Defence mechanism and coping strategy use associated with self-reported eating pathology in a non-clinical sample

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We examined the association of eating disorder traits (i.e., anorexia nervosa [AN] and bulimia nervosa [BN] traits) with the self-reported use of defence mechanisms and coping strategies. We also identified the specific mechanisms that best predicted eating disorder traits. The Eating Attitudes Test-40, Defense Style Questionnaire-40, and COPE scales were administered to a non-clinical sample of 429 students and staff. In general, individuals with elevated AN and BN traits reported using more immature/maladaptive defences and coping strategies. Linear regression models revealed that this association was more pronounced for defence mechanisms than coping strategies. High self-reported use of certain maladaptive defence mechanisms, particularly somatisation and displacement, most consistently predicted higher levels of self-reported AN and BN traits; coping strategies, however, were less frequently predictive of self-reported eating pathology. Some differences were also observed between males and females, particularly concerning levels of eating disorder traits. The results indicate that individuals reporting high levels of eating disorders are likely to display features of personality dysfunction; most prominently, high frequency use of maladaptive defences.

Keywords: anorexia nervosa, bulimia nervosa, coping strategies, defence mechanisms, eating disorders

BACKGROUND

Eating disorders are recognised as persistent disruption of regular eating behaviours, often intended to regulate or control weight that contributes to the debilitation of physical health or psychosocial functioning (Fairburn & Walsh, 2002). Eating disorders can exact a devastating physical toll with an estimated 5.10 deaths annually from anorexia nervosa (AN) and 1.70 deaths from bulimia nervosa (BN) per 1,000 people (Arcelus, Mitchell, Wales, & Nielsen, 2011). The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5; American Psychiatric Association [APA], 2013) defines the symptom profile of AN as an unwillingness to maintain body weight within a normal range, bearing an extreme fear of gaining weight, and giving body weight or shape unnecessary priority in self-evaluation. In turn, the DSM-5 symptom profile of BN includes both recurrent episodes of binge eating and recurrent behaviours to prevent weight gain (e.g., self-induced vomiting, misuse of laxative or other medications, or excessive exercise) after binge eating episode (APA, 2013).

The psychological processes of eating pathology have important implications for aetiology, assessment, and intervention. Accordingly, the personality characteristics of individuals with eating disorders have received empirical attention with poor interoceptive awareness, perfectionism, and obsessiveness found to be particularly salient examples (Leon, Fulkerson, Perry, & Early-Zald, 1995; Santonastaso, Friederici, & Favaro, 1999). Comparatively, little research exists however, on other psychological processes that can have clinical ramifications such as defence mechanisms and coping strategies employed by those with elevated levels of eating disorder traits. Additionally, this study examined which class of mechanism (i.e., defence or coping), and the specific psychological processes associated with each, had particular prominence in the prediction of eating pathology. This, to our knowledge, has been noticeably lacking in previous research, and is a gap in the literature the present study sought to address.

Defence mechanisms and coping strategies: A brief overview

Defence mechanisms refer to involuntary psychological processes that function to alter how stressful events are perceived, such that drastic changes in one's internal and external environment are significantly reduces (Vaillant, 1994). Defence mechanisms buffer the effects of excessive anxiety, enabling individuals to still somewhat function in these situations while preserving self-esteem (Cramer, 1987; Cramer, 2006). Similarly, coping mechanisms are behaviours intended to reduce or diminish psychological distress or stressful conditions (Fleishman, 1984). As Cramer (1998) notes, the key distinction between the two is that defence mechanisms ostensibly involve unconscious processes, while coping mechanisms employ purposeful, conscious processes. Thus, coping occurs with intentionality while defences presumably do not (McCrae, 1989).

Individual defence mechanisms can be grouped into a hierarchy of defence styles whereby some defences are considered more primitive and linked to personality while others are seen as more complex, healthy, and effective (Cramer, 1987). A common taxonomy is that of immature (i.e., maladaptive), neurotic (i.e., intermediate), and mature (i.e., adaptive) defence styles (Andrews, Singh, & Bond, 1993). Although coping mechanisms are not as readily hierarchically arranged, some effort has been made to group individual mechanisms into aggregates. For instance, 'avoidance coping' (C.J. Holahan, Moos, C.K. Holahan, Brennan, & Schutte, 2005) has several maladaptive correlates such as problem drinking and heightened negative psychological adjustment and physical health in men with prostate cancer (R. Moos, Brennan, Fondacaro, & B. Moos, 1990; Roesch et al., 2005).

Defence mechanisms, coping strategies, and eating pathology

Bouchard and Thériault (2003) proposed two models in an effort to explain the interrelationship of defences and coping mechanisms as their distinctions. Working from the field of marital adjustments, they termed their initial model the *independence hypothesis,* which emphasises the relative importance of defences and coping mechanisms without consideration of their level of adaptiveness. The independence hypothesis is based on the premise that coping mechanisms are more adaptive than defence mechanisms. Applied to eating pathology, this would suggest that defence mechanisms are positively related to AN and BN traits, and coping mechanisms, negatively related. Previous research elsewhere (e.g., Fulde, Junge, & Ahrens, 1995) has found defence mechanisms to better predict AN and BN traits than coping strategies.

Bouchard and Thériault's (2003) second model, termed the *effectiveness hypothesis,* takes adaptiveness into account arranging defences and coping mechanisms into four distinct groups: (i) maladaptive defence; (ii) maladaptive coping; (iii) adaptive defence; and, (iv) adaptive coping. In this model maladaptive defence mechanisms and adaptive coping mechanisms are equally unhealthy while adaptive defences and adaptive coping mechanisms are equally salutary. Applied to eating pathology, the effectiveness hypothesis would suggest that maladaptive defence and coping mechanisms should be associated with increased AN and BN traits, while adaptive defence and coping mechanisms should be negatively associated. Prior research elsewhere (e.g., Mayhew & Edelman, 1989; Steiger & Houle, 1991) has demonstrated that maladaptive strategies, in general, to be a better predictor of increased AN and BN traits than adaptive strategies.

In all, the limited research has indicated that individuals with an eating disorder diagnosis or at least prominent traits are more likely to employ maladaptive and immature defence mechanisms (e.g., denial, projection, and passive aggression) than individuals without eating pathology (Gothelf et al., 1995; Poikolainen, Kanerva, Marttunen, & Lönnqvist; Steiger, Goldstein, Mongrain, & van der Feen, 1990; Stein, Bronstein, & Weizman, 2003). Individuals high in eating disorder traits have not necessarily been diagnosed with an eating disorder, but display similar key attributes as diagnosed individuals. The extant literatures has also demonstrated that individuals with AN and BN diagnoses or traits more frequently employ maladaptive avoidance coping mechanisms (e.g., Koff & Sangani, 1997; Mayhew & Edelman, 1989; Troop, Holbrey, & Treasure, 1998; Troop, Holbrey, Trowler, & Treasure, 1994). To our knowledge, however, only one study has examined defence mechanisms, coping mechanisms, and eating disorders concurrently. Several years ago, Smith, Feldman, Nasserbakht, and Steiner (1990) examined the defence and coping mechanisms employed by individuals with AN near their diagnosis and six years post-diagnosis. As anticipated, they found that those with AN, who continued to have psychiatric problems at the six-year follow-up, used more immature defence mechanisms and avoidance coping mechanisms than those without eating disorders. BN diagnoses were not examined however, nor did the researchers attempt to establish which types of mechanism better predicted eating pathology.

Present study and hypotheses

Given the limited work examining coping and defence correlates of eating pathology, the current study examined the extent to which individuals with eating disorder traits reported using maladaptive defence and coping mechanisms with greater frequency than individuals who scored lower on eating disorder traits. Moreover, the study also identified which mechanisms (i.e., defence or coping) best predicted eating disordered traits. Accordingly, we proposed the following hypotheses:

1. High AN and BN traits will be positively associated with self-reported usage of maladaptive immature defence mechanisms.

2. High AN and BN traits will be positively associated with self-reported usage of individual coping mechanisms related to maladaptive avoidance coping, specifically behavioural disengagement, mental disengagement, and substance abuse.

3. Based on the independence hypothesis, it is anticipated that self-reported usage of selected defence mechanisms (i.e., those that demonstrated significant bivariate associations in previous analyses) will predict increased AN and BN symptoms. Conversely, self-reported usage of selected coping strategies will predict decreased AN and BN symptoms.

4. Based on the effectiveness hypothesis, it is anticipated that self-reported usage of selected maladaptive defence mechanisms and coping strategies will uniquely predict increased AN and BN traits. In turn, self-reported usage of selected adaptive defence mechanisms and coping strategies will uniquely predict decreased AN and BN traits.

5. Given the elevated base rate of eating pathology among females in epidemiological findings, it is anticipated that females will have higher levels of self-reported eating pathology than males and associated defences and coping strategies

METHOD

Participants

Participants included 429 university students and staff members (356 females, 71 males) from a Midwestern Canadian medical doctoral university. Participants' ages ranged from 17 to 57 ($M = 23$, $SD = 6.50$ years). They primarily self-identified as White (75%, $n = 319$), followed by Asian (11%, $n = 49$), Aboriginal (4%, $n = 17$), Black (3%, $n = 13$), or other (5%, $n = 22$). Respondents' body mass index (BMI) ranged from 15.60 to 53.20 ($M = 24.62$, $SD = 5.79$). Participants were recruited through the university's psychology research participant pool for course credit or the university's online bulletin.

Measures

Eating Attitudes Test-40 (EAT-40) and Eating Attitudes Test-26 (EAT-26). Permission was obtained to use the EAT-40 and EAT-26 scales in the present study. The EAT-40 (Garner & Garfinkel, 1979) and its shorter version, the EAT-26 (Garner, Olmsted, Bohr, & Garfinkel, 1982), are self-report inventories designed to measure participants' symptomatologies, behaviours, and attitudes associated with eating disorders. An overall eating pathology score, as well as three subscales scores (Dieting, Oral Control, Bulimia and Food Preoccupation) can be obtained. The subscales are based exclusively on 26-items used in the EAT-26 ($\alpha = .91$, 95% CI [.90, .92]). We administered the EAT-40 but only analysed the item content for the EAT-26 in order to examine the subscales. The subscales are listed as follows with internal consistency (Cronbach alpha) values from the present data reported for each. The 13-item Dieting subscale and 7-item Oral Control subscale assess traits commonly associated with AN, for example, 'I am terrified about being overweight.' (Dieting subscale; $\alpha = .85$, 95% CI = .83, .87) and 'I feel that others would prefer if I ate more.' (Oral Control subscale; $\alpha = .77$, 95% CI = .73, .80), while the Bulimia and Food Preoccupation subscale assesses traits commonly associated with BN (e.g., 'I vomit after I have eaten.' $\alpha = 81.95\%$ CI = .78, .83).

Items are rated on a 6-point scale Likert-type scale (1 = *always* to 6 = *never*). These responses, however, are scored using a 0–3 format, with problematic responses receiving a score of 1.2, or 3 based on severity, while the other non-problematic options (i.e., the other three response options) are scored 0. Item responses are summed with higher scores representing greater eating pathology (an overall score at or above 30 indicates high eating pathology), although item means are presented in the current study. Psychometric research (Garner et al., 1982; Garner & Garfinkel, 1979) has demonstrated support for the internal consistency of the EAT-40 overall score ($\alpha = .79$), Dieting subscale ($\alpha = .90$), Oral Control subscale ($\alpha = .83$), and Bulimia and Food Preoccupation subscale ($\alpha = .84$).

Defense Style Questionnaire-40 (DSQ-40). The DSQ-40 (Andrews, Singh, & Bond, 1993) is a 40-item self-report inventory designed to measure participants' defence styles and defence mechanisms through assessing the conscious derivatives of defence mechanisms (Bond, Gardner, Christian, & Sigal, 1983). An individual defence score for 20 defence mechanisms (two items for each individual defence), as well as three higher-order factor scores (mature, neurotic, immature) can be obtained. Sample items, such as 'Sticking to the task at hand keeps me from feeling depressed or anxious' (sublimation, mature) and, 'People say I tend to ignore unpleasant facts as if they didn't exist (denial, immature) are rated on a 9-point Likert scale (1 = *strongly disagree* to 9 = *strongly agree*). The score range from 2 to 18 for individual defences and vary based on the defence style (e.g., range from 24 to 216 for immature defences), with higher scores indicating greater usage of the defence mechanism. Final scores for each individual defence mechanism and defence style are calculated by computing the means of the relevant responses. In the present sample, adequate internal consistency reliability (Cronbach's alpha [95%]), based on Cicchetti et al. (2006) criteria, was obtained for the mature (α = .65 [.59, .69]); neurotic (α = .59 [.53, .65]); and, immature (α = .80 [.77, .83]) defence styles, consistent with past finding (Andrews et al., 1993; Ruutu et al., 2006). The defence mechanism scales, as opposed to the broader defence styles, were the primary foci of analysis in the present study.

COPE. The COPE (Carver, Scheier, & Weintraub, 1989) is a 60-item self-report inventory used to measure participants' individual coping mechanisms. Specifically, it measures a broad range of strategies that individuals may engage in when experiencing stress. The current study employed a dispositional format, such that individuals reported the extent in which they usually engage in the items listed when they are experiencing stress. COPE measures 15 individual coping mechanisms (four items per coping mechanism) listed as follows with internal consistency values (Cronbach alpha [95% CI]) from the present data computed for each: Focus on and Venting of Emotion (α = .81 [.78, .84]); Mental Disengagement (α = .38 [.28, .47]); Substance Use (α = .95 [.94, .96]); Denial (α = .80 [.77, .83]), Positive Reinterpretation and Growth (α = .76 [.72, .80]); Use of Instrumental Social Support (α = .85 [.83, .87]); Active Coping (α = .76 [.72, .79]); Religious Coping (α = .97[.96, .97]); Humour (α = .92 [.91, .93]); Restraint (α = .75[.71, .78]); Use of Emotional Social Support (α = .90[.89, .92]); Acceptance (α = .73[.69, .77]); Suppression of Competing Activities (α = .58[.51, .64]); and, Planning (α = .84[.82, .87]). Items are rated on a 4-point Likert-type scale (1 = *I usually don't do this at all; 4 = I usually do this a lot*). Sample items include: 'I get upset and let my emotions out' (Focus on and Venting of Emotion), 'I make a plan of action' (Planning), 'I laugh about the situation' (Humour), and 'I use alcohol or drugs to make myself feel better (Substance Use). Scores range from 4 to 16 for each coping mechanism with higher scores indicating greater use of the mechanism. Final scores for each individual coping mechanism are generated by computing the individual's item mean of the relevant responses. Psychometric research reported reliabilities of the subscales ranging from α = .45 – .92 (Carver et al., 1989).

Procedure

Participants completed the entire study online and anonymously using their own electronic devices at a time and location of their own discretion. The study title along with a link to the study was provided on the university's participant pool website and the university's online bulletin. Once participants clicked the provided link, they had access to the survey via FluidSurveys. Ethical approval was obtained from the Psychology Research Ethics Committee (Psy-REC #15-60), which is a fully-sanctioned Institutional Review Board designate of the university's Research Ethics Board. On the first page of the survey, participants were presented with consent information. They were informed that the study examined the association of distinct eating behaviours, personal attitudes, and responses to stress. Additionally, they were ensured anonymity, as well as confidentiality if anonymity was lost, and informed of the right to answer those questions that they feel comfortable with. They were informed that by completing and submitting the survey their consent was implied. On the following page, participants were first

presented with instructions for each questionnaire (demographic, EAT-40, DSQ-40, and COPE) that followed, and completed them accordingly. The order of the questionnaire was counterbalanced. At the top of each page, the scale of the questionnaire was displayed, as well as a progress bar to inform participants of how many pages they had left to complete. After participants submitted their data, a debriefing page was displayed. Participants were thanked, given further information of the nature of the study, and reassured of anonymity and confidentiality, should anonymity be lost. The study took less than 30 minutes.

Data analytic plan

Four primary sets of analyses were employed. First, we conducted an exploratory factor analysis (EFA) of the EAT-26 scale items to examine the factor structure of the scale in the present sample. The purpose was to extend prior research on the psychometric properties of the EAT-26 and to ascertain if the factor structure observed with the current sample paralleled that obtained in the construction sample (Garner et al., 1982). For this procedure, we used Mplus 7.4 (Muthén & Muthén, 2015), which employs default robust weighted least squares (WLSMV) estimation and Geomin rotation (an oblique method) to generate fit indices for EFA. The Comparative Fit Index (CFI) and Root Mean Residual Squared Error of Approximation (RMSEA) were computed to evaluate model fit and to compare fit across different factor solutions. Hu and Bentler (1999) note that CFI values around .95 and RMSEA values around .06 indicate acceptable model fit. EFA was used instead of confirmatory factor analysis (CFA) in order to obtain the best solution and fit generated by the data at hand, rather than attempting to force-fit the original EAT-26 scale structure to the data through CFA and potentially adding several parameters post hoc to maximise fit.

All remaining analyses were performed with SPSS 22.0. Second, bivariate associations were examined through correlating COPE and DSQ-40 scales with the EAT-26 AN scales and the BN scale. These analyses were intended to examine to what extent more maladaptive defence mechanisms and coping strategies were associated with increased self-reported eating pathology, and whether more salutary mechanisms and strategies were associated with decreased eating pathology. Correlations between continuous variables were evaluated in terms of effect size magnitudes of .10, .30, and .50, corresponding to effect sizes of small, medium, and large, respectively (Cohen, 1992). Third, three sets of regressions were computed in which linear combinations of defence mechanisms, coping strategies, or a combination of the two, were examined in the prediction of self-reported eating pathology (i.e., scores on the three EAT-26 subscales). Variables with significant bivariate relations with eating pathology were selected as candidate predictors in each of the regression models. As such, we based the regressions on theory in terms of how they were structured (re: the independence and effectiveness hypotheses) and the empirical results of the bivariate analyses to determine which predictors specifically were entered. This test would examine to what extent defence mechanisms and coping strategies were uniquely associated with self-reported eating pathology criteria as per the independence and effectiveness hypotheses.

Finally, we examined gender differences on each of the study measures through t-tests with Cohen's d computed to provide a measure of effect size, in which standardised mean differences values of .20, .50, and .80 correspond to effect sizes of small, medium, and large, respectively (Cohen, 1992). Missing data in the present study was extremely minimal with less than 0.3% of data points missing from the three study measures, and thus being 99.7% complete. As such, mean substitution was used as the most parsimonious method to estimate missing values for analysis.

RESULTS

Exploratory factor analysis of the EAT–26

Exploratory factor analysis was performed to identify latent constructs that underpinned the EAT-26 items and parallels in the factor structure in the factor structure between the current sample and the original construction sample. The Kaiser-Meyer-Olkin value of .923 met the acceptable threshold for factorability and three factors were extracted accounting for 47.2% of the total variance prior to rotation. The three-factor model generated acceptable fit to the data with CFI = .982 and RMSEA = .044 (95% CI = .037 to .050). Chi square difference tests demonstrated this model to be a significant improvement in fit beyond a tow factor model, χ^2 (24) = 168.49, p < .001, which in turn was an improvement over a one-factor model χ^2 (25) = 391.53, p < .001. As seen in the factor loading matrix (Table 1), each of the 26 items loaded on one of the three factors using a minimum loading criterion of .32, in which a given variable would account for a minimum of 10% of the variance in the factor upon which it load (Tabachnick & Fidell, 2007). In all, 16 of the 26 items (61.5%) loaded most highly on their original scale, while 21 of the 26 items (80.8%) loaded significantly on their targeted scale. Specifically, all 7 Oral Control subscale items loaded on the same factor, while 4 of the 6 Bulimia items loaded on a similarly named Dieting factor, with the remaining items cross loading. For continuity with past research, the original EAT-26 scale composition was retained for subsequent analyses.

Table 1

Exploratory Factor Analysis of EAT-26 Items: Factor Loading Matrix

Original scale loading	EAT-26 item	Current sample item loadings		
		Oral Control	Bulimia	Dieting
Dieting	I am terrified about being overweight.	.005	-.108	*.930**
Oral Control	I avoid eating when I am hungry.	*.580**	*.391**	.093
Bulimia	I find myself preoccupied with food.	*-.390**	*.923**	.008
Bulimia	I have gone on eating binges where I feel that I may not be able to stop.	.019	*.774**	-.036
Oral Control	I cut my food into small pieces.	*.490**	.007	.101
Dieting	I am aware of the calorie contents of foods that I eat.	-.103	.085	*.465**
Dieting	I particularly avoid food with a high carbohydrate content (i.e., bread, rice, potatoes, etc.).	*.365**	*.445**	.067
Oral Control	I feel that others would prefer if I ate more.	*.946**	-.027	-.045
Bulimia	I vomit after I have eaten.	*.751**	*.500**	*-.180**
Dieting	I feel extremely guilty after eating.	.245*	*.468**	.359*
Dieting	I am preoccupied with a desire to be thinner.	.005	.056	*.865**
Dieting	I think about burning up calories when I exercise.	-.003	.012	*.528**
Oral Control	Other people think that I am too thin.	*.919**	-.009	-.185
Dieting	I am preoccupied with the thought of having fat on my body.	.296*	.013	*.787**
Oral Control	I take longer than others to eat my meals.	*.599**	-.063	.058
Dieting	I avoid foods with sugar in them.	*.348**	*.610**	-.107
Dieting	I eat diet foods.	*.519**	*.494**	-.069
Bulimia	I feel that food controls my life.	.027	*.866**	.024
Oral Control	I display self-control around food.	*.322**	*-.249**	.029
Oral Control	I feel that others pressure me to eat.	*.847**	.026	.121
Bulimia	I give too much time and thought to food.	-.133	*.753**	.243*
Dieting	I feel uncomfortable after eating sweets.	.243*	*.554**	.145
Dieting	I engage in dieting behaviour.	*.362**	*.544**	.091
Dieting	I like my stomach to be empty.	*.651**	.252*	.208*
Dieting	I enjoy trying new rich foods	*.427**	*-.247**	.277*
Bulimia	I have the impulse to vomit after meals.	*.711**	*.376**	.004

Note: * denotes significant loadings; loadings are italicized for items loading on a given factor; loadings in bold for items loading highest on a given factor. Scale name associated with the original item loading are bolded for items that load on their target scale in both the current and original construction samples.

Defence mechanisms, coping strategies, and eating pathology

Bivariate associations. To test Hypothesis 1, Pearson correlation coefficients were computed to examine the associations between participants' dimensional scores for each individual maladaptive defence mechanism with the Dieting and Oral Control subscales (AN traits) and the Bulimia and Food Preoccupation subscale (BN traits). As seen in Table 2, Projection, Splitting, and Somatisation correlated significantly with all three scales, while Passive Aggression, Acting Out, Displacement, and Rationalisation (negatively) correlated significantly with the Dieting, Bulimia, and Food Preoccupation subscales. The observed correlations were small to moderate in magnitude, and indicate that elevated eating disorder traits are associated with an increased use of predominantly immature defence mechanisms. Among the neurotic defence mechanisms, Undoing was significantly positively correlated

with the Dieting, Bulimia, and Food Preoccupation subscales while Pseudo-altruism was significantly associated with increased Bulimia and Food Preoccupation scores. Finally, Humour was significantly inversely associated with the Dieting scale, and was the only mature defence mechanism to be significantly associated with EAT-26 scores. Otherwise, very few of the mature or neurotic defence mechanisms demonstrated significant associations with eating pathology.

To test Hypothesis 2, Pearson correlations were computed to examine associations between participants' dimensional COPE scores with AN and BN traits. Contrary to Hypothesis 2, Substance Use was not significantly correlated with any eating disorder traits, although significant positive correlations were found between Behavioural Disengagement and two out of three EAT-26 subscales, and Mental Disengagement with Bulimia and Food Preoccupation scores. The pattern of finding suggested a small but significant association between the presence of traits indicative of eating pathology with self-reported use of mental and behavioural coping strategies. Although not formally predicted as part of Hypothesis 2, Focus on and Venting of Emotion and Denial were significantly positively correlated with scores from Dieting and the Bulimia and Food Preoccupation subscales, while Positive Reinterpretation and Growth and Restraint were associated with lower Dieting scores; Religious Coping was associated with increased Bulimia and Food Preoccupation scores.

Table 2

Correlations between Eating Attitudes Test-26 with Defense Style Questionnaire-40 and COPE Scale Scores

Measures	Eating Attitudes Test-26		
	Oral Control	Dieting	Bulimia
Defense Styles Questionnire-40			
Mature	.04	-.05	-.02
Sublimation	.03	.03	.06
Humour	-.01	-.12*	-.08
Anticipation	.03	-.01	-.01
Suppression	.05	-.05	-.03
Neurotic	.09	.12**	.13**
Undoing	.05	.17***	.18***
Pseudo-altruism	.09	.08	.10*
Idealisation	.12	.06	.04
Reaction formation	-.03	.02	.01
Immature	.13**	.22***	.17***
Projection	.11*	.26***	.17***
Passive aggression	.05	.14**	.10*
Acting out	.06	.16***	.14**
Isolation	.09	.07	-.01
Devaluation	.03	.08	.01
Autistic fantasy	.03	.10*	.09
Denial	.02	.05	.03
Displacement	.09	.26***	.26***
Dissociation	.03	-.04	-.03
Splitting	.13**	.14***	.11*
Rationalisation	-.09	-.13**	-.12*
Somatisation	.13**	.24***	.24***
COPE			
Focus on and venting of emotion	.01	.18***	.17***
Mental disengagement	.04	.08	.12*
Behavioural disengagement	.01	.12*	.10*
Substance use	.04	.07	-.01
Denial	.07	.14**	.13**
Positive reinterpretation and growth	.01	-.11*	-.05
Use of instrumental social support	.01	.04	.10*
Active coping	.01	-.03	-.02
Religious coping	.11*	.08	.08
Humour	-.01	-.07	-.04
Restraint	.01	-.12*	-.08
Use of emotional social support	.00	.06	.09
Acceptance	-.05	-.07	-.06
Suppression of competing activities	.10	.02	.05
Planning	-.01	-.02	-.02

Note. * $p < .05$. ** $p < .01$, *** $p < .001$

Regression models. To examine the first component of the third hypothesis (defence mechanism prediction of eating pathology), the DSQ-40 scales with significant bivariate associations were entered simultaneously to predict AN and BN traits through separate sets of regressions. Given that the Dieting scale had considerably larger number of significant associations with the DSQ-40 and COPE subscales than the Oral Control subscale, these two subscales were examined as separate AN measures in the regression analyses. Predictors with unique associations of $p < .10$ or stronger were retained for testing in the final defence mechanism-coping strategy model (to test Hypothesis 4).

The final defence mechanism regression models are shown in Table 3 to illustrate the most informative predictors of eating pathology. The linear combination of defence mechanisms significantly predicted each of the eating disorder criteria, accounting for 3% to 4% of the total variance. Splitting usage uniquely predicted Oral Control (i.e., food restriction) while Projection and Displacement usage were significant unique predictors of increased Dieting scores (i.e., fear of becoming fat, preoccupation with thinness) uniquely predicted higher levels of BN traits. Across all three models, Somatisation usage predicted each eating pathology criterion at $p = .067$ to $.088$, and thus this variable was retained for testing in the final combined defence mechanism/coping strategy model.

To examine the second component of Hypothesis 3 (coping mechanism prediction of eating pathology), a regression was conducted in which selected COPE scales based on significant bivariate associations were entered simultaneously to predict eating disorder traits (Table 4). Religious Coping, as the only significant predictor of Oral Control, naturally predicted scores on this scale via regression, while Focus on and Venting of Emotion and Denial significantly predicted Dieting (with Positive Reinterpretation and Growth retained for the final model). The two former variables uniquely predicted BN traits. Overall, the models tended to be weaker predictors of eating pathology than the defence mechanism scales, accounting for 1% to 7% of the total variance.

To examine Hypothesis 4, three sets of hierarchical regression analyses were conducted to predict eating disorder traits through entering the defence mechanism and COPE predictors that uniquely predicted these criteria from the previous regression analyses. As shown in Table 5, the Somatisation and Splitting defence mechanism usage along with Religious Coping incrementally and uniquely predicted Oral Control scores, accounting for 4% of the variance. In turn, self-reported use of the defence mechanisms of Projection, Displacement, and Somatisation incrementally predicted increased Dieting scores, while Positive Reinterpretation and Growth uniquely predicted decreased Dieting scores, accounting for 14% of the variance. Finally, only Displacement and Somatisation defence mechanism usage significantly and uniquely predicted BN traits, accounting for 10% of the variance. These results suggest that, consistent with Hypothesis 4, selected maladaptive defence mechanism uniquely predicted increased AN and BN traits. Contrary to Hypothesis 4, however, maladaptive coping strategies tended to bear little association with self-reported eating pathology, although in some instances, healthy coping predicted decreased eating pathology, specifically, fears of gaining weight/preoccupation with thinness (i.e., Dieting scores).

Table 3

Regression: Prediction of Anorexia Nervosa Traits (Oral Control and Dieting), and Bulimia Nervosa Traits by Individual Defence Mechanisms

Oral Control (AN)			
Model predictors	B	β	p
Splitting	.11	.10	.049
Somatisation	.10	.10	*.067*
Projection	.05	.04	.442
(Constant)	1.58		
	$R = .03$, $F(3, 425) = 4.30$, $p = .005$		
Dieting (AN) model predictors			
Displacement	.38	.19	.001
Projection	.40	.18	.001
Autistic fantasy	–.16	–.10	*.075*
Somatisation	.17	.09	*.088*
Splitting	.16	.08	.110
Rationalisation	–.19	–.08	.127
Humour	–.13	–.06	.205
Passive aggression	–.12	–.05	.337
Undoing	.07	.03	.518
Acting out	.06	.03	.562
(Constant)	3.11		
	$R = .14$, $F(10, 418) = 6.87$, $p < .001$		
Bulimia nervosa (BN) model predictors			
Displacement	.18	.18	.001
Rationalisation	–.13	–.11	.033
Somatisation	.09	.10	*.072*
Splitting	.08	.08	.135
Passive aggression	–.10	–.08	.152
Pseudo-altruism	.08	.07	.161
Undoing	.07	.06	.242
Projection	.06	.05	.354
Acting out	.01	.01	.784
(Constant)	–.53		
	$R = .11$, $F(9, 419) = 5.92$, $p < .001$		

Note: Significant predictors and p-values in bold font. Predictors associated at $p < .10$ retained for combined model in italics.

Table 4

Regression: Prediction of Anorexia Nervosa Traits (Oral Control and Dieting), and Bulimia Nervosa Traits by Individual Coping Strategies

Oral Control (AN)			
Model predictors	B	β	p
Religious coping	.11	.11	.019
(Constant)	2.80		
	$R = .01$, $F(1, 427) = 5.53$, $p = .019$		
Dieting (AN) model predictors			
Focus on/venting of emotion	.35	.15	.002
Denial	.37	.13	.028
Positive reinterpretation and growth	-.28	-.08	.061
Restraint	-.23	-.08	.101
Behavioural disengagement	.05	.02	.799
(Constant)	6.30		
	$R = .07$, $F(5, 423) = 6.09$, $p < .001$		
Bulimia nervosa (BN) model predictors			
Focus on/venting of emotion	.16	.13	.016
Denial	.15	.10	.088
Mental disengagement	.09	.07	.213
Instrumental social support	.06	.05	.323
Behavioural disengagement	.00	.00	.981
(Constant)	-1.64		
	$R = .05$, $F(5, 423) = 4.27$, $p = .001$		

Note: Significant predictors and p-values in bold font. Predictors associated at $p < .10$ retained for combined model in italics.

Table 5

Regression: Prediction of Anorexia Nervosa Traits (Oral Control and Dieting), and Bulimia Nervosa Traits by a Combined Defence Mechanisms and Coping Strategies Model

Oral Control (AN)			
Model predictors	B	β	p
Somatisation	.11	.11	.021
Religious Coping	.10	.10	.035
Splitting	.11	.10	.042
(Constant)	1.14		
$R^2 = .04$, $F(3, 425) = 5.63$, $p = .001$			
Dieting (AN) model predictors			
Projection	.39	.18	.001
Displacement	.35	.17	.001
Somatisation	.19	.11	.042
Positive reinterpretation and growth	−.26	−.10	.042
Autistic fantasy	−.15	−.09	.072
Focus on/venting of emotion	.16	.07	.159
COPE Denial	.12	.04	.395
$R^2 = .14$, $F(7, 421) = 9.49$, $p < .001$			
Bulimia nervosa (BN) model predictors			
Displacement	.18	.17	.001
Somatisation	.12	.13	.014
Focus on/venting of emotion	.08	.07	.171
Rationalisation	−.08	−.06	.175
COPE Denial	.08	.06	.243
(Constant)	.62		
$R^2 = .10$, $F(5, 423) = 9.52$, $p < .001$			

Note: Significant predictors and p–values in bold font. Predictors associated at $p < .10$ retained for combined model in italics

Summary of predictive associations. Figure 1 provides a basic summary graphic illustration of the prediction effects across correlation and regression analyses for the number of statistically significant associations of individual defence mechanisms and coping strategies with eating disorder trait scores. As seen in this figure, the core maladaptive defence mechanisms of Somatisation, Projection, Displacement, and Splitting generated the largest number of significant associations with eating disorder traits. By contrast, the individual coping strategies were less frequently significantly associated with eating pathology across the range of correlation and regression analyses; the individual coping strategies were both less frequently predictive as well as simply having a smaller raw number of observed associations proportionate to the number of strategies measured and statistical tests conducted.
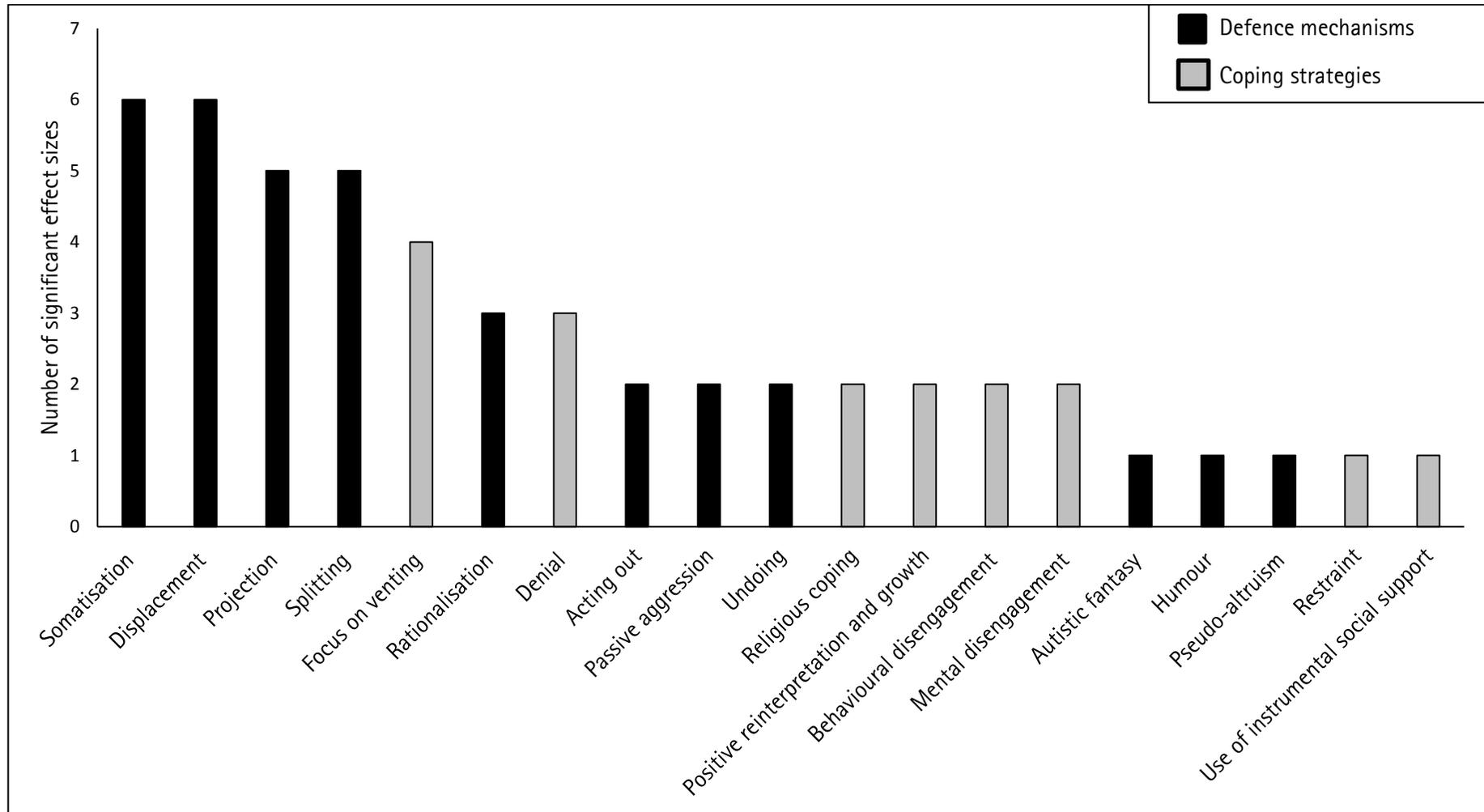
Figure 1. Graphic Illustration of Number of Statistically Significant Effect Sizes in the Prediction of Eating Disorder Traits for Individual Defence Mechanisms and Coping Strategies.

Gender comparisons on self–report measures

To compare differences between females and males on all self-report measures, a series of independent samples t-tests were conducted. Cohen's *d* was also computed as a measure of effect size (Table 6). For both genders, the mean scores for EAT-26 total and subscale scores were below the midpoints, indicating that participants had relatively low degrees of eating disorder traits. For the DSQ40, participants' mean scores were above the midpoint for mature defence mechanisms, approached the midpoint for most neurotic defences, and below the midpoint for immature defence mechanisms, indicating frequent self-reported use of mature defence mechanisms and relatively infrequent use of immature defences. In turn, participants' mean scores on individual coping strategies were below the midpoint for Behavioural Disengagement, Substance Use, Denial, Religious Coping, and Humour, suggesting infrequent self-reported use of these strategies relative to other coping mechanisms, which were above the midpoint.

Gender comparisons demonstrated that females scored significantly higher than males for eating disorder traits by nearly one-third to one-half of a standard deviation (*d* = .29 to .44), particularly on Dieting and Bulimia subscales, consistent with hypothesis 5; there were no differences in Oral Control. For many of the defence and coping mechanisms, differences between females and males were small and frequently non-significant; however, males reported significantly higher self-reported use of individual defence mechanisms that include humour, suppression, denial, dissociation, splitting, and rationalisation while females reported higher use of displacement and somatisation (i.e., the unique predictors of eating disorder traits). For the individual coping mechanisms, females self-reported greater use of Focus on and Venting of Emotion, Use of Instrumental Social Support, and Use of Emotional Social Support while male reported significantly greater use of Substance Use, Humour, Restraint, and Acceptance.

DISCUSSION

The present study examined the extent to which individuals with higher levels of eating disorder traits reported greater usage of maladaptive defence mechanisms and coping strategies compare to those reporting fewer eating disorder traits. This study also aimed to identify the specific mental mechanisms that best predicted eating disorder traits, which to our knowledge has been sparse in previous research. Consistent with study hypotheses, individuals with elevated AN and BN traits, broadly speaking, reported using more maladaptive defences and coping strategies; however, this association was more evident for defence mechanisms given the larger number of predictors which were also higher in their relative magnitudes of association. As per Hypotheses 3 and 4, maladaptive defence mechanisms, specifically displacement and somatisation, were the most consistent predictors of eating disorder traits, given that high self-reported use of these mechanisms most frequently incrementally predicted eating criteria after controlling for coping mechanisms and other associated defences. This was most evident for those AN traits associated with fears of gaining weight and preoccupation with thinness (i.e., Dieting subscale); however, pertaining to food restriction (i.e., Oral Control subscale), although somatisation was predictive, there were no largely predictive defence or coping mechanisms. Although self-reported use of maladaptive defence mechanisms were the strongest predictors, self-reported use of adaptive coping (i.e., positive reinterpretation and growth) also predicted decreased eating pathology concerning fears of gaining weight and preoccupation with thinness.

Some psychological processes of eating pathology

The finding that individuals with elevated AN and BN traits reported a high use of defences that can be subsumed by the maladaptive defence style more than those with fewer traits is consistent with previous research examining eating disorders and eating disorder traits (e.g. Steiger et al., 1990; Steiger & Houle, 1989; Stein et al., 2003; Steiner, 1989). While maladaptive individual defence mechanisms of projection and displacement have been linked to eating pathology in past research (Gothelf et al., 1995; Poikolainen et al., 2001), the present study also identified several other maladaptive individual defence mechanisms used more heavily by those with elevated AN and BN traits including somatisation, splitting, and passive aggression. These specific mechanisms have been less examined in previous literature, perhaps given that measures of individual defence mechanisms are highly variable.

Moreover, that individuals with certain elevated AN and BN traits are more inclined to report to use maladaptive avoidance coping (e.g., mental and behavioural disengagement) more frequently than individuals with lower traits is consistent with prior findings (e.g., Mayhew & Edelmann, 1989; Troop et al., 1994) although the associations observed in the present study were comparatively small in magnitude. While the present study conducted a more nuanced examination of specific coping strategies, such as the individual mechanism that underpin avoidance coping, previous research has tended to examine avoidance coping more broadly to generate larger in magnitude findings (Troop et al., 1994; Troop et al., 1998). In addition, other individual coping mechanisms that had somewhat greater use by those with elevated AN and BN traits, including Focus on and Venting of Emotion and Denial, may not be particularly adaptive. Carver et al. (1989), for instance, found that Focus on Venting of Emotion and Denial were moderately correlated with mental disengagement, behavioural disengagement, and substance use and inversely correlated with more adaptive strategies (e.g., active coping). It seems reasonable then that such defences may have maladaptive behavioural correlates.

The present study findings have implications regarding some possible psychological processes of eating pathology, although they do not exactly confirm or refute either of the models depicted earlier regarding the interrelations of defence and coping mechanisms. For instance, in terms of the independence hypothesis, it was demonstrated that selected defence mechanisms broadly predicted increased AN and BN traits while selected coping mechanisms seldom predicted decreased AN and BN traits (i.e., Positive Reinterpretation and Growth, exclusively for the AN trait of fear of gaining weight and preoccupation with thinness). In terms of the effectiveness hypothesis, it was found that certain individual maladaptive defence mechanisms uniquely and incrementally predicted increased AN and BN traits while one adaptive coping mechanism did not predict increases nor did adaptive defences predict decreases in AN and BN traits. It seems at least in the present sample, more weight may be afforded to the effectiveness model given that there were distinctions within defence mechanism usage to demonstrate that maladaptive defence mechanisms were unique from other defence styles, particularly, the adaptive defence style, in the prediction of eating disorder traits.

At a broad level, there seemed to be more similarities than differences in the psychological processes associated with higher levels of AN, specifically fear of gaining weight and preoccupation with thinness, and BN traits. While many defences and individual coping strategies emerged at the bivariate level, the immature defences of displacement and somatisation, most noticeably, uniquely and significantly predicted higher levels of most traits in the final regression model after controlling for relevant coping mechanisms and associated defences. In displacement, an impulse is behaviourally expressed toward a less threatening target; as this may apply to eating pathology, this may be conceptualised as punishment turned inward with the self-posing as more suitable and less threatening target than the original sources of the stressor, such as an authority figure (e.g., parent, supervisor).

Somatisation, however, refers to the generation of bodily reactions to stress, for instance, developing a physical illness or symptoms, which in turn may reflect an unresolved conflict. There is a highly visceral

component to eating disorders and this defence; an interpretation is that eating pathology is a somatic response to psychological conflicts and emotional disturbances expressed through behaviours such as restricting, purging, and weight fluctuation that characterise these syndromes. Clinical testimonials of patients with eating disorders attest to the restricting behaviours done to wrest some form of control (particularly for AN), in this case over one's own body, within the context of a chaotic world where they have little control over much else. On the other hand, it is possible that somatisation is simply a consequence of a high level of eating disorder traits, which naturally can lead to a host of physical symptoms and medical complications. Given the correlational nature of this research design one cannot know if these defences are a potential cause or consequence of eating disorder traits.

Although the present study featured a non-clinical sample, findings such as these can have clinical relevance. Arguably, any observed links between self-reported defence usage and eating disorder traits may be intensified if clinically diagnosed and non-diagnosed individuals were compared. Tentatively, the present study findings could suggest that individuals with AN and BN have indicators of personality pathology. A high self-reported use of certain maladaptive defence mechanisms may be risk factors for, or at the same time consequence of, eating disorders. On the other hand, the concordant self-reported use of adaptive defence and coping mechanisms, with the exception of Positive Interpretation and Growth for some AN traits, do not appear to be protective against eating pathology, at least not in the present sample. Given that individuals with eating disorders often report more stress that non-eating disordered individuals (Miller, 1988), interventions that focus on how patients manage stressful experiences would seem to be a natural intervention target. For instance, therapeutic foci that include decreasing reliance on maladaptive defence mechanisms, replacing these with more adaptive mental mechanisms, may have salutary effects. The benefit may lie less in the use of adaptive strategies than in relinquishing certain maladaptive strategies.

Limitations, future directions, and conclusions

There are some design features of the present study that constitute limitations such as the limited generalizability (e.g., owing to a predominantly White, female, young, university sample) that is endemic to university-based behavioural research, an inability to form causal inferences (i.e., due to the correlational nature of the design as already discussed), and the potential for impression management biases to influence results (i.e., owing to a reliance on self-report measures). Additionally, multiple ANOVA employed to analyse differences between females and males may have slightly increased the likelihood of Type 1 error. Nevertheless, two additional limitations specific to this research seem germane.

First, the present study did not receive approvals to solicit information from participants as to whether they had ever received a formal eating disorder diagnosis or to employ a clinical sample. Rather, linear relations were examined with dimensional measures of eating disordered traits, and potential theoretical, aetiological, and clinical influences were made from this. Although individuals with high eating disorder traits and those diagnosed with an eating disorder are not necessarily the same, we believe meaningful inferences can still be drawn, as similar findings to these have been obtained elsewhere when eating pathology has been operationalised at the trait-level or by clinical diagnosis (e.g., Stein et al., 2003; Steiner, 1989). A second potential limitation concerns the use of a self-report questionnaire to assess defence mechanisms; as defence mechanisms are ostensibly unconsciously activated, scholars dispute to what degree self-report questionnaires are a valid medium for assessing such domains of psychological functioning (Andrews et al., 1993; Cramer, 1998). Andrews et al. (1993) cogently argued that individuals are often aware (usually in hindsight) of the outcomes of operations of unconscious processes. Additionally, certain attitudes and beliefs are indicators of the consistent use of a defence because repeated usage of a defence mechanism could leave measurable traces in these systems (Andrews et al., 1993).

There are also several avenues for future directions in this area of research. First, one could certainly replicate and extend the study with individuals clinically diagnosed with AN and BN. Few studies, if any, have ascertained which personality features (e.g., defence or coping mechanisms) are a better predictor of eating disorder diagnosis or which may predispose someone to this. Thus, replicating this study with clinically diagnosed individuals could create therapeutically meaningful inroads. A second avenue involves employing a similar research paradigm but using different measures. As discussed above, self-report measures, particularly for defence mechanisms, are controversial. Future research may benefit from the use of projective tests, such as the thematic appreciation test, to assess defence mechanism usage. Such further strategies may help refine models that describe the relation between defence and coping strategies in terms of eating disorder traits to have achieved greater theoretical and clinical impact.

In conclusion, the present study collected quality data on a non-clinical university sample to inform the interrelations of defence mechanisms, coping strategies, and eating pathology. Research on eating pathology is crucial given that, compared to many other mental illness, individuals with eating disorders have one of the highest risks of premature death (Harris & Barraclough, 1998). Future applied research may inform clinical efforts to intervene with this important health problem.

Acknowledgement

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