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AN IDEA WORTH RESEARCHING

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A call to experimentally study acute affect-regulation mechanisms specific to driven exercise in eating disorders

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Abstract

Driven exercise (i.e., feeling compelled to exercise to control one's weight or shape, to obtain other positive consequences of exercising, or to avoid other negative consequences of not exercising) is a common phenomenon in individuals with eating disorders (EDs), typically associated with negative clinical outcomes. Current theoretical models of driven exercise highlight the short-term affect-regulating outcome of acute driven exercise, which is implicated to maintain this symptom either by positive or negative reinforcement. However, few studies have actually investigated cognitive, affective, and psychobiological mechanisms related to acute driven exercise. In particular, experimental studies that directly test mechanisms leading to the short-term affective improvement after acute driven exercise are scarce. In this article, we therefore propose potential cognitive, affective, and psychobiological mechanisms that could explain the affect-regulating function of driven exercise in individuals with EDs. In addition, we suggest examples of experimental studies that could directly test these mechanisms in individuals with EDs, as recent studies have demonstrated the safety of supervised exercise in EDs research. Our aim of stimulating research on the underlying causes and maintenance factors of driven exercise in EDs has the potential to critically inform treatment development for this high-risk population.

KEYWORDS

anorexia nervosa, bulimia nervosa, driven exercise, eating disorders, emotion regulation, experimental, physical activity, psychobiology

1 | INTRODUCTION

Exercising in a driven manner (i.e., driven exercise [DEx], feeling compelled to engage in physical activity or an urgency to exercise to control one's weight or shape, to obtain other positive consequences of exercising, or to avoid other negative consequences of not exercising), is a common phenomenon affecting up to 80% of individuals with anorexia nervosa (AN; Dalle Grave, Calugi, & Marchesini, 2008), and 66% of those with bulimia nervosa (BN; Stiles-Shields, Bamford, Lock, & Le Grange, 2015), associated with numerous negative clinical outcomes. On a behavioral level, DEx is described as predominantly aerobic exercise, and excessive with regards to intensity, duration and frequency (Solenberger, 2001). Furthermore, patients with eating disorders (EDs) often engage in DEx even despite adverse consequences such as pain, risk of injury or significant impact on daily life, leading to further impairment due to prolonged weight-loss or loss of social interactions (Noetel, Dawson, Hay, & Touyz, 2017).

In the last few decades, several conceptualizations of DEx as a symptom of EDs have emerged (Davies, 2015), with two prominent models characterizing DEx either as an exercise dependence (Hausenblas & Downs, 2002) or as compulsive exercise (Meyer, Taranis, Goodwin, & Haycraft, 2011). The exercise dependence model assumes that, similar to behavioral addictions, DEx in EDs is associated with initially engaging in exercise to experience a positive affective state, which will then lead to tolerance (i.e., increase in exercise intensity needed to experience these states) and withdrawal symptoms (e.g., anxiety, depression, guilt). The compulsive exercise model emphasizes the association of DEx with perfectionism and rigidity and describes compulsively engaging in DEx as a means to reduce or avoid negative affective states or feared negative consequences (e.g., weight gain). There is empirical evidence for the validity of both models from cross-sectional studies (Bratland-Sanda et al., 2011; Taranis & Meyer, 2011). Interestingly, both models agree on momentary affect regulation as the main psychological outcome of DEx, which aligns well with etiological models that conceptualize ED behaviors as maladaptive attempts to regulate negative affect induced by ED-specific events and cognitions (Haynos & Fruzzetti, 2011). In line with reinforcement theory (Skinner, 1968), these short-term affect-regulating consequences (decrease of negative affect, increase of positive affect) are considered the main motivators and reinforcers of maladaptive behaviors (Swerdlow, Pearlstein, Sandel, Mauss, & Johnson, 2020).

A psychological mechanism is defined as "a composite system whose activity is responsible for the target phenomenon" (Bechtel & Wright, 2009, p. 119). An example of a psychological mechanism germane to DEx might be the activation of a specific cognition such as "this exercise helps me lose weight" during DEx. This thought is then responsible for a subsequent affective improvement during or at the end of the current DEx episode, perhaps by decreasing fear of weight gain and increasing a sense of pride or accomplishment. Although both models agree on the affect-regulating outcome of DEx, exercise dependence and compulsive exercise diverge in that they propose several distinct key mechanisms that drive *momentary* affective changes associated with DEx that subsequently reinforce this symptom. In addition, improved affect is an outcome of acute exercise in general (Bernstein & McNally, 2018). General mechanisms that lead to affect improvement after acute exercise might promote short-term affective changes after engaging in DEx as well; however, for those with EDs, the cognitions and affects that underpin behavior maintenance might be more ED-specific (see Figure 1 for an overview).

To date, an integration of key mechanisms (ED-specific and general) into a comprehensive biobehavioral model of DEx is lacking. Further, consensus regarding which of the proposed mechanisms are the main causal factors that lead to short-term affective improvement (thereby reinforcing and maintaining DEx) has not been reached. The need for further research in this domain presents in part because physical activity has been discouraged within ED treatment and research for some time (Quesnel et al., 2018). While cessation of physical activity is appropriate in the context of medical acuity, a historically cautionary approach to DEx might have inadvertently led to unnecessary discouragement of experimental studies on exercise in EDs.

2 | OUR IDEA WORTH RESEARCHING: MORE EXPERIMENTAL STUDIES ON THE AFFECTIVE-REGULATING MECHANISMS OF DRIVEN EXERCISE

Hence, our idea worth researching is to begin to experimentally test potential key mechanisms responsible for the affect-regulating function of DEx. Doing so will lead to a better understanding of this symptom and ultimately inform (a) an improved biobehavioral model of DEx, and (b) psychological and possibly pharmacological treatments that explicitly target the mechanisms that serve to maintain this

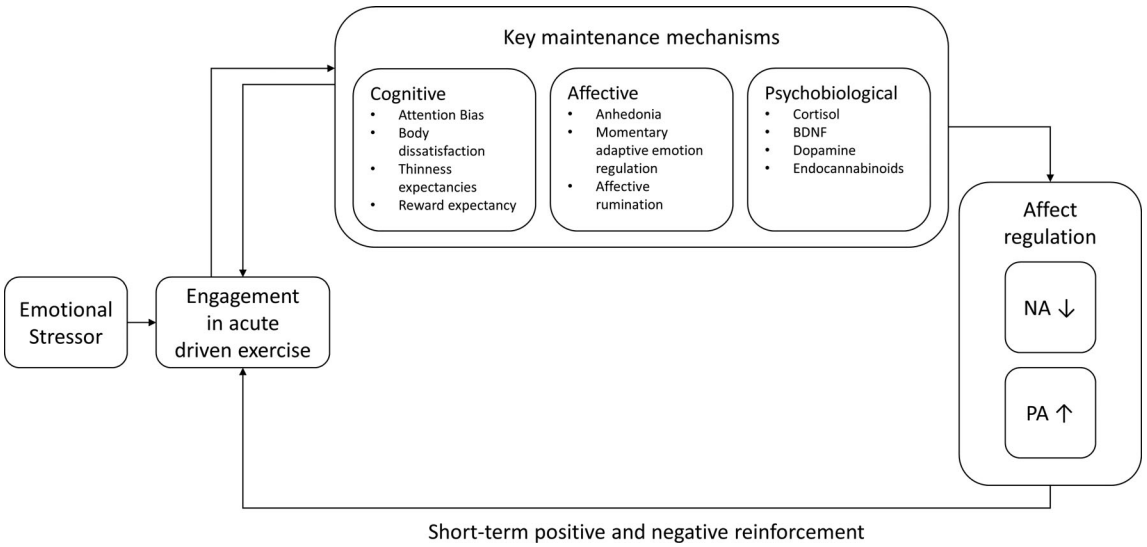

FIGURE 1 Proposed key mechanisms of the short-term affect regulating outcome of acute driven exercise in individuals with eating disorders. BDNF, brain-derived neurotrophic factor; NA, negative affect; PA, positive affect

behavior. Similar to the position taken by Jansen (2016) for a general approach to investigate EDs, and more recently by Glashouwer et al. (2020) in the context of anorexia nervosa, we particularly argue for more *experimental* studies on DEx. We also propose a focus shift from investigating primarily negative consequences of prolonged DEx, towards studying contemporaneous mechanisms that contribute to *acute* affective change, as those effects that are short-term in nature most likely act as positive and negative reinforcers for DEx. To do so, we propose to build a valid laboratory model of acute response to exercise in EDs, or *momentary* DEx. The formulation of this in-lab model depends on which mechanisms of DEx one believes should be prioritized for investigation. Specifically, we argue that prescribed or ad libitum aerobic exercise after an emotional stressor might be a useful in-lab model to investigate DEx (as this mimics key components of the theoretical model, see Figure 1). Below, we propose key cognitive, affective, and psychobiological mechanisms responsible for affective change as an acute response to DEx as a theoretical starting point for this model. We specifically focus on mechanisms that have been implicated in promoting affect regulation following exercise in not only restrictive EDs but also in physically active individuals in general; in so doing, we pay particular attention to those mechanisms that may be more reinforcing and applicable to DEx. We highlight how experimentally testing these ideas in participants with EDs could increase understanding of *why* DEx may be critically implicated in the etiology and maintenance of EDs. Table 1 provides an overview of potential mechanisms, their current state of evidence, and examples of how these mechanisms could be experimentally tested.

2.1 | Cognitive mechanisms

Several dysfunctional ED cognitions have been linked to DEx, such as thinness expectancies (i.e., belief that thinness will improve quality of life, Garner et al., 2014) or body dissatisfaction (Reichert et al., 2020), which are also associated with negative affect. However, it is unclear exactly how these cognitions might contribute to the affect-regulating consequence of DEx. Furthermore, individuals with EDs exhibit high attention biases towards food and body image cues that are often perceived as threatening (Aspen, Darcy, & Lock, 2013). Given preliminary evidence for reduced attention biases towards threat in anxious participants following aerobic exercise (Cooper & Tomporowski, 2017), a cognitive avoidance mechanism of DEx might lead to affective change via shifting attention to a more rewarding activity (i.e., the current exercise) to both reduce momentary uncomfortable attention biases and anticipate improved affect during and following this activity.

Thus, experimental research on cognitive mechanisms of DEx might assess whether manipulating ED-specific cognitions is associated with the intensity or duration of acute exercise (or vice versa), or if exercise is perceived as more rewarding (by way of negative reinforcement) after inducing state body dissatisfaction or thinness expectancies. Intensity and duration of exercise can be important behavioral indicators of the severity of DEx, which could inform further research investigating whether momentary reductions of body dissatisfaction during exercise is a mechanism of DEx in service of acute affective change. Investigating whether DEx acutely attenuates attentional bias in EDs would be of particular interest, as such a short-term relief from focusing on, for example, feared body parts might be negatively reinforcing in that it results in reduced negative affect. Exercise may also be reinforced by affective change following positively-valenced cognitions, for example, anticipation of weight loss and body image improvement during exercise, as well as an experience of pride following its completion. As such, inducing (compared to dampening) positive exercise- and ED-related cognitions may also contribute to heightened in-lab exercise.

2.2 | Affective and emotion regulation mechanisms

Cross-sectional research demonstrates associations between DEx and affect-regulation difficulties (Goodwin, Haycraft, & Meyer, 2014), as well as heightened anhedonia (Davis & Woodside, 2002). Given that emotion regulation is not only a trait, but also a state (Lavender, Tull, DiLillo, Messman-Moore, & Gratz, 2017), a potential affect-regulating mechanism of DEx might involve acute improvement in emotion regulation capabilities, leading to improved affect which positively reinforces DEx. Alternatively, DEx may also be negatively reinforced by affective change after attenuating maladaptive emotion regulation processes, like affective rumination. Affective rumination refers to ruminative processes that focus repetitively on negative affective states, and has been conceptualized as a maladaptive emotion regulation strategy (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Such a mechanism has been observed in individuals with emotion regulation difficulties after acute exercise (Bernstein & McNally, 2018), but remains untested in individuals with EDs. This could be assessed by measuring rumination and affect following a negative mood induction, prior to, during and after acute exercise, compared to resting. To assess whether DEx momentarily improves adaptive emotion regulation as a positively reinforcing mechanism, one may expose participants to controlled doses of exercise followed by emotion regulation tasks (e.g., Stroop tests, cognitive reappraisal tasks), assessing differences in affective change after use of adaptive emotion regulation strategies compared to no exercise. An additional study might investigate if exercise is (objectively or subjectively) more pleasurable for individuals with EDs, particularly for those experiencing anhedonia (Davis & Woodside, 2002), and if so, whether a manipulation of reward response (e.g., by antagonizing dopamine) could modulate the use of exercise for pleasure (O'Hara et al., 2016).

2.3 | Psychobiological mechanisms

Potential affect-regulating psychobiological mechanisms for the maintenance of DEx are numerous, including modulation in hormones such as cortisol or brain-derived neurotrophic factor (BDNF). Cortisol (i.e., adrenal glucocorticoid secreted in response to stress and low

TABLE 1 Potential mechanisms of affective change after driven exercise, their current state of evidence and examples of experimental designs for assessment in eating disorders

Potential mechanism	Evidence	Possible experimental designs for eating disorders
Cognitive mechanisms		
Driven exercise might attenuate high momentary levels of thinness expectancies and body dissatisfaction, leading to increased positive affect and to experience exercise as more rewarding	Cross-sectional (Garner, Davis-Becker, & Fischer, 2014) and EMA studies (Reichert et al., 2020) in ED samples	1. Compare whether participants with high vs. low state levels of thinness expectancies differ in a progressive ratio task assessing exercise reward 2. Assess differences in intensity or duration of exercise needed until affect improvement after experimental induction of state body dissatisfaction
Exercise may attenuate attention bias towards threat-stimuli resulting in reduction of negative affect	In anxious participants (Cooper & Tomporowski, 2017)	Dot-probe task with body-shape or high-caloric food stimuli (fear of weight gain) after either exercise or resting
Affective mechanisms		
Acute driven exercise buffers emotion regulation difficulties	In healthy participants with emotion regulation difficulties (Bernstein & McNally, 2018)	1. Cognitive reappraisal task after exercise to assess changes in affect after adaptive emotion regulation, compared to resting 2. Measuring affective change after a negative mood induction followed by either exercise or resting controlling for rumination during the exercise/resting condition
Intense exercise might be more pleasurable/rewarding than other activities for individuals with EDs	Anhedonia is linked to driven exercise (Davis & Woodside, 2002) Exercise is perceived as more rewarding compared to monetary compensation (Klein et al., 2010)	Pleasure ratings and facial EMG prior/during engagement in exercise and other activities (reading, music listening, animal petting)
Driven exercise may help to avoid core ED-related fears such as fear of weight gain	EMA studies in ED samples (Reichert et al., 2020)	Inducing fear of weight gain (i.e., by imagining eating high-caloric food) followed by either exercise or resting to measure changes in anxiety and fear levels
Psychobiological mechanisms		
Exercise-stimulated BDNF secretion as a mechanism for affective changes during acute driven exercise	In healthy adults after aerobic training (Dinoff et al., 2016)	Measuring BDNF levels before and following supervised aerobic exercise
Exercise may modulate affective responses by blunting cortisol secretion after subsequent emotional stressors	Correlational study in patients with anorexia nervosa (Klein, Mayer, Schebendach, & Walsh, 2007) In depressed and anxious individuals (Zschucke, Renneberg, Dimeo, Wüstenberg, & Ströhle, 2015)	Assessing salivary cortisol levels before, during and after experimentally-induced activity and a subsequent emotional stressor (e.g., social stressor, negative mood induction)
Endocannabinoid may modulate increase in positive affect after engaging in driven exercise	In healthy participants (Dietrich & McDaniel, 2004) Physical activity increased plasma endocannabinoids in individuals with PTSD (Crombie, Brellenthin, Hillard, & Koltyn, 2018)	Assessing endocannabinoid plasma levels prior to and after exercise and their temporal association with positive affect changes

Abbreviations: BDNF, brain-derived neurotrophic factor; ED, eating disorders; EMA, ecological momentary assessment; EMG, electromyography; PTSD, posttraumatic stress disorder.

blood-glucose), is elevated in AN, especially in patients with high levels of DEx (Klein et al., 2007), and acute exercise has been associated with a resulting blunted cortisol response to stress-inducing tasks, indicating a possible psychobiological correlate for the affect-regulating function of exercise (Zschucke et al., 2015). Still, it is unclear if cortisol reactivity to stressors after acute exercise is similar in participants with EDs. BDNF (i.e., modulator of neural plasticity, depression and anxiety) shows elevated levels after exercise in

humans in general (Dinoff et al., 2016) and its expression in the mesolimbic reward system is altered in the rodent model of AN after wheel running (Ho, Klenotich, McMurray, & Dulawa, 2016). To our knowledge, BDNF has not been examined in relation to DEx in humans and it is unclear to what extent BDNF secretion after exercise is associated with momentary affective changes in individuals with EDs.

Other affect-related psychobiological lines of inquiry indicated in patients with DEx include investigating dopamine (DA; Gorrell, Collins, Le Grange, & Yang, 2020) and endocannabinoids (eCB; Crombie et al., 2018). Across samples, physical activity modulates the experience of pleasure and reward (DA), anxiety (eCB) (Matta Mello Portugal et al., 2013), and euphoria and well-being (eCB) (Dietrich & McDaniel, 2004). In clinical samples with post-traumatic stress disorder, experimentally-manipulated acute exercise impacts neurotransmitter release via eCBs (Crombie et al., 2018); to date, eCB modulation related to acute exercise has not been specifically examined in individuals with EDs. While dysregulated D2 receptor function has been generally recognized in patients with AN (Bergen et al., 2005), this phenomenon as it may relate to DEx has been focused in nonhuman samples. Rodent studies, including those treating mice with olanzapine (i.e., atypical antipsychotic that blocks DA), demonstrate that DA alteration, and specifically D2/D3 receptor antagonism, reduce wheel running activity (Hillebrand, van Elburg, Kas, van Engeland, & Adan, 2005; Klenotich, Ho, McMurray, Server, & Dulawa, 2015).

A study might therefore independently assess either cortisol or plasma BDNF levels prior to and after acute exercise, or after an emotional stressor following acute exercise in patients with EDs to assess their respective modulatory and/or mediating roles in the affect-regulating outcome of DEx. Similarly, assessing plasma levels of eCB prior to and after acute exercise in ED patients with high levels of DEx might further enlighten the potential role of eCB in increased positive affect after exercise in these patients. Finally, further investigation of DA signaling, and receptor antagonism specifically related to exercise behavior among humans might be worthwhile to further explore the affect-regulation function of DEx.

3 | CONCLUSION

We presented several potential cognitive, affective, and psychobiological mechanisms that have been implicated in initiating and maintaining DEx in individuals with EDs. We focused on the affect-regulating function of DEx as this is most likely the primary short-term positive and negative reinforcer of this behavior.

In light of recent research that found no serious adverse events related to supervised exercise in individuals with AN (Dittmer et al., 2020), we argue for the urgent necessity of beginning to test potential affect-regulating mechanisms of DEx with experimentally-induced activity. However, as there are potential cardiac complications associated with EDs in general (Casiero & Frishman, 2006), participants' heart rate should be closely monitored during the exercise, exclusion criteria should be clearly defined (e.g., exclusion of pericardial effusion or extreme bradycardia) and prescribed exercise paradigms should consider moderate intensity levels especially when participants are underweight.

Given that current research on the two prominent psychosocial models of DEx in EDs, exercise dependence (Hausenblas & Downs, 2002) and compulsive exercise (Meyer et al., 2011), has been mostly cross-sectional, we propose to test potential affect- regulating mechanisms derived from these and other DEx models to answer exactly *how* DEx leads to short-term affective change. For example, finding that exercise activates adaptive emotion regulation followed by increases in positive affect immediately after the DEx episode would increase our understanding of the mood improvement function of DEx proposed by Meyer et al. (2011). Subsequently, successful identification of core mechanisms can be integrated into a future, mechanistic model of DEx. We are confident that experimentally testing these mechanisms will increase the understanding of the role of DEx in the etiology and maintenance of EDs and subsequently inform treatment development which may lead to improved clinical outcomes for individuals with EDs.

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CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

DATA AVAILABILITY STATEMENT

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

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