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Compulsive Exercise and Weight Suppression: Associations with Eating Pathology in Distance Runners

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Abstract

Some athletes, particularly those who participate in activities that emphasize leanness such as competitive running, are at greater risk for eating disorder (ED) pathology. Compulsive exercise (CE) is linked with ED pathology among female athletes, with evidence that CE for weight control is most strongly correlated with ED thoughts and behaviors. Weight suppression (WS), the discrepancy between highest adult weight and current weight, is also linked with ED symptoms. Taken together, runners with greater WS may be more likely to engage in CE to influence their weight or shape and may subsequently report other associated ED symptoms. As such, the current study investigated associations between CE and ED pathology in a mixed-gender sample of adult distance runners ($N = 277$, 51% female), and tested the impact of WS on this association. Results indicated that females reported greater CE and ED pathology, whereas males reported greater WS. A significant interaction effect for CE x WS, ($p < .05$) indicated that the relation between CE and ED scores was stronger among male runners who reported higher WS. In contrast, WS did not impact the association between CE and ED pathology for female runners. Results from the current study have important implications for the assessment and treatment of ED in athletes, specifically runners, and suggest that WS is an important factor to consider when working with male athletes.

Keywords

Compulsive Exercise; Weight Suppression; Eating Pathology; Athletes; Distance Runners

1. Introduction

Athletes engaging in sports with a leanness focus report elevated eating disorder (ED) symptoms as compared to participants in sports that do not emphasize leanness (Bratland-Sanda & Sundgot-Borgen, 2013; Kong & Harris, 2015). Further, high-level athletes are at increased risk for ED as compared to the general population (Sundgot-Borgen & Torstveit, 2004), and to less competitive athletes (Byrne and McLean, 2002; Kong & Harris, 2015). Specifically for runners, a goal to attain competitive advantage with an idealized body

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composition (i.e., slender with minimal body fat, traditionally considered advantageous in distance running; Rüst et el., 2011) may lead athletes without a naturally lean physique to attempt to modify their build by engaging in ED behaviors (Reel, SooHoo, Petrie, Greenleaf & Carter, 2010; Sundgot-Borgen & Torstveit, 2004).

1.1 Compulsive exercise and risk for ED pathology

Compulsive exercise (CE) is a specific risk factor for ED pathology (Schroff et al., 2006; Meyer, Taranis, Goodwin & Haycraft, 2011) in clinical (Dalle Grave, Calugi, & Marchesini, 2008; Holtkamp, Hebebrand, & Herpertz-Dahlman, 2004) and non-clinical samples (Elbourne & Chen, 2007; Lipsey, Barton, Hulley, & Hill, 2006). Cross-sectional investigation has demonstrated links between CE and ED pathology in athletes (Plateau et al., 2014), and also between CE and other known ED risk factors (e.g., athletic identity; Turton, Goodwin & Meyer, 2017). While further longitudinal research is needed to establish etiological associations, existing evidence suggests that CE is a relevant factor related to ED pathology among athletes.

1.2 Weight suppression and risk for ED pathology

The extent to which exercise is undertaken primarily to change weight or shape is a dimension of CE most strongly associated with ED pathology (e.g., Mond, Hay, Rodgers, Owen, & Beumont, 2004; Mond, Hay, Rodgers, & Owen, 2006; Mond, Myers, Crosby, Hay, & Mitchell, 2008). Weight suppression (WS), the discrepancy between highest adult and current weight (Lowe, 1993), has been examined as a key construct related both to ED pathology and weight management (Gorrell et al., 2018). In a community sample, higher baseline WS predicted increased bulimic symptoms at 20-year follow-up; at 10-year follow-up, elevated drive for thinness mediated this effect (Bodell, Brown & Keel, 2017). These findings suggest that those with greater WS may be more motivated to manipulate their weight or shape, a desire which may exacerbate ED-related behaviors, including CE.

1.3 Gender differences in risk for CE and ED pathology

Interest in the study of athletes and EDs has increased over the past two decades, with increasing attention to gender differences (Baum, 2006). Consistent with population prevalence of EDs (Udo & Grilo, 2018), high-level multi-sport female athletes were more likely than males to report lifetime history of EDs (Schaal et al., 2011), and CE is more strongly related to ED pathology among women (Cunningham, Pearman & Brewerton, 2016). Among runners, individuals exercising excessively with elevated ED risk were also more likely to be female (Di Lodovico, Dubertret, & Ameller, 2018).

Gender differences in links between CE and eating pathology depend on the type of measures used. In particular, measures that include muscle dysmorphia, a symptom that has demonstrated strong conceptual links with eating pathology (Murray, Rieger, Touyz, & De la Garza García, 2010), may be particularly relevant in the assessment of male populations (Forbush et al., 2013). As such, a different rationale for increased exercise (i.e., for purposes of muscle building) has been supported with increased scores on muscle building and excessive exercise among males (Forbush, Wildes, & Hunt, 2014).

Males may also be more likely to engage in CE as a weight control behavior. Specifically, males with EDs are more likely to have significantly higher premorbid weights and a history of obesity (Welch, Ghaderi, & Swenne, 2015). Further, a recent investigation in a non-clinical, mixed-gender sample indicated that males with higher WS were more likely to engage in extreme weight control behaviors (Burnette et al., 2017). These findings suggest that although CE may contribute to ED pathology more commonly among female athletes, males with elevated WS may be at similar risk for this maladaptive exercise behavior.

CE is associated with ED pathology in athletes (Plateau et al., 2014) and CE motivated by weight and shape concerns is the most insidious in contributing to ED risk (e.g., Mond et al, 2004); however, to date, no work has explicitly investigated links between CE, weight history, and ED pathology among athletes. For athletes in higher-risk, leanness-focused sports such as distance running, a better understanding of the role of WS as a precipitant for CE could improve ED screening efforts. Further, with growing evidence that male athletes demonstrate a unique profile of ED pathology that includes the pivotal symptom of unhealthy exercise (e.g., Chatterton & Petrie, 2013; Torstveit, Fahrenholtz, Lichtenstein, Stenqvist & Melin, 2019), research examining these associations separately in males and females is critical (Chapman & Woodman, 2016).

1.4 The current study

The current study aimed to test the relation between CE and ED pathology in male and female runners, both to examine associations within each gender, and to test the potential moderating role of WS in each model. Based upon prior work (Di Lodovico et al., 2018), it was hypothesized that athletes with greater CE scores would report higher levels of ED pathology. An interaction effect was also anticipated, whereby those with increased WS would be more likely to demonstrate a significant association between CE and ED symptoms. Although men with elevated WS may be more likely to engage in extreme weight control behaviors (Burnette et al., 2017), given the nascent evidence to date on gender differences in WS and CE among athletes, moderation analyses remained exploratory.

2. Materials and Methods

2.1 Participants

Participants were adult runners ($N = 277$; 51% female) registered for a half- ($n = 109$) or full-marathon ($n = 168$) in upstate New York. Runners' ages ranged 19 – 73 (M [SD] = 43 [11]). Reported racial identity was White (91%), Asian (3%), Black (2%), Hispanic/Latino (1%), and 3% chose not to respond.

2.2 Measures

2.2.1 Demographic questionnaire.—Participants completed a demographic questionnaire with items including age, sex, and racial identity, as well as self-reported height and weight, used to calculate BMI (in kg/m^2). Supporting the validity of self-reported BMI, prior work with adult athletes did not indicate differences between self-report and objectively measured BMI (Gay, Monsma, & Torres-McGehee, 2009). Another item asked participants to report highest weight at adult height; current weight was subtracted from highest weight to calculate WS.

2.2.2 Compulsive Exercise Test (CET; Taranis, Touyz & Meyer, 2011).—

The CET is a 24-item self-report measure that assesses exercise beliefs and behaviors; the global score was used in the current study as a general measure of CE. The CET uses a Likert scale (anchors: 0 [*never true*]-5 [*always true*]); with higher scores reflecting greater CE. The CET demonstrated good internal consistency in prior research (Taranis et al., 2011); in the current study Cronbach's α = .85.

2.2.3 Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn & Beglin, 2008).—

The EDE-Q is a 28-item self-report questionnaire that assesses disordered eating attitudes and behaviors over the previous 28 days. Responses are on a 7- point scale (anchors: 0 [*no days*]- 6 [*every day*]); higher scores reflect greater ED pathology. The measure provides a global score calculated from the mean of four subscales; the current study used the global score as a measure of ED pathology. The EDE-Q demonstrated high internal consistency and test-retest reliability in prior work (Luce & Crowther, 1999); in our sample, Cronbach's α = .86.

2.3 Procedure

On the day preceding scheduled running events, runners were approached at the packet pick-up site and invited to participate in the study. Interested participants provided informed consent, and completed self-report questionnaires. All study procedures were approved by the University Institutional Review Board.

2.4 Analytic Plan

Bivariate correlations examined associations between variables of interest. Following missing data diagnosis, missing data were confirmed missing completely at random; missing data were imputed with maximum likelihood imputation, averaged over 11 imputations, if ≤ 10% of items were missing from the original item pool of the EDE-Q and CET. A full model predicting EDE-Q global scores was evaluated, with main and interaction effects for CET scores, WS, and gender; BMI and race length were entered as covariates. A one-way analysis of variance (ANOVA) evaluated between-group differences to confirm rationale for testing gender-specific regression models. With Hayes' PROCESS macro (Hayes, 2013), two sets of linear regression analyses, one per gender group, examined regressors for EDE-Q global scores, and whether relations between CE and ED pathology were moderated by WS; covariates in each model included BMI and race length (i.e., half- or full-marathon). Analyses were conducted with SPSS v. 26.

3. Results

3.1 Preliminary analyses.

Data were screened for univariate and multivariate normality and adherence to assumptions of homogeneity of variance; no variables warranted transformation. Males reported significantly higher CET total scores $F(1, 275) = 18.93$, $p < .001$, and WS, $F(1, 266) = 4.83$,

$p = .03$. Females reported significantly higher EDE-Q global scores $F(1, 258) = 11.49$, $p = .001$. Eighteen males and 18 females (5.6% of each gender group) demonstrated EDE-Q global scores above a clinical cut-off (i.e., 1.68 for males (Schaefer et al., 2018) and 2.3 for females (Mond, Hay, Rodgers, Owen, & Beumont, 2004)). Seventy-six females (59%) reported CET scores exceeding a proposed clinical cut-off of 15 (Meyer et al., 2016); while a clinical cut-off has not been published across gender, 47 males (38%) reported CET scores above this threshold. Ninety-four females and 101 males (70% of the full sample) reported WS > 7 pounds, a community-sample mean from prior work (Bodell et al., 2017). Descriptive statistics and bivariate correlations are presented in Table 1.

3.2 Regression analyses

Full model results indicated CET ($p = .01$), BMI ($p < .001$), and race length ($p < .001$), were significant predictors of EDE-Q global scores; there were no significant interaction effects. ANOVA results indicated significant between-gender differences in CET ($p < .001$), EDE-Q ($p = .001$), and WS ($p = .03$), confirming rationale for testing gender-specific regression models.

3.2.1 Female Model.

—The overall model was significant, $F(5, 123) = 10.84$, $p < .001$, accounting for 31% of EDE-Q score variance (Table 2). CET was significantly related to global EDE-Q, $b = .10$, $se(b) = .05$, $p = .04$. BMI, $b = .10$, $se(b) = .03$, $p < .001$ and race length, $b = -.51$, $se(b) = .16$, $p = .002$, were significant covariates. WS did not demonstrate significant main or interaction effects.

3.2.2 Male Model.

—The overall model was significant, $F(5, 118) = 11.82$, $p < .001$, accounting for 33% of EDE-Q score variance (Table 2). An interaction effect emerged for CET x WS, $b = .003$, $se(b) = .001$, $p = .02$, such that the relation between CET and EDE-Q scores was stronger among male runners who reported higher WS (Figure 1).

4. Discussion

Findings from this study inform the understanding of the association between CE and ED pathology in athletes by examining the importance of weight history in a mixed-gender sample of distance runners. Consistent with study hypotheses and recent research, CE was significantly associated with ED symptoms for both males and females. Additionally, on average, males reported significantly lower levels of CE and ED pathology and higher levels of WS. While a full model did not indicate significant moderation effects, results likely reflect insufficient power in the current sample to examine both 2- and 3-way interactions (Heo & Leon, 2010; McClelland & Judd, 1993). Examination of the impact of WS on the association between CE and ED pathology indicated differences when examined separately, within gender. For females, those with higher CET reported higher EDE-Q scores but WS did not impact the association between CE and ED pathology, suggesting that CE represents a risk factor for ED pathology regardless of weight history. In contrast, CE and ED pathology were more strongly related for male runners with higher levels of WS. Specifically, at lower levels of WS, men with *lower* CET scores endorsed greater eating pathology. In contrast, at higher levels of WS, males with higher CET scores also reported significantly elevated EDE-Q scores. Findings may reflect males who are motivated to exercise compulsively based upon a fear of returning to a higher weight, accompanied by elevated weight and shape concern. Males in the current study had significantly greater WS than their female counterparts; an important line of future inquiry may be to explore how weight history may motivate patterns in unhealthy exercise and eating behavior among men.

Overall, these results align with previous research indicating gender differences in relations between CE and ED pathology (Cunningham et al., 2016), as well as WS and ED pathology (Burnette et al., 2017). Findings further suggest that the impact of weight history on ED risk differs between male and female runners. In light of research indicating that men may experience unique risk factors for ED pathology (Chapman & Wood, 2016), results from the current study have important implications for gender-specific assessment and treatment of EDs in runners and suggest that WS is an important factor to consider when working with male athletes.

4.1 Limitations

This study is the first of its kind to specifically examine the impact of weight history and CE on ED pathology in a high-risk, leanness-focused, athlete sample. Strengths of this study include its sample size and ability to examine these relations specific to gender. Despite these strengths, the current study had limitations to note. First, while we controlled for race-length, we did not specifically account for training level; future work might include specific attention to athletic status (e.g., elite vs. recreational). Second, WS was calculated via self-report and as such, weight history in the current study was subject to recall bias. In addition, the questionnaire item that queried WS in the current study did not ask participants to exclude pregnancy or medical complications (e.g., use of prescription corticosteroids) in estimating their highest weight at adult height, which is counter to what has been recommended in prior work (e.g., Witt, Katterman, & Lowe, 2013). Finally, while the EDE-Q is an efficient measure that has demonstrated validity among female athletes (Pope, Gao, Bolter & Pritchard, 2015), it does not include specific assessment of muscle dysmorphia; use of a measure that captures this construct (e.g., Eating Pathology Symptoms Inventory; Forbush et al., 2013) may better inform future investigation of weight history, exercise and eating pathology in samples that include males.

4.2 Conclusions

In a mixed-gender sample of distance runners, females reported greater CE and eating pathology, whereas males reported higher levels of WS. For male runners, the association between CE and eating pathology was stronger among those with elevated WS. In contrast, regardless of weight history, female runners exhibiting CE behaviors demonstrated increased report of ED pathology. Results from the current study have important implications for the assessment and treatment of ED in runners, and suggest that WS is a critical factor to consider when working specifically with male athletes.

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Abbreviations:

ED	Eating disorder
CE	Compulsive exercise
WS	Weight suppression
BMI	body-mass index

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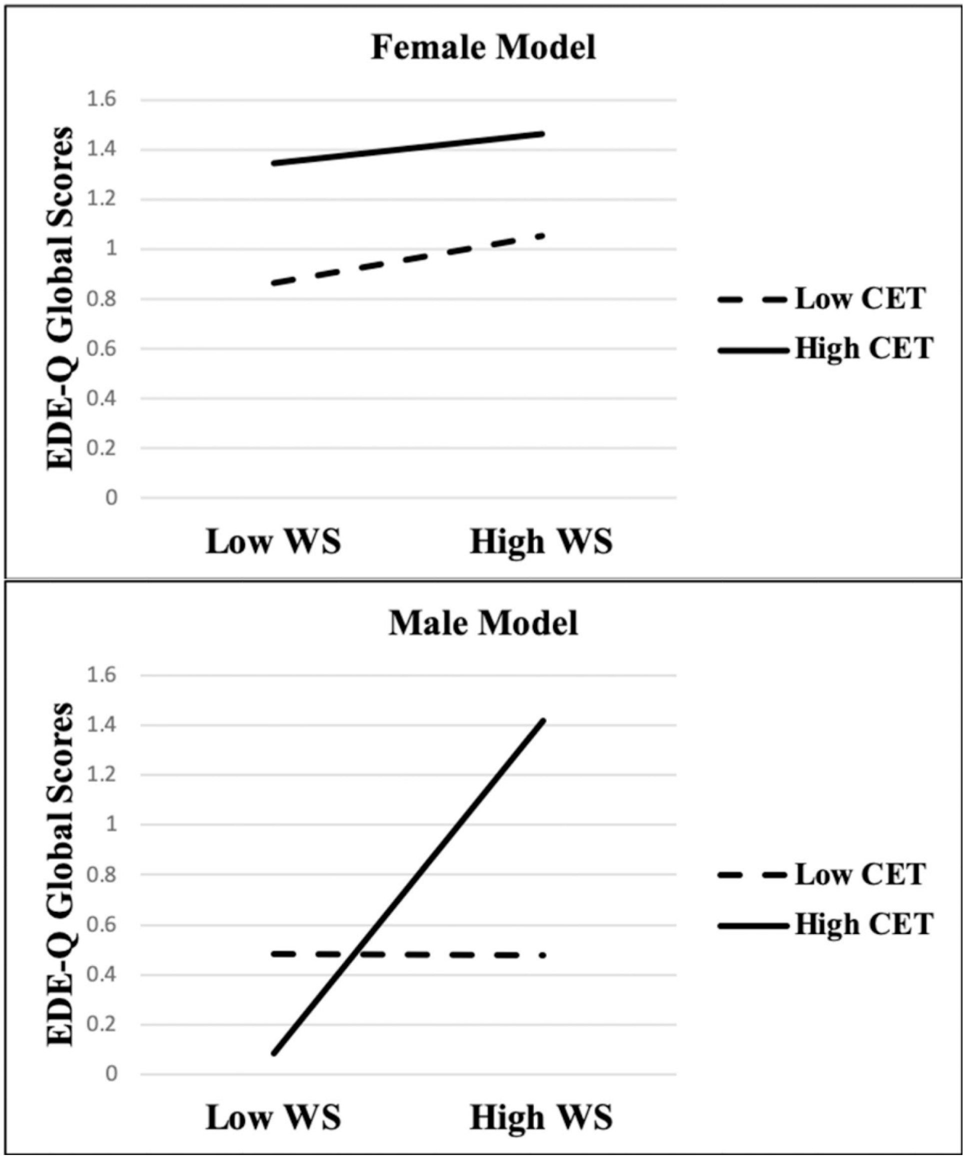

Figure 1.

Association between CET and EDE-Q moderated by WS

Note: CET = Compulsive Exercise Test total score; EDE-Q = Eating Disorder Examination- Questionnaire global score; WS = weight suppression

Table 1.

Descriptive Statistics and Bivariate Correlations for Variables of Interest

Variable	Range	*M (SD)*	Skewness (*SE*)
CET			
Females	8.03 – 21.00	15.21 (2.39)	−.22 (.20)
Males	7.93 – 20.66	13.89 (.23)	.12 (.23)
WS			
Females	.00 – 103.00	19.50 (17.97)	1.52 (.21)
Males	.00 – 136.00	25.04 (23.03)	1.76 (.21)
EDE-Q			
Females	.00 – 4.74	1.19 (.98)	1.00 (.21)
Males	.00 – 4.68	.81 (.82)	1.80 (.22)

Variable	1	2	3	4	5
1 CET	-	−.14	.27**	.11	−.09
2 WS	.14	-	.17	.26**	−.03
3 EDE-Q	.42**	.34**	-	.44**	−.37**
4 BMI	.14	.27**	.24**	-	−.36**
5 Race Length	−.17*	−.15	−.24**	−.24**	-

Note: WS = weight suppression; CET = total scores on Compulsive Exercise Test; EDE-Q = global scores on Eating Disorders Examination-Questionnaire; Race length = full v. half marathon. Correlations for Male sample are presented below the diagonal.

* Asterisk indicates significance of $p < .05$

** Double asterisk indicates significance of $p < .01$.

Table 2.

Female and Male Regression Models for EDE-Q Global Scores

Model	Variable	R^2	*F*	*b*	*SE(b)*	*t*	*p*	CI (95%)
Female (*n* =129)		.31	10.84				< .001[a]	
	CET			.10	.05	2.11	.04*	.007, .189
	WS			.01	.03	.39	.70	−.05, .07
	CET × WS			.00	.002	−.22	.83	−.004, .003
	BMI			.10	.03	3.56	< .001[a]	.05, .16
	Race Length			−.51	.16	−3.19	.001**	−.82, −.20
Male (*n* = 124)		.33	11.82				< .001[a]	
	CET			.05	.04	1.56	.12	−.01, .12
	WS			−.03	.02	−1.80	.07	−.07, .003
	CET × WS			.003	.001	2.34	.02*	.00, .005
	BMI			.04	.02	1.83	.07	−.003, .08
	Race Length			−.22	.14	−1.58	.11	−.50, .05

Note: EDE-Q = global scores on Eating Disorders Examination- Questionnaire; CET = total scores on Compulsive Exercise Test; WS = weight suppression; BMI = body mass index

*Asterisk indicates significance of $p < .05$

**Double asterisk indicates significance of $p < .01$

[a]Indicates significance of $p < .001$.