Caregaro, Burlina, & Santonastaso, 2000; Guarda, Schreyer, Boersma, Tamashiro, & Moran, 2015) and naturally rewarding (Davis & Woodside, 2002; Guarda et al., 2015) properties, likely play a pivotal role in the development and perpetuation of ME. Broadening this possibility, Flack, Pankey, Ufholz, Johnson, and Roemmich (2019) reported a genetic contribution to exercise reinforcement, tolerance for exercise intensity, and moderate-vigorous physical activity. Similarly, a positive genetic correlation between AN and measured physical activity suggests an underlying shared genetic etiology between these two traits (Watson et al., 2019). As such, comprehensive multi-level study of proposed psychosocial models of ME, in tandem with neurocognitive, psychobiological, and genetic underpinnings of exercise behavior, are critical lines of further research.

5 | CONCLUSIONS

Evidence from longitudinal studies suggests that several psychosocial factors contribute to the development of ME in the context of EDs. In particular, findings provide support for weight and shape concerns and negative affect in their precursory role in increasing risk for ME across varied samples. Preliminary evidence was provided for a dynamic relationship between dietary restriction and ME, underscoring the importance of further psychobiological research regarding the multi-faceted association between activity and intake. The majority of evidence in the current review was methodologically constrained by limited assessment of ME, highlighting the primary importance of the consistent implementation of empirically validated measures of ME in future investigation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

REFERENCES

Achamrah N, Coëffier M, & Déchelotte P (2016). Physical activity in patients with anorexia nervosa. Nutrition Reviews, 74(5), 301–311. 10.1093/nutrit/nuw001 [PubMed: 27052638]

Adams J, & Kirkby RJ (2002). Excessive exercise as an addiction: A review. Addiction Research & Theory, 10(5), 415–437. 10.1080/1606635021000032366

Adkins EC, & Keel PK (2005). Does “excessive” or “compulsive” best describe exercise as a symptom of bulimia nervosa. The International Journal of Eating Disorders, 38, 24–29. 10.1002/eat.20140 [PubMed: 15991218]

Allen KL, Byrne SM, Forbes D, & Oddy WH (2009). Risk factors for full- and partial-syndrome early adolescent eating disorders: A population-based pregnancy cohort study. Journal of the American Academy of Child & Adolescent Psychiatry, 48(8), 800–809. 10.1097/chi.0b013e3181a8136d [PubMed: 19564799]

Allen KL, Crosby RD, Oddy WH, & Byrne SM (2013). Eating disorder symptom trajectories in adolescence: Effects of time, participant sex, and early adolescent depressive symptoms. Journal of Eating Disorders, 1(1), 32. 10.1186/2050-2974-1-32 [PubMed: 24999411]

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders. doi:10.1176/appi.books.9780890425596

Anderluh MB, Tchanturia K, Rabe-Hesketh S, & Treasure J (2003). Childhood obsessive-compulsive personality traits in adult women with eating disorders: Defining a broader eating disorder phenotype. American Journal of Psychiatry, 160(2), 242–247. 10.1176/appi.ajp.160.2.242

Asmundson GJG, Fetzner MG, DeBoer LB, Otto MW, & Smits JAJ (2013). Let's get physical: A contemporary review of the anxiolytic effects of exercise for anxiety and its disorders. Depression and Anxiety, 30(4), 362–373. 10.1002/da.22043 [PubMed: 23300122]

Bamber DJ, Cockerill IM, Rodgers S, & Carroll D (2003). Diagnostic criteria for exercise dependence in women. British Journal of Sports Medicine, 37, 393–400. [PubMed: 14514528]

Booth A, Clarke M, Dooley G, Ghersi D, Moher D, Petticrew M, & Stewart L (2012). The nuts and bolts of PROSPERO: An international prospective register of systematic reviews. Systematic Reviews, 1(1), 1–9. [PubMed: 22587946]

Boyd A, Golding J, Macleod J, Lawlor DA, Fraser A, Henderson J, … Davey Smith G (2013). Cohort profile: The “children of the 90s”—The index offspring of the Avon longitudinal study of parents and children. International Journal of Epidemiology, 42(1), 111–127. 10.1093/ije/dys064 [PubMed: 22507743]

Brewerton TD, Stellefson EJ, Hibbs N, Hodges EL, & Cochrane CE (1995). Comparison of eating disorder patients with and without compulsive exercising. The International Journal of Eating Disorders, 17(4), 413–416. [PubMed: 7620482]

Bühren K, Schwarte R, Fluck F, Timmesfeld N, Krei M, Egberts K, … Herpertz-Dahlmann B (2014). Comorbid psychiatric disorders in female adolescents with first-onset anorexia nervosa: Comorbidity in adolescent anorexia nervosa. European Eating Disorders Review, 22 (1), 39–44. 10.1002/erv.2254 [PubMed: 24027221]

Bulik CM, Berkman ND, Brownley KA, Sedway JA, & Lohr KN (2007). Anorexia nervosa treatment: A systematic review of randomized controlled trials. International Journal of Eating Disorders, 40(4), 310–320. 10.1002/eat.20367

Calogero RM, & Pedrotty KN (2004). The practice and process of healthy exercise: An investigation of the treatment of exercise abuse in women with eating disorders. Eating Disorders, 12(4), 273–291. 10.1080/10640260490521352 [PubMed: 16864521]

Carei TR, Fyfe-Johnson AL, Breuner CC, & Brown MA (2010). Randomized controlled clinical trial of yoga in the treatment of eating disorders. Journal of Adolescent Health, 46(4), 346–351. 10.1016/j.jadohealth.2009.08.007

Carek PJ, Laibstain SE, & Carek SM (2011). Exercise for the treatment of depression and anxiety. International Journal of Psychiatry in Medicine, 41(1), 15–28. 10.2190/PM.41.1.c [PubMed: 21495519]

Casper RC (2006). The ‘drive for activity’ and “restlessness” in anorexia nervosa: Potential pathways. Journal of Affective Disorders, 92(1), 99–107. 10.1016/j.jad.2005.12.039 [PubMed: 16448703]

Casper RC (2018). Not the function of eating, but spontaneous activity and energy expenditure, reflected in “restlessness” and a “drive for activity” appear to be Dysregulated in anorexia nervosa: Treatment implications. Frontiers in Psychology, 9, 2303. 10.3389/fpsyg.2018.02303 [PubMed: 30532724]

Chowdhury TG, Chen Y-W, & Aoki C (2015). Using the activity-based anorexia rodent model to study the neurobiological basis of anorexia nervosa. Journal of Visualized Experiments, 104, 52927. 10.3791/52927

Cook B, Hausenblas H, Crosby RD, Cao L, & Wonderlich SA (2015). Exercise dependence as a mediator of the exercise and eating disorders relationship: A pilot study. Eating Behaviors, 16, 9–12. [PubMed: 25464059]

Cook B, Karr TM, Zunker C, Mitchell JE, Thompson R, Sherman R, … Wonderlich SA (2013). Primary and secondary exercise dependence in a community-based sample of road race runners. Journal of Sport and Exercise Psychology, 35(5), 464–469. 10.1123/jsep.35.5.464 [PubMed: 24197714]

Cook B, & Leininger L (2017). The ethics of exercise in eating disorders: Can an ethical principles approach guide the next generation of research and clinical practice? Journal of Sport and Health Science, 6(3), 295–298. 10.1016/j.jshs.2017.03.004 [PubMed: 30356587]

Cook BJ, Wonderlich SA, Mitchell JE, Thompson R, Sherman R, & McCallum K (2016). Exercise in eating disorders treatment: Systematic review and proposal of guidelines. Medicine and Science in Sports and Exercise, 48(7), 1408–1414. 10.1249/MSS.0000000000000912 [PubMed: 26909533]

Cunningham HE, Pearman S, & Brewerton TD (2016). Conceptualizing primary and secondary pathological exercise using available measures of excessive exercise: Conceptualizing pathological exercise. International Journal of Eating Disorders, 49(8), 778–792. 10.1002/eat.22551

Dakanalis A, Clerici M, Caslini M, Gaudio S, Serino S, Riva G, & Carrà G (2016). Predictors of initiation and persistence of recurrent binge eating and inappropriate weight compensatory behaviors in college men: College Men's ED Symptoms and predictors. International Journal of Eating Disorders, 49(6), 581–590. 10.1002/eat.22535

Dalle Grave R, Calugi S, & Marchesini G (2008). Compulsive exercise to control shape or weight in eating disorders: Prevalence, associated features, and treatment outcome. Comprehensive Psychiatry, 49(4), 346–352. 10.1016/j.comppsych.2007.12.007 [PubMed: 18555054]

Danielsen M, Rø Ø, & Bjørnelv S (2018). How to integrate physical activity and exercise approaches into inpatient treatment for eating disorders: Fifteen years of clinical experience and research. Journal of Eating Disorders, 6(1), 1–9. [PubMed: 29344359]

Davis C, & Kaptein S (2006). Anorexia nervosa with excessive exercise: A phenotype with close links to obsessive-compulsive disorder. Psychiatry Research, 142(2–3), 209–217. 10.1016/j.psychres.2005.11.006 [PubMed: 16697469]

Davis C, & Woodside DB (2002). Sensitivity to the rewarding effects of food and exercise in the eating disorders. Comprehensive Psychiatry, 43 (3), 189–194. 10.1053/comp.2002.32356 [PubMed: 11994836]

Davis C, & Fox J (1993). Excessive exercise and weight preoccupation in women. Addictive Behaviors, 18(2), 201–211. 10.1016/0306-4603(93)90050-J [PubMed: 8506791]

Davis HA, Guller L, & Smith GT (2016a). Developmental trajectories of boys' driven exercise and fasting during the middle school years. Journal of Abnormal Child Psychology, 44(7), 1309–1319. 10.1007/s10802-015-0119-3 [PubMed: 26707543]

Davis HA, Guller L, & Smith GT (2016b). Developmental trajectories of compensatory exercise and fasting behavior across the middle school years. Appetite, 107, 330–338. 10.1016/j.appet.2016.08.098 [PubMed: 27544806]

Davis HA, Ortiz AML, & Smith GT (2018). The occurrence and covariation of binge eating and compensatory behaviors across early to mid-adolescence. Journal of Pediatric Psychology, 43(4), 402–412. 10.1093/jpepsy/jsx113 [PubMed: 29048479]

Dittmer N, Jacobi C, & Voderholzer U (2018). Compulsive exercise in eating disorders: Proposal for a definition and a clinical assessment. Journal of Eating Disorders, 6(1). 10.1186/s40337-018-0219-x

Dittmer N, Voderholzer U, Mönch C, Cuntz U, Jacobi C, & Schlegl S (2020). Efficacy of a specialized group intervention for compulsive exercise in inpatients with anorexia nervosa: A randomized controlled trial. Psychother Psychosom, 6, 1–13. 10.1159/000504583

Elbourne KE, & Chen J (2007). The continuum model of obligatory exercise. Journal of Psychosomatic Research, 62(1), 73–80. 10.1016/j.jpsychores.2004.12.003 [PubMed: 17188123]

Epling WF, & Pierce WD (1989). Excessive activity and anorexia in rats. In Pirke KM, Wuttke W, & Schweiger U (Eds.), The menstrual cycle and its disorders (pp. 79–87). Berlin, Heidelberg: Springer. 10.1007/978-3-642-74631-4_9

Erskine HE, Whiteford HA, & Pike KM (2016). The global burden of eating disorders. Current Opinion in Psychiatry, 29(6), 346–353. 10.1097/YCO.0000000000000276 [PubMed: 27532942]

Fairburn CG, Cooper Z, & O'Connor M (1993). The eating disorder examination. International Journal of Eating Disorders, 6, 1–8.

Favaro A, Caregaro L, Burlina AB, & Santonastaso P (2000). Tryptophan levels, excessive exercise, and nutritional status in anorexia nervosa. Psychosomatic Medicine, 62(4), 535–538. 10.1097/00006842-200007000-00012 [PubMed: 10949099]

Flack K, Pankey C, Ufholz K, Johnson L, & Roemmich JN (2019). Genetic variations in the dopamine reward system influence exercise reinforcement and tolerance for exercise intensity. Behavioural Brain Research, 375, 112148. 10.1016/j.bbr.2019.112148 [PubMed: 31404557]

Frank GKW, & Shott ME (2016). The role of psychotropic medications in the management of Anorexia Nervosa: Rationale, evidence and future prospects. CNS Drugs, 30(5), 419–442. 10.1007/s40263-016-0335-6 [PubMed: 27106297]

Godfrey KM, Gallo LC, & Afari N (2015). Mindfulness-based interventions for binge eating: A systematic review and meta-analysis. Journal of Behavioral Medicine, 38(2), 348–362. 10.1007/s10865-014-9610-5 [PubMed: 25417199]

Goodwin H, Haycraft E, & Meyer C (2014a). Sociocultural risk factors for compulsive exercise: A prospective study of adolescents: Compulsive exercise. European Eating Disorders Review, 22(5), 360–365. 10.1002/erv.2309 [PubMed: 25059109]

Goodwin H, Haycraft E, & Meyer C (2014b). Psychological risk factors for compulsive exercise: A longitudinal investigation of adolescent boys and girls. Personality and Individual Differences, 68, 83–86. 10.1016/j.paid.2014.03.048

Goodwin H, Haycraft E, & Meyer C (2014c). Emotion regulation styles as longitudinal predictors of compulsive exercise: A twelve month prospective study. Journal of Adolescence, 37(8), 1399–1404. 10.1016/j.adolescence.2014.10.001 [PubMed: 25448836]

Goodwin H, Haycraft E, Willis AM, & Meyer C (2011). Compulsive exercise: The role of personality, psychological morbidity, and disordered eating. International Journal of Eating Disorders, 44(7), 655–660.

Guarda AS, Schreyer CC, Boersma GJ, Tamashiro KL, & Moran TH (2015). Anorexia nervosa as a motivated behavior: Relevance of anxiety, stress, fear and learning. Physiology & Behavior, 152 (B, 466–472. 10.1016/j.physbeh.2015.04.007 [PubMed: 25846837]

Harris A, Hay P, & Touyz S (2020). Psychometric properties of instruments assessing exercise in patients with eating disorders: A systematic review. Journal of Eating Disorders, 8(1), 45. 10.1186/s40337-020-00315-2 [PubMed: 32884810]

Hausenblas HA, Cook BJ, & Chittester NI (2008). Can exercise treat eating disorders. Exercise and Sport Sciences Reviews, 36, 43–47. [PubMed: 18156953]

Hausenblas HA, & Downs DS (2002). How much is too much? The development and validation of the exercise dependence scale. Psychology and Health, 17(4), 387–404.

Hausenblas HA, & Downs DS (2002). Exercise dependence: A systematic review. Psychology of Sport and Exercise, 3(2), 89–123. 10.1016/S1469-0292(00)00015-7

Hay P, Touyz S, Arcelus J, Pike K, Attia E, Crosby RD, … Meyer C (2018). A randomized controlled trial of the compuLsive exercise activity TheraPy (LEAP): A new approach to compulsive exercise in anorexia nervosa. International Journal of Eating Disorders, 51(8), 999–1004. 10.1002/eat.22920

Haynos AF, & Fruzzetti AE (2011). Anorexia nervosa as a disorder of emotion dysregulation: Evidence and treatment implications. Clinical Psychology: Science and Practice, 18(3), 183–202. 10.1111/j.1468-2850.2011.01250.x

Heyman E, Gamelin FX, Goekint M, Piscitelli F, Roelands B, Leclair E, … Meeusen R (2012). Intense exercise increases circulating endocannabinoid and BDNF levels in humans—Possible implications for reward and depres-sion. Psychoneuro, 37(6), 844–851.

Holtkamp K, Hebebrand J, & Herpertz-Dahlmann B (2004). The contribution of anxiety and food restriction on physical activity levels in acute anorexia nervosa. The International Journal of Eating Disorders, 36 (2), 163–171. 10.1002/eat.20035 [PubMed: 15282686]

Homan K (2010). Athletic-ideal and thin-ideal internalization as prospective predictors of body dissatisfaction, dieting, and compulsive exercise. Body Image, 7(3), 240–245. 10.1016/j.bodyim.2010.02.004 [PubMed: 20226748]

Klok MD, Jakobsdottir S, & Drent ML (2007). The role of leptin and ghrelin in the regulation of food intake and body weight in humans: A review. Obesity Reviews, 8(1), 21–34. 10.1111/j.1467-789X.2006.00270.x [PubMed: 17212793]

Kolar DR, & Gorrell S (2020). A call to experimentally study acute affect-regulation mechanisms specific to driven exercise in eating disorders. International Journal of Eating Disorders, 54(3), 280–286. 10.1002/eat.23427

LaSalle VH, Cromer KR, Nelson KN, Kazuba D, Justement L, & Murphy DL (2004). Diagnostic interview assessed neuropsychiatric disorder comorbidity in 334 individuals with obsessive-compulsive disorder. Depression and Anxiety, 19(3), 163–173. 10.1002/da.20009 [PubMed: 15129418]

Lavender JM, Brown TA, & Murray SB (2017). Men, muscles, and eating disorders: An overview of traditional and muscularity-oriented disordered eating. Current Psychiatry Reports, 19(6). 10.1007/s11920-017-0787-5

Linardon J, Gleeson J, Yap K, Murphy K, & Brennan L (2019). Meta-analysis of the effects of third-wave behavioural interventions on disordered eating and body image concerns: Implications for eating disorder prevention. Cognitive Behaviour Therapy, 48(1), 15–38. 10.1080/16506073.2018.1517389 [PubMed: 30307377]

Lock J, & Le Grange D (2018). Family-based treatment: Where are we and where should we be going to improve recovery in child and adolescent eating disorders. International Journal of Eating Disorders, 52(4), 481–487. 10.1002/eat.22980

McCabe M, & Ricciardelli L (2004). A longitudinal study of pubertal timing and extreme body change behaviors among adolescent boys and girls. Adolescence, 39(153), 145–166. [PubMed: 15230071]

McCabe MP, & Ricciardelli LA (2006). A prospective study of extreme weight change behaviors among adolescent boys and girls. Journal of Youth and Adolescence, 35(3), 402–411. 10.1007/s10964-006-9062-5

McCabe MP, & Ricciardelli LA (2009). Extreme weight change behaviours: Are overweight and normal weight adolescents different, and does this vary over time? European Eating Disorders Review, 17(4), 301–314. 10.1002/erv.929 [PubMed: 19378347]

Meyer C, Plateau CR, Taranis L, Brewin N, Wales J, & Arcelus J (2016). The compulsive exercise test: Confirmatory factor analysis and links with eating psychopathology among women with clinical eating disorders. Journal of Eating Disorders, 4(1), 22. 10.1186/s40337-016-0113-3 [PubMed: 27547403]

Meyer C, Taranis L, Goodwin H, & Haycraft E (2011). Compulsive exercise and eating disorders. European Eating Disorders Review, 19(3), 174–189. 10.1002/erv.1122 [PubMed: 21584911]

Micali N, Hilton K, Natatani E, Heyman I, Turner C, & Mataix-Cols D (2011). Is childhood OCD a risk factor for eating disorders later in life? A longitudinal study. Psychological Medicine, 41(12), 2507–2513. 10.1017/S003329171100078X [PubMed: 21733209]

Moher D, Liberati A, Tetzlaff J, Altman DG, & Prisma Group. (2009). Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. PLoS Medicine, 6(7), e1000097. [PubMed: 19621072]

Mond JM, & Calogero RM (2009). Excessive exercise in eating disorder patients and in healthy women. Australian & New Zealand Journal of Psychiatry, 43(3), 227–234.

Monell E, Levallius J, Forsén Mantilla E, & Birgegard A (2018). Running on empty – A nationwide large-scale examination of compulsive exercise in eating disorders. Journal of Eating Disorders, 6, 11. 10.1186/s40337-018-0197 [PubMed: 29942510]

Negrin AR, Skemp KM, & Baumann DD (2018). The effect of media exposure on body satisfaction and drive for muscularity in women. Health Primary Care, 2, 1–8.

Newnham JP, Evans SF, Michael CA, Stanley FJ, & Landau LI (1993). Effects of frequent ultrasound during pregnancy: A randomised controlled trial. The Lancet, 342(8876), 887–891. 10.1016/0140-6736(93)91944-h

Noetel M, Dawson L, Hay P, & Touyz S (2017). The assessment and treatment of unhealthy exercise in adolescents with anorexia nervosa: A Delphi study to synthesize clinical knowledge. International Journal of Eating Disorders, 50(4), 378–388. 10.1002/eat.22657

Phillips KFV, & Power MJ (2007). A new self-report measure of emotion regulation in adolescents: The regulation of emotions questionnaire. Clinical Psychology & Psychotherapy, 14(2), 145–156. 10.1002/cpp.523

Pinto A, Mancebo MC, Eisen JL, Pagano ME, & Rasmussen SA (2006). The brown longitudinal obsessive compulsive study: Clinical features and symptoms of the sample at intake. The Journal of Clinical Psychiatry, 67(05), 703–711. 10.4088/JCP.v67n0503 [PubMed: 16841619]

Pritchard ME, & Beaver JL (2012). Do exercise motives predict obligatory exercise? Eating Behaviors, 13(2), 139–141. 10.1016/j.eatbeh.2011.11.012 [PubMed: 22365798]

Quesnel DA, Libben M, Oelke D, Clark M, Willis-Stewart S, & Caperchione CM (2018). Is abstinence really the best option? Exploring the role of exercise in the treatment and management of eating disorders. Eating Disorders, 26(3), 290–310. [PubMed: 29131718]

Rizk M, Lalanne C, Berthoz S, Kern L, & Godart N (2015). Problematic exercise in anorexia nervosa: Testing potential risk factors against different definitions. PLoS One, 10(11), e0143352. 10.1371/journal.pone.0143352 [PubMed: 26618359]

Salbach-Andrae H, Lenz K, Simmendinger N, Klinkowski N, Lehmkuhl U, & Pfeiffer E (2008). Psychiatric comorbidities among female adolescents with anorexia nervosa. Child Psychiatry and Human Development, 39(3), 261–272. 10.1007/s10578-007-0086-1 [PubMed: 17987378]

Sawyer SM, Whitelaw M, Le Grange D, Yeo M, & Hughes EK (2016). Physical and psychological morbidity in adolescents with atypical anorexia nervosa. Pediatrics, 137(4), e20154080. 10.1542/peds.2015-4080 [PubMed: 27025958]

Schaal K, Tafflet M, Nassif H, Thibault V, Pichard C, Alcotte M, … Toussaint J-F (2011). Psychological balance in high level athletes: Gender-based differences and sport-specific patterns. PLoS One, 6(5), e19007. 10.1371/journal.pone.0019007 [PubMed: 21573222]

Scharmer C, Gorrell S, Schaumberg K, & Anderson D (2020). Compulsive exercise or exercise dependence? Clarifying conceptualizations of exercise in the context of eating disorder pathology. Psychology of Sport and Exercise, 46,101586. 10.1016/j.psychsport.2019.101586

Schaumberg K, Brosof LC, Lloyd EC, Yilmaz Z, Bulik CM, Zerwas SC, & Micali N (2020). Prospective associations between childhood neuropsychological profiles and adolescent eating disorders. European Eating Disorders Review, 28(2), 156–169. 10.1002/erv.2721 [PubMed: 31994257]

Schleien JL, & Bardone-Cone AM (2016). Competitiveness as a moderator of the relation between appearance-related factors and disordered eating behaviors. Body Image, 17, 30–37. 10.1016/j.bodyim.2016.01.009 [PubMed: 26922697]

Scott CL, Haycraft E, & Plateau CR (2020). The influence of social networks within sports teams on athletes' eating and exercise psychopathology: A longitudinal study. Psychology of Sport and Exercise, 52, 101786. 10.1016/j.psychsport.2020.101786

Scott CL, Plateau CR, & Haycraft E (2020). Teammate influences, psychological well-being, and athletes' eating and exercise psychopathology: A moderated mediation analysis. International Journal of Eating Disorders, 53(4), 564–573. 10.1002/eat.23222

Seigel K, & Hetta J (2001). Exercise and eating disorder symptoms among young females. Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity, 6, 32–39.

Shroff H, Reba L, Thornton LM, Tozzi F, Klump KL, Berrettini WH, … Bulik CM (2006). Features associated with excessive exercise in women with eating disorders. The International Journal of Eating Disorders, 39(6), 454–461. 10.1002/eat.20247 [PubMed: 16637047]

Smith AR, Fink EL, Anestis MD, Ribeiro JD, Gordon KH, Davis H, … Joiner TE (2013). Exercise caution: Over-exercise is associated with suicidality among individuals with disordered eating. Psychiatry Research, 206(2–3), 246–255. 10.1016/j.psychres.2012.11.004 [PubMed: 23219104]

Solenberger SE (2001). Exercise and eating disorders: A 3-year inpatient hospital record analysis. Eating Behaviors, 2(2), 151–168. [PubMed: 15001043]

Solmi F, Mascarell MC, Zammit S, Kirkbride JB, & Lewis G (2019). Polygenic risk for schizophrenia, disordered eating behaviours and body mass index in adolescents. British Journal of Psychiatry, 215(1), 428–433.Embase Medline. 10.1192/bjp.2019.39

Steinhausen HC, Grigoroiu-Serbanescu M, Boyadjieva S, Neumärker KJ, & Winkler Metzke C (2008). Course and predictors of rehospitalization in adolescent anorexia nervosa in a multisite study. The International Journal of Eating Disorders, 41(1), 29–36. 10.1002/eat.20414 [PubMed: 17647278]

Stiles-Shields C, DclinPsy BB, Lock J, & Le Grange D (2015). The effect of driven exercise on treatment outcomes for adolescents with anorexia and bulimia nervosa. International Journal of Eating Disorders, 48(4), 392–396. 10.1002/eat.22281

Swerdlow BA, Pearlstein JG, Sandel DB, Mauss IB, & Johnson SL (2020). Maladaptive behavior and affect regulation: A functionalist perspective. Emotion, 20(1), 75–79. 10.1037/emo0000660 [PubMed: 31961182]

Taranis L, Touyz S, & Meyer C (2011). Disordered eating and exercise: Development and preliminary validation of the compulsive exercise test (CET). European Eating Disorders Review, 19(3), 256–268. 10.1002/erv.1108 [PubMed: 21584918]

Thomas BH, Ciliska D, Dobbins M, & Micucci S (2004). A process for systematically reviewing the literature: Providing the research evidence for public health nursing interventions. Worldviews on Evidence-Based Nursing, 1(3), 176–184. 10.1111/j.1524-475X.2004.04006.x [PubMed: 17163895]

Udo T, & Grilo CM (2019). Psychiatric and medical correlates of *DSM-5* eating disorders in a nationally representative sample of adults in the United States. International Journal of Eating Disorders, 52(1), 42–50. 10.1002/eat.23004

Van Hoeken D, & Hoek HW (2020). Review of the burden of eating disorders: Mortality, disability, costs, quality of life, and family burden. Current Opinion in Psychiatry, 33(6), 521–527. 10.1097/yco.0000000000000641 [PubMed: 32796186]

Watson HJ, Yilmaz Z, Thornton LM, Hübel C, Coleman JRI, Gaspar HA, … Bulik CM (2019). Genome-wide association study identifies eight risk loci and implicates metabo-psychiatric origins for anorexia nervosa. Nature Genetics. 51, 1207–1214. 10.1038/s41588-019-0439-2 [PubMed: 31308545]

Yilmaz Z, Schaumberg K, Halvorsen M, Goodman EL, Brosof LC, Crowley JJ, Anorexia Nervosa Genetics Initiative, Eating Disorders Working Group of the Psychiatric Genomics Consortium, Tourette Syndrome/Obsessive-Compulsive Disorder Working Group of the Psychiatric Genomics Consortium, Mathews CA, Mattheisen M, Breen G, Bulik CM, Micali N, & Zerwas SC (2020). Predicting eating disorder and anxiety symptoms using anorexia nervosa and obsessive-compulsive disorder polygenic scores [preprint]. Psychiatry and Clinical Psychology. 10.1101/2020.07.02.20142844

Young S, Touyz S, Meyer C, Arcelus J, Rhodes P, Madden S, … Hay P (2018). Relationships between compulsive exercise, quality of life, psychological distress and motivation to change in adults with anorexia nervosa. Journal of Eating Disorders, 6(1), 10.1186/s40337-018-0188-0

Zhan C, Heatherington L, & Klingenberg B (2020). Disordered eating- and exercise-related behaviors and cognitions during the first year college transition. Journal of American College Health, 1–12. 10.1080/07448481.2020.1775608

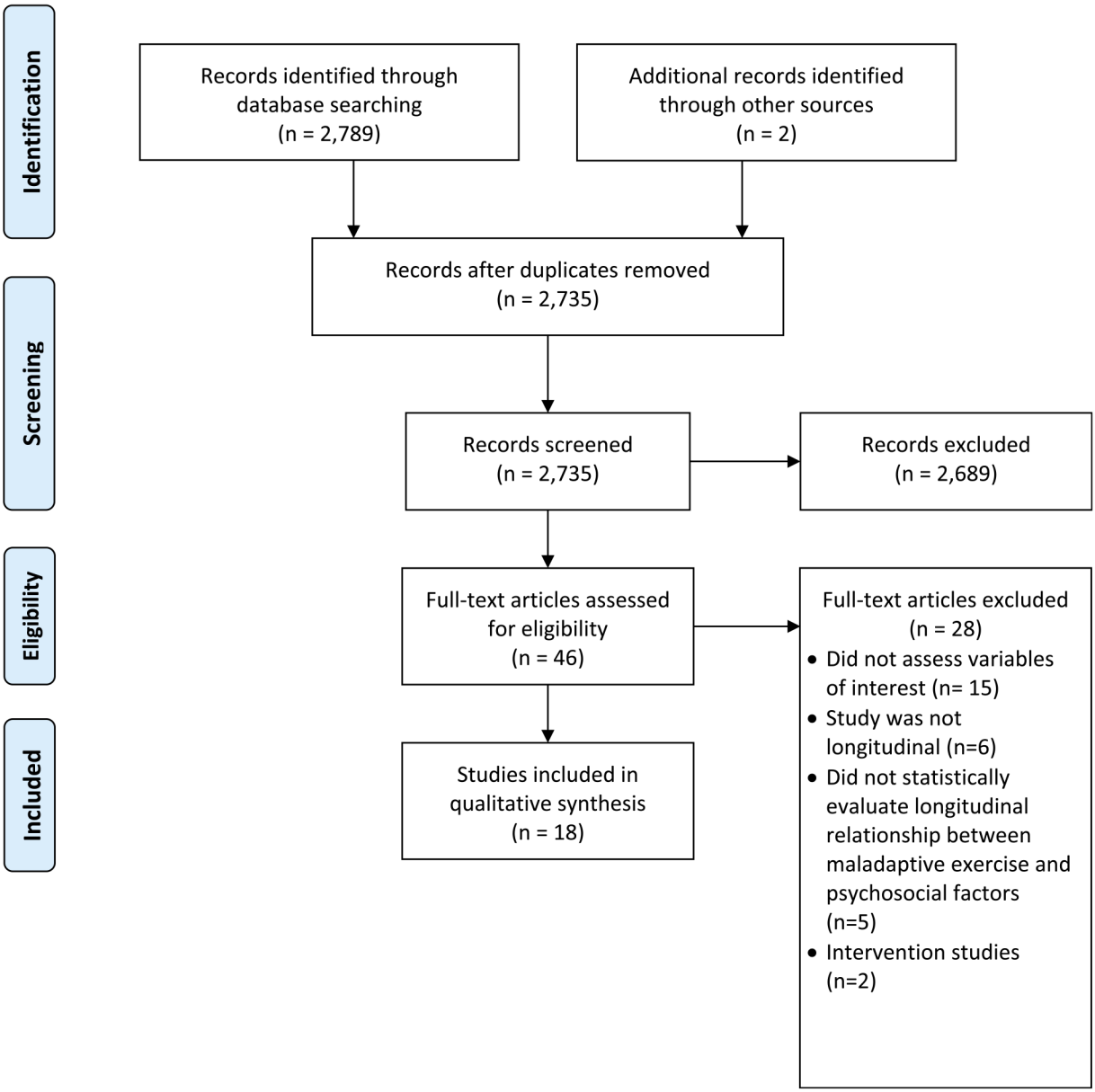

FIGURE 1.
PRISMA flow diagram

TABLE 1

Study characteristics, key findings, and overall quality rating of eligible studies

Study and location	Sample and age	Race/ ethnicity	SES	% Female	*N* follow-up (% baseline *N*)	Study design	Exercise measure	Eating disorder measure and other relevant study measure	Brief summary of findings and main effects when available	Global quality rating
Allen, Crosby, Oddy, and Byrne (2013) Australia	Western Australian pregnancy cohort (Raine) study T1 M_{age} = 14.01 years (*SD* 0.19)	Not detailed in current study; full report in Newnham, Evans, Michael, Stanley, and Landau (1993) and Allen, Byrne, Forbes, and Oddy (2009)	Not detailed in current study; full report in Newnham et al. (1993) and Allen et al. (2009)	51	1,383; 59.4%	Prospective secondary data analysis of birth cohort 1989–1991; assessed at cohort age 14, 17, and 20. Accounted for BMI and income; no specification of T1 ME as covariate	Single binary item (presence/ absence): Hard exercise specifically for weight control inthe last month Response scale of 0, 1, 2, 3 where 1 = some of the time; behavior coded present if occurred at least "some of the time (once per week / a few times per	Items adapted from child eating disorder examination (ChEDE) Eating disorder examination questionnaire (EDE-Q)	For boys, ME was stable over time; for girls, ME increased by ~10% from age 14 to 17, then remained stable to 20. Depressive symptoms at age 14 had a positive main effect on ME for young men at 17 and 20; no effect was found for girls. For boys, effect of depression at age 14 on later ME: OR (95% CI): 2.29 (1.21–4.31)	Moderate
Dakanalis et al. (2016) Italy	First year college men M_{age} not reported	Caucasian (87%), Hispanic/ Latino (6.5%), Asian (4.0%), other/mixed (2.5%)	61% middle class, 20.5% lower-middle class, 18.5% upper-middle class	0	2,555; 95.4%	Longitudinal; two assessments over 9 months, 2013–2014 Covariates unspecified	Binary item from EDDS, (presence/ absence); asked how many times "engaged in more intense exercise" (regardless of duration/ frequency of exercise) "specifically to counteract the effects of overeating." Behavior was coded "present" if participants reported it had occurred ≥1 time in the past 3 months	Eating Disorder Diagnostic Scale (EDDS), Italian translation	ME was largely stable in those endorsing this behavior over time. Elevated negative affectivity, body dissatisfaction, self-objectification, and lower self-esteem at T1 were predictive of both the initiation and persistence of ME and other ED behaviors at T2. OR (95% CI) Negative affectivity: 1.50 (1.14–1.98 Self-objectification: 3.61 (2.39–4.51) Body dissatisfaction: 1.67 (1.21–2.01) Low self-esteem: 1.70 (1.15–2.06)	Strong
Davis, Guller, and Smith (2016a) United States	Middle school boys T1 M_{age} = 10.33 years	European American (60.9%), African	Not reported	0	631; 100% with imputation	Longitudinal; assessed once in fifth grade and six times	Two questions: "Over the past 2 weeks, have you exercised a lot as	EDE-Q; thinness and restricting expectancy inventory (TREI);	ME did not increase over time. At T1, 25% reported high ME (greater frequency);	Strong

Study and location	Sample and age	Race/ ethnicity	SES	% Female	*N* follow-up (% baseline *N*)	Study design	Exercise measure	Eating disorder measure and other relevant study measure	Brief summary of findings and main effects when available	Global quality rating
		American (18.7%), Hispanic (8.2%), Asian (2.9%), middle eastern (0.4%), other (8.8%)				over the following 3 years (through eighth grade) Covariates unspecified	a way to control your weight or because you ate a lot?" (yes/no). If yes, then completed a second item: "On how many days of the last 14 have you done this?" six response options ranging 1–2 days through 14 days/every day. Two items were combined, such that 0 reflected no ME, 1 reflected having done so 1–2 days of the last 14, etc.	eating expectancy inventory (EEI)	of these boys, 9% decreased ME over time. Compared to those reporting no ME, boys who maintained or decreased ME had elevated levels of depression, eating expectancies, and thinness expectancies, and were more likely to have experienced pubertal onset. Cohen's *d*: Depression: $d = 0.30$ Eating expectancies: $d = 0.21$ Thinness expectancies: $d = 0.50$ Phi coefficient: Pubertal onset: $\Phi = 0.14$	
Davis, Guller, and Smith (2016b) United States	Middle school girls T1 M_{age} = 10.33 years	European American (60.9%), African American (18.7%), Hispanic (8.2%), Asian (2.9%), middle eastern (0.4%), other (8.8%)	Not reported	100	564; 100% with imputation	Longitudinal; assessed once in fifth grade and 6 times over the following 3 years (through eighth grade) Covariates unspecified	Two questions: "Over the past 2 weeks, have you exercised a lot as a way to control your weight or because you ate alot?" (yes/no). If yes, then completed a second item: "On how many days of the last 14 have you done this?" six response options ranging 1–2 days through 14 days/ every day. Two items were combined, such that 0 reflected no ME, 1 reflected having done so 1–2 days of the last 14, etc.	EDE-Q; TREI; EEI	At each wave, at least 19% endorsed ME. Depression and thinness expectancies were associated with temporally proximal engagement in ME. Middle school girls engaging in ME or fasting behavior had higher depression scores and more strongly endorsed eating and thinness expectancies in elementary school than girls who did not engage in the behaviors. Depression: OR (95% CI): 1.05 (1.01–1.10) Thinness expectancies: OR (95% CI): 1.34 (.04–1.72)	Strong

Study and location	Sample and age	Race/ ethnicity	SES	% Female	*N* follow-up (% baseline *N*)	Study design	Exercise measure	Eating disorder measure and other relevant study measure	Brief summary of findings and main effects when available	Global quality rating
Davis, Ortiz, and Smith (2018) United States	Children from 23 public elementary schools in Kentucky T1 M_{age} = 10.86 years	European–American (60.9%); African–American (18.7%); Hispanic (8.2%); Asian (2.9%); middle eastern (0.4%); other (8.8%)	Schools represented urban, suburban and rural areas	49	1906; 100% with imputation of missing data	Longitudinal; nine time points over 5 years (2009–2014) T1 ME entered in SEM model	Single binary item (yes/no): Over the past 2 weeks, have you exercised a lot as a means to control your weight or because you ate a lot?	EDE-Q, adapted for children	Rates of ME declined over 5 yrs for girls and boys; ME covaried with other ED behaviors such that engagement in one behavior seemed to increase chances of engaging in others Wave 1 fasting predicts wave 2 ME (b = .08, p <.01) Wave 1 ME predicts wave 2 fasting (b = .12, p <.001).	Strong
Elbourne and Chen (2007) Australia	Female recreational triathletes M_{age} = 30.7 years	Not reported	Not reported	100	42, 69%	Longitudinal; 3 time points over 1 year Covariates unspecified	Obligatory exercise questionnaire (OEQ), with ≥50 for clinical cutoff	Eating disorder inventory (EDI); weight feelings, shape feelings, and eating behavior for weight and shape examination scores from the eating and exercise examination — Computerized (EEE-C)	Obsessive–compulsiveness, increased physical activity, and food restriction were not found to be significant predictors of obligatory exercise. Although self-reported obligatory exercise did not predict eating pathology, preoccupation with weight and shape was a significant predictor of both obligatory exercise and ED behavior.	Weak
Goodwin, Haycraft, and Meyer (2014a) England, United Kingdom	Adolescents from 5 schools Ages 13–15 T1 M_{age} = 13.97 years (*SD* 0.69)	White British (93.8%)	Not reported	56	332, 40.1% of available sample— Participants only included if completed both time points	Longitudinal; 2 time points over 1 year Accounted for T1 ME	Compulsive exercise test (CET)	Eating disorder Inventory-2 (EDI-2) Modified perceived sociocultural influences on body image and body change questionnaire Perceived sociocultural pressure scale	Family and peer messages to become more muscular predicted ME in boys, whereas feeling pressure from the media to be thin was a significant predictor of ME in girls. Boys, muscularity: b = .87, p = .002 Girls, media pressure: b = .99, p = .001 EDI scores were not associated with CET scores for boys.	Moderate
Goodwin, Haycraft, and Meyer (2014b) England,	Adolescents from 6 schools Aged 12–14	White British (93.2%)	Participating schools were in areas of average to low	60	369, 100% — participants only	Longitudinal; 2 time points over 2 years	Compulsive exercise test (CET)	EDI-2: Drive for thinness, bulimia, and body dissatisfaction	For boys, higher levels of obsessive–compulsiveness and self-perfectionism	Weak

Study and location	Sample and age	Race/ ethnicity	SES	% Female	*N* follow-up (% baseline *N*)	Study design	Exercise measure	Eating disorder measure and other relevant study measure	Brief summary of findings and main effects when available	Global quality rating
United Kingdom	T1 M_{age} = 12.89 years (*SD* .69)		levels of socioeconomic status		included in analysis if they completed both time points	Accounted for T1 ME		subscales Child and adolescent perfectionism scale (CAPS) Spence child anxiety scale–obsessive compulsive subscale (SCASOC)	predicted greater ME after controlling for baseline levels of ME; lower anxiety predicted greater engagement in ME. Obsessive–compulsive: $b = .35$, $p < .001$ Perfectionism: $b = .18$, $p < .05$ Anxiety: $b = -.21$, $p < .05$ For girls, psychological predictors did not predict ME.	
Goodwin, Haycraft, and Meyer (2014c) England, United Kingdom	Adolescents from 5 schools Aged 12–14 T1 M_{age} = 13.16 years (*SD* .73)	White British (95.4%)	Not reported	56	572, 70.4% of available sample, participants only included with two time points	Longitudinal; 2 time points over 1 year Accounted for T1 ME	Compulsive exercise test (CET)	Regulation of emotions questionnaire (REQ) Compulsive exercise test (CET)	ME was predicted by internal dysfunctional emotion regulation in girls and boys, even after controlling for initial levels of ME. Internal dysfunctional emotion regulation predicts ME: Boys: $b = .48$, $p < .001$ Girls: $b = .51$, $p < .001$	Moderate
Homan (2010) United States	Undergraduate women T1 M_{age} = 19.2 years (*SD* 1.1)	White (97%)	Private liberal arts college	100	156, 68%	Longitudinal; 2 time points over 7 months Unspecified, but report accounted for T1 outcomes in analyses	Four items from the obligatory exercise questionnaire (OEQ) that address guilt or depression associated with missed exercise	Athlete subscale of the sociocultural attitudes toward appearance Questionnaire-3 (SATAQ-3) Thin internalization subscale of the sociocultural attitudes toward appearance questionnaire (SATAQ) Body areas satisfaction subscale of the multidimensional body-self relations questionnaire (MBSRQ)	T1 athletic-ideal internalization predicted subsequent increases in ME, but not body dissatisfaction or dieting.Initial thin-ideal internalization predicted subsequent body dissatisfaction, dieting, and ME. Athletic-ideal internalization: $b = .11$, $p = .02$ Thin-ideal internalization: $b = .07$, $p = .01$ Neither thin-ideal nor athletic-ideal internalization predicted unique variance in ME in combined model.	Moderate
McCabe and Ricciardelli	Adolescents from 10 high schools	Not reported	Varied socioeconomic status	51	861, 82%	Longitudinal; two time points over 8 months	Exercise dependence scale	EDI-2 bulimia subscale Body image and	For boys ($b = .25$) and girls ($b = .53$), regardless of pubertal	Moderate

Study and location	Sample and age	Race/ ethnicity	SES	% Female	*N* follow-up (% baseline *N*)	Study design	Exercise measure	Eating disorder measure and other relevant study measure	Brief summary of findings and main effects when available	Global quality rating
(2004) Australia	Aged 12–17 T1 M_{age} = 13.3					Accounted for T1 ME		body change inventory	timing, strategies to lose weight predicted ME. For some boys, strategies to increase muscle also predicted ME (*b* = .29). For girls, but not for boys, body dissatisfaction predicted ME (*b* = .33). Early maturing boys and girls were more likely to report ME than others.	
McCabe and Ricciardelli (2006) Australia	Adolescents from 10 high schools Aged 12–15 T1 M_{age} = 14.5 years	Mostly White	Majority middle class	52	847, 76.8%	Longitudinal; three time points over 16 months Accounted for T1 ME	Exercise dependence scale	EDI-2 drive for thinness and bulimia subscales Body image and body change inventory	For boys, significant predictors of ME included changes in positive affect and body importance. Positive affect: *b* = −.12, *p* <.05 Body importance: *b* = .26, *p* <.001 For girls, significant predictors of ME included body dissatisfaction, changes in positive affect, and body importance. Body dissatisfaction: *b* = .15, *p* <.01 Positive affect: *b* = −.13, *p* <.05 Body importance: *b* = .19, *p*.001	Moderate
McCabe and Ricciardelli (2009) Australia	Adolescents from nine schools Aged 11–16 T1 M_{age} = 14.5 years	Majority Anglo-Saxon	A broad range of socio-economic backgrounds was represented	52	792	Longitudinal; two time points over 16 months Accounted for T ME	Exercise dependence scale	EDI-2 bulimia subscale	No differences were evidenced in report of ME across relative to weight status or gender. In a model that included both genders, significant predictors of ME at T2 were negative affect, and body importance; in this model, pubertal timing and body dissatisfaction were not related to ME. Negative affect: *b* = .09, *p* <.05 Body importance: *b* = .37, *p* <.05	Moderate

Study and location	Sample and age	Race/ ethnicity	SES	% Female	*N* follow-up (% baseline *N*)	Study design	Exercise measure	Eating disorder measure and other relevant study measure	Brief summary of findings and main effects when available	Global quality rating
Schleien and Bardone-Cone (2016) United States	Undergraduate women Aged 17–24 T1 M_{age} = 18.71 years (*SD* = 1.01)	Caucasian/ White (73.2%), African American/ Black (9.1%), hispanic/ Latina (8.0%), Asian (5.0%), other (0.4%), multiple race/ ethnicities (4.3%).	Highest parental education was used as a proxy for SES: Mean of 17.01 years (*SD* 2.67), with 16 = 4-year college degree	100	237; 67.3%	Longitudinal; two time points over 1 year Accounted for T1 ME	Single item from EDE-Q: "How many times have you exercised in a "driven" or "compulsive" way as a means of controlling your weight, shape or amount of fat, to burn off calories?" Reported frequency over the past 28 days	Internalization: Thin/low body fat subscale of the SATAQ-4 The appearance subscale of the contingencies of self-worth scale Hypercompetitive attitude scale (HCA) Restraint subscale of the EDE-Q	After controlling for T1 levels of the dependent variables, neither thin-ideal internalization nor appearance contingent self-worth interacted with competitiveness to predict dieting or ME.	Weak
Scott, Haycraft, et al. (2020) England, United Kingdom	Adolescent and young adult athletes T1 M_{age} = 18.38 years (*SD* = 2.70)	White (85%)	Not reported	62	199, 56% of original sample, participants only included with three time points	Longitudinal; three time points over 7 months Accounted for T2 ME with T3 ME as DV	Compulsive exercise test—Athlete version (CET-A)	EDI-2 Social network – Self report data	Convergence of report of ME varied over time (relative to season of play) and gender. Being friends with teammates who themselves are well connected, connecting two groups of teammates, and being part of a cohesive team protected against ME. Measure of cohesion: β = −0.13, p = .01	Weak
Scott, Plateau, et al. (2020) England, United Kingdom	Adolescent and young adult athletes T1 M_{age} = 18.35 years (*SD* = 2.66)	Not reported	Not reported	56	195, 56% of fully available sample, participants only included with three time points	Prospective; three time points over 7 months Did not account for T1 ME	Compulsive exercise test—Athlete version (CET-A)	EDI-2 Rosenberg's self esteem scale (RSES) Hospital anxiety and depression scale (HADS) Bulimic Modelling scale; adapted for athletes (BMS-A)	The extent to which an individual modelled the binge-eating or purging behavior of their team peers was the only teammate influence mechanism to significantly correlate (positively) with ME (CET-avoidance of negative affect, CET-weight control). Higher anxiety mediated the association between peer influence specific to bulimic behavior and CET for avoidance of negative affect β = .31, p = .04	Weak

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Study and location	Sample and age	Race/ ethnicity	SES	% Female	*N* follow-up (% baseline *N*)	Study design	Exercise measure	Eating disorder measure and other relevant study measure	Brief summary of findings and main effects when available	Global quality rating
									Avoidance of negative affect; gender did not moderate this mediation model.	
Solmi, Mascarell, Zammit, Kirkbride, and Lewis (2019) England, United Kingdom	Avon longitudinal study of parents and children	Not detailed in current study; full report in Boyd et al. (2013)	Not detailed in current study; full report in Boyd et al., 2013	54	734; 12% of full sample (*N* = 6,361), participants only included if they reported psychotic symptoms at age 13	Secondary data analysis; longitudinal; two time points over 5 years Did not account for T1 ME	Single binary item from youth risk behavior surveillance system questionnaire (presence/ absence): Exercising frequently for weight loss even when sick, or feeling guilty when not exercising	Binary items from youth risk behavior surveillance system questionnaire (presence/absence): Binge eating, purging, fasting	Evidence of association between psychotic experiences at Age 13 and binge eating, fasting, and purging at Age 18; no association between psychotic experiences and ME; authors cite low power as a possible explanation.	Weak
Zhan, Heatherington, and Klingenberg (2020) United States	First-year college students	Non-Hispanic White (54.6%), Asian (16.6%), biracial/ mixed (10.5%), Hispanic/ Latino/a (10.0%), black/ African American (7.9%), native American (0.4%)	Highest education level attained by parent was an advanced degree (67.3%), college degree (20.4%), no college degree (12.4%)	61	229, 56.8% of available sample—Participants only included if they completed at least two time points	Longitudinal; three time points over 6 months Did not account for T1 ME	Compulsive exercise test – CET "avoidance and rule-driven exercise" and "weight-control exercise" subscales	Contingencies of self-worth scale (CSW) Eating attitudes test-26 – "Belief" items and "behavior" items	Self-worth based upon appearance predicted scores on the CET exercise for weight control subscale. Athletes scored significantly higher than non-athletes on the CET avoidance and rule-driven exercise subscale at T2. $b = .65$, $p < .001$ In contrast, non-athletes scored significantly higher than athletes on the CET exercise for weight control subscale at T2. $b = -.41$, $p < .01$ Neither model demonstrated a significant difference according to gender.	Weak

Note: In this study by Goodwin et al. (2014c), scores on the Compulsive Exercise Test were conceptualized as an eating disorder symptom. Standard deviations for age and effect sizes for main outcomes are reported, when available. Abbreviations: OR, odds ratio; SES, socio-economic status.

TABLE 2

Psychosocial variables demonstrating association with ME in multiple studies and overall quality ratings

Variable	Number of studies	Nature of effect on ME (with time between assessments)	Quality ratings across studies (*n*)
Weight and shape concerns	7	Positive effect of body dissatisfaction on ME: -for adolescent girls, not boys (8 months; McCabe & Ricciardelli, 2004; 16 months; McCabe & Ricciardelli, 2006) -for college men (9 months; Dakanalis et al., 2016) No effect of body dissatisfaction on ME for adolescent boys and girls (16 months; McCabe & Ricciardelli, 2009) Positive effect of body importance (i.e., the degree to which body weight and shape reportedly impact self-identity and/or mood) on ME: -for adolescent boys and girls (16 months; McCabe & Ricciardelli, 2006, 2009) -In male and female college athletes, self-worth based upon appearance predicted ME for weight control (6 months; Zhan et al., 2020) -In triathlete women, preoccupation with weight and shape positively predicted obligatory exercise status (those above a clinical cutoff score on the Obligatory Exercise Questionnaire) (1 year; Elbourne & Chen, 2007) *Muscularity*: -For some adolescent boys, and for girls with an athletic focus, strategies to increase muscle predicted ME (16 months; McCabe & Ricciardelli, 2006) -Family and peer messages to become more muscular predicted ME in adolescent boys; feeling pressure from the media to be thin predicted ME in adolescent girls (1 year; Goodwin et al., 2014a)	Strong (1) Moderate (4) Weak (2)
Thin-ideal internalization, thinness, and eating expectancies	4	-In middle school boys and girls, greater thinness expectancies and eating expectancies predicted ME (3 years; Davis et al., 2016a, 2016b) -In college women: -*athletic*-ideal internalization predicted ME, but did not predict body dissatisfaction or dieting; in contrast, *thin*-ideal internalization predicted all three outcomes (7 months; Homan, 2010) -neither thin-ideal internalization nor appearance contingent self-worth interacted with competitiveness to predict ME (1 year; Schleien & Bardone-Cone, 2016)	Strong (2) Moderate (1) Weak (1)
Affect	8	Positive effect of depression on ME for: -middle school boys (3 years; Davis et al., 2016a) -middle school girls (3 years; Davis et al., 2016b) -late adolescent boys/young men (3 and 6 years; Allen et al., 2013) -college men (9 months; Dakanalis et al., 2016) Positive effect of negative affect on ME: -adolescent boys and girls (16 months; McCabe & Ricciardelli, 2009) Negative effect of positive mood on ME: -for adolescent boys and girls, (16 months; McCabe & Ricciardelli, 2006) Negative effect of depression on ME: -for girls, but not for boys (16 months; McCabe & Ricciardelli, 2006) -In male and female college athletes, elevated anxiety, not depression or self-esteem, contributed to increased report of ME—CET subscale for avoidance of negative affect (8 months; Scott, Plateau, et al., 2020) -In adolescent boys and girls, ME was predicted by internal dysfunctional emotion regulation (Goodwin et al., 2014c)	Strong (3) Moderate (4) Weak (1)
Obsessive-compulsive (OC) temperament	2	No effect of OC traits on ME -for triathlete women (1 year; Elbourne & Chen, 2007) Positive effect of OC traits and self-perfectionism and negative effect of anxiety on ME -for adolescent boys (2 years; Goodwin et al., 2014b)	Moderate (1) Weak (1)