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2017

Accepted Manuscript

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PII: S1471-0153(16)30126-X
DOI: doi: 10.1016/j.eatbeh.2016.11.001
Reference: EATBEH 1130

To appear in: Eating Behaviors

Received date: 20 June 2016
Revised date: 6 November 2016
Accepted date: 9 November 2016

Please cite this article as: Egan, S.J., Bodill, K., Watson, H.J., Valentine, E., Shu, C. & Hagger, M.S., Compulsive exercise as a mediator between clinical perfectionism and eating pathology, Eating Behaviors (2016), doi: 10.1016/j.eatbeh.2016.11.001

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Compulsive exercise as a mediator between clinical perfectionism and eating pathology

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Abstract

The aim of this study was to examine whether compulsive exercise mediates the relationship between clinical perfectionism and eating pathology, based on the cognitive behavioral model of compulsive exercise. Participants were 368 adults who participated regularly in sport/exercise and completed online measures of perfectionism, compulsive exercise and eating disorders. In support of the well-established link between perfectionism and eating disorders, clinical perfectionism predicted eating pathology both directly and indirectly mediated by compulsive exercise. In addition, there were also direct effects of clinical perfectionism on the avoidance/rule-driven behavior, weight control, and mood improvement subscales of the Compulsive Exercise Test (CET). There was a direct effect of the CET weight control subscale on eating pathology and a negative direct effect of the CET subscale mood improvement on eating pathology. Findings lend support to the cognitive behavioral model of compulsive exercise in which clinical perfectionism is conceptualized as related to eating disorders directly and indirectly.
through the mediation of compulsive exercise. Compulsive exercise was also found to have a direct effect on eating disorders. Compulsive exercise may be a symptom of eating pathology, rather than an antecedent, however causal inferences could not be established given the correlational design. Longitudinal research using cross-lagged panel designs to examine a bidirectional relationship between compulsive exercise and eating disorders is needed.

**Keywords:** compulsive exercise; eating disorder; mediation; perfectionism

**Compulsive exercise as a mediator between clinical perfectionism and eating pathology**

Exercise increases prior to and during an eating disorder (Davis, Kennedy, Ralevski, & Dionne, 1994), and is a method of weight and shape control (Fairburn, Cooper, & Shafran, 2003a). Prevalence of eating disorders in athletes ranges from 18% in non-leanness (Sundgot-Borgen, 2003) to 47% in leanness sports (Torstveit, Rosenvinge, & Sundgot-Borgen, 2008), relative to 0.5 to 3% in the general population (Hagger & Chatzisarantis, 2005). A construct which is relevant to eating pathology is compulsive exercise, which is defined as continual rigid and extreme urges to exercise, and difficulty stopping, despite negative consequences, such as injury (Taranis, Touyz, & Meyer, 2011). Compulsive exercise is multidimensional and incorporates several domains including exercise to regulate emotions, compulsivity towards exercise, weight and shape driven exercise, and exercise rigidity (Meyer, Taranis, Goodwin, & Haycraft, 2011; Taranis et al., 2011). While some research has investigated
Compulsive exercise is important to understand given the links that have been demonstrated with eating pathology. The Compulsive Exercise Test (CET; Taranis et al., 2011) is correlated with the Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994) in competitive athletes (Plateau et al., 2014) and regular exercisers (Bodill, Watson, Kane, Hagger, & Egan, 2016). These findings support the link between compulsive exercise and eating pathology. Understanding compulsive exercise is important as it has been associated with higher eating disorder symptoms in both non-clinical (Elbourne & Chen, 2007; Goodwin, Haycraft, Taranis, & Meyer, 2011; Lipsey, Barton, Hulley, & Hill, 2006) and clinical eating disorder samples (Formby, Watson, Hilyard, Martin, & Egan, 2014; Shroff et al., 2006). Further, compulsive exercise is a risk factor for relapse in anorexia nervosa, and linked with higher suicidal behavior, treatment drop-out and longer hospital admissions (Formby et al., 2014; Meyer et al., 2011).

A cognitive behavioral model of compulsive exercise was put forward by Meyer et al. (2011) which suggests that compulsive exercise is more than a mere symptom of an eating disorder. Meyer et al. (2011) argued that compulsive exercise is associated with both perfectionism (Meyer et al., 2011; Taranis & Meyer, 2010) and eating disorders (Boyd, Abraham, & Luscombe, 2007; Davis, Blackmore, Katzman, & Fox, 2005). In the model, a link between perfectionism and eating pathology is proposed to be mediated by compulsive exercise, although a direct
link is also proposed. Meyer et al. (2011) suggest that compulsive exercise and perfectionism are theoretically linked due to the relationship between compulsive exercise, rigidity and self-criticism, which are central components of perfectionism.

Perfectionism has a strong association with eating disorders (Egan, Wade, & Shafran, 2011). Clinical perfectionism involves striving to achieve high standards despite adverse consequences and judging self-worth on achievement (Shafran, Cooper, & Fairburn, 2002). Clinical perfectionism is one of four key maintaining factors in the transdiagnostic model of eating disorders (Fairburn et al., 2003a). Perfectionism has also been measured on the Multidimensional Perfectionism Scales (FMPS; Frost, Marten, Lahart, & Rosenblate, 1990; HMPS; Hewitt & Flett, 1991) which are linked to eating pathology (Egan et al., 2011). Perfectionism has been found to be associated with compulsive exercise (Taranis & Meyer, 2010). Specifically, a range of studies have found individuals engaging in exercise with elevated perfectionism have higher eating pathology than those with lower perfectionism (e.g., Penniment & Egan, 2012). Perfectionism has been found to be the risk factor with the strongest effects on disordered eating attitudes in female athletes (Hopkinson & Lock, 2004). Further, increases in eating psychopathology in athletes have been found to be a result of increases in perfectionism (Shanmugam & Davies, 2015).

There has been little research examining the validity of Meyer et al.’s (2011) model. Only one study to date (McLaren et al., 2001) has examined whether compulsive exercise mediates the relationship between perfectionism and eating pathology. McLaren et al. (2001) found in a sample of female university students that compulsive exercise did not moderate the relationship between perfectionism and eating pathology and concluded both perfectionism and compulsive exercise are associated with eating pathology. When controlling for body mass index, the relationship between perfectionism and dietary restraint was partially mediated by compulsive exercise, accounting for some, but not all, of the relationship between perfectionism and eating pathology (McLaren et al., 2001). Further, only some of Meyer et al.’s dimensions of
compulsive exercise have been associated with eating pathology, particularly, exercise driven by rigid rules, avoidance of affective withdrawal symptoms, and exercise to control weight. The mood improvement factor has not received consistent support (Goodwin et al., 2011; Meyer et al., 2011; Plateau et al., 2014; Taranis et al., 2011; Taranis & Meyer, 2011).

The aim of this study was to examine whether compulsive exercise mediates the association between perfectionism and eating pathology. Mediation analysis is important as it provides formative evidence to inform interventions by identifying key targets for intervention and the pathways by which those interventions work (Hagger, Chan, Protogerou, & Chatzisarantis, 2016). This study will build on McLaren et al.’s (2001) findings by using measures which may account for more variance in eating pathology and a measure of clinical perfectionism. The rationale for examining the mediating role of compulsive exercise between perfectionism and eating disorders is based on the association between compulsive exercise and eating pathology (Elbourne & Chen, 2007; Formby et al., 2014; Goodwin et al., 2011; Lipsey et al., 2006; Shroff et al., 2006), perfectionism and compulsive exercise (Hopkinson & Lock, 2004; Penniment & Egan, 2012; Shanmugam & Davies, 2015; Taranis & Meyer, 2010) and perfectionism and eating disorders (Egan et al., 2011).

Our proposed model is presented in Figure 1. We predicted that a partial mediation model (Figure 1, solid lines) in which perfectionism predicted eating pathology directly and indirectly through compulsive exercise will better fit the data than a full mediation model in which the direct effect of perfectionism on eating pathology was fixed to be zero. Specifically, we hypothesized that the effects of clinical perfectionism and concern over mistakes on the compulsive exercise dimensions of avoidance and rule-driven behavior and weight control exercise will be positive and statistically significant. We also predicted that two of the subscales from the CET subscales, avoidance and rule driven behavior and weight control exercise, will have positive, statistically significant direct effects on eating pathology. We expected positive and significant
indirect effects of perfectionism on eating disorders through the avoidance and rule driven behavior and weight control exercise dimensions of the CET. We anticipate that the direct effect of mood improvement on eating pathology will not be statistically significant (Figure 1, broken line).

**Method**

**Participants**

The population was adults over the age of 18 years who participated in a diverse range of sport and exercise. The inclusion criterion of exercise twice or more per week was chosen to include people who exercised regularly but the criterion was relatively low as amount of exercise is not the defining feature of compulsive exercise (Adkins & Keel, 2004). Our definition of regular exercise was more stringent than Taranis et al. (2011) who defined regular exercise as some form of sport or exercise over the past 4 weeks.

The sample comprised 368 participants; 50% females, 37% males (gender was not reported by 13% of the sample), aged 18-65 years (M = 32.24, SD = 10.49). Participants engaged in a mean of 1.63 sports (SD = 1.14, range 1-9 sports). For participants who reported engaging in more than one sport, the first sport that they reported was recorded as the sport that they engaged in.

[INSERT TABLE 1 HERE]

**Procedure**
Participants were recruited online through non-random sampling via sporting organizations, for example triathlon and marathon running clubs and social networking. Once participants had consented to take part in the study, they were provided an online link the questionnaires. Participants were debriefed after the completion of the survey and provided with referral details for consumer information on eating disorders websites.

Measures

**Compulsive exercise.** The 24-item CET (Taranis et al., 2011) consists of five original subscales: Avoidance and rule-driven behavior (e.g. "I feel guilty if I miss an exercise session"); Weight control exercise (e.g. "I exercise to burn calories and lose weight"); Mood improvement (e.g. "I feel less anxious after I exercise"); Lack of exercise enjoyment (e.g., "I find exercise a chore") and Exercise rigidity (e.g. "I follow a set routine for my exercise"). In line with previous research (Bodill et al., 2016; Plateau et al., 2014), the two originally proposed CET subscales, lack of exercise enjoyment and exercise rigidity, were excluded.

**Eating pathology.** The EDE-Q (Fairburn & Beglin, 1994) is a widely used reliable and valid measure of eating pathology with four subscales: Restraint, Eating Concern, Weight Concern, and Shape Concern.

**Perfectionism.** The nine-item Concern over Mistakes (CM) subscale from the FMPS (Frost et al., 1990) was used to measure perfectionism. The subscale has good construct validity, internal consistency and test-retest reliability (Frost et al., 1990). The Clinical Perfectionism Questionnaire (CPQ; Fairburn, Cooper, & Shafran, 2003b) is a 12-item self-report measure of clinical perfectionism with good internal consistency and validity.
(Egan et al., 2016). This scale was used in addition to CM given the relevance of clinical perfectionism to eating disorders (Fairburn et al., 2003a).

Data Analysis

Prior to analysis, missing values were imputed using multiple imputation in SPSS version 23, values were imputed where less than 5% of values were missing. A structural equation model using a maximum likelihood method was conducted with Mplus version 7.31. The psychological variables were represented as latent variables indicated by multiple items from their questionnaire measures or, in the case of the EDE-Q, the four composite scales. Gender and age were control variables which predicted all other variables in the model. The hypothesized relations among the variables in the proposed model are summarized in Figure 1. At the measurement level, construct validity of the latent factors was established using the average variance extracted (AVE), which measures the amount of variance captured by a factor in relation to that due to measurement error, and composite reliability coefficient ($\rho$), which should exceed .50 and .70, respectively (Diamantopolous & Siguaw, 2000). Adequacy of the hypothesized model was established using the comparative fit index (CFI) and the Tucker-Lewis Index (TLI) index, with values approaching .90 typically considered appropriate for adequate model fit, the standardized root mean squared residuals (SRMSR) with a cutoff value of .08 representative of a well-fitting model, and the root mean squared error of approximation (RMSEA) and its 90% confidence intervals (CI90), with a cutoff value equal to or less than .05 and narrow confidence intervals indicative of an adequately-fitting model (Marsh, Hau, & Wen, 2004). Hypothesized mediation effects were tested by calculating indirect effects with bootstrapped standard errors.

[INSERT FIGURE 1 HERE]

Results
Preliminary Analyses

Factor correlations, average variance extracted and reliability coefficients for study variables are presented in Table 2. Prior to evaluating hypotheses in the structural equation model, we examined the solution estimates to ensure that the psychological constructs were sufficiently well defined. AVE was less than .50 for only one variable, the clinical perfectionism measure (CPQ). This is consistent with previous research which has revealed that this measure tends to have some low factor loadings resulting in low variance extracted for some items and attenuating the average (Egan et al., 2016; Stoeber & Damian, 2014). Composite reliability estimates were adequate for all factors (ρ > .70, see Table 2).

With respect to the CET (Taranis et al., 2011), we found weak correlations between two of the originally proposed subscales, lack of exercise enjoyment and exercise rigidity, and the CET total. These findings are similar to previous research (Bodill et al., 2016; Plateau et al., 2014) which has indicated a three-factor solution for the CET comprising avoidance/rule-driven behavior, mood improvement, and weight control exercise subscales, with the lack of exercise enjoyment and exercise rigidity subscales exhibiting weak correlations with the total CET score. The weak correlations indicate that these constructs do not adequately contribute to the overall concept of compulsive exercise. Consistent with these findings, we elected to drop these factors from our analysis and focused on the avoidance/rule-driven behavior, mood improvement, and weight control exercise subscales only.

Structural Equation Model
Standardized parameter estimates for the structural relations among the proposed model are given in Figure 2. Overall, the model indicated adequate fit across multiple criteria, CFI = .87, TLI = .86, RMSEA = .06, p < .001 (CI90 lower limit = .06; CI90 upper limit = .06), SRMSR = .07. In addition, the model accounted for a statistically significant amount of variance in the key dependent variables: eating pathology ($R^2 = .75, p < .001$), avoidance/rule-driven behavior ($R^2 = .37, p < .001$), weight control exercise ($R^2 = .21, p < .001$), and mood improvement ($R^2 = .13, p = .001$).

There were statistically significant direct positive effects of clinical perfectionism on avoidance/rule-driven behavior ($\beta = .55, p < .001$), weight control exercise ($\beta = .31, p < .001$), and mood improvement ($\beta = .34, p < .001$). There was also a statistically significant positive direct effect of concern over mistakes on weight control exercise ($\beta = .21, p = .016$). There was a statistically significant positive direct effect of weight control exercise on eating pathology ($\beta = .75, p < .001$), and a statistically significant negative direct effect of mood improvement on eating pathology ($\beta = -.18, p = .001$). There was also a statistically significant positive direct effect of clinical perfectionism on eating pathology ($\beta = .23, p < .001$). In terms of indirect effects, we found a statistically significant positive indirect effect of clinical perfectionism on eating pathology through weight control exercise ($\beta = .23, p < .001$). There was also a statistically significant negative indirect effect of clinical perfectionism on eating pathology through mood improvement ($\beta = -.06, p = .019$). There was therefore a statistically significant overall positive indirect effect of clinical perfectionism on eating pathology comprising the two indirect effects ($\beta = .16, p = .024$). There was also a statistically significant total effect of clinical perfectionism on eating pathology comprising the direct and indirect effects ($\beta = .39, p < .001$). We also found a statistically significant indirect effect of concern over mistakes on eating pathology via weight control exercise ($\beta = .15, p = .019$).
Discussion

We aimed to examine whether compulsive exercise mediated the relationship between perfectionism and eating pathology. Direct and indirect effects were found between clinical perfectionism and eating pathology through compulsive exercise. Specifically, there were direct effects of clinical perfectionism on avoidance/rule-driven behavior, weight control exercise, and mood improvement. There was also a direct effect of the weight control exercise subscale on eating pathology and a negative direct effect of the mood improvement subscale on eating pathology.

The direct effect of clinical perfectionism on avoidance/rule-driven behavior and weight control exercise, and mood improvement, suggests that clinical perfectionism is associated with the essential components of compulsive exercise and lends support to Meyer et al.’s (2011) model where perfectionism leads to compulsive exercise. We also found a direct effect of perfectionism measured by concern over mistakes on weight control but not the other two CET subscales, whereas clinical perfectionism had a direct effect on all CET subscales. Similarly, clinical perfectionism had a direct effect on eating pathology, which supports the transdiagnostic model (Fairburn et al., 2003a) while no direct relationship was found with concern over mistakes. Thus, clinical perfectionism explains more variance in compulsive exercise and eating pathology than concern over mistakes. Hence, the CPQ may be useful in future research given previous research has only used the multidimensional perfectionism measures (e.g., Plateau et al., 2014; Taranis et al., 2011).

The direct and indirect effects of clinical perfectionism on eating pathology is consistent with the well-established link between perfectionism and eating disorders (Egan et al., 2011; Taranis et al., 2011). These findings support our prediction of partial mediation and, given
there was not full mediation of the effect of compulsive exercise on perfectionism and eating disorders, perfectionism may also have a direct impact on eating disorders, independent of compulsive exercise. However, this direct effect may also be due to other extraneous mediators which were not measured and reflect other processes by which perfectionism relates to eating pathology independent of compulsive exercise.

The indirect effects of clinical perfectionism and concern over mistakes on eating pathology via the weight control subscale suggests that compulsive exercise has value in explaining the relationship between perfectionism and eating disorders as predicted by Meyer et al. (2011). It would be useful for future research to further validate Meyer and colleagues’ model in a clinical eating disorder sample, as generalizations regarding the model in clinical samples cannot be made given our sample was non-clinical. It would also be useful to determine whether interventions for both perfectionism and compulsive exercise may help to improve the efficacy of standard treatments for eating disorders, above the gains already found in efficacious treatments such as CBT-E (Fairburn et al., 2009) where clinical perfectionism is a focus of treatment if it is elevated. Future research could also examine the efficacy of specific stand-alone treatments for perfectionism (e.g., Egan et al., 2014), for compulsive exercise and perfectionism in eating disorders.

We found a direct effect of the weight control exercise subscale on eating pathology consistent with Meyer et al.’s (2011) model. These findings support previous research where weight control exercise has been found to be strongly associated with EDE-Q scores (Bodill et al., 2016; Plateau et al., 2014; Taranis et al., 2011), which is unsurprising given the subscale represents items measuring compulsive exercise being used as a method to control weight and shape.

The finding that the mood improvement subscale of the CET had a direct, negative effect on eating disorder symptoms is in contrast to previous research in athletes and non-athletes which has reported either no association (Taranis et al., 2011; Plateau et al., 2014; Taranis &
Meyer, 2011) or weak positive association (Goodwin et al., 2011) and is in the opposite direction to Meyer et al.’s prediction (2011). One interpretation is that individuals who engage in exercise who report that exercise does not improve mood already have an optimal level of positive and negative affect, which may be protective against eating pathology. In a recent study it was reported that while perfectionistic concerns positively predicted CET weight control, perfectionistic strivings positively predicted CET mood improvement (Madigan, Stoeber, & Passfield, 2016). Madigan et al. (2016) suggested that in contrast to engaging in exercise for weight control, exercising for mood improvement may have adaptive aspects, given perfectionistic strivings has been associated with positive outcomes and mood improvement has been found to be unrelated to disordered eating (Adkins & Keel, 2005). Further, given only one study has found a weak positive association between mood improvement and eating pathology (Goodwin et al., 2011) and all others have found no relationship (Plateau et al., 2014; Taranis et al., 2011; Taranis & Meyer, 2011) engaging in exercise to improve mood may not be a central feature of compulsive exercise.

**Strengths and Limitations**

The relatively large sample size is a strength as it permitted the use of confirmatory latent-variable analyses. The use of a measure of clinical perfectionism was also a strength as previous research has only used multidimensional perfectionism scales, and clinical perfectionism was found to have a stronger relationship with both compulsive exercise and eating pathology than concern over mistakes.

There are several limitations. Participant recruitment was not random or stratified which limits generalizability. Participants were recruited from sources known to the research group, particularly triathlon and marathon running clubs. The sample, therefore, comprised mainly of individuals who are actively involved in one particular type of athletic pursuit: individual endurance sports and particularly running. The
sample may, therefore, not be representative of individuals involved in sport and exercise in general. In addition, the study was cross-sectional, therefore the bidirectional relationship between eating pathology and compulsive exercise could not be assessed as this requires a longitudinal design. While we hypothesized that perfectionism would affect compulsive exercise, which would impact eating pathology, it could equally be argued that perfectionism influences eating pathology, which then influences compulsive exercise (i.e., compulsive exercise is a symptom of eating pathology, rather than an antecedent). Indeed, Meyer et al.’s (2011) model proposed bidirectional links between eating pathology and compulsive exercise. Meyer et al. (2011) argued that dieting and exercise reinforce one another due to social reinforcers which accompany the initial weight loss from regular exercise, which then intensifies eating, shape and weight concerns. Future research should measure perfectionism, compulsive exercise and eating pathology at two time points using a panel design and include cross-lagged effects to determine the bidirectional (reciprocal) relations between variables.

Conclusions

In summary, we found support for Meyer et al.’s (2011) model with clinical perfectionism being associated both directly to eating pathology and indirectly through compulsive exercise. The findings highlight the importance of research on targeting perfectionism in eating disorders, and further research on compulsive exercise.

Funding Acknowledgements

This research was supported by a grant from the School of Psychology and Speech Pathology, Curtin University
References


Figure 1. Proposed structural equation model illustrating a partial mediation model of effects among perfectionism, Compulsive Exercise Test, and eating disorder factors. Effects of gender and age as control variables on each variable in the model omitted for clarity. Broken lines illustrate pathways estimated but expected to be null or zero.
Figure 2. Standardized parameter estimates of a structural equation model of effects among perfectionism, CET, and eating disorder factors. Statistically significant indirect effects not shown in model: Clinical perfectionism → Weight control → Eating disorders (β = .231, p < .001); Clinical perfectionism → Mood improvement → Eating disorders (β = -.060, p = .019); Concern over mistakes → Weight control → Eating disorders (β = .158, p = .019). Effects of gender and age as control variables on each variable in the model not depicted in diagram: Gender → Clinical perfectionism (β = .272, p < .001); Gender → Concern over mistakes (β = .333, p < .001); Gender → Avoidance/Rule-driven behaviour (β = .058, p = .233); Gender → Weight control (β = -.125, p = .025); Gender → Mood improvement (β = -.024, p = .691); Gender → Eating disorders (β = .258, p < .001); Age → Clinical perfectionism (β = -.114, p = .034); Age → Concern over mistakes (β = -.096, p = .060); Age → Avoidance/Rule-driven behaviour (β = -.013, p = .777); Age → Weight control (β = -.031, p < .560); Age → Mood improvement (β = -.208, p < .001); Age → Eating disorders (β = -.041, p = .250).

* p < .05 ** p < .01 *** p < .001

Table 1

A description of sport and exercise participation in the sample (N = 368)

<table>
<thead>
<tr>
<th>Sport participation</th>
<th>Percentage (n%)</th>
</tr>
</thead>
</table>
Running 121 (33%)
Triathlon 70 (19%)
Ball sports* 59 (16%)
Gym 52 (14%)
Other** 20 (5%)
Cycling 14 (4%)
Dance, yoga 12 (3%)
Athletics 11 (3%)
Rowing, swimming 9 (2%)

Note: * Ball sports = volleyball, football, baseball, basketball, golf, rugby, squash, netball, cricket, hockey, tennis, badminton. ** Other = water polo, sailing, surf lifesaving, pilates, skateboarding, hula hooping, equestrian.

Table 2 Zero-Order Intercorrelations and Reliability Coefficients for Study Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>AVE</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Clinical perfectionism</td>
<td>.324</td>
<td>(.819)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Concern over mistakes</td>
<td>.514</td>
<td>.700***</td>
<td>(.904)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Avoidance/rule driven behavior</td>
<td>.562</td>
<td>.606***</td>
<td>.460***</td>
<td>(.909)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Weight control</td>
<td>.460</td>
<td>.426***</td>
<td>.389***</td>
<td>.500**</td>
<td>(.805)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Mood improvement</td>
<td>.438</td>
<td>.297***</td>
<td>.165**</td>
<td>.590***</td>
<td>.303***</td>
<td>(.793)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Eating disorders</td>
<td>.703</td>
<td>.535***</td>
<td>.468***</td>
<td>.431***</td>
<td>.779***</td>
<td>.117*</td>
<td>(.901)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Age</td>
<td>–</td>
<td>-.114*</td>
<td>-.096</td>
<td>-.081</td>
<td>-.086</td>
<td>0.239***</td>
<td>-.084</td>
<td>-.006</td>
<td>–</td>
</tr>
</tbody>
</table>
Note. AVE = Average variance extracted of latent factors. Correlations with psychological constructs are latent factor correlations. Composite reliability index (CRI) displayed on principal diagonal; *Dichotomous variable coded as 1 = Male, 2 = Female.

* p < .05  ** p < .01  *** p < .001
Highlights

- Structural equation modelling was used to examine a model of compulsive exercise.
- Clinical perfectionism predicted eating pathology and was mediated by compulsive exercise.
- Compulsive exercise had direct effects on eating pathology.
- The results support the cognitive-behavioural model of compulsive exercise.
- Research should investigate treatments for perfectionism and compulsive exercise.